

Global Health Action Special Volume 2009

Climate change and global health: linking science with policy

Heat, work and health: implications of climate change

Guest Editor: Tord Kjellstrom

Climate change and infectious diseases

Guest Editor: Rainer Sauerborn

Global Health Action

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CONTENTS

Foreword

Maria Neira, World Health Organization 4

Invited Editorial

Health as a crucial driver for climate policy
Rainer Sauerborn, Tord Kjellstrom and Maria Nilsson 5

Heat, work and health: implications of climate change

The thermal environment of the human being on the global scale
Gerd Jendritzky and Birger Tinz 10

Issues in health risk assessment of current and future heat extremes
Yasushi Honda and Masaji Ono 22

Comparing approaches for studying the effects of climate extremes – a case study of hospital admissions in Sweden during an extremely warm summer
Joacim Rocklöv and Bertil Forsberg 28

Maintaining health, comfort and productivity in heat waves
Ken Parsons 39

Workplace heat stress, health and productivity – an increasing challenge for low and middle-income countries during climate change
Tord Kjellstrom, Ingvar Holmer and Bruno Lemke 46

Effects of heat on workers' health and productivity in Taiwan
Ro-Ting Lin and Chang-Chuan Chan 52

Work-related heat stress concerns in automotive industries: a case study from Chennai, India
Ramalingam Ayyappan, Sambandam Sankar, Paramasivan Rajkumar and Kalpana Balakrishnan 58

Heat stress assessment among workers in a Nicaraguan sugarcane farm
Orlando Delgado Cortez 65

A pilot field evaluation on heat stress in sugarcane workers in Costa Rica: What to do next?
Jennifer Crowe, Berna van Wendel de Joode and Catharina Wesseling 71

The 'Hothaps' programme for assessing climate change impacts on occupational health and productivity: an invitation to carry out field studies
Tord Kjellstrom, Sabine Gabrysch, Bruno Lemke and Keith Dear 81

Climate change and infectious diseases

Climate change, its impact on human health in the Arctic and the public health response to threats of emerging infectious diseases
Alan J. Parkinson and Birgitta Evengård 88

Effects of climate change on tularaemia disease activity in Sweden
Patrik Rydén, Anders Sjöstedt and Anders Johansson 91

Milder winters in northern Scandinavia may contribute to larger outbreaks of haemorrhagic fever virus
Magnus Evander and Clas Ahlm 98

Local scale prediction of *Plasmodium falciparum* malaria transmission in an endemic region using temperature and rainfall
Yazoumé Yé, Moshe Hoshen, Catherine Kyobutungi, Valérie R. Louis and Rainer Sauerborn 103

Meningococcal disease and climate <i>Helena Palmgren</i>	116
Climate variability and increase in intensity and magnitude of dengue incidence in Singapore <i>Yien Ling Hii, Joacim Rocklöv, Nawi Ng, Choon Siang Tang, Fung Yin Pang and Rainer Sauerborn</i>	124
Climate impacts on environmental risks evaluated from space: a conceptual approach to the case of Rift Valley Fever in Senegal <i>Yves M. Tourre, Jean-Pierre Lacaux, Cécile Vignolles and Murielle Lafaye</i>	133
Vulnerability to episodes of extreme weather: Butajira, Ethiopia, 1998–1999 <i>Anders Emmelin, Mesganaw Fantahun, Yemane Berhane, Stig Wall and Peter Byass</i>	140
Using high spatial resolution remote sensing for risk mapping of malaria occurrence in the Nouna district, Burkina Faso <i>Peter Dambach, Ali Sié, Jean-Pierre Lacaux, Cécile Vignolles, Vanessa Machault and Rainer Sauerborn</i>	149
Cooking fuels and the push for cleaner alternatives: a case study from Burkina Faso <i>Shelby Yamamoto, Ali Sié and Rainer Sauerborn</i>	156
Climate change influences infectious diseases both in the Arctic and the tropics: joining the dots <i>Birgitta Evengård, and Rainer Sauerborn</i>	165
Climate change and population health in Africa: where are the scientists? <i>Peter Byass</i>	173

It is not by coincidence that this special volume on climate change and human health is being launched just before the COP-15, the crucial meeting of policy-makers from 192 countries, in Copenhagen from December 8th to 19th 2009, to negotiate future climate policy.

Although invoked frequently, the health argument has not been sufficiently firmly put forward, accompanied by hard evidence, as a driver for the climate policy community. Against this perspective of fostering dialogue between the global health and climate policy communities, the editors have chosen the COP-15 meeting as an occasion to present this special volume.

It is now almost 20 years since WHO published its first report on the health implications of climate change (1). Since that time, the Organisation has worked with researchers around the world to build the evidence base describing the linkages between climate change and health. In 2008, the 193 nations which constitute the World Health Assembly called for stronger action to respond to this emerging threat, and specified the need for more applied research. WHO has since coordinated an international consultation to define a global research agenda that responds to these needs (2). This agenda is action-oriented, stresses links with other, non-health, sectors and highlights the importance of research in and by low and middle-income countries.

This volume is an excellent contribution to such truly global, action and policy-oriented research. Firstly, the editors and authors highlight the needs for better and more scientific evidence on how climate change affects human health and what can and should be done about it. Secondly, this special volume goes well beyond health in a

narrow sense. For example, new evidence is provided of how heat and heat waves reduce not only health and well-being but also work productivity of farmers and industrial workers. Thirdly, the volume contains papers on infectious disease impacts in the Arctic and the tropics, and therefore generates an innovative global view of health impacts. Finally, I am particularly pleased to see such a strong interest and representation of authors from low and middle-income countries.

I trust it will trigger debates between health researchers and policy-makers and help to highlight the need for a consensus on effective climate policies. Without effective and equitable international action, we will be unable to protect future generations from the long-term adverse health effects of a changing climate.

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References

1. WHO. Potential health effects of climate change: report of a WHO Task Group. Geneva: World Health Organization; 1990. Available from: http://whqlibdoc.who.int/hq/1990/WHO_PEP_90_10.pdf [cited 5 October 2009].
2. WHO. Protecting health from climate change: global research priorities. Geneva: World Health Organization; 2009. Available from: <http://www.who.int/globalchange/publications/9789241598187/en/index.html> [cited 5 October 2009].

Health as a crucial driver for climate policy

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Health impacts of climate change and the need to prevent them should be at centre stage of the ongoing debate on climate policies (1). We have specifically prepared this series of papers to be available for the COP15¹ conference in Copenhagen, to which the world looks to agree on targets and procedures to reduce greenhouse gas (GHG) emissions on the basis of fair burden-sharing between high and low-income countries.

Fig. 1 illustrates how GHG emissions, health impacts and climate policies are linked through a cascade of causes and effects. It highlights, how evidence of health impacts (top right arrow) can and should influence the debate on mitigation and adaptation (bottom left arrow).

There are three strong reasons for the climate policy community to consider health impacts:

- 1) the impacts are large, increasing and inequitably distributed;
- 2) the majority of people everywhere are concerned about the protection of their own and their children's health and are hence prepared to support mitigation policies; and
- 3) certain mitigation policies have significant positive health 'co-benefits', and these should be quantified and promoted to support mitigation arguments.

We take up these three points below and conclude with suggestions to better link health research and climate policy.

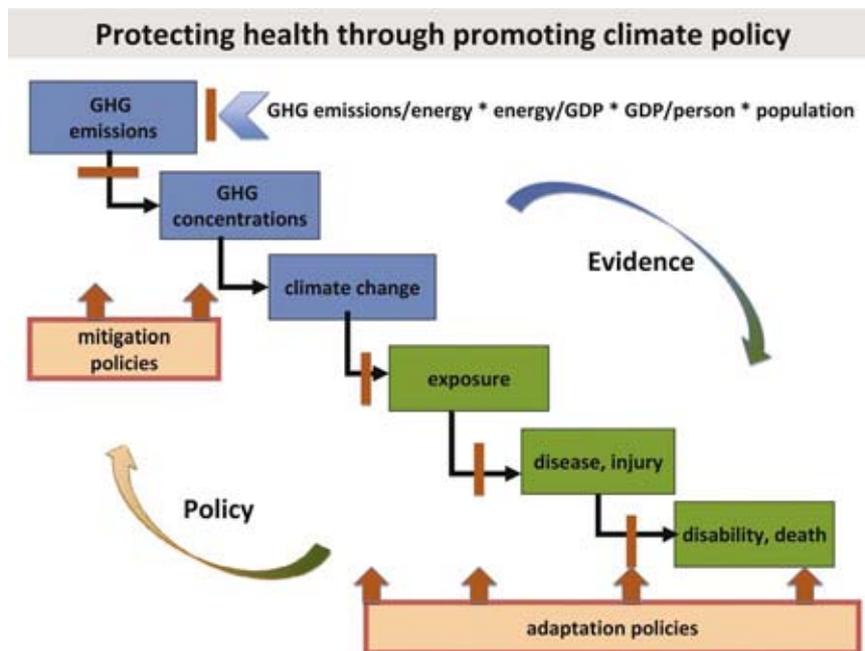


Fig. 1. Relationships between climate policy and health.

The factors in the upper part of the graph leading to GHG emissions are known as the 'Kaya identity', although the formula by Kaya and Yakobori (2) captures only energy-related emissions and was originally developed for CO₂ emissions. Put in words: How can we decarbonise energy production and increase energy efficiency while protecting economic growth particularly in low and middle-income countries for a growing population?

Health impacts are large, increasing and inequitably distributed

There are a large number of documented and imputed effects of climate change on health, operating through an even larger set of direct and indirect processes. We refer the reader to a recent review (8). An assessment by WHO in 2004 concluded that approximately 166,000 deaths occurred in the year 2000 due to climate changes that had occurred between 1990 and 2000 (3). An increase over time is very likely. Updated calculations of the global burden of disease are in progress and the Centre for Global Health Research at Umeå University will contribute to that work, which is intended to estimate current health impacts and forecast future health impacts of climate change up to 2030.

Furthermore, populations in low-income countries are the most vulnerable to adverse health effects of climate change. This raises major ethical and political concerns (4–7). There are a large number of documented and imputed effects of climate change on health, operating through an even larger set of direct and indirect processes. We refer the reader to a recent review (8).

Important in the mitigation debate, but discussed very little, is the major contribution due to health damage to estimates of the full economic costs of climate change on human welfare under any climate scenario. Stern (32) included health as one of the non-market climate impacts in one of his models. When those were added to his economic model of climate change impacts, the long-term economic impact up to the year 2200 increased from 7.3 to 13.8% of GDP per capita.

Health is a potentially excellent motivator for change behaviour and policies

The concern of citizens about their own and their children's health is arguably the most powerful motivator to accept changes in lifestyle or to accept the inconvenience and costs involved with climate policies. As Jay and Marmot (33) recently put it: 'Crucially for winning hearts and minds in richer countries, what is good for the climate is good for health'. Surprisingly, apart from some studies (from the high-income countries) on the individual or public willingness-to-pay (WTP) for climate policies, which reported a 50% increase in individual WTP for improved climate protection between 2003 and 2006 (34), we could not identify any significant literature on this issue. On the other hand, studies on the perception of climate change and individual behaviour change do exist, albeit mainly from the high-income countries (35).

Careful and science-based communication of the health risks of climate change and the co-benefits of climate policies may therefore be an important approach for convincing both lawmakers and the general public of the urgency of climate policy. Studies on which channels

and messages might be most effective in doing so would be welcome.

Mitigation policies will generate health 'co-benefits'

There are potentially large health co-benefits of mitigation policies, which should be entered into the economic calculations of the costs of mitigation. While such co-benefits have been amply documented to accrue in other sectors, such as agriculture, technology and forestry, the quantification of *health* co-benefits is at a particularly early stage and requires strong research effort and methodological development, such as proposed by Smith and Haigler (36). An innovative angle put forward by Yamamoto et al. (29) is to view policies for reducing biomass-burning for cooking as climate policies, which have huge health co-benefits. It is not widely known that indoor air pollution arising from cooking currently kills far more people than the consequences of outdoor air pollution.

The way forward

Fill the research gaps

Many knowledge gaps have been identified, for example, in the IPCC assessment report (37) as well as in other reviews (38, 39). The most recent international attempt to identify gaps and recommend future research was a meeting in Madrid in October 2008 organised by WHO, the United Nations Foundation, the US National Institute of Environmental Health Sciences and the Ministry of Health of Spain (40).

An example of an important gap is the lack of research on the impact of climate change-related increases in heat exposure on working people (14). Such impacts can be expected to have direct consequences for local economic development potential in exposed populations (9).

Although still small compared to other climate-relevant sectors, research on the climate–health nexus is rapidly evolving. Universities are beginning to move such research up their priority list and courses, both introductory and research oriented, are offered both in the high-income countries (for example at Umeå University, University of Heidelberg and Australian National University) and soon in the low and middle-income countries (for example, at BRAC University in Bangladesh). The involvement of researchers from low-income countries is still low (31), but growing, as reflected in this series of papers.

Develop and monitor adaptation strategies

Research evidence is still scant for answering some very policy-relevant questions, particularly in the context of developing countries: Which adaptation policies work best for which priority diseases and at what cost in a specific

country context? How do we best set up early warning systems? How should we protect health infrastructures? Which drugs and vaccines need accelerated research and development? Which current health interventions need to be delivered with much more impetus in view of climate change? How can we focus health policies and interventions on the most vulnerable groups? And finally, most importantly, but very mundanely, how can we strengthen health systems in general to face yet another challenge to population health?

'Use' health as a driver for global climate policies

Nilsson et al. (41) compared the development of climate policy with that of tobacco control. The main difference between the two health threats lies in the fact that climate change has effects on the globe's entire population. Yet, victims and perpetrators of climate change suffer in very different degrees. The main lesson learnt from tobacco control for Copenhagen is: do not wait! More than 50 years elapsed between the scientific evidence that smoking has strong negative health effects and the signing of the Framework Convention for Tobacco Control. Delay to act is deadly, as each year smoking claims 5.4 million lives (41, 42).

Our strong belief is that the world cannot afford to wait until all the evidence is in, as the smoking policy example has taught us. On the other hand, and opportunistically for the COP15 conference, we hope to bring the argument of protecting health through mitigation closer to centre stage in the mitigation debate. The question is as simple as it is crucial:

Will the policy-makers gathering in Copenhagen later this year live up to the challenge and agree on measures to effectively and fairly reduce GHG emissions, in order to protect our our children's health?

Overview of this special volume

The 23 articles in this special volume focus on two large groups of climate-exacerbated adverse health effects: the first is a direct effect, the second largely indirect.

- 1) The effects of *heat on human health*. These *direct* effects are potentially magnified by climate change. A particular perspective is that heat and humidity do not only act on human physiology, but also reduce work productivity, particularly in developing countries (9). The collection of articles on 'Heat and Health' focuses on direct human exposures to extreme heat, which will be an increasing condition in most of the world with climate change. Excessive heat exposure is a health risk for all age groups and the paper by Jendritzky and Tinz (10) shows with innovative maps the extent to which different parts of the world are now at risk and will be at greater risk in 2050. Honda and Ono (11) have developed an improved method to quantify heat-related mortality risks, and Rocklöv and Forsberg (12) compare different methods for quantifying mortality impacts during heat waves. Parsons presents practical approaches for reducing health risks during heat waves (13). The main focus in the other articles is on the vulnerable group of adults carrying out heavy labour in hot working environments: outdoors or indoors. Kjellstrom, Holmer and Lemke (14) describe the physiological mechanisms behind the health and productivity effects. Examples of these types of occupational health concerns are given by Lin and Chan (Taiwan) (15), Ayyappan, Sankar, Rajkumar and Balakrishnan (India) (16) and Delgado (Nicaragua) (17). Crowe, van Wendel de Joode and Wesseling (18) discuss in detail the possibilities of investigating such concerns in Costa Rica, and Kjellstrom, Gabrysch, Lemke and Dear (19) present the 'High Occupational Temperature Health And Productivity Suppression' (Hothaps) study programme, and invite interested scientists to participate. This programme will investigate global climate change impacts on heat and occupational health, a new concept publicised for the first time via this journal.
- 2) The effects on *infectious diseases*. These are indirect effects of climate change. Using a decidedly global lens, we report on increases in various infectious diseases both in the Arctic (Evengård and Parkinson (20), Rydén et al. (21) and Evander and Ahlm (22)) and in tropical countries (Yé et al. (23), Palmgren (24), Ling et al. (25), Tourre et al. (26), Emmelin et al. (27) and Dambach et al. (28)). The authors examine a wide range of diseases of parasitic, bacterial and viral origin. The focus, however, is on action for health systems: (i) assessing the dynamic, magnitude and nature of health impacts (22, 23); (ii) identifying most vulnerable populations (20, 26, 27); and (iii) contributing to the development of new tools for health systems for surveillance and early warning (21, 23, 25, 26, 28) The paper by Yamamoto et al. (29) examines the link between climate change and indoor air pollution, two seemingly unrelated public health threats. Evengård and Sauerborn (30) 'connect the dots' by pointing to a set of six common scientific and policy challenges in the Arctic and the tropics with regard to climate-sensitive infectious diseases. These papers thus stress common ground in research and policy challenges in what otherwise are extremely different settings: the cold high-income countries and the warm low and middle-income countries. Byass (31) finally reviews and portrays the dearth of research on climate

change and health in Africa and looks at ways of stimulating more work in this field, particularly by African scientists.

Note

1. COP means ‘Conference of the Parties’ and was created by Article 7 of the United Nations Framework Convention on Climate Change (UNFCCC), signed in Rio de Janeiro in 1992 and ratified by 193 countries. The COP is the ‘supreme body of this convention’ and mandated ‘to review the implementation of the Convention and any legal instrument . . . and shall make . . . the decisions necessary to promote the effective implementation of the Convention’. The conference convened in Copenhagen between December 8th and 19th 2009 will be the 15th such conference, hence COP15.

References

1. Lim V, Stubbs JW, Nahar N, et al. Politicians must heed health effects of climate change (Letter). *BMJ* 2009; 339: b3672.
2. Kaya Y, Yokobori K. Environment, energy, and economy: strategies for sustainability. Tokyo, Japan: United Nations University Press; 1998.
3. McMichael AJ, Campbell-Lendrum DH, Corvalan CF, Ebi KL, Githeko A, Scheraga JD, et al., editors. Climate change and human health – risks and responses. Geneva: WHO; 2003.
4. McMichael AJ, Campbell-Lendrum D, Kovats A, Edwards S, Wilkinson P, Wilson T, et al. Global climate change. In: Ezzati M, Lopez AD, Rodgers A, Murray CJL, eds. Comparative quantification of health risks. Global burden of diseases attributable to selected major risk factors. Geneva: World Health Organization; 2004.
5. Friel S, Marmot M, McMichael AJ, Kjellstrom T, Vågerö D. Global health equity and climate stabilization: a common agenda. *Lancet* 2008; 373: 1677–82.
6. Patz J, Campbell-Lendrum D, Gibbs H, Woodruff R. Health impact assessment of global climate change: expanding on comparative risk assessment approaches for policy making. *Annu Rev Public Health* 2008; 29: 27–39.
7. Campbell-Lendrum D, Corvalán C, Neira M. Global climate change: implications for public health policy. *Bull World Health Organ* 2007; 85: 161–244.
8. Costello A, Abbas M, Allen A, Ball S, Bell S, Bellamy R, et al. Managing the health effects of climate change: Lancet and University College London Institute for Global Health Commission. *Lancet* 2009; 373: 1693–733.
9. Kjellstrom T. Climate change, direct heat exposure, health and well-being in low and middle-income countries. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.1958
10. Jendritsky G, Tinz B. The thermal environment of the human being on the global scale. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2005
11. Honda Y, Ono M. Issues in health risk assessment for current and future heat extremes. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2043
12. Rocklöv J, Forsberg B. Comparing approaches for studying the effects of climate extremes – a case study of hospital admissions in Sweden during an extremely warm summer. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2034
13. Parsons K. Maintaining health, comfort and productivity in heat waves. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2057
14. Kjellstrom T, Holmer I, Lemke B. Workplace heat stress, health and productivity – an increasing challenge for low and middle-income countries during climate change. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2047
15. Lin R-T, Chan C-C. Effects of heat on workers’ health and productivity in Taiwan. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2024
16. Ayyappan R, Sankar S, Rajkumar P, Balakrishnan K. Work-related heat stress concerns in automotive industries: a case study from Chennai, India. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2060
17. Delgado CO. Heat stress assessment among workers in a Nicaraguan sugarcane farm. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2069
18. Crowe J, van Wendel de Joode B, Wesseling C. A pilot field evaluation on heat stress in sugarcane workers in Costa Rica: what to do next? *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2062
19. Kjellstrom T, Gabrysch S, Lemke B, Dear K. The ‘Hothaps’ programme for assessing the impacts of climate change on occupational health: an invitation to carry out field studies. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2082
20. Parkinson AJ, Evengård B. Climate change, its impact on human health in the Arctic and the public health response to threats of emerging infectious diseases. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2075
21. Rydén P, Sjöstedt A, Johansson A. Effects of climate change on tularaemia disease activity in Sweden. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2063
22. Evander M, Ahlm C. Milder winters in northern Scandinavia may contribute to larger outbreaks of hemorrhagic fever virus. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2020
23. Ye Y, Hoshen M, Kyobutungi C, Louis VR, Sauerborn R. Local scale prediction of *Plasmodium falciparum* malaria transmission in an endemic region using temperature and rainfall. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.1923
24. Palmgren H. Meningococcal disease and climate. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2061
25. Ling HY, Rocklöv J, Ng N, Sauerborn R, Siang TC, Yin PF. Climate variability and increase in intensity and magnitude of dengue incidence in Singapore. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2036
26. Tourre YM, Lacaux J-P, Vignolles C, Lafaye M. Climate impacts on environmental risks evaluated from space: a conceptual approach to the case of Rift Valley Fever in Senegal. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2053
27. Emmelin A, Fantahun M, Berhane Y, Wall S, Byass P. Vulnerability to episodes of extreme weather: Butajira, Ethiopia, 1998–1999. *Global Health Action* 2008. DOI: 10.3402/gha.v1i0.1829
28. Dambach P, Sié AN, Lacaux JP, Vignolles C, Machault V, Sauerborn R. Using high-spatial resolution remote sensing for risk mapping of malaria occurrence in the Nouna district, Burkina Faso. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2094
29. Yamamoto S, Sié A, Sauerborn R. Cooking fuels and the push for cleaner alternatives: a case study from Burkina Faso. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2088
30. Evengård B, Sauerborn R. Climate change influences infectious diseases both in the Arctic and in the tropics: joining the

- dots. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2106
31. Byass P. Climate change and population health in Africa: where are the scientists? *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2065
 32. Stern N. *The economics of climate change*. Cambridge: Cambridge University Press; 2007.
 33. Jay M, Marmot MG. Health and climate change (editorial). *BMJ* 2009; 339: b3669.
 34. Curry TE. A survey of public attitudes towards climate change and climate change mitigation technologies in the United States: analysis of 2006 results. Cambridge, MA: MIT, Laboratory for Energy and the Environment, Publication No LFEE 2007-01-WP; 2007.
 35. Semenza JC, Hall DE, Wilson DJ, Bontempo BD, Sailor DJ, George LA. Public perception of climate change voluntary mitigation and barriers to behavior change. *Am J Prev Med* 2008; 35: 479–87.
 36. Smith KR, Haigler E. Co-benefits of climate mitigation and health protection in energy systems: scoping methods. *Ann Rev Pub Health* 2008; 29: 11–25.
 37. Inter-Governmental Panel on Climate change. *Fourth Assessment Report IPCC, Volume 2: Impacts, Adaptation and Vulnerability*. Geneva, Cambridge: Cambridge University Press; 2007. Available from: www.ipcc.ch
 38. McMichael T. Environmental change, climate and population health: a challenge for inter-disciplinary research. *Environ Health Prev Med* 2008; 13: 183–6.
 39. Sauerborn R. Global environmental change-an agenda for research and teaching in public health – invited editorial. *Scand J Public Health* 2007; 35: 561–3.
 40. Neira M. Foreword. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2109
 41. Nilsson M, Beaglehole R, Sauerborn R. Climate policy: lessons from tobacco control. *Lancet* 2009; submitted.
 42. WHO report on the global tobacco epidemic, The MPOWER package. Geneva: World Health Organization; 2008.

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The thermal environment of the human being on the global scale

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Background: The close relationship between human health, performance, well-being and the thermal environment is obvious. Nevertheless, most studies of climate and climate change impacts show amazing shortcomings in the assessment of the environment. Populations living in different climates have different susceptibilities, due to socio-economic reasons, and different customary behavioural adaptations. The global distribution of risks of hazardous thermal exposure has not been analysed before.

Objective: To produce maps of the baseline and future bioclimate that allows a direct comparison of the differences in the vulnerability of populations to thermal stress across the world.

Design: The required climatological data fields are obtained from climate simulations with the global General Circulation Model ECHAM4 in T106-resolution. For the thermo-physiologically relevant assessment of these climate data a complete heat budget model of the human being, the 'Perceived Temperature' procedure has been applied which already comprises adaptation by clothing to a certain degree. Short-term physiological acclimatisation is considered via Health Related Assessment of the Thermal Environment.

Results: The global maps 1971–1980 (control run, assumed as baseline climate) show a pattern of thermal stress intensities as frequencies of heat. The heat load for people living in warm-humid climates is the highest. Climate change will lead to clear differences in health-related thermal stress between baseline climate and the future bioclimate 2041–2050 based on the 'business-as-usual' greenhouse gas scenario IS92a. The majority of the world's population will be faced with more frequent and more intense heat strain in spite of an assumed level of acclimatisation. Further adaptation measures are crucial in order to reduce the vulnerability of the populations.

Conclusions: This bioclimatology analysis provides a tool for various questions in climate and climate change impact research. Considerations of regional or local scale require climate simulations with higher resolution. As adaptation is the key term in understanding the role of climate/climate change for human health, performance and well-being, further research in this field is crucial.

Keywords: *climate impact research; thermal assessment; acclimatisation; heat load; cold stress; bioclimate mapping; Perceived Temperature*

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Climate and climate change is predominately considered based on standard climate parameters such as air temperature or precipitation in time and space. Even in epidemiology, most of the studies relate just air temperature to data of health outcome (e.g. 'temperature-related mortality') (1). This is remarkable since the relationship of human beings to the thermal environment requires a more comprehensive consideration. The term 'thermal environment' encompasses both the atmospheric heat exchanges with the body (thermal stress) and the body's physiological response (thermal strain).

The close relationship of humans to the thermal component of the atmospheric environment belongs to

everybody's daily experience. The hot summer of 2003 in Europe (2) when about 55,000 heat-related extra deaths occurred (3) is a good example of the significance of the thermal environment for human health (1, 4, 5). Balancing the human heat budget to variable environmental and metabolic heat loads is controlled by a very efficient (for healthy people) autonomous thermoregulatory system. This is supported by behavioural adaptation (e.g. eating and drinking, activity and resting, clothing, exposure, housing and migration) driven by conscious sensations of thermal discomfort. Such adaptation enables the human being to live and work in virtually any climate zone on Earth, albeit with varying degrees of discomfort and strain.

Internal heat is produced via metabolic activity required to perform mental and physical activities. The body exchanges heat by convection (sensible heat flux), conduction (contact with solids), evaporation (latent heat flux), radiation (long and short-wave) and respiration (latent and sensible). Thus, assessing the human thermal environment requires application of a complete heat budget model that takes all mechanisms of heat exchange into account (6–9).

Similar statistical relationships between mortality and thermal stress are found across the world (10, 11). However, populations living in different climates respond differently to heat stress due to some degree of physiological and behavioural acclimatisation. Fig. 1 shows, based on the application of a complete heat budget, that the impact of different thermal stress categories on relative mortality (defined as the number of deaths relative to the expected mortality) varies between European cities and areas (10). During days with no thermal stress or only slight deviation to the warm side (i.e. no thermal strain), small deviations of mortality from the expected value $EV = 100\%$ occur (Fig. 1). As thermal stress (heat or cold) becomes more extreme, mortality increases in different ways for ‘Normally cold’ places and ‘Normally hot’ places.

With the help of thermal assessment models, climatological information can be transformed into bioclimatological information. The climate data (observations at weather stations or output of numerical modelling) can be spatially presented in maps using geographical information system (GIS) techniques or grid data from model outputs. In this way, the bioclimate of heat and/or cold stress has been presented for New Zealand (12), Canada (13), Northern China (14) and Germany (15, 16). The German bioclimate maps were based on Fanger’s (6) Predicted Mean Vote

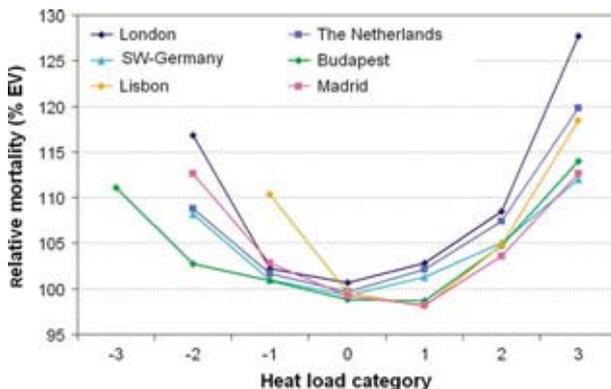


Fig. 1. Thermal stress-related mortality as deviation from the expected value for selected European cities and areas 1986–1996 during different thermal stress categories (after 36 and 37). –3, strong cold stress; –2, moderate cold stress; –1, slight cold stress; 0, thermal comfort (no thermal stress); 1, slight heat load; 2, moderate heat load; 3, strong heat load, taking acclimatisation into account.

(PMV)-equation using 30 years of hourly data from weather stations. A similar approach was used for Greece (17) and Austria (18). Some modern approaches were also combined for the human bioclimatic atlas for Mexico (19). Based on 30 years time series of three-hourly meteorological data of more than 900 weather stations across Europe, Perceived Temperature (PT) maps were produced presenting the frequency of heat load and cold stress using an acclimatisation approach (20).

In a first attempt to describe human thermal stress on a global scale the PMV-equation was applied to simulation data from the global General Circulation Model (GCM) ECHAM4/T106 of the Max Planck-Institute for Meteorology Hamburg/Germany (21). However, the PMV approach underestimated the role of humidity in warm conditions. Recently, global maps of the distribution of seasonal mean values of the Physiological Equivalent Temperature (PET) (22) for the current and a future climate were published (23). These are an improvement, but from a health impact point of view the frequency of exceeding thresholds may be more appropriate than the mean PET values. This report will demonstrate maps of the global distribution of threshold exceeding using improved thermal stress estimates.

Materials and methods

The thermal assessment procedure

The heat exchange between the human body and the thermal environment can be described in the form of the energy balance equation, an application of the first theorem of thermodynamics applied to the body’s heat sources (metabolism and environmental) and the various avenues of heat loss to environment (see Method box 1) (24).

Among the advanced heat budget models, Fanger’s PMV-equation (6, 26) can be considered as appropriate if Gagge et al.’s (27) improvement in the description of latent heat fluxes by the introduction of PMV^* is applied. Together with a radiation model this approach combines the operational thermal assessment procedure ‘Klima-Michel’-model (KMM) (28–30) of the German national weather service Deutscher Wetterdienst (DWD) with the output parameter (descriptive term) ‘Perceived Temperature, PT’ (31). PT follows the PET approach (22, 32), Standard Effective Temperature (SET) (27), or Outdoor Standard Effective Temperature (Out_SET) (33, 34) in order to achieve a descriptive term in °C. PT is defined as the air temperature of a standard environment (wind calm, air temperature = mean radiant temperature, relative humidity (RH) = 50%, metabolic rate 2.3 MET = 135 W m^{-2} , which means walking at 4 km h^{-1}) that would produce the same thermal stress as the actual environment (Table 1). Additionally, optimal behavioural adaptation by clothing (along the scale from the thermal

Method box 1. Heat exchange

$$M - W - [Q_H(T_a, v) + Q^*(T_{mrt})] - [Q_L(e, v) + Q_{SW}(e, v)] - Q_{Re}(T_a, e) \pm S = 0 \quad (1)$$

M metabolic rate (activity)
W mechanical power
S storage (change in heat content of the body)

Peripheral (skin) heat exchanges

Q_H turbulent flux of sensible heat
*Q** radiation budget
Q_L turbulent flux of latent heat (diffusion of water vapour)
Q_{SW} turbulent flux of latent heat (sweat evaporation)

Respiratory heat exchanges

Q_{Re} respiratory heat flux (sensible and latent)

Thermal environmental parameters

T_a air temperature
T_{mrt} mean radiant temperature
v wind velocity relative to the body
e water vapour pressure

The meteorological input variables include air temperature *T_a*, water vapour pressure *e*, wind velocity *v* and mean radiant temperature *T_{mrt}* including short and long-wave radiation fluxes, in addition to metabolic rate and clothing insulation. In Eq. (1) the appropriate meteorological variables are attached to the relevant energy fluxes in W/m². The physiological (internal) variables, such as the temperature of the core or the skin, the sweat rate, and the skin wetness, which all interact with the environmental heat conditions, are not mentioned here. An overview on the basics in thermo-physiology and heat exchange modelling with respect to adaptation is given e.g. in (25).

resistance of summer clothing (0.5 clo) to winter clothing (1.75 clo) (1 clo = 0.155 Km²W⁻¹) is considered based on ISO 9920 (35). It is assumed that people adapt their behaviour in order to gain comfortable conditions. The PT scale (Table 1) refers to the ASHRAE-Scale (ISO 7730) (26) extended to a nine-step scale.

The PT procedure incorporates the acclimatisation approach Health Related Assessment of the Thermal Environment (HeRATE) (36). HeRATE is a conceptual model of short-term acclimatisation based on findings in adaptation studies. The procedure modifies the absolute PT thresholds of Table 1 by superimposing the (relative) historic experience of the population in terms of PT of the previous weeks. Using the example of Lisbon 2003, Fig. 2 shows how the actual thresholds for the different thermal stress categories change from the initial values of Table 1. This procedure has the advantage that the index can be used without modification in different climate regions and during different times of the year without the need to artificially define seasons and calibrate it to a particular locale.

Climatological input

For global bioclimate maps no sufficient observational data base is available. However, the necessary meteorological input data for the global scale can be taken either from global GCMs or from reanalyses of Numerical Weather Prediction (NWP) models (e.g. European Centre for Medium-Range Weather Forecasts (ECMWF), Reading/UK, or National Centers for Environmental Prediction (NCEP), Camp Springs/USA). In this investigation we use two runs of the global GCM ECHAM4 (38) at the relatively high resolution T106 (39), about 100 km grid-point distance in middle latitudes (see Method box 2).

Due to restricted computer power, only two ‘time slices’ with a length of 10 years each are available in the high-resolution version of the GCM. In the first experi-

Table 1. Perceived Temperature, Predicted Mean Vote (According to (6)), and corresponding thermal stress (Initial conditions without acclimatisation; negative values are ‘cold stress’ and positive values are ‘heat load’)

Thermal stress category	Perceived Temperature (PT) in °C	Predicted Mean Vote (PMV)	Thermo-physiological stress
4	PT ≥ +38	Very hot	Extreme heat load
3	+32 ≤ PT < +38	Hot	Strong heat load
2	+26 ≤ PT < +32	Warm	Moderate heat load
1	+20 ≤ PT < +26	Slightly warm	Slight heat load
0	0 < PT < +20	Comfortable	None
-1	-13 < PT ≤ 0	Slightly cool	Slight cold stress
-2	-26 < PT ≤ -13	Cool	Moderate cold stress
-3	-39 < PT ≤ -26	Cold	Strong cold stress
-4	PT ≤ -39	Very cold	Extreme cold stress

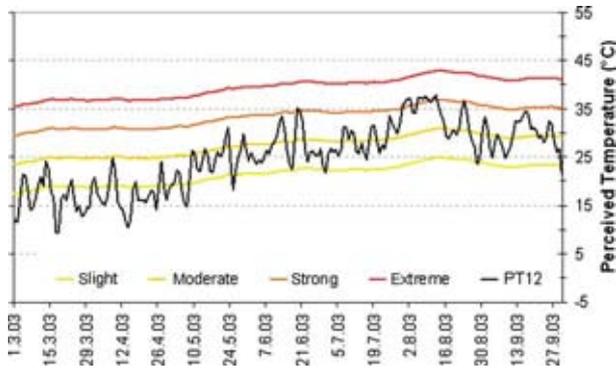


Fig. 2. Example of daily values of Perceived Temperature at 12 UTC (black line) for the weather station Lisbon (Portugal) from March to September 2003 (36). Compared to Table 1 the coloured lines indicate the variable thresholds for the different thermal stress categories specifying acclimatisation.

ment (control run) the concentration of greenhouse gases is set constant at the observed level 1971–1980 (recent climate conditions). In the second run the concentration of greenhouse gases is taken from the IPCC-scenario IS92a ‘business-as-usual’ (40) for the period 2041–2050. This scenario is between the two middle range scenarios A1B and A2 used in the report of the Intergovernmental Panel on Climate Change (IPCC) (41).

Calculation of the thermal environment

Using the model output data of ECHAM4/T106, the values of PT were calculated for the different time periods for each land-grid point and four Universal Times (UTC) at 00.00, 06.00, 12.00 and 18.00. In order to obtain comparable PT values, the predominant effect of the daily thermal variation was considered by recalculating corrected PT values for 12 Mean Local Time (MLT) for all grid points by a nonlinear interpolation procedure

Method box 2. ECHAM4-details

The global GCM ECHAM4 applies the relatively high resolution T106, about 100 km grid-point distance in middle latitudes (39). In both experiments (time slices), the surface of the earth is represented by 320×160 grid points which means a distance of about 1.1° in zonal and meridional direction. The GCM-data are available at fixed times 00.00, 06.00, 12.00 and 18.00 Universal Time (UTC). The meteorological variables required for the assessment are air temperature T_a , dew point T_d (to derive water vapour pressure) and wind velocity v , which are directly used to calculate the sensible and latent heat flux. In addition to these, for the parameterisation of the long-wave and short-wave radiant fluxes, cloudiness information such as total cloud cover N and cloud cover in the different tropospheric levels (low, middle and high) are used to calculate mean radiant temperature T_{mrt} .

centred around the four neighbouring PT values at the given UTC times. For the given six-hour intervals the maximum mean error of this procedure is in the order of 1 K ($=1^\circ\text{C}$), which was tested with observational data of a couple of European weather stations with a time resolution of one hour.

The maps of PT values are related to people staying outdoors at noon (12 MLT). Because PT_{max} usually occurs one or two hours later, the PT value at 12 MLT can be considered as a mean value over a certain time period starting some time before 12 MLT until some time after the same PT value as at 12 MLT occurs once more in the afternoon.

From these 12 MLT PT-time-series at every land-grid point the mean annual frequency (number of days p.a.) of comfort conditions (no thermal stress) or different intensities of thermal stress, respectively, has been derived for the two ECHAM4 time slices 1971–1980 and 2041–2050, taking acclimatisation into account according to HeRATE. The actual PT value for a certain thermal stress level at a given point and a given date always depends on the previous thermal conditions. The difference between the time periods (2041–2050) and (1971–1980) can be interpreted as the change in the bioclimate.

Results

The current thermal conditions

The map of the frequency distribution of thermal comfort (i.e. no thermal stress) 1971–1980 shows that the mid-latitude regions are most comfortable, in particular in the maritime affected areas (Fig. 3). This is true for Western Europe, New Zealand, the southern parts of Australia, Chile and Argentina as well as a narrow coast strip in the west of North-America where 300 days with thermal comfort at 12 MLT can be taken as typical. Similar conditions can be found in higher elevations of the Andes and in the area of the foothills of the Himalayas. With increasing continental influence in mid-latitudes or increasing subtropical influence, the number of days with comfort conditions decreases considerably. In the lowlands of the humid tropics thermal comfort is not possible at 12 MLT. This is also true for the ice shield regions of Antarctica and Greenland due to the limit to maximum clothing insulation of 1.75 clo which is used here.

The general relationship of heat load conditions to latitude (solar climate) is evident (Fig. 4a), although it is influenced by altitude. Heat load is the predominant thermal state in the tropics throughout the year. With increasing latitude the heat load probability declines, but even in moderate climates a considerable number of days with heat load can still be expected. Here the maritime influence in the area of the western coast lines is reproduced.

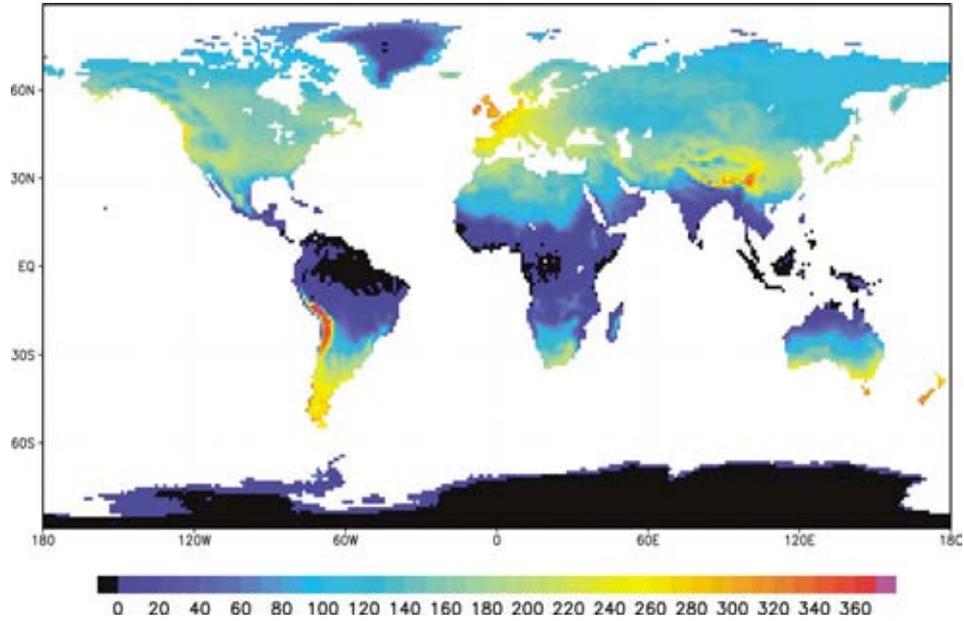


Fig. 3. Mean annual frequency of comfortable conditions (no thermal stress) at 12 MLT (=noon) taking acclimatisation into account based on ECHAM4/T106-data 1971–1980.

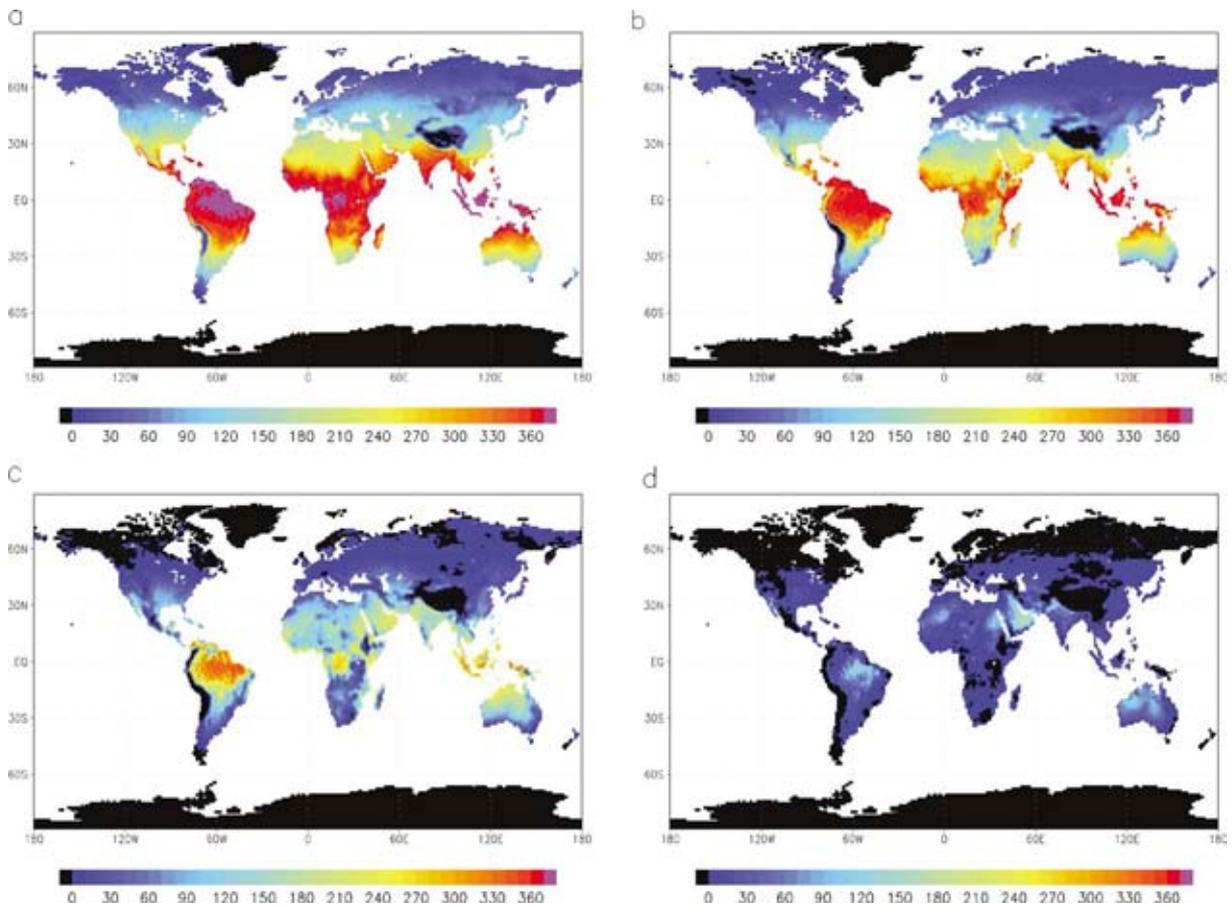


Fig. 4. (a–d) Mean annual frequency of exceeding the threshold for (a) heat load, (b) moderate heat load, (c) strong heat load and (d) extreme heat load at 12 MLT taking acclimatisation into account, based on ECHAM4/T106-data 1971–1980.

From a health point of view, higher heat load intensities become more relevant. Compared to Fig. 4a the area with at least moderate heat load conditions (Fig. 4b) is of course smaller and now almost covers those regions of the world known for tropical or subtropical climate. In the equator region even moderate heat load occurs almost every day. The presentations of the frequency of at least strong (Fig. 4c) and extreme (Fig. 4d) heat load isolate the regions further. It is interesting, for example, that Saudi Arabia shows the most days with extreme heat load (>170 days) while the threshold for at least strong heat load is more frequently exceeded in the Amazon basin and in Indonesia (>300 days).

The zonal variation in the thermal conditions is also found in the distribution of cold stress ('in spite of winter clothing with 1.75 clo!'; Fig. 5a). While almost every day cold stress occurs in Antarctica, followed by less than one-third of the time span of a year over the southern tip of South-America (Tierra del Fuego), the northern hemisphere shows a more differentiated pattern due to superimposed topography and position relative to the sea. The frequency of cold stress in the tropical and subtropical regions is zero or close to that. When the

thresholds for cold stress are tightened in the given definitions (Fig. 5b–d) the borders recede considerably step by step. Extreme cold stress can only be found some distance away from the coast over the ice shields of Antarctica almost every day (Fig. 5d). In central Greenland, the most extreme region in the northern hemisphere, the frequency of such cold conditions is 'only' marginally above 200 days a year. With respect to health consequences it is interesting to note that the regions showing at least moderate cold stress (Fig. 5b) have low population densities.

The spatial distribution of the thermal conditions across Europe looks very similar to maps based on data from 918 European weather stations 1971/2000 (20, 21).

Thermal stress conditions in a future climate

The ECHAM4/T106 simulation of the climate in the period 2041–2050 ('Future') uses the IPCC IS92a 'business-as-usual' scenario. Figs. 6–8 show the difference between the predicted future bioclimate and the 1971–1980 thermal conditions.

Both the humid tropics and Antarctica are characterised by already lacking comfort conditions (permanent

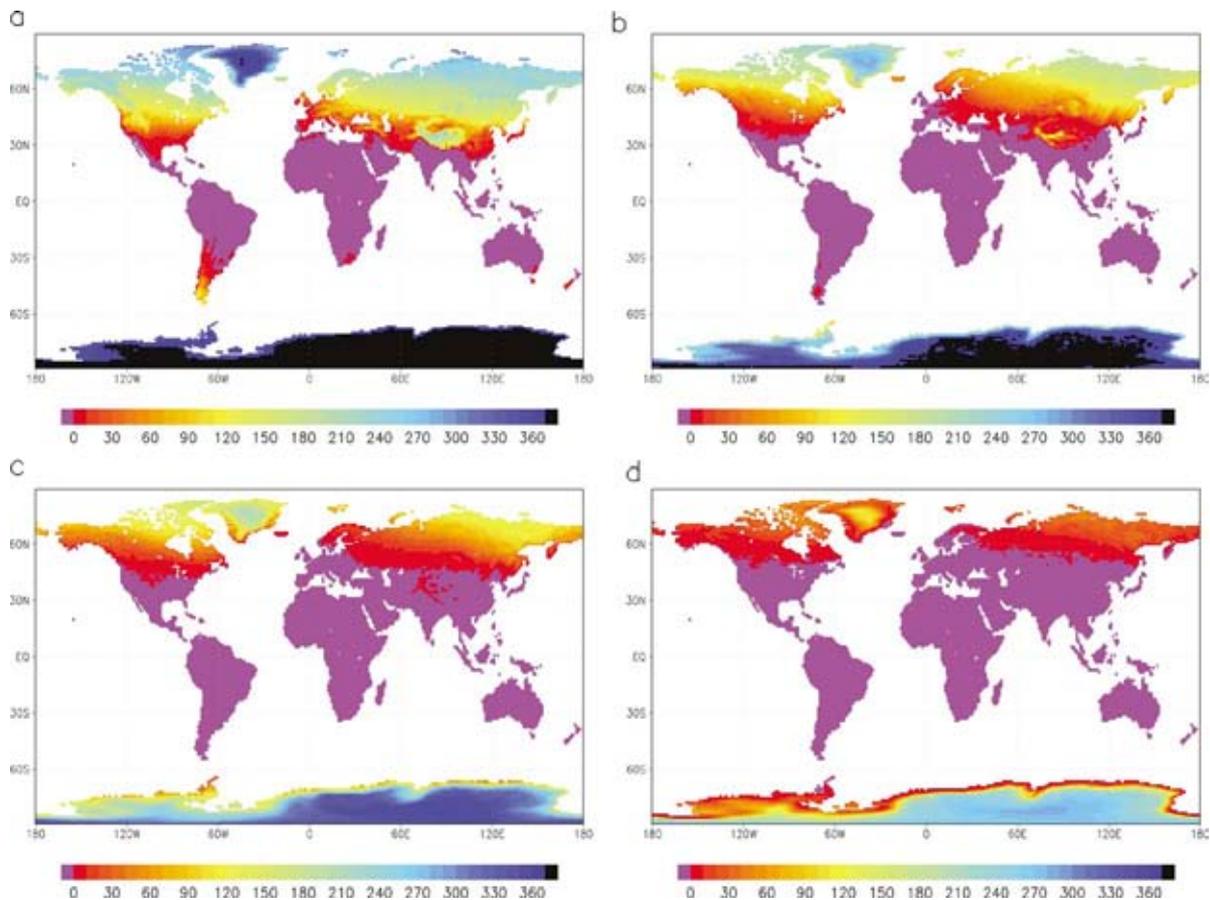


Fig. 5. (a–d) Mean annual frequency of exceeding the threshold for (a) cold stress, (b) moderate cold stress, (c) strong cold stress and (d) extreme cold stress at 12 MLT taking acclimatisation into account, based on ECHAM4/T106-data 1971–1980.

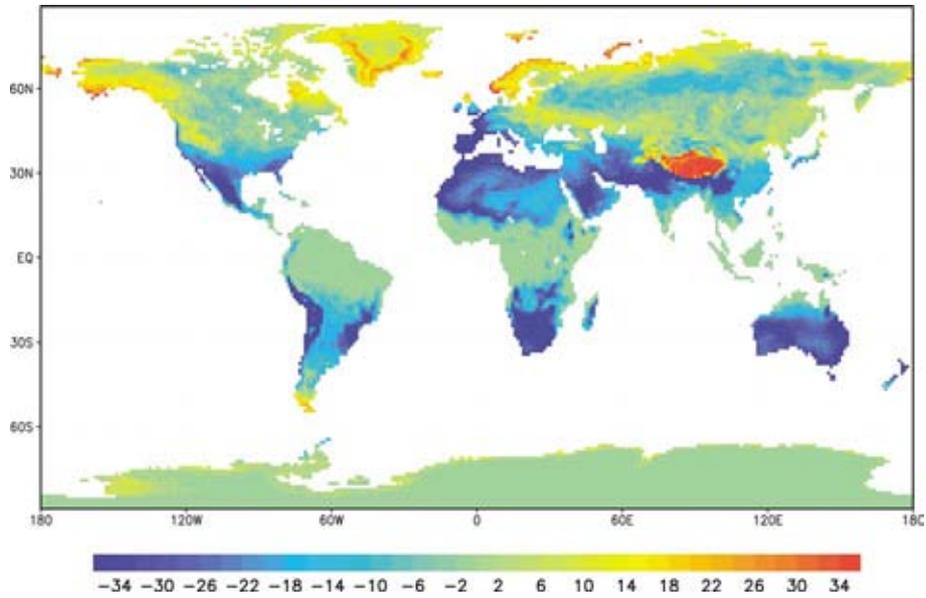


Fig. 6. Difference of the mean annual frequency of comfortable conditions (no thermal stress) at 12 MLT between future and recent climate taking acclimatisation into account, based on ECHAM4/T106-data (2041–2050 and 1971–1980).

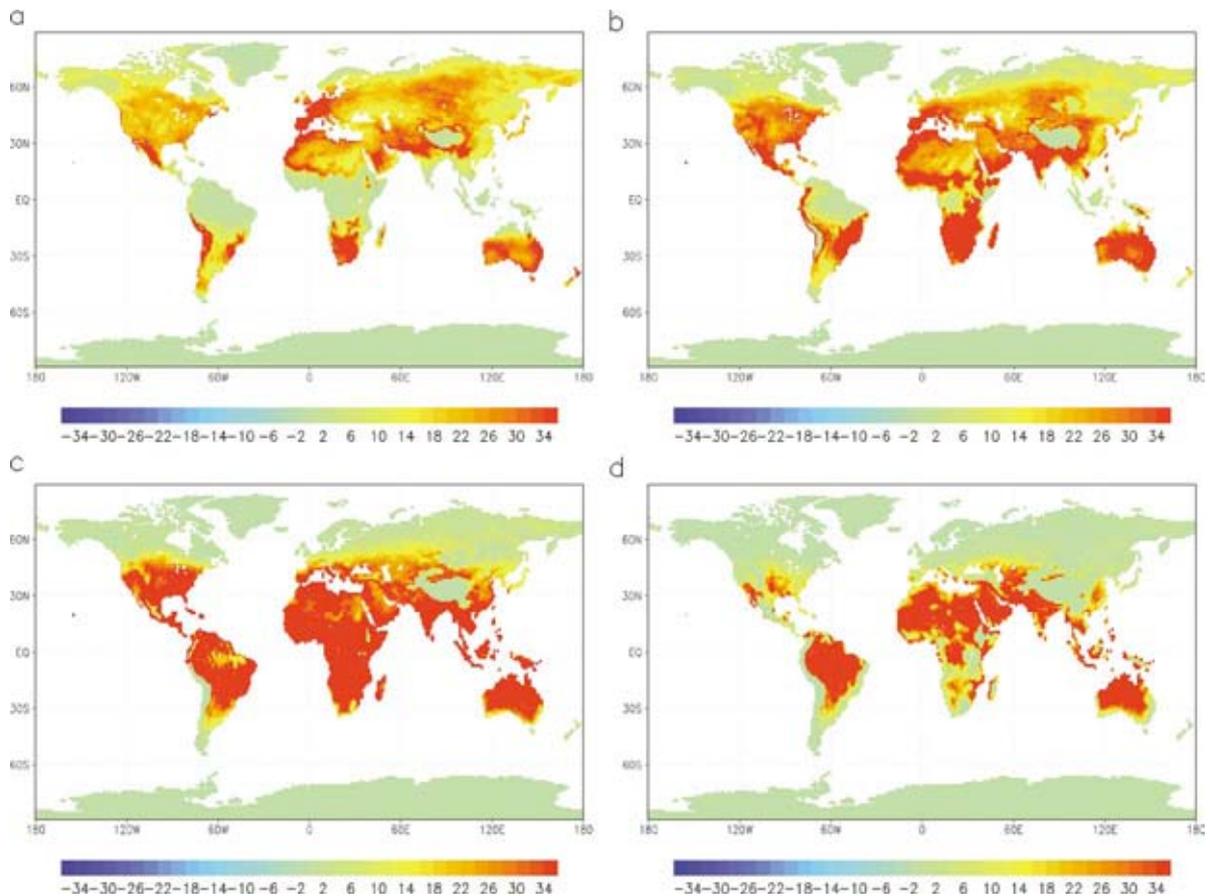


Fig. 7. (a–d) Difference of the mean annual frequency of exceeding the threshold for (a) heat load, (b) moderate heat load, (c) strong heat load and (d) extreme heat load at 12 MLT taking acclimatisation into account, based on ECHAM4/T106-data (2041–2050 and 1971–1980).

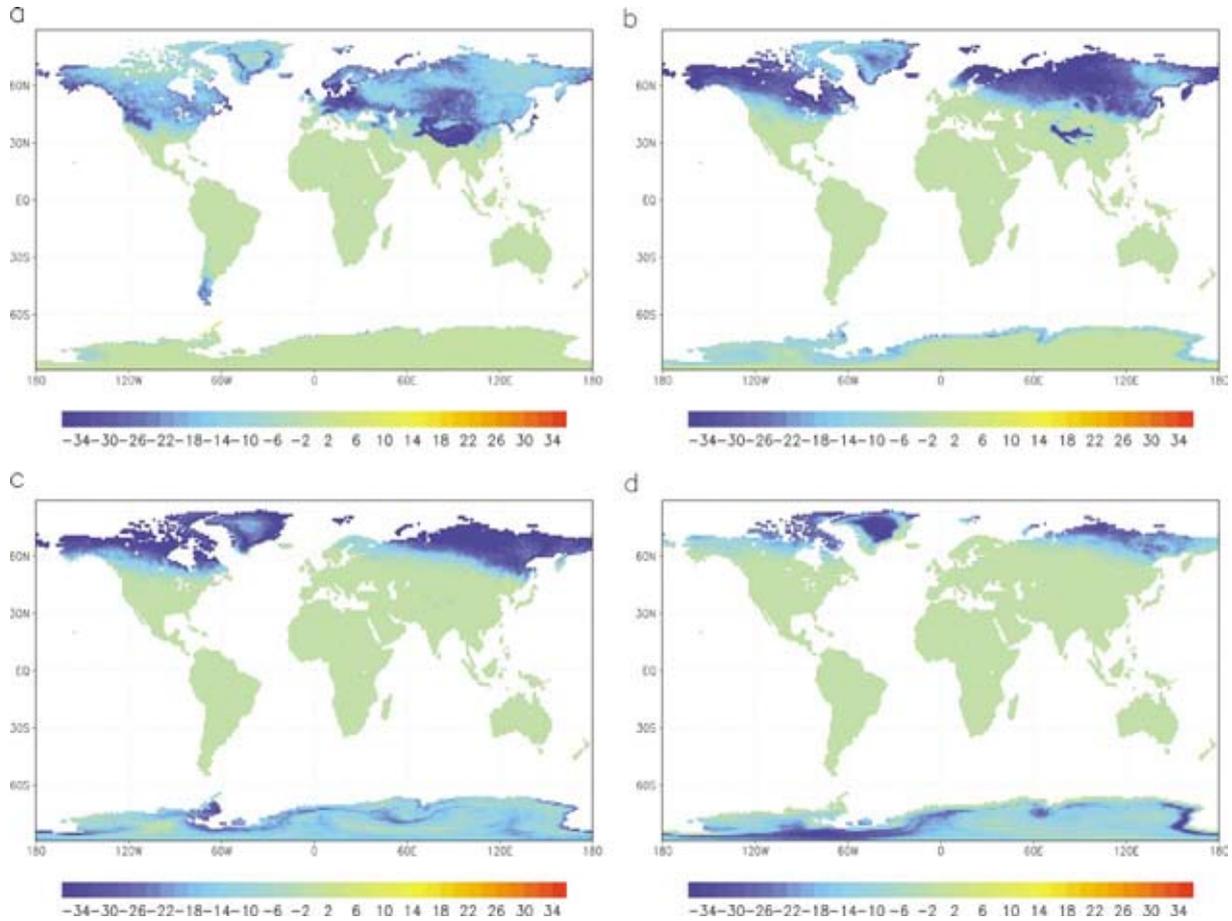


Fig. 8. (a–d) Difference of the mean annual frequency of exceeding the threshold for (a) cold stress, (b) moderate cold stress, (c) strong cold stress and (d) extreme cold stress at 12 MLT taking acclimatisation into account, based on ECHAM4/T106-data (2041–2050 and 1971–1980).

heat load or cold stress conditions, respectively) (Fig. 6), and these will remain. The most pronounced decrease in comfort will happen in the subtropics up to the middle latitudes (moderate climate). An increase in comfortable conditions is mainly limited to cold areas with low population density, maybe with the exception of southern Scandinavia, parts of Russia, China, the northwest of the USA, and British Columbia in Canada.

The distribution of the changes of the annual frequencies of heat load (Fig. 7a–d) shows that almost all regions will become hotter. In the tropics there is no increase of the ‘all heat load conditions’ (Fig. 7a) or ‘at least moderate heat load’ (Fig. 7b) as these are already exceeded. There is a considerable increase in heat load in the subtropics and higher latitudes. While the affected areas are becoming smaller with tightening thresholds the opposite is the case in the tropics and subtropics (Fig. 7c and d). Although the characteristics are different, almost all densely populated areas are affected by the increase of adverse thermal conditions due to climate change.

As is nowadays the case, no cold stress will occur in the tropics and subtropics also in a future climate (Fig. 8a–d). The most densely populated areas will never be affected by extreme or even strong cold stress. In moderate climates a considerable reduction in the frequency of cold stress can be expected (Fig. 8a). The ‘at least moderate cold stress’ situation in the area of the Great Lake District in North-America, Scandinavia and Russia will be improved (Fig. 8b) while a significant decrease in stronger cold stress will occur in practically non-populated regions (Fig. 8c and d).

Discussion

Climate modelling provides global-wide time series of grid data for every day at several (here four) hourly time points, and here we used ECHAM4 time-slice modelling assuming the ‘business-as-usual’ green house gas scenario. In order to estimate impacts of climate or climate change on human health and well-being, a specific physiologically relevant assessment of the climate data is required. This assessment must be based on heat exchange theory taking

all mechanism of heat exchange into account, which results in heat budget models such as the PT procedure. Such models have the ability to correctly assess the climatological variables: air temperature, mean radiant temperature, wind velocity and water vapour pressure. It is likely that our assessment procedure can be applied across the world because thermo-physiology functions are fundamentally the same in all populations. At the population level (neglecting age, gender and inter-individual differences) the susceptibility to thermal stress is mainly controlled by physiological and behavioural adaptation. The psychological aspects of how people assess and prefer climate sensation (see e.g. 42, 43) are not subject of this study. Thermal comfort in our study stands for 'no stress' which means no physiological strain.

Societies have always adapted their cultures to meet the climate requirements (11, 44). Clothing (see e.g. 'adaptive model' by 45, 46), building design, food and drinking habits, working hours (siesta), avoiding outdoors activities during the hottest time of the day, lowering metabolic rate, etc. are some examples of behavioural adaptation that reduces exposure to thermal stress. If acclimatisation were perfect no adverse health effects of climate would be expected, but numerous publications report health impacts of climate indicating that societies are not able to adapt completely.

Acclimatisation (=adaptation to climate) is here considered firstly by assuming reasonable behaviour of the target group 'general population' with respect to clothing. The range of clo-values between 0.5 and 1.75 refers to moderate climate experience. Although there is some evidence that in cold winter climates the general population is not used to wearing more protective clothing thus limiting obviously their time for staying outdoors, this is certainly not true for people (not considered here) with particular activities such as soldiers, rangers, hunters, skiers who are able to avoid any cold stress by appropriate special clothing. Typical Inuit clothing (4.0 clo) would actually produce approximate comfort under the predominant conditions in extreme cold winter climates. The applied lower value 0.5 clo is already almost at the lower possible end. Shorts instead of jeans as often used by people in the tropics reduce the clo-value to 0.4 which would slightly reduce heat load.

Secondly, the acclimatisation approach HeRATE adjusts the initial thermal thresholds automatically to the prevailing local climate. In the warm-humid tropics and in the hot subtropics this often results in an increase of the thresholds for the different heat load intensities of 6–8°C. However, possible long-term (over years and decades) acclimatisation has not been considered due to the lack of a quantitative approach. Additionally the assessment procedure focuses on the population level

which means people walking outdoors with a given metabolic rate of 2.3 MET (135 W m^{-2}). Reducing the MET activity could be assumed under heat load conditions as an adaptation measure of the population if no specific work intensities are required. This was not included in the PT standardisation. Thus, the calculated heat load conditions are probably overestimated. On the other hand there are many outdoor working conditions requiring much higher metabolic rates. In tropical and sub-tropical climates the obstruction of heat release from the body can very quickly produce extreme heat load conditions in a working individual which significantly differ from the assessments in our analysis for an assumed general public.

With the help of PT and HeRATE, the current and predicted thermal environment is described in health-related frequencies of exceeding locally adapted thresholds based on daily values at a fixed time (noon = 12 MLT) which is representative for a few hours around noon. This makes a big difference to the usual consideration of climate and climate change based on monthly or annual mean values of air temperature. In terms of both comfort and the absence of intense thermal stress, the mid-latitude areas are 'privileged' while comfortable or only slight heat load conditions only rarely occur in the humid tropics. The distribution of heat load follows the solar climate to a great extent, while it is modified by maritime or continental influences and altitude. In the tropics and subtropics a huge number of human beings are affected by heat stress, particularly in Asia and Africa. In tropical and sub-tropical areas, where extreme heat load is already common, climate change will make 'extreme heat load' even more 'extreme' with significant impacts on health and well-being of populations living in these areas.

Of course these societies apply some behavioural adaptation measures but the dramatically increased – and from a greenhouse gas point of view contra productive – use of air-conditioning systems in these countries by those who have the financial capabilities indicates that they are not satisfied with the existing climate. The problem increases because in rapidly growing cities traditional buildings which relied on local experience in climate-related building design, is no longer realised. In addition, the urban heat island effect in large cities is already greater than the predicted climate change until the end of this century, and the public health risks are significant. By contrast, major cold stress is limited to sparsely populated areas.

When considering the fact that (a) almost the entire population of the world lives in areas indicated by yellow or red colours in the presentation of the changes (see Fig. 7c) and (b) already exceeding the threshold for moderate heat load would increase mortality (at least based on studies in moderate climates), the public health

issue of this aspect of climate change becomes evident. This would also be true if, due to an underestimation of the effect of acclimatisation in the applied procedure, the thresholds for the different heat load intensities were actually somewhat higher. The predominant part of the world's population will belong to the 'climate losers,' being faced with more frequent and more intense adverse thermal conditions, while few countries or areas will belong to the 'climate winners,' a view on the problem that first was introduced by Auliciems in 1994 (47).

The interpretation of the results must consider the geographic scale. When looking at individual pixels, the coarse resolution (approx. 100 km in middle latitudes, 125 km at the equator) becomes evident. The daily living and working space of a human being is much smaller. The described assessment procedure can also be applied to regional climate models (see e.g. 48) are nested into global models, thus providing bioclimatological conditions with higher resolution (down-scaling). An important aspect that could be added is the urban heat island effect. Further improvements of these assessments can be expected by applying the almost finalised Universal Thermal Climate Index (UTCI; (COST 730) (49)), which is based on human response-related thermo-physiological modelling with the help of a multi-segmental, multi-layered representation of the human body (50, 51), to the new generation of climate simulation models including down-scaling.

Conclusions

The application of the thermo-physiological assessment procedure PT to climate data broadens the usual consideration of climate maps presenting only single climate data such as air temperature. The approach results in a spatial assessment of thermal stress categories which relate climate to the health and well-being of humans. This provides specific problem-oriented information to the climate and climate change, and human health relationships in general and for the estimation of the vulnerability of different populations.

Optimal conditions in terms of no or at most slight thermal stress differ significantly between the individual continents. Europe is obviously 'privileged' while particularly Africa and Australia suffer from heat load with highest frequency at the extremes. Asia and both the Americas lie approximately in between. In the predicted future climate, based on the ECHAM4 simulations, the most pronounced changes will occur at the extreme heat load category which will then become the most frequent condition for America as it is already now for Africa and Australia. Favourable conditions will decrease basically all over the world.

A better understanding of acclimatisation as the adaptation to climate and climate change in different time scales remains an important research issue.

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References

1. Kovats SR, Jendritzky G. Heat-waves and human health. In: Menne B, Ebi KL, eds. *Climate change and adaptation strategies for human health*. Darmstadt, Germany: Steinkopff; 2006, pp. 63–97.
2. Schär C, Jendritzky G. Hot news from summer 2003. *News and views. Nature* 2004; 432: 559–60.
3. Kosatsky T. The 2003 European heat waves. *Euro Surveill* 2005; 10: 148–9.
4. Laschewski G, Jendritzky G. Effects of the thermal environment on human health: an investigation of 30 years of daily mortality data from SW Germany. *Clim Res* 2002; 21: 91–103.
5. Koppe C, Kovats S, Jendritzky G, Menne B, Baumüller J, Bitan A, et al. *Heat waves – risks and responses. Health and global environmental change, Series, 2*. Copenhagen, Denmark: World Health Organization, Regional Office for Europe; 2004.
6. Fanger PO. *Thermal comfort, analysis and application in environment engineering*. Copenhagen, Denmark: Danish Technical Press; 1970.
7. Landsberg HE. *The assessment of human bioclimate, a limited review of physical parameters*. World Meteorological Organization, Technical Note No. 123, WMO-No. 331; 1972.
8. Driscoll DM. Thermal comfort indexes. *Current uses and abuses. Nat Weather Digest* 1992; 17: 33–8.
9. Parsons KC. *Human thermal environments: the effects of hot, moderate, and cold environments on human health, comfort and performance*. London and New York: Taylor & Francis; 2003.
10. Kirch W, Menne B, Bertollini R. *Extreme weather events and public health responses*. WHO. Heidelberg, Germany: Springer; 2005.
11. Menne B, Ebi KL, editors. *Climate change and adaptation strategies for human health*. WHO. Darmstadt, Germany: Steinkopff; 2006.
12. de Freitas CR. Bioclimates of heat and cold stress in New Zealand. *Weath Climat* 1987; 7: 55–60.
13. Auliciems A, de Freitas CR. Cold stress in Canada: a human climatic classification. *Int J Biometeorol* 1976; 20: 287–94.
14. de Freitas CR. Human climates of Northern China. *Atm Env* 1979; 13: 71–7.
15. Jendritzky G. *Das Bioklima in der Bundesrepublik Deutschland [The bioclimate of FR Germany]*. Gütersloh: Flöttmann; 1988.
16. Grätz A, Jendritzky G. *Bioklima – die Bedeutung des Klimas für den Menschen [Bioclimate – the significance of climate for the human being]*. In: *Nationalatlas Bundesrepublik Deutschland. Klima, Pflanzen- und Tierwelt [National atlas of FR Germany. Climate, flora and fauna]*. Leibniz-Institut für Länderkunde (Hrsg.). Heidelberg, Berlin: Spektrum Akad Verl; 2003.

17. Matzarakis A. Human-biometeorological assessment of the climate of Greece. Dissertation in Greece, School of Geology, Aristotelian University of Thessaloniki; 1995.
18. Koch E, Matzarakis A, Rudel E, Zygmuntowski M. Human-Bioklima in Österreich – Die thermische Komponente [Human bioclimate in Austria – the thermal component]. In: Koch E, Marktl W, Matzarakis A, Nefzger H, Rudel E, Schunder-Tatzber S, Zygmuntowski M, eds. Klimatherapie in Österreich. Broschüre zu den Potentialen der Klimatherapie in Österreich [Climate therapy in Austria. Brochure on the potentials of climate therapy in Austria]. Vienna: Bundesministerium für Wirtschaft und Arbeit; 2005, pp. 7–26.
19. Morillon-Galvez D, Saldana-Flores R, Tejeda-Martinez A. Human bioclimatic atlas for Mexico. *Sol Energy* 2004; 76: 781–92.
20. Tinz B, Jendritzky G. Macro- and mesoscale maps of the thermal environment. 17th International Congress of Biometeorology. *Ann Meteorol* 2005; 41: 641–3.
21. Jendritzky G, Tinz B. Human bioclimate maps for climate impact research. Proceedings of International Conference of Biomet and International Conference on Urban Climate. ICB-ICUC '99, Sydney 1999. Geneva: WCASP; Vol. 50, WMO/TD No. 1026; 2000, pp. 63–83.
22. Höppe P. The physiological equivalent temperature – a universal index for the biometeorological assessment of the thermal environment. *Int J Biometeorol* 1999; 43: 71–5.
23. Matzarakis A, Amelung B. Physiologically equivalent temperature as indicator for impacts of climate change on thermal comfort of humans. In: Thomson MC, Garcia-Herrera R, Beniston M, eds. Seasonal forecasts, climate change and human health. *Advances in global change research* 30. Berlin: Springer Sciences and Business Media; 2008, pp. 161–72.
24. Büttner K. Physikalische Bioklimatologie. Probleme und Methoden [Physical bioclimatology. Problems and methods]. Leipzig, Germany: Akad Verl Ges; 1938.
25. Jendritzky G, de Dear R. Adaptation and the thermal environment. In: Ebi KL, Burton I, McGregor G, eds. *Biometeorology for adaptation to climate variability and change: research frontiers and perspectives*. Heidelberg, Germany: Springer; 2009, pp. 9–32.
26. ISO 7730. Ergonomics of the thermal environment – analytical determination and interpretation of thermal comfort using calculation of the PMV and PPD indices and local thermal comfort criteria; 2005.
27. Gagge AP, Fobelets AP, Berglund PE. A standard predictive index of human response to the thermal environment. *ASHRAE Trans* 1986; 92: 709–31.
28. Jendritzky G, Sönning W, Swantes HJ. Ein objektives Bewertungsverfahren zur Beschreibung des thermischen Milieus in der Stadt- und Landschaftsplanung ('Klima-Michel-Modell') [An objective assessment procedure to specify the thermal environment in urban and landscape planning ('Klima-Michel model')]. *Beiträge Akad Raumforschung Landesplanung Hannover* 1979; 28: 1–85.
29. Jendritzky G, Nübler W. A model analysing the urban thermal environment in physiologically significant terms. *Arch Met Geoph Biokl B* 1981; 29: 313–26.
30. Jendritzky G. Bioklimatische Bewertungsgrundlage der Räume am Beispiel von mesoskaligen Bioklimakarten [Regional bioclimatological assessment procedure using meso-scale bioclimate maps as example]. In: Jendritzky G, Schirmer H, Menz G, Schmidt-Kessen W, eds. *Methode zur raumbezogenen Bewertung der thermischen Komponente im Bioklima des Menschen (Fortgeschriebenes Klima-Michel-Modell)* [Method of a region-oriented assessment of the thermal component of human bioclimate (Updated Klima-Michel model)]. *Beiträge Akad Raumforschung Landesplanung Hannover* 1990; 114: 7–69.
31. Staiger H, Bucher K, Jendritzky G. Gefühlte Temperatur. Die physiologisch gerechte Bewertung von Wärmebelastung und Kältestress beim Aufenthalt im Freien in der Maßzahl Grad Celsius [The physiological adequate assessment of heat load and cold stress in outdoor conditions in terms of degree Celsius]. *Ann Meteorol* 1990; 33: 100–7.
32. Mayer H, Holst J, Dostal P, Imbery F, Schindler D. Human thermal comfort in summer within an urban street canyon in Central Europe. *Meteorol Z* 2008; 17: 241–50.
33. Pickup J, de Dear R. An outdoor thermal comfort index (OUT_SET*) – part I – the model and its assumptions. In: de Dear R, Kalma J, Oke T, Auliciems A, eds. *Biometeorology and urban climatology at the turn of the millennium*. Geneva: WCASP 50: WMO/TD No. 1026; 2000, pp. 279–83.
34. De Dear R, Pickup J. An outdoor thermal environment index (OUT_SET*) – part II – applications. In: De Dear R, Kalma J, Oke T, Auliciems A, eds. *Biometeorology and urban climatology at the turn of the millennium. Selected Papers from the Conference ICB-ICUC'99, Sydney, 8–12 November 1999*. Geneva: WMO, WCASP; Vol. 50, 2000, pp. 258–90.
35. ISO 9920. Ergonomics of the thermal environment – estimation of the thermal insulation and evaporative resistance of a clothing ensemble; 1995.
36. Koppe C, Jendritzky G. Inclusion of short-term adaptation to thermal stresses in a heat load warning procedure. *Meteorol Z* 2005; 14: 271–8.
37. Koppe C. Gesundheitsrelevante Bewertung von thermischer Belastung unter Berücksichtigung der kurzfristigen Anpassung der Bevölkerung an die lokalen Witterungsverhältnisse [Health related assessment of thermal stress considering short-term adaptation of the population to local climate]. *Ber d Deutschen Wetterdienstes Offenbach* 2005; 226: 1–168.
38. Roeckner E, Arpe K, Bengtsson L, Christoph M, Claussen M, Dümenil L, et al. The atmospheric general circulation model ECHAM4: model description and simulation of present-day climate. Reports of the Max-Planck-Institute. Hamburg 1996; 218: 1–90.
39. Stendel M, Roeckner E. Impacts of horizontal resolution on simulated climate statistics in ECHAM4. Reports of the Max-Planck-Institute. Hamburg 1998; 253: 1–57.
40. Kattenberg A, Giorgi F, Grassl H, Meehl GA, Mitchell JFB, Stouffer RJ, et al. Climate models – projections of future climate. In: Houghton JT, MeiraFilho LG, Callander BA, Harris N, Kattenberg A, Maskell K, eds. *Climate change 1995: the science of climate change*. New York: Cambridge University Press; 1996, p. 572.
41. Solomon S, Qin D, Manning M, Chen Z, Marquis M, Averyt KB, et al., editors. *Climate change 2007: the physical science basis. Contribution of Working Group I to the fourth assessment report of the intergovernmental panel on climate change*. Cambridge, UK and New York: Cambridge University Press; 2007. Available from: <http://www.ipcc.ch/ipccreports/ar4-wg1.htm> [cited 27 September 2009].
42. Nikolopoulou M, Baker N, Steemers K. Thermal comfort in outdoor urban spaces: understanding the human parameter. *Sol Energy* 2001; 3: 227–35.
43. Knez I, Thorsson S, Eliasson I. Psychological mechanism in outdoor place and weather assessment: towards a conceptual model. *Int J Biometeorol* 2009; 53: 101–11.
44. Parry ML, Canziani OF, Palutikof JP, van der Linden PJ, Hanson CE, editors. *Climate change 2007. Impacts, adaptation and vulnerability. Contribution of Working Group II to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change*. Cambridge, UK and New York: Cambridge

- University Press; 2007. Available from: <http://www.ipcc.ch/ipccreports/ar4-wg2.htm> [cited 27 September 2009].
45. De Dear R, Brager G. Developing an adaptive model of thermal comfort and preference. *ASHRAE Trans* 1998; 104: 145–67.
 46. Brager GS, Paliaga G, de Dear R. Operable windows, personal control and occupant comfort. *ASHRAE Trans* 2004; 110: 510–26.
 47. Auliciems A. Thermoregulatory adaptation to global warming – winners and losers. Proceedings of the 13th International Congress of Biometeorology, Calgary, Canada, 12–18 September 1993, pp. 109–122.
 48. Jacob D, Bärring L, Christensen OB, Christensen JH, Hagemann S, Hirschi M, et al. An inter-comparison of regional climate models for Europe: design of the experiments and model performance. *Clim Chang* 2007; 81: 31–52.
 49. COST 730. Towards a Universal Thermal Climate Index UTCI for assessing the thermal environment of the human being. MoU of COST Action 730. Available from: <http://www.utci.org;2004>. [cited 27 September 2009].
 50. Fiala D, Lomas KJ, Stohrer M. Computer prediction of human thermoregulatory and temperature responses to a wide range of environmental conditions. *Int J Biometeorol* 2001; 45: 143–59.
 51. Fiala D, Lomas KJ, Stohrer M. First principles modelling of thermal sensation responses in steady-state and transient conditions. *ASHRAE Trans: Res* 2003; 109: 179–86.

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Issues in health risk assessment of current and future heat extremes

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Background: In assessing health risks relating to current and future heat extremes, it is important to include developing countries, because these countries are considered to be vulnerable to the impact of climate change due to inadequate public health infrastructure, nutritional status and so forth. However, it is usually difficult to obtain relevant information from these countries, also because of insufficient public health infrastructure.

Objectives: We invented a method that can be used for developing countries to assess the health risks of current and future extremes, but there still are some issues. We introduce and discuss these issues.

Design: We analysed time-series data with non-parametric regression models including generalised additive models, which controlled for time trends.

Results: When we controlled for year, the temperature–mortality relation was V-shaped, but when we controlled for season as well as year, the left side of the V-shape disappeared. Our month-specific analysis also revealed that winter months had higher mortality rates than other months, but there was no relation between mortality rate and temperature within each month during winter.

Conclusions: This suggests that, unlike heat effects, risks due to cold effects may not be ameliorated even if global warming occurs. We need to investigate the mechanism behind high mortality during winter months.

Keywords: *heat extremes; Japan; epidemiology; excess mortality; generalised additive models*

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In conducting global assessments of health risks associated with the impacts of ongoing climate change, one of the largest problems is that we need to address not only developed countries, but also many developing countries. In developing countries, the impact may be more serious than that in developed countries, yet the necessary data are sparse in general.

Among the various impacts, we have been assessing the health risk effect of current and future heat extreme. In this case also, there are problems in addressing developing countries in the assessment. Direct heat effects are usually evaluated with time-series studies or case-cross-over studies. In both types of studies, we need daily data for meteorological variables and outcome variables (mortality or morbidity). Also, for better prediction, we may need age, sex and cause-specific outcomes. However, it is impossible to obtain the daily outcome data for many developing countries. Hence, while it is important to develop sophisticated multivariate statistical models for developed countries, it is also very important to develop a simple method that can be applied to developing countries.

Thus, we have established our approach for prediction without using daily mortality data. The outline is as follows:

Reports in various areas in the world have shown that temperature–mortality relation is V-shaped, i.e. the mortality rate is high at both extremes of temperature, and between the extremes, there is a temperature at which the mortality is lowest. This temperature is called ‘optimum temperature’ (OT). Based on this relation, the excess mortality can be defined as shown in Fig. 1.

Some researchers also reported that the OT level is related to climate (1, 2). Curriero and colleagues reported that southern US cities had higher OT than northern US cities. To incorporate this characteristic in the prediction, we need a good climate index that can be used to infer the OT level. After exhaustive searching, we found that the 80–85th percentile value of daily maximum temperature is the best index for OT inference (3). Roughly speaking, if the 80th percentile value of daily maximum temperature in a certain area is 25°C, OT is also close to 25°C.

Based on the above finding and other assumptions explained in detail in Appendix A, we projected present

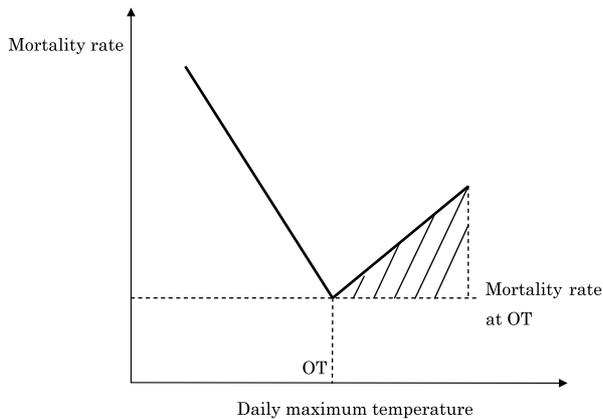


Fig. 1. A schema for excess mortality due to heat.

and future risk due to heat-related deaths (4). In the assessment, however, we encountered some challenges: the days with extremely high temperatures were rare and the risk for these days could not be precisely estimated, hence we needed to pool the data; but pooling across areas was difficult because the OT levels were climate dependent. In our analysis, then, we pooled across chronological time.

In this article, we consider whether or not pooling across time was appropriate, and show another problem we found in controlling for time trend by generalised additive model. Also, we briefly discuss the net effect of climate change, making assessments that evaluate not only heat-related effects, but also cold-related effects.

Present investigation

Methods

Data

As we described above, we made our model as simple as possible to be generalisable to developing countries. Hence, we used daily maximum temperature as the only meteorological variable, and daily crude mortality rate (or daily number of deaths) as an outcome variable. Notably, our study showed that relative humidity did not confound the temperature–mortality relation in Japan (5).

The data for daily maximum temperature were obtained from Meteorological Agency, Japan, and that for daily mortality data were obtained from Ministry of Health, Labour and Welfare. The period of observation was from 1972 to 1995 (except for Okinawa, for which the period was from 1973 to 1995).

Statistical methods

We evaluated temperature–mortality relations by prefecture. In Japan, we have 47 prefectures; the northernmost prefecture is Hokkaido (43°N) and the southernmost prefecture is Okinawa (26°N). Tokyo is located in the middle of these two prefectures in terms of latitude (35°N). In the evaluation, we used smoothing spline (smooth.

spline) function from the statistics package of R language and environment for statistical computing and graphics (6). Degrees of freedom is another issue in semi-parametric regression; fewer degrees of freedom yield smoother curves, but if too few, then true relationships can be missed. Based on our exhaustive search, we set the degrees of freedom as six to obtain the best bias–variance trade-off (7). Using the above model, we determined the OT.

Year-adjusted mortality rate

In drawing smoothing spline curves, we encountered a problem with non-V-shaped relations in some prefectures (7). We could not initially explain why this phenomenon had occurred, but now we have found that it is due to the mechanism explained in Appendix B.

To illustrate, we replicate the temperature–mortality relation in Hiroshima, and compare it with the similar relation in which we replaced the ordinary mortality rate with year-adjusted mortality rate as described below.

In calculating year-adjusted daily mortality rate (YaM), we calculate two mean mortality rates in addition to ordinary daily mortality rate (DM): grand mean mortality rate (GM), i.e. average of DM for the entire observation period and year mean mortality rate (YM), i.e. average of the DM for each year. YaM is defined as:

$$\text{YaM} = \text{DM} - \text{YM} + \text{GM}$$

This procedure sets the average of YaM for each year identical to GM. In other words, this procedure explicitly eliminates the year trend.

Generalised additive models

Another method used to control for non-linear time trends was generalised additive models (8). In the analysis we used ‘mgcv’ package (9) from the R statistical environment. In controlling for time trend, we tried to make the degrees of freedom closer to the number of observation years to control for year trend, and seven times as large as the number of observation years to control for year and seasonal trend. Since time trend is explicitly addressed by adding the time term in the generalised additive models, we used the daily number of deaths, instead of the year-adjusted mortality rate.

Month-specific analysis

As will be described in the Results section, the year-controlled temperature–mortality relation and the year and season-controlled relations showed substantial differences. In order to explore these differences further, we evaluated month-specific temperature–mortality relations using smoothing spline curves.

Results

Fig. 2 shows the relation between daily maximum temperature and mortality in Hiroshima. Although we

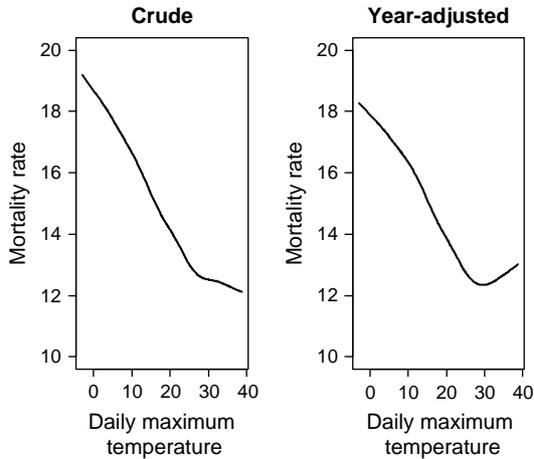


Fig. 2. Relation between daily maximum temperature and mortality rate (Hiroshima, 65+ years old males, 1972–1995). Mortality for the right panel is the ordinary daily mortality rate (daily number of deaths divided by population for the year) and that for the left panel is the year-adjusted mortality rate.

used identical datasets except for mortality rate, the left panel shows a non-V-shaped relation, whereas the right panel clearly shows a V-shaped relation. This comparison suggests that not controlling for year fails to form a V-shaped relation.

To provide additional information for discussion, we show Fig. 3. Here, the distribution of daily maximum temperatures for Hiroshima was shown for 1972–1979, 1980–1989 and 1990–1995. During 1972–1979, there were some days with daily maximum temperature below zero,

but no such day during 1990–1995. In contrast, number of days with daily maximum temperature beyond 35°C was much larger during 1990–1995 than that during 1972–1979. In Hiroshima, OT was in the upper twenties. The frequency distribution for OT appeared stable over time compared with the above difference for extremely cold and hot temperatures.

Fig. 4 illustrates the result of generalised additive model analyses. The top two panels show the results when only year was controlled for, and the bottom two panels show the results when year and season were controlled for. When only year was controlled for, the temperature–mortality relation was V-shaped, and the number of deaths was increasing over time. In contrast, when year and season were controlled for, the left-hand side of the V-shape disappeared. On the other hand, the high mortality rate during the cold season was observed in the time trend panel; with varying magnitude, every year had a winter spike.

Fig. 5 shows the relation between daily maximum temperature and year-adjusted mortality rate by month for some selected prefectures. The solid black, red and green lines on the left side are for December, January and February, respectively. The solid black, red and green lines on the right side are for June, July and August, respectively. Despite some fluctuations, the lines for winters do not appear to be temperature-related. In contrast, the lines for summer show higher mortality at higher temperatures. Another finding is that, not only within the prefecture, but also across prefectures, mortality rates within particular months in winter were

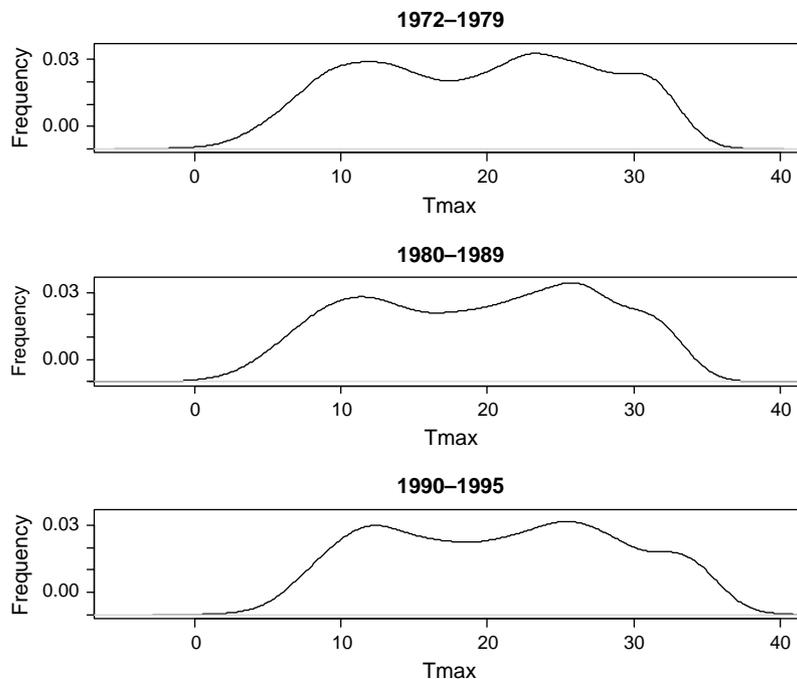


Fig. 3. Distribution of daily maximum temperature in Hiroshima by period.

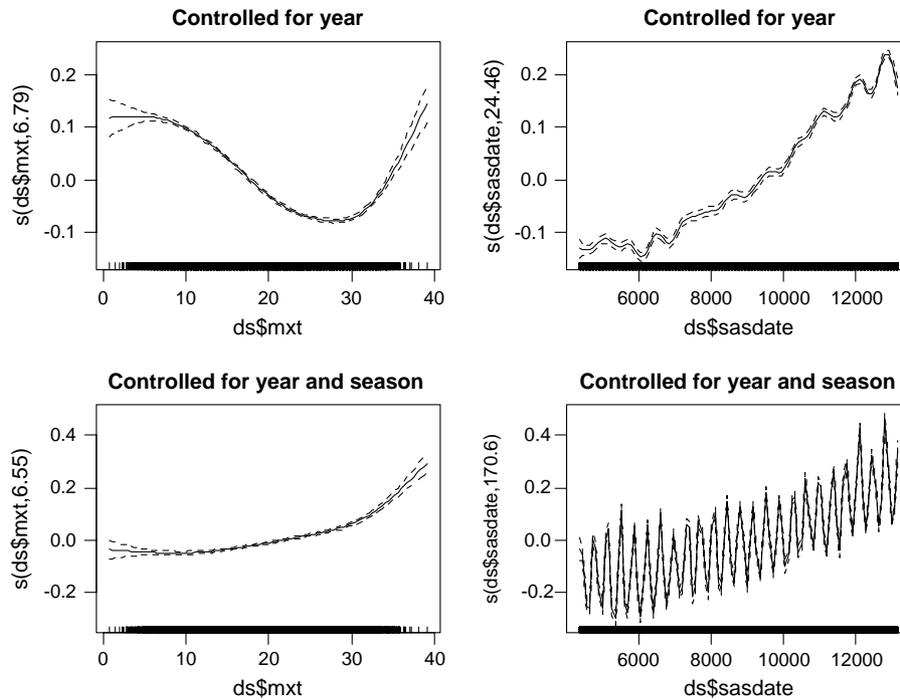


Fig. 4. Result of generalised additive model analyses for Tokyo.

Number of daily deaths was predicted with daily maximum temperature ($ds\$mxt$) and time trend ($ds\$sasdate$). In controlling for year only, degrees of freedom for time trend were close to the number of years observed, and the degrees of freedom were about seven times as large when controlling for year and season.

more or less similar. Hokkaido, the coldest prefecture in Japan, had a much lower temperature range than other prefectures, yet its mortality rates in colder months were similar to those of the other prefectures.

Discussion

As Fig. 2 clearly shows, not controlling for time trends resulted in bias, with the non-V-shape problem. As explained in Appendix B, simultaneous changes in

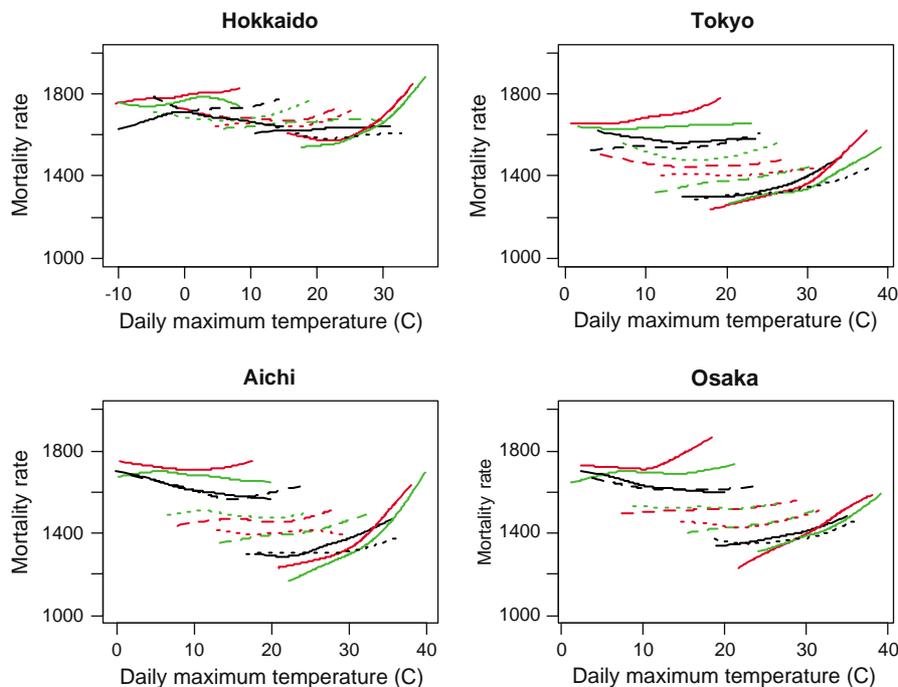


Fig. 5. Relation between daily maximum temperature and year-adjusted mortality rate by month for some selected prefectures.

mortality rate and temperature distribution over time confounded the V-shaped temperature–mortality relation. Since temperatures have been rising due to climate change and mortality rates have been decreasing in many countries, it is not desirable to pool the data across time. This leaves us a problem of statistical instability for the relative risk on days with extremely high temperature. One possible solution, besides using generalised additive models, is to adjust for OT. This would at least reduce the variance of the relative risk on days with temperatures beyond the OT.

Another challenge we encountered in generalised additive models is how we can consider cold effects. Our analysis revealed that left side of the V-shape disappeared in all the prefectures; no prefecture showed higher mortality in colder days than in warmer days during winter when controlling for year and season. Some reports showed that V-shaped relations remained after controlling for season (2, 10). We are unsure why Curriero and colleagues' results showed very high mortality on colder days, but in some figures from McMichael and colleagues' report, a similar pattern to ours can be observed. Also Armstrong's paper showed relation between cardiovascular mortality (which is the major cause of death that forms the V-shaped relation) and daily temperature in London, and the cold effect decreased substantially when controlling for influenza epidemics and other factors (11). At least our monthly analysis clearly showed that, for the colder months, temperature levels did not affect mortality within each month. Armstrong's finding and our monthly analysis suggest that the cold side of the V-shape is mainly formed by influenza epidemic or other seasonal factors. This may incur reconsideration of 'net effect' evaluation of climate change on mortality, because global warming increases the number of extremely hot days and hence heat-related mortality increases, but decreasing numbers of very cold days may not attenuate high mortality in winter. There are other issues in the inference of health impact of climate change in developing countries, but our finding on cold effects showed the necessity of further investigations on the mechanism of high mortality during winter. Lag effect analysis is one of these investigations. At least we should not simply use the V-shaped curve in projecting net effect of climate change.

In the risk assessment of future climate change impact, how we address adaptation is always challenging. Adaptation can be divided into two categories; explicit policies to reduce the impact and spontaneous, collective phenomenon. The former cannot be estimated without conducting intervention studies, but the latter may be estimated using our finding, i.e. the relation between the 80th percentile value of the daily maximum temperature and OT; shifting the V-shaped curve from left to right makes the population less vulnerable to heat. For example, if the 80th percentile

value of the daily maximum temperature rose from 26 to 28°, then setting the OT estimate at 26° would imply a 0% shift and 28° would imply a 100% shift.

Conclusion

In order to assess the risks due to climate change in developing countries, our finding that the 80th percentile value of daily maximum temperature was a good estimate of the optimum daily maximum temperature can be used, but there are some challenges.

We found that simultaneous changes in mortality rate and temperature distributions confounded the temperature–mortality relation in Japan. Since the temperature has been rising due to climate change and mortality rate has been changing in many of the countries, it is not desirable to pool the data across time.

The colder side of the V-shaped relation was formed due to higher mortality in winter months, but there was no cold effect within each month. This finding suggests that simple calculations of the net effect of global warming using V-shaped curves are questionable.

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Conflict of interest and funding

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References

1. Honda Y, Ono M, Uchiyama I, Sasaki A. Shift of the short-term temperature–mortality relationship by a climate factor – some evidence necessary to take account in estimating the health effect of the global warming. *J Risk Res* 1998; 1: 209–20.
2. Curriero FC, Heiner KS, Samet JM, Zeger SL, Strug L, Patz JA. Temperature and mortality in 11 cities of the eastern United States. *Am J Epidemiol* 2002; 155: 80–7.
3. Honda Y, Kabuto M, Ono M, Uchiyama I. Determination of optimum daily maximum temperature using climate data. *Environ Health Prev Med* 2007; 12: 209–16.
4. Takahashi K, Honda Y, Emori S. Assessing mortality risk from heat stress due to global warming. *J Risk Res* 2007; 10: 339–54.
5. Honda Y, Ono M, Uchiyama I. Humidity does not confound temperature–mortality relationship. *Jpn J Biometeorol* 2000; 37: 113–8.
6. Ihaka R, Gentleman R. R: a language for data analysis and graphics. *J Comp Graph Stat* 1996; 5: 299–314. Available from: <http://www.R-project.org> [cited 21 October 2009].
7. Likhvar VN, Honda Y. Choice of degree of smoothing in fitting nonparametric regression models for temperature–mortality relation in Japan based on a priori knowledge. *J Health Sci* 2008; 54: 143–53.
8. Hastie TJ, Tibshirani RJ. Generalized additive models. London: Chapman & Hall; 1990 pp. 136–73.

9. Woods S. The mgcv package. Version 1.4–1. 2008, pp. 17–24. Available from: <http://www.R-project.org> [cited 21 October 2009].
10. McMichael AJ, Wilkinson P, Kovats SR, Pattenden S, Hajat S, Armstrong B, et al. International study of temperature, heat and urban mortality: the 'ISOTHURM' project. *Int J Epidemiol* 2008; 37: 1121–31.
11. Armstrong B. Models for the relationship between ambient temperature and daily mortality. *Epidemiology* 2006; 17: 624–31.

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Appendix A. Assumptions for the climate change risk projection

As described in the main text, it is often the case that the only available data is crude mortality rate in developing countries. Even when age and sex-specific mortality rate or cause-specific mortality rate are available, often the data are not sufficiently reliable. Hence, we tried to estimate the mortality rate at OT (MRot) using annual crude mortality rate (MRa). In our study (4), we used Japanese dataset. For each prefecture, we computed the ratio MRot/MRa, and obtained 0.9 for the average ratio of the 47 prefectures.

Another parameter we need is the relative risk for the excess mortality due to heat. To overcome statistical instability, we adjusted for OT in obtaining the relative risk estimate. We also recognised that the relative risk was close to unity if the temperature was close to OT, but became larger when the temperature was much higher. Hence we calculated relative risks for two temperature categories; our relative risk estimate based on Japanese 47 prefectures were 1.02 for daily maximum temperature between OT and OT+5 and 1.10 for daily maximum temperature beyond OT+5.

The existence of OT difference across the prefectures implies that OT may become higher when the global

warming occurs. However we have no clue how fast this OT shift occurs, thus we assumed that OT is stable even when the global warming occurs.

Other assumptions we made were future scenarios; we used SRES A1B scenario (1) and Japanese climate model, CCSR/NIES/FRCGC Atmosphere–Ocean General Circulation Model.

Appendix B. Mechanism of forming non-V-shaped relations

As shown in Fig. 3, number of days with OT (around 28°) did not change substantially, but number of days with extremely high temperature considerably increased over time. This suggests that the proportion of days in later years is higher for days with extremely high temperature than for days with OT. Also, the mortality rate has been decreasing in Japan.¹ Hence the mortality rates were lower for more recent years. The combined effect of these two trends makes the pooled mortality rate for days with extremely high temperature lower than that for days with OT when the data are pooled across time.

For most of the prefectures, the effect was such that the slope of the right side of V was less steep; only for several prefectures, the V-shaped relation disappeared. However, even when there is V-shaped relation, the slope is confounded, and may not be valid if we pool the data across time.

Note

1. Fig. 2 is for 65+ years old age group and the mortality rate was decreasing. In contrast, the total number of deaths has been increasing as shown in Fig. 4.

Reference

1. IPCC. Special report on emissions scenarios. Cambridge: Cambridge University Press; 2000.

Comparing approaches for studying the effects of climate extremes – a case study of hospital admissions in Sweden during an extremely warm summer

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Background: Health effects induced by climate, weather and climatic change may act directly or indirectly on human physiology. The future total burden of global warming is uncertain, but in some areas and for specific outcomes, mortality and morbidity are likely to increase. One likely effect of global warming is an increasing number of extreme weather events, such as floods, storms and heat waves. The excess numbers of specific health outcomes attributable to climate-induced events can be estimated. This paper compares approaches for estimating excess numbers of outcomes associated with climate extremes, exemplified by a case study of hospital admissions during the extremely warm summer of 2006 in southern Sweden.

Materials and methods: Daily hospital admission data were obtained from the Swedish National Board of Health and Welfare for six hospitals in the Skåne region of southern Sweden for the period 1998 to 2006. Daily temperature data for the region were obtained from the meteorological station in the city of Malmö. We used four established approaches for estimating the daily excess numbers associated with extreme heat. Time series of daily event rates were assumed to follow a Poisson distribution. Excess event rates were compared by using several approaches, such as standardised event ratios and generalised additive models to estimate the health risks attributable to the extreme climate event.

Results: The four approaches yielded vastly different results. The estimates of excess were considerably biased when not accounting for time trends in previous years' data. Three of four approaches showed a significant increase in excess hospitalisation rates attributable to the heat episode in Skåne in 2006. However, modelling the effect of temperature failed to describe the risks induced by the extreme heat.

Conclusion: Estimates of excess events depend greatly on the approach used. Further research is needed to identify which method yielded the most accurate estimates. However, one of the approaches used generally seem to perform better than the others in estimating the excess rates associated with the heat episode. Further on, estimating relative risks of temperature or other determinants of disease may fail to incorporate the unique characteristics of particular weather events, such as the effect caused by very persistent heat exposure. Unless this can be incorporated into predictive models, such models may be less appropriate to use when predicting the future burden of heat waves on human health.

Keywords: *hospital admission; temperature; heat; surveillance; weather; climate change; extreme event*

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Establishing associations between weather, climate and different health outcomes are fundamental to better understandings of the complex associations between human wellbeing and a changing climate. Weather and climate can affect health on different timescales, by different pathways, and associations can be roughly divided into direct or indirect effects accord-

ing to whether an extreme weather event acts directly on human physiology (e.g. cardiovascular effects of heat waves, cold spells and extreme weather events) or indirectly via pathogens, allergens or vectors (e.g. vector and water-borne diseases, mould and pollens) (1). The links between weather, climate and health are still largely unexplored, but in some areas researchers have

established various associations between heat events and health outcomes, such as patterns of cholera transmission (2), tick-borne disease (3) and heat-induced mortality (4, 5).

According to the fourth assessment report by the Intergovernmental Panel of Climate Change (IPCC), climate and health experts expect the global burden of disease to increase due to extreme climatic events, such as floods, heat waves and storms (1). Health outcomes during such events are in some cases monitored by surveillance centres, with excess risks or frequencies being estimated routinely or subsequently characterised in more detailed and specific scientific publications. Often risks of an outcome during an extreme event are compared with the expected risks for the same outcome; for example, in heat wave observational studies this may correspond to the observed number subtracted (O–E) or divided by (O/E) the expected number, where the expected is based on the mean frequency in preceding years (4, 6–9). Another frequently applied study design compares observed frequencies with those predicted (expected) from previous years in time series regression models including trends and possibly taking into account calendar time (10–13). Both approaches mentioned above are common in observational studies and offer a fast and comprehensive way to augment scientific knowledge of climate induced hazards. A third approach used in studies of temperature and health is the incorporation of parametric or non-parametric spline functions (smooth functions) in time series regression models (14). Such functions estimate the excess health outcomes (O–E) during the extreme events, taking into account calendar time variables, such as seasonality, time trend, weekday patterns and national holiday patterns plus additional potential confounders, e.g. air pollution levels during an heat wave (15). With such a study design the excess ratio (O/E), estimated by the smooth function, is directly related to the levels expected during the same time period as the event given by covariates of the model. A fourth approach frequently used when establishing a direct relation between temperature and mortality, and morbidity is modelling the variability of daily deaths as dependent on the variability in daily and previous days' temperatures, adjusting for a range of determinant factors, such as calendar time patterns and time trends (14).

The direct effect of heat on morbidity and mortality

Heat-related health events are likely to increase with global warming unless adaptation occurs. Studies of heat impacts on human physiology have a longstanding history (5). The largest proportion of heat effects are not reported cases of hyperthermia, but mainly increases in cardiovascular and respiratory hospitalisations or deaths (5). Cardiovascular events are mainly a consequence of increased heart rate due to regulation of body

temperature by sweating, which in turn leads to a reduction in blood volume (6). The mechanisms for respiratory deaths are less clear. Recent studies have identified several high risk groups, such as living on top floors, living in institutions, engaging in outdoor activities involving exertion, being very young or very old and being in populations not adapted to heat (5, 7, 16–20). Heat waves are also often associated with increased incidence of dehydration, renal failure and electrolyte disorders, as well as increases in neurological disorders (5, 7, 18).

The number of papers on heat events and mortality has grown rapidly, but the number of studies assessing heat effects on morbidity, for example, the number of hospitalisations, are fewer (9, 21–25). Many studies estimate the general effect of high temperature during summer, utilising distributed lag models on a daily basis, but not the specific effect of extreme heat where there is little or no relief from the heat during the event period. Such events do not necessarily correspond to the same increase in relative risk. The risks are often much higher during heat waves. This was, for example, illustrated by the large excess mortality in France during the heat wave of 2003 (15). In developed countries, where most research on this subject has been undertaken, the effect of heat has been more severe among the very elderly in the population. The opposite pattern has been observed in regions with poorer health conditions, where heat has been shown to be a more important predictor of childhood mortality, for example, in India where infectious diseases dominate causes of mortality. Moreover, the overall effects of high temperatures on hospitalisations have shown different patterns in heat waves (9, 21) compared to general summer temperatures (21, 22). However, few papers have investigated the excess morbidity both during extreme heat events and during summers in general, to show the difference between such exposures.

Established associations, climate and the extreme nature of the 2006 Swedish heat event

The climate in Sweden is rather moderate during the summer. Nonetheless, we have previously established associations between high temperatures, relative humidities and respiratory, cardiovascular and total mortality (excluding external causes), as well as increasing susceptibility over time in Stockholm (27, 28). However, no particular heat extreme or heat wave has been studied. During the summer of 2006, several meteorological stations in the south of Sweden recorded the highest summer mean and July mean temperature since measurements started in the middle of the nineteenth century. At the meteorological station in Lund the mean temperature for July was 21.7°C, against a reference level for the period 1961–1990 of 16.7°C and corresponding standard deviation 1.3 (Personal communication: Markku Rummukainen &

Erik Kjellström, Rossby Center, SMHI). In the wider context, the temperature in Lund for July 2006 was 3.8 standard deviations from the reference mean level and was therefore less extreme in comparison with the 2003 European heat wave, when levels reached almost six standard deviations from the reference mean level (29).

Objective

In this paper we aim to estimate the excess number of daily hospital admissions (all causes excluding external; respiratory and cardiovascular causes) caused by the heat event in 2006 in Skåne, Sweden, by employing four different approaches commonly used in the literature to estimate the impact of heat events on mortality and morbidity:

- (1) standardised event ratios assuming the same frequencies as the mean of the two previous years;
- (2) time series Poisson regression approach predicting the expected excess numbers from trends and calendar patterns;
- (3) time series Poisson regression approach estimating excess numbers with a smooth function; and
- (4) time series Poisson regression approach incorporating predictors of temperature effect in lag strata.

Population and data

We collected cause-specific data on acute (unplanned) hospitalisations at the hospitals in Malmö, Lund, Helsingborg, Trelleborg, Ystad and Landskrona from the National Board of Health and Welfare during the period 1998–2006. Data were aggregated at the daily level by counts of total (excl. external) causes, cardiovascular causes [ICD10:I] and respiratory causes [ICD10:J] for all ages.

Data on daily mean temperatures during the period 1998–2006 were collected from Malmö meteorological station maintained by the Swedish Meteorological and Hydrological Institute.

Methods

Extreme temperatures during 2006 ranged over the whole summer period, so we defined the heat event as the months June–August, to incorporate a potential early summer heat effect due to lack of acclimatisation and lag effects in the later part of the summer.

In the first approach we calculated differences (O–E) and ratios (O/E) of observed and expected counts during the heat event in the study region according to the cause-specific groups studied. The expected count in 2006 was calculated as the mean of the observed counts for the same time period as the heat event during the two preceding years. The observed and the expected counts in 2006 were summed by week, starting June 1st and ending August 30th (total 13 weeks) as were the

standardised event ratios. Confidence intervals (CI; 95%) were calculated for the total number of excess admissions with variance derived from the Poisson distribution for weekly mean frequencies during the reference period (for Poisson distributed events the variance equals the mean) and assuming normal properties for the sum of frequencies.

For the three following approaches we estimated the excess numbers and ratios during the heat event in 2006 by establishing Poisson regression models incorporating smooth functions of between-year and within-year trends (seasonality), and factors describing weekday patterns and national holidays.

Additionally in the second approach we established a generalised additive Poisson model and predicted the expected frequencies during summer 2006 according to time trends, weekday and holiday patterns estimated from the two preceding years. The smooth functions for within-year and between-year trends were allowed a restrictive fixed degree of freedom (df) of 2 and 7, respectively.

Additionally in the third approach we established generalised additive Poisson regression models for the period 1998–2006 and incorporated a smooth function over time (from July 1st until August 30th in 2006) to model the excess risks during the heat event period, as well as a smooth function for within-year and between-year trends. The two latter functions were allowed a fixed df of 12 and 9, respectively. The smooth function for the excess risks during summer 2006 was allowed 9 df as a maximum and was penalised for wiggleness. CIs (95%) for the total excess numbers were derived from the regression models.

The fourth and final approach undertaken was modelling the effect of mean daily temperature on hospitalisation in a time series generalised additive Poisson regression model using smooth functions of moving average temperature lag strata 0–1, 2–6 and 7–13. These functions were penalised if too wiggly and maximally allowed 7 df. In this model we controlled for seasonality, 7 df, and between year time trends, 9 df, by smooth functions with fixed degree of freedom. The smooth functions were less relaxed this time to avoid the control for seasonality affecting the estimates for the daily effect of temperatures. Additional variables included were factors for weekday and holiday patterns. The fourth approach was based on data from the period 1998–2006. A 95% CI for the total excess frequency was derived from standard deviations from the regression model.

The analysis was done in R³⁰ using the *mgcv* package. The generalised cross-validation criteria were used to fit the best model. The assumption on the flexibility of the smooth function for excess risks (approach III) was relaxed allowing more degrees of freedom. However, this resulted only in minor changes in the estimated

Table 1. Descriptive statistics for daily summer temperatures in Malmö 2004–2006

Daily mean temperature (°C) in summer	Mean	Maximum	Minimum
2004	16.0	22.2	11.3
2005	16.7	22.6	10.7
2006	18.4	24.2	11.1

excess effects. The smooth functions for within-year trends as well as the smooth functions for excess risk during the heat event 2006 were based on cyclic spline functions; the other smooth functions were based on cubic splines. The adjusted *R*-squared was between 50 and 75% in all the models, with the highest *R*-squared in

models of all-cause morbidity. In all models fitted we allowed for over-dispersion. Model diagnostics, such as heteroskedacity, normality and response vs. fitted values were graphically examined.

All calculations were based on daily data. However, to simplify interpretation we aggregated some of the results to a weekly basis.

Results

The 95th percentile of daily temperatures in Malmö was 19.4°C over the study period. During the extremely warm summer of 2006 the daily mean temperatures in Malmö were generally above 20°C from July 2nd to August 1st. The maximum daily mean temperature was above the 90th percentile of summer temperature during July, with exception of the period July 12th–15th when the

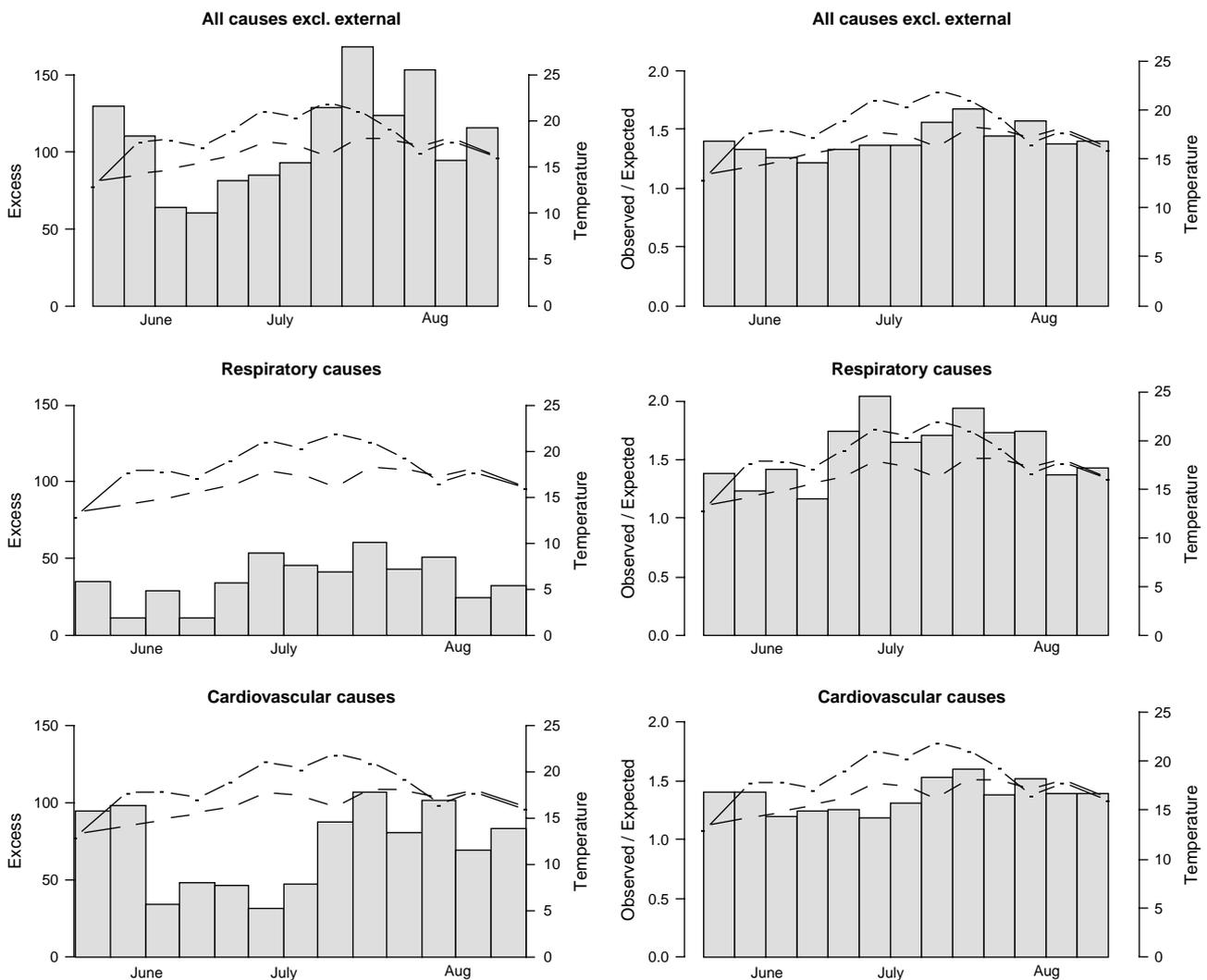


Fig. 1. To the left; excess admissions in summer 2006 derived from observed numbers in summer 2006 minus observed numbers during the two preceding summers. To the right; corresponding ratios of observed to expected numbers. The dot-dashed lines correspond to weekly mean temperatures during summer 2006 and the dashed lines to the weekly mean temperatures during the two preceding summers.

Table 2. The heat event attributable excess admissions in all and cause-specific groups corresponding to the different approaches of estimating excess frequencies with 95% confidence intervals ()

Approach	Total excess admission summer 2006 attributed to heat – all causes excl. external	Total excess admission summer 2006 attributed to heat – respiratory causes	Total excess admission summer 2006 attributed to heat – cardiovascular causes
I Comparing observed expected from means of 2004, 2005	1,406 (1,280, 1,532)	476 (410, 541)	931 (822, 1,039)
II Comparing observed with expected from model with trends and calendar effects 2004–2005	753 (715, 790)	305 (287, 323)	403 (370, 436)
III Attributed to smooth function of excess of summer 2006	157 (122, 193)	146 (128, 164)	–15 (–36, 8)
IV Attributed to short-term variations in temperature 2006	32 (6, 58)	26 (14, 38)	–2 (–24, 20)

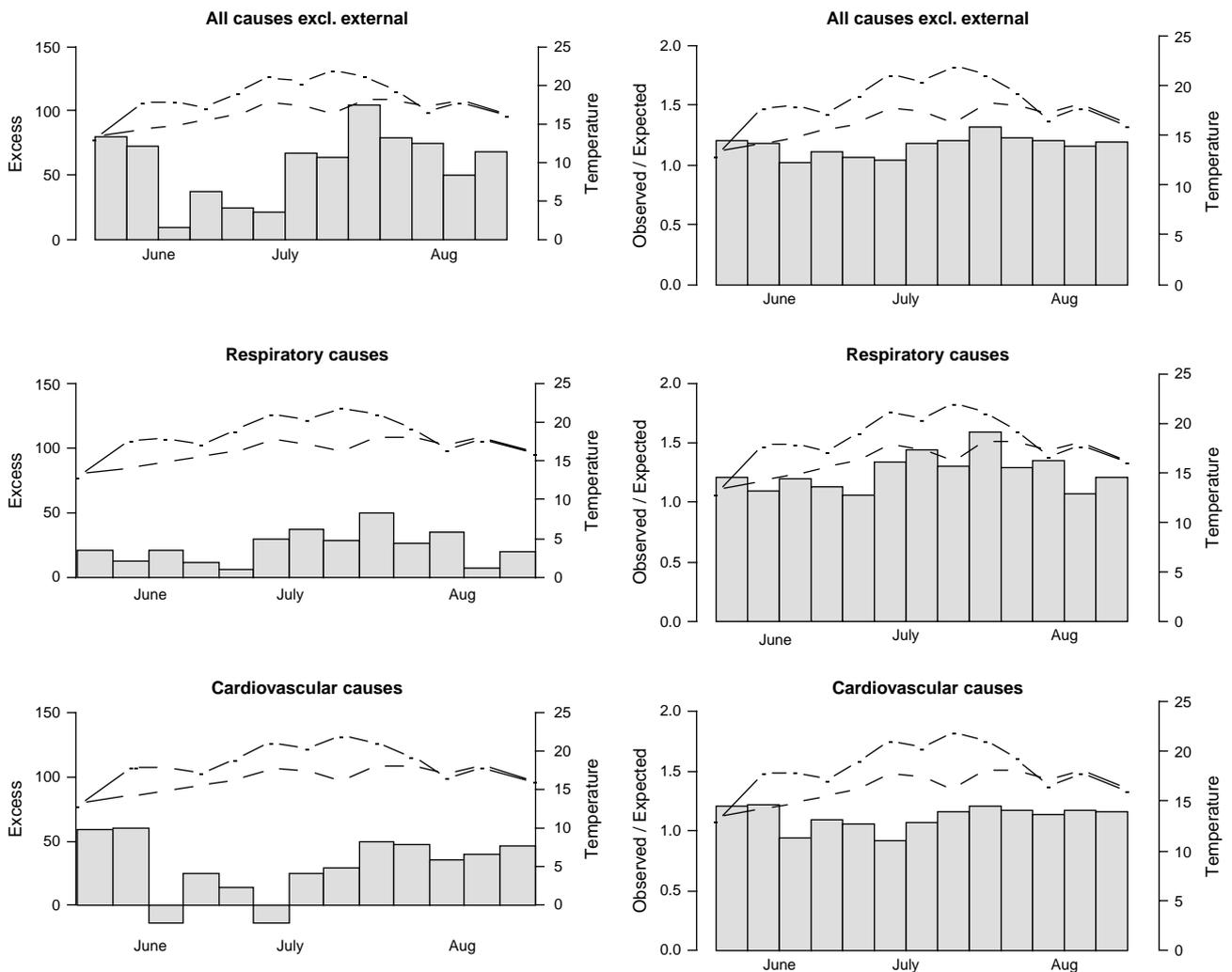


Fig. 2. To the left; excess admissions during summer 2006 as observed minus expected, and to the right; ratios of observed to expected, with expected as predicted from a regression model based on patterns during the two preceding years. The dot-dashed lines correspond to weekly mean temperatures during summer 2006 and the dashed lines to the weekly mean temperatures during the two preceding summers.

temperatures were just below the 90th percentile. The summer mean temperature in Malmö, 2006, was about 2°C warmer compared to the two preceding years (see Table 1).

In Fig. 1, weekly comparison of hospitalisations in all-cause excl. external, respiratory and cardiovascular causes from the first approach are presented as excess numbers (O–E) and standardised admission ratios (O/E) during summer 2006 compared with the two previous summers (2004–2005). The weekly mean temperatures during summer 2006 (dot-dashed) and the mean of the two preceding years (dashed) are also contrasted. According to this approach, the excess number of admissions was generally greatly higher during the whole summer with the largest increase after the warmest period at the end of July and the beginning of August. The relative increase was largest for respiratory admissions

where the risks were up to twice those during the reference period. Generally, during the whole summer, risks of admission were higher than during the two preceding years. The total number of excess admissions during summer 2006 was estimated as 1,406 (95% CI: 1,280–1,532), 476 (95% CI: 410–541) and 931 (95% CI: 822–1,039) in groups of total, respiratory and cardiovascular causes, respectively (see Table 2). The increased risk of cardiovascular admissions was unexpectedly high at the beginning of summer. However, not accounting for an increasing trend in admissions during the study period is likely to bias the estimates from the first approach.

In Fig. 2, the excess numbers and ratios of observed and expected from the second approach are presented for the three groups of hospital admissions. Here the frequencies increased during the warmest part of the summer, but for cardiovascular causes, also at the beginning of summer.

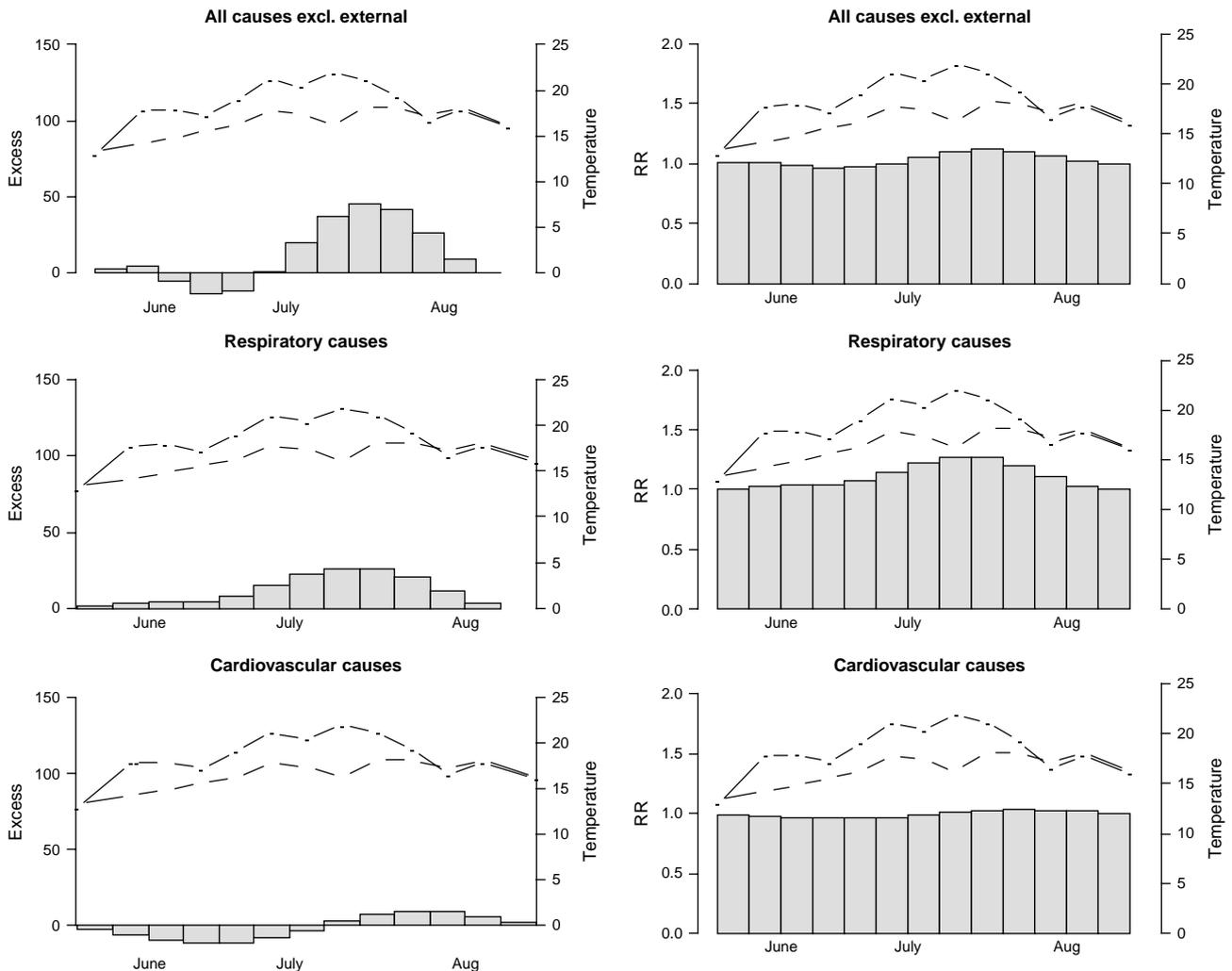


Fig. 3. To the left; excess admissions in summer 2006 derived from a regression model incorporating a smooth function of time of the heat event to capture associated risks, and to the right; corresponding ratios of modelled frequencies (RR) associated with the heat event divided by expected frequencies if no heat event. The dot-dashed lines correspond to weekly mean temperatures during summer 2006 and the dashed lines to the weekly mean temperatures during the two preceding summers.

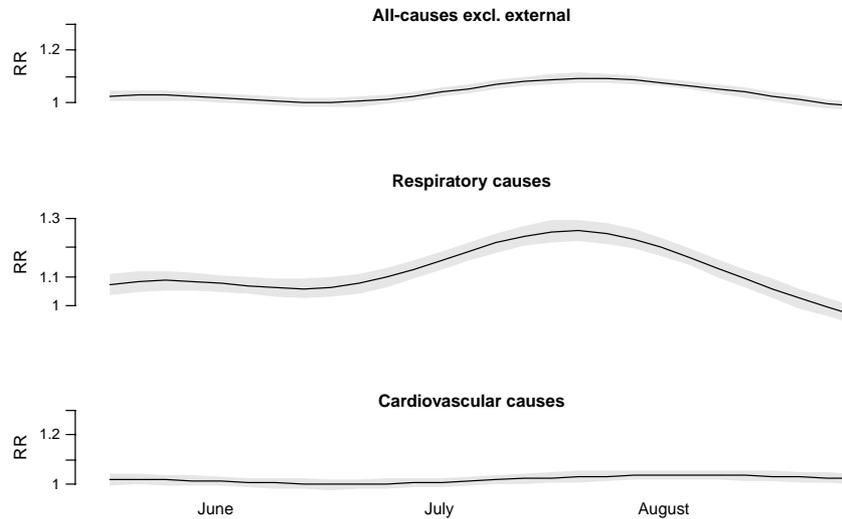


Fig. 4. Smooth functions of excess risk for hospitalisation during the record breaking warm summer of 2006 in cause-specific groups. The shaded region is the 95% confidence intervals.

The relative risk estimates were normalised to increase from approximately one. The total number of excess admissions during summer 2006 was estimated as 753 (95% CI: 715–790), 305 (95% CI: 287–323) and 403 (95% CI: 370–436) in groups of total, respiratory and cardiovascular causes, respectively (see Table 2). In Fig. 3, the excess numbers and ratios associated with the heat event

from the third approach corresponding to the smooth functions of the risks in 2006 are presented. The relative increase was largest for respiratory admissions. Both the group of respiratory admissions and the group of all-causes showed statistically significant increases which were probably due to the heat event. This effect was less apparent in the cardiovascular event group.

In Fig. 4, the smooth functions from this model are presented with 95% CIs in grey. In Fig. 5, the weekly residuals of the model of all-causes are plotted. As can be seen, there were small residual patterns after having fitted the model. However, there were a few disturbing peaks, one of them at the time of the highest temperature levels, that was not accounted for by this function. The total number of excess admissions during the summer of 2006 was estimated as 157 (95% CI: 122–193), 146 (95% CI: 128–164) and –15 (95% CI: –36–8) for all cause, respiratory and cardiovascular admissions, respectively (see Table 2).

In Fig. 6, the fourth approach was applied and the predicted excess attributable to the summer 2006 temperatures was compared with the expected levels of admissions predicted from the mean daily temperature during the two preceding years (all other covariates being the same as 2006). Here, the frequencies increased very little compared with estimates from previous approaches. The total number of excess admissions during summer 2006 was estimated as 32 (95% CI: 6–58), 26 (95% CI: 14–38) and –2 (95% CI: –24–20) for all cause, respiratory and cardiovascular admissions, respectively (see Table 2).

In Fig. 7, the smooth function of the effect of temperature in each lag strata for all causes is presented as risk relative to the minimum point at the curve, with its 95% CI. Generally, all of these functions were significant at the 95% level, except the lag 0–1 function for

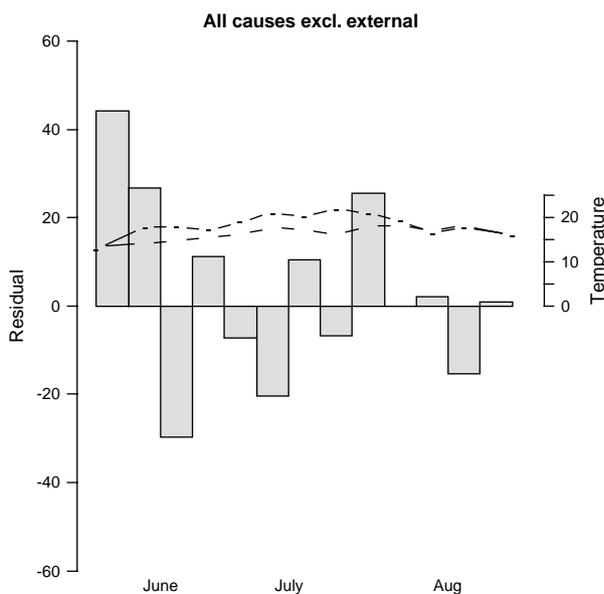


Fig. 5. The observed weekly count of hospital admissions in summer 2006 subtracted from the predicted count from the time series regression model with smooth functions describing excess risks during the heat event, also corresponding to the model residuals during summer 2006. The dot-dashed line corresponds to weekly mean temperatures during summer 2006 and the dashed line to the weekly mean temperatures during the two preceding summers.

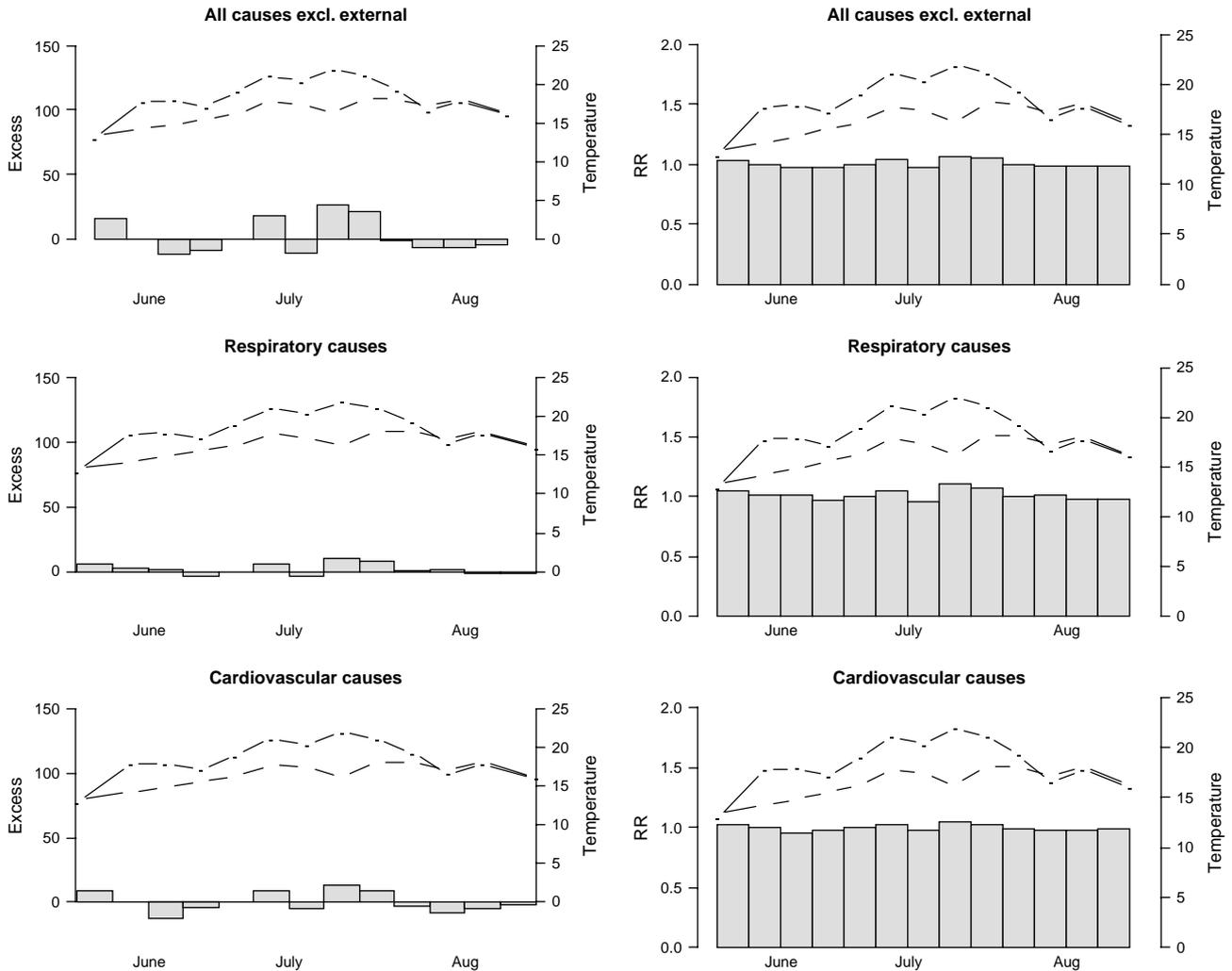


Fig. 6. To the left; weekly admissions in summer 2006 attributed to daily variation of temperature compared with mean temperature levels in 2004 and 2005. The model incorporates smooth functions of temperature effect in lag strata 0–1, 2–6 and 7–13, and to the right; corresponding risks relative to the risk attributed to mean daily temperatures of 2004–2005. The dot-dashed lines correspond to weekly mean temperatures during summer 2006 and the dashed lines to the weekly mean temperatures during the two preceding summers.

respiratory causes. For all causes, the effect of temperature on admission rates was rather weak in the lag strata 0–1, v-shaped in the lag strata 2–6 (increasing with both low and high temperatures) and increased with decreasing temperature in the lag strata of 7–13. It should be noted, however, that these effects of temperature have not been controlled for the influence of influenza, but instead the trend functions were more relaxed to incorporate such effects.

Discussion

We used four different approaches to study the effects of a climatic extreme on hospital admissions, taking the case of a record-breaking warm summer. The results from the four approaches differed to a very large extent, and uncertainties in the effect estimates (CIs) should be of little concern compared to the differences induced by the

study methodologies. The largest excess numbers were estimated in the first approach, not incorporating the increasing frequency of admissions with time. The estimates of the first approach were therefore likely to overestimate excess numbers and risk ratios associated with the heat event. The second approach, which is commonly used, also yielded quite large estimates of excess in all disease groups studied. However, here the ratios were more accurately normalised to the baseline levels of 2006 due to the modelling of the time trend. In the third approach, the excess frequencies due to heat in 2006 were rather low, and may have fallen victim to natural constraints for smooth functions (being restricted to be smooth and not sufficiently well incorporating peaks). It is therefore likely that such a function may have underestimated the effects of the extremely warm summer on hospitalisation rates. This suspicion was augmented

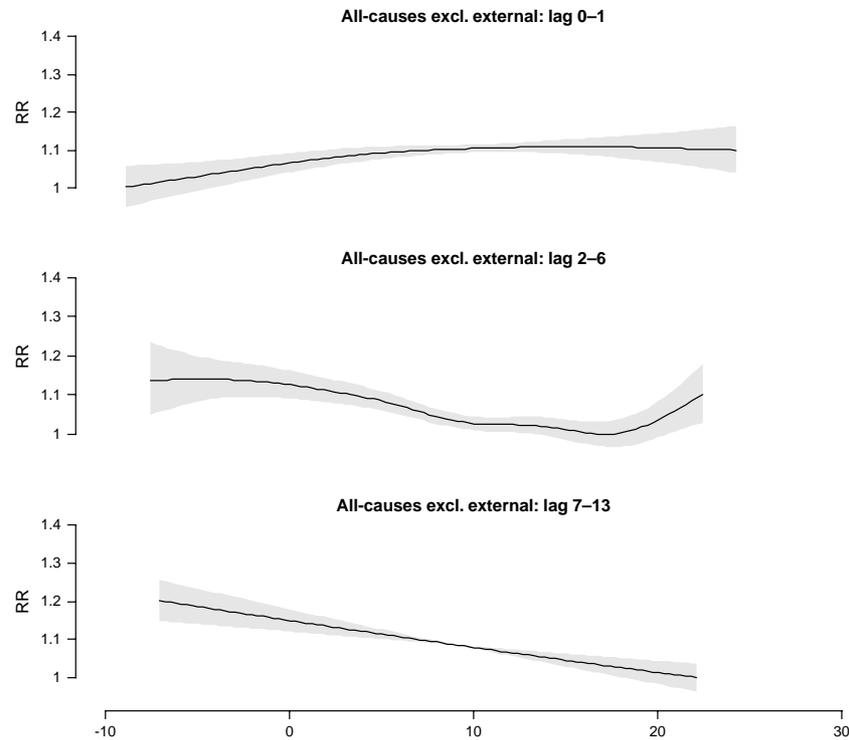


Fig. 7. The risks of hospital admission as a function of temperature in lag strata of 0–1, 2–6 and 7–13. The shaded areas are 95% confidence intervals.

after having plotted the residuals of the model for summer 2006. The fourth approach predicted the rates in 2006 based on modelling the lagged relationship between temperature and hospitalisations during the period 1998–2006. However, this approach (which is well established in studies of heat on mortality) failed to predict any increases in the frequencies of hospitalisation at all. The explanation for this is probably that the approach did not incorporate effects of persistent exposure, which is a unique characteristic in this data only occurring during heat waves, but rather described the short-term variation in admissions due to short-term variability in temperature in general. Previous heat waves have indicated the risks during such extreme conditions may increase much more than predicted from temperature–mortality studies (5, 15). All models showed increased risks during the end of July and start of August, the period when the temperature had been at an extremely high level for about two weeks. According to the second approach, which probably yielded the best estimates, the admissions rates during the last week in July were about 30% higher than normal for this time of year. The time lag between heat and peaking event rates was also a fact during the 2003 heat wave in France (15).

The most obvious difference between the estimates from the second and the third approach was that the second approach estimated high excess levels during the start of June 2006 mainly in cardiovascular disease, while

the third did not. These effects could be due to early summer warm weather, when the population was not acclimatised to high temperatures, but it could also be a pattern caused by something else that started before June 1st and which is therefore not estimated by the smooth function for excess, since this function assumed the excess started on or after June 1st.

In the fourth approach the effect of high temperatures was estimated to be mainly in the lag strata of 2–6, showing a more delayed effect of heat for hospitalisation than for mortality in Sweden (27). Looking at other lag strata we can confirm the contrasting patterns of admissions and temperature found previously (21, 24). We can also confirm the stronger relationship between respiratory admissions and high temperatures previously established (22). This was also the case for mortality and high temperatures in Sweden (27). This, together with the fact that many cardiovascular disease events may have occurred outside of hospitals, may indicate a shortcoming in the health system; a very rapid development of disease states or fatigue/disorientation that made people less likely to seek help and therefore die at home (24). However, the fewer cardiovascular causes may still represent a larger proportion of attributed cases because the incidence rate of cardiovascular disease is about four times greater than the attributed cases of respiratory diseases.

To judge which of these approaches is preferable, based on these data, is hard, but future simulation results may predict the more favourable approach to use. So far, it seems the second approach performed better than the others according to trends and predictions and taking into account the residual peaks of the third approach. It is clear, however, that confidence limits based on a single approach indicate little of the uncertainty that we should show around estimates of excess.

An additional but rarely used method applied to hospital admission rates during heat waves is to define the reference period as a period before the event and a period after the event (23). This approach resembles the third approach used in this study (with a smooth function estimating the excess rates), but avoids the problem with the smooth function not being flexible enough to including spikes caused by the exposure. However, if the heat event episode runs over a longer period of time (as in this case) a seasonal confounding may bias such estimates. Because of this, we did not incorporate that approach into this study.

In the future, climate is likely to show increased frequencies of extreme events. Methodologies for studying health outcomes associated with weather, climate and climate change incorporating time are also necessary when estimating the efficacy of climate change intervention measures. We need to study and reduce such effects as much as possible, not only with a regional but also with a global perspective which targets interventions to areas most affected by climatic change, especially with respect to younger age groups. The effects of climate change on health are complex and a general preparedness should be reached in all countries, irrespective of the scenarios and outcomes, since these may be very different from future impacts. However, when studying the effects of extreme climate on health we need to choose methodological approaches carefully.

Conclusion

Different approaches for studying the effects of an extreme climate event on health can result in highly variable estimates. Further research is needed to identify methods yielding the most accurate estimates. However, one of the approaches used here (the second) seems to have performed better than the others in estimating the excess rates associated with this heat episode. The three first approaches foretold a significant increase in hospital admissions related to the heat episode. However, estimating relative risks of temperature or other determinants of disease may fail to incorporate the specific characteristics of the particular weather event, such as the duration. This means such estimates may be less appropriate for using in predicting the future burden of such events on human health, and in particular the burden of future heat waves.

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References

1. Confalonieri U, Menne B, Akhtar R, Ebi KL, Hauengue M, Kovats RS, et al. Human health. In: Parry ML, Canziani OF, Palutikof JP, van der Linden PJ, Hanson CE, eds. *Climate change 2007: impacts, adaptation and vulnerability. Contribution of Working Group II to the fourth assessment report of the Intergovernmental Panel on Climate Change*. Cambridge: Cambridge University Press; 2007, pp. 391–431.
2. Koelle K, Rodo X, Pascual M, Yunus M, Mostafa G. Refractory periods and climate forcing in cholera dynamics. *Nature* 2005; 463: 696–700.
3. Lindgren E, Gustafson R. Tick-borne encephalitis in Sweden and climate change. *Lancet* 2001; 358: 1731–2.
4. Kovats S, Hajat S. Heat stress and public health. *Annu Rev Public Health* 2008; 29: 41–55.
5. Basu R, Samet JR. Relation between elevated ambient temperature and mortality: a review of the epidemiologic evidence. *J Epidemiol Rev* 2002; 24: 190–202.
6. Michelozzi P, De'Donato F, Bisanti L, Russo A, Cadum E, DeMaria M, et al. The impact of the summer 2003 heat waves on mortality in four Italian cities. *Euro Surveill* 2005; 10: 161–5.
7. Johnson H, Kovats RS, McGregor G, Stedman J, Gibbs M, Walton H. The impact of the 2003 heat wave on daily mortality in England and Wales and the use of rapid weekly mortality estimates. *Euro Surveill* 2005; 10: pii = 558.
8. Conti S, Meli P, Minelli G, Solimini R, Toccaceli V, Vichi M, et al. Epidemiologic study of mortality during the summer 2003 heat wave in Italy. *Environ Res* 2005; 98: 390–9.
9. Semenza JC, McCollough JE, Flanders D, McGeehin MA, Lumpkin JR. Excess hospital admissions during the July 1995 Heat Wave in Chicago. *Am J Prev Med* 1999; 16: 269–77.
10. Whitman S, Good G, Donoghue ER, Benbow N, Shou W, Mou S. Mortality in Chicago attributed to the July 1995 Heat Wave. *Am J Public Health* 1997; 87: 1515–8.
11. Garssen J, Harmsen C, De Beer J. The effect of the 2003 heat wave on mortality in the Netherlands. *Euro Surveill* 2005; 10: 165–8.
12. Grize L, Huss A, Thommen O, Schindler C, Braun-Fahrlander C. Heat wave 2003 and mortality in Switzerland. *Swiss Med Wkly* 2005; 135: 200–5.
13. Rey G, Jougl E, Fouillet A, Pavillon G, Bessemoulin P, Frayssinet P, et al. The impact of major heat waves on all-cause and cause-specific mortality in France from 1971 to 2003. *Int Arch Occup Environ Health* 2007; 80: 615–26.
14. Armstrong B. Models for the relationship between ambient temperature and daily mortality. *Epidemiology* 2006; 17: 624–31.
15. Le Tertre A, Le Franc A, Eilstein D, Declercq C, Medina S, Blanchard M, et al. Impact of the 2003 heat wave on all-cause mortality in 9 French cities. *Epidemiology* 2006; 17: 75–9.
16. Parsons K. *Human thermal environment. The effects of hot, moderate and cold temperatures on human health, comfort and performance*, 2nd ed. New York: CRC; 2003.

17. Kovats RS, Ebi KL. Heatwaves and public health in Europe. *Eur J Public Health* 16: 592–99.
18. Fouillet A, Rey G, Laurent F, Pavillon G, Bellec S, Guihennec-Jouyau C, et al. Excess mortality related to the August 2003 heat wave in France. *Int Arch Occup Environ Health* 2006; 80:16–24.
19. Vandentorren S, Bretin P, Zeghnoun A, Mandereau-Bruno L, Croisier A, Cochet C, et al. August 2003 heat wave in France: risk factors for death of elderly people living at home. *Eur J Public Health* 2006; 16: 583–91.
20. Pirard P, Vandentorren S, Pascal M, Laaidi K, Le Tertre A, Cassadou S, et al. Summary of the mortality impact assessment of the 2003 heat wave in France. *Euro Surveill* 2005; 10: 153–6.
21. Kovats RS, Hajat S, Wilkinson P. Contrasting patterns of mortality and hospital admissions during hot weather and heat waves in Greater London, UK. *Occup Environ Med* 2004; 61: 893–8.
22. Michelozzi P, Accetta G, De Sario M, D'Ippoliti D, Marino C, Baccini M, et al. High temperature and hospitalization for cardiovascular and respiratory causes in 12 European cities. *Am J Respir Crit Care Med* 2009; 179: 383–9.
23. Knowlton K, Rotkin-Ellman M, King G, Margolis HG, Smith D, Solomon G, et al. The 2006 California heat wave: impacts on hospitalizations and emergency department visits. *Environ Health Perspect* 2009; 117: 61–7.
24. Matsrangelo G, Hajat S, Fadda E, Buja A, Fedeli U, Spolaore P. Contrasting patterns of hospital admissions and mortality during heat waves: are deaths from circulatory disease a real excess or an artifact. *Med Hypotheses* 2006; 66: 1025–8.
25. Hansen AL, Peng B, Ryan P, Nischke M, Pisaniello, Tucker G. The effect of heat waves on hospital admissions for renal disease in a temperate city of Australia. *Int J Epidemiol* 2008; 37: 1359–65.
26. Hajat S, Armstrong BG, Gouveia N, Wilkinson P. Mortality displacement of heat-related deaths – a comparison of Delhi, São Paulo, and London. *Epidemiology* 2005; 16: 613–20.
27. Rocklöv J, Forsberg B. The effect of temperature on mortality in Stockholm 1998–2003: a study of lag structures and heat wave effects. *Scand J Public Health* 2008; 36: 516–23.
28. Rocklöv J, Forsberg B, Meister K. Winter mortality modifies the heat-mortality association the following summer. *Eur Respir J* 2009; 33: 245–51.
29. Schär C, Luigi Vidale P, Luthi D, et al. The role of increasing temperature variability in European summer heat waves. *Nature*. 2004; 427: 332–6.
30. R development core team. R: a language and environment for statistical computing. R foundation for statistical computing. Vienna, Austria. Available from: <http://www.R-project.org>; 2007.

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Maintaining health, comfort and productivity in heat waves

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Background: The aim of this paper is to summarise what is known about human response to heat and to use this knowledge to provide guidance on how to maintain the health, comfort and performance of people in heat waves.

Design: The use of power and especially water are critical in providing cooling. A practical method of cooling people in a water bath is described. A warm bath slowly cooled will provide effective cooling but not thermal trauma.

Result: It is concluded that for sedentary and light activities, it is not necessary to cool offices or homes below 25°C for thermal comfort.

Conclusion: To compare the costs due to loss of productivity caused by a heat wave, with the cost of taking action, more research is needed into the relationship between levels of heat stress and how much distraction and 'time off task' it causes.

Keywords: *heat physiology; heat waves; health; comfort; productivity*

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The principles behind how people respond to heat and conditions which would influence health, comfort and human performance have been extensively studied and are well understood (1). To avoid unacceptable heat strain in specific populations and in specific contexts requires the application of those principles into guidelines and heat management systems. How to do that is not well understood.

Heat stress is determined by the *metabolic heat production* of the body, which increases with level of activity (even at rest a person produces around 100 W of heat) and the ability to lose heat to the environment so that heat stored in the body does not raise body temperature to unacceptable levels. *Clothing* will restrict heat loss (or gain) between the skin and the environment, which is determined by *air temperature, radiant temperature, humidity* and *air velocity*. It is the interaction and combined effect of these six factors that determines the level of heat stress.

Heat strain is a consequence of the response of the body to heat stress. Increased heart rate and blood flow to the skin, and sweating can cause illness and death in vulnerable people even when internal body temperatures are at what would normally be considered to be acceptable levels. Sweating can lead to dehydration and as well as increased body temperature can lead to collapse, heat stroke and death.

This paper provides a summary of physiological responses to heat and presents methods for reducing heat strain. The aim of the paper is to consider the application of knowledge of human response to heat, to provide methods for maintaining the health, comfort and productivity of people during a prolonged and atypical period of hot weather (a heat wave).

Physiological response to heat

In heat stress the body temperature may rise and receptors sensitive to change in temperature in the skin, muscle, stomach and other areas of the central nervous system, as well as in the anterior hypothalamus itself, all send signals via the central nervous system to the anterior hypothalamus. The ratio of sodium to calcium ions is also monitored. Where temperatures are above 'set point' levels, blood circulation is controlled in specific areas of the body through the sympathetic nervous system which dilates the cutaneous vascular bed, and hence increases skin blood flow and invokes the sweating mechanism if necessary. This provides greater potential for heat to flow from the body and hence maintain body temperature. Because the heart cannot supply blood to all of the body's organ systems, the autonomic nervous and endocrine system control allocation of blood to competing organs.

During exercise there is an initial sympathetic vasoconstriction, so that blood may flow to active muscles. If

heat is required to be dissipated there is an increased cutaneous blood flow. During continuous work, in the heat, central nervous blood volume decreases as the cutaneous vessels dilate. The stroke volume falls and the heart rate must increase to maintain cardiac output. The effective circulatory volume also decreases as water is lost through sweating.

Sweat glands are stimulated by cholinergic sympathetic nerves and secrete sweat onto the surface of the skin. Sweat rates of 1 L per hour are common and for each litre evaporated, 675 W of heat are lost per hour (2). However, large sweat losses reduce body water content and hence thermoregulatory effectiveness. During sweating, salt is lost at about 4 g per litre (1 g per litre in acclimatised persons). As a normal diet provides 8–14 g of salt per day, then a normal diet is often sufficient. Salt tablets can irritate the stomach and heavier use of salt at meals is preferred, but salt supplementation will normally not be required (2). Potassium is also lost in sweat and a high salt intake may increase potassium loss. In most cases, however, potassium will be replaced by a normal diet (especially fruits and vegetables).

The overall physiological response for continued heat storage is therefore vasodilatation to increase skin temperature and then sweating leading to profuse sweating (including ineffective dripping of some sweat losing insignificant heat but important water). As 'core' temperature continues to rise and the skin is completely wet, hidromeiosis (a reduction in sweating) may occur due to swelling and blocking of sweat glands in the wet humid conditions (3). This is often confused with so-called sweat gland fatigue. The decrease in sweating promotes a further, often rapid, increase in 'core' temperature to beyond 38–39°C, where collapse may occur to above 41°C (rectal temperature) and heat stroke may occur. There will be mental confusion, behavioural changes, failure in central nervous thermoregulation and sweating, and death with eventual denaturing of body protein. National Institute for Occupational Safety and Health (NIOSH) (2) consider age, gender, body fat, drugs (including alcohol) and other non-thermal disorders as important individual factors. There is a large individual variability in the mechanisms of response, which are not fully understood. Physical fitness, however, has been shown to be of great importance. The mnemonic, 'SHAFTS' can be used to advise people how to increase tolerance to heat. This is: Sensible (i.e. appropriate behaviour; reduce exercise or work activity); Hydrated; Acclimatised; Fit; Thin; and Sober (including avoidance of alcohol and other drugs).

Heat physiology and health

Leithead and Lind (4) conclude that heat disorders occur for one or more of three reasons:

- 1) the existence of factors such as dehydration or lack of acclimatisation;
- 2) the lack of proper appreciation of the dangers of heat, either on the part of the supervising authority or of the individuals at risk; and
- 3) accidental or unforeseen circumstances leading to exposure to very high heat stress.

They conclude that many heat-related deaths can be attributed to neglect and lack of consideration and that even when disorders do occur much can be done if all the requirements for the correct and prompt remedial treatment are available. In climates such as those found in Singapore, military personnel are exposed to hot, humid conditions and must carry out essential tasks in protective clothing. Although heat stroke will occur, severe consequences have been avoided by organisational methods including extensive training of personnel and an efficient back-up system to transport casualties rapidly to hospital.

There are a number of classification systems for heat disorders. The mechanisms are summarised in Fig. 1 (5) and a description is provided in Table 1 (6).

There are a number of other complaints related to heat exposure. For example, in mildly sunburnt skin, sweat can be trapped and accumulate under the dead surface layer and cause discomfort as well as reducing evaporative efficiency. In industries where chemicals and particulates are present in the air, they may interact with sweat on the skin surface to cause complaints. The interaction between chemical substances in the air and a sweating person has yet to be fully explored but can be significant. Pollution and poor air quality may be an additional hazard along with heat and in combination may cause and exacerbate respiratory disorders. Protective clothing and equipment may promote sweating and will reduce the ability to evaporate sweat to cool down. This will often increase the risk for the person the clothing is protecting against and dangerous levels can occur at what would normally be considered to be moderate temperatures.

Acclimatisation

Acclimatisation refers to the increased ability of a person to reduce heat strain due to experience of exposure to heat. It takes two forms: behavioural and physiological. Behavioural acclimatisation is most effective and is mainly gained in the context of the heat exposure. If we go to a hot climate, we soon learn to drink appropriately and keep out of the sun. Physiological acclimatisation involves adjustments to our ability to thermoregulate and allow a greater capacity for and more efficient vasodilatation and in particular, sweating. When people become exposed to heat they increase their ability to sweat (7). Significant changes occur even after only three days of exposure. It is possible to gain physiological acclimatisation by exposing

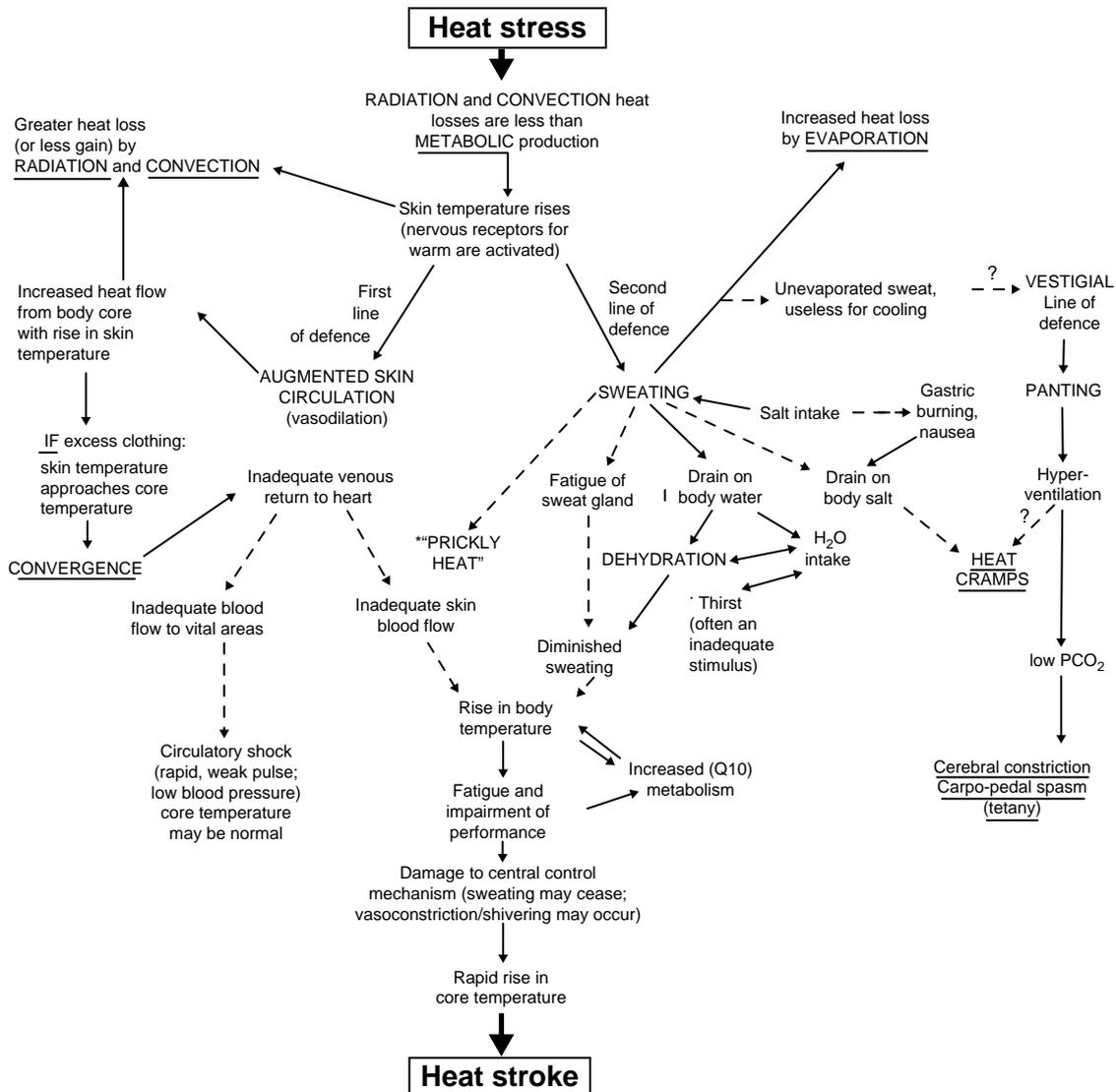


Fig. 1. Causes of heat stroke (from Ref. (5)).

people for a few hours per day to high levels of heat (e.g. in a laboratory – called acclimation) and this is often used as a technique to reduce strain when people are about to travel to a hot climate.

In short, if a person is used to being exposed to heat then he/she will adjust physiologically to some extent and learn how to behave to tolerate the heat. In extremely hot tropical environments this ‘behavioural change’ will include a reduction of physical activity workload, and an increase of frequency and length of rest breaks. If they are not used to the heat and when the weather suddenly becomes hot, then they will be more vulnerable to heat strain.

Maintaining health in heat waves

There is no reason why a person should suffer ill health in a heat wave as the principles of reducing heat strain are

well understood. The requirement is that we have adequate management systems based upon these principles. Physiological responses to heat have generally been studied on fit young males and under conditions that could be regarded as very hot and towards the extremes of weather conditions. For less fit and vulnerable people, physiological responses such as increased heart rate, prolonged sweating and respiratory responses (related to air quality or dryness) will pose a threat. Increased body temperature will eventually be a threat to health, but it may not be the primary cause of illness and death in a heat wave.

So what we do to maintain health? We set up a management system to ensure that people are not exposed to unacceptable heat stress. Heat stress is determined by the interaction of air temperature, radiant temperature, air velocity and humidity. It is also greatly

Table 1. Classification, medical aspects and prevention of heat illness (from (6))

Category and clinical features	Predisposing factors	Underlying physiological disturbance	Treatment	Prevention
1. Temperature regulation heatstroke Heatstroke: (1) hot dry skin usually red, mottled or cyanotic; (2) t_{re} , 40.5°C (104°F) and over; (3) confusion, loss of consciousness, convulsions, t_{re} continues to rise; fatal if treatment delayed	(1) Sustained exertion in heat by unacclimatised workers; (2) lack of physical fitness and obesity; (3) recent alcohol intake; (4) dehydration; (5) individual susceptibility; and (6) chronic cardiovascular disease	Failure of the central drive for sweating (cause unknown) leading to loss of evaporative cooling and an uncontrolled accelerating rise in t_{re} , there may be partial rather than complete failure of sweating	Immediate and rapid cooling by immersion in chilled water with massage or by wrapping in wet sheet with vigorous fanning with cool dry air, avoid overcooling, treat shock if present	Medical screening of workers, selection based on health and physical fitness, acclimatisation for 5–7 days by graded work and heat exposure, monitoring workers during sustained work in severe heat
2. Circulatory hypostasis heat syncope Fainting while standing erect and immobile in heat	Lack of acclimatisation	Pooling of blood in dilated vessels of skin and lower parts of body	Remove to cooler area, rest recumbent position, recovery prompt and complete	Acclimatisation, intermittent activity to assist venous return to the heart
3. Water and/or salt depletion (a) Heat exhaustion (1) Fatigue, nausea, headache and giddiness; (2) skin clammy and moist; complexion pale, muddy or hectic flush; (3) may faint on standing with rapid thready pulse and low blood pressure; (4) oral temperature normal or low but rectal temperature usually elevated (37.5–38.5°C) (99.5–101.3°F); water restriction type; urine volume small, highly concentrated; salt restriction type; urine less concentrated, chlorides less than 3 g/L	(1) Sustained exertion in heat; (2) lack of acclimatisation; and (3) failure to replace water lost in sweat	(1) Dehydration from deficiency of water; (2) depletion of circulating blood volume; (3) circulatory strain from competing demands for blood flow to skin and to active muscles	Remove to cooler environment, rest recumbent position, administer fluids by mouth, keep at rest until urine volume indicates that water balances have been restored	Acclimatise workers using a breaking-in schedule for 5–7 days, supplement dietary salt only during acclimatisation, ample drinking water to be available at all times and to be taken frequently during work day
(b) Heat cramps Painful spasms of muscles used during work (arms, legs or abdominal); onset during or after work hours	(1) Heavy sweating during hot work; (2) drinking large volumes of water without replacing salt loss	Loss of body salt in sweat, water intake dilutes electrolytes, water enters muscles, causing spasm	Salted liquids by mouth or more prompt relief by I-V infusion	Adequate salt intake with meals; in unacclimatised workers supplement salt intake at meals

Table 1 (Continued)

Category and clinical features	Predisposing factors	Underlying physiological disturbance	Treatment	Prevention
4. Skin eruptions				
(a) Heat rash (miliaria rubra; 'prickly heat')				
Profuse tiny raised red vesicles (blister-like) on affected areas, pricking sensations during heat exposure	Unrelieved exposure to humid heat with skin continuously wet with unevaporated sweat	Plugging of sweat gland ducts with retention of sweat and inflammatory reaction	Mild drying lotions, skin cleanliness to prevent infection	Cool sleeping quarters to allow skin to dry between heat exposures
(b) Anhydrotic heat exhaustion (miliaria profunda)				
Extensive areas of skin which do not sweat on heat exposure, but present gooseflesh appearance, which subsides with cool environments; associated with incapacitation in heat	Weeks or months of constant exposure to climatic heat with previous history of extensive heat rash and sunburn	Skin trauma (heat rash; sunburn) causes sweat retention deep in skin, reduced evaporative cooling causes heat intolerance	No effective treatment available for anhydrotic areas of skin, recovery of sweating occurs gradually in return to cooler climate	Treat heat rash and avoid further skin trauma by sunburn, periodic relief from sustained heat
5. Behavioural disorders				
(a) Heat fatigue – transient				
Impaired performance of skilled sensorimotor, mental or vigilance tasks, in heat	Performance decrement greater in unacclimatised and unskilled worker	Discomfort and physiologic strain	Not indicated unless accompanied by other heat illness	Acclimatisation and training for work in the heat
(b) Heat fatigue – chronic				
Reduced performance capacity, lowering of self-imposed standards of social behaviour (e.g. alcoholic over-indulgence), inability to concentrate, etc.	Workers at risk come from temperate climates, for long residence in tropical latitudes	Psychosocial stresses probably as important as heat stress, may involve hormonal imbalance but no positive evidence	Medical treatment for serious cases, speedy relief of symptoms on returning home	Orientation on life in hot regions (customs, climate, living conditions, etc.)

influenced by the clothes a person is wearing and the activity they are performing. Consideration of these six factors must form the basis of any management programme. A full discussion of management programme for heat waves is provided in (8). Of particular importance will be the availability of household water and electrical power. Moving people away from hot homes to cooler areas (e.g. public libraries or modern shopping centres) will reduce heat strain but may not be practicable. The use of water provides an effective and often practical method of keeping cool in the heat. Simply plunging the hands and feet into cool water reduces thermal strain. Immersing hands and arms in cool water is a practical way for vulnerable people to avoid health problems in the heat. For those with a bathtub, running a normal warm bath (e.g. 38°C water temperature) to half full and getting in, then running in cool water to fill the bath allows a non-stressful method of keeping cool. The initial warm water will promote vasodilatation and will not cause thermal shock, which will occur with cool water and raise blood pressure. The body will not detect the slow cooling and at around 30°C a 'cool' bath will greatly enhance heat loss.

Spraying water on the face or exposed skin and enhancing evaporative cooling with fans, for example, will reduce heat loss even when the air temperature is above desired internal body temperature. The direction and strength of fans, however, has to be controlled as evaporation of mucous from the airways and moisture from the eyes can cause irritation and respiratory problems.

The use of air conditioning is very effective in reducing air temperature and hence thermal strain. It requires electrical power, however, and if electric grid systems cannot meet capacity then lack of power can cause a critical problem. The above discussion provides methods for keeping cool in a heat wave. It is not exhaustive and the reader is referred to (8) for a complete discussion.

Maintaining comfort in heat waves

Conditions for thermal comfort are well described by Fanger (9) and were later adopted worldwide in ISO 7730 (10). They are, that for a person to be in thermal comfort, he/she must be in *heat balance* (maintaining internal body temperature) and that *skin temperatures and sweat rates must be within comfort limits*. A method is derived from those three premises that allows the prediction (from air temperature, radiant temperature, air velocity, humidity, clothing and activity) of thermal comfort conditions and of any thermal discomfort. The predicted mean vote (PMV) index predicts the mean rating of a large group of people exposed to thermal conditions (six factors above) on the scale: +3 hot; +2 warm; +1 slightly warm; 0 neutral; -1 slightly cool; -2 cool; -3 cold. For a given

PMV value, the predicted percentage of dissatisfied (PPD) provides the likely level of dissatisfaction.

Parsons (1) and Hodder and Parsons (11) extend the scale from 7 to 11 points by adding very hot/cold and extremely hot/cold on either end. They then provide a simple rule of thumb method for taking account of the direct sun on a person. For every 200 Wm⁻² of solar radiation (from 0 Wm⁻² totally cloudy to 600 blue sky to 1,000 absolute maximum), the PMV (now PMV_{solar}) is increased by one scale value. It is emphasised that this rule of thumb provides a simplistic practical method which accounts for main effects. For a more accurate assessment, direct and diffuse radiation, depending upon turbidity and cloudiness, elevation of the sun, posture of the person and more, also play a role but involve increasingly complex analysis.

To take account of the ability for people to change behaviour or adapt to the environment, Parsons (1) suggested an I_{equiv} index which allows the clothing insulation value, used in the calculation of PMV, to be adjusted to take account of the effects of any adaptive opportunities in the environment (e.g. ability to reduce clothing, open windows, etc.). The I_{equiv} method provides a simplistic but direct method of accounting for adaptive opportunities available to people. Estimates of adaptive opportunity, however, remain necessarily subjective. There are other, less rational or 'causal' adaptive models which relate indoor comfort temperatures to outside conditions based upon data from thermal comfort surveys (e.g. 12). The 'causal' model assumed in such models is that the effects of the adaptive opportunity, such as that used in the I_{equiv} method, are captured in the behaviour of people to outside conditions, for example, a reduction in clothing when outside temperatures are high. The I_{equiv} method suggests that a consequence of this is to provide the suggestion that for reasonable levels of adaptive opportunity, it will not be necessary to cool offices or homes below 25°C to provide thermal comfort. This measure is related to sustainable thermal comfort discussions (13) and will also have the effect of significantly reducing electrical power requirements and hence avoiding power failure. It is interesting to note that in Japan a few years ago, the government in Tokyo ran a campaign to set a lower limit for cooling offices to 28°C air temperature. The Prime Minister at the time advised businessmen not to wear a tie. The 28°C limit clearly requires less energy to achieve in hot outside conditions than a limit of 25°C or below, however, it is likely to cause some discomfort. The practical point is that it is a common practice to cool rooms to around 20°C (and often causes cold discomfort) in hot outside conditions. This is an unnecessary and inefficient use of energy. Energy saving by not cooling below 25°C would be highly significant worldwide and it is generally agreed would still allow thermal comfort.

Maintaining productivity in heat waves

The most effective way to maintain human performance and productivity during a heat wave is to provide thermal comfort conditions. This is not always possible, however, and in hot conditions productivity is likely to fall. This is by no means certain, however, and motivation, morale and team spirit will all be significant factors. There have been numerous studies into the effects of heat on manual dexterity and cognitive performance. The most direct and measurable effect on productivity, however, is ‘time off task’ (1) caused by work having to be stopped or due to heat causing a distraction to people (e.g. concentrating or finding fans or adjusting control systems or simply paying attention to the heat). This will apply to people in homes, offices and other work places. The relationship between the level of distraction caused by heat and the level of heat stress is not known. Further investigation is needed if the full economic costs of heat waves are to be determined. For practical purposes the costs in terms of health and loss in productivity can be weighed against the costs of systems for ensuring the avoidance of unacceptable thermal strain during heat waves.

Conclusions

- 1) Much is known about human response to heat and basic principles can be used to provide guidance on effective heat management systems.
- 2) With electrical power and especially water, it is possible to avoid health problems due to heat.
- 3) For sedentary and light activities it is not necessary to cool rooms below 25°C to provide thermal comfort.
- 4) There is a need to determine the relationships between levels of heat stress and how much distraction and time off work it causes.

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References

1. Parsons K. Human thermal environments. London: Taylor & Francis; 2003.
2. NIOSH. Occupational exposure to hot environments. DHHS (NIOSH) Publication No. 86-113. Washington, DC: National Institute for Occupational Safety and Health; 1986.
3. Kerslake D Mck. The stress of hot environments. Cambridge: Cambridge University Press; 1972.
4. Leithead CS, Lind AR. Heat stress and heat disorders. London: Cassell; 1964.
5. Belding HS. The search for a universal heat stress index. In: Hardy JD, Thomas CC, eds. Physiological and behavioural temperature regulation. IL: Springfield; 1970.
6. Goldman RF. Standards for human exposure to heat. In: Mekjavic IB, Banister EW, Morrison JB, eds. Environmental ergonomics. London: Taylor & Francis; 1988. pp. 99-136.
7. Clark RP, Edholm OG. Man and his thermal environment. London: Edward Arnold; 1985.
8. Menne B, Ebi KI. Climate change and adaptation strategies for human health. Darmstadt: Springer; 2006.
9. Fanger PO. Thermal comfort. Copenhagen: Danish Technical Press; 1970.
10. ISO 7730. Ergonomics of the thermal environment – analytical determination and interpretation of thermal comfort using calculation of the PMV and PPD indices and local thermal comfort. Geneva, Switzerland: ISO; 2005.
11. Hodder S, Parsons KC. The effects of solar radiation on thermal comfort. *Int J Biometeorol* 2006; 51: 233-50.
12. De Dear RJ, Brager GS. Developing an adaptive model of thermal comfort and preference. *ASHRAE Transactions*; 1998.
13. Parsons K. Sustainable thermal comfort. In: Hall M, ed. Materials for energy efficiency and comfort in occupied spaces. Woodhead Publishing; in press.

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Workplace heat stress, health and productivity – an increasing challenge for low and middle-income countries during climate change

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Background: Global climate change is already increasing the average temperature and direct heat exposure in many places around the world.

Objectives: To assess the potential impact on occupational health and work capacity for people exposed at work to increasing heat due to climate change.

Design: A brief review of basic thermal physiology mechanisms, occupational heat exposure guidelines and heat exposure changes in selected cities.

Results: In countries with very hot seasons, workers are already affected by working environments hotter than that with which human physiological mechanisms can cope. To protect workers from excessive heat, a number of heat exposure indices have been developed. One that is commonly used in occupational health is the Wet Bulb Globe Temperature (WBGT). We use WBGT to illustrate assessing the proportion of a working hour during which a worker can sustain work and the proportion of that same working hour that (s)he needs to rest to cool the body down and maintain core body temperature below 38°C. Using this proportion a 'work capacity' estimate was calculated for selected heat exposure levels and work intensity levels. The work capacity rapidly reduces as the WBGT exceeds 26–30°C and this can be used to estimate the impact of increasing heat exposure as a result of climate change in tropical countries.

Conclusions: One result of climate change is a reduced work capacity in heat-exposed jobs and greater difficulty in achieving economic and social development in the countries affected by this somewhat neglected impact of climate change.

Keywords: *climate change; work; heat; occupational health; productivity*

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Global climate change will affect living and working environments, and create health threats for millions of people (1, 2). The average global temperature is increasing and it is estimated that it will go up a further 1.8–4.0°C (estimated average 3.0°C) by 2100 (1), depending on actions to limit greenhouse gas emissions. The extent of *local climate change* will vary depending on geographic and local meteorological conditions. Modern urban development can add several degrees to local temperatures through heat absorption in concrete buildings, road tar-seal, etc.: the 'urban heat island effect' (3).

Increasing local ambient temperature means higher human exposure to heat, which during hot seasons in hot parts of the world can create very unhealthy

environments for people who are not able to protect themselves with air conditioning or other cooling methods. Both general living environments and working environments are affected. The latter may create impacts both on workers' health and on economic conditions (4). Workers in low and middle-income tropical countries are likely to be at highest risk of excessive heat exposure.

The aim of this paper is to briefly introduce the most commonly used occupational heat stress index and how working people are likely to be affected at different heat exposure levels, an important aspect of the health effects of climate change (5). We will highlight the potential impacts of increasing heat exposure due to climate change in low and middle-income tropical countries.

Physiological and clinical impacts of heat stress

The human body is designed to maintain a core body temperature of 37°C. A person carrying out physical activity (for instance while working) creates metabolic heat inside the body, which needs to be transferred to the person's external environment in order to avoid a dangerous increase of core body temperature (6). The body heat balance is determined by the 'six fundamental factors' (6):

- (1) air temperature;
- (2) radiant temperature;
- (3) humidity;
- (4) air movement (wind speed);
- (5) clothing; and
- (6) the metabolic heat generated by human physical activity.

If cooling via sweating and convection (via contact with cooler air and air movement) is not sufficient, the metabolic heat generation needs to be reduced to avoid heat strain and heat stroke (7). This creates limits to the extent to which physical activity and work output can be maintained without rest periods.

When physical activity is high in a hot working environment, the worker is at risk of increased core body temperature (above 38°C), diminished physical work capacity (8, 9), diminished mental task ability (10), increased accident risk (11) and eventually heat exhaustion or heat stroke (12). The main factor underlying these effects is the increased core body temperature (13), but dehydration due to sweating and inadequate liquid intake is also of major importance (14). Symptomatic exhaustion and clinical diseases, particularly kidney disease (14), can be the result of excessive dehydration (9).

When body temperature exceeds 39°C, acute heat disorders (heat stroke) may occur, and above 40.6°C life-threatening 'severe hyperpyrexia' starts to occur (13). Many of these references may look outdated, but the fact is that much of the relevant research was carried out several decades ago, and more recent studies have confirmed the understanding of the basic mechanisms (9).

Assessing workplace heat stress

To protect workers from the effects of heat exposure 'heat stress indices' and protective guidelines have been developed (7). The most commonly used in occupational health is the Wet Bulb Globe Temperature (WBGT) index developed by the US Army many decades ago (15). This index takes into account air temperature, radiant temperature, humidity and air movement, and is the basis for time limitations of work in different heat exposure standards. Other indices of heat exposure (e.g. heat stress

index, index of thermal stress, predicted four-hour sweat rate) (8) are likely to be correlated to WBGT.

The WBGT is a combination of three local climate measurements (16): the natural wet bulb temperature, T_{nwb} ; the globe temperature, T_g ; and the air temperature, T_a . $WBGT\text{-outdoors} = 0.7 \times T_{nwb} + 0.2 \times T_g + 0.1 \times T_a$; $WBGT\text{-indoors} = 0.7 \times T_{nwb} + 0.3 \times T_g$. Special equipment is required to measure T_{nwb} and T_g (6), and these variables are not routinely measured at weather stations. Descriptions of the equipment and the physical science basis for the three temperature measurements can be found on web-sites or in textbooks (6).

In order to assess time trends of human heat exposure in the past and likely future trends during climate change, it would be very useful if available weather station data could be used to estimate WBGT. However, the relationships between the different variables are complex.

Different mathematical models to calculate WBGT from weather station data have been developed and a recent one, based on a detailed analysis of the physical principles behind heat and energy transfer (17), appears the most accurate. The computer software can be obtained from the authors on request (17). This elaborate model makes it possible to compare calculated WBGT estimates for different seasons and places.

Occupational heat exposure guidelines based on WBGT (16, 18) state maximum heat exposures in jobs at different work intensity (in Watts). The international standard (18) presents the proportions of work hours during which workers need to take rest periods, depending on work intensity and WBGT, in order to avoid the core body temperature exceeding 38°C for an average worker. A table in the international standard presents WBGT 'reference values' (the point at which some preventive action should be taken) (Table 1) shows the WBGT levels that require no hourly rest, or rest to the extent of 25, 50 and 75% (rest/work ratios) during the working hour. The US guidelines (16) also includes a WBGT level at which no work should be carried out without special protective clothing at a higher level of heat exposure. These standards have been summarised in Table 2.

At light work intensity (200 Watts) the need for rest periods each hour starts at a WBGT of 31°C, while at heavy work intensity (500 W) this threshold occurs at a WBGT of approximately 25.5°C (Table 2). The need for preventive actions to avoid excessive heat exposure starts even earlier in accordance with the 'reference values' (30 and 23°C, respectively; Table 2).

The US Army and Air Force has issued advice on heat effect prevention (15) that is similar to the levels in Table 1, but this advice naturally assumes that a soldier can cope with somewhat more heat. For example, at $WBGT = 32^\circ\text{C}$ a soldier doing moderate work in relatively light clothing

Table 1. Reference values for WBGT (°C) at different work intensity levels (in Watts = W), light clothing

Metabolic rate class ^a (work intensity)	0 (rest)	1 (light work)	2 (medium work)	3 (intense work)	4 (very intense work)
Approximate metabolic rate, M (W)	100	200	300	400	500
WBGT reference values ^b (°C)	33	30	28	25	23

^aThe metabolic rate classes are: 0 =resting, M <117 W; 1 =light work, 117 <M <234 W; 2 =sustained medium level work, 234 <M <360 W; 3 =intense work, 360 <M <468 W; 4 =very intense work, M >468 W.

^bThe ISO standard (18) says: 'If these values are exceeded, it is necessary either to reduce the direct heat stress at the workplace, or to carry out a detailed analysis of exposure and prevention.'... 'These values represent the mean effect', so short peak exposures may be acceptable. However, the values are set to avoid over-heating (>38°C) in 'almost all individuals'. Thus, some people would be more sensitive and risk over-heating.

Note: Based on recommendations from the United States National Institute of Occupational Safety and Health (NIOSH) (16) and the International Standards Organization (ISO) (18), if the worker uses heavier clothing or protective clothing, these values need to be reduced, see ISO (18).

(425 W) is allowed to work 20 minutes of every hour, while the National Institute for Occupational Safety and Health (NIOSH) recommendation is for 15 minutes work per hour. As pointed out in Table 2, all of these recommendations depend on the clothing worn. The heavier the clothing is, the more rest time is required.

The WBGT is not considered ideal as an occupational heat stress index for individual work situations (19, 20) and other alternatives have been proposed; e.g. the Required Sweat Rate index (21), the Predicted Heat Strain index (22) and the Thermal Work Limit (23, 24). For the purposes of this paper we used WBGT primarily as an illustration of one of the potential consequences at population level of global climate change.

Table 2 shows a strikingly narrow WBGT range between the heat exposure level that is acceptable for continuous workplace exposure (e.g. 25.5°C at 500 W) and a 75% rest time requirement (31°C at 500 W). Kjellstrom (4) defined work capacity as the percentage of a working hour that a worker can perform his/her intended work. If no rest time is needed, because of heat, during a working hour, then the work capacity is 100%. If 75% rest time is needed, the work capacity is 25%, etc. Using this

approach, Table 2 can be used to estimate loss of work capacity in heat exposed occupations for every hour of the day.

Impacts of workplace heat exposure on clinical health

Heat-related deaths at work have occasionally been reported, starting with classical studies in South Africa (25). A recent study of 423 heat-related deaths among agricultural workers in the USA, 1992–2006 (26) shows that the problem is still occurring. There are few systematic studies in low and middle-income countries of deaths or serious clinical heat stroke among heat-exposed workers, except for India where considerable research on the physiology of heat exposure and preventive approaches has been carried out by Nag and colleagues at the National Institute of Occupational Health. For instance, one study using experimental exposure chambers quantifies the 'tolerance time' of work at different intensities until core body temperature reaches 39°C (27). At a WBGT of 34°C, the tolerance time in heavy work goes below one hour, and it reduces by 4–5 minutes per 1°C increase of WBGT (27). These WBGT thresholds for 'safe' hourly continuous work

Table 2. Recommended maximum WBGT exposure levels (°C) at different work intensities and rest/work ratios for an average acclimatised worker wearing light clothing^a

Metabolic rate class (work intensity)	1 (light work) WBGT (°C)	2 (medium work) WBGT (°C)	3 (heavy work) WBGT (°C)	4 (very heavy work) WBGT (°C)
Continuous work, 0% rest/hour	31	28	27	25.5
25% rest/hour	31.5	29	27.5	26.5
50% rest/hour	32	30.5	29.5	28
75% rest/hour	32.5	32	31.5	31
No work at all (100% rest/hour) ^b	39	37	36	34

^aThese WBGT values are taken from a graphic in the international standard (ISO, 18) and are approximate.

^bFrom recommendations by NIOSH (16).

are higher than those in Tables 1 and 2, but this is a natural result of using 39°C as an acceptable core body temperature rather than 38°C as in the tables. The latter temperature provides a greater safety margin for heat stroke among the workers who are sensitive to heat exposure.

The physiological basis for the different levels of clinical health damage was described briefly above and more details are given by Parsons (6) and Bridger (9). Beyond the acute heat stress, more chronic effects on the heart and kidneys may develop after repeated excessive body heating or dehydration (9).

Impacts of workplace heat exposure on worker productivity

The relationship between occupational heat exposure and productivity was pointed out as long ago as 1974 by Axelsson (28) and was further commented upon by Holmer (29), but very little research has been carried out aiming at quantifying this relationship in work situations where workers are 'self-paced'. The slowing down of work as a defence mechanism during severe heat exposure is labelled 'autonomous adaptation' by climate change researchers (30). Productivity has also been analysed for indoor climates in relation to air conditioning needs (6). The first report on this issue in the context of global climate change (4) likened the heat effect on work output to the 'disability' caused by defined diseases, and concluded that this effect may contribute to disability in a population to a greater degree than most diseases.

A number of recent studies have analysed different aspects of the effects of heat exposure on productivity. In indoor environments, increased heat exposure reduces performance (31–33) and reducing humidity of office air in the tropics was shown to improve the perception of the work environment (34). In Bangladesh, heat reduced work performance in metal workshops (35).

Protective clothing increased heat stress and reduced performance (36).

Heat stress is likely to be common during hot seasons, but culturally accepted methods to reduce impacts on health and work capacity (such as 'siesta') are generally effective in avoiding serious health impacts. However, these culturally accepted methods will undoubtedly reduce the hourly productivity of the exposed workers. More research to document these conditions is needed to make accurate estimates of the impacts of climate change.

Modelling the impacts of workplace heat exposure

Lemke and Kjellstrom (to be published) used the model by Liljegren et al. (17) to calculate WBGT based on daily weather data for a selection of cities in countries with hot seasons. The WBGT levels during the hot seasons are very high in outdoor work where sun exposure is a major contributor to high WBGTs. For instance, in Delhi the calculated WBGTs during afternoons in May (the hottest month) reach above 30°C on average (Fig. 1). The resulting work capacity during different hours for a person who works at a heavy work intensity of 500 W is very low: on average only 20% of work capacity remains at 12 noon (Fig. 1). In order to avoid the midday work capacity loss, people use 'siesta', night work, or similar approaches to work primarily during less hot parts of each 24-hour period. However, night work is not possible for workers who rely on daylight to see their worksite (e.g. poor farmers in tropical countries). Additional examples are provided in recent technical reports (37, 38) where more detail about the methods used and the results for major cities are given.

Climate change and heat stress trends in tropical countries

The ongoing global climate change has until now been described primarily in terms of the average global

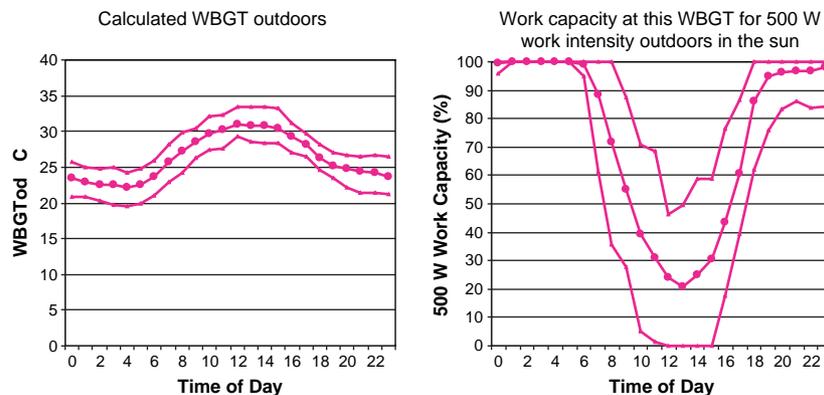


Fig. 1. Calculated WBGT outdoors in the sun and resulting work capacity at 500 W work intensity during different hours of a 24-hour period in Delhi, India, May 1999 (based on hourly weather data from the NOAA database; the middle line is the monthly mean, the other lines the 5 and 95 percentiles of recordings on individual days during that month).

Table 3. Summary of recent temperature trends in selected cities from 1980 to 2007 according to regression lines based on NOAA database weather station data (source (37))

City	Time trends, fitted line, temperature increase for		
	Maximum temp. (°C/century)	Average temp. (°C/century)	Minimum temp. (°C/century)
Johannesburg	+2.97	-1.27	-3.86
Atlanta	-0.89	+1.85	+3.77
Managua	+0.27	+1.29	+3.51
Cairo	+2.62	+4.84	+6.58
Athens	+4.09	+5.30	+5.14
Delhi	+2.08	+0.53	+0.18
Chennai	+2.87	+0.41	-0.43
Bangkok	+4.52	+5.19	+5.37
Chiang Mai	-1.13	+0.22	+0.77
Osaka	+4.76	+5.25	+5.96
Shanghai	+7.06	+7.77	+11.79

Note: Temperature change per century presented like this is comparable with the IPCC estimates for the twenty-first century, but this approach does not imply that the changes will be linear and continuous for 100 years; change per decade could be more appropriate.

temperature change. To describe changes in workplace, heat stress requires trend analysis of local temperature, humidity, wind speed and solar radiation. We will show temperature trends for selected locations as indicators of the likely trends of actual heat stress, but more detailed analysis is required for projections of future heat stress impacts.

The highest expected temperature increase due to climate change is expected close to the North Pole (1), which creates major problems for the environment, but few people live there. Densely populated areas around the planet that are expected to get the highest temperature increases due to global climate change are mainly inland areas within the large continents with an increase of 1–3°C by 2020 and 3–5°C by 2080 (1). In many of these areas the maximum temperatures during the hottest part of the year are already close to 40°C (examples in Kjellstrom et al. (37) and increasing over time (Table 3). An additional 3–5°C will make heavy work (e.g. in agriculture and construction work) very difficult during the hottest periods in most of these cities and in tropical countries in general. The ongoing changes of temperature are caused partly by the ‘heat island effect’ in many of these cities and may only partly be due to global atmospheric change.

Modelling by the Intergovernmental Panel on Climate Change (1) forecasts substantial increases of future annual average temperatures (and in many places also increases of humidity) in areas populated by billions of people, and it is likely that for many workers increasing WBGT index levels will affect their work capacity and create health risks. The eventual occupational impacts of such increasing heat exposure are dependent on shading from trees or roofs, clothing, radiated heat and

wind speed in workplaces, but it is most likely that global climate change is a threat to safe, comfortable and productive thermal working environments for a significant part of the global population. To limit these impacts, urban planning and workplace design should consider the impacts of climate change.

Conclusions

The impact on human function and health in work situations is a ‘neglected’ effect of global climate change. The potential health risks and worker productivity reductions due to climate change are substantial. The lack of attention until recently may well be due to the fact that this is mostly a problem in low and middle-income tropical countries where climate change impacts during this century will be prominent and air conditioning is not widely available, while in high-income countries air conditioning is already very common in workplaces.

The increasing heat exposure due to local climate changes is likely to create occupational health risks and to have a significant impact on the productivity of many workers, unless effective preventive measures (‘adaptation’) reducing the occupational heat stress are implemented. This may be practically and economically possible for indoor environments, but it is much more difficult for outdoor environments. Eventually, this will hamper economic and social development in affected countries unless appropriate preventive measures are taken in the planning processes for workplaces and urban development.

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References

1. IPCC. Fourth assessment report. Geneva, Inter-governmental Panel on Climate Change. Cambridge: Cambridge University Press; 2007. Available from: www.ipcc.ch [cited 18 October 2008].
2. Costello A, Abbas M, Allen A, Ball S, Bell S, Bellamy R, et al. Lancet-University College London Institute for Global Health Commission). Managing the health effects of climate change. *The Lancet* 2009; 373: 1693–733.
3. Oke TR. City size and the urban heat island. *Atmosph Environ* 1973; 7: 769–79.
4. Kjellstrom T. Climate change, heat exposure and labour productivity. *Epidemiology* 2000; 11: S144.
5. Kjellstrom T. Climate change, direct heat exposure, health and well-being in low and middle-income countries. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.1958.
6. Parsons K. Human thermal environment. The effects of hot, moderate and cold temperatures on human health, comfort and performance, 2nd edition. New York: CRC Press; 2003.
7. Ramsey JD, Bernard TE. Heat stress. Chapter 22. In: Harris RL, ed. *Patty's industrial hygiene*, 5th edition. New York: John Wiley and Sons; 2000, pp. 925–84.
8. Kerslake DM. The stress of hot environments. Cambridge: Cambridge University Press; 1972.
9. Bridger RS. Introduction to ergonomics, 2nd edition. London: Taylor & Francis; 2003.
10. Ramsey JD. Task performance in heat: a review. *Ergonomics* 1995; 38: 154–65.
11. Ramsey JD, Burford CL, Beshir MY, Jensen RL. Effects of workplace thermal conditions on safe working behavior. *J Safety Res* 1983; 14: 105–14.
12. Hales JRS, Richards DAB. Heat stress-physical exertion and environment. Amsterdam: Excerpta Medica; 1987.
13. Leithead CS, Lind AR. Heat stress and heat disorders. London: Cassell; 1964.
14. Schrier RW, Hano J, Keller HI, Finkel RM, Gilliland PF, Cirksena WJ, et al. Renal, metabolic, and circulatory responses to heat and exercise. *Ann Int Med* 1970; 73: 213–23.
15. USDAAF. Heat stress control and heat casualty management. Technical bulletin TB MED 507/AFPAM 48-152 (I). Washington, DC: US Department of the Army and Air Force; 2003.
16. NIOSH. Criteria for a recommended standard: occupational exposure to hot environments. NIOSH Publication No. 86-113. Atlanta, GA: National Institute of Occupational Health; 1986.
17. Liljegren JC, Carhart RA, Lawday P, Tschopp S, Sharp R. Modeling the Wet Bulb Globe Temperature using standard meteorological measurements. *J Occup Environ Hyg* 2008; 5: 645–55.
18. ISO. Hot environments – estimation of the heat stress on working man, based on the WBGT-index (wet bulb globe temperature). ISO Standard 7243. Geneva: International Standards Organization; 1989.
19. Malchaire J, Gebhardt HJ, Piette A. Strategy for evaluation and prevention of risk due to work in thermal environments. *Ann Occup Hyg* 1999; 43: 367–76.
20. Malchaire J, Kampmann B, Havenith G, Mehnert P, Gebhardt HJ. Criteria for estimating acceptable exposure times in hot working environments: a review. *Int Arch Occup Environ Health* 2000; 73: 215–20.
21. ISO. Hot environments – analytical determination and interpretation of thermal stress using calculation of required sweat rate. ISO Standard 7933. Geneva: International Standards Organization; 1989.
22. Miller VS, Piette A, Kampmann B, Mehnert P, Gebhardt H, Havenith G, et al. Development and validation of the Predicted Heat Strain Model. *Ann Occup Hyg* 2001; 45: 123–35.
23. Brake DJ, Bates GP. Limiting metabolic rate (Thermal Work Limit) as an index of thermal stress. *Appl Occup Environ Hygiene* 2002; 17: 176–86.
24. Miller VS, Bates GP. The Thermal Work Limit is a simple reliable heat index for the protection of workers in thermally stressful environments. *Ann Occup Hyg* 2007; 51: 553–61.
25. Wyndham CH. A survey of the causal factors in heat stroke and of their prevention in the gold mining industry. *J S African Inst Mining and Metallurg* 1965; 66: 125–55.
26. MMWR. Heat-related deaths among crop workers – United States, 1992–2006. *JAMA* 2008; 300: 1017–8.
27. Nag PK, Nag A, Ashtekar SP. Thermal limits of men in moderate to heavy work in tropical farming. *Ind Health* 2007; 45: 107–17.
28. Axelsson O. Influence of heat exposure on productivity. *Work Environ Health* 1974; 11: 94–9.
29. Holmer I. Assessment and prevention of heat stress at work. UFA Bulletin No. 4. Stockholm, Sweden: National Institute of Working Life; 1996.
30. Ebi KL, Smith JB, Burton I, eds., Integration of public health with adaptation to climate change. New York: Taylor & Francis; 2005.
31. Fisk WJ. Health and productivity gains from better indoor environments and their relationship with building energy efficiency. *Ann Rev Energy Environ* 2000; 25: 537–66.
32. Witterseh T, Wyon DP, Clausen G. The effects of moderate heat stress and open-plan office noise distraction on SBS symptoms and on the performance of office work. *Indoor Air* 2004; 14: 30–40.
33. Wyon DP. The effects of indoor air quality on performance and productivity. *Indoor Air* 2004; 14: 92–108.
34. Gunnarsen L, Santos AMB. Reduced heat stress in offices in the tropics using solar powered drying of supply air. *Indoor Air* 2002; 12: 252–62.
35. Ahasan MR. Work related problems in metal handling tasks in Bangladesh: obstacles to the development of safety and health measures. *Ergonomics* 1999; 42: 385–96.
36. Bernard TE. Heat stress and protective clothing: an emerging approach from the United States. *Ann Occup Hyg* 1999; 43: 321–7.
37. Kjellstrom T, Lemke B, Dear K. Climate change, urban heat exposure, and occupational health impacts. Report from National Centre for Epidemiology and Population Health, Australian National University (in press).
38. Kjellstrom T. Global climate change and health – a new theme for research in environmental medicine. Technical report. Stockholm, Sweden: National Institute of Environmental Medicine; 2009. Available from: <http://www.imm.ki.se/klimatrapport> [cited 23 October 2009]

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Effects of heat on workers' health and productivity in Taiwan

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Background: The impact of global warming on population health is a growing concern and has been widely discussed. The issue of heat stress disorders and consequent productivity reduction among workers has not yet been widely addressed. Taiwan is an island straddling the Tropic of Cancer in the West Pacific and has both subtropical and tropical climates. As of 2008, the economy of Taiwan accounts for 1.1% of the world gross domestic product at purchasing power parity and is listed as 19th in the world and eighth in Asia, according to International Monetary Fund data.

Objective: The aim of this paper is to identify occupations at risk and the potential health impacts of heat on workers in Taiwan.

Design: Historical data relating to meteorology, population, the labour force and economy were obtained from publicly available databases from the Taiwanese government.

Results: Hot seasons with an average maximum temperature above 30°C and relative humidity above 74%, lasting for four to six months from May to October, pose health threats to construction, farming and fishery workers. In particular, populations of ageing farmers and physically overloaded construction workers are the two most vulnerable worker categories in which high temperature impacts on health and productivity.

Conclusions: Currently, regulations and preventive actions for heat relief are difficult to enforce for several reasons, including lack of equipment for measuring environmental conditions, lack of awareness of potential hazards and strict time constraints imposed on workers. There is an urgent need to systematically and comprehensively assess the impact of a warming climate on workers' health and productivity to provide effective prevention strategies for a better working and living environment in Taiwan.

Keywords: *occupational health; global warming; hot temperature; heat stress disorders; productivity*

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In 2007, three consecutive days of high temperatures, from 33 to 34°C, at the end of May resulted in the deaths of three 80-year-old farm workers in a rural area in southern Taiwan while they were working outdoors (1). In July 2009, a 40-year-old live-line worker died of heatstroke. He had begun work at noon, conducting electrical equipment maintenance, wearing protective clothing and a helmet at a temperature of 33–34°C (2). Such incidents highlight how excessive heat exposure can cause death during hot summer seasons in Taiwan. During 2006–2007, a total of 22 deaths were officially attributed to excessive heat (E900 as classified by ICD-9) by the Department of Health in Taiwan (3). However, the exact issue of how the climate affects population health and productivity is still unclear because there has been no nationwide study or surveillance of this issue.

Taiwan's geography and climate

Taiwan, with a population of 23 million, is an island located between 21.5–25.2°N and 120–122°E. This island straddles the Tropic of Cancer in the western Pacific, with both subtropical and tropical climates. The climate of Taiwan is characterised by relatively high year-round temperatures and humidity, usually accompanied by heavy rain and tropical cyclones during the summer.

Taipei (25.5°N) and Kaohsiung (22.4°N) are the two largest municipalities in Taiwan and are located to the north and south of the Tropic of Cancer, respectively. These two areas have the largest populations and the most ongoing construction projects in Taiwan, such as new high-rise apartments, commercial centres and underground transportation systems. Their respective population sizes are 6.5 million in Taipei and 2.8 million in

Kaohsiung (4). The major economic activities of these two areas are commercial and light manufacturing industries, for instance the information technology industry (Standard Industrial Classification (SIC) codes 357, 365–367, 369, 481–484 and 489) in Taipei and heavy manufacturing industries, such as the petrochemical industry (SIC code 29), in Kaohsiung. Taichung (24.9°N), with a population of 2.6 million, is the third largest metropolitan area in Taiwan. Taichung is located in central Taiwan and has a combination of commercial, industrial and agricultural economic activities (4). Chiayi (23.3°N) and Tainan (23.1°N), which are the two largest farming areas in Taiwan, are located to the south of the Tropic of Cancer and have a population of about 2.7 million in total. Their main economic activities include the production of rice, fruits and vegetables from cultivated fields and fishery products from inland fish farms, accounting for 18–25% of agriculture production and 24% of fishery production, as measured in metric tonnes, in Taiwan (4, 5).

Over the past 30 years, meteorological data from the three largest metropolitan areas in Taiwan (i.e. Taipei, Taichung and Kaohsiung) have shown hot seasons with an average maximum temperature above 30°C, lasting four to five months, with a mean relative humidity ranging from 74 to 82% (Fig. 1A–C) (6). Meteorological data from the two largest farming areas (i.e. Chiayi and Tainan) also show similar high temperatures in hot seasons, with an average maximum temperature above 30°C, lasting five to six months, and even higher humidity, ranging from 77 to 85% (Fig. 1D and E) (6).

Typically, the temperature of Taiwan varies during the four seasons and peaks in July. On average, monthly mean temperatures range from 15.8 to 29.2°C in metropolitan areas and from 16.1 to 29.0°C in farming areas (6). However, a recent nationwide report indicated that the average temperatures measured at five Taiwan weather stations in July have shown an increase in temperature by 1.6°C over the past century (7).

Occupations and industries at risk of heat stress in Taiwan

Labourers who work in hot indoor or outdoor environments in Taiwan include construction workers (roads and roofs), farming and fishing workers, cooks (in bakeries and kitchens), metal and glass manufacturers (for instance, in steel and bicycle factories), and transportation workers and material movers (for example, postal deliverers and traffic policemen). Of these, construction (SIC Division C) workers accounted for the largest portion of the labour force that works outdoors, with around 842,000 workers (8.1% of the labour force), whereas agriculture, forestry and fishing (SIC Division A) workers accounted for 535,000 workers (5.1%) in 2008 (Table 1) (8). A total of 11,000 agriculture, forestry, fishing and construction workers are foreign labourers from Thailand, Indonesia,

the Philippines and Vietnam (9). Foreign workers are recruited to Taiwan mainly because of cheaper labour costs, but also on account of the notion that they have greater heat stress endurance than local workers. Anecdotal stories of sudden deaths, possibly due to excess heat during work, have been occasionally reported in the past. Workers in construction, farming and fishery industries may also suffer from high occupational temperature impacts on health and productivity due to climate change, as suggested by Kjellstrom (10). These workers are a potentially high-risk population in Taiwan because they have to work under hot and humid summer conditions, which last for more than five months. More than a million workers are employed in these industries, and they contributed around 3.4% of the real GDP of Taiwan in 2008 (11). The effect of heat on these workers' health and work output is an important public health issue in Taiwan.

Surveys of employees' perceptions of safety and health in the work environment have been routinely conducted via questionnaires every three years since 1988, primarily by the Taiwan Institute of Occupational Safety and Health. The most recent survey of 19,000 workers indicated that 42% of employees perceived a risk of excessive heat at the workplace (12). The proportion of employees who perceived risk was particularly high in the construction industry (76.3%) and in the agriculture, forestry and fishing industries (71.3%) (12). Compared to data from 2001, these rates were higher in both sets of industries in 2007 (Fig. 2), whereas the rate for the total workforce decreased slightly (12–14). A decrease in perceived risk of excessive heat amongst the total workforce may be due to the increased use of air conditioning in indoor workplaces. Note that the proportion of employees who perceived a risk of excessive heat at the workplace is higher in males (50.97%) and in those with lower educational levels (63.51–78.15% for junior high level or below) (12).

However, information regarding actual exposure to hot work environments for these workers remains unclear, especially for outdoor construction sites and farming operations during hot summer months, raising concern about the effect of heat stress on workers' health and productivity. Two studies have attempted to investigate heat exposure levels and workers' symptoms of fatigue in hot workplaces in Taiwan (15, 16). One investigation on workers' heat exposure at hot workplaces during summer months (i.e. from July to September) showed that averages of the wet bulb globe temperature (WBGT) index at 54 workplaces ranged from 21.4 to 40.1°C, and 51.4% of the measured WBGT values exceeded the recommended WBGT level for the 212 workers in these workplaces (15). The unusually high WBGT levels measured in this survey were caused by a combination of heat generated indoors and natural heat outdoors (15). Another study showed that subjective symptoms of shoulder pain, lower back pain and thirst in 151 workers

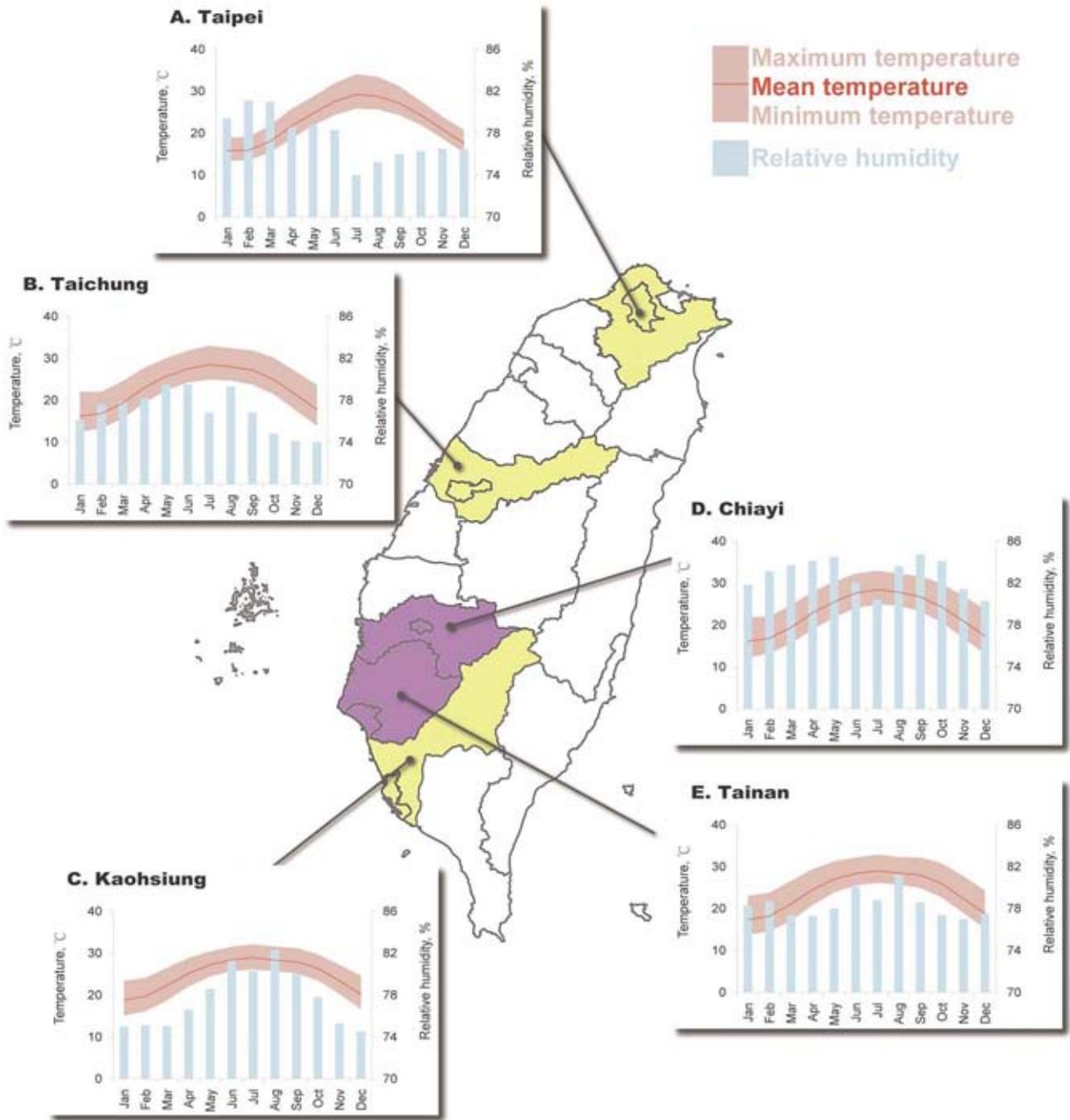


Fig. 1. Meteorological data showing monthly averages for daily mean, minimum, and maximum temperature and monthly averages for relative humidity in three metropolitan areas (A–C) and two farming areas (D and E) over the past 30 years (Source 6).

in steel, glass and porcelain manufacturing plants were related to high WBGT indexes (range: 19.6–34.6°C) at their worksites (16).

Regulations and prevention of excessive heat exposure in workplaces and households

The Labour Safety and Health Act in Taiwan prevents workers from being exposed to unusually high

temperatures for more than six hours per day (17). According to the WBGT-based Work-Rest Regime Standards for Workers in Hot Workplaces in Taiwan, permissible heat exposure in hot environments is based on the type of work being performed; for example, the WBGT of an area of continuous work should not exceed 30.6°C for light workloads, 28.0°C for moderate workloads, or 25.9°C for heavy workloads (18). Employers

Table 1. Description of all working populations and workers in industries at risk of excessive heat in Taiwan, in 2008 (Sources 8, 11)

Industries, by sex	Employed persons (in thousands)	Work hours per week	Educational level equal to junior high school or below (%)	Real GDP, new Taiwan dollars (in millions)
Both sexes				
All industries	10,403	43.83	25	13,089,718
Construction	842	40.41	48	173,060
Agriculture, forestry and fishing	535	40.24	74	271,594
Males				
All industries	5,902	44.19	27	–
Construction	746	40.37	50	–
Agriculture, forestry and fishing	374	40.40	71	–
Females				
All industries	4,501	43.35	21	–
Construction	96	40.74	33	–
Agriculture, forestry and fishing	162	39.88	80	–

are required to ensure that employees who work in certain environments be provided with personal equipment that protects them from heat exposure, as well as drinking water and salt (18). These places include boiler and furnace rooms, places in which steel and non-ferrous metals are smelted or cast, ceramic, glass and carbide furnace plants and steam tunnels (18). According to the Workplace Environmental Monitoring Regulation, workplaces should be inspected at least once every three months when the WBGT of the workplace exceeds the limit of the criteria (19).

However, such regulation is difficult to enforce in farming, fishing and construction industries, where the WBGT of outdoor workplaces is not directly measured

and can only be indirectly inferred from local meteorological monitoring data. About 83% of farming and fishery workers are self-employed or household labourers (8) and are not equipped to measure the WBGT themselves. Around 84% of construction workers are employees (8), and they have to work during hot days to fulfil the required tasks for a set period of time.

On average, construction workers worked 40.41 hours per week in 2008 (8). In Taiwan, workers take one hour off for lunch, usually from 12:00 to 13:00, at shady places to avoid direct sun exposure. In addition to following the criteria of work and rest, these workers are usually shirtless and wear helmets, quenching their thirst with either alcohol, energy drinks or herbal teas.

Construction workers usually live in dormitories near construction sites. The dormitories are usually tin-plate huts and are sometimes not furnished with air conditioners. Workers often feel uncomfortable living in such conditions, especially during the hot and humid summer (20). The common way to reduce heat stress at these dormitories is to open windows to increase natural ventilation or to use fans when air conditioners are not available.

The working period for farmers depends on the season of harvest. The first crop of rice, for example, is harvested during the hot season from May to August, followed by the second crop, harvested from September to January (21). The average working hours in agriculture, forestry and fishing industries were 40.24 hours per week in 2008 (8). Farmers may work shirtless and wear wide-brimmed hats to avoid overheating at work. However, some farmers wear protective clothes and masks to reduce pesticide exposure (22), which may increase the risk of heat-related illness. Most farmers live in their own houses and use fans

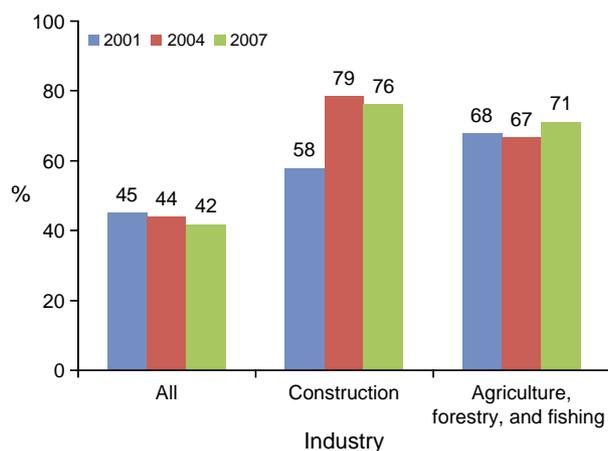


Fig. 2. Percentage of employees who perceived a risk of excessive heat at the workplace: all industries; construction industries and agriculture, forestry and fishing industries in 2001, 2004 and 2007 (Sources 12–14).

or air conditioners to reduce heat exposure. Commuting from their houses to the farms may also increase heat exposure, because farmers travel to their workplace on foot or by bicycle or motorcycle.

Conclusions

Epidemiological studies and reports in Taiwan have highlighted the impact of warmer climate on several infectious diseases, such as dengue fever and enteroviral infection (23–25). However, the impact of a warmer climate on workers' health and productivity has not been analysed. Farmers' heat-related deaths exemplify the vulnerability of older farmers and fishermen working in hot climates. The fact that agricultural workers tend to be older raises the question of whether they have appropriate protection in overheated environments (Fig. 3) (8). Moreover, tight protective clothing protecting against pesticide exposure may increase the risk of heat stress among farm workers. Heavy physical workloads can also pose a threat of heat fatigue among construction workers, which is likely to increase the risk of accidents and injuries at construction worksites.

This report has outlined the risks that heat poses for workers' health. Construction and farming workers, especially elderly workers, are the most vulnerable occupational groups that may be exposed to heat at worksites for long durations. Their health and work capacity are among the major concerns for the nation's economic development.

As a result of global warming, we should assess the impact of local climate change on workers' health and productivity in Taiwan, both qualitatively and quantitatively. New studies can provide labour and health policymakers, as well as safety and health practitioners, with essential information about the effects of heat on health and productivity. Such studies can address prevention strategies that would provide these workers with a better working and living environment.

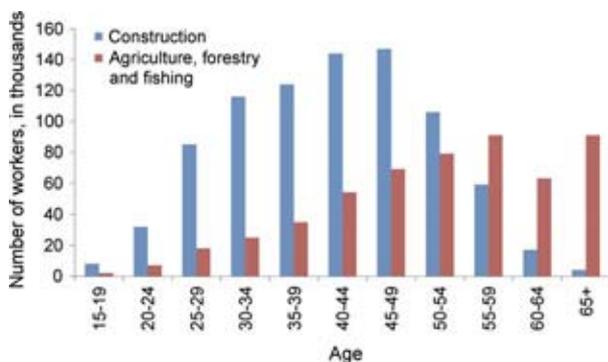


Fig. 3. Age distribution of employed persons by industry in 2008 (Sources 8).

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References

- Ju HJ. Three farm workers' heat deaths in three consecutive days. *United Daily News*, A3, 29 May 2007, (In Chinese).
- Yang DY. A Taipower live-line worker has died in suspected heat-related death on Great Heat day. *United Daily News*, A14, 24 July 2009. (In Chinese)
- Department of Health, Executive Yuan, Taiwan. Causes of death statistics. Available from: <http://www.doh.gov.tw/statistic/index.htm>; 2006, 2007 [cited 26 December 2008]. (In Chinese)
- Directorate-General of Budget, Accounting and Statistics, Executive Yuan, Taiwan. Static statistics of population. Available from: <http://sowf.moi.gov.tw/stat/month/m1-06.xls>; 2008 [cited 29 May 2009].
- Agriculture and Food Agency, Council of Agriculture, Executive Yuan, Taiwan. Index of agricultural production. Yearly report of Taiwan's agriculture. Available from: <http://eng.coa.gov.tw/list.php?catid=17848>; 2007 [cited 18 August 2009].
- Central Weather Bureau of Taiwan. Monthly climate statistics. Available from: <http://www.cwb.gov.tw/eng/index.htm>; 1971–2000. [cited 26 September 2009].
- Chen YL. Climate change in Taiwan from 1901 to 2006. *Sci Develop* 2008; 424: 6–11. (In Chinese)
- Directorate-General of Budget, Accounting and Statistics, Executive Yuan, Taiwan. Manpower survey. Available from: <http://www.stat.gov.tw/ct.asp?xItem=18844&ctNode=4944>; 2008 [cited 21 August 2009]. (In Chinese)
- Council of Labor Affairs, Executive Yuan, Taiwan. Labor statistics. Available from: http://www.cla.gov.tw/cgi-bin/SM_theme?page=41761dc1; 2008 [cited 29 May 2009]. (In Chinese)
- Kjellstrom T. Climate change, heat exposure and labour productivity. 12th Conference of the International Society for Environmental Epidemiology (ISEE), Buffalo, USA, 19–23 August 2000. *Epidemiology* 2000; 11: S144.
- Directorate-General of Budget, Accounting and Statistics, Executive Yuan, Taiwan. Annual estimates of gross domestic product by industry group. Available from: <http://www.dgbas.gov.tw/ct.asp?xItem=14616&CtNode=3566>; 2008 [cited 29 May 2009]. (In Chinese)
- Hsu JH, Li YS. Survey of perceptions of safety and health in the work environment in 2007. Taipei, Taiwan: Institute of Occupational Safety and Health (IOSH); 2008. Report No. IOSH96-M319. (In Chinese)
- Tai CF, Yang SC, Yeh WY. Survey of employees' perceptions of safety and health in the work environment in 2001 Taiwan. Taipei, Taiwan: Institute of Occupational Safety and Health (IOSH); 2002. Report No. IOSH90-H304. (In Chinese)
- Shih TS, Chen CJ, Chang CP. Survey of employees' perceptions of safety and health in the work environment in 2004 Taiwan. Taipei, Taiwan: Institute of Occupational Safety and Health (IOSH); 2005. Report No. IOSH93-H302. (In Chinese)

15. Ho ST, Liou YW, Yeh WY, Cheng SS, Wei MC. Study on heat exposure dosage of labours in hot workplace. Taipei, Taiwan: Institute of Occupational Safety and Health (IOSH); 1994. Report No. IOSH83-H242. (In Chinese)
16. Mao IF, Chen ML, Chen CJ, Yeh WY, Yang KY, Chen YC, et al. The study of the work environmental temperature and worker's fatigue. *J Occup Safe Health* 1996; 4: 1–12. (In Chinese)
17. Council of Labor Affairs, Executive Yuan, Taiwan. Labor Safety and Health Act. 1974. Available from: <http://laws.cla.gov.tw/eng/flaw/flawdat01.asp?lsid=FL015013> [cited 18 August 2009]
18. Council of Labor Affairs, Executive Yuan, Taiwan. WBGT-based Work-Rest Regime Standards for Workers in Hot Workplaces, 1974. Available from: <http://laws.cla.gov.tw/Chi/FLAW/FLAWDAT01.asp?lsid=FL015019> [cited 26 December 2008]. (In Chinese)
19. Council of Labor Affairs, Executive Yuan, Taiwan. Workplace Environmental Monitoring Regulation, 1992. Available from: <http://laws.cla.gov.tw/Chi/FLAW/FLAWDAT01.asp?lsid=FL015045> [cited 18 August 2009]. (In Chinese)
20. Fan GY. Cost control in the construction industry. Taipei, Taiwan: Chan's Arch Books; 1997. (In Chinese)
21. Council of Agriculture, Executive Yuan, Taiwan. Available from: http://www.coa.gov.tw/show_product.php?id=9_cikuo_20060322161311 [cited 12 June 2009]. (In Chinese)
22. Li HP. Safety on spraying agricultural chemicals. In: Yeh Y and Lin TC, eds. *Taiwan agriculture encyclopedia*. Crop edition. 3rd ed. Taipei, Taiwan: Council of Agriculture, Executive Yuan, Taiwan; 2005, pp. 531–3. (In Chinese)
23. Wu PC, Guo HR, Lung SC, Lin CY, Su HJ. Weather as an effective predictor for occurrence of dengue fever in Taiwan. *Acta Trop* 2007; 103: 50–7.
24. Chen HY. Ecological and seasonal variations of enteroviruses infection in Taiwan, 2000–2004. Taipei, Taiwan: National Taiwan University; 2008. (In Chinese)
25. Su HJ, Lin CK, Chen PS. The impact of climate changes on public health. *Sci Develop* 2008; 421: 12–7. (In Chinese)

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Work-related heat stress concerns in automotive industries: a case study from Chennai, India

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Background: Work-related heat stress assessments, the quantification of thermal loads and their physiological consequences have mostly been performed in non-tropical developed country settings. In many developing countries (many of which are also tropical), limited attempts have been made to create detailed job-exposure profiles for various sectors. We present here a case study from Chennai in southern India that illustrates the prevalence of work-related heat stress in multiple processes of automotive industries and the efficacy of relatively simple controls in reducing prevalence of the risk through longitudinal assessments.

Methods: We conducted workplace heat stress assessments in automotive and automotive parts manufacturing units according to the protocols recommended by NIOSH, USA. Sites for measurements included indoor locations with process-generated heat exposure, indoor locations without direct process-generated heat exposure and outdoor locations. Nearly 400 measurements of heat stress were made over a four-year period at more than 100 locations within eight units involved with automotive or automotive parts manufacturing in greater Chennai metropolitan area. In addition, cross-sectional measurements were made in select processes of glass manufacturing and textiles to estimate relative prevalence of heat stress.

Results: Results indicate that many processes even in organised large-scale industries have yet to control heat stress-related hazards adequately. Upwards of 28% of workers employed in multiple processes were at risk of heat stress-related health impairment in the sectors assessed. Implications of longitudinal baseline data for assessing efficacy of interventions as well as modelling potential future impacts from climate change (through contributions from worker health and productivity impairments consequent to increases in ambient temperature) are described.

Conclusions: The study re-emphasises the need for recognising heat stress as an important occupational health risk in both formal and informal sectors in India. Making available good baseline data is critical for estimating future impacts.

Keywords: *work-related heat stress; WBGT; climate change; automotive industry*

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The prevalence of heat-related health risks in any strenuous job performed in hot and humid environments is well known and has been the basis for development of work-related heat stress standards (1, 2). While the prevalence of exposures to heat stress is common in many occupations throughout the world, the quantification of thermal loads and their physiological consequences have mostly been performed in non-tropical settings. In developing countries (many of which including India are also tropical), limited attempts have been made to create detailed job-exposure profiles for various sectors. For the large workforce employed in these settings, especially in small and medium enterprises

and non-industrial settings (including farming), the contributions from environmental exposures, varying workloads and physiological differences remain poorly characterised. Against this backdrop of a well-known occupational risk factor, there now looms an additional health threat from potential heat stress contributions related to global climate change (3). Climate change impacts related to heat stress are often examined in relation to heat wave-mediated effects on the general population but the recognition that climate change may exacerbate occupational heat-related risks is yet to develop. With the majority of workplace settings in developing countries being heavily influenced by outdoor

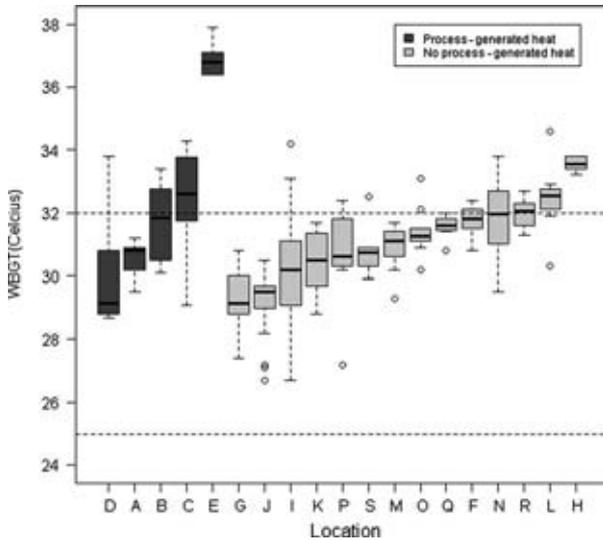


Fig. 1. Box plots illustrating the distribution of measured WBGT values at various indoor locations in automotive or automotive parts manufacturing units (dashed lines indicate the range of outdoor WBGT values across locations; dark boxes indicate locations with process-generated heat contributions and light boxes indicate locations without process heat contributions (i.e. only ambient temperature contributions), respectively, to heat stress.

Work locations key: With process heat contributions – A: PTCS (varnishing oven), B: cab furnace, C: paint shop, D: fuel injection manufacturing, E: tube manufacturing, F: canteen (boiler area). Without process heat contributions – G: body shop (general shop floor), H: fuel injection manufacturing (general shop floor), I: paint shop (general shop floor), J: stamping, K: wheel alignment and engine deck, L: material storage and stores, M: PTCS (starter, armature and shaft areas), N: team meeting areas, O: plastic moulding area, P: utility areas, Q: canteen (general), R: brazing, S: trim and chassis.

temperatures (in the absence of mechanical cooling), it can be expected that both indoor and outdoor workers may experience heat stress. Even relatively modest increases in ambient temperatures could be expected to tip large worker populations exposed to ‘near limit values’ of heat stress over the threshold into the realm of experiencing heat stress-related health risks.

The Indian automotive industry is a major economic sector and has undergone rapid expansion since 1991 with Governmental deregulation of the sector. For instance, production of vehicles has increased nearly five-fold from about 2 million in 1991 to 9.7 million in 2006. Chennai, formerly Madras, is the fourth largest metropolis in India and is the capital of the southern coastal state of Tamil Nadu. Chennai accounts for 60% of the country’s automotive exports and is sometimes referred to as ‘the Detroit of India’. The sector employs nearly 250,000 workers. Although most manufacturing plants in this sector are large units that are well

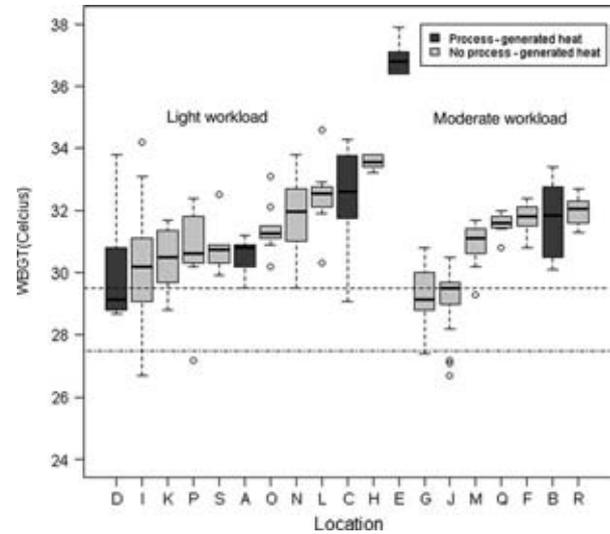


Fig. 2. Workloads at various locations in relation to WBGT indices (dark boxes indicate locations with process-generated heat contributions and light boxes indicate locations without process heat contributions (i.e. only ambient temperature contributions), respectively, to heat stress; dashed lines indicate TLV for fully acclimatised light work and dotted-dashed lines indicate TLV for fully acclimatised moderate work).

Work locations key: With process heat contributions – A: PTCS (varnishing oven), B: cab furnace, C: paint shop, D: fuel injection manufacturing, E: tube manufacturing, F: canteen (boiler area). Without process heat contributions – G: body shop (general shop floor), H: fuel injection manufacturing (general shop floor), I: paint shop (general shop floor), J: stamping, K: wheel alignment and engine deck, L: material storage and stores, M: PTCS (starter, armature and shaft areas), N: team meeting areas, O: plastic moulding area, P: utility areas, Q: canteen (general), R: brazing, S: trim and chassis.

Many locations with light workloads were still in excess of TLVs (indicating the need for engineering controls). Other locations with moderate workloads were close to or exceeded the TLVs (indicating opportunities for both administrative and engineering controls).

regulated for most occupational health and safety hazards, heat stress exposure remains quite prevalent in many processes. Chennai temperatures also range from around 21°C (between December and February) to around 37°C (between March and September). Some months record temperatures as high as 42°C. Although most processes in the automotive sector are performed indoors, lack of controls within the work environment and outdoor jobs make workers prone to heat stress from both ambient temperatures as well as process-generated heat.

We present here a case study from Chennai in southern India that illustrates the prevalence of work-related heat stress within multiple processes of automotive industries and the efficacy of relatively simple controls in reducing

prevalence of risks through longitudinal assessments. We also made limited assessments in two other sectors to estimate the likely percentages of workers at risk from heat stress in various processes. Since newly established plants have routine monitoring facilities, the choice of newly established automotive plants to conduct this pilot in a rapidly expanding sector allowed us to identify opportunities to create longitudinal baseline data for assessing efficacy of interventions as well as modelling future impacts from climate change.

Materials and methods

We conducted workplace heat stress assessments in automotive and automotive parts manufacturing units according to the protocols recommended by NIOSH, USA. Locations for measurements were selected based on the initial survey results; these included indoor locations with process-generated heat exposure, indoor locations without direct process-generated heat exposure and outdoor locations. Nearly 400 measurements of heat stress were made over a four-year period at more than 100 locations within eight units involved with automotive or automotive parts manufacturing in the greater Chennai metropolitan area. Since most workplace locations were not air-conditioned and therefore likely to be influenced by outside temperature and time of day/season, measurements were always made during the hottest part (11:00–14:30) of the day in the months of May or June, with repeated annual assessments. Measurements were used to recommend interventions at selected locations in the automotive units and multiple longitudinal measurements were made at locations where controls were implemented in order to assess their efficacy.

In addition, cross-sectional assessments were made in multiple processes in glass manufacturing and textile industries. We then collected information on workforce strengths in all three sectors to estimate likely percentages in each sector that were likely to be at risk from work-related heat stress.

The measurements were carried out using an area heat stress monitor (Model Questemp^o 34, manufactured by Quest Technologies, USA) that calculates the wet bulb globe temperature (WBGT) to assess heat stress. The instruments used for the measurements comply with the standards set out by American Conference of Governmental Industrial Hygienists (ACGIH). The necessary information on workload, clothing worn, worker’s time-activity pattern and acclimatisation was collected on-site, to make appropriate adjustments to the measured WBGT value. The threshold limit value (TLV) was computed by taking spot readings throughout the work-shift and on the basis of worker description of workload, using a ‘clo’ factor of 0.6 for summer work uniforms. This ‘clo’ factor contributes to a WBGT correction factor of 0°C. For light workloads and full acclimatisation of the workers, a

Table 1. Improvements in heat stress-related exposures at select locations

Location	Work-load	Mean WBGT (in °C) 2005	Mean WBGT (in °C) 2006	Mean WBGT (in °C) 2007	Mean WBGT (in °C) 2008	TLV (in °C)	Recommendation implemented
Stamping (n = 24)	Moderate	30.5	29.6	29.2	27.1	27.5	Improvement of cross-ventilation by installing more windows on the wall
Body shop (n = 48)	Moderate	29.9	30.7	–	28.9	27.5	Improvement of cross-ventilation by installing more windows on the wall
Paint shop (loading/unloading) (n = 54)	Light to moderate	32.2	–	31.2	29.1	27.5	Provision of lime juice and milk during the hot season Installation and maintenance of air cooling ducts
Paint shop (oven operations) (n = 24)	Light	34.2	–	–	29.2	29.5	Thermoinsulation of the oven
Engine/chassis/wheel alignment (n = 34)	Light	32.0	–	33.8	31.4	29.5	Increasing the number of breaks during summer
Batch and hold yard shed (n = 38)	Light	33.4	33.3	32.2	31.7	29.5	Installation and maintenance of air cooler

Note: WBGT measurements were made during the hottest part of the day.

Table 2. Exposure profiles for heat stress in select processes of automotive, glass and textile manufacturing sectors in southern India

Industrial sector	Location	Name of the process	Physical workload	Number of workers	Average WBGT (in °C)	TLV for WBGT (in °C)	Exceeding TLV	Estimated % of population at risk	
Automobile/automotive parts manufacturing	Indoor with process heat	Paint shop	Moderate	178	30.4	27.5	Yes	100	
		Indoor without process heat	Stamping	Moderate	90	29.1	27.5	Yes	51.3
	Indoor without process heat	Body shop	Moderate	340	28.9	27.5	Yes		
		TCF	Moderate	312	31.3	27.5	Yes		
		Engine plant	Moderate	172	30.6	27.5	Yes		
		Launch	Moderate	15	30.2	27.5	Yes		
		MP & L	Light	45	29.4	29.5	No		
		Moderate		100	29.3	27.5	Yes		
			P & D	Light	100	28.2	29.5	No	
		Moderate		26	28.3	27.5	Yes		
		Admin	Light	1,000	25.6	29.5	No		
		Maintenance	Moderate	150	29.7	27.5	Yes		
		Outdoor	Maintenance	Moderate	50	30.9	27.5	Yes	100
			Gardening	Moderate	25	30.9	27.5	Yes	
			Heavy		50	30.9	26	Yes	
Glass manufacturing	Indoor with process heat	Furnace area	Light	25	31.9	29.5	Yes	100	
		Port 3	Light	14	37.8	29.5	Yes		
		Bay 1	Light	12	38	29.5	Yes		
		Bay 2	Light	25	40.9	29.5	Yes		
		Bay 17	Light	48	34.9	29.5	Yes		
		Annealing area 1	Light	7	35.6	29.5	Yes		
		Annealing area 2	Light	6	38	29.5	Yes		
		Cold end	Light	9	32.4	29.5	Yes		
		Mirror plant – hot	Light	7	29.8	29.5	Yes		
		Indoor without process heat	Mirror plant – wet	Light	12	28.5	29.5	No	31.2
			Bay loading area	Moderate	76	29.3	27.5	Yes	
		Admin	Light	200	26	29.5	No		
		Maintenance	Moderate	20	29.8	27.5	Yes		
	Outdoor	Maintenance	Moderate	10	31	27.5	Yes	100	
		Gardening	Moderate	18	31.2	27.5	Yes		
Heavy			10	31.1	26	Yes			
Textile manufacturing	Indoor without process heat	Blowing	Moderate	19	26.8	27.5	No	28.1	
		Carding/drawing/roving	Moderate	15	28.8	27.5	Yes		

Table 2 (Continued)

Industrial sector	Location	Name of the process	Physical workload	Number of workers	Average WBGT (in °C)	TLV for WBGT (in °C)	Exceeding TLV	Estimated % of population at risk
		Combing	Moderate	22	26.5	27.5	No	
		Spinning	Moderate	46	30.3	27.5	Yes	
		Winding/reeling/doubling	Moderate	31	27.2	27.5	No	
		Weaving	Moderate	47	27	27.5	No	
		Admin	Light	50	26.8	29.5	No	
		Maintenance	Moderate	5	29.6	27.5	Yes	
		Maintenance	Moderate	5	29.7	27.5	Yes	100
	Outdoor	Gardening	Moderate	2	29.6	27.5	Yes	
			Heavy	3	29.6	26	Yes	

TLV of 29.5°C was used and in case workstations did not require workers to stay permanently, a TLV of 29.5°C was used assuming light work (e.g. inspection work) and full acclimatisation. The adjusted values were compared to the prescribed TLVs recommended by the ACGIH.

Descriptive statistical analysis was done using the software ‘R’.

Results

Heat stress exposure in various processes of automotive and automotive parts manufacturing

Fig. 1 shows the distribution of measured heat stress indices across locations. Many indoor locations were found to be close to or exceeded the recommended TLVs. Further, indoor WBGT indices were observed to be largely driven by outdoor temperatures as they were uniformly high even in locations with no process-generated heat components.

Workloads prevalent at each of the locations shown in Fig. 1 (that were used to compute the corresponding WBGT index) are illustrated in Fig. 2.

Qualitative assessment of work practices and implementation of controls

In order to make specific recommendations to the units for heat stress exposure reduction, we undertook an observational qualitative assessment for existing work practices and existing controls. A number of recommendations ranging from provision of hydration breaks to improved natural ventilation and installation of air cooling devices were made. Based on longitudinal measurements at the same facilities, some of the key post-intervention improvements are shown in Table 1.

Estimating potential for work-related heat stress across select sectors

As an attempt to understand the potential scale of impacts related to work-related heat stress, in order to understand possible ramifications for climate change-related exacerbation, an estimate of the proportion of workers at risk are provided for selected sectors. This is based on measurements that were made and information collected as part of the routine occupational hygiene monitoring services provided by the investigators’ University department. While the case study summarised above had the single largest set of longitudinal measurements, the other sectors had a smaller number of cross-sectional measurements. The exposure implications for select processes in three such sectors, namely automotive parts manufacturing, glass manufacturing and textiles are detailed in Table 2.

Discussion

Results from over 400 measurements across multiple locations and industries clearly indicate that many processes even in organised large-scale industries have yet to control heat stress-related hazards adequately. Although a systematic review is not available, studies conducted in many other sectors in India reveal a high prevalence of heat-related exposures in both the formal and informal sectors, including farming, glass manufacturing, stone quarrying and crushing, mining, etc. (4–6).

While indoor work without process-generated heat exposures should be relatively less hazardous, because of the tropical climatic conditions in India, and particularly in the south, and the lack of controlled built environments, ambient temperatures influence work-related heat exposures even in indoor settings. This is further compounded by manual handling and other ergonomic hazards, also widely prevalent and poorly controlled in many industrial processes. Outdoor work is very common in India. Many jobs in the service sector (transport and local trade), construction, municipal administration and small businesses, in addition to specific processes in manufacturing and mining, are performed outdoors and here the impacts of high ambient temperatures can be particularly detrimental.

Exposure information available from selected studies in India is summarised in Table 3 along with estimates of worker populations employed in these sectors. While reliable measurements are not available in many sectors to estimate worker populations at risk, the sectors profiled in this paper serve to illustrate the likely widespread prevalence of such risks. Although the measured values reported in many studies, including this one, have not been able to capture the full range of exposures that may be experienced across seasons and at different times during the day, the observed prevalences of work-related heat stress reported in Table 2 are likely to be at the low end of exposure spectrum as they were limited to large scale and relatively newly established units.

As illustrated by the efficacy of relatively simple controls in the units included for assessment, there exist

several options to install and/or improve existing controls. It is particularly important to recognise that while administrative controls appear more attractive (as they do not require initial large capital investments), the loss in productivity could be substantial if one were to genuinely implement controls to ensure health and comfort of workers. The cost–benefit thus should duly address health and quality of work impacts while comparing across control strategies. In developing countries there is also a socio-cultural dimension of ‘risk perception’ that argues against provision of air-conditioned work spaces in the shop floor. The added value of having comfortable work spaces insulated from external climate vagaries for health and productivity thus remain largely uncharacterised.

Given the large propensity of workplaces that expose workers to near or more than permissible levels of heat stress, it could be expected that even modest increases in temperature resulting from climate change could significantly alter the distribution of exposures and related health impacts. Work ability at even the lowest intensities of work may be severely limited if WBGT indices are increased beyond the already high values recorded in workplaces. The effects are also likely to make poorer workers even more vulnerable on account of their poorer health status, limitations in accessing controlled (air-conditioned) workplaces/homes and greater likelihood of engaging in heavy work. Although work-related heat stress information is frequently collected in many workplaces, many variables can influence measured values and accompanying heat stress such as time of day, month, location of measurement, workloads and availability and efficacy of controls. While it could be expected that increase in work-related heat stress may hamper productivity (for example, due to increased frequency of rest breaks, diminished work output and lost work days), the quantitative exposure relationship between heat stress and productivity remains to be characterised across work settings. In order to maintain adequate surveillance on workplaces, modelling approaches are needed that could use routinely collected weather station data in relation to

Table 3. Heat stress and worker profiles for selected industry sectors in India

Sector	Range of heat stress values (WBGT) measured (°C)	Estimated worker population in 1,000s (as per Indian National Sample Survey, 2000)	Reference
Agriculture	34.4–42.2	237,786	Nag et al. (5)
Glass manufacturing	30–40	Not available	Srivastava et al. (6)
Ceramics	43–54	Not available	Parikh et al. (7)
Mining	25–31	2,263	Mukerjee et al. (8)
Tanning	28–41	1,081	Conroy et al. (9)
Textiles	27–39	10,480	Sankar et al. (10)

measured WBGT indices at workplaces over a local region to estimate population level impacts on productivity and health. The development of such Population Heat Exposure Profiles (PHEPs) are being explored (Kjellstrom and Lemke, unpublished) and will likely allow monitoring of trends in ambient temperature and related implications for work-related heat stress across space and time as well as across multiple work place configurations in developing country settings, where routine workplace data is not always available and accessible.

Conclusions

The present case study serves to re-emphasise the need for recognition of heat stress as an important occupational health risk in both formal and informal sectors in India. Control of heat stress may have multiple co-benefits in terms of better health, improved productivity, lower rates of accidents, lower rates of morbidity and improved sense of comfort and social well-being. With the threat of climate change-related impacts looming large on developing countries including India, there is an imminent need to include this set of heat-related impacts while modelling health effects related to climate change. Making available good baseline data is critical for estimating future impacts and the case study presented here represents one such pilot effort in southern India.

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References

1. National Institute of Occupational Safety and Health. Occupational exposure to hot environments. Revised criteria. Washington, DC: US Department of Health, Education & Welfare; 1986.
2. ISO7933. Ergonomics of the thermal environmental analytical determination and interpretation of heat stress using calculation of the predicted heat stress. Geneva: ISO; 2004.
3. Kovats RS, Hajat S. Heat stress and public health: a critical review. *Annu Rev Public Health* 2008; 29: 41–55.
4. National Institute of Occupational Health. Health status of tea plantation workers with special reference to their occupation. Annual Report. Ahmedabad, India; 1979, pp. 153–75.
5. Nag PK, Sebastian NC, Malvankar MG. Occupational workload in Indian agricultural workers. *Ergonomics* 1980; 23: 91–102.
6. Srivastava A, Kumar R, Joseph E, Kumar A. Heat exposure study in a glass manufacturing unit in India. *Ann Occ Hyg* 2000; 44: 449–53.
7. Parikh DJ, Ghodasara NB, Ramanathan NL. A special heat stress problem in ceramic industry. *Eur J App Phy Occ Phy* 1978; 40: 63–72.
8. Mukherjee A, Bhattacharya S, Saiyed HN. Assessment of respirable dust and its free silica contents in different Indian coal mines. *Ind Health* 2004; 43: 277–84.
9. Conroy L, Forst L, Nickels L, Krantz A, Balakrishnan K. WHO modules in occupational health: economic sector – manufacturing; Instructor Manual; 2005. Available from: www.who.int/entity/occupational_health/publications/whomodules/en [cited 31 July 2009].
10. Sankar S, Padmavathi R, Rajan P, Ayyappan R, Arnold J, Balakrishnan K. Job-exposure-health profile for workers exposed to respirable dusts in textile units of Tamilnadu, India: results of preliminary investigations. Abstract presented at ISEA–ISEE (International Society of Exposure Analysis – International Society for Environmental Epidemiology); 2002 Aug. 11–15; Vancouver, Canada. *Epidemiol.* 2002; 13: S231; 846.

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Heat stress assessment among workers in a Nicaraguan sugarcane farm

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Background: Heat illness is a major cause of preventable morbidity worldwide. Workers exposed to intense heat can become unable to activate compensation mechanisms, putting their health at risk. Heat stress also has a direct impact on production by causing poor task performance and it increases the possibility of work-related morbidity and injuries. During the sugarcane harvest period, workers are exposed to excessive sunlight and heat from approximately 6 am to 3 pm. A first assessment of heat stress during the 2006/2007 harvesting season served to redesign the existing rehydration measures. In this project, sugarcane workers were provided with more rehydration solutions and water during their work schedule.

Objective: To assess heat stress preventive measures in order to improve existing rehydration strategies as a means of increasing productivity.

Methods: A small group of 22 workers were followed up for 15 days during working hours, from 6 am to 3 pm. Selection criteria were defined: to have worked more than 50% of the day's working schedule and to have worked for at least 10 days of the follow-up period. A simple data recollection sheet was used. Information regarding the amount of liquid intake was registered. Production output data was also registered. Temperature measurements were recorded by using a portable temperature monitoring device ('EasyLog', model EL-USB-2).

Results: The average temperature measurements were above the Nicaraguan Ministry of Labour thresholds. Seven workers drank 7–8 L of liquid, improving their production. Output production increased significantly ($p = 0.005$) among those best hydrated, from 5.5 to 8 tons of cut sugarcane per worker per day.

Conclusions: Productivity improved with the new rehydration measures. Awareness among workers concerning heat stress prevention was increased.

Keywords: *heat stress; climate measurements; hydration; sugarcane harvest; production output*

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The incidence of heat-related disorders increases with higher ambient temperatures (1). Internationally, different authors and Governmental institutions have assessed heat stress effects on workers and also have described different methods to measure its negative impact on productivity as well as mechanisms to control and diminish these effects (2–6). Heat stress in Nicaragua has not been given the importance it deserves, especially in relation to carrying out work in outdoor settings, such as agricultural activities.

In Nicaragua, sugarcane harvesting is done manually using machetes. Workers employed in the Montelimar farm are exposed to intense sunlight and heat while working on sugarcane plantations. Usually they work from 6 am to 2 or 3 pm, but some take more time and finish around 5 pm. This schedule starts on Monday and finishes on Sunday, so it can restart similarly the following

week. Consequently on an average they have a high and/or very high metabolic rate. Traditionally, the average productivity rate per worker has been 5.5 tons per day.

Currently, the harvesting season lasts six months. During this time, every foreman is responsible for handing out to each farm worker three small bags of 250 mL of rehydration solution, every day for as long as the harvest period process lasts. However, there is a lack of properly designed policies or measures destined to guarantee an adequate distribution and consumption of the rehydration solutions.

In 1997, Perez Lopez et al. (7) evaluated hydro-electrolyte modifications in sugarcane workers in western Nicaragua after they were given rehydration solutions while harvesting sugarcane.

They designed a study in which they evaluated 15 workers supplied with an electrolyte-balanced rehydration

solution and an equal number of workers given regular tap water. Some slight signs of dehydration were found in 80% and 73% of the two groups, respectively, while more severe dehydration was found in 33% of the tap water group and only among 20% of the rehydration solution group.

Ten years later, Dr. Solis Zepeda (8) conducted a controlled clinical trial on the same working population in western Nicaragua. His main objective was to evaluate the impact of preventive measures used to avoid damage to renal function caused by heat syncope (8). The analysed measures were rehydration solution (treated group) or regular water (control group) intake, provided by the company they work for. He evaluated 218 workers who drank rehydration solutions and 187 workers who drank regular water. Blood and urine samples were taken.

Serum electrolytes before beginning and after ending the day's work were monitored. The control group showed significant differences ($p=0.002$) in serum electrolyte levels (5 mEq/L sodium difference; -0.5 mEq/L potassium difference) compared to the treated group (1 mEq/L sodium difference; -0.6 mEq/L potassium difference).

When it came to analysing diurnal differences in serum creatinine, the differences were greater in the control group (creatinine levels of 0.50 mg/dL initially and 0.55 mg/dL at the end of the working day) than among cases (creatinine levels of 0.85 mg/dL initially and 0.94 mg/dL at the end of the working day) ($p<0.001$). Glomerular filtration rate was significantly impaired in the control group (-16 cc/min difference) compared with the treated group (-3.2 cc/min difference, $p<0.001$).

The initial proposal for this project was to improve rehydration measures by increasing the amount of rehydration solutions and their distribution only in two farms (El Zapote and Montelimar) as a pilot intervention plan. Results were to be evaluated afterwards and if they were successful, then in the next harvesting season (2007/2008) these policies were to be implemented in all the other farms.

Sampling methods were used in order to properly evaluate the heat load to which sugarcane workers

are exposed. Heat stress indexes such as WBGT were measured. A globe thermometer with a 15 cm diameter hollow copper sphere painted in black on the outside was used.

All measured values were above threshold limit values (TLV) considered normal by the Nicaraguan Ministry of Labour (9). It is important to mention that the Nicaraguan Ministry of Labour does not have its own TLVs and therefore it uses as national references those from the American Conference of Industrial Hygienists (ACGIH) and the National Institute for Occupational Safety and Health (NIOSH) (5).

The overall objective was to assess heat stress prevention measures in order to improve existing rehydration strategies as a means of increasing productivity.

Methods

Twenty-two workers were followed up from 15th April to 30th April 2008 during working hours, from approximately 6 am to 3 pm, for a total of 160 working hours. This included only workers who had worked more than 50% of each day's working period and had worked for at least 10 days of the follow-up period.

A simple data collection sheet was used. Information regarding the amount of liquid intake expressed (in litres, L) and daily productivity output (in tons) were recorded. The latter was facilitated through foremen's productivity data sheets.

The rehydration strategies were re-structured by decision makers participating in the project and the author of this paper. For an eight working hour schedule, all workers were instructed to drink 1 L of the rehydration solutions or tap water at least 30 minutes before they began to work. They were also encouraged to drink water and rehydration solutions (250 mL) every 30–45 minutes. In total, they were each asked to drink 10 L of liquid (tap water and rehydration solutions) per day.

In order to facilitate the monitoring of basic heat stress indexes during the 2007/2008 harvesting season, temperature, humidity and dew point were measured by using a portable temperature monitoring device ('EasyLog',

Table 1. Wet bulb globe temperature (WBGT) values during the 2007 harvest season at Montelimar farm

Time	Globe temperature	Dry bulb	Natural wet bulb	Measured WBGT	Permitted WBGT	Heat stress (%)
7:40 am	41	29.5	24.0	28.0	30.6	91.3
8:40 am	42	31.5	23.5	28.0	30.6	91.5
9:40 am	40	32.0	24.0	28.0	30.6	91.5
10:40 am	45	33.0	24.0	29.1	30.6	95.1
11:40 am	49	34.5	24.5	30.4	30.6	99.3
12:40 pm	45	34.0	25.0	29.9	30.6	97.7
1:40 pm	45	34.5	25.0	30.0	30.6	97.9

model EL-USB-2). This device was used to make hourly measurements. The data collected were downloaded to a PC for later analysis.

Variables such as heart rate and weight were evaluated at the beginning and at the end of the working day. Neither the amount of liquid intake nor productivity was validated.

Wet bulb globe temperature measurements recorded at Montelimar farm are shown in Table 1. Wind velocity was not evaluated due to the lack of proper equipment for measurement.

Statistical analysis

Descriptive statistics were used to analyse climate data (temperature (°C); relative humidity (%); dew point (°C)); liquid intake and output production. Chi-squared test was used to evaluate liquid intake versus output production by using SPSS version 13.

Results

Fig. 1 shows *in situ* hourly climate variation direct from the sugarcane fields while workers harvested the crop. As can be seen, temperature and relative humidity values oscillated from 23.5 to 34.5°C and from 40% to 64%, respectively, reaching maximum values as early as 8–10 am. This meant that water distribution had to be started at around that time on an hourly basis.

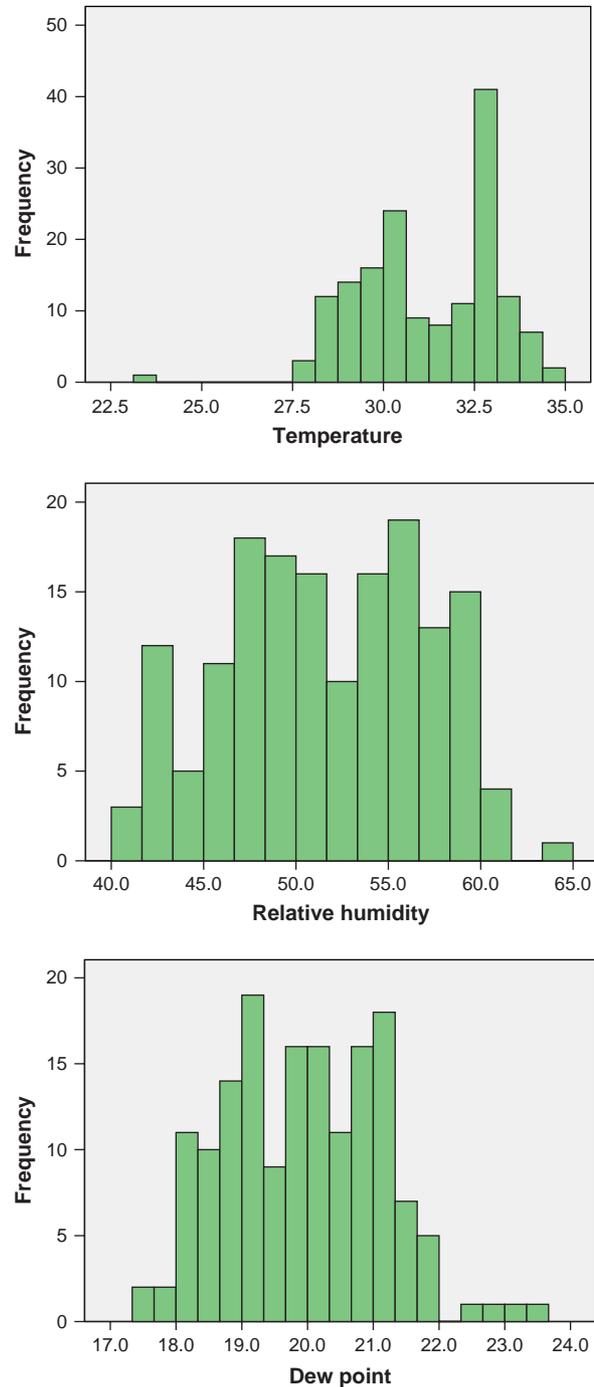
Table 2 shows the amount of liquid intake (including both regular tap water and specially formulated rehydration drinks). Seven workers drank from 7 to 8 L as temperature increased. What is of great concern is that, although temperature increased to maximum values early in the morning, many workers did not follow the rehydration measures and drank less than 6 L, a potentially dangerously low volume.

Fig. 2 shows relationships between daily production output and liquid intake. This showed that 13 workers who had the highest production output (range 6–8 tons, average 7.45 tons) were those who drank more than 6 L of liquid (regular tap water and/or specially formulated rehydration solutions) in comparison to those who drank less liquid and had a smaller production output. This finding is statistically significant ($p=0.005$).

Measurements of heart rate and body weight at the start and end of the working day showed that workers experienced increases in heart rate and loss of body weight as they worked in these hot conditions.

Discussion

Historically, monitoring of toxins in the work environment has been the primary focus for identifying risks. Some potential biomarkers linked to cell injury are immunological factors, lymphokines, growth factors, prostaglandins, endothelins, collagen, adhesion mole-



	Temperature (°C)	Relative Humidity (%)	Dew point (°C)
Mean	31.2	51.5	19.9
Standard deviation	1.9	5.4	1.2

Fig. 1. Climate measurements during 160 working hours of follow-up for 22 sugarcane workers at the Montelimar farm. San Rafael del Sur, Managua, Nicaragua, April 2008.

Table 2. Comparison between temperature measurements and daily water intake for 22 sugarcane workers at the Montelimar farm. San Rafael del Sur, Managua, Nicaragua, April 2008

	Daily water intake		
	Less than 6 L	6-7 L	7-8 L
Temperature (°C)	<i>N</i>	<i>N</i>	<i>N</i>
23.5-27.2	1	0	0
27.2-30.8	3	5	2
>30.8	5	1	5

cules, thromboxanes, leukotrienes, platelet activating factors and heat shock proteins (10).

As mentioned earlier, heat illness is a major cause of preventable morbidity worldwide (1) and although human beings possess considerable ability to compensate for naturally occurring heat stress, many occupational environments and/or physical activities expose workers to heat loads which are so excessive as to threaten their health and productivity (11). It is important to remember that the normal human body contains approximately 60% of water, about 34-40 L in an adult person (12).

The present project evaluated climatic conditions at sugarcane plantations located at sea level, on the southwest coast of Nicaragua. Monitored climate indices (temperature and relative humidity) values varied from 23.5 to 34.5°C and from 40% to 64%, respectively, reaching maximum values as early as 8 am.

According to the Nicaraguan Institute of Territorial Studies (INETER) (13), relative humidity values for the whole month of April varied between 69 and 79% all along Nicaragua’s Pacific coastline from San Juan del Sur (Southern Region) to Chinandega (Western Region). All sugarcane mills are located along the Pacific coastline. Managua lies between the two regions; the Montelimar sugar farm is located 62 km from Managua. However, the INETER humidity data are quite different from the relative humidity registered on the farm located in western Managua. Perhaps the fact that only 15 days were registered influenced this variation. Temperature data were not available at INETER’s website.

Although only 22 subjects were followed-up for a short period of time in this study, important results were obtained. Other authors have shown the relationship between heat stress health effects and the ability to perform different tasks, as well as the increased risk of suffering work-related injuries (14). In this study, the workers drank more liquid as temperature values

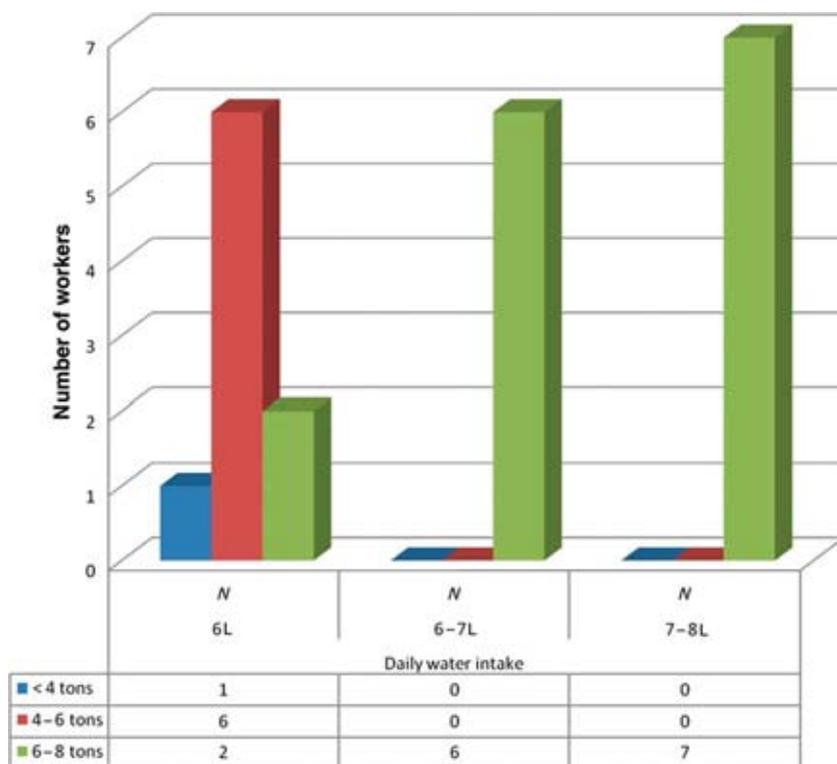


Fig. 2. Relationship between production output and daily water intake for 22 sugarcane workers at the Montelimar farm. San Rafael del Sur, Managua, Nicaragua, April 2008.

increased to maximum peaks. This was part of a rehydration process which was well planned in advance by the company's decision makers. Unlike during past harvesting seasons, when water and rehydration solutions were distributed quite randomly, during this harvesting season cool water and specially formulated drinks were distributed or intended to be distributed to workers, who received 1 L just before they began their working day and then 500 mL every 30 minutes.

The basis of this principle is that drinking to satisfy thirst is not enough to keep a person well hydrated. Most of the people become aware of thirst once they have lost 1–2 L of body water and persons highly motivated to perform hard work may incur losses of 3–4 L before serious thirst forces them to stop and drink. Since dehydration reduces the capacity for absorption from the gut, workers must be educated regarding the importance of drinking enough water during work and continuing generous rehydration during off-duty hours (14).

Productivity was positively influenced by the new rehydration measures. There was a significant increase of production, with up to 8 tons per worker during the follow-up period compared to the normal 5.5 tons per worker prior to the change in rehydration measures.

This important change in rehydration policies and increase in production output is the result of various efforts of training workers, foremen and managers on heat stress prevention, proper hydration measures and quality of (working) life carried out by occupational health and safety professionals (physicians and engineers), human resources departments and top management at sugarcane farms.

However, this was not an easy task. Often workers rejected the new rehydration measures, most of the time because it was difficult for them to understand thoroughly the dehydration and physiological compensatory mechanisms. Some of the reasons for this can be attributed to their low educational level, and feeling that 'nothing bad has ever happened to me before', etc.

Certainly more effort in terms of intervention strategies and scientific investigation needs to be carried out among workers in Nicaragua who perform jobs in which they are exposed to high ambient temperatures. These include farm workers, construction workers, miners and fishermen, especially those employed in the informal sector, which occupies about half of Nicaragua's economically active population.

More funds should also be designated by companies' decision makers for improving basic working conditions, in order to increase overall productivity (and workers' satisfaction in terms of better wages). This would also

translate into safer and healthier workers, less absenteeism from sick leave, fewer accidents and other incidents.

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References

1. Jason Hoppe DO. University of New York Downstate Medical Centre, Kings County Hospital. Heat exhaustion and heat-stroke. March 2006. Emergency medicine. Available from: www.emedicine.com/emerg/topic236.htm [cited 25 April 2009].
2. Bernard TE, Cross RR. Heat stress management: case study in an aluminium smelter. *Ind J Ergonom* 1999; 23: 609–20.
3. Rodahl K. Occupational health conditions in extreme environments. *Ann occup Hyg* 2003; 47: 241–52.
4. NIOSH. Working in hot environments. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 86–112. Available from: <http://www.cdc.gov/niosh/hotenvt.html>; 1986 [cited 25 April 2009].
5. Occupational Safety and Health Administration. Heat stress. Technical manual, Section III, Chapter 4. Occupational Safety and Health Administration, Washington, DC. Available from: www.osha.gov/dts/osta/otm/otm_iii/otm_iii_4.html [cited 15 April 2009].
6. Tierney LM, Jr., McPhee SJ, Papadakis MA. Heat disorders. Current medical diagnosis and treatment. 1999. Mexico: Manual Moderno; 2000. (Spanish edition)
7. Pérez López R. Water and electrolytes balance changes among sugar cane farmers after given an electrolyte formulated solution at the "San Antonio Sugar Mill". Final Thesis report. School of Medicine, Universidad Nacional Autónoma de Nicaragua-León, León, Nicaragua, 1997.
8. Solís Zepeda Guillermo A. Impact of preventive measures designed to avoid the deterioration of renal function due to Heat Disease among farmers at the "San Antonio Sugar Mill" in western Nicaragua, during the 2005/2006 harvest period. Final Thesis Report for the specialty of Internal Medicine, Universidad Nacional Autónoma de Nicaragua-León, León, Nicaragua, 2007.
9. Nicaraguan Ministry of Labour. Compilation of laws and policies concerning occupational hygiene and safety. Project of the World Fund. March 2008.

10. Hemstreet GP. Biomarkers of nephrotoxicity. Renal-urinary systems. Encyclopedia of occupational health and safety, 4th ed. Geneva: International Labour Office; 1998.
11. Nunneley SA. Prevention of heat stress. Encyclopedia of occupational health and safety, 4th ed. Geneva: International Labour Office; 1998.
12. Nielsen B. Effects of heat stress and work in the heat. Encyclopedia of occupational health and safety, 4th ed. Geneva: International Labour Office; 1998.
13. Nicaraguan Institute of Territorial Studies, INETER. Climate tables for the month of April 2008. Available from: www.ineter.gob.ni [cited 13 June 2009].
14. Staal Wästerlund D. A review of heat stress research with application to forestry. Applied Ergonomics. UK: Elsevier Science Ltd; 1998.

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A pilot field evaluation on heat stress in sugarcane workers in Costa Rica: What to do next?

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Background: Climate change is producing major impacts including increasing temperatures in tropical countries, like Costa Rica, where the sugarcane industry employs thousands of workers who are exposed to extreme heat.

Objectives: This article outlines a pilot qualitative evaluation of working conditions and heat in the sugarcane industry.

Design: A literature review, direct observations and exploratory interviews with workers were conducted to reach a preliminary understanding of the dimensions of heat-related health issues in the sugarcane industry, as a basis for the design of future studies.

Results: The industry employs temporary workers from Nicaragua and Costa Rica as well as year-round employees. Temporary employees work 12-hour shifts during the harvest and processing ('zafra') season. In many cases, sugarcane field workers are required to carry their own water and often have no access to shade. Sugar mill workers are exposed to different levels of heat stress depending upon their job tasks, with the most intense heat and workload experienced by the oven ('caldera') cleaners.

Conclusions: Research is needed to achieve better understanding of the multiple factors driving and interacting with heat exposures in the sugarcane industry in order to improve the health and safety of workers while maintaining worker productivity.

Keywords: *sugarcane; Central America; Costa Rica; heat exposure; heat stress; agriculture; climate change*

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Climate change is producing major impacts worldwide including increasing temperatures, particularly in tropical countries (1). Increasing temperature raises environmental as well as serious human health concerns. Although the human body is remarkably capable of maintaining its core temperature around 37°C, a combination of conditions such as high environmental temperature and high humidity (which decreases the efficiency of sweating), clothing that increases core temperature (and reduces ability to sweat), physical exercise and dehydration can disturb the equilibrium. This can result in heat stress and, when severe, can cause heat stroke and death (2).

Exposure to excessive heat is common in sectors like agriculture, mining, construction and manufacturing plants ('maquiladoras'), particularly in tropical countries where these sectors comprise a large proportion of the workforce. Increasing temperatures can mean increasing

exposure to extreme heat and increasing risk for negative health outcomes for worker populations (3). Although governments, the private sector, NGOs and academia have begun to focus on the economic and environmental impacts of global climate change, there has been little attention to what effects global heating will have on the health and productivity of the worldwide labour force (2).

The sugarcane industry in Costa Rica is an important contributor to the national economy through the production of multiple products for national and international consumption as well its contribution to recent regulatory requirements for bioethanol to be included as a constituent of petroleum-based fuels. The production and processing of sugarcane is a major source of employment in Costa Rica for both Costa Ricans as well as Nicaraguans who travel to work in the harvest and processing ('zafra') period. According to the Sugarcane Industrial Agriculture Association (LAICA), the sugar

industry in Costa Rica employs 20,000 permanent workers (4). Temporary workers come from both Costa Rica and Nicaragua and are either employed directly by the company or by an individual who offers subcontracting services to companies. Estimates for the number of temporary workers employed by the sugarcane industry vary enormously, without verifiable numbers. In some companies, the number of temporary workers needed for the harvest has actually gone down due to increased use of mechanised harvest techniques (4). During the zafra season, all efforts concentrate on the successful harvest and processing of the year's crop. The large number of workers needed to achieve this goal creates occupational health issues that are complicated by the focus on the harvest, the intense heat and the influx of temporary workers.

Sugarcane is grown in multiple regions throughout the country, with the most sugarcane being produced in Guanacaste in northwest Costa Rica, approximately 250 km from the capital city of San José. In Guanacaste, there are three large plantation companies (with sugar mills and fields) which, together with numerous independent farmers, are collectively responsible for approximately 59% of the total sugarcane harvested and 55% of the total sugar produced in Costa Rica (4).

Guanacaste is one of the hottest and driest regions of Costa Rica and has two main seasons: the rainy season (May–November) and the dry season (December–April). Sugarcane harvesting, planting and processing take place during the hottest (dry) season. Between 1973 and 1994, the monthly averages of daily maximum temperature ranged from 31.1°C in October to 36.0°C in April (Table 1) (5). Official data for more recent years is not publicly available.

This report outlines a pilot qualitative evaluation to explore the dimensions of heat-related health issues in the sugarcane industry.

Present study

Methods

A brief qualitative assessment of factors related to heat stress and its health effects was conducted between November 2008 and March 2009. Published and unpublished documents about sugarcane production in Central America and heat-related human health effects were identified by searching PubMed as well as Costa Rican and Central American popular press available online. Brief exploratory interviews were held with:

- (1) four occupational health researchers with experience in the sugarcane industry;
- (2) one occupational health professional from a sugarcane company in Guanacaste;
- (3) a company nurse and physician with experience treating sugarcane workers; and
- (4) 17 workers (12 sugar mill and five field workers).

The interviews contained questions about the tasks that workers complete; the heat conditions they face; strategies for reducing heat-related health effects; the possibilities for measuring worker productivity; health effects and heat stress in future studies; and whether or not they considered it important to conduct research about heat-related health conditions. In addition, during the 2008–2009 harvest, direct observation of the conditions and work loads present in the sugar mill and the field was conducted on multiple visits to one company.

Table 1. Climate data from the National Meteorological Institute of Costa Rica (Daniel Oduber Airport), Liberia, Guanacaste, Costa Rica

Month	Average maximum temperature (°C) (1973–1994)	Average minimum temperature (°C) (1973–1994)	Average monthly precipitation (mm) (1957–1994)
January	33.4	20.8	0.8
February	34.5	21.1	2.0
March	35.5	21.6	4.5
April	36.0	22.4	13.9
May	34.2	23.3	191.8
June	32.1	23.0	254.2
July	32.0	22.7	150.2
August	32.0	22.5	201.5
September	31.3	22.3	319.4
October	31.1	22.0	290.6
November	31.7	21.4	91.2
December	32.6	21.1	9.8

Results

The sugarcane industry includes two main areas in which workers are exposed to extreme heat: field work and processing plant (sugar mill) work (Fig. 1). Both areas include work during the zafra season as well as the maintenance (non-zafra) season. For the purpose of this report, *field work* includes harvesting and planting during zafra and maintenance of the crop during no-zafra. Work in the *sugar mill* includes all tasks beginning with unloading the harvested sugarcane from the trucks and ending with the production of refined sugar products during zafra season, and during the non-zafra period repairs and preparations of the machinery and infrastructure in the sugar mill for the next harvest period.

Literature review

Very few scientific findings exist about the risks faced by sugarcane workers in Central America. There have been a few studies in the Americas on specific risks among sugarcane workers such as cancer (6, 7), respiratory conditions (8) and musculoskeletal injuries due to repetitive motion and machete use (9), but none of these studies make links to heat or climate change. There has been some important work done regarding socio-economic and gender-related issues for sugarcane workers (10, 11) which although not directly linked to heat stress, provides important context for understanding heat-related health issues.

Some technical reports have been published on general risks present in the industry, including heat (12, 13). For example, a report from the International Labor Organization (ILO) highlights the exposure of workers to noise; vibration; contact with agricultural chemicals; repetitive

motion; machinery-produced heat; environmental heat (temperature and humidity); ultraviolet solar radiation; and visible light in addition to climatic conditions such as rain, wind and lightning (13).

Likewise, within the Program on Work and Health in Central America (SALTRA), a participatory methodology was developed to reduce the risk of accidents and various occupational exposures in the sugarcane industry. Eight participatory workshops were held with workers and managers to identify hazards along with possible solutions. In total, workers identified 311 hazards and mentioned one or more solutions for 89% of the hazards. In addition to exposures such as noise and dust exposure, heat-stress was identified as a main occupational health problem in these workshops (12).

At the regional level, research has been conducted and is ongoing concerning an important human health outcome that directly affects many sugarcane workers and which may be linked to climate. Epidemics of chronic kidney disease (CKD) in sugarcane workers have been reported in El Salvador and Nicaragua (14–18, 25), while Costa Rica and other countries in Central America report higher than expected occurrence of CKD in sugarcane workers (19). One repeatedly mentioned hypothesis is that the high number of sugarcane workers suffering from CKD may be at least partially due to chronic dehydration related to working conditions.

Although there has been little research regarding heat-related health issues, there have been some studies regarding climate change in the Guanacaste Region. A report produced by the Central American Integration System (SICA) (20) as well as the popular press (21) have warned that the northern part of the country will

Month	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec	Jan	Feb	Mar	Apr
Monthly mean max outside temp. (°C)	34.2	32.1	32.0	32.0	31.3	31.1	31.7	32.6	33.4	34.5	35.5	36.0
Work period	Non-zafra							Zafra				
Field work (Outside)	Maintenance irrigation: in sunshine											
								Harvesting: in sunshine				
								Planting: in sunshine				
Mill work (Inside)	Maintenance											
								Clarifiers: hot tanks				
								Evaporators: hot tanks				
								Calderas: very hot furnaces				
								Centrifuges: hot tanks				
								Tachos: hot tanks and steam				
							Patio: unloading in sunshine					

Fig. 1. Division of work in the sugarcane industry in Costa Rica.

experience temperatures as much as 3°C higher over the next 90 years and there will be a reduction in rainfall of up to 11%.

Nationally, there has been a small increase in research regarding climate change and its links to production of food and export products (22). This is due not only to the increased awareness of climate change, but also the economic crisis, which has fostered renewed interest in national, sustainable and self-sufficient food production (23, 24).

Information from interviews and observations

Harvest. Sugarcane harvesting by hand is an intense job that involves constant exposure to heat and sunlight (see Fig. 2). Many farms burn the sugarcane in the evening or night hours, before the harvest. This is done before both manual and automated (tractor) harvest to decrease the sharpness of the leaves. Sugarcane farmers also report that the burning decreases the probability of snake bites during manual harvest and reduces the weight of the cane for transportation purposes. Some say burning increases the quality of the sugar. Burning is carried out by a group of approximately six workers that start and control the fires making sure that they burn only the amount that is possible to harvest the next morning, to avoid the risk of sugar content in the cane decreasing while waiting for harvest.

Harvesting starts in the morning and lasts until the early afternoon hours, usually in fields still smoking from the previous night's burning. When the sugarcane is cut by hand with machetes, workers are usually paid by the ton or by the number of rows they cut in teams of three workers. As noted in the ILO report on working conditions in the sugarcane industry in Costa Rica (13), the heat stress for sugarcane harvesters is increased because of the high physical demands on them. In-



Fig. 2. Sugarcane harvesting in Costa Rica takes place under direct sun, often in fields that have been burnt the night before.



Fig. 3. Automated sugarcane harvesters with air-conditioned cabs are increasingly used in many farms, but many workers are still employed to cut sugarcane by hand.

ingly, larger farms are using automated sugarcane harvesters (see Fig. 3), but still rely on manual labour in sections of fields that are too small or too uneven for the tractors. Tractor drivers are enclosed within air-conditioned cabs.

The harvest activities in the field also include the transportation of the sugarcane to the sugar mill. Depending upon the farm or company, transportation may be done with large 'semi' trucks with air-conditioned cabs or with tractors (without cabs) that pull a large open wagon containing the harvested sugarcane.

Planting. Sugarcane plants have a productive life between one and five years and when a field needs re-planting, it is done by hand. Planting usually takes place the day after the harvest. In most cases, planting is done between 05:00 and 14:00 and involves several tasks. Some workers prepare sacks of 'seed' (the shoots are referred to as 'seed' in Costa Rica, although they are actually seedlings or 'shoots'). Other workers distribute the sacks in the field; while a third group of workers plants the shoots. Most companies sub-contract the planting and a typical contractor employs approximately 150 workers that are responsible for three hectares per day. Reportedly, the workers that prepare the sacks often work longer hours in order to fulfil the quota (approximately 5,000 sacks) that must be ready for the following day. The group of planters travel to a new field each day and workers are usually paid by the number of rows or metres that they plant. Later, planters are sometimes employed to 're-cover' ('re-tapar') the plants with soil, a process done days or weeks after the initial planting in which workers make sure each plant is sufficiently covered with soil at its roots. Planting also requires individuals employed as 'counters', responsible for confirming the number of metres planted by each worker. Traditionally

only men are employed in the tasks related to planting, but there are anecdotal reports that an increasing number of women are also employed in many of these tasks.

Irrigation and general field work. Sugarcane companies directly hire both permanent and temporary workers to carry out irrigation and general field up-keep tasks during the harvest period. The vast majority of these workers are employed starting with the harvest (December), but continue after the harvest ends (usually in the beginning of April) until the rainy season starts (usually in May). During the dry season, irrigation takes place continuously and in most companies, shifts run from 06:00 to 18:00 and then from 18:00 to 06:00. In some companies, workers rotate one week on the day shift and one week on the night shift for the duration of the harvest and planting season. The rotating schedule allows all workers to earn overtime and all of the workers consulted preferred the night shift to the day shift because of the decreased heat and the increased pay.

Irrigation may be water or may be water combined with fertiliser (such as ash or other by-products of the processing, which provide nitrogen and other nutrients to the plants). Irrigators often work alone in large fields. They are exposed to direct sunlight with no trees or other sources of shade. The company bus drops them off at their assigned area for the day and a supervisor passes by throughout the shift to check on progress. Irrigation usually requires continuous walking and considerable physical effort throughout the majority of the 12-hour shift.

Specific tasks for irrigation workers include hooking up the tubes, unplugging rubber stoppers to allow water to flow to a specific region of the field, using a tarp to direct water and continually levelling the soil between the rows to allow the water to enter undisturbed. Irrigation workers carry a shovel with an extra-long handle which they use to level the ground and also to help them cross large irrigation ditches. Most workers consulted for this exploratory study reported taking or not taking breaks depending on '*how my [irrigation] water is going*'. In other words, if the water is flowing well, workers are able to take short breaks, but if the rows where the water enters require a great deal of work with the shovel, it is not possible to take breaks.

Other workers are hired by the company for general maintenance and tasks in the field. These workers include both those hired seasonally as well as those who work for a particular company year-round. Typical tasks include: picking up rocks in the field before the harvesting tractor enters, cutting weeds and grass with a machete, picking up sugarcane left behind by the tractor harvesters in the field, fixing leaks in the irrigation system and keeping the irrigation canals free of debris.

Finally, workers are employed to carry out application of different kinds of pesticides and fertilisers. Depending on the company, these workers can be inside an enclosed tractor cab, use a knapsack sprayer, a 'boom' sprayer, a mechanical sprayer behind the back of a four-wheeled motorcycle or in a combination of workers on foot and in a tractor. Both general maintenance workers and pesticide applicators usually work alone and are exposed to direct sunlight, although on some days, they may be near trees or other sources of shade under which they can seek brief respite or where they can leave their lunches and water jugs.

Maintenance (non-zafra) period. The maintenance (non-zafra) period is a calmer period that employs considerably less workers, but still involves working in conditions of exposure to considerable heat and solar radiation. Workers during the maintenance season are almost always full-time, year-round employees of a company hired to carry out tasks such as the application of pesticides and fertilisers, manual weed control and the maintenance of irrigation canals. The majority of the non-zafra season coincides with the rainy season, which tends to be somewhat cooler (see Table 1). Additionally, there is far less pressure to complete tasks at a rapid rate during 12-hour shifts.

Heat-related issues and solutions for field workers

With the maximum daily temperature almost never dropping below 31°C and the intense direct sunlight as a result of Costa Rica's latitude, the potential for heat stress in field workers is high. Furthermore, the fields probably retain additional heat following burning and, as one person stated, '*you can literally see the ground smoking and fuming*' the morning following a burning. There are anecdotal reports of heat stroke victims resulting in hospitalisation in at least some companies. Intravenous rehydration treatment is reportedly relatively common in the medical clinic of large companies.

Occupational health workers pointed out that severe dehydration cases are often linked to complex social issues, such as workers who fear seeking medical care because they lack documentation and who, therefore, may work in the fields while sick and after not having eaten for several days.

It is speculated that chronic dehydration, exacerbated by exposure to extreme heat may be a serious problem for sugarcane field workers and may, therefore, be linked to the high prevalence of CKD among sugarcane workers (14–19, 25). Workers mention the need to drink water '*for your kidneys*' and one worker reported '*many co-workers are affected by kidney problems*'.

The main strategy currently in place for dealing with heat stress in the field is hydration, however, health and safety personnel in the sugarcane industry have expressed

concern that hydration just with water is not sufficient and that there may be a need to consider nutritional factors and electrolyte levels when providing hydration for workers. In many companies, field workers, whether employed directly by the company or by contractors, are responsible for carrying the water they need for the entirety of their shift. Most field workers consulted said they take between 2 and 10 liters of water into the field with them. Since there is no shade available for most workers, they wrap their water jugs in a wet cloth when they leave their house to keep the water from over-heating in the direct sunlight.

Most workers reported drinking either coffee or 'fresco' (fruit juice mixed with water and sugar) during the day and most reported drinking at least one glass of water or fresco before leaving the house and when returning home at the end of the shift. One worker reported that one must be careful not to consume too much water before doing machete work, as to avoid '*jumbling up and bothering the stomach*'. In general, the amount of liquid consumed by workers varied considerably, but it is likely that, in the majority of cases, it is far below the amount of liquid lost by perspiration under the working conditions of the zafra. It is common to hear workers saying they believe the drinking water available to them at home and work is contaminated and some of these workers believe that the water may be related to the high incidence of kidney disease in sugarcane workers.

In order to protect themselves from solar radiation (as well as the sharp sugarcane leaves) workers use long sleeves, long pants and neck covering either as a cap with a flap in the back or a cap with a handkerchief hanging to cover the neck. Most wear rubber boots and many wear two shirts (one long-sleeved 'button-down' shirt underneath and one short-sleeved 'button-down' shirt on top) to lessen the burning sensation created by solar radiation.

A half-an-hour lunch break and two 15-minute breaks are required for eight-hour shifts in Costa Rica. However, during the harvest and planting, temporary field workers are trying to make as much money as possible during the four to five months they have employment in the sugarcane industry. Temporary workers said that they are paid by the amount of work they complete and are, therefore often reluctant to take breaks. Temporary and permanent workers said that sometimes their workload prevents them from taking a full lunch period or the permitted 15-minute breaks.

When asked whether they believe the heat or 'especially hot days' affect their productivity, most field workers said that they feel they get tired faster, but most of them also qualified their statement saying that they manage to complete their tasks regardless.

Description of sugar mill work

The sugar mill is an intense work environment during the harvest and processing season due to the high number of workers, the 24-hour work schedule, the intense heat and the volume of work, whereas there are very few workers and much calmer conditions during the maintenance period. Workers in the sugar mills may be temporary or year-round, but all are employed directly by the company. Shifts during the zafra period are 12-hour shifts as described above for the field workers.

Typically, the sugar mills are large three-storey buildings with corrugated metal exterior walls and roofs. The infrastructure, including stairs and handrails, is made of metal and the flooring is cement. There are different sections of the plant, some of which have ventilation in the form of an open section in one of the exterior walls while other sections of the plant are more closed off. Most of the processes produce heat and, as a result, the entire plant is warm and many areas are filled with hot vapour. The stairs and handrails are warm to the touch.

The sugar mills are run by electricity produced onsite using steam created by large ovens which burn 'bagasse', the parts of the sugarcane plant left after crushing the cane to extract the juice. The ovens are located within the sugar mill and create extremely hot working conditions. The temperature in the sugar mill remains high during day time and night time because the ovens ('calderas') keep running all night.

In order to maintain the machines and tubing through which the vapour travels, there are periodic purges of steam, usually several times per shift. Some of these purges are directed outside the plant, but others are actually purged inside the plant. Previous measurements taken in Nicaragua and Costa Rica by the SALTRA programme during the harvest season demonstrate the wet bulb thermometer temperature varies between 30.1 and 37.0°C.

Patio. The 'patio' workers (where the sugarcane is unloaded from trucks into the sugar mill) are the only sugar mill workers exposed to direct sunlight as a part of their regular tasks. One worker remains in an air-conditioned control cabin, but the others help with the unloading process and are on their feet in the direct sunlight (during the day shift) for most of the 12 hours.

Tachos. Approximately two workers per shift typically work as 'tacheros', controlling the quality of the sugar by controlling pressure and volume in large tanks. The job is quite detail-oriented and requires experienced workers. In a typical sugar mill, the tachos area is located on the third floor which absorbs heat from the tin roof as well as accumulating heat that rises from below, but does have large open spaces at either end that allow for a breeze on windy days. Tacheros are on their feet for most of the

shift, but are able to sit for brief periods on wooden benches located near the open spaces. This area of the sugar mill is particularly exposed to the purges of steam (described above). The tachos area is the only one which has an air-conditioned chamber where a computer is stored and where some of the tacheros occasionally seek relief from the heat. Most, however, are reluctant to do so as they feel the extreme changes in temperature are unhealthy.

Centrifuges, clarifiers and evaporators. The centrifuge, ‘clarifying’ and evaporating areas are located within the sugar mill. In all three areas, workers are on their feet and must pay attention to detail, but they are also able to sit down for short periods of time. The centrifuge area is particularly hot because the large tanks present release both heat and steam on a continuous basis.

Calderas (ovens). The calderas that burn bagasse to produce electricity must be cleaned of ash. A team of 4–5 workers is dedicated to this task which involves opening a large iron door of an oven at 250–270°C. One worker sprays the burning ash with a garden hose until it stops burning, while the others use metal poles and shovels to pull the smouldering ash out and place it on the palette of a forklift. The process takes approximately 2.5 hours and is done twice per 12-hour shift. These workers are exposed to extreme heat, risk of burns and inhalation of particulate matter but also to an extremely intense physical work load. When they are not cleaning the ovens, these workers are responsible for general upkeep tasks or for helping other workers wherever it may be necessary.

Maintenance (‘non-zafra’) period. During the maintenance period, there are fewer workers employed by the company. Those that are employed carry out tasks related to the maintenance of machinery. The plant does not burn material to produce electricity during the maintenance period, significantly reducing the temperature in the sugar mill and the rainy season from May to November also brings decreasing outside temperatures.

Heat-related issues and strategies for sugar mill workers

Hydration is the principal strategy for reducing heat effects for sugar mill workers and in some companies, water tanks are located in several spots throughout the three-storey plant. They are filled with tap water. The workers consulted said that, depending on where the tap is located, the drinking water comes out hot. Some suspect the tap water is contaminated, with one worker even reporting that he and his co-workers add bleach to the water to purify it. Workers said they receive plastic ‘sandwich’ bags full of a rehydration drink once per 12-hour shift and that sometimes if they get two bags, they

share them with the workers in the hottest areas. Some workers also mentioned going to the company cafeteria to purchase soft drinks or juice drinks. A few of the workers indicated that it is important not to drink water that is ‘too hot’ or ‘too cold’ because it is ‘bad for the organism’. The caldera workers interviewed reported being able to get a juice drink at the cafeteria for free. Workers varied as to how much liquid they reported drinking, with the average being about 5.5 liters of water per 12-hour shift. Caldera workers reported drinking up to 7 liters during the 2.5 hours it takes them to clean the ovens each time. Most workers, however, were unsure how much liquid they consume.

Workers in the plant reported that the heat affects their productivity, particularly in the case of the workers whose jobs require attention to detail because the heat affects their ability to concentrate. Several workers reported feeling tired from the heat, and said that sometimes they take a caffeinated aspirin pill to combat this feeling, whereas others take acetaminophen for headaches. Most workers wear long sleeves and a hardhat. It is common to see workers take off their gloves due to the heat. Oven cleaners wear a helmet, safety glasses, long sleeves, long pants, rubber boots, a hat and a handkerchief or other piece of cloth (sometimes the hood portion that has been cut off of a jacket which they zip or velcro in front of the mouth and nose). Approximately half of the oven cleaners wear gloves while cleaning the oven, while the other half remove them due to heat. The oven cleaners reported either dumping water on their heads or putting their feet (while in rubber boots) in cool water in order to cool off. All workers said that they preferred working at night because it is cooler.

Discussion

One of the most pressing occupational health hazards for sugarcane workers is exposure to extreme heat. The effects of global climate change have already been shown to be affecting the Guanacaste region of Costa Rica (21, 22) and more negative effects are expected in the coming years (24). Additionally, there have already been studies on the potential effect of climate change or climate variability on the economics, production and pest-related problems in the sugarcane industry (22, 27). There are, however, no studies investigating the effect of climate change and increasing heat on workers’ productivity or health in Costa Rica. The high numbers of Costa Rican and Nicaraguan workers employed in the industry, as well as the economic importance of the industry, make research of utmost importance and urgency, particularly in light of the expected climate changes.

Costa Rica has a regulation in place to protect workers from extreme heat; however, it is a vague document that simply states: ‘The temperature and humidity level of the environment in enclosed workplaces should be maintained

(when the nature of the industry allows), between limits that are neither uncomfortable nor dangerous for the health of workers' (28). The National Advisory Board responsible for determining workplace temperature and humidity limits recommends that companies follow norms set by the Institute for Technical Norms in Costa Rica. This document defines the maximum temperature and humidity levels dependent upon the type of work being carried out (26). These norms do not set limits by industry or by type of work, but rather provide information for making appropriate calculations for work and rest periods. In Costa Rica, each company is responsible for setting its own limits and there is almost no government-level enforcement of the vague guidelines. There is interest on the part of management in most sugarcane companies to look for solutions to reduce workers' exposure to extreme heat as it affects both health and productivity.

Although some potential solutions are already in place within some companies, such as providing water dispensers inside sugar mills, their effectiveness has not been studied. One potential solution that has been suggested is providing break periods in air-conditioned rooms. Interestingly, this solution may present cultural barriers, and therefore may require a holistic interdisciplinary and participatory approach to research. In particular, there is a strongly held belief in Costa Rica that moving from hot to cold temperatures will cause one to become sick (for example, catch a cold or develop arthritis). Similar beliefs exist about drinking water or other liquids that are cold. There have been no studies to investigate the viability of rest chambers or the provision of cold drinks in terms of the tendency of Costa Ricans to avoid temperature extremes. Other strategies implemented by workers at the individual level may be having negative effects (for example taking caffeinated aspirin pills) or be positive initiatives (for example cooling water in the field by wrapping water jugs in wet cloth, or cooling feet by placing them in cool water while wearing rubber boots) that could be encouraged on a larger scale.

Hydration is another example of a commonly used approach already in place within the sugar mills to decrease potential negative effects of heat exposure. However, health and safety personnel indicated that they believe factors such as nutrition and electrolyte levels should be incorporated into hydration programmes in order to make them more effective. Although companies have expressed interest in a more integral nutrition-based approach to hydration as a means of combating heat stress, there have been no studies to explore the dietary and hydration habits already in place or the possibility of implementing hydration programmes in the sugarcane industry. Particularly in Guanacaste, hydration is complicated by what appears to be a widespread belief that the water is unsafe and can lead to kidney problems. This

possibility warrants an integrated research model that includes testing the drinking water for nephrotoxins.

When developing and evaluating solutions for heat stress in the sugarcane industry, it is important to recognise that there is a lack of data on the heat conditions that the sugarcane workers face. It is necessary to measure heat and other climatic conditions (such as humidity, dew point and wind speed) that affect the body's ability to regulate heat. Additionally, it is critical that the workload of each person be described fully and that the work of women in the field is included in the documentation. Finally, it is critical that the socio-economic and cultural dimensions of current and potential solutions be considered, particularly taking into account the workers' experiences, knowledge and perspectives. Without these indicators, it is difficult to create and evaluate potential solutions that address worker health and productivity. It is also essential that the health-related effects of heat stress be documented through a cohort study that could measure factors ranging from biomarkers of heat stress to psychological effects of heat exposure. Likewise, it is important that climate conditions are being appropriately measured and recorded in order to measure possible climate change.

There seems to be increasing political and public awareness about the link between global climate change and health as well as an appropriate political environment for action (21, 24, 29–31). The Costa Rican government is currently in the process of planning and policy making for adaptation to climate change. Unfortunately, there is a severe lack of research data upon which to base these critical policy-related decisions, making the need to produce data particularly important.

Meanwhile, some solutions for apparent problems can already be implemented, such as creating ways for workers to access to shade in the fields or establishing and enforcing rest breaks for workers and implementing improved access to water or integrated hydration plans. These interventions should be evaluated for both their effectiveness as well as their potential for cultural acceptance.

Strengths and limitations

This brief exploratory description of the heat-related issues faced by sugarcane workers falls short as it does not include heat measurements, nor detailed information from the temporary sugarcane harvesters and planters. This group of workers is probably the most exposed and the most vulnerable, yet it is the least studied. This population also faces socio-economic conditions that require an in-depth understanding of the issues related to their social, living and working environments (4, 10, 11). Integrated, holistic methodologies such as an ecosystem health approach (32) are necessary to understand

the multiple dimensions of the environmental, health and socioeconomic issues that require sustainable solutions.

Conclusion

The sugarcane industry is extremely important to Costa Rica's economy. Sugarcane workers in Costa Rica face exposure to extreme heat under current climatic conditions, which are likely to worsen with predicted climatic changes. At present, there is strong national political interest in preparing for any negative health effects of climate change. Research is needed to achieve better understanding of the multiple factors driving and interacting with the sugarcane industry in order to improve the health and safety of workers while simultaneously maintaining worker productivity.

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References

1. IPCC. International Panel on Climate Change. Fourth assessment report. Geneva, Switzerland: Inter-governmental Panel on Climate Change; 2007. Report No.: AR4. Available from: http://www.ipcc.ch/publications_and_data/publications_ipcc_fourth_assessment_report_synthesis_report.htm [cited 23 October 2009].
2. Kjellstrom T. Climate change, health exposure and productivity. ISEE 2000, 12th conference of the society for environmental epidemiology, August 2000. Buffalo, NY. *Epidemiology* 2000; 11: S144.
3. Kjellstrom T. Climate change, direct heat exposure, health and well-being in low and middle-income countries. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.1958
4. Acuña-González G. Diagnostico: la agroindustria de la caña de azúcar en Costa Rica: características, organización y condiciones laborales [The agricultural industry in Costa Rica: characteristics, organization and labor conditions]. San José, Costa Rica: asociación servicios de promoción laboral [Association services for labor promotion] (ASEPROLA); 2004.
5. Stolz-España W, Agüero Porras J. Climatología aeronáutica: aeropuerto internacional Daniel Oduber Quiros [Aeronautic climatology: Daniel Oduber Quiros International Airport]. San José, Costa Rica: Instituto Meteorológico Nacional and El Ministerio de Ambiente y Energía [National Meteorological Institute and the Ministry of Environment and Energy]; 2008.
6. Coble J, Morris-Brown L, Hayes R, Huang W-Y, Winn D, Gridley G, et al. Sugarcane farming, occupational solvent exposures, and the risk of oral cancer in Puerto Rico. *J Occup Environ Med* 2003; 45: 869–74.
7. Sinks T, Goodman MT, Kolonel LN, Anderson B. A case-control study of mesothelioma and employment in the Hawaii sugarcane industry. *Epidemiology* 1994; 5: 466–8.
8. Sinks T, Hartle R, Boeniger M, Mannino D, Boyd JE, Fernback J, et al. Exposure to biogenic silica fibers and respiratory health in Hawaii sugarcane workers. *Occup Med* 1994; 36: 1329–34.
9. Clementson CL, Hansen AC. Pilot study of manual sugarcane harvesting using biomechanical analysis. *J Agric Saf Health* 2008; 14: 309–20.
10. Rossi-Rocha F, Palucci Marziale M, do Carmo Cruz Robazzi M. Poverty as a predisposing factor of illness tendencies in sugarcane workers. *Rev Latino-Am* 2007; 15: 736–41.
11. Loría R. De Nicaragua a Costa Rica y a Nicaragua. La ruta crítica de las mujeres migrantes nicaragüenses: una mirada desde la zona norte fronteriza [From Nicaragua to Costa Rica to Nicaragua. The critical route for women migrants from Nicaragua: a look from the North Frontier Zone]. San José, Costa Rica: Alforja; 2002.
12. Vergüizas-Valverde M, van Wendel de Joode B, Rojas Garbanzo M. Metodologías participativas: prevención de riesgos laborales en la agroindustria de caña de azúcar [Participatory methods: prevention of occupational risks in the agricultural industry of sugarcane]. Heredia, Costa Rica: SALTRA Technical Series on Work & Health 5; 2007. Available from: <http://www.saltra.info/index.php?module=Pagesetter&func=viewpub&tid=11&pid=5> [cited 23 October 2009].
13. Chinchilla-Vargas E. Estudio del proceso de trabajo y operaciones. Su perfil de riesgos y exigencias laborales en el cultivo e industrialización de la caña de azúcar [Study of work processes and operations. An outline of risk and work demands in the cultivation and industrialization of sugarcane]. San José, Costa Rica: International Labor Organization (ILO). Proyecto: promoción de la seguridad y salud del trabajo agrícola en América Central [Project: promotion of safety and health in agricultural work in Central America]; 1998.
14. García-Trabanino R, Aguilar R, Reyes S, Ortiz M, Leiva M. Nefropatía terminal en pacientes de un hospital de referencia en El Salvador [Terminal nephropathy in patients in a reference hospital in El Salvador]. *Revista Panamericana de Salud Pública [Pan Am J Pub Health]* 2002; 12: 202–6. Available from: <http://www.scielosp.org/pdf/rpsp/v12n3/12875.pdf> [cited 23 October 2009].
15. García-Trabanino R, Dominguez J, Jansa J, Oliver A. Proteinuria e insuficiencia renal crónica en la costa de El Salvador [Proteinuria and chronic kidney disease on the coast of El Salvador]. *Nefrología* 2005; 25: 31–8. Available from: <http://www.revistanefrologia.com/mostrarfle.asp?ID=2144> [cited 23 October 2009].
16. Peraza S, Aragón-Benavides A, García-Trabanino R, Hogstedt C, Leiva R, Wesseling C. Prevalence of chronic kidney disease in five communities of El Salvador. Book of abstracts of the 20th international conference on epidemiology and occupational health and 10th international symposium on neurobehavioral methods and effects in occupational and environmental health; 8–13 June 2008; Heredia, Costa Rica; 2008, p. 164.
17. Flores-Reyna R, Jenkins Molieri J, Vega Manzano R, Chicas Labor A, Leiva Merino R, Calderón G, et al. Enfermedad renal terminal: Hallazgos preliminares de un reciente estudio en El Salvador [Terminal kidney disease: preliminary findings from a recent study in El Salvador]. San Salvador, El Salvador: OPS-Ministerio de Salud de El Salvador; 2003.
18. Callejas-Callejas L, Alonso-Medrano C, Mendoza B. Insuficiencia renal crónica: Una prioridad en salud pública en zonas de la costa del pacífico de Nicaragua, Mayo-Septiembre 2003 [Chronic kidney insufficiency: a priority for public health in coastal zones of the Pacific region of Nicaragua, May–September 2003] Managua, Nicaragua: US Centers for Disease Control and Prevention (CDC)-Ministerio de Salud Nicaragua [Ministry of Health Nicaragua]. (MINSAL-Nicaragua); 2003.
19. Cuadra S, Jakobsson K, Hogstedt C, Wesseling C. Chronic kidney disease: assessment of current knowledge and feasibility for regional research collaboration in Central America. SALTRA Technical Series on Work & Health 2, Heredia, Costa Rica; 2006. Available from: <http://www.saltra.info/index.php?>

- module=Pagesetter&func=viewpub&tid=11&pid=4 [cited 23 October 2009].
20. SICA. Impacts and adaptation to climate change and extreme events in Central America. San Jose, Costa Rica: University of Costa Rica Geophysical Research Center; 2006. Available from: <http://www.cifor.cgiar.org/trofcca/america/docs/CC%20adaptation%20in%20Central%20America%20-%20AIACC%202006.pdf> [cited 23 October 2009].
 21. Fonseca P. Cambio climático producirá sequías e inundaciones aquí [Climate change will produce droughts and floods here]. La Nación. 2007 August 14, 2007. Available from: http://www.nacion.com/ln_ee/2007/agosto/14/aldea1199145.html [cited 23 October 2009].
 22. Moreno M, Alfaro E. Variabilidad climática y producción de caña de azúcar en Costa Rica [Climate variability and the production of sugarcane in Costa Rica]. *Ambientico* 2007; 156: 15–8. Available from: <http://www.ambientico.una.ac.cr/165.pdf> [cited 23 October 2009].
 23. Charpentier F. Crisis alimentaria en Costa Rica y cómo salir de ella [The food crisis in Costa Rica and how to avoid it]. *Ambientico* 2008; 176: 10–11. Available from: <http://www.ambientico.una.ac.cr/176.pdf> [cited 23 October 2009].
 24. Estado de la nación en desarrollo humano sostenible [The state of the nation in sustainable human development]. San Jose, Costa Rica: Programa Estado de la Nación [Program The State of the Nation]; 2008.
 25. Alonso-Medrano A, Perea W. Insuficiencia Renal Crónica (ERC) en trabajadores de caña de azúcar, Chinandega, Nicaragua: Febrero–Marzo 2002 [Chronic kidney insufficiency in sugarcane workers, Chinandega, Nicaragua: February–March 2002]: US Centers for Disease Control and Prevention (CDC)-Ministerio de Salud Nicaragua [Ministry of Health Nicaragua] (MINSAL-Nicaragua); 2002.
 26. INTECO. Higiene y seguridad ocupacional: Exposición a ambientes con sobrecarga térmica [Hygiene and occupational health: exposure to environments with excess heat]. San Jose, Costa Rica: Instituto de Normas Técnicas de Costa Rica [Institute for Technical Norms in Costa Rica]; 2002.
 27. Retana JA, Solera M, Solano J, Alvarez H. Efecto de la variabilidad climática sobre la fluctuación poblacional de la rata cañera (*Sigmon hispidus*) en Cañas, Guanacaste. [Effect of climate variability on the population fluctuation of cane rat (*Sigmon hispidus*) in Cañas, Guanacaste] San José, Costa Rica: Boletín Meteorológico del Insituto Meteorológico Nacional. 1999; 22: 1–11. Available from: http://www.imn.ac.cr/publicaciones/estudios/Variab_clima_ratasCR.pdf [cited 23 October 2009].
 28. Reglamento general de seguridad e higiene del trabajo [General regulation of occupational safety and hygiene]. San Jose, Costa Rica: Ministerio de Trabajo y Seguridad Social [Ministry of Labor and Social Security].
 29. Ávila ML. La salud y el cambio climático [Health and climate change]. La Nación; 1970. Available from: http://www.nacion.com/ln_ee/2008/octubre/28/opinion1753073.html [cited 28 October 2008].
 30. UNDP. PNUD aprueba 450 millones para cambio climático en Costa Rica [UNDP approves 450 million for climate change in Costa Rica]. San Jose, Costa Rica: UNDP; 2007.
 31. UNDP. PNUD advierte que cambio climático ya golpea a habitantes de Costa Rica [UNDP announces that climate change has already affected inhabitants of Costa Rica. Available from: http://www.pnud.or.cr/index.php?option=com_content&view=article&id=242%3Apnud-advier-te-que-cambio-climco-ya-golpea-a-habitantes-de-costa-rica&catid=46%3Adesarrollo-humano&Itemid=1; 2007 [cited 2 December 2008].
 32. Boischio A, Sanchez A, Orosz Z, Charron D. Health and sustainable development: challenges and opportunities of ecosystem approaches in the prevention and control of dengue and Chagas disease. *Cad Saude Publica* 2009; 25: S149–54. Available from: <http://www.scielo.br/pdf/csp/v25s1/14.pdf> [cited 23 October 2009].

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The 'Hothaps' programme for assessing climate change impacts on occupational health and productivity: an invitation to carry out field studies

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The 'high occupational temperature health and productivity suppression' programme (Hothaps) is a multi-centre health research and prevention programme aimed at quantifying the extent to which working people are affected by, or adapt to, heat exposure while working, and how global heating during climate change may increase such effects. The programme will produce essential new evidence for local, national and global assessment of negative impacts of climate change that have largely been overlooked. It will also identify and evaluate preventive interventions in different social and economic settings.

Hothaps includes studies in any part of the world where hourly heat exposure exceeds physiological stress limits that may affect workers. This usually happens at temperatures above 25°C, depending on humidity, wind movement and heat radiation. Working people in low and middle-income tropical countries are particularly vulnerable, because many of them are involved in heavy physical work, either outdoors in strong sunlight or indoors without effective cooling. If high work intensity is maintained in workplaces with high heat exposure, serious health effects can occur, including heat stroke and death.

Depending on the type of occupation, the required work intensity, and the level of heat stress, working people have to slow down their work in order to reduce internal body heat production and the risk of heat stroke. Thus, unless preventive interventions are used to reduce the heat stress on workers, their individual health and productivity will be affected and economic output per work hour will be reduced. Heat also influences other daily physical activities, unrelated to work, in all age groups. Poorer people without access to household or workplace cooling devices are most likely to be affected.

The Hothaps programme includes a pilot study, heat monitoring of selected workplaces, qualitative studies of perceived heat impacts and preventative interventions, quantitative studies of impacts on health and productivity, and assessments of local impacts of climate change taking into account different applications of preventative interventions.

Fundraising for the global programme is in progress and has enabled local field studies to start in 2009. Local funding support is also of great value and is being sought by several interested scientific partners. The Hothaps team welcomes independent use of the study protocols, but would be grateful for information about any planned, ongoing or completed studies of this type. Coordinated implementation of the protocols in multi-centre studies is also welcome. Eventually, the results of the Hothaps field studies will be used in global assessments of climate change-induced heat exposure increase in workplaces and its impacts on occupational health and productivity. These results will also be of value for the next assessment by the Intergovernmental Panel on Climate Change (IPCC) in 2013.

Keywords: *climate change; heat; work; health; productivity*

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Background: key issues concerning climate change and health

Global climate change is making already hot seasons in hot parts of the world even hotter (1). Since 1980, many populated places (particularly cities) with hot climates (temperatures regularly above 35°C) have already recorded 1–2°C increases in average temperature (2). Additional 2–4°C increases can be expected in these places during this century according to the Intergovernmental Panel on Climate Change (IPCC) (1). In urban areas, with rapid development of buildings, roads and other major physical structures, the temperature increase is likely to proceed faster and to higher levels due to the ‘urban heat island effect’ (3, 4).

A range of health impacts related to climate and climate change have been identified (1, 5, 6). These include heat exhaustion, heat stroke, kidney disease, effects of additional air pollution, injuries and mental stress from extreme weather events, vector-borne diseases, diarrhoea and malnutrition. Many of these can create higher health risks among working people.

Heat exhaustion and heat stroke are of particular importance for occupational health. A working person creates heat internally in the body (particularly through muscular work), which adds to the heat stress in hot environments (7). If cooling methods in the workplace are insufficient, the only way for a working person to reduce heat stress is to take breaks or slow down their work. This reduces ‘work capacity’ (and daily work output) and economic productivity (8–10).

Even daily physical activities in the household can cause heat stress and may need to be reduced, resulting in less ability to carry out tasks on hot days. Travelling to and from work is another daily source of heat exposure for many people (7). Adults carrying out heavy labour are exposed (10), while children and the elderly are likely to be particularly sensitive to over-heating.

These non-fatal impacts on people exposed to heat are the focus of the ‘Hothaps (high occupational temperature health and productivity suppression)’ research and prevention programme introduced in this article.

Background: key issues concerning workplace heat, health and productivity

The Hothaps research programme contributes to the ongoing gathering of evidence on climate change and health. The idea for these studies emerged at an IPCC meeting in Delhi in December 1998 and was later presented at a conference (9). Numerous review reports on climate change and health (1, 5, 6, 11, 12) have highlighted the increasing human health risks due to heat waves, extreme weather conditions, increased air pollution, lack of access to safe water and food, spread of disease vectors and human displacement due to flooding,

drought or sea-level rise. Analysis of the non-fatal impacts of direct heat exposure on people, particularly when they work in physically demanding jobs, has so far been very limited. The IPCC report (1) mentions this issue only briefly.

The physiological basis for the direct effects of heat on humans and their work capacity is well understood (e.g. 7, 10, 13). The core body temperature of all human beings needs to be close to 37°C. Temperatures a few degrees higher than this can cause malfunction of body systems (7). The human body has physiological heat control mechanisms that can maintain the core body temperature even when the external air temperature is greater than 37°C. These mechanisms rely heavily on sweating, which becomes less efficient as the relative humidity of the air increases.

High heat exposure initially causes heat strain, which is subjectively perceived as unpleasant or dangerous, and this can progress into heat exhaustion (fatigue), heat stroke (7, 10) and even death (14, 15, 16). The main determinants of heat exhaustion are increases of body temperature and heart rate, as well as dehydration due to sweating without liquid replacement (7).

The climate variables used to define heat exposure and ‘microclimate’ are primarily temperature, humidity, direct heat radiation, diffuse heat radiation and wind speed at the work place (10). A combined measure of these is the Wet Bulb Globe Temperature (WBGT, recorded as °C), the indicator used in the International Standards Organization occupational exposure standard (17). Where WBGT measurements are not available, it is possible to model these approximately using data from weather stations (18; Lemke and Kjellstrom, to be published).

A natural reaction of a working person to heat is to reduce physical activity, which reduces the body’s internal heat production. This preventive reaction leads to reduced work capacity during exposure to heat (13, 16). Thus, the impact of increasing heat exposure on work capacity is a result of the natural preventive actions of working people to reduce heat stroke risk by slowing down work (‘autonomous adaptation to climate change’, 19). The degree of reduced work capacity in relation to heat exposure (as measured by the heat index WBGT) is shown schematically in Fig. 1, which also highlights the influence of clothing on the heat impact.

At WBGT values over 25°C, work capacity starts to decrease, and at WBGT values over 40°C it is very difficult to carry out any physical activity at all. Depending on air humidity, wind speed and heat radiation (e.g. from the sun), a WBGT range of 25–40°C occurs at air temperatures in the range 30–45°C. Such temperatures are common during the hot season in tropical and some sub-tropical countries. Many work situations are such that air conditioning is not feasible

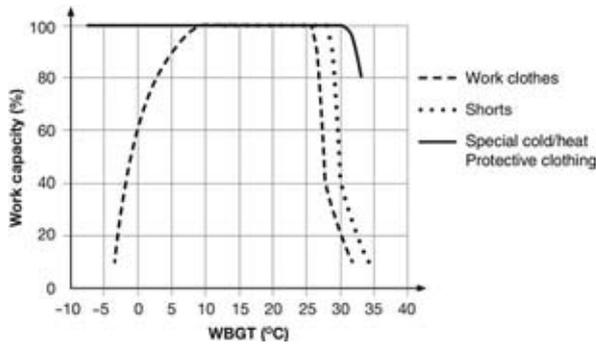


Fig. 1. Conceptual distribution of work capacity in relation to heat exposure (WBGT) and clothing based on ergonomic practice (7) and the international standard for work in hot environments (17). (Shorts or other very light clothing are not expected to be worn for work at WBGT below 15°C; common work clothes provide protection down to below 10°C, while they increase heat stress somewhat at WBGT above 25°C; at extreme hot and cold exposures special clothing can protect work capacity).

(work outdoors or in very large open indoor spaces), and fans are not sufficient when the heat exposure is very high (fans even increase the heat exposure when the air temperature exceeds skin temperature; 10).

The Hothaps team is developing a Population Heat Exposure Profile (PHEP), an analysis tool that presents the current heat situation and time trends for relevant climate and heat exposure variables for each month of the year (Kjellstrom and Lemke, to be published). Based on daily climate data from 13,000 weather stations around the world, acquired from websites at the US National Oceanic and Atmospheric Administration (NOAA) and National Aeronautics and Space Administration (NASA), the PHEPs present seasonal and intra-daily (hourly) variations in heat exposure. These can be used to estimate the associated health and productivity impacts. Fig. 2 demonstrates examples of data from Delhi, India, in August 1999 (August being one of the hottest months in Northern India).

Hothaps (high occupational temperature health and productivity suppression) programme overview (towards IPCC 2013)

Achievements to date: problem definition and preliminary global assessments

The Hothaps team has identified this problem associated with climate change and raised awareness of this effect of climate change via annual conference presentations, published journal articles (16), book chapters (20, 21) and monographs (2, 22), in order to alert interested people to the potential occupational health problems that increased heat exposure might cause. The physiological and ergonomic mechanisms behind the effects of heat on

humans have been reviewed (23, 24). A first global assessment of the impact of workplace heat on productivity has been carried out (25). Plans for future Hothaps work have been developed and fundraising for this research has started.

Current activities: methodological development, model design and study planning

Specific methods for improving heat exposure estimation (for instance the calculation of WBGT from weather station data) and the PHEP is under development and will be tested and promoted as a new tool for improved human exposure assessment. Modelling of heat exposure and the related effects based on existing physiological models is providing first stage estimates of how the increasing human heat exposure may impact on different population groups in different parts of the world. Preliminary global assessments of heat-related productivity loss or burden of disease are indicating how large the Hothaps effect may become with climate change, and its geographical distribution.

The different elements of health impact analysis (for instance burden of disease elements) are currently reviewed by the Hothaps team and new models for calculating impacts will be developed and tested. Besides WBGT, other relevant heat stress indices will also be reviewed. Human variability in the heat–health relationships will be quantified by using statistical distribution concepts and incorporated into statistical models for impact analysis. The need for preventive interventions in occupational health is also under study, including assessments of the prevention provided by international guidelines and standards. The plan and protocols for local Hothaps field studies in different parts of the world are being tested and funding for research is being sought. Four pilot studies are currently being carried out in India, Thailand and Costa Rica.

Future plans: field studies to assess local conditions in different countries, 2009–2011

In as many places as possible, Hothaps field studies will be carried out in as many places as possible, generating new evidence about health and productivity effects, local heat warning systems and preventive occupational health programmes. The results will be pooled to produce new quantitative exposure–response relationships for effects of excessive heat exposure on occupational health and productivity. Heat exposure descriptions based on local PHEPs will, together with climate change models, be developed into projected future occupational heat exposure scenarios and combined with exposure–response relationships to produce local or national climate change occupational health and productivity impact assessments.

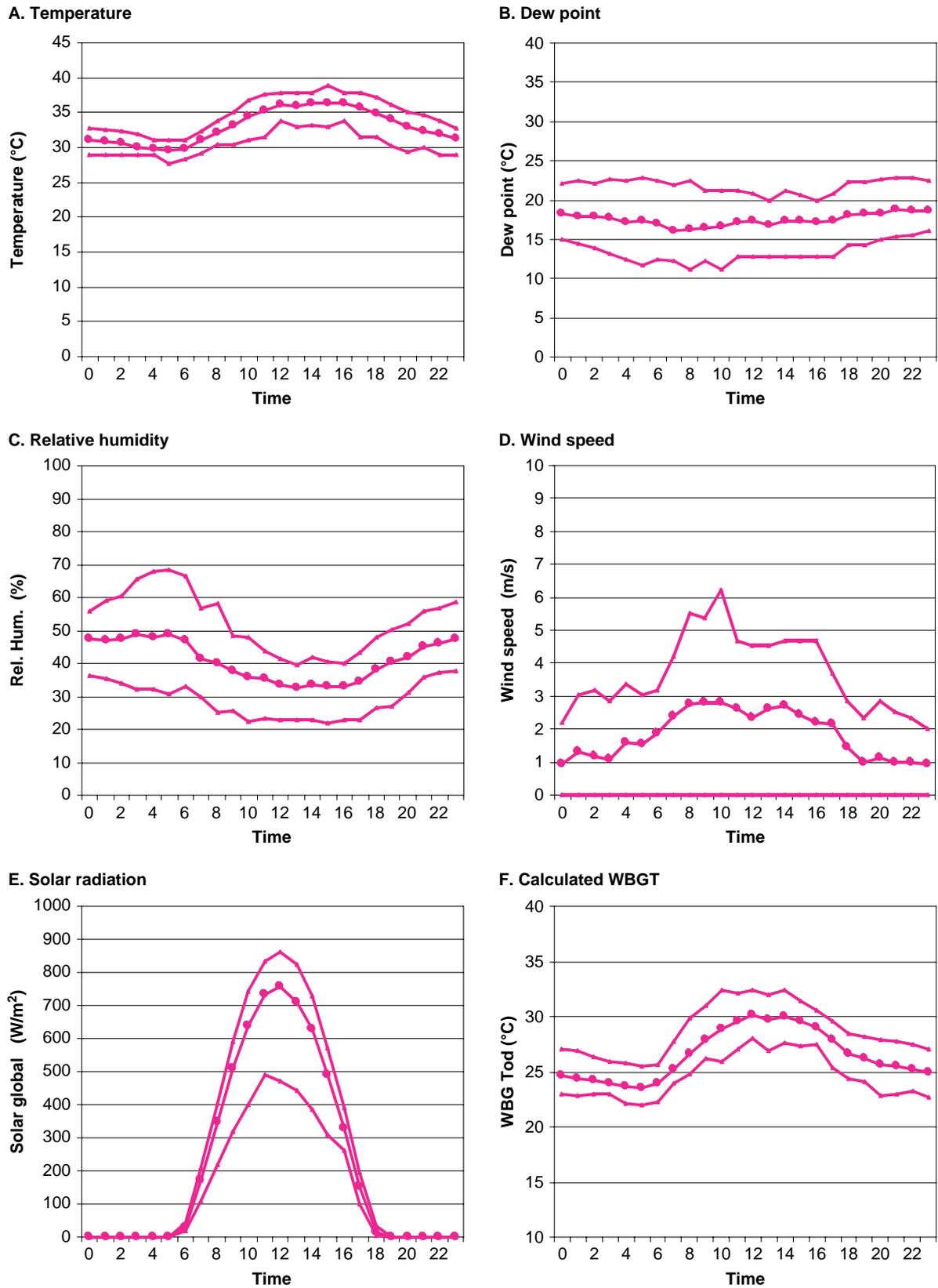


Fig. 2. Examples of hourly data in a Population Heat Exposure Profile for Delhi, August 1999. Includes shade temperature, dew point, relative humidity, wind speed, solar radiation (global) and WBGT outdoors (middle curve, averages; bottom and top curves, 5th and 95th percentiles of values for each hour on individual days during the month).

Future plans: detailed quantitative global assessments and preventive strategies, 2011–2012

Global occupational heat stress maps will be produced based on PHEPs and climate change predictions. The results of Hothaps field studies and heat exposure mapping will be used to make global health and productivity impact assessments. New preventive interventions ('adaptation' approaches) for occupational health will be devised. The results of the Hothaps global occupational health impact analysis will be delivered to the IPCC.

Aims and objectives for the Hothaps (high occupational temperature health and productivity suppression) field studies

Overall aims

The Hothaps international research programme aims at characterising and quantifying the effects of heat on occupational health and work capacity in different parts of the world, taking future climate change into account. It also aims at building knowledge about and finding effective preventive interventions (adaptation) against increasing occupational heat stress due to global climate change.

Specific objectives

- 1) Develop improved methods to quantify heat exposure relevant to human health and performance (work capacity), based on an established heat stress index and routine weather station data.
- 2) Identify heat exposure situations that create health risks and reduce the ability of people to carry out daily tasks or work, and identify the locations in the world where such effects are already common during the hot season.
- 3) Identify occupations in countries in different climate zones that are particularly vulnerable to heat-induced health risks and work capacity suppression, due to the work being outdoors during the hot season or indoors in places where heat reducing interventions are not in place.
- 4) Measure the impact of heat exposure on current work output and daily life activities, and, if possible, on relevant physiological and psychological indices as well as clinical disease.
- 5) Determine in selected countries how small shifts in temperature and heat exposure due to climate change or climate variability may impact on occupational health risks and work capacity in different occupations and assess how this compares to other 'dis-abilities' caused by disease or injury.
- 6) Model the empirical relationship between recorded weather variables and observed workplace heat

stress, to enable estimation of occupational heat stress in countries where field studies are yet to be carried out, and thereby to evaluate the global impact of the Hothaps effect.

- 7) Provide input into analyses of climate change impact on population health, worker productivity and economic conditions at local, national and global level.
- 8) Identify preventive measures ('adaptation') that can be taken to reduce the current and future climate impacts on occupational health and quantify their effectiveness in different countries and settings.

Overview of field study components

The Hothaps field study programme contains five components briefly described below (detailed protocols can be acquired from the Hothaps team). The proposed methods will need to be adapted to local conditions, while harmonisation of exposure and effect variables is important for combined analysis of the global Hothaps impact. Ideally, a Hothaps pilot study should be completed before carrying out the subsequent field studies. However, the components can be carried out separately or in combination. The field studies should ideally be carried out using participatory approaches with key target groups in order to ensure that the studies take their concerns into account.

Pilot study for Hothaps field studies

The pilot study will describe the general heat exposure situation for people in the country, make initial comments on how people cope with heat, and tentatively identify occupational and community groups that might already be affected by heat and therefore particularly vulnerable to increasing heat caused by future climate change. The first stage would be a literature review of any local information on heat problems at work, warning systems and preventive interventions (the review of published and unpublished information should ideally go back to the 1950s as in some countries studies were carried out this long ago). Existing workplace heat exposure data can be complemented by climate measurements in selected workplaces to compare with published data or weather station data.

A list of broad questions will be used to collect information from key informants about the heat exposure situation and impacts on health and work capacity in a specific location (a suggested questionnaire will be provided by the Hothaps team). The key informants could include occupational health practitioners, public health professionals, social scientists, community organisation representatives and well-informed people from different economic sectors.

The questionnaire study will identify common observations of the way people describe the impacts of heat on

their health, and will collect ‘anecdotal’ evidence about how work and daily life are affected by heat and what measures people take to reduce the impact of heat. These qualitative data can be used to generate hypotheses about specific factors that influence heat stress, to identify potential preventive interventions and to inform quantitative studies. The information about the types of heat protection interventions used in the study location can be combined with the literature review information to propose heat warning systems and other preventive interventions.

The pilot study report is an important first step towards the other components of the programme and related fundraising activity. The report should ideally include a proposed timetable and budget for each of the other programme components the research group is considering.

Basic local heat monitoring and occupational exposure assessment

The basic contributors to human heat exposure are air temperature and humidity, wind speed and heat radiation (which in outdoor locations is usually from solar radiation). Thousands of weather stations around the world measure at least the first three of these variables. However, spatial and temporal variations at local level are great, and to estimate human heat exposure accurately, local measurements at workplaces are needed.

The preferred heat stress index is WBGT. There are two components of the WBGT that are not measured routinely at weather stations, the natural wet bulb temperature and the globe temperature. If specialised equipment can be acquired, it is possible to carry out measurement of all the WBGT components. However, without such equipment, measurement of air temperature and humidity is a good starting point for calculating WBGT using the methods used in the PHEPs with assumptions about wind speed and heat radiation exposure.

Exploratory qualitative studies of occupational heat impacts and preventive approaches

In this component groups of heat exposed workers are asked to answer questions in a questionnaire similar to the one used in the pilot-study with key informants. A participatory approach can be used to adapt the questionnaire to local concerns, and data collection may be carried out via individual questionnaires or via focus groups. The results will help generate hypotheses that can be investigated with quantitative studies, and they will produce examples of the type of heat prevention interventions used by workers. The application of special heat warning systems, occupational heat exposure guidelines and organised re-hydration programmes can also be analysed.

Quantitative studies of occupational health impacts, work capacity and prevention

This study component measures the actual difference in health and work capacity effects of heat between seasons with different levels of heat exposure in the same groups of workers. Occupational groups to include are those that would be vulnerable to heat exposure as identified in the pilot-study or qualitative study. The results can be expressed as quantitative exposure–response relationships for different types of effects. The quantitative study provides a basis for estimating future trends in occupational health risks and daily work capacity and economic impacts when climate conditions change. This type of study can also quantify the effectiveness of different methods to reduce heat exposure and impacts (preventive interventions or adaptation methods).

Health and productivity impact assessment of local climate change on occupational health

With the exposure-response relationships identified with the quantitative studies and local heat exposure estimates, it will be possible to calculate the future impact not only on individuals, but also at population level based on the estimated climate change at a particular place. Additional input data required are the estimated future age distribution and occupation distribution of the population of interest, and the extent to which they are working outdoors and/or indoors, with or without air conditioning or other effective cooling technologies. The results are estimates of the current and future health risks, the lost work hours due to reduced work capacity (a ‘trade-off’ between health risk and work productivity), and potential economic impacts as a function of the degree of climate change at the locality.

Population-based estimates of climate change impacts on occupational health, worker productivity and the economy will enable estimates of how different cooling mechanisms can influence each of these variables, as well as the cost–benefit relationship of various preventive adaptation approaches. The Hothaps team will provide detailed guidance on how each component of the field studies should be carried out.

Conclusions and invitation

Climate change will pose a number of health threats. The effects on working people have not yet been fully analysed. The aim of the Hothaps programme is to assess the effects of a hotter climate on occupational health and productivity. The physiological and ergonomic basis of these potential effects of climate change is well known, but the impacts in different countries and occupational groups have as yet been only poorly quantified.

The threats to health, well-being and the economy from the ‘Hothaps effect’ strengthens the need for

mitigation of climate change and for preventive interventions (adaptation) through design of urban areas, housing and workplaces that reduce heat exposure, and through public and occupational health programmes that protect individuals at risk.

This article invites interested scientists around the world to join the research programme by carrying out field studies in their locality. Of particular interest are locations in tropical countries where large population groups are working in hot environments without adequate protection from heat.

Conflict of interest and funding

The authors have not received any funding or benefits from industry to conduct this study.

References

1. IPCC. Fourth assessment report. Geneva: Inter-governmental Panel on Climate Change, Cambridge: Cambridge University Press; 2007. Available from: www.ipcc.ch [cited 18 October 2008].
2. Kjellstrom T, Lemke B, Dear K. Climate change, urban heat exposure, and occupational health impacts. Report from National Centre for Epidemiology and Population Health, Australian National University (in press)..
3. Oke TR. City size and the urban heat island. *Atmos Environ* 2003; 7: 769–79.
4. USEPA. Heat island effect. Available from: <http://www.epa.gov/heatislands> [cited 23 September 2009].
5. McMichael A, Campbell-Lendrum D, Ebi K, Githeko A, Scheraga J, Woodward A. Climate change and human health: risks and responses. Geneva: World Health Organization; 2003.
6. Costello A, Abbas M, Allen A, Ball S, Bell S, Bellamy R, et al. (Lancet-University College London Institute for Global Health Commission). Managing the health effects of climate change. *The Lancet* 2009; 373: 1693–733.
7. Bridger RS. Introduction to ergonomics, 2nd ed. London: Taylor & Francis; 2003.
8. Axelsson O. Influence of heat exposure on productivity. *Work Environ Health* 1974; 11: 94–9.
9. Kjellstrom T. Climate change, heat exposure and labour productivity. *Epidemiology* 2000; 11: S144.
10. Parsons K. Human thermal environment. The effects of hot, moderate and cold temperatures on human health, comfort and performance, 2nd ed. New York: CRC Press; 2003.
11. McMichael AJ, Haines A, Sloof R, Kovats S, editors. Climate change and human health. Geneva: World Health Organization; 1996.
12. GHF. The anatomy of a silent crisis. Geneva: Global Humanitarian Forum; 2009.
13. Kjellstrom T, Holmer I, Lemke B. Workplace heat stress and health – an increasing challenge for low and middle income countries during climate change. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2047
14. Wyndham DH. A survey of the causal factors in heat stroke and their prevention in the gold mining industry. *J South African Inst Mining Metallurgy* 1965; 66: 125–55.
15. MMWR. Heat-related deaths among crop workers – United States, 1992–2006. *JAMA* 2008; 300: 1017–18 (also: *MMWR* 2008; 57: 649–653).
16. Kjellstrom T. Climate change, direct heat exposure, health and well-being in low and middle-income countries. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.1958
17. ISO. Hot environments – estimation of the heat stress on working man, based on the WBGT-index (wet bulb globe temperature). ISO standard 7243. Geneva: International Standards Organization; 1989.
18. Liljegren JC, Carhart RA, Lawday P, Tschopp S, Sharp R. Modeling the wet bulb globe temperature using standard meteorological measurements. *J Occup Environ Hyg* 2008; 5: 645–55.
19. Ebi KL, Smith JB, Burton I, editors. Integration of public health with adaptation to climate change. New York: Taylor & Francis; 2005.
20. Kjellstrom T, Hogstedt C. Global situation concerning work-related injuries and diseases. In: Elgstrand K, Pettersson I, eds. OSH for development. Stockholm: Royal Institute of Technology; 2009, pp. 741–61.
21. Kjellstrom T, Monge P. Global climate change and cities. In: Vlahov D, Ivey Bufford J. Urban health: a global perspective. New York: New York Academy of Medicine; 2009 (in press).
22. Kjellstrom T. Global climate change and health. A new theme for research in environmental medicine. Technical report. Stockholm: Institute of Environmental Medicine (IMM); 2009. Available at: <http://www.imm.ki.se/klimatrapport091020.pdf> [cited 25 October 2009].
23. Kjellstrom T, Holmer I. Climate change and occupational heat stress. Proceedings of the International Congress on Biometeorology, Tokyo, Japan, 22–26 September 2008.
24. Kjellstrom T, Lemke B, Holmer I. Climate change and occupational heat stress. Proceedings of the ICEE 2009 International Conference on Environmental Ergonomics, Boston, USA, 2–6 August 2009.
25. Kjellstrom T, Kovats S, Lloyd SJ, Holt T, Tol RSJ. The direct impact of climate change on regional labour productivity. *Arch Environ Occup Health* 2009 (in press).

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Climate change, its impact on human health in the Arctic and the public health response to threats of emerging infectious diseases

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The Arctic has warmed substantially over the last few decades. A recent study shows that temperatures over the last century increased almost three times faster in the Arctic than elsewhere in the Northern Hemisphere, reversing a 2000-year cooling trend, and outpacing current climate model predictions (1). This rapid warming trend is anticipated to continue into the next century with temperature increases exceeding those predicted in the rest of the Northern Hemisphere and will result in accelerated loss of land and sea ice, and an increased rate of sea level rise, with global consequences. These changes are already impacting local communities, which have observed profound changes in their local environments, and are leading to significant economic and cultural upheaval particularly for the indigenous peoples of the Arctic (2). Because climate change is more advanced in the Arctic than other regions of the world, the Arctic can play a vital role in preparing the world for what is to come.

Resident indigenous populations of the Arctic are uniquely vulnerable to climate change because of their close relationship with, and dependence on, the land, sea and natural resources for their well-being (3). Direct health threats from climate change include morbidity and mortality resulting from increasing extreme events (storms, floods, increased heat and cold) and an increased incidence of injury and mortality associated with unpredictable ice and storm conditions. Indirect effects include increased mental and social stress related to changes in environment and loss of traditional lifestyle; potential changes in bacterial and viral diseases; and decreased access to quality water sources (4, 5). Some regions are at risk for increasing illness due to failing sanitation infrastructure resulting from changes in permafrost and storm surges. Some regions will also experience changes in diet resulting from changes in subsistence species distribution and accessibility (6). This may result in a shift away from a

traditional subsistence diet to a more Western diet. While this shift may be beneficial, providing a more varied and reliable diet, the increased accessibility to processed foods, high in saturated fats and sugar, may result in an increase in the incidence of obesity, diabetes, cardiovascular disease and cancer (7, 8). Projected warming will affect the transport, distribution and behaviour of contaminants, further threatening the safety of the traditional food supply and potentially increasing human exposure (9). Higher temperatures at lower latitudes will increase volatilisation of contaminants resulting in increased delivery of contaminants to the Arctic. As precipitation increases over land, river flow will increase resulting in greater delivery of contaminants to the coasts and oceans.

These health impacts are taking place in the context of ongoing cultural and socioeconomic changes occurring in Arctic communities. Climate change represents another of many sources of stress on these northern societies and cultures as it affects the relationship between the people and the land and environment, which will further stress communities and individual psychosocial health. The potential impact on human health will differ from place to place depending on regional, and even local, differences in climate change as well as variations in health status and adaptive capacity of different populations (3).

Arctic populations have a long history of both endemic and epidemic infectious diseases (10). Despite advances in antimicrobial therapy, and availability of vaccines, high rates of invasive diseases caused by *Streptococcus pneumoniae*, *Haemophilus influenzae* and *Mycobacterium tuberculosis* persist. Sharp seasonal epidemics of viral respiratory infections also commonly occur. The over-use of antimicrobial drugs in some regions has led to the emergence of multi-resistant *S. pneumoniae*, *Helicobacter pylori* and methicillin-resistant *Staphylococcus aureus*.

The impact of climate on the incidence of these existing infectious disease challenges is unknown. However, it is

known that inadequate housing and sanitation are already important determinants of infectious disease transmission in many Arctic regions. Damage to the sanitation infrastructure by melting permafrost or flooding may therefore result in increased rates of hospitalisation among children for respiratory infections, as well as an increased rate of skin infections, and diarrhoeal diseases caused by bacterial, viral and parasitic pathogens (11).

Some infectious diseases are unique to the Arctic and lifestyles of the indigenous populations, and may increase in a warming Arctic. For example, many Arctic residents depend on subsistence hunting, fishing and gathering for food, and a predictable climate for food storage. Food storage methods often include above ground air-drying of fish and meat at ambient temperature, below ground cold storage on or near the permafrost, and fermentation. Changes in climate may prevent the drying of fish or meat, resulting in spoilage. Similarly, loss of the permafrost may result in spoilage of food stored below ground. Outbreaks of food-borne botulism occur sporadically in communities in the Arctic and are caused by ingestion of improperly prepared fermented traditional foods (12–14). Because germination of *Clostridium botulinum* spores and toxin production occurs at temperatures above 4°C, it is possible that warmer ambient temperatures in these regions associated with climate change may result in an increase the rates of food-borne botulism. Outbreaks of *Vibrio parahaemolyticus* gastroenteritis are commonly associated with sea water temperatures above 15°C. An outbreak of gastroenteritis caused by *V. parahaemolyticus* was documented among cruise ship passengers consuming raw oysters in Prince William Sound, Alaska (15) and provides direct evidence of an association between rising sea water temperature and onset of illness. In order to prevent further oyster farm outbreaks, a water temperature monitoring and shell-fish testing programme has been recommended. No additional outbreaks have been reported.

Warmer temperatures may allow an infected host animal species to survive winters in larger numbers, increase in population and expand their range of habitation and thus increase the opportunity to pass infections to humans. For example, the climate-related northern expansion of the boreal forest in Alaska and northern Canada has favoured the steady northward advance of the beaver, extending the range of *Giardia lamblia*, a parasitic infection of the beaver that can infect other mammals, including humans who use untreated surface water (2). Similarly, warmer temperatures in the Arctic and sub-Arctic regions could support the expansion of the geographical range and populations of foxes and voles, common carriers of *Echinococcus multilocularis* and the cause of alveolar echinococcus in humans (16, 17).

Climate change may influence the density and distribution of animal hosts and mosquito vectors which could

result in an increase in human illness or a shift in the geographical range of disease caused by these agents. West Nile virus entered the USA in 1999, and in subsequent years infected human, horse, mosquito and bird populations across the USA, and as far north as northern Manitoba (18). In the Russian Federation infected birds and humans have been detected as far north as the region of Novosibirsk (19). In Sweden the incidence of tick-borne encephalitis (TBE) has substantially increased since the mid-1980s. This increase corresponds to a trend of milder winters and an earlier onset of spring resulting in an increase in the tick population (*Ixodes ricinus*) that carries the virus responsible for TBE and other potential pathogens. Similarly in Northeastern Canada, climate change is projected to result in a northward shift in the range of *Ixodes scapularis*, a tick that carries *Borrelia burgdorferi* the etiologic agent of Lyme disease (20). Major increases in the prevalence of hantavirus and tick-borne infections in human populations in northern Europe and central Asia during the last decade have been associated with rodent population irruptions linked to a series of exceptionally warm winters (21, 22). In the unexpected outbreak of Puumala virus (a Hanta virus) in northern Sweden in 2007, the incidence was found to be 313/100,000 inhabitants in Västerbotten County. The increase in the rodent population, milder weather and less snow cover were probably contributing factors (21). Similar outbreaks have been noted in the Russian Federation (19). Whether or not disease in humans is a result of these climate-induced alterations of vector range depends on many other factors, such as land-use practices, human behaviour, human population density and adequacy of the public health response.

The public health response to these emerging microbial threats should be focused regionally include enhancing the public health capacity to monitor diseases with potentially large public health impacts, including respiratory diseases in children, skin infections and diarrhoeal diseases, particularly in communities being undermined by melting permafrost that is damaging water and sewage systems. Monitoring certain vector-borne diseases, such as West Nile virus, Lyme disease, TBE and Puumala virus should be priorities in areas at the margins of focal regions known to support both animal and insect vectors and where climate change may promote the geographic expansion of vectors.

Because Arctic populations are relatively small and widely dispersed, region-specific detection of significant trends in emerging climate-related infectious diseases may be delayed. This difficulty may be overcome by linking regional monitoring systems for the purposes of sharing standardised information on climate-sensitive infectious diseases of mutual concern over larger areas. Efforts should be made to harmonise notifiable disease registries, laboratory methods and clinical surveillance definitions

across administrative jurisdictions to allow comparable disease reporting and analysis. An example of such a network is the International Circumpolar Surveillance system for emerging infectious diseases. This network links hospital and public health laboratories together for the purposes of monitoring invasive bacterial diseases and tuberculosis in Arctic populations (10).

Public health capacity should be enhanced to promptly respond to infectious disease food and water-borne outbreaks (botulism or gastroenteritis caused by *G. lamblia*, *Cryptosporidium* or *V. parahemolyticus*). Public health research is needed to determine the baseline prevalence of potential climate-sensitive infectious diseases (e.g. West Nile virus, Puumala virus, *Borrelia spp.*, *Brucella spp.*, *Echinococcus spp.*, *Toxoplasma spp.*, and intestinal protozoa) in both human and animal hosts in regions where emergence may be expected. Such studies can be used to accumulate additional evidence of the effect of climate change or weather on infectious disease emergence, to guide early detection and public health intervention strategies, and to provide science-based support for public health actions on climate change.

Conflict of interest and funding

The authors have not received any funding or benefits from industry to conduct this study.

References

- Kaufman DS, Schneider DP, McKay NP, Ammann CM, Bradley RS, Briffa KR, et al. Recent warming reverses long-term arctic cooling. *Science* 2009; 325: 1236–9.
- ACIA. Arctic Council 2005. Arctic climate impact assessment scientific report. Cambridge: Cambridge University Press; 2005, pp. 863–960.
- Hess J, Malilay J, Parkinson AJ. Climate change: the importance of place and places of special risk. *Am J Prev Med* 2008; 35: 468–78.
- Parkinson AJ. Climate change and infectious diseases: the arctic environment. IOM Institute of Medicine. Global climate change and extreme weather events: understanding the contributions to infectious disease emergence. Washington, DC: The National Academies Press; 2008.
- Parkinson AJ, Berner J. Climate change and impacts on human health in the Arctic: an international workshop on emerging threats and the responses of arctic communities to climate change. *Int J Circumpolar Health* 2009; 68: 88–95.
- Vors SL, Boyce MS. Global decline in caribou and reindeer. *Global Change Biology* 2009. doi: 10.1111/j.1365-2486.2009.01974.x.
- Orr P, Lorencz B, Brown R, Kielly R, Holton D, Clugstone H, et al. An outbreak of diarrhea due to verotoxin-producing *E. coli* in the Canadian Northwest Territories. *Scand J Infect Dis* 1994; 26: 675–84.
- Bjerregaard P, Young KT, Dewailly E, Ebbesson SOE. Indigenous health in the Arctic: an overview of the circumpolar Inuit population. *Scand J Pub Health* 2004; 32: 390–5.
- Kraemer LD, Berner JE, Furgal CM. The potential impact of climate on human exposure to contaminants in the Arctic. *Int J Circumpolar Health* 2005; 64: 498–508.
- Parkinson AJ, Bruce M, Zultz T. International circumpolar surveillance, and Arctic network for surveillance of infectious diseases. *Emerg Infect Dis* 2008; 14: 18–24.
- Hennessey TW, Ritter T, Holman RC, Bruden DL, Yorita KL, Bulkow L, et al. The relationship between in-home water service and the risk of respiratory tract, skin, and gastrointestinal tract infections among rural Alaska Natives. *Amer J Public Health* 2008; 98: 2072–8.
- Sobel J, Tucker N, Sulka A, McMaughlin J, Maslanka S. Foodborne botulism in the United States, 1990–2000. *Emerg Infect Dis* 2004; 10: 1606–11.
- Proulx JF, Milor-Roy V, Austin J. Four outbreaks of Botulism in Ungava Bay Nunavik, Quebec. *Can Commun Dis Rep* 1997; 23: 30–2.
- Sørensen HC, Albøge K, Misfeldt JC. Botulism in Ammassalik. *Ugeskrift for Laeger* 1993; 115: 108–9.
- McLaughlin JB, Depoala A, Bopp CA, Martinek KA, Napolilli N, Allison C, et al. Emergence of *Vibrio parahaemolyticus* gastroenteritis associated with consumption of Alaskan oysters and its global implications. *New England J Med* 2005; 353: 1463–70.
- Rausch R. Cystic echinococcosis in the Arctic and sub-Arctic. *Parasitology* 2003; 127: S73–85.
- Castrodale LJ, Beller M, Wilson JF, Schantz PM, McManus DP, Zhang LH, et al. Two atypical cases of cystic echinococcosis (*Echinococcus granulosus*) in Alaska 1999. *Am J Trop Med Hyg* 2002; 66: 325–7.
- Parkinson AJ, Butler JC. Potential impact on climate change on infectious disease emergence in the Arctic. *Int J Circumpolar Health* 2005; 64: 478–86.
- Revich BA. Climate change alters human health in Russia. *Stud Russ Econ Dev* 2008; 19: 311–7.
- Ogden NH, Maarouf A, Barker IK, Bigras-Poulin M, Lindsay LR, Morshed MG, et al. Climate change and the potential for range expansion of the Lyme disease vector *Ixodes scapularis* in Canada. *Int J Parasitol* 2005; 36: 63–70.
- Pettersson L, Boman J, Juto P, Evander M, Ahlm C. Outbreak of Puumala virus infection, Sweden. *Emerg Infect Dis* 2008; 14: 808–10.
- Gray JS, Dautel H, Estrada-Peña A, Kahl O, Lindgren E. Effects of climate change on ticks and tick-borne diseases in Europe. *Interdiscip Perspect Infect Dis* 2009; 593232, Epub 2009 Jan 4. Available from: <http://www.hindawi.com/journals/ipid/2009/593232.html> [cited 19 April 2009].

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Effects of climate change on tularaemia disease activity in Sweden

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Tularaemia is a vector-borne infectious disease. A large majority of cases transmitted to humans by blood-feeding arthropods occur during the summer season and is linked to increased temperatures. Therefore, the effect of climate change is likely to have an effect on tularaemia transmission patterns in highly endemic areas of Sweden. In this report, we use simulated climate change scenario data and empirical data of temperatures critical to tularaemia transmission to forecast tularaemia outbreak activity. The five high-endemic counties: Dalarna, Gävleborg, Norrbotten, Värmland and Örebro represent only 14.6% of the total population of Sweden, but have recorded 40.1–81.1% of the number of annual human tularaemia in Sweden from 1997 until 2008. We project here earlier starts and a later termination of future tularaemia outbreaks for the time period 2010–2100. For five localised outbreak areas; Gagnef (Dalarna), Ljusdal (Gävleborg), Harads (Norrbotten), Karlstad (Värmland) and Örebro municipality (Örebro), the climate scenario suggests an approximately 2°C increase in monthly average summer temperatures leading to increases in outbreak durations ranging from 3.5 weeks (Harads) to 6.6 weeks (Karlstad) between 2010 and 2100. In contrast, an analysis of precipitation scenarios indicates fairly stable projected levels of precipitation during the summer months. Thus, there should not be an increased abundance of late summer mosquitoes that are believed to be main vectors for transmission to humans in these areas. In conclusion, the results indicate that the future climate changes will lead to an increased burden of tularaemia in high-endemic areas of Sweden during the coming decades.

Keywords: tularaemia; climate; epidemiology; models; statistical; temperature; rain; forecast; Sweden; *Francisella tularensis*

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Since the first description in Sweden in the 1930s (1), tularaemia has occurred predominantly in some northern and central areas of the country but, recently, the disease has extended southwards resulting in a considerable number of human cases in emerging areas. In 2003, for example, 700 individuals were diagnosed with tularaemia and half of them were affected in areas south of the river Dalälven cutting across central Sweden from the mountains in the west to its draining areas in the Botnian Sea in the east (2). The large majority of humans that contract tularaemia get infected in late summer and autumn (3, 4). Tularaemia is a zoonotic, often vector-borne disease occurring in a wide range of mammals and caused by the intracellular bacterium *Francisella tularensis* (5). In Europe, the etiological agent is *F. tularensis* subsp. *holarctica* (Type B) that appears to have a strong

association with water since many humans report to have contracted disease around lakes and rivers. The precise relationship between water and the persistence and spread of *F. tularensis* is, however, not well characterised, much due to the fact that the bacterium is difficult to culture from water samples (6, 7). Its presence in water and sediments has been proven through inoculation of samples into laboratory animals, from which culturable bacteria subsequently have been isolated.

Worldwide, a wide range of arthropods has been identified in the transmission of tularaemia to mammalian hosts, including ticks, tabanid flies (horseflies and deerflies), fleas and mosquitoes (8). Subspecies *holarctica* is believed to be heavily dependent on transmission by ticks, mites, tabanid flies and mosquitoes. In central Europe, blood-feeding mites and ticks are believed to be

most important vectors for rodent transmission of *F. tularensis* and to maintain epizootic tularaemia foci (9). In particular, *Dermacentor reticulatus* has been implicated to play a significant role in transmission of *F. tularensis* among small mammals in Europe but it does not feed on humans. Only *Ixodes ricinus* is capable of transmitting tularaemia to humans but overall, tick-borne human cases appear to be infrequent in central and northern Europe. As human tularaemia vectors, tabanid flies and mosquitoes appear to be essential in northern Europe including Sweden (4). In Northern Scandinavia, ticks are rare while tularaemia is sometimes common. According to clinical data, the predominant route of transmission to humans in Sweden is through mosquito bites. Some 10 mosquito species have been found to be naturally infected but the exact roles of different species for the spread of tularaemia in Sweden and other Scandinavian countries are not known.

In view of this background, it is apparent that much research is required before a complete understanding of the epidemiology and the risk factors for human tularaemia outbreaks can be obtained. Thorough field and experimental research on the ecology of *F. tularensis*, its transmission routes to humans, and biotic and abiotic prerequisites for tularaemia outbreaks will be invaluable to create models for disease prediction. Before robust field and experimental data exists, it is still possible to develop statistical projections for the purpose based on existing knowledge of correlations between climate parameters and human tularaemia cases. One such attempt using a large-scale climate approach has been performed. Using the North Atlantic Oscillation (NAO) index representing the dominant mode of winter climate variability in the whole North Atlantic region, it was concluded that a low NAO-index indicating cold winters, and low water flow in rivers during the following summer was associated with high numbers of human cases of tularaemia in Sweden two years later (10). Considering the scarcity of studies, additional work seems warranted. There is precedence from other parts of the world that the current wealth of climate data and models of future climate conditions can be used as a foundation to predict future transmission patterns. For example, a recent study generated models based on historical data on tularaemia and plague from the USA that were consistent in relation to known climate changes over the period. The models were then used to forecast likely shifts in patterns of transmission over the next 50 years (11).

The present study aims to utilise regional climate change scenario data for Sweden and regional data on past human tularaemia infections to create a model that forecast the magnitude of future human outbreaks.

Materials and methods

Data on human tularaemia

Tularaemia in humans is a disease that is mandatory to report. The figures used here were based on annual numbers of human tularaemia cases reported to County Medical Officers for Communicable Diseases in all regions of Sweden and are available at the website of the Swedish Institute for Infectious Disease Control (<http://www.smittskyddsinstitutet.se>). Demographic data for Sweden year 2008 was obtained from Statistics Sweden available at <http://www.ssd.scb.se>.

Climate change scenario data

A 140-year model run of regional climate simulations for Sweden performed at the Rossby Center, SMHI, Sweden, was used (12). Scenario data for the period 2000–2100 using the regional climate model RCA3 and the Intergovernmental Panel on Climate Change (IPCC) Special Report on Emissions Scenario B2 was downloaded at <http://www.smhi.se>. The resolution was given as 50 × 50 km squares. The data was arranged to regions by use of the WGS84 coordinates for the square centres. A square with its centre coordinate within a regional border was assigned to that region.

Empirical data for deciding temperatures suitable for tularaemia transmission to humans

Reported date of disease onset for 379 individuals 1981–2007 in Dalarna County was obtained from the local County Medical Office and used for prediction of time and temperature ranges suitable for tularaemia transmission in Sweden. Outbreaks with five or more cases were used to determine the date for first and last case of the epidemic, respectively. For these time points, the average temperatures (± 3 days around the day of onset of disease) were used to determine the temperature critical for the start and the termination of an epidemic.

Visualisation of trends in climate change scenario data

At the regional level, monthly average values were calculated for temperature and precipitation using the corresponding 50 × 50 km squares. The data was smoothed by a non-parametric method for estimating local regression surfaces, loess smoother, with the span-parameter set to 0.75 for each variable and county and visualised using in-house S-Plus scripts (13).

Outbreak duration calculated from scenario data

For each year, geographical region and local outbreak area, the first and last human tularaemia case was predicted using scenario data as follows: The temperature profile of an area was obtained by linear interpolation of

monthly smoothed scenario mean temperatures from May to October. Time points for first and last case of an epidemic was predicted with the intersections of the temperature profile and the empirically determined temperatures critical for start and end of a human tularaemia epidemic. The duration of an epidemic was obtained as the estimated interval.

Results

The five investigated counties reported 40.1–81.1% of the total number of annual human tularaemia in Sweden 1997–2008 (Fig. 1A). The counties Dalarna, Gävleborg, Norrbotten, Värmland and Örebro contain 14.61% of the total population of Sweden (1,352,558/9,256,347 inhabitants) with annual tularaemia incidences per 100,000 inhabitants of 0.70–78.05 (Dalarna), 0.70–66.04 (Gävleborg), 0.38–20.82 (Norrbotten), 0–24.86 (Värmland) and 0–54.78 (Örebro) during the same time period. The geographical distribution of tularaemia within counties was uneven with a large fraction of disease reports from repeated outbreaks in more restricted geographical areas; namely, Gagnef and adjacent villages (Dalarna), Ljusdal (Gävleborg), Harads (Norrbotten), Karlstad (Värmland) and Örebro municipality (Örebro) (Fig. 1B).

An analysis of summer temperatures was performed under the assumption that late summer epidemics of tularaemia in Sweden require temperatures critical for replication of the infectious agent and dispersal by blood-feeding arthropods. Temperature scenarios projected for the five tularaemia hotspot areas showed an increase in average monthly summer temperatures during the period 2010–2100 (Fig. 2). The analysis suggests that a critical

time point for the start of a summer epidemic will occur earlier in the future. Simultaneously, a temperature-dependent termination of a summer epidemic will be displaced to a later time point. This overall scenario is similarly projected for all five endemic areas. The scenario suggests an approximately 2°C increase in monthly average summer temperatures during the years 2010–2100. Then, an analysis of projected summer precipitation was performed under the assumption that vector abundance in late summer (mainly flood mosquitoes) will be dependent on local spring and summer rains. The precipitation analysis showed a fairly stable projected level of precipitation during the summer months (Fig. 3). A slight increase was projected for late October, an effect that will be much more pronounced during winter months according to the used climate scenario (data not shown). Projections of temperature and precipitation changes were also performed at the regional scale for Dalarna, Gävleborg, Norrbotten, Värmland and Örebro, and very similar trends were obtained at this larger geographical scale (data not shown).

A projected temperature-dependent increase in duration of human tularaemia epidemics for five tularaemia high-endemic areas is shown in Fig. 4. An analysis of the projected displacement in time of temperatures critical to the start and termination of human tularaemia outbreaks suggests that the effects may be pronounced. The projection indicates increases in outbreak durations as follows: 3.5 weeks (for Harads), 4.9 (Örebro), 5.5 (Ljusdal), 6.0 (Gagnef) and 6.6 (Karlstad) during the time period 2010–2100.

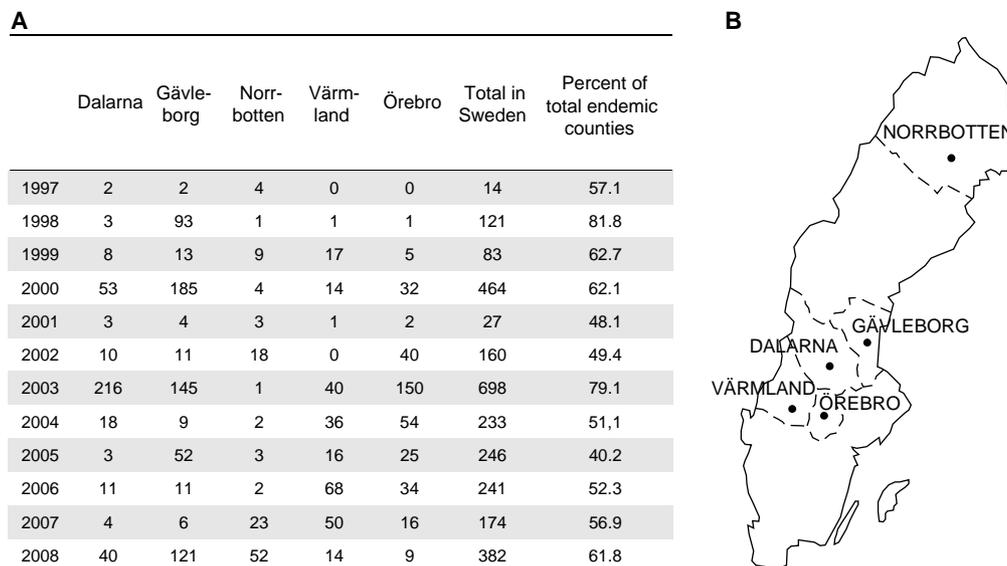


Fig. 1. Number of human tularaemia infections in Sweden 1997–2008. Panel A shows the number of individuals for each of five high-endemic counties. Panel B shows the geographic locations of the five counties and the five black dots indicate localised outbreak areas within the respective counties. The indicated areas are the following: Harads (Norrbotten), Ljusdal (Gävleborg), Gagnef (Dalarna), Karlstad (Värmland) and Örebro municipality (Örebro).

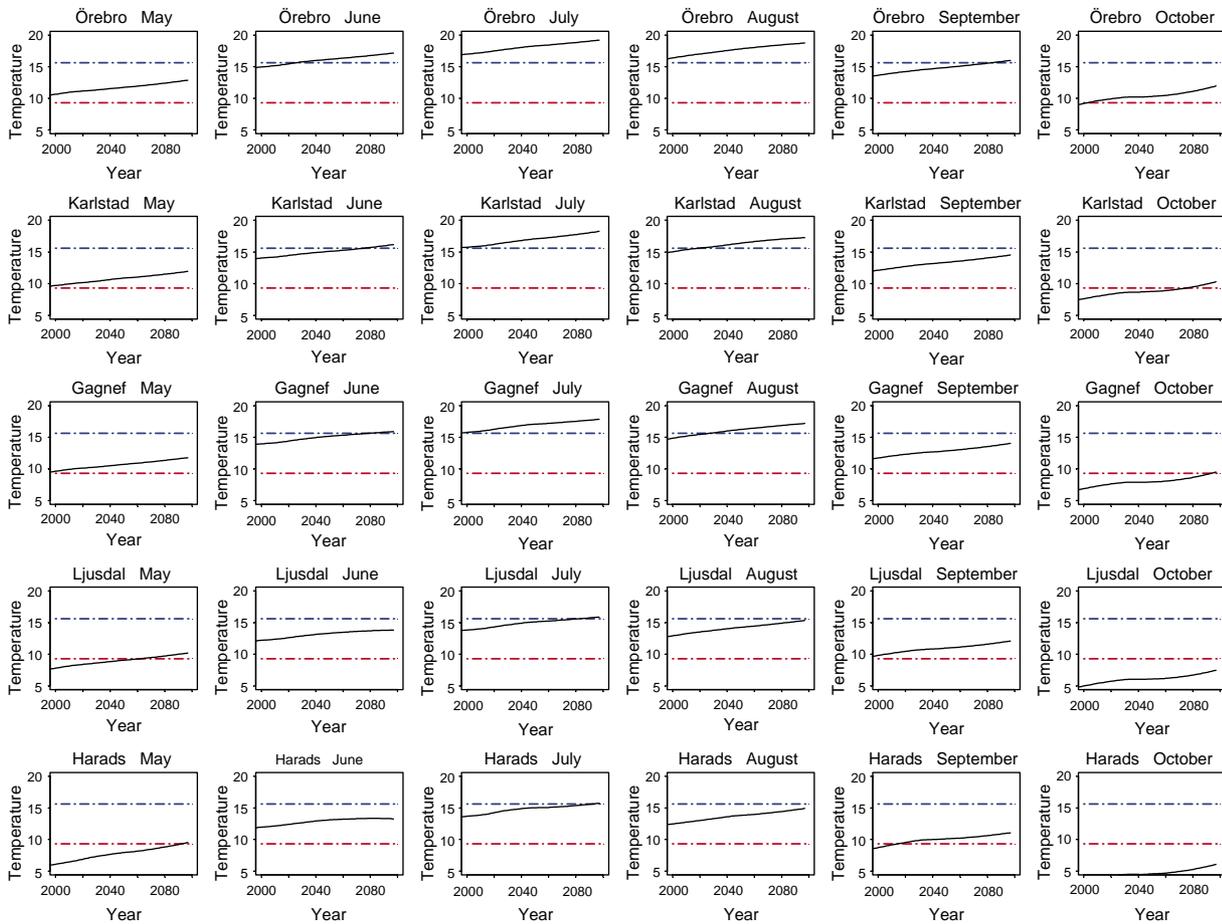


Fig. 2. Projected monthly average temperatures (°C) for the period 2000–2100. The projections (black lines) for the localised outbreak areas Örebro, Karlstad, Gagnef, Ljusdal and Harads are based on the IPCC climate scenario B2. The permissive thresholds for transmission, for the start of epidemic (blue) and termination of epidemic (red), are estimated from empirical data from Dalarna.

Discussion

Tularaemia is a disease which occurs only in the Northern hemisphere and in most countries presenting as isolated cases. However, in a few countries there exist regions where tularaemia has been endemic for many decades, most notably in Sweden, Finland and Russia (14). In Sweden, it is well known that certain regions have been affected since the 1930's and with such high incidences that has become a public health problem. The most notable examples in this regard are the county of Ljusdal along the river Ljusnan in Gävleborg and a few counties and villages, e.g. Gagnef, along the river Västerdalälven in Dalarna. The existing data on tularaemia cases in Sweden, even when going back many decades, demonstrate that an absolute majority have occurred during a narrow span of the year, commencing in late July and terminating in mid-September, sometimes during an even more narrow time span (15). Thus, the seasonality unequivocally demonstrates that a number of climate factors must be intimately linked to the outbreaks. The most obvious factor is temperature, since the seasonal changes in Sweden are very marked and the periods of the

outbreaks are characterised by much higher temperatures than those during the remainder of the year. Although our knowledge is incomplete as to why there is such strong association with temperature, there are many temperature-affected parameters that are intimately linked to the transmission, such as precipitation, water temperatures and abundance of blood-feeding arthropods, e.g. mosquitoes, which may transfer disease to humans.

In view of the predicted highly significant temperature increases by all existing climate models, we therefore postulated that these increases will coincide with extended annual time spans for tularaemia outbreaks. To this end, we simulated future changes in temperature and precipitation for today's endemic areas, Karlstad, Örebro, Gagnef, Ljusdal and Harads. Our analysis demonstrates that there will be much longer time spans with temperatures allowing tularaemia outbreaks in the coming decades (Fig. 4). The model predicted that the extension to some extent will be dependent on the geographical location. The most pronounced increases will be in the areas of Karlstad and Gagnef followed by Ljusdal (Fig. 1B and 4). Measured as a

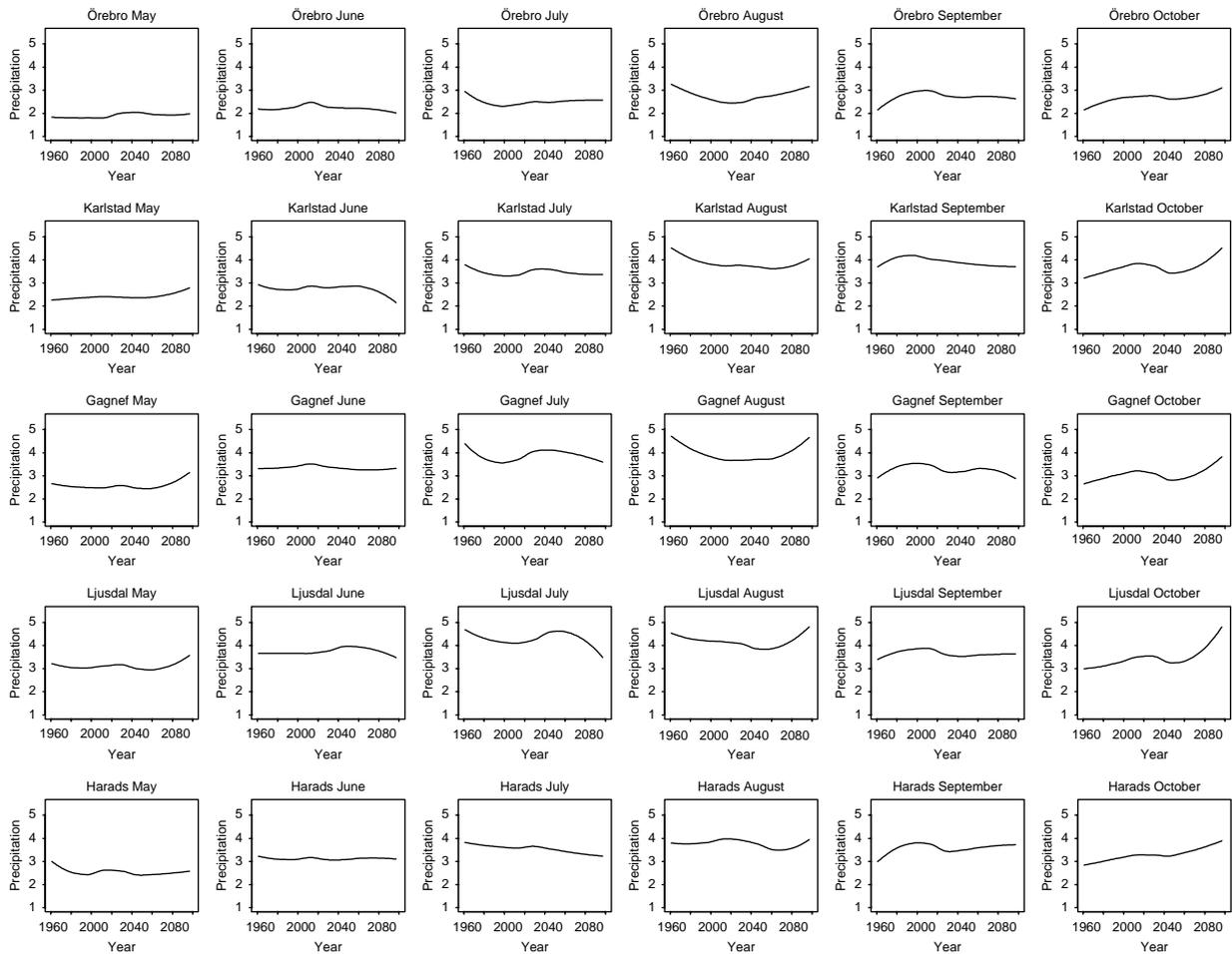


Fig. 3. Projected monthly average precipitation (mm/day) for the period 2000–2100. The projections (black lines) for the localised outbreak areas Örebro, Karlstad, Gagnef, Ljusdal and Harads are based on the IPCC climate scenario B2.

relative increase compared with current permissive transmission periods, the projected increase is largest for the area of Ljusdal. Our analyses suggest a two-fold increase of the period suitable for tularaemia transmission in Ljusdal with a following increase of the disease burden. In this study, we chose to use Rossby centre climate simulation data (12) which are based on the climate change scenario B2 prepared by the IPCC (16). The decision to use the B2 scenario meant that our projections are conservative as it assumes lower CO₂ concentrations, a smaller human population, lower energy consumption and less change in land use as compared with another frequently used scenario, named A2. The use of the less conservative scenario A2 for this study would have produced more pronounced climate change effects including greater and more rapid temperature increases.

In contrast to previous studies using the NAO-index which models very large geographical scale climate changes (10, 17), we selected to use a regional simulation model with a resolution of only 50 × 50 km. A smaller geographical scale approach to climate change seems

warranted in attempts to forecast tularaemia outbreaks since the disease is known to be highly localised. Both past (18) and more recent research have suggested that tularaemia is included among the diseases that display a pronounced ‘natural nidality’ (nidus means nest, home or habitat). Such nidi could be as large as a landscape zone, such as the flat land adjacent to a river, or as small as a single rodent burrow (‘a microfocus’). Permanent foci (‘elementary foci’) are present where there is an environmental reservoir of an infectious agent. The maintenance of several vector-borne infections, such as plague and tick-borne encephalitis appear to be best explained by natural nidality (19). In a recent field survey on Martha’s Vineyard, Massachusetts, ticks infected by *F. tularensis* were found in a natural microfocus with a diameter of only a few hundred metres where the agent of tularaemia stably persisted for a period of four years (20). Similarly, previous studies of human infections in Ljusdal (Gävleborg) and in villages along Västerdalälven (Dalarna) in Sweden have shown that the same genotype of *F. tularensis* overwinters between outbreaks within geographical scales of 50 km

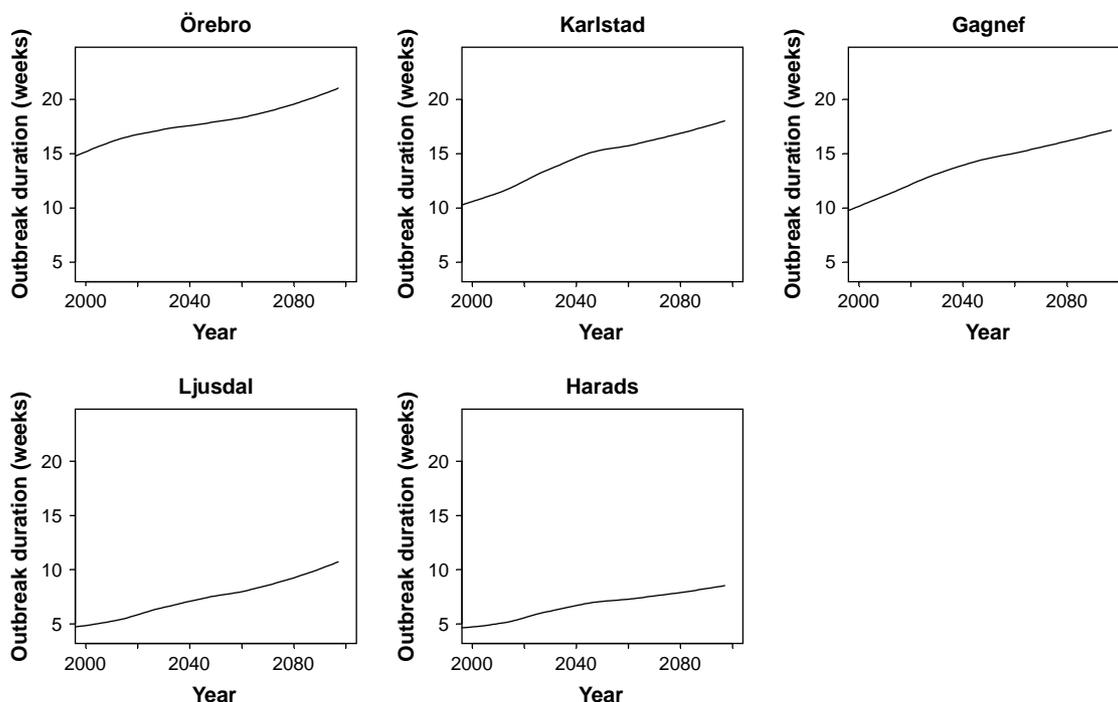


Fig. 4. Projected outbreak duration (in weeks) for the period 2000–2100. The projections for the localised outbreak areas Örebro, Karlstad, Gagnef, Ljusdal and Harads are based on the IPCC climate scenario B2.

and cause infections in humans for a time period of more than 17 years (21, 22). Collectively, these results strongly suggest that local climate parameters in tularaemia high-endemic areas should be better for forecasting outbreak activity than global scale climate parameters.

The analysis indicated only marginal changes in the precipitation, albeit changes will occur during the middle of the modelled period (Fig. 3). In an ongoing eco-epidemiological project, we have postulated that *F. tularensis* has an enzootic life cycle in Sweden. Its natural habitat is predicted to be wetlands and natural waters, where the organism constitutes a natural part of the microbial food web in interplay with protozoa and biofilms. Mosquitoes would become infected already during their larval stages in water and thus constitute the link between the aquatic and the terrestrial environments. In our hypothesis, rodents and lagomorphs are dead-end hosts for *F. tularensis* and rarely disseminate tularaemia to other animals and to humans and only by direct contact. A change of environmental factors, such as an increased abundance of nutrient-rich wetlands in close proximity to populated areas, is predicted to be an important factor for the incidence and magnitude of tularaemia outbreaks. Thus, if the hypothesis is true, then precipitation during the summer months will be an important factor affecting the occurrence of late summer mosquitoes and thereby the transmission of tularaemia. Our current findings indicated, however, that changes in precipitation in the tularaemia hotspot areas will be subtle and therefore

should not be decisive for the future outbreaks of tularaemia. Still, this prediction has a number of caveats. First, the role of precipitation for mosquito abundance may be complex, so projections based on overall precipitation may be too simplistic. Additional parameters probably influence the mosquito abundance considerably, e.g. forerunner wetness, soil type, and rates of evaporation and transpiration. Moreover, excessive rainfall can decimate some mosquito populations by flushing larval habitats or antecedent drought may reduce competitor species of mosquito larva leading to elevated rates of mosquito production (23). The proportion of mosquitoes that are infectious may also be variable and reflect the distance from their breeding habitats and the age structures of mosquito populations (24). Thus, a detailed and comprehensive modelling of mosquito populations in the future will require more sophisticated models. Finally, the exact relationship between the occurrence of specific mosquito species and transmission of tularaemia needs to be clarified.

Apart from climate-driven changes in ecology, like replication habitats for *F. tularensis* and blood-feeding disease vectors, it will also be important to determine how humans adapt to a warmer climate. These issues should be addressed in future research on forecasting the human burden of vector-borne infectious diseases including tularaemia. It is likely that human responses to climate change, e.g. changes in residential patterns in proximity to active disease transmission areas, in the period of time spent

outdoors versus indoors and in the extent of restoration of wetlands and the future use of irrigation, affect the burden of vector-borne diseases as much as ecological changes.

Considering the increasing temperatures during the coming decades, inevitable changes in the flora and fauna will result and most likely this will affect the geographical distribution of tularaemia. Currently, we are lacking the detailed information required to identify the denominators of hotspot regions. Since these regions encounter very high incidences of tularaemia, it may be warranted to implement local surveillance systems and preventive health measures such as vaccination of the populations. Therefore, the development of models to precisely identify regions at risk to become tularaemia hotspots will be of high priority. The current model is a first step in the development of the required, more sophisticated future models. Even the current simplistic model unambiguously indicates that the future climate changes will lead to an increased burden of tularaemia in Sweden during the coming decades and in view of this, additional research will be required to more specifically understand the prerequisites of tularaemia outbreaks, enabling the development of sophisticated models that can forecast risks of local outbreaks so appropriate preventive actions can be undertaken.

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References

- Olin G. Occurrence and mode of transmission of tularemia in Sweden. *Pathol Microbiol Scand* 1942; 19: 220–47.
- Payne L, Arneborn M, Tegnell A, Giesecke J. Endemic tularemia, Sweden, 2003. *Emerg Infect Dis* 2005; 11: 1440–2.
- Christenson B. An outbreak of tularemia in the northern part of central Sweden. *Scand J Infect Dis* 1984; 16: 285–90.
- Eliasson H, Lindbäck J, Nuorti JP, Arneborn M, Giesecke J, Tegnell A. The 2000 tularemia outbreak: a case-control study of risk factors in disease-endemic and emergent areas, Sweden. *Emerg Infect Dis* 2002; 8: 956–60.
- Sjöstedt AB. Family XVII. *Francisellaceae*, Genus I. *Francisella*. 2 ed. In: Brenner DJ, Krieg NR, Staley JT, Garrity GM, eds. *Bergey's manual of systematic bacteriology*, Vol. 2. New York: Springer; 2005, pp. 663–67.
- Parker RR. Contamination of natural waters and mud with *Pasteurella tularensis*. Washington: U.S. Government Printing Office; 1951.
- Petersen JM, Carlson J, Yockey B, Pillai S, Kuske C, Garbalena G, et al. Direct isolation of *Francisella* spp. from environmental samples. *Lett Appl Microbiol* 2009; 48: 663–7.
- Hopla CE, Hopla AK. Tularemia. In: Beran GW, ed. *Handbook of zoonoses*, 2nd ed. Boca Raton, FL: CRC Press; 1994 p. 113–26.
- Keim P, Johansson A, Wagner DM. Molecular epidemiology, evolution, and ecology of *Francisella*. *Ann N Y Acad Sci* 2007; 1105: 30–66.
- Palo T, Ahlm C, Tärnvik A. Climate variability reveals complex events for tularemia dynamics in man and mammals. *Ecol Soc* 2005; 10: 22–9.
- Nakazawa Y, Williams R, Peterson AT, Mead P, Staples E, Gage KL. Climate change effects on plague and tularemia in the United States. *Vector Borne Zoonotic Dis* 2007; 7: 529–40.
- Kjellström E, Barring L, Gollvik S, Hansson U, Jones C, Samuelsson P, et al. A 140-year simulation of European climate with the new version of the Rossby Centre regional atmospheric climate model (RCA3). Norrköping, Sweden: SMHI2005 Contract No.: 108.
- Cleveland WS, Devlin SJ. Locally-weighted fitting: an approach to fitting analysis by local fitting. *JASA* 1988; 83: 596–610.
- Sjöstedt A. Tularemia: history, epidemiology, pathogen physiology, and clinical manifestations. *Ann N Y Acad Sci* 2007; 1105: 1–29.
- Tärnvik A, Sandström G, Sjöstedt A. Epidemiological analysis of tularemia in Sweden 1931–1993. *FEMS Immunol Med Microbiol* 1996; 13: 201–4.
- Nakicenovic N, Swart R, editors. Special report on emissions scenarios. A special report of Working Group III of the Intergovernmental Panel on Climate Change. Cambridge: Cambridge University Press; 2000.
- Hubalek Z. North Atlantic weather oscillation and human infectious diseases in the Czech Republic, 1951–2003. *Eur J Epidemiol* 2005; 20: 263–70.
- Pavlovsky EN. Natural nidity of transmissible diseases [Translation from the Russian edition, by Plous FK, Jr. English translation edited by Levine ND, editor]. Urbana, IL: University of Illinois Press; 1966.
- Ostfeld RS, Glass GE, Keasing F. Spatial epidemiology: an emerging (or re-emerging) discipline. *Trends Ecol Evol* 2005; 20: 328–36.
- Goethert HK, Telford SR, 3rd. Nonrandom distribution of vector ticks (*Dermacentor variabilis*) infected by *Francisella tularensis*. *PLoS Pathog* 2009; 5: e1000319.
- Johansson A, Farlow J, Larsson P, Dukerich M, Chambers E, Byström M, et al. Worldwide genetic relationships among *Francisella tularensis* isolates determined by multiple-locus variable-number tandem repeat analysis. *J Bacteriol* 2004; 186: 5808–18.
- Johansson A, Göransson I, Larsson P, Sjöstedt A. Extensive allelic variation among *Francisella tularensis* strains in a short-sequence tandem repeat region. *J Clin Microbiol* 2001; 39: 3140–6.
- Chase JM, Knight TM. Drought-induced mosquito outbreaks in wetlands. *Ecol Lett* 2003; 6: 1017–24.
- Smith DL, Dushoff J, McKenzie FE. The risk of a mosquito-borne infection in a heterogeneous environment. *PLoS Biol* 2004; 2: e368.

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Milder winters in northern Scandinavia may contribute to larger outbreaks of haemorrhagic fever virus

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The spread of zoonotic infectious diseases may increase due to climate factors such as temperature, humidity and precipitation. This is also true for hantaviruses, which are globally spread haemorrhagic fever viruses carried by rodents. Hantaviruses are frequently transmitted to humans all over the world and regarded as emerging viral diseases. Climate variations affect the rodent reservoir populations and rodent population peaks coincide with increased number of human cases of hantavirus infections. In northern Sweden, a form of haemorrhagic fever called nephropathia epidemica (NE), caused by the Puumala hantavirus (PUUV) is endemic and during 2006–2007 an unexpected, sudden and large outbreak of NE occurred in this region. The incidence was 313 cases/100,000 inhabitants in the most endemic areas, and from January through March 2007 the outbreak had a dramatic and sudden start with 474 cases in the endemic region alone. The PUUV rodent reservoir is bank voles and immediately before and during the peak of disease outbreak the affected regions experienced extreme climate conditions with a record-breaking warm winter, registering temperatures 6–9°C above normal. No protective snow cover was present before the outbreak and more bank voles than normal came in contact with humans inside or in close to human dwellings. These extreme climate conditions most probably affected the rodent reservoir and are important factors for the severity of the outbreak.

Keywords: *hantavirus; climate; bank vole; Sweden*

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Viewpoint

Climate change may affect the spread of zoonotic infectious diseases in several ways. The geographic distribution and thereby the population at risk as well as the transmission of these diseases may increase due to climate factors such as temperature, humidity and precipitation (1). The group of zoonotic infectious diseases includes large and increasing number of bacterial, viral and parasitic infections, each with its specific characteristics, reservoir host and mode of transmission (2–6). Several of these infections are recognised as emerging diseases and pose a serious threat to human public health (7). These diseases are most commonly transmitted to humans either directly from animals (e.g. rodents) (4) or by vectors (e.g. mosquitoes and ticks) (2, 3, 8).

Hantaviruses are globally spread haemorrhagic fever viruses carried by various rodents (9). These viruses are frequently transmitted to humans globally and are recognised as emerging viral diseases. Hantaviruses can

cause two febrile illnesses in humans: haemorrhagic fever with renal syndrome (HFRS) in Asia and Europe and hantavirus cardiopulmonary syndrome (HCPS) in the Americas, depending on the species of hantavirus (9). HFRS accounts for more than 200,000 cases and thousands of deaths annually, whereas HCPS is much less frequent although more severe with a mortality rate above 40% (9). Severe forms of HFRS are prevalent in China and Korea (Haantan virus) and the Balkans (Dobrava virus) and a milder form, nephropathia epidemica (NE), is most prevalent in Russia, Finland, Sweden, Germany, France, Belgium and Norway. NE is caused by Puumala virus (PUUV) and is transmitted to humans via inhalation of infectious aerosols containing rodent saliva, urine and/or faeces from bank voles (*Myodes glareolus*) (10).

The bank vole is one of the most abundant mammal species in Europe. Variations in availability of plants and berries due to climate variations affect the vole populations, and in Central Europe these abundance peaks are often related to high tree seed production, which is

supposedly triggered by specific weather conditions (11, 12). In northern Fennoscandia, the bank vole population shows pronounced seasonal and multi-annual fluctuations in population density (13). The cyclic activity of the population dynamics has been explained by variations in predator populations and availability of food (14). The voles serve as prey for several predators, making them an important part in the ecological web in Fennoscandia (15). The bank vole population peaks coincide with increased number of human cases of hantavirus infections (16). In contrast to humans, rodent reservoirs become persistently infected showing no signs of disease or clinical pathology. In humans, after exposure to PUUV there is a 1–5 week incubation period before the NE disease symptoms appear. The disease is characterised by acute onset of high fever, headache, backache, myalgia and abdominal pain (17). Renal impairment is common and there is increasing evidence for a correlation between previous PUUV infection and later chronic renal affection and hypertension. One-third of the patients in Sweden have haemorrhagic symptoms and severe bleedings may occur. Some patients with more severe forms of HFRS (5–10%) die from hypotensive shock or bleeding.

During the last days of December 2006 and the first months of 2007, northern Sweden experienced a sudden and large outbreak of NE with high numbers of NE patients that resulted in a considerable burden on public health services (18). According to the Swedish Communicable Disease Act, NE must be reported and records show that the outbreak peaked during the first three months of 2007 (Fig. 1). In Sweden during 2007 and during 2008, a record-breaking 2,195 NE cases were recorded and more NE cases were diagnosed although not in the similar frequencies as in early 2007. The incidence in one of the most endemic areas – Västerbotten County for 2007 – was 313 recognised cases/100,000

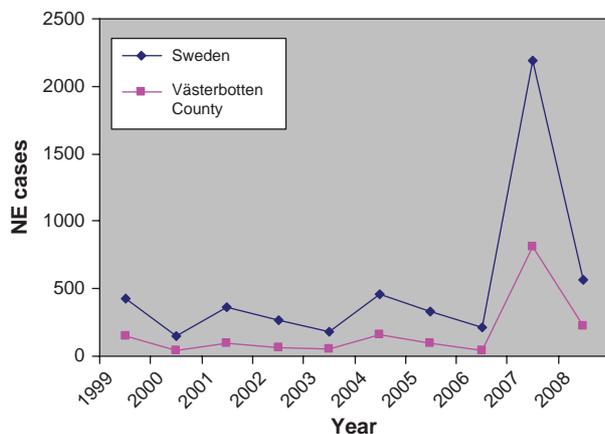


Fig. 1. Annual incidence of human hantavirus infection in Sweden and Västerbotten County from 1999 through 2008.

inhabitants. In Sweden during the peak (January–March 2007), 972 cases were recorded, 474 cases in Västerbotten County alone. The NE patients showed all the classical HFRS symptoms and displayed the whole spectra from mild to severe disease requiring hospitalisation and occasionally intensive care. Of the diagnosed patients, 30% were hospitalised and three known fatalities (0.24% case fatality) were recorded in the two most northern counties in Sweden during 2007. Previous NE peaks in 1999 (73/100,000), 2002 (38/100,000) and 2005 (61/100,000) had much lower incidence compared to the record year 2007 (313/100,000) (Fig. 1) even though the bank vole prevalence was similar (18, 19).

One important factor that influences hantavirus transmission to humans is the increased exposure of humans to infected rodent excreta. Several reports from inhabitants in areas where bank voles normally dwell indicated that more bank voles were found in traps inside houses than normal (18). This led to an investigation regarding the weather conditions during this period: interestingly, December 2006 was exceptional with respect to the mild weather with no or very little snow and hard ice-cover in the coastal area of northern Sweden (Fig. 2A). During two previous NE peak periods (2001–2002 and 2004–2005), when records of snow cover were available, the ground was already covered in early winter (Fig. 2A). In December 2006, Sweden experienced an average temperature 4.5–9.5°C warmer than normal and in the most endemic region, Västerbotten County, the average temperature for December was 6.0–9.0°C warmer than normal (Fig. 2B). In previous peak years (December 2001–2002 and 2004–2005), the average temperature was normal (18).

In Sweden during 2007, the NE outbreak seemed to have at least two main determinants, a peak year for bank voles and an extreme climate in the affected regions with a record-breaking warm winter that registered temperatures 6–9°C above normal. The number of NE cases depends on the size of the vole population, which peaks every third to fourth year (16, 20) and an increase in the bank vole population was reported in northern Sweden in the autumn of 2006, similar to the preceding autumn of two NE peaks (1998–1999 and 2004–2005) (19). Thus the bank vole population was high, but not more than previous peak years and could not alone explain the very high number of NE cases in early 2007.

Snow cover, an important factor for bank vole survival, provides insulation from the cold, protection from predators and access to food below the snow (13). Lately, the winters in the regions in northern Sweden where most cases of NE occur have been unusually mild (21). They have been characterised by late snowfall, periods with no snow and sometimes rain followed by freezing temperatures (21) destroying the protective effect of the snow cover. These extreme climate conditions with absence of

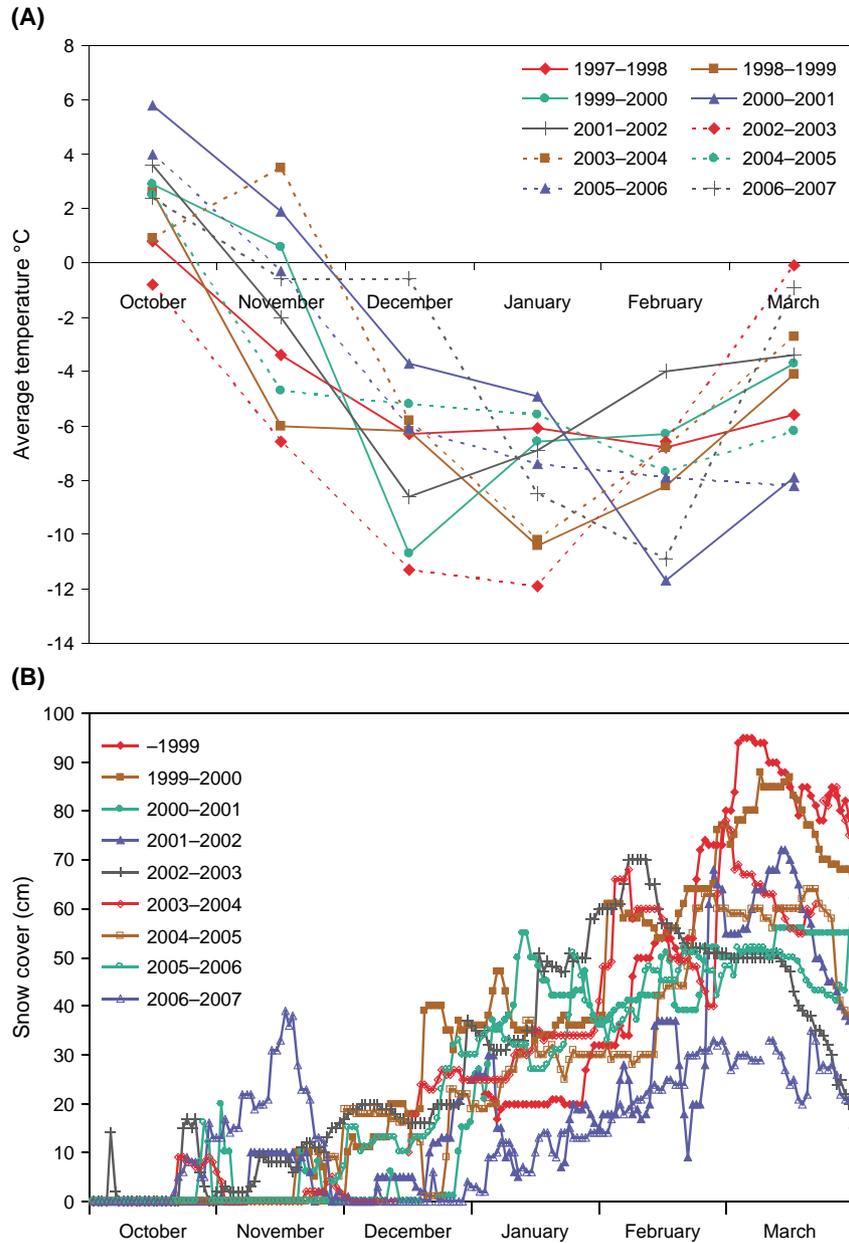


Fig. 2. Climate conditions December 1999–2007 in the NE outbreak area of Västerbotten County. The figures show number of days with snow cover and the average temperature. Snow cover was defined as snow depth >0 cm. The measurements were made in locations approximately 30 km from the coast.

the snow cover in northern Sweden during December 2006 and January 2007 probably contributed to the transmission of PUUV from bank voles to humans. The number of NE cases among humans as well as the PUUV prevalence among bank voles is linked to variables favouring the survival of the virus in the environment, such as indirect transmission facilitated by low winter temperatures (22). The increased PUUV transmission in northern Sweden seemed to be facilitated by more frequent contact between bank voles and humans. It is conceivable that during December 2006,

when there was no snow cover for a long period, bank voles sought refuge in barns and houses and other buildings, a behaviour that increased the exposure for humans. In 2007 and 2008, there were still rather high numbers of diagnosed NE patients (Fig. 2), but these increases most probably were related to the bank vole population peak during this period (23). Furthermore, the food availability for bank voles could also be affected by climate conditions. In Belgium, the relationship between tree seed production, climate and NE incidence has been analysed and NE epidemics are preceded by

abundant tree seed production (24). Moreover, a direct link between climate and NE incidence was found, with high summer and autumn temperatures, two years and one year, respectively, before NE occurrence, related to high NE incidence (24). Of course, another possible factor for the outbreak could be that the virus itself has mutated and to study this we have compared the PUUV genome from 2007 with previous years. Virus RNA from three patient samples were sequenced and revealed that the obtained sequences were highly homologous to previous rodent PUUV isolates from the area (18, 25).

The bank vole and other rodents play an important role as reservoir for many pathogens that may cause disease in humans and domestic animals (26). Many of these pathogens are recognised as emerging zoonotic infections (7). Climatic changes may greatly affect the rodent population size and behaviour, increasing the risk for transmission of several human pathogens (1, 4). Similarly, other vector-borne infections may increase due to climate change (1, 2, 5, 8). To minimise threats to public health and to maximise preparedness and proper actions, surveillance of reservoirs, vectors and pathogens are of the greatest importance. Attempts to forecast disease outbreaks have focused on the impact of climate variability on infectious diseases (27). Using El Niño/Southern Oscillation-related climate anomalies, vector-borne infections – such as dengue fever, malaria, Rift Valley fever, West Nile fever and hantavirus disease – have been predicted to occur in several parts of the world (28). For the vector-borne Rift Valley fever virus, the risk-mapping model using these climate data predicted areas where outbreaks of Rift Valley fever in humans and animals were expected and occurred in the Horn of Africa from December 2006 to May 2007 (29).

Conclusion

A combination of a mild climate at the beginning of winter, loss of protective snow cover and high rodent reservoir numbers likely caused the sudden and dramatic increase of NE cases in the winter 2006/2007 in northern Sweden. The globally spread, rodent-borne hantaviruses should be regarded as an increasing threat to health since future climate change scenarios predict higher temperatures.

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References

1. Semenza JC, Menne B. Climate change and infectious diseases in Europe. *Lancet Infect Dis* 2009; 9: 365–75.
2. Gould EA, Higgs S. Impact of climate change and other factors on emerging arbovirus diseases. *Trans R Soc Trop Med Hyg* 2009; 103: 109–21.
3. Jaenson TG, Eisen L, Comstedt P, Mejlon HA, Lindgren E, Bergström S, et al. Risk indicators for the tick *Ixodes ricinus* and *Borrelia burgdorferi* sensu lato in Sweden. *Med Vet Entomol* 2009; 23: 226–37.
4. Klempa B. Hantaviruses and climate change. *Clin Microbiol Infect* 2009; 15: 518–23.
5. Nakazawa Y, Williams R, Peterson AT, Mead P, Staples E, Gage KL. Climate change effects on plague and tularemia in the United States. *Vector Borne Zoonotic Dis* 2007; 7: 529–40.
6. Patz JA, Graczyk TK, Geller N, Vittor AY. Effects of environmental change on emerging parasitic diseases. *Int J Parasitol* 2000; 30: 1395–405.
7. Kuiken T, Leighton FA, Fouchier RA, LeDuc JW, Peiris JS, Schudel A, et al. Public health. Pathogen surveillance in animals. *Science* 2005; 309: 1680–1.
8. Zeman P, Beneš C. A tick-borne encephalitis ceiling in Central Europe has moved upwards during the last 30 years: possible impact of global warming? *Int J Med Microbiol* 2006; 293: 48–54.
9. Schmaljohn C, Hjelle B. Hantaviruses a global disease problem. *Emerg Infect Dis* 1997; 3: 95–104.
10. Vapalahti O, Mustonen J, Lundkvist Å, Henttonen H, Plyusnin A, Vaheri A. Hantavirus infections in Europe. *Lancet Infect Dis* 2003; 3: 653–61.
11. Piechotowski I, Brockmann SO, Schwarz C, Winter CH, Ranft U, Pfaff G. Emergence of hantavirus in South Germany: rodents, climate and human infections. *Parasitol Res* 2008; 103: S131–7.
12. Clement J, Vercauteren J, Verstraeten WW, Ducoffre G, Barrios JM, Vandamme AM, et al. Relating increasing hantavirus incidences to the changing climate: the mast connection. *Int J Health Geogr* 2009; 8: 1.
13. Hansson I, Henttonen H. Gradients in density variations of small rodents: the importance of latitude and snow cover. *Oecologia* 1985; 67: 394–402.
14. Crespin L, Verhagen R, Stenseth NC, Yoccoz NG, Prévot-Julliard A-C, Lebreton J-D. Survival in fluctuating bank vole populations: seasonal and yearly variations. *Oikos* 2002; 98: 467–79.
15. Hörnfeldt B. Cycles of voles, predators, and alternative prey in boreal Sweden. Umeå University Dissertation, Umeå, Sweden; 1991.
16. Olsson GE, Dalerum F, Hörnfeldt B, Elgh F, Palo TR, Juto P, et al. Human hantavirus infections, Sweden. *Emerg Infect Dis* 2003; 9: 1395–401.
17. Settergren B. Clinical aspects of nephropathia epidemica (Puumala virus infection) in Europe: a review. *Scand J Infect Dis* 2000; 32: 125–32.
18. Petterson L, Boman J, Juto P, Evander M, Ahlm C. Outbreak of Puumala virus infection, Sweden. *Emerg Infect Dis* 2008; 14: 808–10.
19. Olsson GE, Hörnfeldt B, Hjertkvist M, Lundkvist Å. Nephropathia epidemica: high risk in Norrland during winter. *Läkartidningen* 2007; 104: 3450–3. [In Swedish].
20. Brummer-Korvenkontio M, Vapalahti O, Henttonen H, Koskela P, Kuusisto P, Vaheri A. Epidemiological study of nephropathia epidemica in Finland 1989–96. *Scand J Infect Dis* 1999; 31: 427–35.

21. Alexandersson H. Temperature and precipitation in Sweden 1860/2002. Norrköping: SMHI; 2002. Meteorologi rapport 104. [In Swedish with English abstract].
22. Linard C, Tersago K, Leirs H, Lambin EF. Environmental conditions and puumala virus transmission in Belgium. *Int J Health Geogr* 2007; 6: 55.
23. Olsson GE, Hjertqvist M, Lundkvist A, Hörnfeldt B. Predicting high risk for human hantavirus infections, Sweden. *Emerg Infect Dis* 2009; 15: 104–6.
24. Tersago K, Verhagen R, Servais A, Heyman P, Ducoffre G, Leirs H. Hantavirus disease (nephropathia epidemica) in Belgium: effects of tree seed production and climate. *Epidemiol Infect* 2009; 137: 250–6.
25. Pettersson L, Klingström J, Hardestam J, Lundkvist A, Ahlm C, Evander M. Hantavirus RNA in saliva from patients with hemorrhagic fever with renal syndrome. *Emerg Infect Dis* 2008; 14: 406–11.
26. Davis S, Calvet E, Leirs H. Fluctuating rodent populations and risk to humans from rodent-borne zoonoses. *Vector Borne Zoonotic Dis* 2005; 5: 305–14.
27. Epstein P. Climate change and infectious disease; stormy weather ahead. *Epidemiology* 2002; 13: 373–5.
28. Anyamba A, Chretien JP, Small J, Tucker CJ, Linthicum KJ. Developing global climate anomalies suggest potential disease risks for 2006–2007. *Int J Health Geogr* 2006; 5: 60.
29. Anyamba A, Chretien JP, Small J, Tucker CJ, Formenty PB, Richardson JH, et al. Prediction of a Rift Valley fever outbreak. *Proc Natl Acad Sci USA* 2009; 106: 955–9.

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Local scale prediction of *Plasmodium falciparum* malaria transmission in an endemic region using temperature and rainfall

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Background: To support malaria control strategies, prior knowledge of disease risk is necessary. Developing a model to explain the transmission of malaria, in endemic and epidemic regions, is of high priority in developing health system interventions. We develop, fit and validate a non-spatial dynamic model driven by meteorological conditions that can capture seasonal malaria transmission dynamics at the village level in a malaria holoendemic area of north-western Burkina Faso.

Methods: A total of 676 children aged 6–59 months took part in this study. Trained interviewers visited children at home weekly from December 2003 to November 2004 for *Plasmodium falciparum* malaria infection detection. *Anopheles* daily biting rate, mortality rate and growth rate were evaluated. Digital meteorological stations measured ambient temperature, humidity and rainfall in each site.

Results: The overall *P. falciparum* malaria infection incidence was 1.1 episodes per person year. There was strong seasonal variation in *P. falciparum* malaria infection incidence with a peak observed in August and September, corresponding to the rainy season and a high number of mosquitoes. The model estimates of monthly mosquito abundance and the incidence of malaria infection correlated well with observed values. The fit was sensitive to daily mosquito survival and daily human parasite clearance.

Conclusion: The model has demonstrated potential for local scale seasonal prediction of *P. falciparum* malaria infection. It could therefore be used to understand malaria transmission dynamics using meteorological parameters as the driving force and to help district health managers in identifying high-risk periods for more focused interventions.

Keywords: local scale; modelling; prediction; *Plasmodium falciparum* malaria; under five years; endemic region

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Malaria continues to be a deadly disease and action towards its control remains challenging for researchers and policymakers. To support control strategies, prior knowledge of disease risk is necessary. Developing a model to explain the transmission of malaria, in endemic and epidemic regions, is of high priority in developing health system interventions. As malaria is a vector-borne disease, the life cycle of its vector, the female *Anopheles* mosquito, drives the transmission. The life cycles of both the vector and the parasite within the vector depend on the microclimate.

Since the early 20th century, there have been attempts to understand malaria transmission dynamics, through

mathematical modelling, to support control efforts. Ross developed the first model to predict malaria transmission and spread of the disease, and later concluded that increasing vector mortality significantly could eradicate malaria (1, 2). In the 1950s, George MacDonald, building on Ross' model, concluded that, at equilibrium, the weakest link in the cycle of malaria transmission is the adult female *Anopheles* (3). His conclusions formed the basis of the global malaria eradication campaign, with DDT targeted at adult female *Anopheles*. In the 1970s, Dietz and Molineaux, in the Garki project, developed a more sophisticated model, clearly considering human immunity interacting with transmission (4–6).

Further, Halloran and colleagues considered the population-level effects of potential stage-specific vaccines (7). Since then, malaria modelling has drawn significant attention. Populations are modelled as large numbers of interacting individual humans and individual mosquitoes, each with its own characteristics and dynamics (6). Further steps towards biological realism have included the effects of weather (8–16). With the shift back from malaria control to elimination and possible eradication (17–20), a number of current models are focusing on drug resistance (21, 22) and vaccine development (23).

The lack of data in many components of malaria transmission has restricted modelling efforts to a regional scale, since a significant pool of data is needed to test and fit the different sets of parameters. Even though available models are informative for developing global, regional or national malaria control strategies, they are limited in their applicability at local sites. However, local conditions are the main drivers of malaria transmission (24). Thus, better understanding of these conditions and transmission dynamics through modelling may be more informative and relevant for local control efforts.

This study elected to develop and validate a non-spatial dynamic model, driven by meteorological conditions, which can capture seasonal malaria transmission dynamics, at the scale of a single village. This was achieved by using comprehensive field data that included incident cases of human *Plasmodium falciparum* (*Pf*) malaria infection, as well as entomological and meteorological data. The focus for human infection was on children under five years, since they are the most vulnerable, and because most infections in this age group will be symptomatic and, therefore, more easily detected.

Methods

Study sites

This study was conducted in the town of Nouna and the villages of Cissé and Goni. These three sites are part of the Nouna Demographic Surveillance System (DSS) area (25). A detailed description of the study sites is given elsewhere (26).

Study population

A total of 676 children (Cissé: 171, Goni: 240 and Nouna: 265), aged 6–59 months, took part in this study. The children were selected in each site by systematic cluster sampling of households from the DSS database. A detailed description of the study population is given elsewhere (27).

Active case detection: *Plasmodium falciparum* infection

In each site, site-based interviewers visited the children weekly to assess their *Pf* malaria infection status and

collect housing conditions data. The case detection methods are extensively described by Yé and colleagues (27).

The outcome measure was a *Pf* infection episode, defined as an axillary temperature of at least 37.5°C plus a positive malaria parasite test.

Entomological data

Mosquito population abundance was monitored by using a standard Center for Disease Control (CDC) Light Trap (LT) (28) from December 2003 through November 2004. Mosquitoes were captured on the first and second day of each month at each site in four randomly selected houses.

LTs fitted with incandescent bulbs were installed close to human volunteers sleeping under untreated mosquito nets in these houses for two consecutive nights from 18:00 to 06:00 hours. In addition, we used the Human Landing Collection (HLC) method, which involves one person sitting inside an uninhabited house and another outside, collecting mosquitoes that land on their exposed legs by using torchlight and test tubes. This was done in two shifts (18:00–24:00 hours and 24:00–06:00). HLC volunteers gave informed consent. They were given malaria prophylaxis and checked for fever for a fortnight after their participation in the study.

Field supervisors transported the mosquitoes caught to the laboratory in a cold-box. A technician in entomology counted and sorted the specimens by species. He classified mosquitoes caught by LT and HLC as ‘unfed’, ‘partly-fed’, ‘fully fed’, ‘semi-gravid’ or ‘gravid’ by external inspection (LT) or dissection (HLC). The technician checked for parity the ovaries of unfed HLC mosquitoes as described by Detinova (29) and Gilles and Warrell (30).

The age structure of the *Anopheles gambiae* population was assessed by calculating the parity (number of times eggs laid previously). A high fraction of nulliparous mosquitoes (mosquitoes that had never laid eggs) signifies a young population. This is used to estimate the proportion of infectious vectors to calculate the value of the infectious bite rate parameter.

Indoor human bite rates (3) were calculated for each month and site, as follows: Human bite rate: $ma = Bs/P/n$, where Bs is the number of *A. gambiae* caught indoors by HLT; P is the number of people involved in the capture and n is the total number of nights.

A. gambiae mortality (k -value) was calculated for each month and site. This expresses the number of vectors surviving from the egg stage to the adult stage. The monthly number of vectors was transformed into a natural logarithm. For a month with no vectors, the logarithm of one was calculated. Based on previous studies, we assumed the maximum number of eggs oviposited by individual mosquitoes was, $e = 100$ eggs (31, 32) on average. To calculate k -value, the following

formula (33) was used:

$$\log(\text{potential_eggs, month } 1) = \log(\text{adults_mosquito} \\ + 1, \text{ month } 1) + \log(e) \text{ and } k\text{-value}_{\text{month } 1} = p \cdot \log \\ (\text{adults-mosquito, month } 2)$$

The resulting k -value was used to calculate the monthly mortality rate (m), an important parameter of our model, by using the formula: $m = 1 - 10^{-k\text{-value}}$.

Measurement of meteorological parameters

Three site-based meteorological units measured rainfall, temperature and relative humidity on the ground. Units were set for 10-second measurement cycles and 10-minute recording cycles. Details are given elsewhere (27).

Model development

Model description

We used the so-called ‘‘compartmental model’’ developed by Ross (1) and adapted by MacDonald (3). These models were based on the assumption that the human population can be subdivided into three compartments: (1) *susceptible* (do not have malaria); (2) *infected* (have the parasite, but it has not yet developed to the gametocyte stage); and (3) *infectious* (are symptomatic and have the parasite at the gametocyte stage). Similarly, the vector population can be classified as: (1) *susceptible* (do not carry the parasite); (2) *infected* (fertilisation and sporogony); and (3) *infectious* (sporozoites in the salivary glands). The transmission process starts when an infected vector takes a blood meal from a human. The changes among the subpopulations in each compartment are determined by a set of parameters, like mosquito mortality, bite rate, growth rate, sporogony and gonotrophic cycle duration, and human malaria-induced mortality and parasite clearance rates. Most malaria models were constructed on these basic assumptions, as was the model by McKenzie and others (34) from which our model is derived. In our model, the mosquito population was divided into two subpopulations, non-infected and infected, since we assumed that every mosquito that feeds on an infected human would have 100% probability of becoming infectious if it survived long enough. The state and transition of the model (Fig. 1) shows the changes in each subpopulation given different parameters. These parameters are labelled with Greek characters and defined in Table 1. This model is an extension of a previous model, which was set to detect malaria in the dry season (35). That model was driven by entomological data and did not simulate the dynamics of the vector population. This current one has vector population dynamics, which is driven by temperature and rainfall. Since the dry season in the study region is characterised by total absence of rainfall, a model driven by rainfall would not have been appropriate to capture transmission. Appendix 1 provides the details of the

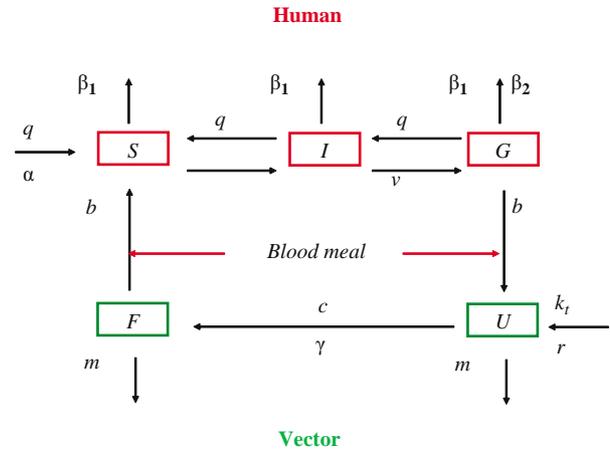


Fig. 1. State and transition of the dynamic model. Human: S , susceptible; I , infected; G , infectious. Vector: U , susceptible; F , infectious.

mathematical expressions of the model and the specific assumptions.

Model implementation, prediction and testing

The model was driven by temperature, which defines the sporogonic and gonotrophic cycles, and by rainfall. Both meteorological values were used to calculate the carrying capacity (k_f) described in Appendix 1. To train the model most of the parameters were estimated using field data collected in 2004. Because we did not have data for 2005, the outputs of the model, which consist of monthly mosquito numbers and cases of malaria infection, were compared with data from 2004. The model outputs were normalised to allow comparison with observed values. The normalisation was done by multiplying the monthly value of the model outputs with a constant obtained by dividing the highest value of the observed with the highest value of the model output.

The model was implemented in a Microsoft Excel spread sheet using a set of difference equations with one day step. Each of the variables representing the human and mosquito subpopulations was followed in a separate column. In addition, at each stage, the model calculated the daily changes of these variables. An offset function was used for processes with delay, such as mosquitoes becoming infectious at the end of the sporogonic cycle.

The model's *goodness of fit* Δ was determined by using the residual sum of squares (SS) of the difference between the predicted and the observed values of all months. The value of each parameter was determined successively by minimising SS (Table 2). This was continued for all parameters, until no further improvements in fit were possible, which was the common minimum for all parameters. Around the determined joint-optimal value for all parameters, each parameter was varied in turn to determine whether the fit was highly sensitive to the

Table 1. Definition of model parameters

Parameters	Definition	Source
α	Daily natural per-capita human birth rate	DSS, recalculated in daily birth rate
β_1	Daily natural per-capita human death rate	DSS, recalculated in daily death rate
β_2	Daily malaria-induced per capita death rate in humans	Noun DSS, recalculated in daily death rate
q	Daily malaria clearance rate in humans	Fitted and compared with field data
ν	Time delay for human host, from becoming infected to becoming infectious	Dietz et al. (4)
m	Daily mortality rate of vectors	Calculated and fitted
r	Daily mosquito per-capita intrinsic growth rate	Theoretical maximum of 10, precise value fitted from model
B	Daily bite rate of vectors	The lower bound if 1/gonotrophic cycle, precise value fitted from model
b	Daily rate at which vectors bite humans	$b = B \times \text{HBI}$
γ	Daily probability of vector becoming infected after infectious bite	Fitted
c	Time delay for vector from infection to infectious stage	Sporogonic cycle, calculated using Detinova formula 111/ ($T^\circ\text{C} - 18$)
K_t	Environmental carrying capacity	$K_t = Pmm \times akt$

parameter values. A parameter was 'sensitive' if 10% variation in the parameter value caused 30% variation in Δ . This process was employed in lieu of sufficient data to allow calculation of confidence intervals. The Microsoft Excel 'Solver Add-In' function, which uses the Generalized Reduced Gradient (GRG2) method, was used for this process.

The model predicted mosquito abundance and malaria incidence for each month and site for the year 2004. Output values were normalised versus the expected, by multiplying each predicted monthly value by a ratio which was obtained by dividing the observed highest value by the predicted value. The variances for the

normalised prediction and observed values were calculated to assess the fit of the model for each site. Small variance suggests good representation of the field data by the model. The fit was also presented graphically, by plotting the monthly predicted and observed values.

Results

During follow up, out of the 676 children, 20 (3.0%) left the cohort, either because of death (11) or migration out of the study sites (9). Children were not always present at each visit; therefore, the overall person-years (PY) observed were 594.9.

Plasmodium falciparum malaria infection incidence

Out of 1,274 fever episodes, 635 were positive for *Pf* malaria infection, giving an infection incidence of 1.1 episodes per PY. The lowest incidence was observed in Nouna (0.8 per PY). In Cissé and Goni, the incidences were 1.2 and 1.3, respectively, but not significantly different. There was strong seasonal variation in the incidence, with higher rates in August and September (Table 3).

Entomological patterns

Using the LT and HLC method combined, with all species included across all sites, 16,657 mosquitoes were caught. The largest proportion of captured mosquitoes was *Culex* (72.19%), followed by *A. gambiae* (15.57%), *Aedes* (6.3%), *Mansonia* (4.6%), *Anopheles funestus* (1.5%) and *Anopheles nili* (0.1%). The highest number of *A. gambiae* was caught in Goni ($n = 1,431$), followed by Cissé ($n = 598$) and Nouna ($n = 565$).

Table 2. Model parameter values and bounds

Parameters	Cissé [bounds]	Goni [bounds]	Nouna [bounds]
α	0.000126	0.000126	0.000126
β_1	0.000096	0.000096	0.000096
β_2	0.000041	0.000041	0.000041
q	0.12 [0.10– 0.17]	0.12 [0.10– 0.17]	0.12 [0.10–0.17]
ν	10 days [9–15]	10 days [9–15]	10 days [9–15]
r	2	2	2
m	0.15 [0.06– 0.20]	0.15 [0.07– 0.22]	0.14 [0.05–0.22]
b	0.56 [0.5–0.6]	0.56 [0.5–0.6]	0.56 [0.5–0.6]
γ	0.79	0.79	0.79
c	10.6 days [9–14]	13.3 days [9–14]	9.9 days [9–14]

Table 3. *Plasmodium falciparum* malaria infection incidence rates, per 1,000, per month and site

Months	Villages			All
	Cissé	Goni	Nouna	
Dec-03	159.2	122.0	88.9	136.6
Jan-04	43.6	37.6	34.1	37.3
Feb-04	137.9	38.1	57.5	69.1
Mar-04	123.4	85.4	42.6	82.6
Apr-04	14.6	59.2	125.3	72.0
May-04	6.7	31.0	22.0	26.2
Jun-04	6.3	29.1	12.4	20.9
Jul-04	14.2	111.4	35.6	58.0
Aug-04	268.6	220.7	83.2	220.2
Sep-04	163.7	272.6	107.0	223.7
Oct-04	129.6	200.1	126.5	152.5
Dec-04	87.1	112.6	58.5	84.7
Total	1166.4	1278.7	692.1	1067.3

Meteorological conditions

All sites presented a similar pattern of meteorological conditions. The rainfall was concentrated in the months from May to October. The total amount of rainfall was higher in Nouna than in Cissé or Goni. The relative humidity pattern followed that of rainfall. The mean temperature was more or less similar in all sites. The average mean temperature for the whole period was lower in Goni, however, with high variation as compared to Cissé and Nouna. A detailed description of the meteorological conditions is given elsewhere (27).

Model simulation

Simulation of daily *Anopheles gambiae* abundance

In all three sites, rainfall was followed by an increase in the mosquito population two weeks later (Fig. 2). In Cissé, mosquitoes were few (fewer than 10 per day) over the first 120 days of the year, corresponding to January through April. The first peak in mosquito numbers was observed on the 122nd day of the year, followed by a second peak, one month later. These peaks were all observed after a peak in rainfall. Two other peaks in mosquito abundance were observed after the second peak. These increases corresponded to July and August, months with high rainfall. From August on, the vector population decreased significantly and continued to do so towards the end of year, after the end of the rainy season.

In Goni, the simulation produced several peaks in the vector population, following each peak in rainfall. As in Cissé, these peaks were clustered within a period from the 121st to 301st days of the year. This period corresponds with May through October. In contrast to Cissé, although

there was some daily variation, the vector population remained high over this period, probably because of the higher amounts of rainfall. After the end of the rainy season, we observed a drop in the mosquito population.

The Nouna site had about the same pattern of mosquito abundance and distribution as Goni, even though rainfall was more abundant. The mosquito population increased shortly after the onset of the rainy season. It remained high (about 100/day), with some variation until the end of the rainy season, when levels decreased to less than 10 mosquitoes daily. As at the other two sites, the highest peak in the mosquito population was observed about two weeks after the highest peak of rainfall in August.

Monthly prediction of *Anopheles gambiae* abundance compared to observed vector numbers

The model predicted a peak in vector numbers for all sites in September, matching the observations for Goni and Nouna (Fig. 3). In Cissé, the peak in the number of caught mosquitoes was observed one month earlier, in August and this, therefore, did not match the prediction. Consistent across all sites, the model prediction matched with observed numbers from January through April, though the numbers were small. In June, in Cissé and Goni, there was a predicted increase in mosquito population which was not observed in the field. At all three sites, there was a significant decline (both predicted and observed) in the vector population in October, and both remained low in November and December.

Overall, the model predictions fit the observed data. The fit was better in Nouna, where we observed the least variance ($\Delta = \sum(O_i \times P_i)^2 = 1696.5$, $SD = 8.8$); where O_i is the observed number in the vector population in a month, and P_i is the number predicted by the model. The variances for Goni and Cissé were 11,630.4 and 35,292.2, respectively.

Monthly predicted *Plasmodium falciparum* malaria infection episodes compared to observed

Incident cases of *Pf* malaria infection among children were also simulated by the model, per site and per month (Fig. 4). For all sites, there was a seasonal pattern in *Pf* infection incidence. From December through June, the incidence decreased progressively, and then increased from July through September, after which another decrease was observed. Although the predicted and observed incidences were similar, there were some specific variations, expressed by the variation Δ . The model predictions matched the observed episodes better in Goni, where the smallest variance was observed ($\Delta = 626.8$, $SE = 6.6$), versus Nouna ($\Delta = 733.7$, $SE = 4.8$) and Cissé ($\Delta = 882.8$, $SD = 6.7$).

Sensitivity of the model to different parameters

The dependence of the variance on the various parameters is presented in Fig. 5(a–f). Each parameter (X axis) is plotted against the variance (Y axis). The best value of the parameter is the one that causes the smallest variance. For instance in Fig. 5a: m shows that a value below 13% as well

as values above cause high variance, but this stabilises after 40%; in Fig. 5b: the best value of b is 0.6 (one bite every two days); in Fig. 5c: the best value of v is 13 days; in Fig. 5d: the best value of c is nine days; in Fig. 5e: the best value of q is 11.6% and in Fig. 5f: the best value of γ is 71%.

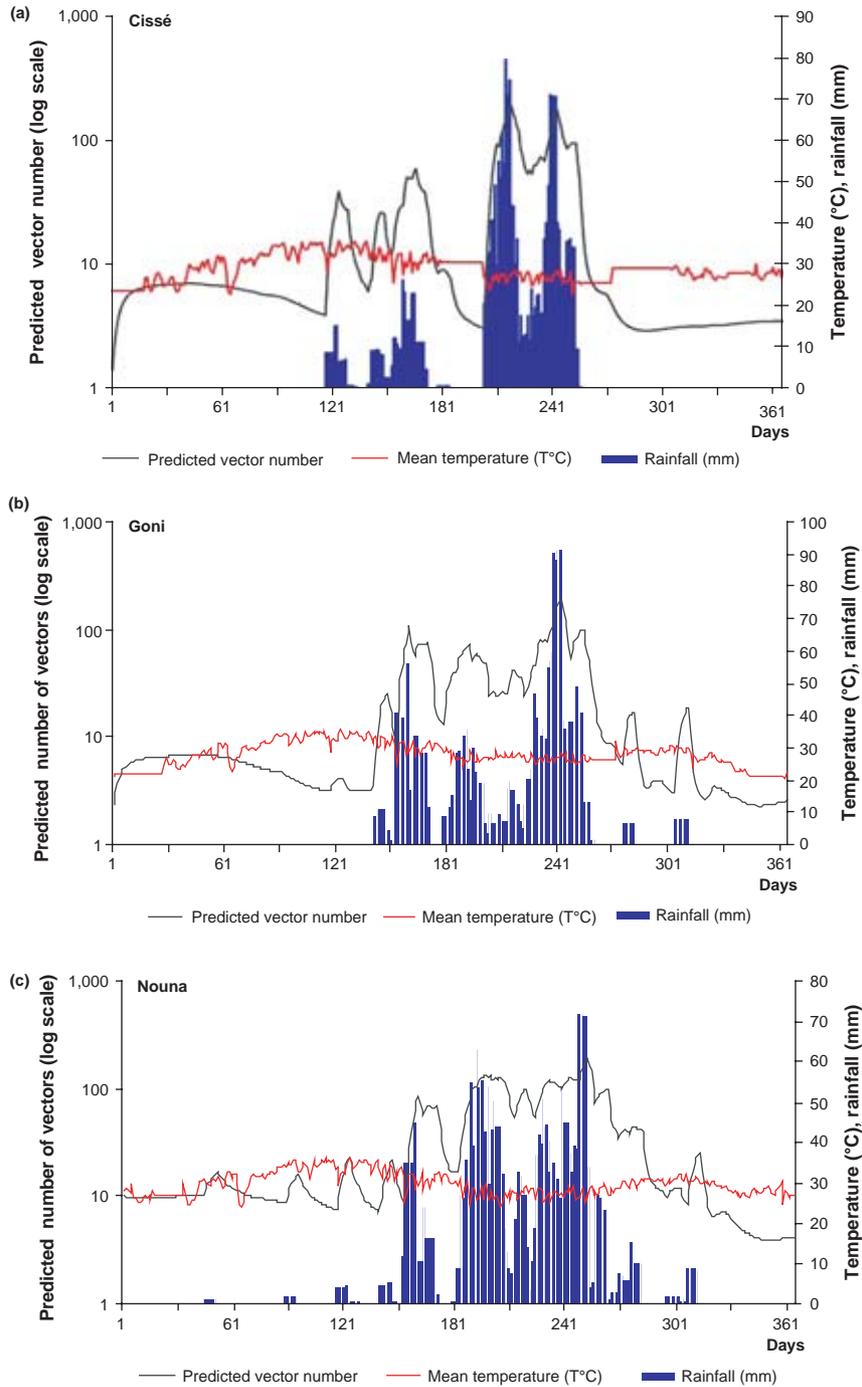


Fig. 2. Mean temperature and rainfall-based predictions of *A. gambiae* population abundance for each site: (a) Cissé, (b) Goni and (c) Nouna. Simulated *A. gambiae* population abundance (black curve) is plotted against the daily temperature (red curve) and the preceding two weeks' cumulative rainfall (blue curve).

Discussion

A dynamic model to predict malaria transmission among children under age five was developed. The model is composed of five difference equations that express changes in infectious status of the human and vector populations given temperature and rainfall conditions. The model simulated the vector population abundance and the human *Pf* malaria infection incidence for each of three ecological settings over one year. Most of the model parameters were calculated based on field data, and then fitted. The model was a good representation of *Pf* malaria infection in the region. The predicted mosquito populations and *Pf* malaria infection incidences were close to observed values.

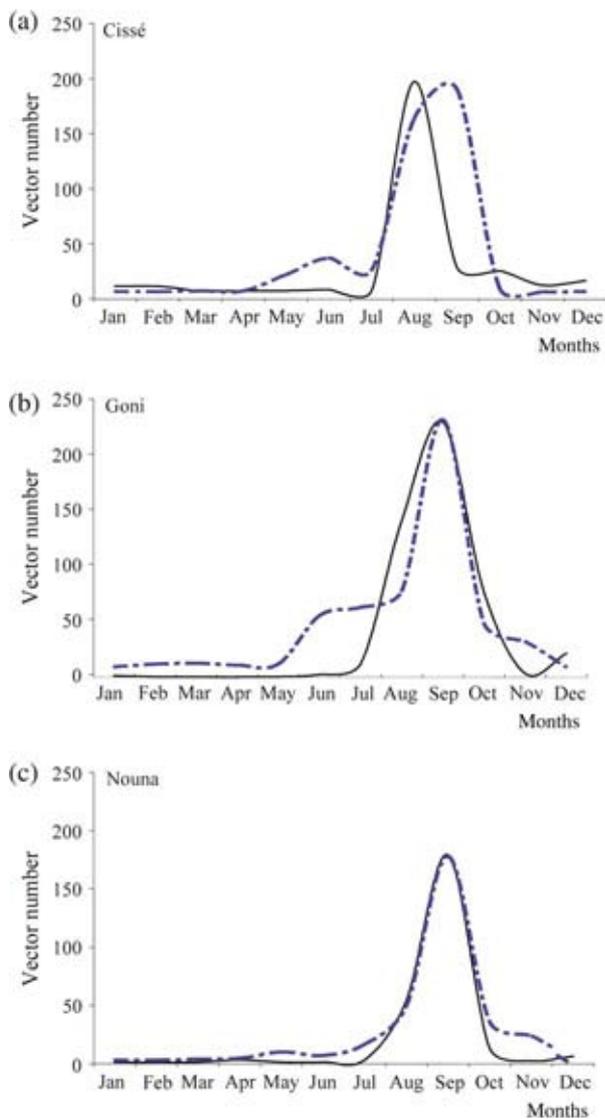


Fig. 3. Predicted monthly *A. gambiae*, compared to observed vector numbers in Cissé (a), Goni (b) and Nouna (c). The monthly prediction (broken line) of *A. gambiae* is compared with those caught in the field (full line).

Simulation of mosquito dynamics

Rainfall and temperature drive the vector population abundance. The dynamic model represented this adequately in all sites. Peak vector numbers observed about two weeks after a peak in rainfall are characteristic of the vector–rainfall relationship. Indeed, in ideal temperatures (28°C) and conditions, the development of *A. gambiae* from the egg to adult stage takes about 14 days (32, 36). The presence of water pools generated by rainwater allows the mosquitoes to lay their eggs, which then develop into adult mosquitoes if the water pools are sustained for at least 14 days. Some potential breeding sites could be expected in the area surrounding wells throughout the year. This is because of the constant spillage of water when people are fetching it. Sometimes, intentional pools are created for purposes of watering cattle. However, these pools are not common and only support a few mosquitoes. Because of the dry conditions in the area, the most important source of breeding sites remains rainfall water, and this explains the high

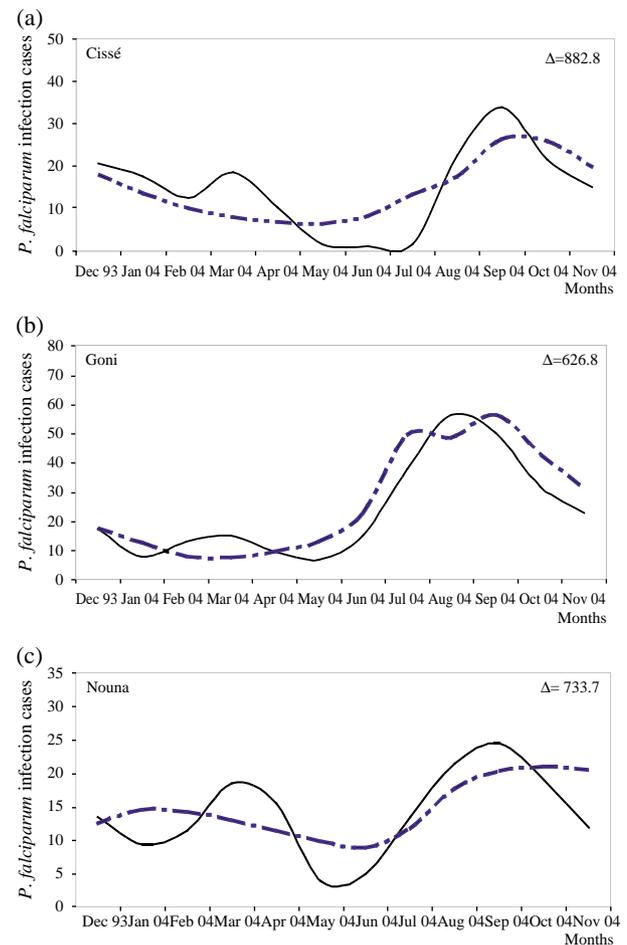


Fig. 4. Predicted monthly *Plasmodium falciparum* infection episodes versus observed episodes in Cissé (a), Goni (b) and Nouna (c). The monthly prediction (broken line) of episodes is compared with those observed in the field (full line).

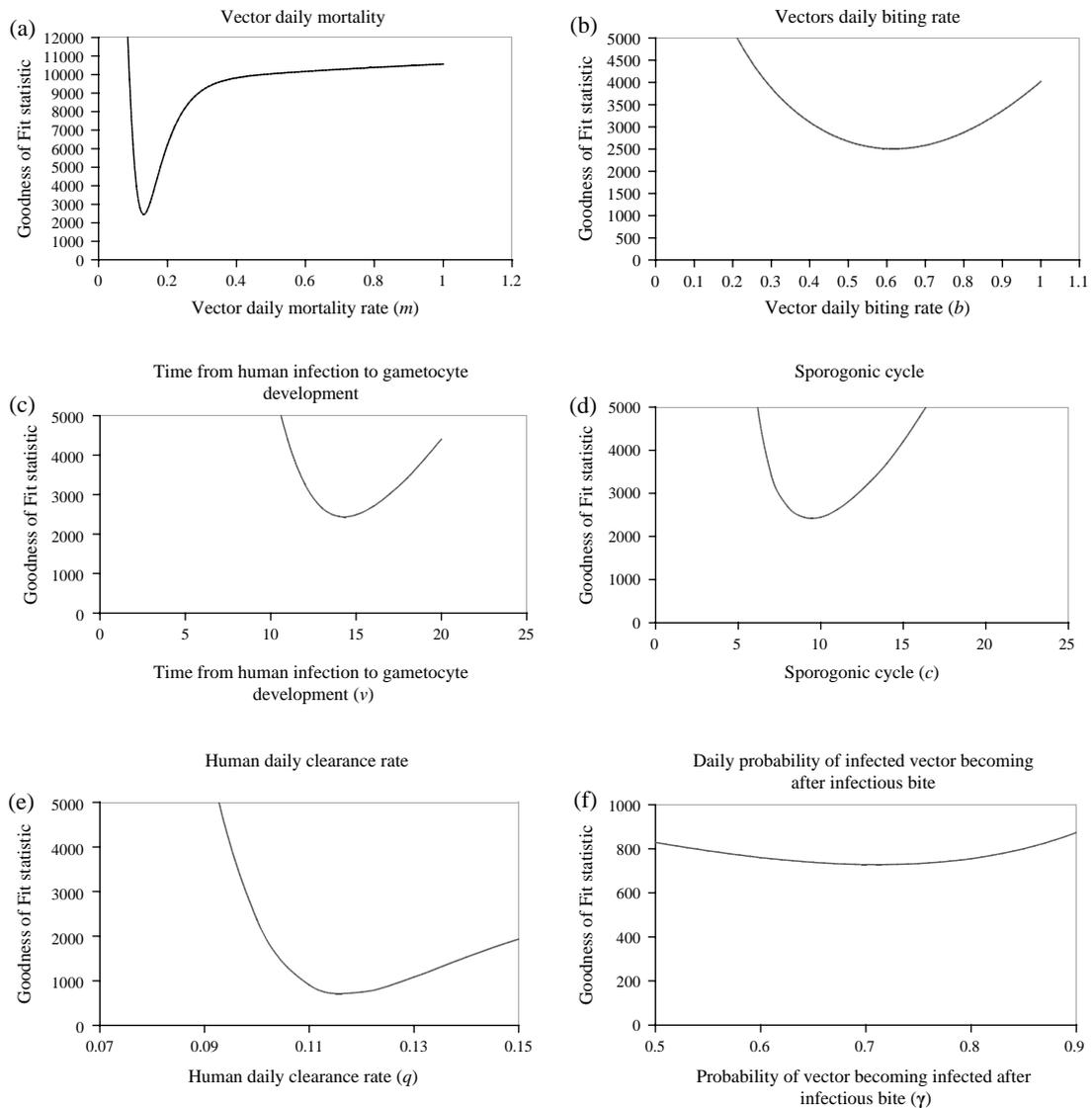


Fig. 5. Variation between the observed *Plasmodium falciparum* infection and the model output for single parameters.

abundance of mosquitoes during the rainy season. Rainfall was the main driver of vector abundance. As expected, in all sites the model detected few vectors (<10) during the dry season but vectors persisted, despite the total absence of rainfall during this season, probably because of breeding sites created around wells.

Monthly predictions of the number of vectors fit the numbers caught in the field, and, both predicted and observed numbers followed a similar pattern at all sites. This suggests that the model is a good representation of mosquito population dynamics. Some difference in the timing of peak abundance was observed in Cissé; where there was a deviation of one month between the predicted (September) and observed (August) peak. This may have been because of the soil texture in Cissé, which probably is not able to hold water on the surface long enough to allow vector development. However, this was not captured by

the current model. Consistent across all sites, the model predicted vectors in May and June, though no vectors were observed in the field. This could be explained by the model being sensitive to any amount of rainfall; whereas, in the field, the quantity of rainfall in May and June was not enough to keep vector breeding sites.

Although the model produced a fair representation of the mosquito population, it could be improved by also simulating the immature stage (eggs, larvae and pupae) of the vector, which are strictly dependant on surface water availability. Mosquitoes need water to reproduce and the oviposition rate is assumed to be proportional to mosquito numbers and the daily rainfall filling local water pools (16). Further, direct correlation of rainfall amount with mosquito abundance could result in some estimation bias. This is because the availability and duration of surface water are also dependant upon the

evaporation index, soil texture and moisture index. High evaporation will cause quick drying out of pools, whereas a lower consistency of soil texture and dry soil will lead to faster infiltration.

Simulation of *Plasmodium falciparum* malaria infection cases

Although some monthly differences were observed, probably due to the small number of cases, the general seasonal pattern was represented well by the model. However, the model is not sensitive to the sporogonic cycle. This implies that a small variation in ambient temperature would not result in major changes in incidence, and that time from human infection to gametocyte development is not a key in determining incidence rates.

The daily vector bite rate was found to be 0.56 per day. This would represent a gonotrophic cycle of 1.5 days, if every bite achieves a full blood meal. However, this is not always the case, as mosquitoes often return for second bites, if interrupted during their meal. Thus, the gonotrophic cycle may be longer than predicted by this model. The model is insensitive to precise values of b , (human bites per day) and this reduces the validity of the model as an estimator of gonotrophic cycle length. In addition, the model was developed assuming all vectors are *anthropophilic*, which is not necessarily the case. In fact, we expect this parameter to vary from one season to another (37).

The incidence of *Pf* malaria is dependent on two key parameters, which are the daily mortality rate of the vector and the parasite clearance rate in humans. These parameters can both be influenced by public health interventions. The daily mortality rate of the vector can be increased by vector control methods, such as indoor residual spraying, and vector numbers can be reduced by removing breeding sites. Effective treatment of patients will increase the malaria clearance rate in human (q), by protecting not only sick individuals, but also the surrounding population. The parasitological clearance rate (12%) was slightly slower than can be deduced from Müller and others (38), who witnessed 27% seven-day parasitological failure with chloroquine treatment. This would reflect 17% daily clearance. This discrepancy probably is a result of Müller and colleagues (38) having measured the asexual form clearance, while our focus was on the sexual form.

The model is driven by parasitological data for children under five, while the entire population contributes to the transmission. To account for this effect, we would need to survey the general population. This would require checking large numbers of asymptomatic individuals for subclinical infections. This raised technical and ethical issues. Nevertheless, it was assumed that parasite prevalence among children under five was not unlike that of

the general population, even though clinical symptoms would not be present in many older individuals.

The model can be a useful tool for malaria control strategies especially in a low transmission context. It has the ability to quantify the context-specific risk of malaria, a precondition for cost-effective interventions. Although, the model was developed based on data collected in a specific context it can be used in a different setting. In that case the parameters would have to be measured locally and fitted without the need to change the model formulation. The fitting of the model was based on field data to make sure that mathematical formulae are plausible and describe the biological process of the transmission of the disease. For use in predicting malaria incidences in other settings, the critical inputs will be rainfall and temperature data, which nowadays can be obtained from satellite sources. Other parameters to be fitted may be obtained from the literature.

The strength of this model lies in its simplicity and its respect for the biological process of malaria transmission on the ground. However, to be cost-effective, the model's major drivers which are rainfall and temperature could be derived from remote sensing data as ground-based measurements are expensive to implement at local scale.

Although this is an academic work to reproduce the biological process of malaria transmission given different meteorological conditions, the ultimate aim is to produce a tool that can be used to refine malaria control strategies at health district level. The practical use of the model is in its prediction of the expected monthly number of malaria cases among under five children in different villages from given health districts based on rainfall and temperature data from either national meteorological stations or forecasting data from satellite. Such prior prediction of cases will help health planners at local level to better mobilise and allocate scarce resources to areas with most need. We plan to develop user-friendly software with the model in the background. The software will allow the input of basic data in order to produce the estimated monthly cases of malaria for different villages. However, we will first validate the model for different years within the frame of future studies that will generate relevant data for this purpose.

Conclusion

The model shows potential for local-scale seasonal prediction of *Pf* malaria infection rates and distribution. Thus, it could be used to understand malaria transmission dynamics, using meteorological parameters as a driving force, to help local district health bodies to identify the risk period for more focused interventions. However, we do not pretend to have captured 100% of the transmission dynamics. Further improvements to the model can be made.

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References

- Ross R. Studies on malaria. London: John Murray; 1928.
- Utzinger J, Tozan Y, Singer BH. Efficacy and cost-effectiveness of environmental management for malaria control. *Trop Med Int Health* 2001; 6: 677–87.
- MacDonald G. Appendix I. Mathematical statement. In: MacDonald, G., ed. *The epidemiology and control of malaria*. London: Oxford University Press; 1957, p. 201.
- Dietz K, Molineaux L, Thomas A. Malaria model tested in the savannah. *Bull World Health Organ* 1974; 50: 347–57.
- Molineaux L, Gramiccia G. The Garki project. Geneva: World Health Organization; 1980, pp. 1–311.
- McKenzie FE, Samba EM. The role of mathematical modeling in evidence-based malaria control. *Am J Trop Med Hyg* 2004; 71: 94–6.
- Halloran ME, Struchiner CJ, Spielman A. Modelling malaria vaccines. II: population effects of stage-specific malaria vaccines dependent on natural boosting. *Math Biosci* 1989; 94: 115–49.
- RanDolph SE, Rogers DJ. Satellite data and disease transmission by vectors: the creation of maps for risk prediction. *Bull Soc Pathol Exot* 2000; 93: 207.
- Kleinschmidt I, Sharp BL, Clarke GP, Curtis B, Fraser C. Use of generalized linear mixed models in the spatial analysis of small-area malaria incidence rates in Kwazulu Natal, South Africa. *Am J Epidemiol* 2001; 153: 1213–21.
- Hay SI, Omumbo JA, Craig MH, Snow RW. Earth observation, geographic information systems and *Plasmodium falciparum* malaria in sub-Saharan Africa, remote sensing and geographical information system in epidemiology. *Adv Parasitol* 2000; 47: 173–215.
- Rogers DJ, Randolph SE, Snow RW, Hay SI. Satellite imagery in the study and forecast of malaria. *Nature* 2002; 415: 710–5.
- Craig MH, Snow RW, le Sueur D. A climate-based distribution model of malaria transmission in sub-Saharan Africa. *Parasitol Today* 1999; 15: 105–11.
- MARA/ARMA. Towards an atlas of malaria risk in Africa. First technical reports of the MARA/ARMA collaboration. Durban; 1998.
- Martens WJ, Niessen LW, Rotmans J, Jetten TH, McMichael AJ. Potential impact of global climate change on malaria risk. *Environ Health Perspect* 1995; 103: 458–64.
- Lindsay SW, Martens WJ. Malaria in the African highlands: past, present and future. *Bull World Health Organ* 1998; 76: 33–45.
- Hoshen MB, Morse AP. A weather-driven model of malaria transmission. *Malaria J* 2004; 3: 32.
- Hommel M. Towards a research agenda for global malaria elimination. *Malar J* 2008; 7: S1.
- Aguas R, White LJ, Snow RW, Gomes MG. Prospects for malaria eradication in sub-Saharan Africa. *PLoS One* 2008; 12: 3.
- Greenwood B. Can malaria be eliminated? *Trans R Soc Trop Med Hyg* 2009; 103: S2–5.
- Maude RJ, Pontavornpinyo W, Saralamba S, Dondorp AM, Day NP, White NJ, et al. The role of mathematical modelling in malaria elimination and eradication (Comment on: Can malaria be eliminated?). *Trans R Soc Trop Med Hyg* 2009; 103: 643–4.
- Koella JC, Antia R. Epidemiological models for the spread of anti-malarial resistance *Malar J* 2003; 2: 3.
- White LJ, Maude RJ, Pongtavornpinyo W, Saralamba S, Aguas R, Effelterre TV, et al. The role of simple mathematical models in malaria elimination strategy design. *Malar J* 2009; 8: 212.
- Fabrizio T, Nicolas M, Melissa P, Alain S, Thomas AS. Simulation of the cost-effectiveness of malaria vaccines. *Malar J* 2009; 8: 127.
- Garnham PCC. Malaria in Kisumu, Kenya colony. *J Trop Med Hyg* 1929; 32: 207–16.
- Yé Y, Sanou A, Gbangou A, Kouyaté B. INDEPTH. Demography and health in developing countries. Volume 1. Population, health and survival at INDEPTH sites. Chapter 19 Nouna DSS. Canada: IDRC; 2002.
- Yé Y, Kyobutungi C, Louis RV, Sauerborn R. Micro-epidemiology of *Plasmodium falciparum* malaria: is there any difference in transmission risk between neighbouring villages? *Malar J* 2007; 6: 46.
- Yé Y, Louis V, Simoboro S, Sauerborn R. Effect of meteorological factors on clinical malaria risk among children, using village-based meteorological stations and community-based parasitological survey. *BMC Pub Health* 2007; 7: 101.
- Cano J, Berzosa PJ, Roche J, Rubio JM, Moyano E, Guerra-Neira A, et al. Malaria vectors in the Bioko Island (Equatorial Guinea): estimation of vector dynamics and transmission intensities. *J Med Entomol* 2004; 41: 158–61.
- Detinova TS. Méthodes à appliquer pour classer par groupes d'âge les diptères présentant une importance médicale. [Age-grouping methods in Diptera of medical importance]. Geneva: Ser Monogr WHO No. 27; 1963.
- Gilles HM, Warrell DA. Bruce-Chwatt's essential malariology, 3rd ed. London: Edward Arnold; 1993.
- Takken W, Klwoden MJ, Chambers GM. Effect of body size on host seeking and blood meal utilization in *Anopheles gambiae sensu stricto* (Diptera: Culicidae): the disadvantage of being small. *J Med Entomol* 1998; 35: 639–45.
- Depinay JM, Mbogo CM, Killeen G, Knol B, Beier J, Carlson J, et al. A simulation model of African *Anopheles* ecology and population dynamics for the analysis of malaria transmission. *Malar J* 2004; 3: 29.
- Rogers DJ. Estimation of the mortalities of the immature stage. In: Youdeowei A, Service MW, eds. *Pest and vector management in the tropics with particular reference to insects, ticks, mites and snails*. London: Longman; 1983, pp. 139–59.
- McKenzie FE, Wong RC, Bossert WH. Discrete-event simulation models of *Plasmodium falciparum* malaria. *Simulation* 1998; 71: 250–61.
- Yé Y, Sauerborn R, Simoboro S, Hoshen M. Using modelling to assess malaria infection risk during the dry season on a local scale in an endemic area of rural Burkina Faso. *Ann Trop Med Parasitol* 2007; 101: 375–89.
- Jepson WF, Moutia A, Courtois C. The malaria problem in Mauritius: the binomics of Mauritian Anophelines. *Bull Entomol Res* 1947; 38: 177–208.
- Awolola TS, Okwa OO, Hunt RH, Ogunrinade AF, Coetzee M. Dynamics of the malaria vector populations in coastal Lagos, south-western Nigeria. *Ann Trop Med Parasitol* 2002; 96: 75–82.
- Müller O, Traoré C, Becher H, Kouyate B. Malaria morbidity, treatment-seeking behaviour, and mortality in a cohort of young

children in rural Burkina Faso. Trop Med Int Health 2003; 8: 290–6.

39. Detinova TS. Age-grouping methods in Diptera of medical importance with special reference to some vectors of malaria. Geneva: Monogr Ser WHO No 47; 1962.

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Appendix 1. Model description

The dynamic concept in contrast to the static concept, tries to capture the transmission and biological processes of the disease. The model driven by temperature and rainfall was based on the assumption that the human population is divided into three categories: susceptible (S), malaria-infected (I) and infectious (G), and mosquito population is classified into two compartments: non-infections (U) and infectious, strongly affected by temperature and rainfall.

$$\delta S = \alpha(S + I + G) + q(I + G) - \left(1 - \left(\frac{S + I + G - 1}{S + I + G}\right)^{bF}\right) S - \beta_1 S, \quad (1)$$

$$\delta I = \left(1 - \left(\frac{S + I + G - 1}{S + I + G}\right)^{bF}\right) S - (1 - (\beta_1 + \beta_2 + q))^v \times \left[\left(1 - \left(\frac{S + I + G - 1}{S + I + G}\right)^{bF}\right) S\right]_{t-v} - (\beta_1 + \beta_2 + q)I, \quad (2)$$

$$\delta G = (1 - (\beta_1 + \beta_2 + q))^v \times \left[\left(1 - \left(\frac{S + I + G - 1}{S + I + G}\right)^{bF}\right) S\right]_{t-v} - (\beta_1 + \beta_2 + q)G, \quad (3)$$

$$\delta U = \frac{r(U + F)}{\left[1 + \frac{(U + F)}{K_t}\right]} - \left[bU \frac{G}{S + I + G}\right]_t \gamma - mU, \quad (4)$$

$$\delta F = (1 - m)^c \left[bU \frac{G}{S + I + G}\right]_{t-c} \gamma - mF. \quad (5)$$

Equations 1–3 describe the change in the human population while Equations 4 and 5 describe change in vector population. Each term is explained in detail below.

Change in uninfected human population:

$$\delta S = \alpha(S + I + G) + q(I + G) - \left(1 - \left(\frac{S + I + G - 1}{S + I + G}\right)^{bF}\right) S - \beta_1 S. \quad (1)$$

Equation 1 describes the changes in the uninfected human population and includes four terms:

- The first term is the natural growth rate which is expressed by $\alpha(S + I + G)$, assuming people are born healthy and irrespective of the health of the mother. As the model is simulated daily, this is expected to be negligible.
- The second term is the malaria clearance expressed by $q(I + G)$. We assume that people clear the infection at a fixed rate from all stages of the disease. We also assume that there is no immunity and no super-infection (additional infection starts after a new hepatic stage), contrary to Dietz et al. (4).
- The third term is the human infection expressed by $\left(1 - \left(\frac{S + I + G - 1}{S + I + G}\right)^{bF}\right) S$. It expresses the daily new infection within the human population. The expression $\frac{S + I + G - 1}{S + I + G} = 1 - \frac{1}{S + I + G}$ is the probability of a single person not getting a bite from a specific mosquito; bF is the number of infectious mosquito biting in a day, given a daily biting rate per mosquito of b , $\left(\frac{S + I + G - 1}{S + I + G}\right)^{bF}$ is the probability of a specific person not getting bitten by any of the infectious mosquitoes. $1 - \left(\frac{S + I + G - 1}{S + I + G}\right)^{bF}$ is the probability of a specific person getting bitten by one or more of infectious mosquitoes. Multiplying by S gives the number of uninfected peoples being bitten by at least one infectious mosquito in a day.
- The fourth term β_1 is the death rate in the population from all causes except malaria, assuming there is not link with malaria. Then $\beta_1 S$ is the number of death within the uninfected population.

In addition the following assumptions were made:

1. A mosquito bites only once in a gonotrophic cycle.
2. Mosquitoes bite randomly. No specific attraction to any subpopulation.
3. The stage of infection does not influence the mosquitoes biting habits.
4. An infectious bite necessarily causes *Plasmodium falciparum* infection.

Change in infected human population:

$$\delta I = \left(1 - \left(\frac{S + I + G - 1}{S + I + G}\right)^{bF}\right) S - (1 - (\beta_1 + \beta_2 + q))^v \times \left[\left(1 - \left(\frac{S + I + G - 1}{S + I + G}\right)^{bF}\right) S\right]_{t-v} - (\beta_1 + \beta_2 + q)I. \quad (2)$$

Equation 2 describes the changes in the infected (but not infectious) human population and includes three terms:

- The first term is $\left(1 - \left(\frac{S + I + G - 1}{S + I + G}\right)^{bF}\right)S$ and as described above is the number of uninfected people being bitten by at least one infectious mosquito in a day.
- The second term $\left[\left(1 - \left(\frac{S + I + G - 1}{S + I + G}\right)^{bF}\right)S\right]_{t-v}$ represents people that became infected v days ago. They have now mature gametocytes and are infectious. However, not all of those people are still available. They may have either died of malaria or other disease or they may have cleared their infection. For each day the probability of leaving the group early will be $\beta_1 + \beta_2 + q$. The probability of remaining in the group for a day is $1 - (\beta_1 + \beta_2 + q)$. The probability of completing the whole process of v days is $(1 - (\beta_1 + \beta_2 + q))^v$.
- The third term $-(\beta_1 + \beta_2 + q)I$ represents the number of people that leave the infected stage by death or clearance.

In addition, the following assumptions were made:

1. β_2 is constant and does not change according to the stage of the infection. We know the mortality could change per stage. We may leave it out of this equation for biological reasons.
2. q is not specific to the stage of the infection. We have two types of q clearance because of treatment and clearance because of immune system (natural clearance). We could also decide there is no natural clearance. We also know that drugs are stage specific (liver stage and blood stage).

Change in infectious human population:

$$\delta G = (1 - (\beta_1 + \beta_2 + q))^v \times \left[\left(1 - \left(\frac{S + I + G - 1}{S + I + G}\right)^{bF}\right)S\right]_{t-v} - (\beta_1 + \beta_2 + q)G. \quad (3)$$

Equation 3 describes the changes in the infectious human population and includes two terms:

- The first term $(1 - (\beta_1 + \beta_2 + q))^v \left[\left(1 - \left(\frac{S + I + G - 1}{S + I + G}\right)^{bF}\right)S\right]_{t-v}$ is described above.
- The second term $-(\beta_1 + \beta_2 + q)G$ represents the number of people that leave the infectious stage by death or clearance.

Change in the size of uninfected vector population:

$$\delta U = \frac{r(U + F)}{\left[1 + \frac{(U + F)}{K_t}\right]} - \left[bU \frac{G}{S + I + G}\right]_t \gamma - mU. \quad (4)$$

Equation 4 describes the changes in the uninfected vector population and includes three terms:

- The first term $\frac{r(U + F)}{\left[1 + \frac{(U + F)}{K_t}\right]}$ is the maturation of the larval stage. This term describes the number of larvae surviving to become mature mosquitoes. The numerator is the number of larvae expected to survive to maturity under ideal conditions. $U + F$ is the total number of mosquitoes, assuming infectious status does not influence the fertility. r is the per mosquitoes fertility (number of eggs oviposited per day multiplied by the probability of each to develop into a mature mosquito under ideal condition). The denominator reflects the decrease in survival because of non-ideal conditions. The $U + F$ expresses the density dependent limitation on larvae survival. The precise characteristic of this dependence is determined by the carrying capacity K_t . In principle, K_t varies with temperature, rainfall and humidity and should be measured from the field. Thus the number of larvae increases with the number of mosquitoes but is limited by carrying capacity. The number of the larvae surviving is dependent on the surface water available. As at this stage of research a full evapo-transpiration model is not available, K_t is therefore assumed to be proportional to the previous weekly aggregated rainfall. $K_t = Pmm \times akt$. The value of akt is to be determined empirically.
- The second term $bU \frac{G}{S + I + G}$ represents the new infections of mosquito at time t . bU is the number of uninfected mosquitoes biting in a day. The fraction is the probability of a single mosquito biting at random an infectious human out of the total human population. We multiply this by γ to reflect the probability of becoming infected.
- The third term, mU , is the mortality of uninfected mosquitoes or the number of uninfected mosquitoes dying per day. m was calculated from the k -value (log generation mortality). In the study site setting, due to the constantly warm temperature, the gonotrophic cycle varies between two and three days. The survival of mosquitoes depends on the gonotrophic cycle and due to the stability of the cycle m was treated as

constant. The precise value of m was empirically determined by fitting the model.

In addition the following assumptions were made:

1. Mosquitoes bite randomly and independent of their infectious status.
2. Survival is independent of the infectious status.

Change in the size of the infectious vector population:

$$\delta F = (1 - m)^c \left[bU \frac{G}{S + I + G} \right]_{t-c} \gamma - mF. \quad (5)$$

Equation 5 describes the changes in the infected vector population and includes two terms:

- The first term, $(1 - m)^c \left[bU \frac{G}{S + I + G} \right]_{t-c} \gamma$ is the number of mosquitoes infected c days ago, reduced by the survival. c is the sporogonic cycle given by Detinova (39) as $111/(T^\circ - 18)$.
- The second term $-mF$ is the number of infectious mosquitoes dying in a day.

In addition the following assumptions were made:

1. Infectious mosquitoes never clear their infectious status.
2. Mosquitoes are either infected or infectious.

Meningococcal disease and climate

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Invasive meningococcal disease (IMD) is an epidemic infectious disease highly influenced by climatic factors. Climate plays an important role in both the spatial distribution of the disease and in the seasonality of IMD as seen all over the world (1). It is mentioned as one of the infectious diseases likely to be affected by climate change in the Fourth Assessment report of the Intergovernmental Panel on Climate Change (IPCC) (2).

IMD is an airborne disease with humans as its only reservoir. Global warming will change precipitation levels with a combination of more severe droughts in some areas and more frequent heavy precipitation events in others (2), and these are events likely to affect the incidence and geographical distribution of IMD (1). Effects suspected to be the result of climate change are already evident on the distribution of IMD epidemics in Africa (1, 3).

Research in the field of infectious diseases and climate has focused on vector-borne diseases like malaria and dengue fever (1, 4–8). Less is known about the effects of climate on airborne diseases like IMD.

The aim of this review is to give an overview of the current knowledge of how climate affects IMD and to more thoroughly investigate the climate research concerning IMD that has been published in the last decade.

The disease and its prevention

IMD includes meningococcal septicaemia and meningococcal meningitis. The disease is prevalent all over the world. It is caused by *Neisseria meningitidis*, a gram-negative coccoid bacteria. Treatment of IMD is still not a major problem as *N. meningitidis* is sensitive to a number of antibiotics, although betalactamase resistance is seen in some parts of the world (9–12). Despite treatment, 4–17% of the patients die (13–18) and 8–20% of the survivors will suffer from lifelong sequelae like deafness, cognitive impairment and other central nervous system complications (19–22). The age groups most susceptible to the disease are young children, adolescents and young adults (23, 24).

N. meningitidis can be divided into serogroups on the basis of polysaccharide capsular antigens. The major

pathogenic serogroups are A, B, C, W135, X and Y (24, 25). There are differences between the serogroups, both in virulence and in their capacity to cause epidemics. Large-scale epidemics are mainly caused by serogroup A, although serogroups W135 and C have also been implicated in epidemics. Smaller outbreaks and singular cases of the disease are more commonly caused by serogroups B and C and less frequently by other serogroups (24, 26, 27).

IMD is transmitted through respiratory secretion or saliva. *N. meningitidis* is found mainly in the upper respiratory tract (URT). Acquisition of the bacteria can either be transient or result in colonisation of the URT epithelium (carriage) or in invasive disease (24). The epidemiology of IMD is complex due to the great number of symptom-free carriers of the bacteria. In populations studied, 2–70% of people have been found to be carriers of the bacteria in their airways (24, 28–30). In Africa, carrier rates have been shown to be higher during epidemics than in the endemic situation (24, 28, 30, 31) and less frequent in small children than in adolescents and adults (24, 31). Carriage of *Neisseria* plays an important role in the epidemiology of the disease, a role that is still not well understood (29, 32).

Vaccines exist for serogroups A, C, W135, X and Y but no commercial vaccine has yet been produced for serogroup B. Limitations of the vaccines are short duration of immunity, 3–5 years, low immunogenicity in children under two years and no effect on carriage of the bacteria (24, 25). New conjugate vaccines have been developed for serogroups A and C, with prolonged immunity, better immunological effect in young children and with protective effects against carriage (24, 33–36).

Epidemiology of invasive meningococcal disease (IMD)

Meningitis belt

In 1963, the French physician Lapeyssonnie described a geographically well-defined area in Sub-Saharan Africa with an exceptionally high incidence of meningococcal meningitis (37). This area, the classical meningitis belt (Fig. 1), has seen epidemics of the disease at intervals of

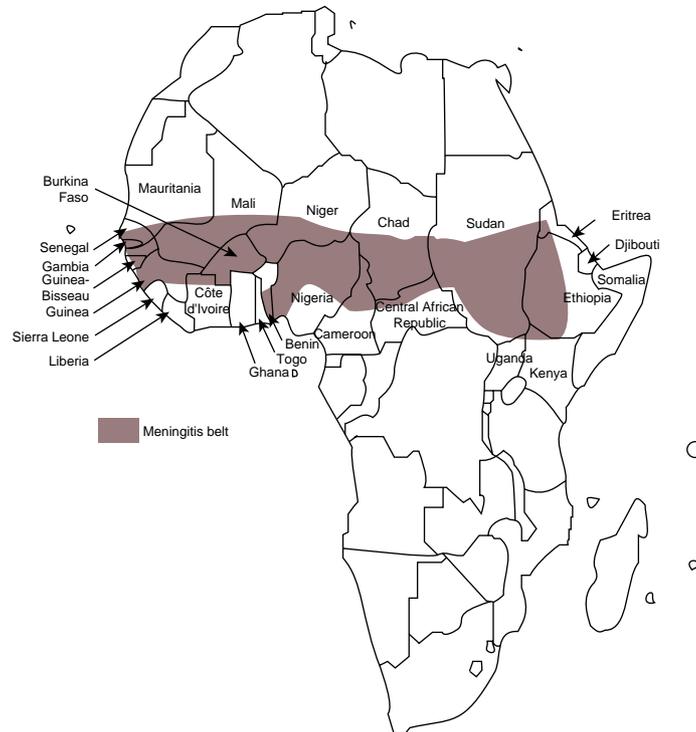


Fig. 1. The classic African meningitis belt.

Source: Control of epidemic meningococcal disease. WHO practical guidelines. 2nd edition 1998. WHO/EMC/BAC/98.3. Available from: <http://www.who.int/csr/resources/publications/meningitis/whoemcbac983.pdf>

5–10 years during the last 100 years (38). The first well-described epidemic in Africa was in Nigeria in 1905 (38). The meningitis belt stretches from Ethiopia and the Sudan in East Africa to Mali, Senegal and Guinea in the west (38). The most common cause of these epidemics is *N. meningitidis* serogroup A (37, 38). In the epidemic years of 2002–2003, serogroup A was substituted by serogroup W135 that caused a large epidemic with its main focus in Burkina Faso (39–42), but since then serogroup A has been the dominant pathogen again (38). In the 2002–2003 serogroup W135 epidemic, as well as in a serogroup A epidemic starting in the Sudan and Chad in 1988, it has been demonstrated that one likely source of introduction of new meningococcal bacterial strains in the meningitis belt is pilgrims returning from the Hajj in Saudi Arabia (43–45).

In the last decades, African countries south of the belt have experienced large IMD epidemics and there has been an extension of the belt into countries like Togo, Cameroon, Côte d'Ivoire and Benin (3, 46, 47). In East Africa, Kenya, Uganda and Tanzania have suffered large-scale IMD epidemics (3, 18, 38, 47–49). The health impact of IMD in Africa makes the disease a main focus for interventions by health agencies in the countries of the meningitis belt. During epidemics in the 1990s an incidence of 1,000 cases per 100,000 was seen (24). An estimated 200,000 people were hit by the disease in the serogroup A epidemic of 1996 (32, 49).

Outside the belt

IMD is a global disease seen in most countries of the world. The incidence of the disease is however lower than in the meningitis belt. In Europe it ranges between 0.2 and 14 cases per 100,000 and 0.2–4 per 100,000 in USA (51). In most countries the disease is endemic with small outbreaks, mainly in crowded settings like schools and military establishments and is mainly caused by serogroups B and C (24, 52). Changes in the epidemiology of IMD due to the introduction of the serogroup C conjugate vaccine are expected to be seen in the future (51). Infrequently epidemics of serogroup A meningitis have been seen outside Africa, for example, in China (53), Nepal (54), India (55) and Russia (56).

Climate and invasive meningococcal disease (IMD)

Two main features of the IMD are influenced by climatic factors: the geographical distribution of high disease incidence with large epidemics in the meningitis belt and the seasonality of the disease seen globally. During the last decade, with growing interest in the effects of climate on health, a number of studies, spatial as well as temporal, of climatic effects on IMD have been conducted and models for predicting epidemics have been proposed. The majority of these studies have concerned IMD in Africa (3, 50, 58–62).

Geographical distribution

Lapeyssonnie described the boundaries of the meningitis belt as equivalent to the annual rainfall isohyets of 300 mm in the north, and 1,100 mm in the south, thereby indicating that climatic factors are involved in the geographical distribution (37). Subsequent research in the meningitis belt have found that the optimal climate for transmission of the disease is the savannah climate south of the Sahel, with an annual precipitation index of 300–1,100 mm, extremely dry but warm winter seasons and a relatively abrupt onset of the rainy season (3, 57). During the last decade climate research concerning the geographical distribution of IMD in Africa has made important progress and has resulted in risk mapping models.

Risk mapping

In a study by Molesworth and colleagues (3) in 2002, the spatial distribution of IMD epidemics in Africa occurring between 1980 and 1999 was mapped using a dataset with published and unpublished epidemics and surveillance data of number of cases reported to the WHO (Fig. 2). Maps showing the location and maximum incidence rates of 144 epidemics in Africa were constructed. The maps demonstrated that the risk of IMD epidemics is almost as high in many geographical areas south of the meningitis

belt, like the Rift Valley and the Great Lakes region, but that the maximum number of cases is higher in the countries in the belt as compared to countries outside the belt. In accordance with the suggested boundaries of the meningitis belt proposed by Lapeyssonnie 40 years previously, the risk map also demonstrated a striking association between IMD epidemics and the 300–1,100 rainfall isohyets in all of Africa, also outside the belt.

The risk models constructed by Molesworth and colleagues have been further developed to investigate the environmental factors driving the IMD epidemics in Africa. In a study by Molesworth and colleagues published in 2003 (58), they analysed the same IMD epidemic and surveillance data as in (3) together with information on climate variables like absolute humidity, dust and rainfall, and data on land-cover type and population density. They found that absolute humidity and land-cover type were the climatic factors that best correlated to IMD epidemics. Among other factors found to be independently associated with IMD epidemics, dust was especially interesting as the dustiness in the meningitis belt increased dramatically due to the Sahelian droughts of the 1970s and 1980s (58). The model also demonstrated that climate zones not having distinct wet and dry seasons, such as deserts and the humid and often

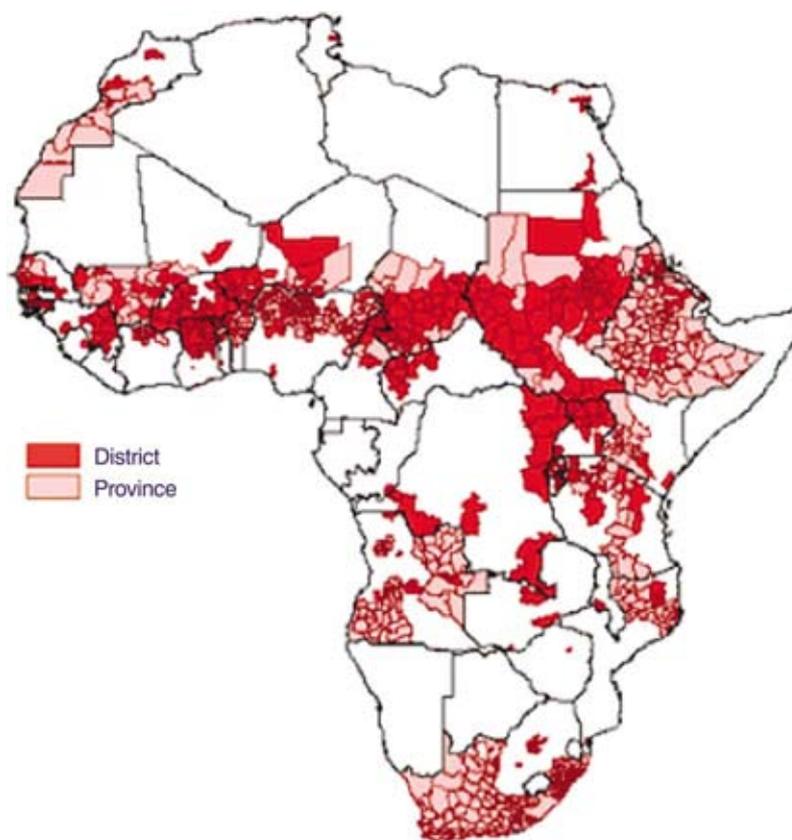


Fig. 2. Risk map for IMD. Districts and provinces experiencing meningococcal meningitis epidemics in Africa 1980–1999. After Molesworth et al. (3). Available from: http://www.liv.ac.uk/researchintelligence/issue15/graphics/15/meningitis_01.gif

forested parts of coastal and central Africa, are less likely to have epidemics than those with contrasting seasons like the semi-arid savannah and grassland found in the Sahel and eastern and southern Africa.

The risk model of Molesworth and colleagues was evaluated in a prospective study of IMD epidemics in Africa 2002–2004 by Savory and colleagues (59). They found that only 59% of the 71 epidemics that occurred during the time period were located within the meningitis belt. Most of the new epidemic districts were however located in areas geographically contiguous to the belt. The epidemics in the meningitis belt had a significantly higher mean number of cases than epidemics outside the belt. The authors concluded that there is an extension of the meningitis belt particularly into districts in Côte d'Ivoire, Togo, the Central African Republic and Cameroon (59). The risk mapping of IMD provides a tool for priority planning of vaccination campaigns, for further research on climate effects on IMD in Africa and a model that can be suitable for the development of early warning systems (EWS) for the disease. It will also be an important instrument for surveillance of the impact of future climate change (57).

Seasonality

Lapeyssonnie also described the seasonality of the disease, with the peak of epidemics during the dry winter season. Frequently the epidemics ceased when the rainy season started, to be resumed in the dry season of the next year (37, 63, 64).

Subsequent studies have suggested low absolute humidity (58) and the dry Harmattan winter winds (62) as the main climatic driver behind the seasonality in the meningitis belt. Sultan and colleagues found a strong correlation between the maximum Harmattan wind index and the onset of IMD epidemics in a study in Mali during 1994–2002 (62). They concluded that the seasonal rise in meningitis cases corresponded to large-scale atmospheric phenomena associated with the Sahelian dry season (62).

The peak of the IMD epidemics comes when the absolute humidity is at its lowest and the epidemics subside with rising humidity before the annual rain period begins (28). In a study in Niger, Jackou-Boulama and colleagues (65) found a negative correlation between rainfall and IMD incidence. The IMD incidence fell when the rain season began. In contrast, incidence of meningococcal carriage has been found to rise with increasing atmospheric humidity in a study by Mueller and colleagues (66) where they investigated carriage rates in the non-epidemic year of 2003 in Burkina Faso. This result stresses the complex role of carriers in the understanding of IMD.

Many countries outside Africa, especially in the Northern hemisphere, show a similar seasonal incidence of the disease as in the meningitis belt (67–71) with peak

incidence during the dry winter months, but the relation between high incidence and low humidity seen in the meningitis belt is not a consistent finding outside the belt. In New Zealand a study showed that the incidence of IMD increased with increasing humidity and cooler temperatures, but declined, as in the meningitis belt, with heavy rain (72). UK studies have shown that IMD outbreaks are correlated with high humidity and rapid changes in relative humidity before outbreaks (73, 74).

The proposed biological explanation for the climate effects on IMD is that low humidity, dry winds and high levels of dust in the air injures the barriers of the URT mucosa, thus facilitating IMD (18, 48). *N. meningitidis* can more easily penetrate injured mucosal membranes and access the blood stream and the meninges, where it causes disease (18). The mechanisms of interaction between *N. meningitidis* and the mucosal epithelial cells are well known (18, 75), but to my knowledge no studies concerning the effects of climatic factors on the pathogenesis and transmission of *N. meningitidis in vivo* have been done, perhaps mainly due to the lack of a reliable animal model for the bacteria (76).

Early warning systems (EWSs)

One of the main aims of EWSs for IMD is to predict epidemics so that mass vaccination can begin in time to curb the spread of disease (57, 59, 61). One tool for identifying epidemics is the IMD case number thresholds presented by WHO as an EWS in the year 2000. It distinguishes between the usual annual rise in IMD incidence and epidemics (77). To find climatic factors that can predict IMD epidemics, Thomson and colleagues investigated a large number of environmental factors in a study in Burkina Faso, Niger, Mali and Togo (61). They found that land-cover type (savannah, grassland and barren areas) was associated with IMD incidence, rainfall and dust. Areas with barren land had the lowest IMD incidence and highest dust levels, and in savannah areas the rainy season started earlier and was more intense than in grassland and barren areas. They could also demonstrate that excess dust in October and a rainfall deficit in January were the best predictors for epidemics. Annual meningitis incidence anomalies (e.g. early cases) at district level were significantly correlated with monthly climate anomalies for rainfall and dust in the pre, post and epidemic seasons, with stronger relationship in savannah areas (57).

The performance of an EWS based on climate indices has recently been investigated in a study by Yaka and colleagues (60). They identified Burkina Faso and Niger as the two countries with the highest risk of IMD epidemics using data from a previously published study by Broutin and colleagues (50). By computing meningitis cases in the two countries with climate variables, they found a significant correlation between IMD incidence

and climatic factors during the winter period, October–January, in Niger. In Burkina Faso the correlation was not significant. This study stresses the importance of including other variables than climate in future EWSs to be able to predict epidemics.

Climate is a driving factor in the seasonality of the disease and in the geographical distribution of IMD in the meningitis belt, but climatic factors alone cannot explain the high disease incidence in the meningitis belt or the periodicity of epidemics. Immunity in the population, carriage rates, vaccination coverage, social interactions and the introduction of new strains of meningococci are other main factors to be considered to fully understand the dynamics of IMD in the meningitis belt (57, 61).

Discussion

The epidemiology of IMD is closely related to climatic factors like air humidity, rainfall and dust (3, 58, 61, 65). The disease is a special challenge to research in the field of disease and climate change as it is spread all over the world and in almost all types of climate zones. The meningitis belt in the Sahel region of Africa has the highest IMD incidence and large epidemics mainly caused by serogroup A meningococci is one of the main health issues for the countries in the belt (30, 78).

Pre-epidemic vaccination against serogroups A and W135, the most common serogroups causing epidemics in the meningitis belt, is feasible, but the vaccine effect only lasts for three to five years, although the new conjugate vaccines now in the pipeline may improve that duration (79, 80). To keep the population constantly immune to the disease is too costly and demands a vaccination infrastructure that does not yet exist in the countries involved. Mass vaccination in the face of a current epidemic is often done too late (81).

In Africa, 350 million people lives in areas at risk for IMD epidemics (57, 58). Only 40 million doses of the new conjugate A vaccine will be available during the first years. Risk models could guide selection of priority areas and demonstrate vaccination efficacy through surveillance. They could also provide support for the control of epidemics in areas where the population has not had access to new vaccines (57).

Meteorological surveillance offers a possibility for developing EWSs for epidemic preparedness (55–59). With the aid of EWSs, vaccination could be made more effective. Attempts at EWSs have been promising but need further refinement (55–57). For future predictive models to be effective, meteorological data must be included in computations together with data on population immunity, changes in population structure and the dynamics of *N. meningitidis* carriers (57, 61). There also needs to be an improvement of disease surveillance data and weather forecasting data in the African continent (57). It is also crucial that health

authorities in the countries at most risk for IMD epidemics are interested in the implementation of EWSs and that economic resources are allocated for them (82, 83, 85). In 2008, the University Corporation for Atmospheric Research (UCAR), working with an international team of health and weather organisations, launched the Meningitis forecast project. The aim of the project is to provide long-term weather forecasts to medical officials in Africa to help reduce outbreaks of meningitis. The forecasts will enable local health providers to target vaccination programmes more effectively (see <http://www.ucar.edu/news/releases/2008/meningitis.jsp>).

Future climate change and invasive meningococcal disease (IMD)

In the 21st century the main projected climate changes in Africa are warming, especially in sub-tropical regions and a decrease in annual rainfall, especially in North Africa and the northern parts of Sahara. By 2080, an increase of 5–8% of arid and semi-arid land in Africa is projected (78). The Sahel, where the meningitis belt is situated, is one of the regions in Africa most vulnerable to climate change. The factors that determine the southern boundary of the Sahara and rainfall in the Sahel have attracted special interest among climate scientists because of the extended drought experienced in this region in the 1970s and 1980s (84). Not only warming and decreased rainfall but also complex feedback mechanisms due to deforestation, land-cover change and changes in atmospheric dust-loading are also playing a role, particularly for drought persistence in the Sahel and its surrounding areas (78).

A likely scenario for the projected increase in temperature and decrease in rainfall is more frequent and longer droughts and thereby a likely increase in the amount of dust in the surface air, together with alterations of atmospheric humidity. These climatic factors will most likely have negative effects on IMD in the meningitis belt, with epidemics of longer duration and maybe also higher incidence. But the effects these climate changes may have on the incidence of meningococcal carriage must also be considered, as carriage incidence plays an important role in the dynamics of IMD epidemics (24, 29, 31). This makes constructing scenarios for IMD more complex, especially as some previous studies have shown negative correlations between carriage and air humidity (66).

Warming and reduced rainfall will also affect the land-cover types in the region with extension of the savannah southwards. Populations at the margin of the current distribution of IMD will be particularly affected (3, 58). The meningitis belt has already expanded (48, 58, 85, 86) and countries south of the belt have suffered from epidemics to an extent not previously reported, for example, in Cameroon (46), Ghana (87) and Togo (88). The population at risk of IMD epidemics is likely to expand, but this prediction is uncertain as droughts

also play a role in human migration and population dislocation (78).

Future research and surveillance

Expansion of the meningitis belt is already evident and is likely to progress with climate change in Africa. Close surveillance of IMD incidence in the areas bordering the meningitis belt, and of other African countries with similar climates, for early detection of climate change effects is therefore of uttermost importance (3). Further development of existing EWSs with inclusion of more demographic data, data on vaccination coverage and natural immunity in the populations and bacteriological surveillance data on meningococcal strains, is necessary for predicting epidemics in time for massvaccination campaigns to be effective (60). Further studies on the relation between climatic factors and IMD, both in the meningitis belt in Africa and in countries outside the belt in the Northern and Southern hemisphere are needed, to be able to refine knowledge on how climate affects both the carrier state and the disease. Of special interest are epidemics outside Africa within the 300–1,100 mm rainfall isohyets (57).

There are differences between different serogroups of meningococci, both in virulence and in their ability to cause epidemics, that need to be investigated to a greater extent to better understand differences in epidemiology between serogroup A in the meningitis belt and other serogroups, mainly C and B in countries outside the belt (24, 26, 27). Molecular biology studies are needed to better understand the effects of climate on the binding and penetration of *N. meningitidis* in mucosal membranes. This includes development of tissue models and animal models for *N. meningitidis*.

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References

- IPCC. Climate change 2007: impacts, adaptation and vulnerability. In: Parry ML, Canziani OF, Palutikof JP, van der Linden PJ, Hanson CE, eds. Contribution of Working Group II to the fourth assessment. Report of the Intergovernmental Panel on Climate Change. Cambridge, UK: Cambridge University Press; 2007, p. 976. Available from: http://www.ipcc.ch/publications_and_data/publications_ipcc_fourth_assessment_report_wg2_report_impacts_adaptation_and_vulnerability.htm [cited 12 September 2009].
- Confalonieri U, Menne B, Akhtar R, Ebi KL, Hauengue M, Kovats RS, et al. Human health. In: Parry ML, Canziani OF, Palutikof JP, van der Linden PJ, Hanson CE, eds. Climate change 2007: impacts, adaptation and vulnerability. Contribution of Working Group II to the fourth assessment report of the Intergovernmental Panel on Climate Change. Cambridge, UK: Cambridge University Press; 2007, pp. 391–431. Available from: <http://www.ipcc.ch/pdf/assessment-report/ar4/wg2/ar4-wg2-chapter8.pdf> [cited 12 September 2009].
- Molesworth AM, Thomson MC, Connor SJ, Cresswell MP, Morse AP, Shears P, et al. Where is the meningitis belt? Defining an area at risk of epidemic meningitis in Africa. *Trans R Soc Trop Med Hyg* 2002; 96: 242–9.
- Patz JA, Epstein PR, Burke TA, Balbus JM. Global climate change and emerging infectious diseases. *JAMA* 1996; 275: 217–23.
- Githeko AK, Lindsay SW, Confalonieri UE, Patz JA. Climate change and vector-borne diseases: a regional analysis. *Bull World Health Organ* 2000; 78: 1136–47.
- Semenza JC, Menne B. Climate change and infectious diseases in Europe. *Lancet Infect Dis* 2009; 9: 365–75.
- Zell R. Global climate change and the emergence/re-emergence of infectious diseases. *Int J Med Microbiol* 2004; 293: 16–26.
- Reiter P. Climate change and mosquito-borne disease: knowing the horse before hitching the cart. *Rev Sci Tech* 2008; 27: 383–98.
- Sáez-Nieto JA, Lujan R, Berrón S, Campos J, Viñas M, Fusté C, et al. Epidemiology and molecular basis of penicillin-resistant *Neisseria meningitidis* in Spain: a 5-year history (1985–1989). *Clin Infect Dis* 1992; 14: 394–402.
- Hedberg ST, Fredlund H, Nicolas P, Caugant DA, Olcén P, Unemo M. Antibiotic susceptibility and characteristics of *Neisseria meningitidis* isolates from the African meningitis belt, 2000 to 2006: phenotypic and genotypic perspectives. *Antimicrob Agents Chemother* 2009; 53: 1561–6.
- du Plessis M, von Gottberg A, Cohen C, de Gouveia L, Klugman KP. *Neisseria meningitidis* intermediately resistant to penicillin and causing invasive disease in South Africa in 2001 to 2005. *J Clin Microbiol* 2008; 46: 3208–14.
- Jorgensen JH, Crawford SA, Fiebelkorn KR. Susceptibility of *Neisseria meningitidis* to 16 antimicrobial agents and characterization of resistance mechanisms affecting some agents. *J Clin Microbiol* 2005; 43: 3162–71.
- Olivares R, Bouyer J, Hubert B. Risk factors for death in meningococcal disease. *Pathol Biol (Paris)* 1993; 41: 164–8.
- Baraff LJ, Lee SI, Schriger DL. Outcomes of bacterial meningitis in children: a meta-analysis. *Pediatr Infect Dis J* 1993; 12: 389–94.
- Dominguez A, Cardeñosa N, Pañella H, Orcau A, Companys M, Alseda M, et al. The case-fatality rate of meningococcal disease in Catalonia, 1990–1997. *Scand J Infect Dis* 2004; 36: 274–9.
- Smith I, Bjørnevik AT, Augland IM, Berstad A, Wentzel-Larsen T, Halstensen A. Variations in case fatality and fatality risk factors of meningococcal disease in Western Norway, 1985–2002. *Epidemiol Infect* 2006; 134: 103–10.
- Veeken H, Ritmeijer K, Hausman B. Priority during a meningitis epidemic: vaccination or treatment? *Bull World Health Organ* 1998; 76: 135–41.
- van Deuren M, Brandtzaeg P, van der Meer JWM. Update on meningococcal disease with emphasis on pathogenesis and clinical management. *Clin Microbiol Rev* 2000; 13: 144–66.
- Smith AW, Bradley AK, Wall RA, McPherson B, Secka A, Dunn DT, et al. Sequelae of epidemic meningococcal meningitis in Africa. *Trans R Soc Trop Med Hyg* 1988; 82: 312–20.
- Pomeroy SL, Holmes SJ, Dodge PR, Feigin RD. Seizures and other neurologic sequelae of bacterial meningitis in children. *N Engl J Med* 1990; 323: 1651–7.
- Al Khorasani A, Banajeh S. Bacterial profile and clinical outcome of childhood meningitis in rural Yemen: a 2-year hospital-based study. *J Infect* 2006; 53: 228–34.
- Schildkamp RL, Lodder MC, Bijlmer HA, Dankert J, Scholten RJ. Clinical manifestations and course of meningococcal disease in 562 patients. *Scand J Infect Dis* 1996; 28: 47–51.

23. Peltola H. Meningococcal disease: still with us? *Rev Infect Dis* 1983; 5: 71–91.
24. Stephens DS, Greenwood B, Brandtzaeg P. Epidemic meningitis, meningococcaemia, and *Neisseria meningitidis*. *Lancet* 2007; 369: 2196–210.
25. Rosenstein NE, Bradley BA, Stephens DS, Popovic T, Hughes JM. Meningococcal disease. *N Engl J Med* 2001; 344: 1378–88.
26. Noah N. Surveillance of bacterial meningitis in Europe 1999–2000. *Eurosurveillance* 2002; 6: pii=2116. Available from: <http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=2116> [cited 11 October 2009].
27. Shigematsu M, Davison KL, Charlett A, Crowcroft NS. National enhanced surveillance of meningococcal disease in England, Wales and Northern Ireland, January 1999–June 2001. *Epidemiol Infect* 2002; 129: 459–70.
28. Greenwood BM, Greenwood AM, Bradley AK, Williams K, Hassan-King M, Shenton FC, et al. Factors influencing susceptibility to meningococcal disease during an epidemic in The Gambia, West Africa. *J Infect* 1987; 14: 167–84.
29. Trotter C, Greenwood B. Meningococcal carriage in the meningitis belt. *Lancet Infect Dis* 2007; 7: 797–803.
30. Roberts L. An ill wind, bringing meningitis. *Science* 2008; 320: 1710–5.
31. Yazdankhah SP, Caugant DA. *Neisseria meningitidis*: an overview of the carriage state. *J Med Microbiol* 2004; 53: 821–32.
32. Greenwood B. Meningococcal meningitis in Africa. *Trans R Soc Trop Med Hyg* 1999; 93: 341–53.
33. Jódar L, LaForce FM, Ceccarini C, Aguado T, Granoff DM. Meningococcal conjugate vaccine for Africa: a model for development of new vaccines for the poorest countries. *Lancet* 2003; 361: 1902–4.
34. Trotter CL, Andrews NJ, Kaczmarski EB, Miller E, Ramsay ME. Effectiveness of meningococcal serogroup C conjugate vaccine 4 years after introduction. *Lancet* 2004; 364: 365–7.
35. Snape MD, Pollard AJ. Meningococcal polysaccharide–protein conjugate vaccines. *Lancet Infect Dis* 2005; 5: 21–30.
36. Siu T, Tang W, Dawar M, Patrick DM. Impact of routine immunization using meningococcal C conjugate vaccine on invasive meningococcal disease in British Columbia. *Can J Public Health* 2008; 99: 380–2.
37. Lapeyssonnie L. La méningite cérébrospinale en Afrique. *Bull World Health Organ* 1963; 28: 3–114.
38. Greenwood B. 100 years of epidemic meningitis in West Africa – has anything changed? *Trop Med Int Health* 2006; 11: 773–80.
39. Decosas J, Koama JB. Chronicle of an outbreak foretold: meningococcal meningitis W135 in Burkina Faso. *Lancet Inf Dis* 2002; 2: 763–5.
40. Nathan N, Rose AM, Legros D, Tiendrebeogo SR, Bachy C, Bjørnløw E, et al. Meningitis serogroup W135 outbreak, Burkina Faso, 2002. *Emerg Infect Dis* 2007; 13: 920–3.
41. Kelly D, Pollard AJ. W135 in Africa: origins, problems and perspectives. *Travel Med Infect Dis* 2003; 1: 19–28.
42. Zombré S, Hacén MM, Ouango G, Sanou S, Adamou Y, Koumaré B, et al. The outbreak of meningitis due to *Neisseria meningitidis* W135 in 2003 in Burkina Faso and the national response: main lessons learnt. *Vaccine* 2007; 25: A69–71.
43. Moore PS, Harrison LH, Telzak EE, Ajello GW, Broome CV. Group A meningococcal carriage in travelers returning from Saudi Arabia. *JAMA* 1988; 260: 2686–9.
44. Salih MA, Danielsson D, Bäckman A, Caugant DA, Achtman M, Olcén P. Characterization of epidemic and non-epidemic *Neisseria meningitidis* serogroup A strains from Sudan and Sweden. *J Clin Microbiol* 1990; 28: 1711–9.
45. Lingappa JR, Al-Rabeah AM, Hajjeh R, Mustafa T, Fatani A, Al-Bassam T, et al. Serogroup W-135 meningococcal disease during the Hajj, 2000. *Emerg Infect Dis* 2003; 9: 665–71.
46. Cunin P, Fonkoua M-C, Kollo B, Bedifeh AB, Bayanak P, Martin PMV. Serogroup A *Neisseria meningitidis* outside meningitis belt in Southwest Cameroon. *Emerg Inf Dis* 2003; 9: 1351–3.
47. Anonymous. Outbreak news. Meningococcal disease, African meningitis belt, epidemic season 2006. *Wkly Epidemiol Rec* 2006; 81: 119–20.
48. Moore PS. Meningococcal meningitis in sub-Saharan Africa: a model for the epidemic process. *Clin Infect Dis* 1992; 14: 515–25.
49. Anonymous. Response to epidemic meningitis in Africa, 1997. *Wkly Epidemiol Rec* 1997; 72: 313–18.
50. Broutin H, Philippon S, Constantin de Magny G, Courel F, Sultan B, Guegan JF. Comparative study of meningitis dynamics across nine African countries: a global perspective. *Int J Health Geo* 2007; 6. DOI: 10.1186/1476-072X-6-29. Available from: <http://www.ij-healthgeographics.com/content/6/1/29> [cited 12 September 2009].
51. Harrison LH, Trotter CL, Ramsay ME. Global epidemiology of meningococcal disease. *Vaccine* 2009; 27: B51–63.
52. Thikomirov E, Santamaria M, Esteves K. Meningococcal disease: public health burden and control. *World Health Stat Q* 1997; 50: 170–7.
53. Wang JF, Caugant DA, Li X, Hu X, Poolman JT, Crowe BA, et al. Clonal and antigenic analysis of serogroup A *Neisseria meningitidis* with particular reference to epidemiological features of epidemic meningitis in the People's Republic of China. *Infect Immun* 1992; 60: 5267–82.
54. Cochi SL, Markowitz LE, Joshi DD, Owens RC Jr, Stenhouse DH, Regmi DN, et al. Control of epidemic serogroup A meningococcal meningitis in Nepal. *Int J Epidemiology* 1987; 16: 91–7.
55. Bhatia SL, Sharma KB, Natarajan R. An outbreak of meningococcal meningitis in Delhi, India. *J Med Res* 1968; 56: 259–63.
56. Achtman M, van der Ende A, Zhu P, Koroleva IS, Kusecek B, Morelli G, et al. Molecular epidemiology of serogroup A meningitis in Moscow, 1969 to 1997. *Emerg Infect Dis* 2001; 7: 420–7.
57. Cuevas LE, Jeanne I, Molesworth A, Bell M, Savory EC, Connor SJ, et al. Risk mapping and early warning systems for the control of meningitis in Africa. *Vaccine* 2007; 25: A12–7.
58. Molesworth AM, Cuevas LE, Connor SJ, Morse AP, Thomson MC. Environmental risk and meningitis epidemics in Africa. *Emerg Inf Dis* 2003; 9: 1287–93.
59. Savory EC, Cuevas LE, Yassin MA, Hart CA, Molesworth AM, Thomson MC. Evaluation of the meningitis epidemics risk model in Africa. *Epidemiol Infect* 2006; 134: 1047–51.
60. Yaka P, Sultan B, Broutin H, Janicot S, Philippon S, Fourquet N. Relationship between climate and year to year variability in meningitis outbreaks: a case study in Burkina Faso and Niger. *Int J Health Geogr* 2008; 7: 34. Available from: <http://www.ij-healthgeographics.com/content/7/1/34> [cited 12 September 2009].
61. Thomson MC, Molesworth AM, Djingarey MH, Yameogo KR, Belanger F, Cuevas LE. Potential of environmental models to predict meningitis epidemics in Africa. *Trop Med Int Health* 2006; 11: 781–8.
62. Sultan B, Labadi K, Guégan J-F, Janicot S. Climate drives the meningitis epidemics onset in West Africa. *PLoS* 2005; 2: 43–9.
63. Greenwood BM, Bradley AK, Wall RA. Meningococcal disease and season in sub-Saharan Africa. *Lancet* 1985; 2: 829–30.
64. Salih MA, Ahmed HS, Karrar ZA, Kamil I, Osman KA, Palmgren H, et al. Features of a large epidemic of group A

- meningococcal meningitis in Khartoum, Sudan in 1988. *Scand J Infect Dis* 1990; 22: 161–70.
65. Jackou-Boulama M, Michel R, Ollivier L, Meynard JB, Nicolas P, Boutin JP. Correlation between rainfall and meningococcal meningitis in Niger. *Med Trop (Mars)* 2005; 65: 329–33.
 66. Mueller JE, Yaro S, Madec Y, Somda PK, Idohou RS, Lafourcade BM, et al. Association of respiratory tract infection symptoms and air humidity with meningococcal carriage in Burkina Faso. *Trop Med Int Health* 2008; 13: 1543–52.
 67. Block C, Roitman M, Bogokowsky B, Meizlin S, Slater PE. Forty years of meningococcal disease in Israel: 1951–1990. *Clin Infect Dis* 1993; 17: 126–32.
 68. Jensen ES, Lundbye-Christensen S, Pedersen L, Sørensen HT, Schönheyder HC. Seasonal variation in meningococcal disease in Denmark: relation to age and meningococcal phenotype. *Scand J Infect Dis* 2003; 35: 226–9.
 69. Michele MM, Alberto M, Liana S, Francesco D. Do environmental factors influence the occurrence of acute meningitis in industrialized countries? An epidemic of varying aetiology in northern Italy. *Eur J Epidemiol* 2006; 21: 465–8.
 70. Fisman DN. Seasonality of infectious diseases. *Annu Rev Public Health* 2007; 28: 127–43.
 71. Kinlin LM, Spain CV, Ng V, Johnson CC, White AN, Fisman DN. Environmental exposures and invasive meningococcal disease: an evaluation of effects on varying time scales. *Am J Epidemiol* 2009; 169: 588–95.
 72. Lindsay AP, Hope V, Marshall RJ, Salinger J. Meningococcal disease and meteorological conditions in Auckland, New Zealand. *Aust N Z J Public Health* 2002; 26: 212–8.
 73. Millon C. When and where are we at risk? The geographical distribution of meningococcal meningitis in England and Wales. BA Thesis, School of Geography, University of Oxford, Oxford, UK, 1983.
 74. Collier CG. Weather conditions prior to major outbreaks of meningococcal meningitis in the United Kingdom. *Int J Biometeorol* 1992; 36: 18–29.
 75. Plant L, Jonsson AB. Contacting the host: insight and implications of pathogenic *Neisseria* cell interactions. *Scand J Infect Dis* 2003; 35: 608–13.
 76. Zarantonelli ML, Szatanik M, Giorgini D, Hong E, Huerre M, Guillou F, et al. Transgenic mice expressing human transferrin as a model for meningococcal infection. *Infect Immun* 2007; 75: 5609–14.
 77. Anonymous. Detecting meningococcal meningitis epidemics in highly-endemic African countries. *WER* 2000; 28: 306–9.
 78. IPCC. Climate change 2007: synthesis report. In: Core Writing Team, Pachauri RK, Reisinger A, eds. Contribution of Working Groups I, II and III to the fourth assessment report of the Intergovernmental Panel on Climate Change. Geneva, Switzerland: IPCC; 2007, p. 104. Available from: http://www.ipcc.ch/publications_and_data/publications_ipcc_fourth_assessment_report_synthesis_report.htm [cited 12 September 2009].
 79. Vogel G. Shortage of meningitis vaccine forces triage in Burkina Faso. *Science* 2003; 299: 1499–501.
 80. LaForce FM, Konde K, Viviani S, Préziosi MP. The meningitis vaccine project. *Vaccine* 2007; 25: A97–100.
 81. LaForce FM, Ravenscroft N, Djingarey M, Viviani S. Epidemic meningitis due to group A *Neisseria meningitidis* in the African meningitis belt: a persistent problem with an imminent solution. *Vaccine* 2009; 27: B13–9.
 82. Armstrong G, Sackey SO, Tetteh C, Bugri S, Perkins BA, Rosenstein NE, et al. Emergency vaccination against epidemic meningitis in Ghana: implications for the control of meningococcal disease in West Africa. *Lancet* 2000; 355: 30–3.
 83. Cox J, Abeku TA. Early warning systems for malaria in Africa: from blueprint to practice. *Trends Parasitol* 2007; 23: 243–6.
 84. Guibourdenche M, Høiby EA, Riou JY, Varaine F, Joguet C, Caugant DA. Epidemics of serogroup A *Neisseria meningitidis* of subgroup III in Africa, 1989–94. *Epidemiol Infect* 1996; 116: 115–20.
 85. DaSilva J, Connor SJ, Mason SJ, Thomson MC. Response to Cox and Abeku: early warning systems for malaria in Africa: from blueprint to practice. *Trends Parasitol* 2007; 23: 243–6.
 86. Varaine F, Caugant DA, Riou JY, Kondé MK, Soga G, Nshimirimana D, et al. Meningitis outbreaks and vaccination strategy. *Trans R Soc Trop Med Hyg* 1997; 91: 3–7.
 87. Leimkugel J, Hodgson A, Forgor AA, Pflüger V, Dangy JP, Smith T, et al. Clonal waves of *Neisseria* colonisation and disease in the African meningitis belt: eight-year longitudinal study in northern Ghana. *PLoS Med* 2007; 4: e101.
 88. Aplogan A, Batchassi E, Yakoua Y, Croisier A, Aleki A, Schlumberger M, et al. An epidemic of meningococcal meningitis in the region of Savanes in Togo in 1997: research and control strategies. *Sante* 1997; 7: 384–90.

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Climate variability and increase in intensity and magnitude of dengue incidence in Singapore

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Introduction: Dengue is currently a major public health burden in Asia Pacific Region. This study aims to establish an association between dengue incidence, mean temperature and precipitation and further discuss how weather predictors influence the increase in intensity and magnitude of dengue in Singapore during the period 2000–2007.

Materials and methods: Weekly dengue incidence data, daily mean temperature and precipitation and the midyear population data in Singapore during 2000–2007 were retrieved and analysed. We employed a time series Poisson regression model including time factors such as time trends, lagged terms of weather predictors, considered autocorrelation and accounted for changes in population size by offsetting.

Results: The weekly mean temperature and cumulative precipitation were statistically significant related to the increases of dengue incidence in Singapore. Our findings showed that dengue incidence increased linearly at time lag of 5–16 and 5–20 weeks succeeding elevated temperature and precipitation, respectively. However, negative association occurred at lag week 17–20 with low weekly mean temperature as well as lag week 1–4 and 17–20 with low cumulative precipitation.

Discussion: As Singapore experienced higher weekly mean temperature and cumulative precipitation in the years 2004–2007, our results signified hazardous impacts of climate factors on the increase in intensity and magnitude of dengue cases. The ongoing global climate change might potentially increase the burden of dengue fever infection in near future.

Keywords: dengue fever; *Aedes aegypti*; weather; mean temperature; precipitation; climate variability; incidence

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Dengue is currently a major epidemiological threat for over 100 countries with about 70% of the 2.5 billion populations at risk living in Asia Pacific region (1, 2). Exact reasons for the re-emergence are not fully comprehended; nevertheless, the US Centers for Disease Control and Prevention has identified factors such as accelerated urbanisation and population growth, weakened public health infrastructure, increased international travel and lack of effective vector control system as main determinants for global emergence of dengue (3).

Dengue is caused by four serotypes of viruses (DENV-1–4) transmitted by female *Aedes aegypti* (principal vector) and *Aedes albopictus*. *A. aegypti* is highly domestic, feeds in the day and lays eggs in artificial containers in and around residential areas. *A. aegypti* may feed on several individuals in order to complete a single blood meal; thus, increase number of infected hosts

in a short period (4). Upon being bitten by a dengue infective mosquito, a host may be infected with dengue virus after 3–14 days of intrinsic incubation period. The host then goes through 2–10 days of viraemic period, during which a female mosquito becomes infected by feeding blood meal on the infective host. The transmission cycle continues after extrinsic incubation period (8–12 days) takes place in the female mosquito and dengue virus is transmitted to other uninfected hosts through the bites of an infective mosquito (4). A patient infected with one serotype of dengue virus will be immune for that serotype, but can be infected again with other serotypes and face higher risk of haemorrhagic symptoms which could lead to systemic failure or death (4).

Studies have shown that ambient temperature could have impact on population size, maturation period, feeding characteristics and survival rate of *Aedes*

mosquitoes (5, 6). Mortality rate of larvae, pupae and adult female mosquitoes as a function of temperature between 10 and 40°C can be represented by a wide-base 'U' graphical shape. At temperature ranging from 15 to 30°C, *Aedes* mosquitoes experience lower mortality rate (5). A recent study in French Guiana indicated that *Aedes* mosquitoes could survive up to 76 days with favourable environment condition (7). Simultaneously, *Aedes* mosquitoes experience shorter reproductive cycle at higher temperature of 32°C and increase feeding frequency more than twofold as compared to temperature at 24°C; pupae development period may reduce from four days at 22°C to less than one day as temperature increases to 32–34°C; thus, mosquito population multiplies swiftly as temperature increases (6). According to a study by Tun-Lin et al., female and male ratio of mosquito offspring could be 4:3 at 30°C (8). Additionally, the extrinsic incubation period of dengue viruses shortens from 12 days at 30°C to seven days as temperature rises to 32–35°C (9). Conversely, heavy precipitation destroys larvae and reduces survival rate of female mosquitoes in the short period but creates abundant breeding sources in long term (7, 10); whereas extremely low precipitation increases ambient temperature, usage of water and air-coolers and water storage that may serve as breeding habitats.

Dengue is endemic in Singapore with all four serotypes of dengue viruses. It was first reported in Singapore in the early 1960s (11), but was curbed through effective vector surveillance and control programmes, public education and law enforcement since 1970s. Dengue re-emerged in the 1990s with increasing incidence rate though overall house index was less than 2% (12). In 2003, dengue cases increased more than tenfold with 108.5 cases per 100,000 populations as compared to 9.3 cases per 100,000 populations in 1988. The surge continued and Singapore experienced the worst historical outbreak of dengue incidence in year 2005 with 326.5 cases per 100,000 populations (13, 14). Besides marked increase in cases, geographical expansion of dengue incidence from the east and southeast zones to west and north zones was also noted since 2004 (14). A study in 2006 revealed that increasing outdoor dengue transmission, lower herd immunity and shift of vector control emphasis were some possible reasons for the upsurge of dengue (12).

Singapore had implemented aggressive vector control programmes since early 1970s. However, there was a change in emphasis from vector surveillance to case detection in the 1990s (12). Upon case detection, National Environment Agency (NEA) would immediately deploy dengue control team(s) to perform source reduction, adulticidal fogging and various prevention activities according to the standard operation procedure. In 2003, Geographical Information System was added to enhance analysis of distribution of dengue cases, *Aedes* mosquitoes and weather data. Since the 2005 outbreak,

NEA had revised its strategy and put in place integrated dengue surveillance and control programme (15, 16). The programme involved active vector surveillance; vector control; public education and community involvement; as well as research and development. In late 2005, NEA rolled out nationwide 'carpet combing' operations to eliminate breeding sources in all constituencies and established an inter-agency dengue task force to enhance communication and corporation between government agencies and private organisations. National vector surveillance and control system were further strengthened in December 2005 to combat the upsurge in dengue incidence (15, 16).

In view of global climate change projections, the future climate may be favourable for greater dengue outbreaks; especially in subtropical and temperate countries. It was estimated that global mean surface temperature would be rising approximately by 1.4–5.8°C in current century; whilst temperature for Asia Pacific region would be increasing around 0.5–2°C by 2030 and 1–7°C by 2070 (17, 18). Furthermore, small island state such as Singapore could be more inclined to experience extreme temperature and changes in rainfall which in turn had additional impact on increase of infectious diseases (19). Nevertheless, the burden of climate change regarding dengue incidence in Singapore is so far uncertain.

Though association between weather and dengue incidence in Singapore is well documented (20, 21), the study on how the association influences the increasing intensity and magnitude of dengue incidence in recent years, especially years 2004–2007, is yet to be established. In this study we apply time series model to establish association between dengue incidence and weather predictors while taking into account the delayed effects of exposure and further discuss how these predictors are associated with increasing intensity and magnitude of dengue cases in Singapore in years 2000–2007.

Materials and method

Study area

Singapore is a highly urbanised small tropical island with approximately 710.2 km² land area. The island has population size of about 4.8 millions and population density of 6,814 persons per km² (22). Average daily minimum and maximum temperature in Singapore ranged from 23.2 to 31.7°C with daily mean temperature around 27°C; however, daily minimum temperature could drop to 20.2°C or rise to maximum 36°C in an extreme cold or hot day. The average annual rainfall was around 2,346 mm (23).

Data collection

Weekly dengue data were collected from the Communicable Diseases Division, Ministry of Health Singapore

and MOH weekly epidemiological publications for year 2000–2007. Dengue fever (DF) or dengue haemorrhagic fever (DHF) was defined as clinical dengue cases with confirmed laboratory blood tests of dengue viral infection of any serotypes. Under the Infectious Diseases Act in Singapore, it is mandatory for all medical practitioners to notify all diagnosed or confirmed dengue cases to the Ministry of Health within 24 hours (24). Laboratories are also required to report all patients with positive blood test for dengue viral infection to the Ministry of Health.

Weather data were obtained from World Data Center for Meteorology, Asheville. Daily mean temperature and precipitation reported by Changi meteorological station was extracted from the World Meteorological Organization (WMO) and NOAA National Climatic Data Center (25). The meteorological data were provided by the Meteorological Services Division of NEA of Singapore through Exchange of Regional Weather Data by the use of Global Telecommunication System (GTS) of WMO. Weekly mean temperature and cumulative precipitation were aggregated from daily weather data. Midyear population during the period 2000–2007 was obtained from Statistics Singapore’s website (26).

Statistical analysis

We established a time series Poisson regression model that simultaneously included time factors such as time trend, lagged terms of weather predictors, lags of dengue cases as auto regressive terms and we accounted for changes in size of the population by offsetting midyear population. We modelled the predictors as smooth cubic spline functions given 3 degrees of freedom (*df*) each, with exception for the smooth function of trend that was allowed 6 *df*. We tested the sensitivity of the *df* of the trend by doubling it. In the Poisson regression models we allowed for over-dispersion.

$$Y(t) \sim \text{Poisson} (\mu(t))$$

$$\begin{aligned} \text{Log} (\mu(t)) = & \beta_0 + \log(\text{pop}_t) + \beta_1 \text{AR}(\text{den}_t) \\ & + \sum_{i=1}^5 (S[\text{temp}_i, df] + S[\text{prep}_i, df]) \\ & + S(\text{trend}, df) \end{aligned}$$

where β = parameter estimates; t = time series in weeks; $\log(\text{pop})$ = offset midyear population; $\text{AR}(\text{den})$ = auto regressive term of dengue cases; S = cubic spline smoothing function with corresponding degree of freedom (*df*); temp_i = weekly mean temperature at specific lag strata, i ; prep_i = weekly cumulative precipitation at specific lag strata, i ; where i corresponds to 1–5 lag strata = week 1–4, 5–8, 9–12, 13–16, 17–20; trend corresponds to week number starting from the first week in year 2000.

Midyear population was included as an offset to adjust for annual population growth or decay in the modelled relative risk. Whereas auto regressive terms ranging from

1 to 8 weeks were estimated by summing average duration of incubation period in infected person, infectious period of host and survival period of female *Aedes* mosquitoes (4, 7). Concurrently, lag terms ranging from 1 to 20 weeks for temperature and precipitation were created to analyse relative risks between weather predictors and dengue with effect of different time lag. Cross-correlation coefficients of each weather variable and dengue cases as well as literature reports were examined to estimate maximum lag terms (21, 27). Trend and seasonality pattern in collected data were identified by using time series plot of dengue cases and to be controlled as an unmeasured confounders by the smooth function of time trend.

Model fit was evaluated by Akaike’s Information Criterion (AIC) and further validated by plotting predicted residuals against observed data, observing residual sequence plot and analysing normality tests. Furthermore, Autocorrelation (ACF) and partial autocorrelation (PACF) were evaluated to avoid confounding of the risk estimates by unknown sources and shrinking of the variance associated with parameter estimates. To account for this, we modelled auto regressive terms. PACF was also examined to avoid over fitting (which could occur if allowing the trend too much flexibility) signalled by extremely high proportion of negative PACF. Data were analysed using R2.8.1 (28).

Results

During the study period, dengue cases (Fig. 1) increased from 673 cases in year 2000 to 4,789 cases in 2003, after which, annual dengue cases increased with greater magnitude and intensity with record of 9,459 cases in year 2004 and peaked in year 2005 with 14,209 cases. The incidence then decreased to 3,131 cases in 2006 and resurged in 2007 with 8,848 cases. The highest dengue cases in the study period were reported in week 38 of year 2005 with 714 cases.

Fig. 2 reveals time series of weekly weather predictors and dengue cases. Singapore experienced the highest

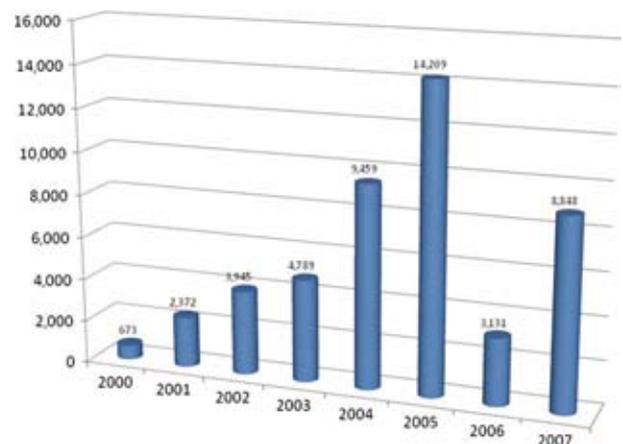


Fig. 1. Annual dengue cases in years 2000–2007.

weekly mean temperature of 30.4°C in year 2005 followed by 30.3°C in year 2002. The highest weekly mean temperature in the study period was recorded in week 17 of year 2005 with zero weekly cumulative precipitation. While the longest dry spell of 40 days occurred in the first two months of 2005, highest weekly cumulative precipitation was recorded as 388 mm in week 51 of year 2006. Overall, weekly mean temperature for year 2000–2003 ranged from 25.7 to 30.3°C; while the range was 25.5–30.4°C for 2004–2007. Concurrently, weekly cumulative precipitation in years 2000–2003 ranged from 0 to 260 mm with average 42 mm and the range was 0–388 mm with average 44.5 mm for years 2004–2007.

As shown in Fig. 3, dengue incidence increased linearly with weekly mean temperature at lag strata 5–8, 9–12 and 13–16; during which the highest increases in relative risk occurred at time lag of 9–12 weeks. Our results showed that decreasing mean temperature below 27.8°C produced opposite effect by increasing relative risk of dengue incidence significantly at lag strata 17–20; while relative risk of dengue increased minimally when mean temperature was above 27.8°C at the same strata. Simultaneously, dengue incidence was associated with weekly cumulative precipitation except with precipitation above 125 mm at lag strata 1–4, 5–8 and 9–12 (Fig. 4). The impact of cumulative precipitation on dengue incidence was negative at lag strata 1–4 as decreasing precipitation corresponded to increasing relative risk with maximum effect at zero weekly cumulative precipitation. Nevertheless, relative risk of dengue increased linearly with weekly cumulative precipitation at lag strata 5–8 and 9–12 and peaked at about 75 mm before declining. Increase in cumulative precipitation showed highest relative risk of

dengue incidence at lag strata 13–16 with a plateau effect when cumulative precipitation was between 75 and 150 mm. Risk function of dengue and cumulative precipitation was shown as a ‘U’ shape at lag strata 17–20. The relative risk of dengue increased linearly when cumulative precipitation was above 75 mm; nevertheless, the risk function indicated opposite effect as cumulative precipitation was below 75 mm.

Post estimation plots indicated good fit of Poisson regression model for the analysis. The model explained 89.9% of the variance in weekly dengue cases and produced good fit of predicted cases when plotted against observed data (Figs. 5 and 6). Residuals sequence plot indicated constant location and scale between observed and predicted values; while histogram of residuals showed approximately normal distribution. Concurrently, ACF and PACF indicated independent residuals. Thus the graphs indicated appropriate choice of model. Furthermore, sensitivity test of df for trend changed little of our results.

Discussion

Though elevated weekly mean temperature preceded dengue incidence at lag of 5–16 weeks, temperature decreasing below 27.8°C exerted increasing risks on dengue incidence at longer time lag of 17–20 weeks. On the other hand, weekly cumulative precipitation was associated with dengue incidence at lag of 1–20 weeks. Decreasing weekly cumulative precipitation posed increasing risk on dengue outbreak at time lag of 1–4 and 17–20 weeks; whereas highest relative risk occurred at 13–20 weeks subsequent to cumulative precipitation above 150 mm.

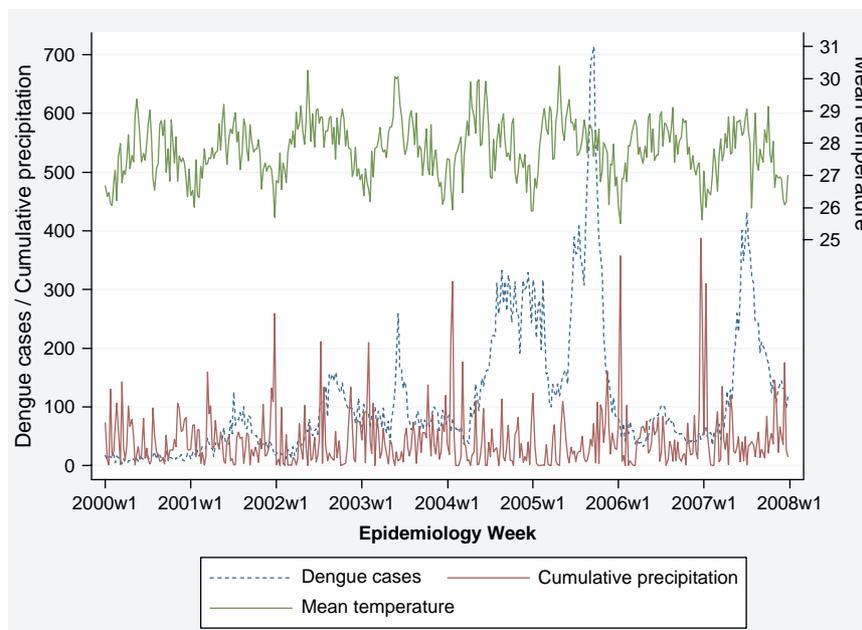


Fig. 2. Weekly dengue cases, mean temperature and cumulative precipitation in 2000–2007.

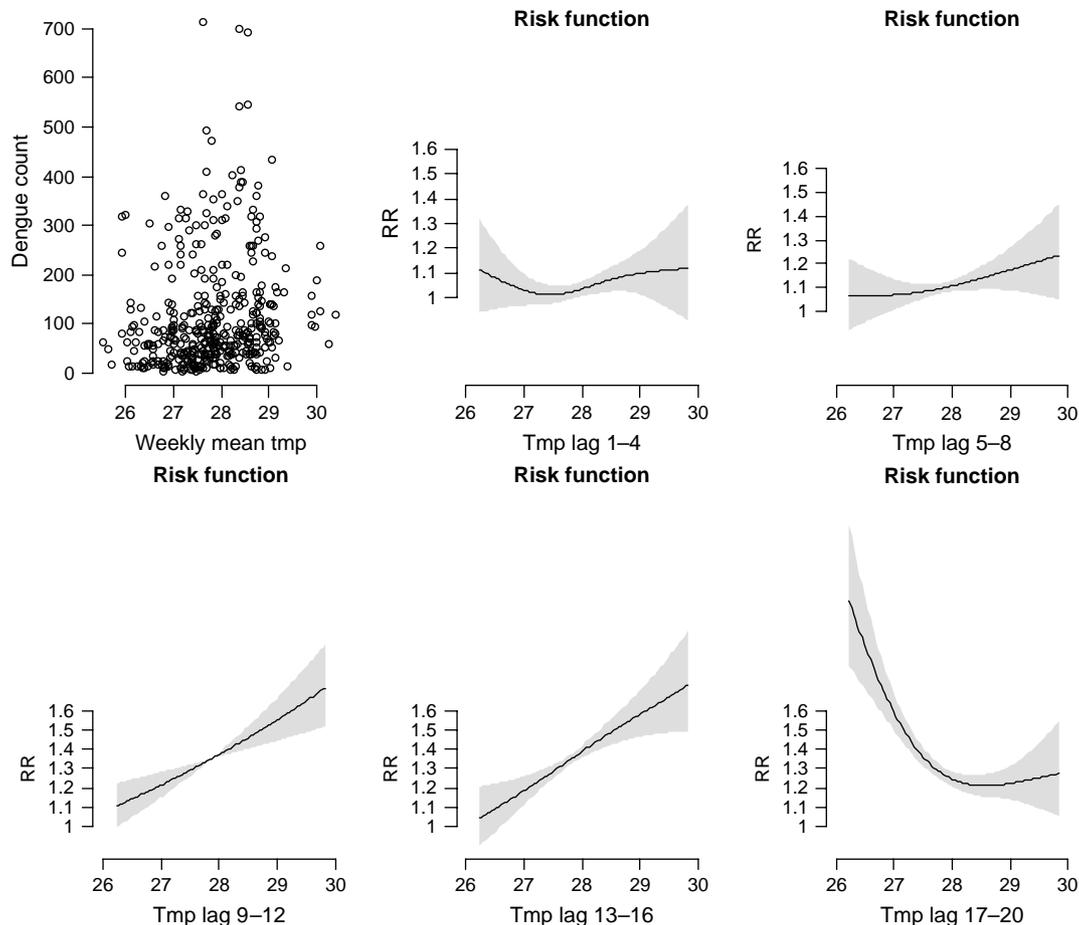


Fig. 3. Relative risks of dengue incidence with weekly mean temperature (Tmp) at various lagged strata.

As Singapore experienced higher weekly mean temperature and cumulative precipitation in year 2004–2007, the results signified positive impact of climate factors on the increase in intensity and magnitude of dengue cases; since the levels of temperature and precipitation were more conducive in these years according to the exposure response relationship estimated. Additionally, high cumulative precipitation during the study period possibly explained a fraction of dengue endemic in Singapore as heavy precipitation created abundant outdoor breeding sources. A previous study by Ooi et al. (12) suggested viral transmission outside homes as one of the reasons for resurgence of dengue in Singapore; while Koh et al. (2008) also stated that 50% of all mosquito breeding habitats, which included discarded receptacles, choked drains, water pump rooms, were located outdoor (20). However, field surveillance data showed that *A. albopictus* was the main species which bred in habitats located outdoor; while the primary vector, *A. aegypti*, which was responsible for majority outbreaks in Singapore, still preferred to breed inside homes. Literature also indicated that *A. albopictus* probably served as a vector maintaining the dengue virus in South-east Asia and Pacific Islands

(29). Nevertheless, Singapore has put in place control measures targeting at both types of breeding habitats to minimise transmission by both species.

The long time lag of 17–20 weeks was consistent with a study by Koh et al. that dengue incidence in year 2005 was highly associated with weekly mean temperature at a lag time of 18 weeks as well as study by Heng et al. that dengue incidence occurred at a lag time of 8–20 weeks following elevated temperature (20, 21). The lagged effect of dengue incidence could be reasoned by incubation periods in host–vector–pathogen transmissions cycle plus reproduction, maturation and survival rate of vector.

This study adopted an approach that allowed the effect of weather to act non-linearly as exposure response function; thus enhanced the flexibility to model risks with time on a weekly resolution. In the analysis we assumed there was no strong seasonal confounding that needs to be controlled, except time trend. This judgment was based on deduction that no further confounder was identified and that PACF suggested over fitting when adjusting for seasonality. Furthermore, auto regressive terms included in the model explained some of such variation. The examination of ACF indicated serial

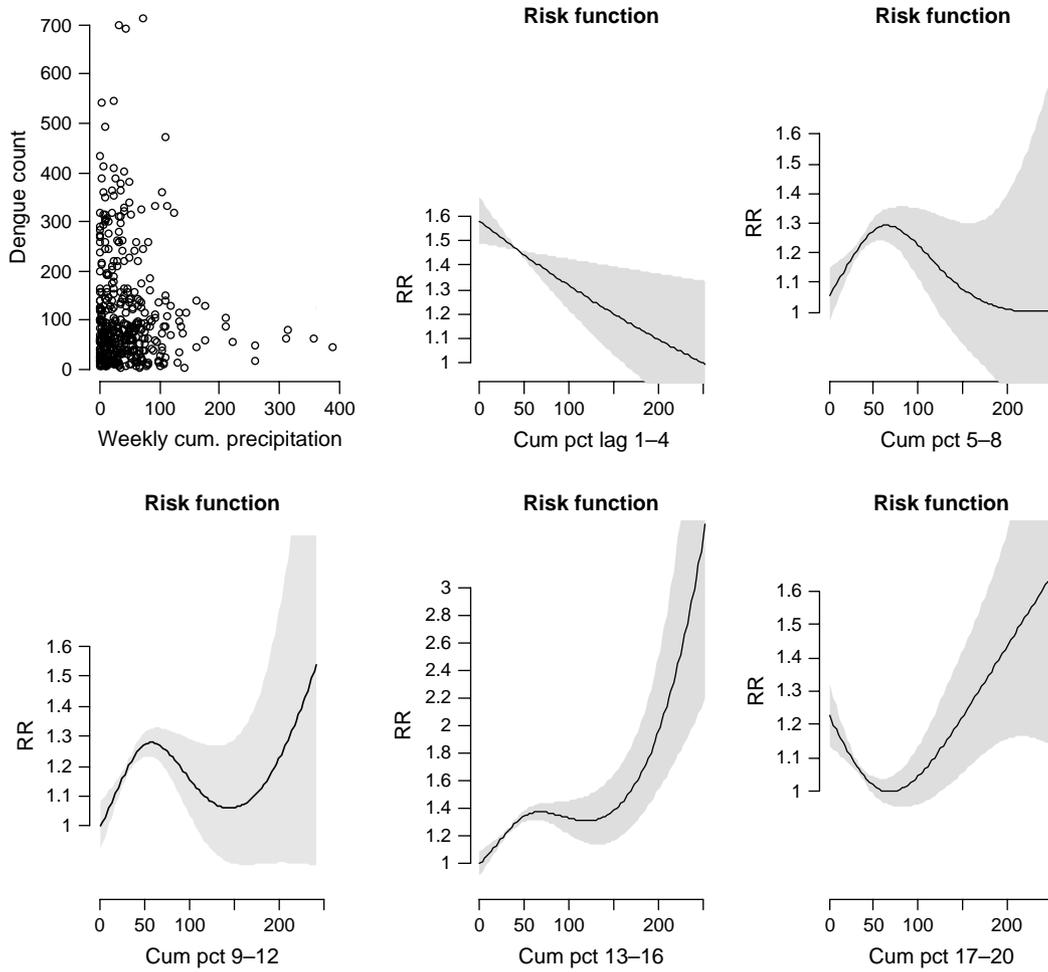


Fig. 4. Relative risks of dengue incidence with weekly cumulative precipitation (Cum pct) at various lagged strata.

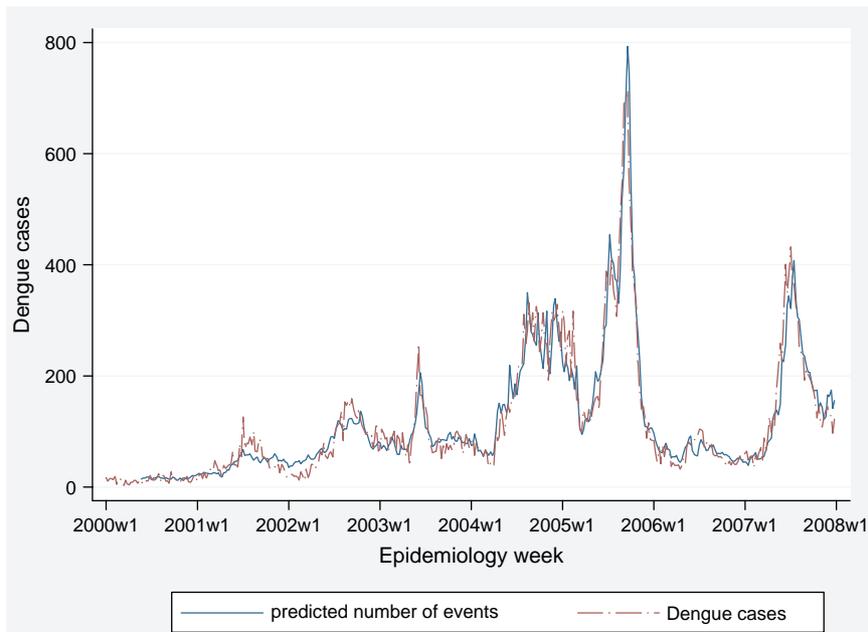


Fig. 5. Predicted cases vs. observed dengue cases in 2000–2007.

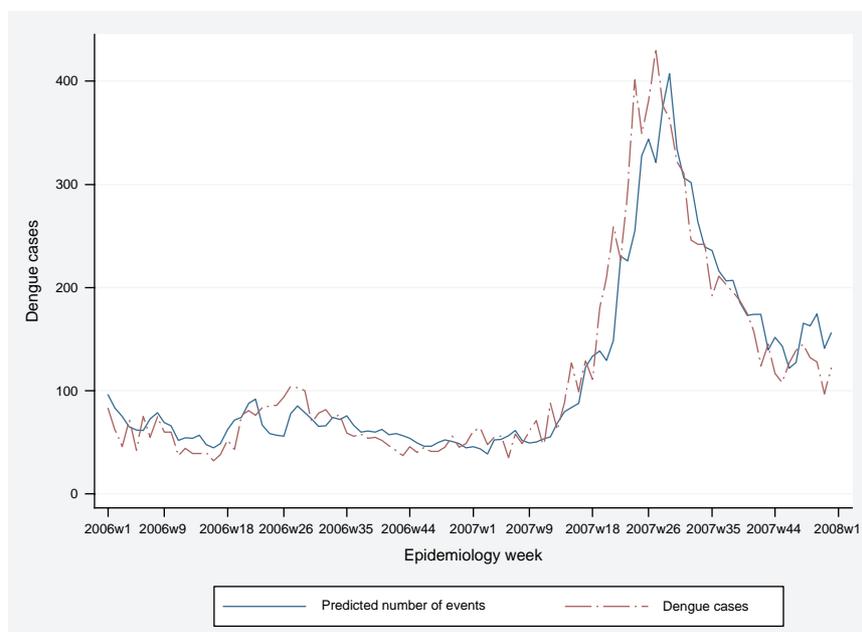


Fig. 6. Predicted cases vs. observed dengue cases in 2006–2007.

correlation for consecutive lags of dengue cases; thus, dengue lag terms were used as autoregressive term in the model. The seasonality of the mosquito population was likely to be driven by temperature and precipitation and the high level of explained variation of the model supported this.

Instead of modelling the mosquito population, we addressed the incidence directly and estimated dengue cases caused by the size of the vector population related to climate factors such as temperature and precipitation. Elements not explained by weather predictors were explained by the trend function. Determinants of the trend function had not been addressed in this study, but were likely attributed to multiple factors such as vector control capacity, surveillance emphasis, herd immunity, socioeconomic, environment, change of dengue serotypes, etc. According to a previous study, non-climate factors such as changes of dengue serotypes partly contributed to the increase in dengue cases during the period 2004–2007 (20). The high cases in 2004 and 2005 were due partly to a change in dengue serotype from DENV-2 to DENV-1; while the change from DENV-1 to DENV-2 in 2007 was also one of the reasons for resurgence of dengue incidence in the year (20). Another factor accounted for in the trend function was the enhancement of pre-emptive vector surveillance during the inter-epidemic periods and inter-sectoral collaborations to conduct systematic source reduction. Since 2006, the NEA (Singapore) has introduced Intensive Source Reduction Exercises over a period of 12 weeks before the expected peak in cases in accordance to the temperature trend. These measures might, to certain extent, contribute to the reduction of dengue cases in year 2006 and

help to curb 2007 dengue cases from escalating into a huge outbreak.

Climate variability between years may have direct impact on dengue incidence in current and subsequent year (30–32). Inter-annual climate variability can be influenced by local weather and irregular warming of sea surface temperature (El Niño Southern Oscillation or ENSO phenomenon). During the study period, average weekly mean temperature was higher in years 2002, 2004 and 2005; simultaneously, ENSO phenomenon was recorded in May 2002–Mar 2003, Jun 2004–Feb 2005 and Aug 2006–Jan 2007 (33). This inter-annual climate variability could have increased relative risk of dengue incidence in Singapore as record showed larger dengue outbreak in years 2004, 2005 and 2007.

In order to avoid camouflage of temporal variations of weather, it is also essential to interpret our findings by analysing both short and long-term weather variations. For instance, year 2005 experienced higher temperature and lower cumulative precipitation in the first 17 weeks as compared to subsequent weeks of the year; thus, relative risk for dengue incidence was higher in second and third quarter as compared to average relative risk for the year. Moreover, as a highly urbanised small island state, Singapore faces threat of urban heat island effect along with extreme temperature and precipitation events which may compound the exponential increase in dengue cases (19, 34, 35).

Limitations in the study of dengue incidence include asymptomatic and unreported cases that lead to under-reported dengue cases. In a survey on seroprevalence in Singapore (2007), it was estimated that asymptomatic cases could be approximately 19 times higher than

reported cases (36). The under-reported cases could surge higher during peak tourism period(s) as tourists from regional countries with asymptomatic dengue infection may infect mosquitoes in Singapore 'silently' and cause greater threat of dengue outbreak in near future.

Climate variability in dissimilar geographical areas may have diverse local effects on dengue outbreak as both climate and non-climate variables influencing dengue transmission may be unique in different locations (37). For instance, a study in Southern Thailand showed relative importance of weather predictors varied with geographical areas. The study indicated mean temperature, rainfall and relative humidity were associated with dengue haemorrhagic fever (DHF) in provinces along Andaman Sea border; while minimum temperature, number of rainy days and relative humidity were associated with DHF in provinces along the Gulf of Thailand border (38). Thus, spatial and transmission heterogeneity secondary to unique local factors may limit generalisation of the study result from one city to another.

Given that temperature and precipitation in Singapore are conducive for increasing vector population size, biting and infective rate and that silent transmission may occur in community, strong health policies that support active dengue surveillance and regular breeding source reduction exercises are vital for effectiveness and sustainability of dengue prevention efforts. As dengue outbreaks are surging in Asia Pacific and dengue viruses are spreading among nations in the region through international travel and trade, the risk of dengue outbreaks in Singapore will possibly be intensified. Therefore, further studies concerning impact of climate factors on both regional and local dengue incidence are essential for analysis of geographical dengue distribution and transmission patterns so as to facilitate formulation of regional adaptation and prevention strategies accordingly.

Conclusion

In view of global warming, efforts to strengthen dengue control programmes and public health interventions are inevitable in order to avert upsurge of dengue incidence which predictably increases national health burden and economic losses. The finding of this study reveals potential transmission of dengue disease as a result of climate variability with possible time lag up to 20 weeks. Currently NEA conducts intensive vector control measures 12 weeks before the anticipated rise in dengue cases based on temperature trending, during which public, government agencies and other stakeholders will be alerted to participate in preventive measures. Nevertheless, the additional use of extreme low or high weekly cumulative precipitation will further enhance the predictive model of dengue transmission and improve the effectiveness of current vector control programmes. A key to eradicate dengue from community is commitment and

participation from individual population; therefore, integrating climatic factors into national dengue prevention/education programmes will help to create public awareness and enable individuals to take necessary precaution.

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References

1. World Health Organization. Dengue and dengue haemorrhagic fever. Fact sheet No. 117. WHO. Available from: <http://www.who.int/mediacentre/factsheets/fs117/en/>; 2009 [cited 22 March 2009].
2. World Health Organization Regional Office for the Western Pacific. Dengue in the Western Pacific region. WHO/WPRO. Available from: http://www.wpro.who.int/health_topics/dengue/; 2009 [cited 4 April 2009].
3. Division of Vector-Borne Infectious Diseases. Dengue fever. Centers for Disease Control and Prevention. Available from: <http://www.cdc.gov/ncidod/dvbid/dengue/index.htm>; 2008 [cited 21 March 2009].
4. Gubler DJ. Dengue and dengue hemorrhagic fever. *Clin Microbiol Rev* 1998; 11: 480–96.
5. Yang HM, Macoris MLG, Galvani KC, Andrighetti MTM, Wanderley DMV. Assessing the effects of temperature on the population of *Aedes aegypti*, the vector of dengue. *Epidemiol Infect* 2009; 137: 1188–202.
6. Focks DA, Brenner RJ, Hayes J, Daniels E. Transmission thresholds for dengue in terms of *Aedes aegypti* pupae per person with discussion of their utility in source reduction efforts. *Am J Trop Med Hyg* 2000; 62: 11–8.
7. Fouque F, Carinci R, Gaborit P, Issaly J, Bicout DJ, Sabatier P. *Aedes aegypti* survival and dengue transmission patterns in French Guiana. *J Vector Ecol* 2006; 31: 390–9.
8. Tun-Lin W, Burkot TR, Kay BH. Effects of temperature and larval diet on development rates and survival of the dengue vector *Aedes aegypti* in north Queensland, Australia. *Med Vet Entomol* 2000; 14: 31–7.
9. Watts DM, Burke DS, Harrison BA, Whitmire RE, Nisalak A. Effect of temperature on the vector efficiency of *Aedes aegypti* for dengue 2 virus. *Am J Trop Med Hyg* 1987; 36: 143–52.
10. National Environment Agency. NEA: dengue control more successful when the community is involved. Singapore: National Environment Agency; 2004.
11. Chew A, Ho Y, Lee YK. A haemorrhagic fever in Singapore. *Lancet* 1961; 1: 307–10.

12. Ooi E-E, Goh K-T, Gubler DJ. Dengue prevention and 35 years of vector control in Singapore. *Emerg Infect Dis* 2006; 12: 887–93.
13. Singapore Ministry of Health. Communicable diseases surveillance in Singapore 2006. Singapore: Ministry of Health; 2007.
14. Singapore Ministry of Health. Report of the expert panel on dengue [monograph on the Internet]. Available from: http://www.moh.gov.sg/mohcorp/uploadedfiles/News/Current_Issues/2005/Oct/Final_Report-dengue_7_Oct_05.pdf; 2005 Oct 7 [cited 6 March 2009].
15. Singapore Ministry of Health. Surveillance and control of dengue vectors in Singapore. *Epidemiol News Bull* 2006; 32: 1–9.
16. Singapore Ministry of Health. Dengue surveillance in Singapore, 2005. *Epidemiol News Bull* 2006; 32: 9–14.
17. Kovats S, Ebi KL, Menne B, Campbell-Lendrum D, Canziani OF, Githeko A, et al. Methods of assessing human health vulnerability and public health adaptation to climate change. Health and global environmental change. Series no.1. Copenhagen, Denmark: WHO/EURO; 2003.
18. Preston BL, Suppiah R, Macadam I, Bathols J. Climate change in the Asia/Pacific region. A consultancy report prepared for the climate change and development roundtable. Aspendale, Australia: CSIRO Marine & Atmospheric Research; 2006.
19. Ebi KL, Lewis ND, Corvalan C. Climate variability and change and their potential health effects in small island states: information for adaptation planning in the health sector. Climate change and human health: national assessments of impacts and adaptation. *Environ Health Perspect* 2006; 114: 1957–63. (Mini-monograph)
20. Koh KWB, Ng LC, Kita Y, Tang CS, Ang LW, Wong KY, et al. The 2005 dengue epidemic in Singapore: epidemiology, prevention and control. *Ann Acad Med Singapore* 2008; 37: 538–45.
21. Heng BH, Goh KT, Neo KS. Environmental temperature, *Aedes aegypti* house index and rainfall as predictors of annual epidemics of dengue fever and dengue haemorrhagic fever in Singapore. In: Goh KT, ed. *Dengue in Singapore*. Singapore: Institute of Environmental Epidemiology, Ministry of Environment. Technical Monograph Series No. 2. 1998, pp. 138–49.
22. Key Annual Indicators. Singapore: Department of Statistics. Available from: <http://www.singstat.gov.sg/stats/keyind.html>; 2008 [cited 12 March 2009].
23. Meteorological Services Division. Climatology of Singapore. Singapore: National Environment Agency. Available from: http://app2.nea.gov.sg/weather_statistics.aspx; 2009 [cited 10 August 2009].
24. Infectious Diseases Act (Chapter 137). Singapore: Ministry of Health; 1977.
25. NNDC Climate Data Online. National Climatic Data Center, NESDIS, NOAA, U.S. Department of Commerce. Available from: <http://www7.ncdc.noaa.gov/CDO/dataproduct>; 2008 [cited 20 July 2008].
26. Monthly Digest of Statistics Singapore, March 2009: demography: population and growth rate. Singapore: Department of Statistics. Available from: <http://www.singstat.gov.sg/pubn/reference/mdsmar09.pdf>; 2009 [cited 8 April 2009].
27. Naumova EN, MacNeill IB. Time-distributed effect of exposure and infectious outbreaks. *Environmetrics* 2009; 20: 235–48.
28. R Development Core Team. R: a language and environment for statistical computing. Vienna, Austria: R Foundation for Statistical Computing. Available from: <http://www.R-project.org>; 2008 [cited April 2009].
29. Gratz NG. Critical review of the vector status of *Ae. albopictus*. *Med & Vet Entomol* 2004; 18: 215–27.
30. Githeko AK, Lindsay SW, Confalonieri UE, Patz JA. Climate change and vector-borne diseases: a regional analysis. *Bull World Health Organ* 2000; 78: 1136–47.
31. Hurtado-Diaz M, Riojas-Rodriguez H, Rothenberg SJ, Gomez-Dantes H, Cifuentes E. Short communication: impact of climate variability on the incidence of dengue in Mexico. *Trop Med Int Health* 2007; 12: 1327–37.
32. Halstead SB. Dengue virus-mosquito interactions. *Annu Rev Microbiol* 2008; 53: 273–91.
33. Climate Prediction Center. Cold & warm episodes by season. National Weather Service. Available from: http://www.cpc.noaa.gov/products/analysis_monitoring/ensostuff/ensoyears.shtml; 2009 [cited 8 April 2009].
34. Campbell-Lendrum D, Corvalan C. Climate change and developing-country cities: implications for environmental health and equity. *J Urban Health* 2007; 84: i109–17.
35. Aniello C, Morgan K, Busbey A, Newland L. Mapping micro-urban heat islands using Landsat TM and a GIS. *Comput Geosci* 1995; 21: 965–9.
36. Ye T, Ang LW, Chow A, Chew SK. Seroprevalence study on past and recent dengue virus infection in Singapore. *Epidemiol News Bull (Singapore)* 2007; 33: 36–41.
37. Johansson MA, Dominici F, Glass GE. Local and global effects of climate on dengue transmission in Puerto Rico. *PLoS Negl Trop Dis* 2009; 3: e382.
38. Promprou S, Jaroensutasinee M, Jaroensutasinee K. Climatic factors affecting dengue haemorrhagic fever incidence in Southern Thailand. *Dengue Bull* 2005; 29: 41–8.

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Climate impacts on environmental risks evaluated from space: a conceptual approach to the case of Rift Valley Fever in Senegal

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Background: Climate and environment vary across many spatio-temporal scales, including the concept of climate change, which impact on ecosystems, vector-borne diseases and public health worldwide.

Objectives: To develop a conceptual approach by mapping climatic and environmental conditions from space and studying their linkages with Rift Valley Fever (RVF) epidemics in Senegal.

Design: Ponds in which mosquitoes could thrive were identified from remote sensing using high-resolution SPOT-5 satellite images. Additional data on pond dynamics and rainfall events (obtained from the Tropical Rainfall Measuring Mission) were combined with hydrological in-situ data. Localisation of vulnerable hosts such as penned cattle (from QuickBird satellite) were also used.

Results: Dynamic spatio-temporal distribution of *Aedes vexans* density (one of the main RVF vectors) is based on the total rainfall amount and ponds' dynamics. While Zones Potentially Occupied by Mosquitoes are mapped, detailed risk areas, i.e. zones where hazards and vulnerability occur, are expressed in percentages of areas where cattle are potentially exposed to mosquitoes' bites.

Conclusions: This new conceptual approach, using precise remote-sensing techniques, simply relies upon rainfall distribution also evaluated from space. It is meant to contribute to the implementation of operational early warning systems for RVF based on both natural and anthropogenic climatic and environmental changes. In a climate change context, this approach could also be applied to other vector-borne diseases and places worldwide.

Keywords: *climate change; public health; remote sensing; risk mapping; vector-borne diseases; Rift Valley Fever; early warning systems; Health Information System*

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Rationale

Variation and change in climate

The climate changes and varies on many spatio-temporal scales. Natural variability in climate is a function of: (i) the relative position of the sun (and its activity such as sunspots, radiation, magnetism, eruption) and the earth; (ii) the Milankovitch cycles (1); and (iii) the interactions between the components of the climate system, i.e. the atmosphere, the hydrosphere, the cryosphere, the biosphere and the lithosphere. For centuries a panoply of climate signals have been noted, ranging from the diurnal, to multi-decadal effects including seasonal, quasi-biennial (QB), El-Niño/Southern Oscillation

(ENSO), quasi-decadal (QD) and inter-decadal (ID) oscillations (2). Adding to these natural cycles and oscillations is the anthropogenic component from population increase, energy needs and associated pollution. Natural climate oscillations interact with the anthropogenic climate change component, and directly impact ecosystems, public health and socio-economic conditions. The natural variability of the global climate during the 20th century is reproduced in Fig. 1 [see also (3)].

Climate change and public health

Climate change *alters* regional and local social and economic dynamics with the potential of bringing additional inequalities all around the world (4). This could

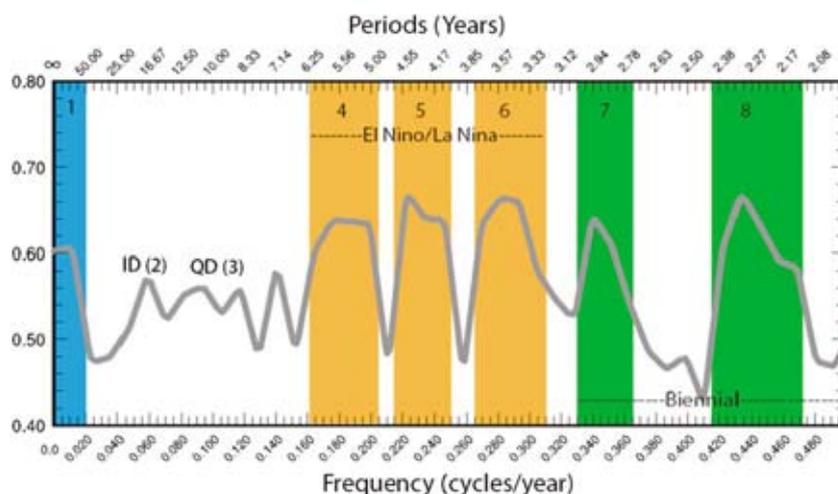


Fig. 1. During the 20th century a global statistical analysis in the frequency-domain of both sea-surface temperature and sea-level pressure, allows identification of natural climate signals. Coloured bands highlight those signals with their percentage of variance displayed on the ordinates, i.e. secular signal or penta-decadal (blue band, signal # 1), El-Niño/Southern Oscillation or ENSO (orange bands, signals # 4, 5 and 6), the quasi-biennial signal (green bands, signals # 7 and 8). Signal # 2 ID or inter-decadal, and signal # 3 QD or quasi-decadal, are more locals and found over the Pacific and Atlantic Oceans, respectively (2). The anthropogenic climate change is interacting and modulates the above climate signals.

result in economic migration, and reduced access to primary resources. Climate change had impacts in historical times with respect to the development of many cultures. Changes have been observed in nutrient budgets and nutrient cycles, with enhanced human pressure through population increase and public health impacts. The total primary energy demand is expected to increase by ~60% during the first quarter of the 21st century. Most of this energy will come from fossil sources, and unfortunately only 1–2% is expected from renewable sources. Such disequilibrium is likely to create socio-economic chaos, regional and local vulnerability in terms of prices and supply, and have considerable impacts on the environment and public health, for example, on infectious diseases, respiratory and circulatory problems, pollution, allergen-related diseases and impaired immune systems. Public health issues will also be exacerbated by poor water quality and malnutrition, leading to huge costs and increasing poverty.

Climate change *perturbs* important physical and biological systems to which human populations are generally biologically and culturally adjusted. The various environmental changes linked to natural and anthropogenic variability and changes in climate, and loss of biodiversity though land-use changes, will all have their own impacts on public health. It is recognised that beneficial impacts such as decreases in cold-related deaths are also anticipated. Direct influence from demographic factors may increase risks of infectious diseases being transmitted from person-to-person. Thus, socio-economic impacts on infectious diseases and public health, arising from climate and environmental changes, require attention. Most

emerging (or re-emerging) infectious diseases (including vector-borne diseases) are due partly to changes in ‘microbial traffic’, for example, the introduction of pathogens from wildlife into human populations already at risk. Changes in transmission of diseases by vectors (such as mosquitoes) may arise from new vector reservoirs in different habitats, the changing climate and environmental determinants of which deserve further investigation. These processes may depend upon ecological and environmental factors, but the spread of diseases is also facilitated by climate variability/change, population migration, demographic crowding effects, sanitation levels and/or breakdowns in public health systems. As of today, the increase in occurrence of many infectious diseases reflects the compounded effects of climatic and environmental changes, population increases, economic, social and technological changes.

Climate change and infectious diseases

The challenge for assessing socio-economic impact of infectious diseases (~75% of actual infectious diseases in humans are zoonoses) cannot be addressed without considering both abiotic and biotic environmental factors that affect the maintenance and transmission of the diseases. The last 25 years have witnessed an explosion of environmentally related diseases and disorders, with strong environmental forcing and adaptation or lack thereof. For infectious diseases, this includes increases in prevalence, incidence and geographical distribution across wide taxonomic ranges, related to climatic and environmental changes and practical changes in land-use. The understanding of these associated changes represents

an important step for moving away from the more traditional individual-centred view of microbiology and medical epidemiology.

Direct health effects of climate variability and change include: (i) changes in mortality and morbidity arising from heat-waves and thermal stress (such as in 2003 over southwest Europe; and to a lesser degree in 2007 over Italy and Greece); (ii) respiratory ailments associated with modified concentrations of particulate matter and aero-allergens (e.g. spores, moulds) and/or air pollutants; and (iii) health consequences from extreme weather events, including storms, floods and gales.

Indirect health effects arise from perturbation of more complex ecological systems, and include changes in the ecology, range and activity of vectors and associated diseases (i.e. malaria, West Nile virus, Rift Valley Fever (RVF), avian flu, chikungunya, dengue fever and others) (5); changes in the environment for water-borne diseases and pathogens (i.e. gastro-intestinal infections, vibrio diseases including cholera, diseases from polluted water and others); changes in the atmospheric boundary layer, and transmission of air-borne diseases (i.e. meningitis, respiratory ailments and others); changes in regional and local agricultural practices and food availability which can lead to malnutrition and lack of fresh water. Public health can also be affected by massive population movements along narrow coastal regions, and by regional conflicts arising from declining agricultural and water resources. Some diseases have already extended their endemic range, such as leishmaniasis in southern Europe and the Maghreb. Climate change may facilitate habitat extension for sandfly and other phlebotome vectors northwards, whilst the ecology and geography of the tick species responsible for transmitting Lyme disease may profoundly change.

Climate change and decision-making

Climate change affects regional socio-economic costs and losses, through changes in temperature and soil moisture, inherent use of fertilisers and pest and pathogen activity. Decision-making models to be used must include:

- (1) Identification of 'normal' impacts of disease (in lives and economic terms).
- (2) Definition of 'climate events' linked to 'health events' (epidemics, endemics, pandemics).
- (3) Definition of 'increased impacts' and socio-economic losses.
- (4) Identification of methods for loss mitigation.
- (5) Definition of real costs for effective implementation of services such as Health Information Systems (HIS).
- (6) Quantification of real savings (including lives) if a well-identified 'health event' does not occur.

Even if regional modelling studies consistently indicate that tropical and sub-tropical countries would be most affected, changing climate and environment at higher latitudes must also be considered. Forecasting climate change impacts on public health requires the development of scenario-based risk assessments which must include generalised assessment of the consequences from complex demographic, social and economical disruptions. Integrated mathematical modelling must be used if one wants to estimate the future impacts of climate change on health (6). Such new modelling requires that each component of the chain of causation: climate, environmental and social change is fully represented.

Uncertainties do remain and are due to future industrial and economic activities, interactions between and within natural systems, and differences in sensitivity of disease systems and vulnerability of populations. Differences in population vulnerability could arise from heterogeneity of human culture, social relations and behaviour. Non-linear uncertainties arise from the stochastic nature of the biophysical systems being modelled. Local anthropogenic deforestation may directly alter the distribution of vector-borne diseases while also cause a local increase in temperature (positive feedback).

Climate and environmental changes and remote sensing

Public health indicators and disease surveillance activities should be integrated with other in-situ monitoring systems developed by the United Nations, such as Global Climate Observing System (GCOS), Global Ocean Observing System (GOOS), Global Terrestrial Observing System (GTOS) and the integrated Global Earth Observation System of Systems (GEOSS). Today, the use of satellites allows monitoring in high resolution of changes in environmental and climatic parameters. This provides an important continuum of observational spatio-temporal scales on both oceanic and terrestrial environmental structures, which should never be interrupted.

Tele-epidemiology

Infectious diseases remain a considerable challenge to public health. In the context of climate change and the rapidly increasing population as mentioned above, some epidemics are emerging or re-emerging such as the RVF over West Africa, dengue fever over northern Argentina and chikungunya in the Indian Ocean and northern Italy, among others.

The conceptual approach

Following the French contribution and presentation during the Johannesburg Summit 2002, a new conceptual approach has been developed: so-called tele-epidemiology

(7). It aims to monitor and study the spread of human and animal infectious diseases which are closely tied to climate and environmental changes. By combining satellite-originated data on vegetation (SPOT), meteorology (Meteosat, TRMM), oceanography (Topex/Poseidon; ENVISAT, JASON) with hydrology data (distribution of lakes, water levels in rivers, ponds and reservoirs), with clinical data from humans and animals (clinical cases and serum use), predictive mathematical models can be constructed.

Lately as a part of the French Ministry of Research's Earth-Space Network, a pilot sentinel network has been deployed in Niger and Burkina Faso for monitoring infectious diseases such as malaria, which is also tied to changing environmental factors. This integrated and multidisciplinary approach of tele-epidemiology includes:

- (1) monitoring and assembling multidisciplinary in-situ datasets to extract and identify physical and biological mechanisms at stake;
- (2) remote-sensing monitoring of climate and environment, linking epidemics with 'confounding factors' such as rainfall, vegetation, hydrology and population dynamics; and
- (3) use of bio-mathematical models for epidemic dynamics, vector aggressiveness and associated risks.

As such an interactive tool contributing to HIS on re-emergent and new infectious diseases (RedGems) was born (8). It constitutes the main pillar of tele-epidemiology by facilitating real-time monitoring of human and animal health and the exchanges of epidemiological, clinical and entomological data. The primary mission of RedGems (www.redgems.org) is to contribute towards the development of early warning systems (EWS) for infectious diseases and contribute to the main three actions of tele-epidemiology presented above. The overall objective is to attempt predicting and mitigating public health impacts from epidemics, endemics and pandemics.

The Rift Valley Fever (RVF) case

The various components of the new conceptual approach described above have been thoroughly tested with regard to the emerging RVF in the Ferlo (Senegal). This successful approach has led the Senegalese government to provide funding, and extend the approach to all risk zones (i.e. hazards+vulnerability) where populations and cattle are exposed (9).

The Ferlo region in Senegal, became prone to RVF in the late 1980s with the appearance of infected vector mosquitoes of the *Aedes vexans* and *Culex poicilipes* species (10, 11). The latter proliferate near temporary ponds and neighbouring humid vegetation. RVF

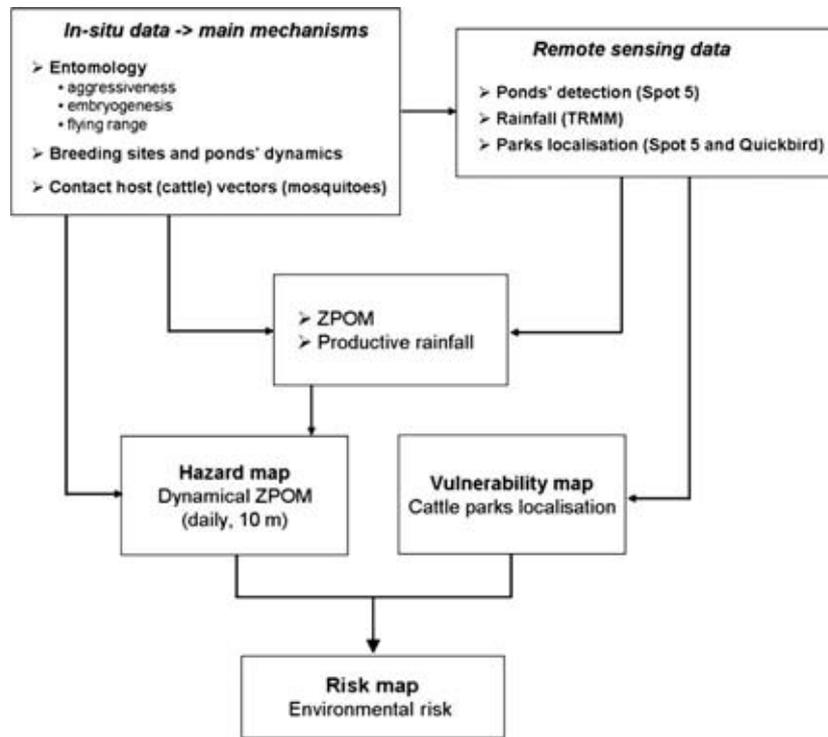


Fig. 2. Integrated conceptual approach. The basic components for the concept are presented in the top three boxes: in-situ data (upper left), remotely sensed data (upper right) and Zone Potentially Occupied by Mosquitoes or ZPOMs and 'productive rainfall' in terms of production of mosquitoes/vectors (centre). The bottom three boxes distinguish between hazards (bottom left), vulnerability (bottom right), both leading to the environmental risks (very bottom).

epizootic outbreaks in livestock cause spontaneous abortions and perinatal mortality. So far, human-related disease symptoms are often limited to flu-like syndromes but can include more severe forms of encephalitis and haemorrhagic fevers. As a result, local socio-economic resources can be seriously affected. The ultimate goal was to use specific Geographical Information System (GIS) tools (12) and remote-sensing (RS) images and data to detect potential breeding ponds and evaluate RVF transmission and areas potentially at risk, characterised as Zones Potentially Occupied by Mosquitoes (ZPOMs).

A schematic design of the integrated conceptual approach to determine the environmental risk levels of RVF is presented in Fig. 2. The upper left box in the figure identifies key entomological factors for *A. vexans* (flying-range, aggressiveness and embryogenesis), environmental factors (rainfall distribution, limnometry and pond dynamics) as well as pastoral data such as the zones where animals are penned at night. From the upper right box, the detection of lead environmental and climatic factors (mainly rainfall) favouring the mechanisms presented are highlighted. For example, localities and optimal pond conditions for the breeding and hatching of *A. vexans* can be modelled (13). The central box refers

to the ZPOMs derived from pond dynamics after a 'productive rainfall' event, and which includes the flying ranges of *A. vexans*. The integration of all the above components leads to the notion of risks: hazards and exposure vulnerability of hosts. This original approach (14) bridges the physical and biological mechanisms, linking environmental conditions to the 'production' of RVF vectors and accompanying potential risks.

Possible hazards in the vicinity of fenced-in hosts are displayed in Fig. 3, where the Barkedji area is shown with the mapped ZPOMs. Thus, parks and villages can easily be identified. Out of 18 rainfall events obtained from TRMM for the 2003 rainy season, seven were considered as 'productive' with regard to *A. vexans* production (based on entomological studies).

Conclusion

Climate variability and change, environmental risks and public health are all associated. In the case of potential RVF epidemics, mechanisms linking rainfall variability (and trends), density and aggressiveness of vectors and vulnerability of hosts are presented. Using observations from space, we constructed the dynamic evolution of ZPOMs [Fig. 4; see also animated on-line version in (9)

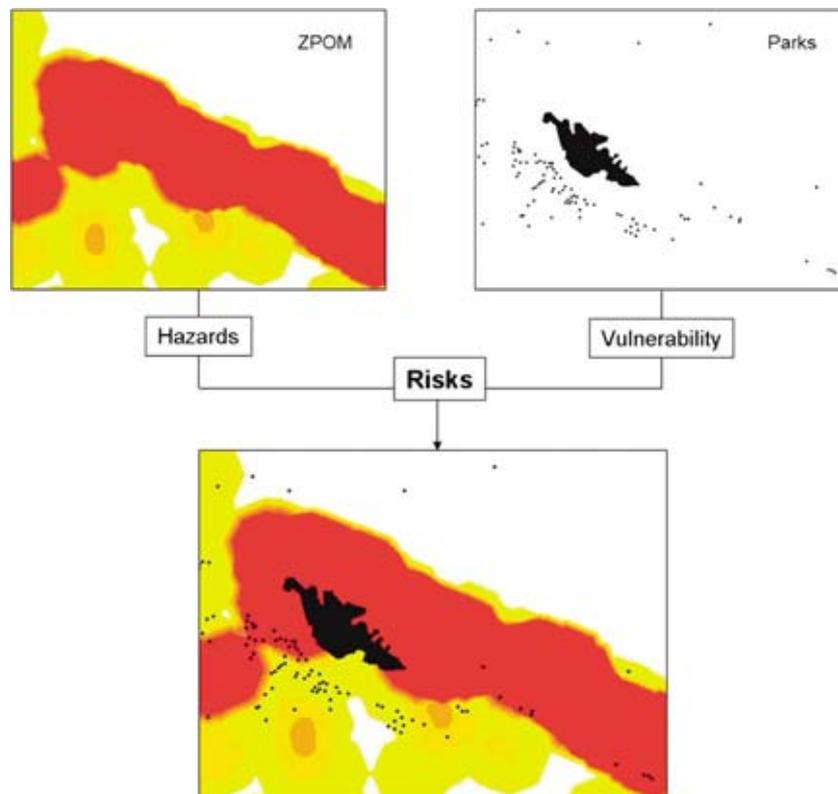


Fig. 3. Zone Potentially Occupied by Mosquitoes, or ZPOMs with ranked hazards from yellow (low hazards) to red (high hazards). ZPOMs in the Barkedji area constructed from the pond distribution after a single rainfall event (top left). Localisation of the Barkedji village and ruminants' fenced-in areas (vulnerability, from QuickBird) in black for the same area and period (top right). Potential risks i.e. hazards+vulnerability are shown by super-imposing the two pictures (bottom).

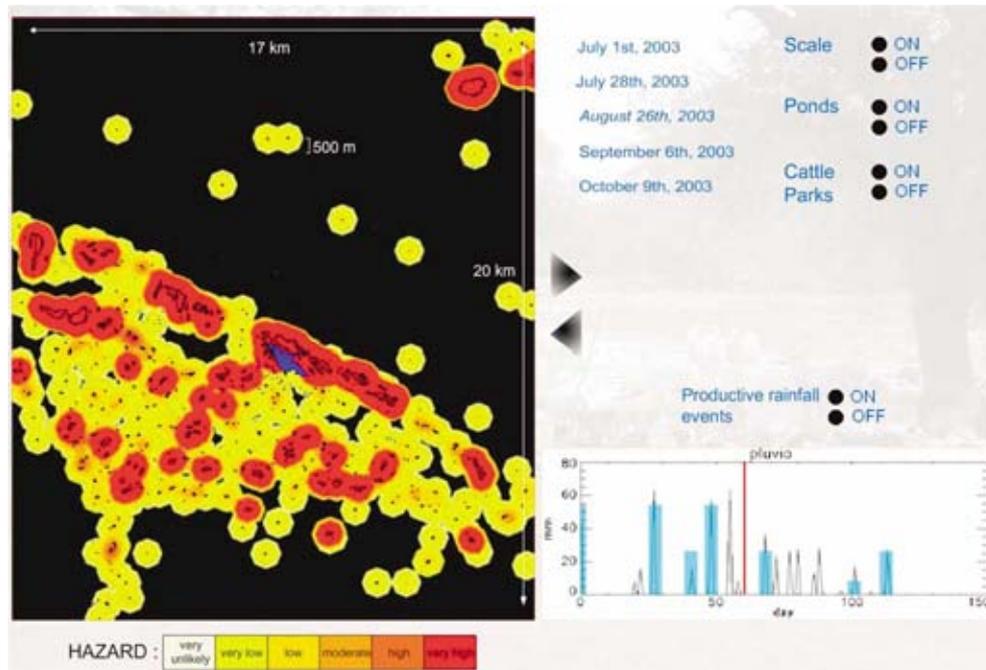


Fig. 4. Dynamic Zones Potentially Occupied by Mosquitoes (ZPOMs) and ranked hazards+risks. Dynamic ZPOMs with ranked hazards (from very unlikely and very low in yellow, to very high in red, bottom scale) and ponds distribution (in blue) during the 2003 rainy season. From the hyperlinked figure available on-line paper, by clicking on the two fat black arrows, animated ZPOMs from ‘productive rainfall’ (highlighted in blue, at the bottom right) are displayed (upper arrow for forward motion, lower arrow for backward motion) along with the relative parks’ locations (vulnerability). The starting date is 28 June 2003. ZPOMs for specific date can also be displayed. The vertical red marker is for accurate time positioning on a daily basis.

available at www.geospatialhealth.unina.it] from the distribution and development of ponds was crucial. It allowed direct identification of RVF risks from discrete and ‘productive rainfall’ events such as local deep atmospheric convections and propagating squall-lines. This remote-sensing approach and the new integrated concept belongs to the so-called tele-epidemiology developed at CNES (14).

Climatic and environmental variability and changes identified from space provide the elements for the mapping of risk zones in which necessary conditions for the RVF virus to circulate and be transmitted exist. The evolution of the ZPOMs during the rainy season reveals areas in which populations and cattle of the Ferlo region in Senegal are exposed. There are many strengths in this approach. It can be used in quasi real-time, and results can be linked with biological modelling for virus transmission and circulation and more classical epidemiological models. Socio-economic risks may be reduced and mitigated, based upon statistical evaluation of the seasonal rainfall forecasts which can be assessed a few months prior to the rainy season and subsequently updated. For example, results can be immediately applied upstream by the Senegalese Direction de l’Elevage (DIREL) though strategic displacement of fenced-in

areas for cattle penned at night, during the course of the rainy season. Nonetheless, socio-economic problems may still arise if the relevant information has not been distributed operationally to all parties involved, through regional HIS. Ultimately, the fully integrated approach should help in understanding the mechanisms leading to potential RVF epidemics and improve the RVF EWS.

The conceptual approach presented might not apply directly for other vector-borne diseases, whose vectors have different behaviours. Thus, physical and biological mechanisms for other infectious diseases and places (including higher latitude regions) need to be studied individually. A similar methodology using space observations may be used, particularly in places where climate and environment are foreseen to change rapidly, as for example currently being implemented for malaria in Burkina Faso.

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References

- Milankovitch M. 1930: Mathematische Klimalehre und Astronomische Theorie der Klimaschwankungen, Handbuch der Klimologie Band 1. Teil A Borntrager: Berlin; 1930.
- Tourre YM, White WB. Global climate signals and equatorial SST variability in the Indian, Pacific and Atlantic oceans during the 20th century. *Geophys Res Lett* 2006; 33: L06716.
- Lacaux J-P, Tourre YM. Impacts des changements climatiques. Le climat et sa variabilité ont-ils un impact sur la santé humaine? *Biofutur* 2006; 270: 22–4.
- Plan Bleu. Climate change and energy in the Mediterranean. Part 1, Chapter 1: Mediterranean Basin: climate change and impacts during the 21st century. Report 2008. Sophia Antipolis, France: Le Plan Bleu; 2008, pp. 52–4.
- Takken W, Martens P, Bogers RJ. Environmental change and malaria risk: global and local implications. New York, LLC: Springer-Verlag; 2006, p. 139.
- Martens P. Climate change 2001: vulnerability and sustainability. IPCC TAR report. Available from: www.grida.no/climate/ipcc_tar/wg2/539.htm [cited September 2009].
- Lafaye M. Nouvelles applications spatiales pour la santé: la télé-épidémiologie pour le suivi des fièvres aviaires. Toulouse cedex, France: CNES Magazine; 2006, 30–31 February 2006.
- Marechal F, Ribeiro N, Lafaye M, Güell A. Satellite imaging and vector-borne diseases: the approach of the French National Space Agency (CNES). *Geospat Health* 2008; 3: 1–5.
- Vignolles C, Lacaux J-P, Tourre YM, Bigeard G, Ndione J-A, Lafaye M. Rift Valley Fever in a zone potentially occupied by *Aedes vexans* in Senegal: dynamics and risk mapping. *Geospat Health* 2009; 3: 211–20.
- Lacaux J-P, Tourre YM, Vignolles C, Ndione J-A, Lafaye M. Classification of ponds from high spatial resolution remote sensing: application to Rift Valley Fever epidemics in Senegal. *J Remote Sensing Env* 2007; 106: 66–74.
- Tourre YM, Lacaux J-P, Vignolles C, Ndione J-A, Lafaye M. Mapping of zones potentially occupied by *Aedes vexans* and *Culex poicilipes* mosquitoes, the main vectors of Rift Valley Fever in Senegal. *Geospat Health* 2008; 3: 69–79.
- Tourre YM, Fontannaz D, Vignolles C, Ndione J-A, Lacaux J-P, Lafaye M. GIS and high-resolution remote sensing improve early warning planning for mosquito-borne epidemics. *Healthy GIS, GIS for Health and Human Services, ESRI; 2007: 1–4.*
- Bicout DF, Porphyre T, Ndione J-A, Sabatier P. Modeling abundance of *Aedes* and *Culex* spp. in rain-fed ponds in Barkedji, Senegal. Proceedings of the 10th International Symposium on Veterinarian Epidemiology and Economics, Vina del Mar, Chile, 17–21 November 2003, p. 84.
- CNES, 2008: Method for tele-epidemiology (Méthode pour la télé-épidémiologie). Patent pending # PCT/FR2009/050735.

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Vulnerability to episodes of extreme weather: Butajira, Ethiopia, 1998–1999

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Background: During 1999–2000, great parts of Ethiopia experienced a period of famine which was recognised internationally. The aim of this paper is to characterise the epidemiology of mortality of the period, making use of individual, longitudinal population-based data from the Butajira demographic surveillance site and rainfall data from a local site.

Methods: Vital statistics and household data were routinely collected in a cluster sample of 10 sub-communities in the Butajira district in central Ethiopia. These were supplemented by rainfall and agricultural data from the national reporting systems.

Results: Rainfall was high in 1998 and well below average in 1999 and 2000. In 1998, heavy rains continued from April into October, in 1999 the small rains failed and the big rains lasted into the harvesting period. For the years 1998–1999, the mortality rate was 24.5 per 1,000 person-years, compared with 10.2 in the remainder of the period 1997–2001. Mortality peaks reflect epidemics of malaria and diarrhoeal disease. During these peaks, mortality was significantly higher among the poorer.

Conclusions: The analyses reveal a serious humanitarian crisis with the Butajira population during 1998–1999, which met the CDC guideline crisis definition of more than one death per 10,000 per day. No substantial humanitarian relief efforts were triggered, though from the results it seems likely that the poorest in the farming communities are as vulnerable as the pastoralists in the North and East of Ethiopia. Food insecurity and reliance on subsistence agriculture continue to be major issues in this and similar rural communities. Epidemics of traditional infectious diseases can still be devastating, given opportunities in nutritionally challenged populations with little access to health care.

Keywords: *climate change; famine; mortality; demographic surveillance; epidemiology*

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Having suffered some apocalyptic famines in the later decades of the twentieth century, Ethiopia acquired a reputation, at least among the international media, of being a land of famine. Fortunately the scale of famine has reduced, perhaps partly due to the end of the civil war in 1991, less widespread droughts, and improvements in water harvesting and storage. However, although famines in Ethiopia are no longer hitting the headlines as they once did, food security remains problematic in some areas and in particular periods. With increasing climate change, the probability will increase of episodes of extreme weather situations, i.e. fluctuations in seasonality and amounts of rainfall. Famine has long been recognised as a result of inequi-

table distribution, not of an actual shortage of food on a global or even a national scale (1). However, there is still a reason to assume that the human consequences of times of food insecurity or the need of aid are not equitably distributed. These consequences are not generally easy to assess in a society where even most births and deaths go unregistered, and where there is no comprehensive health information system.

Famine in Ethiopia is certainly not a new problem, and particular historical episodes have been documented over a long period (2). Much of the documentation of famine has concentrated on its effects on children (3), but there is also evidence of substantial effects of nutritional stress among older Africans (4). Acute episodes of famine

triggering emergency response have been described in terms of morbidity and mortality (5). Famine-associated mortality patterns globally have often been associated with epidemics of infectious disease, such as cholera and malaria, among nutritionally compromised populations (6).

Food insecurity continues to be a problem in sub-Saharan Africa. A national survey by the Ethiopian Central Statistical Authority in 1999, as reported by the International Food Policy Research Institute (7), showed that Ethiopia averages one of the lowest energy per capita per day levels (1,648 kCal) in sub-Saharan Africa, with the majority of the population in a food energy deficient state (76.4%). During 1999–2000, Ethiopia experienced a period of famine due to low and untimely rainfall (8).

The focus of media and organisations has been on pastoralists and semi-pastoralists in eastern Ethiopia. The effects of drought on grazing and on the crops of vegetables and pulses from the small rainy season have been treated as specific to these most drought-prone areas, while the farming communities in the central and western highlands have received less attention (8).

The Butajira Rural Health Programme (BRHP) has maintained a programme of epidemiological surveillance in the Butajira District since 1987 (9), currently named the Meskan and Mareko district, with Butajira town as its administrative centre. Inspection of the BRHP data revealed two peaks of mortality, in 1998 and 1999, well in excess of the normally observed year-to-year variation. Further investigation and enquiry revealed that these related to a serious episode of unseasonal rainfall followed by drought with consequent food insecurity.

The aim of this paper is to characterise the detailed epidemiology mortality of this period, making use of the rare opportunity BRHP affords of having individual population-based data before, during and after the crisis period, on a longitudinal basis. Having access to basic information on household economy we also aim to demonstrate social inequity in vulnerability to climatic events.

Methods

BRHP has operated community-based surveillance on an open-cohort population sample within the Butajira District since 1987. This involved initially selecting a sample of communities within the district using a probability proportional to size method, an initial enumeration of the selected communities in late 1986, and continuous surveillance of vital events by means of household visits (initially monthly, later quarterly) from the start of 1987 (9). The sample includes five highlands, four lowland villages and one of the four administrative units of Butajira town.

Butajira district is located some 130 km to the south of Addis Ababa, in central Ethiopia, and is part of the

Southern Nations, Nationalities and Peoples' Region (SNNPR). The district covers an area of approximately 25 × 25 km. The mountains of the African Rift Valley rise to some 3,500 m above sea level to the west of the district, while the eastern area is a series of plateaux around 1,500 m above sea level. Consequently patterns of agriculture, which are predominantly based on subsistence crops together with some cash crop production, vary considerably across the district. Malaria occurrence is characterised by high altitude endemicity, with more frequent outbreaks in the lowland areas. Butajira town is centrally located at the interface of the highland and lowland areas, where the main activities are small-scale trading and service industries. Most town dwellers operate within a cash economy, albeit at a very basic level, unlike the more subsistence-based rural economy. Electricity, piped water and hospital services are available to many in the town, but not in the surrounding villages.

As the mortality peak associated with this period of untimely rainfall occurred during 1998–1999, the data analysed here relate to the 5-year period 1997–2001 inclusive, in order to see what happened before and after the crisis. BRHP routine data include all deaths, which are registered at the household level, and the informants' opinion as to the cause of death. More recently, verbal autopsy procedures have been introduced, giving more detailed cause of death data (9, 10), but for this period, no more details on circumstances and causes of death are available. In this study, cause of death is as reported by the respondents in the routine surveillance. Household locations are known from GPS data, together with basic rural household characteristics such as household water supply and farm land holding, and linked to details of the individual inhabitants. In an attempt to study the socio-economic distribution of mortality, the rural population has been classified according to land holding as living on smaller or larger farms. The unit used in the BRHP data collection is the Ethiopian *timad*, approximately 0.5 hectares. Based on the distribution itself 'smaller farms' are defined as 0–3 *timad*, and 'larger farms' as 4 *timad* and above.

Monthly rainfall data from the weather station in Butajira for the years 1987–2003 were made available by the National Meteorological Authority. Crop statistics were provided by the District Agriculture Bureau of the Meskan and Mareko District Authority.

BRHP data are managed in a dBase system, which was used to extract the relevant records for this period. The Cohort software (Umeå University) was used for person time-based cohort analysis and Stata (Stata Corporation) for logistic regression analysis. Space–time clustering was modelled using SaTScan v6.1 (11–13).

BRHP research activities have been approved by the National Ethical Clearance Committee and individual

informed consent obtained from each participant and/or their parents/guardians.

Results

Average annual rainfall was 1,211 mm for the period 1987–2003. Annual rainfall showed moderate variation over the period for which data were available, from a maximum of 1,471 mm in 1996 to the minimum of 865 mm in 2000. However, monthly rainfall fluctuated considerably more. Fig. 1 shows the rainfall 1997–2001 by month, plotted against the average for each calendar month of the period 1987–2003. The expected pattern has a smaller rainy season around March–April and a larger mid-June to mid-September. While 1997 and 2001 show average patterns with clear seasonality and average totals, the three intervening years are different. In 1998, the total rainfall was approximately 20% above annual average and there was no break between the small and big rains. Both 1999 and 2000 had less than average annual rainfall, 80 and 70% of average, respectively. In 1999, the small rains nearly failed and the big rains began late and went on into the normally dry harvest months, September–November. The rains in 2000 were approximately on season, but the total was low.

Unseasonal or low amounts of rain would be expected to influence the crop yields. The main harvest season is in October–November and the yield can be affected both by drought during the growth period in June–September and by rains during the harvest. However, the agricultural production reports to the district Bureau of Agriculture do not show any pattern similar to those of the rainfall data. Data for three major cash crops, maize, teff (an indigenous Ethiopian cereal) and pepper were available for five of the nine rural sub-districts included in the demographic surveillance, from the harvests 1997–2001.

Maize shows a drop in yield for the year 1999 but not for 1998 or 2000. Teff and pepper gave average yields in 1998, 1999 and 2000. Overall, there was little variation in the reported agricultural production for the period.

Table 1 shows the mortality experience of the Butajira district population by year, sex age and area. A total of 3,512 deaths occurred in the 5-year period 1997–2001 inclusive, among 222,891 person-years (p-y) of observation, giving an overall mortality rate of 15.8 per 1,000 p-y. However, 1,327 deaths (37.8%) occurred in 1999 and 801 (22.8%) in 1998, giving a mortality rate of 24.5 per 1,000 p-y for 1998–1999, compared with 10.2 per 1,000 p-y in the remainder of the 5-year period. Mortality rates per 1,000 p-y, by quarter for the 5-year period, are shown in Fig. 2, in which the mortality fractions ascribed to malaria and diarrhoea/malnutrition are shown separately. There was no substantial measles outbreak during the period. The mortality peaks in 1998 and 1999 were reflected across all age groups, but were most marked in children under five years of age, who experienced the greatest increase in mortality rates. Deaths among children under 5 accounted for 47.2% of the total. The CDC humanitarian crisis threshold of one death per 10,000 per day (14) was exceeded during the period April–September 1999.

Fig. 3 shows how under-five mortality was distributed among the three different ecological zones (rural highlands, rural lowlands and Butajira town). It is evident that the town population was largely unaffected, with only a very minor increase in mortality in 1998. In the following analyses, the town population has therefore been excluded.

Most of the excess mortality in 1998–1999 appears to be attributed to either malaria or diarrhoea/malnutrition (Fig. 2). Table 2 shows the results of a multivariate

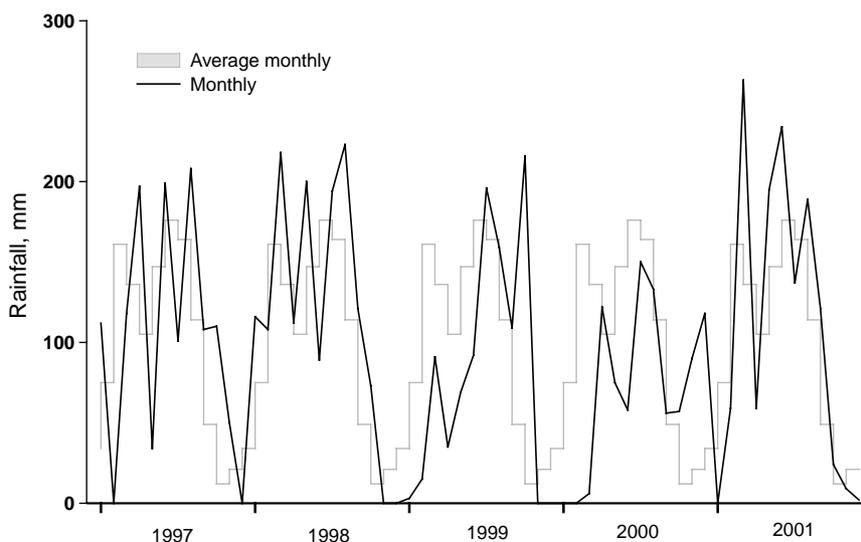


Fig. 1. Patterns of monthly rainfall 1997–2001 and 1987–2003 average rainfall for calendar month.

Table 1. Mortality per 1,000 person-years and number of deaths, 1997–2001 in the Butajira district, by age, sex, geographical area and calendar year

			1997	1998	1999	2000	2001
Sex	Age years		Mortality/1,000 p-y (deaths)				
Highland	F	0–4	30.9 (48)	44.8 (72)	84.4 (131)	26.8 (41)	20.2 (30)
		5–49	6.0 (40)	8.6 (63)	14.6 (111)	5.1 (41)	2.5 (21)
		50+	43.5 (32)	36.9 (29)	117.5 (90)	30.2 (24)	10.3 (9)
M	0–4	44.2 (67)	42.5 (69)	79.1 (127)	26.4 (42)	21.7 (35)	
		5–49	6.0 (38)	9.2 (63)	13.6 (96)	4.8 (36)	2.6 (20)
		50+	49.9 (39)	32.1 (26)	88.9 (70)	31.6 (26)	17.7 (16)
Lowland	F	0–4	49.2 (67)	78.6 (115)	114.9 (161)	32.9 (45)	28.1 (39)
		5–49	6.1 (32)	9.5 (55)	18.5 (111)	5.1 (33)	4.2 (28)
		50+	29.5 (15)	54.1 (29)	76.0 (40)	33.5 (19)	21.4 (13)
M	0–4	43.9 (61)	84.0 (127)	103.4 (150)	36.1 (52)	25.7 (37)	
		5–49	7.1 (36)	11.7 (65)	18.8 (109)	4.4 (28)	3.0 (20)
		50+	21.8 (13)	33.2 (21)	77.3 (48)	31.3 (21)	19.3 (14)
Urban	F	0–4	18.6 (12)	15.4 (11)	28.1 (20)	13.8 (10)	19.8 (13)
		5–49	3.9 (11)	4.3 (14)	1.9 (7)	2.8 (12)	3.0 (14)
		50+	35.4 (11)	34.1 (11)	41.8 (14)	41.7 (15)	26.0 (10)
M	0–4	21.9 (14)	19.7 (14)	24.1 (18)	22.7 (17)	21.5 (15)	
		5–49	3.5 (9)	3.4 (10)	4.8 (16)	3.8 (15)	2.3 (10)
		50+	25.1 (6)	27.9 (7)	30.3 (8)	20.5 (6)	22.4 (7)
Total			14.1 (551)	18.8 (801)	29.9 (1,327)	10.1 (483)	7.1 (351)

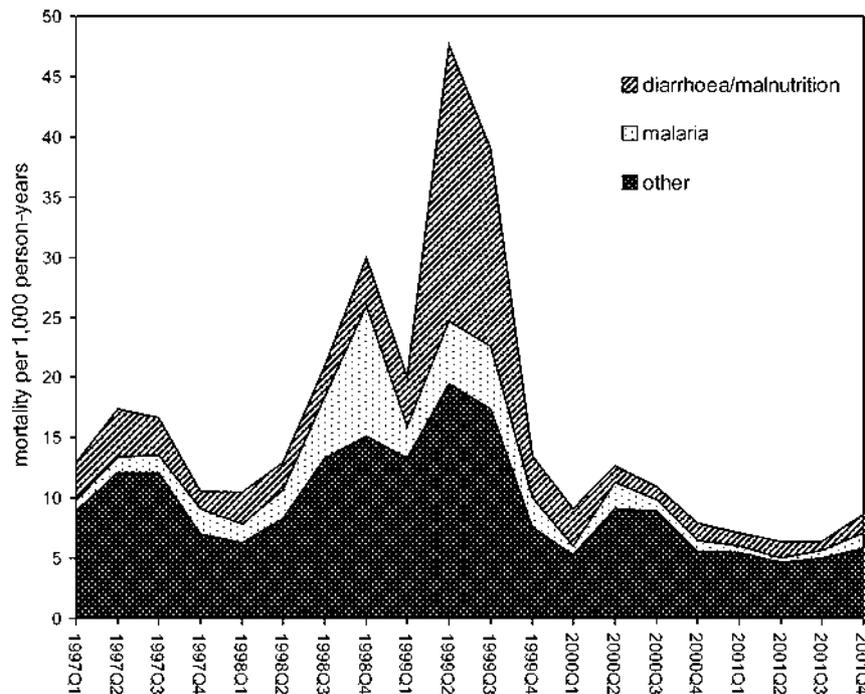


Fig. 2. Overall mortality rates by quarter for the period 1997–2001 in Butajira, showing mortality fractions for deaths reported to be related to malaria and diarrhoea/malnutrition.

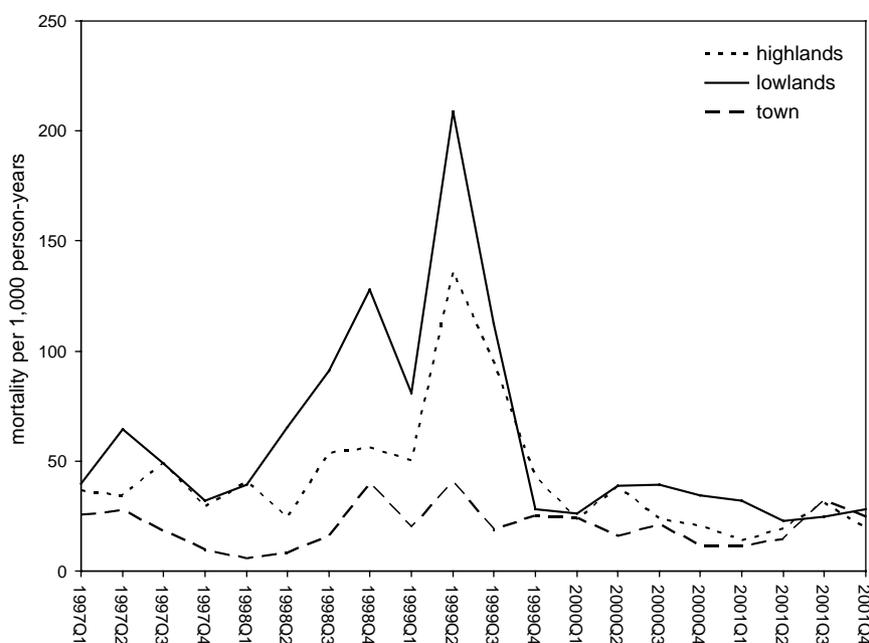


Fig. 3. Under-five mortality rates by quarter for the period 1997–2001 in Butajira.

logistic regression analysis investigating the characteristics of rural people who died during 1998–1999 from malaria ($n=364$) and diarrhoea/malnutrition ($n=614$), respectively, compared with deaths from all other causes ($n=1,000$). Malaria deaths were more likely in those over the age of 5 years, while diarrhoea/malnutrition deaths were less likely between the ages of 5 and 50 years. Deaths from malaria were more likely during 1998 and from diarrhoea/malnutrition during 1999 (Fig. 2). Having a

protected water supply was significantly protective against diarrhoea/malnutrition deaths. Smaller number of household members was a protective factor against malaria deaths as well as against diarrhoea/malnutrition deaths. Living in the lowland areas increased malaria deaths but possibly protected against dying from diarrhoea/malnutrition.

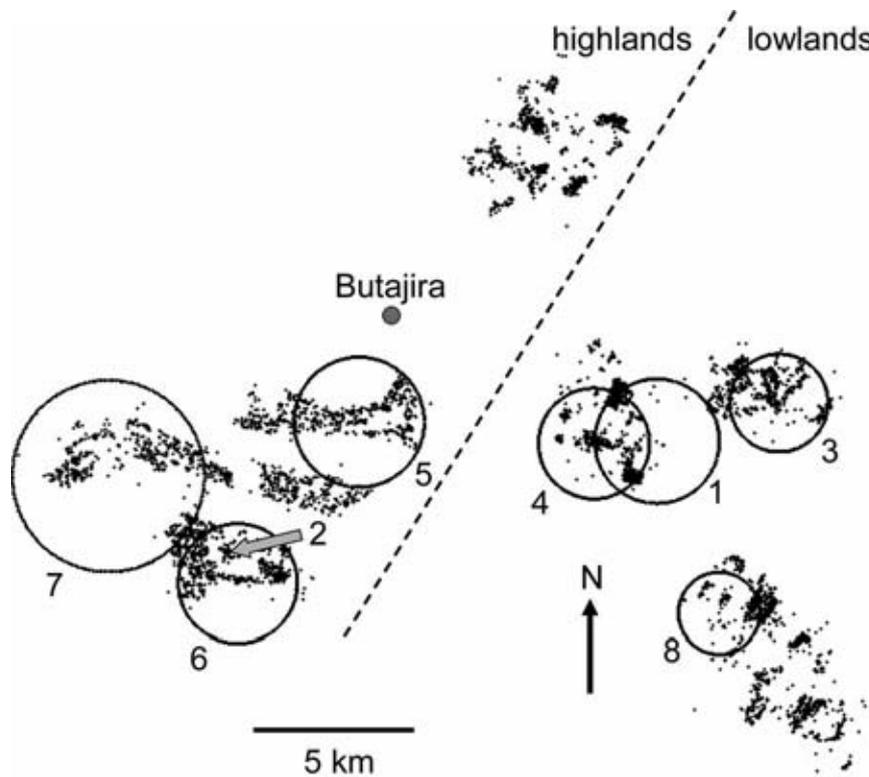
Space-time clustering analyses were undertaken for the 364 malaria deaths and 614 diarrhoea/malnutrition

Table 2. Factors associated with deaths from malaria (364) or diarrhoea/malnutrition (614) compared with deaths from all other causes (1,000 deaths) in the rural areas of Butajira during 1998–1999. Results from multivariate regression analyses*. Confidence intervals are corrected for sampling effect of household clustering

Factor	Level	Malaria		Diarrhoea/malnutrition	
		Multivariate odds ratio	95% CI	Multivariate odds ratio	95% CI
Sex	Female	Ref	–	Ref	–
	Male	1.15	0.89–1.47	0.84	0.68–1.03
Age group	Under 5 yrs	Ref	–	Ref	–
	5–49 yrs	1.73**	1.32–2.27	0.82	0.65–1.04
	50+ yrs	1.87**	1.31–2.65	1.36**	1.03–1.80
Water source	Unprotected	Ref	–	Ref	–
	Protected	0.90	0.65–1.25	0.69**	0.51–0.94
Household members	Less than 5	Ref	–	Ref	–
	5 or more	1.29**	1.01–1.66	1.26**	1.02–1.56
Area	Highland	Ref	–	Ref	–
	Lowland	1.63**	1.25–2.14	0.80**	0.64–1.00

*Models including sex, age, source of drinking water, #household members and area.

**Significant at the 95% level.



cluster	cause	radius (km)	from	to	deaths/ population	RR	p
1	malaria	1.98	10/98	9/99	58/3153	4.03	0.001
2	malaria	0.06	7/99	12/99	5/25	77.6	0.008
3	malaria	1.56	7/99	12/99	26/2851	3.68	0.030
4	diarrhoea	1.76	4/99	9/99	98/3500	7.41	0.001
5	diarrhoea	2.07	4/99	9/99	72/2916	6.25	0.001
6	diarrhoea	1.89	4/99	9/99	56/3426	4.00	0.001
7	diarrhoea	3.05	4/99	6/99	31/3376	4.38	0.001
8	diarrhoea	1.29	4/99	6/99	20/1504	6.27	0.001

Fig. 4. Space–time clustering analysis of malaria deaths ($n=364$) and diarrhoea/malnutrition deaths ($n=614$) among 7,411 households (small dots) during 1998 and 1999 in the Butajira district. All the eight clusters shown represent areas/periods of significantly high mortality, as detailed at the base of the figure.

deaths, respectively, among the 7,411 rural households surveyed. This revealed three clusters with statistically significant raised incidences of malaria deaths and five with raised diarrhoea/malnutrition deaths, as shown in Fig. 4.

The differences in rural overall, all-cause mortality between smaller and larger farmers over the period 1997–2001 is shown in Fig. 5 as adjusted rate ratios (RR) for each quarter of follow-up. There are two periods when the mortality gap widens, the RR increase and are statistically significantly greater than 1. The first is in the third quarter of 1998 and the second in quarters 1–3 of 1999.

These RR peaks nearly coincide with the two mortality peaks (Figs. 2 and 3), but precede them by one month.

The consequences of the famine on reproductive health are shown in Fig. 6, for the rural and town population combined. All-cause mortality among women aged 15–44 years in 1999 (11.63 per 1,000 p-y) was more than double that in the remainder of the 5-year period (5.45 per 1,000 p-y, RR 2.14, 95% CI 1.71–2.67), and at the same time a marked drop in birth rate was evident. Still births showed a later peak, in 2000, of 36.3 per 1,000 live births, a rate more than double that in the remainder of the 5-year

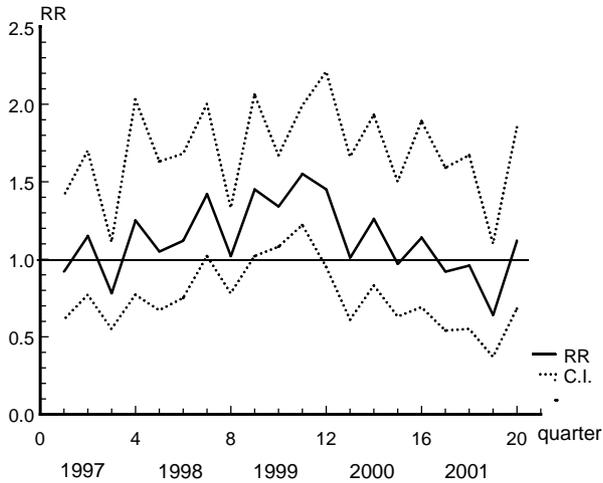


Fig. 5. Overall, all-cause rural mortality rate ratios, smaller vs larger farms, by quarter, Mantel-Haenszel adjusted, 95% CI.

period (14.6 per 1,000 live births, RR 2.50, 95% CI 1.79–3.48). The BRHP cohort is generally too small for year-by-year analysis of maternal deaths, but there were no obvious trends during this period.

Discussion

There is no question that these analyses reveal a serious humanitarian crisis with the Butajira population during 1998–1999, which also met the CDC guideline definition. Nevertheless, Butajira District at the time did not convey the impression of a population in crisis, nor were any substantial humanitarian relief efforts triggered. The

epidemiological analysis reveals that, in common with many historic famines, the direct causes of excess mortality were predominantly diarrhoeal disease and malaria, occurring as two distinct epidemics within the space of a few months.

In the case of the malaria epidemic in 1998, the mortality patterns have to be understood against the complex epidemiological patterns of malaria in highland Ethiopia. Butajira District does not experience the endemic malaria seen elsewhere in Africa, as a result of climate and altitude. The highland areas rarely experience malaria outbreaks at all, but when they do, for example as a result of seeding by gametocyaemic travellers, the effects can be very serious, since there is no substantial immunity in any age group. An extreme example of this is evident from the clustering analysis shown in Fig. 4, where 5/25 residents living within a 60 m radius (cluster 2) died from malaria in one highland village. In the so-called lowland areas, which are still substantially above sea level, there are more frequent outbreaks of seasonal malaria, but relatively low transmission levels and therefore poor individual immunity still mean that there can be considerable all-age mortality as a result. It may also have been the case here that nutritional stress and high mortality increased mobility in the population, either to seek food or attend funeral ceremonies, and this may have exacerbated the spread of malaria outbreaks into the highland villages.

The substantial epidemic of diarrhoeal disease mortality in mid-1999 is notable both for its magnitude and short duration, as well as its drastic effects among

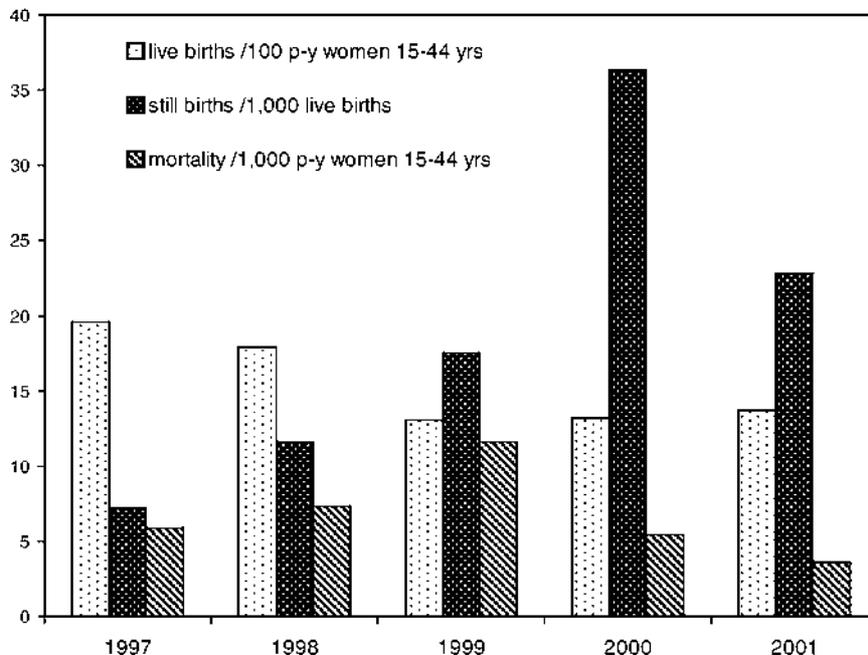


Fig. 6. Effects of famine on reproductive health indices in Butajira during 1997–2001.

children, comparing quarter 2, 1999 to the same quarters before and after (Fig. 3). This is indicative of acute, virulent disease such as cholera or other similar diarrhoeal diseases. Clearly there is no data available on the aetiology of this outbreak, though the substantial survival advantage of protected water sources suggests that it was a water-borne infection. As with malaria, increased mobility at the time may have contributed to the spread of the epidemic from village to village.

For both the diarrhoeal and malarial epidemics, it is clear from the clustering analysis that, as might be expected in relatively short-term epidemics of infectious disease, there were a number of high-incidence areas, while other places were more fortunate. It is notable that neither epidemic took a major hold within Butajira town (Fig. 3), despite being less than 1 km away from a nearby village in which 1 in 40 of the population died from diarrhoea (Fig. 4, cluster 5). Exactly what protected the town is not clear; the availability of better water supplies and housing may have contributed, but it may also have been due to not being part of the subsistence economy to start with, and hence not nutritionally compromised at the time of the epidemic.

The fact that the RR peaks in Fig. 5 precede the mortality peaks in Figs. 2 and 3 may be taken to show that the smaller farmers, at least in the rural areas, respond earlier than those moderately better off to the crisis, with an increasing mortality. 'Smaller farmers', with 0–1.5 hectares of land, in the context is mainly subsistence farmers, with no or little surplus crop to trade for cash.

The effects on reproductive indices (Fig. 6) are striking. Evidence from China has linked nutritional stress to increased foetal loss and decreased birth rates (15), but it is not clear in these data why the peak in stillbirths was as late as 2000, nor indeed whether this late peak was an effect of the same cause.

The Butajira area with its location in the central highlands of Ethiopia has not come into focus as a location for the severe famines reported from the northern and eastern part of the country (8). However, it seems likely that the poorest in the farming communities are as vulnerable as the pastoralists in the North and East. Reports of cash crop yields from Butajira villages show little variation over the period 1997–2001. Together with the fact that the mortality peaks in 1998–1999 were minor in Butajira town, this could indicate that it is those who are most unconnected to a cash economy and are most reliant on subsistence farming that experience most of the excess mortality.

A weakness of this analysis is that we have no primary data on nutritional status. The 1999 national survey (7) included regional-level data, which rated SNNPR as one of the lowest in Ethiopia (and in Africa) at that time, with 1,529 kCal per capita per day and 81.9% of the

population being food-energy deficient. This is a clear indication of the poor nutritional status in the area at the time, and was measured over several seasons of the year, but does not provide any insight as to how food insecurity might have been changing year by year. On the other hand, these levels of mortality have not been seen at any other time since the inception of BRHP in 1987, which, together with local evidence of drought and famine, suggest that this crisis was a major demographic and health event.

The attribution of cause of death during this period was also not totally robust. However, the large excess number of deaths due to diarrhoea and malaria in a short period of time, as shown in Fig. 2, are not likely to have happened by chance. Death following acute diarrhoea, particularly during an epidemic, is not difficult for relatives to recognise and remember. It could be argued that deaths reported as malaria could be mistaken for other acute febrile illnesses. However, the larger proportion of malaria deaths in lowland areas and the seasonal pattern both add verisimilitude to the data. Measles did not play any major part in the observed mortality rates, probably due to relatively good levels of immunisation. The overall pattern of mortality is also very similar to other famines documented in the past. For example, taking the serious famine that Sweden experienced nationally in 1773, annual mortality rates for the period 1771–1775 were 27.7, 37.6, 53.2, 22.3 and 24.7 per 1,000, closely similar to the experience of Butajira more than two centuries later.

With hindsight, it is also clear that the surveillance system in BRHP should have picked up the excessive rates of mortality at the time, and used the information to trigger appropriate interventions (16). This did not happen, and perhaps demographic surveillance sites (DSS) in general should give more thought to implementing mechanisms for raising immediate alarms whenever, for example, the one death per 10,000 per day threshold in crude mortality is reached (17).

In conclusion, the people of Butajira experienced an insidious yet devastating famine at the turn of the millennium, at a time when most of the world has consigned such events to history. Food insecurity and reliance on subsistence rain-fed agriculture continue to be major issues in this and similar rural communities. Epidemics of traditional infectious diseases can still be devastating, given opportunities in nutritionally challenged populations with little access to health care. More action could and should have been taken in this case, not least by using the surveillance system to flag the emergency. With continuing climate change, episodes like the 1998–1999 may become more frequent, thus increasing the need for weather warning systems to be extended to subsistence farmers as well as for local measures to meet local food shortages.

References

1. Sen A. Population and reasoned agency: population growth and food security. *Development* 2007;50(S1):98–102.
2. Webb P, von Braun J, Yohannes Y. Famine in Ethiopia: policy implications of coping failure at national and household levels. Research Report 92. Washington, DC: International Food Policy Research Institute; 1992.
3. Lindtjorn B. Famine in southern Ethiopia 1985–6: population structure, nutritional state and incidence of death among children. *Br Med J* 1990;301:1123–7.
4. Kigutha HN, van Staveren WA, Hautvast JGAJ. Elderly under nutritional stress: a seasonal study on food consumption and nutritional status in Kenya. *Int J Food Sci Nutr* 1998;49:423–33.
5. Salama P, Assefa F, Talley L, Spiegel P, van der Veen A, Gotway CA. Malnutrition, measles, mortality, and the humanitarian response during a famine in Ethiopia. *JAMA* 2001;286:563–81.
6. Macfarlane A. The dimensions of famine. 2002. <http://www.alanmacfarlane.com/savage/A-FAM.PDF> [cited December 2008]
7. Smith LC, Alderman H, Aduayom D. Food insecurity in sub-Saharan Africa: new estimates from household expenditure surveys. Research Report 146. Washington, DC: International Food Policy Research Institute; 2006.
8. Hammond L, Maxwell D. The Ethiopian crisis of 1999–2000: lessons learned, questions unanswered. *Disasters* 2002;26:262–79.
9. Berhane Y, Wall S, Kebede D, et al. Establishing an epidemiological field laboratory in rural areas-potentials for public health research and interventions. The Butajira Rural Health Programme 1987–1999. *Ethiop J Health Dev* 1999;13:1–47.
10. Fantahun M, Fottrell E, Berhane Y, Wall S, Högberg U, Byass P. Assessing a new approach to verbal autopsy interpretation in a rural Ethiopian community: the InterVA model. *Bull World Health Organ* 2006;84:204–10.
11. Kulldorff M. A spatial scan statistic. *Commun Statist Theory Meth* 1997;26:1481–96.
12. Kulldorff M and Information Management Services, Inc. SaTScanTM v6.1: Software for the spatial and space-time scan statistics. <http://www.satscan.org/>, 2006.
13. Ghebreyesus TA, Byass P, Witten KH, et al. Appropriate tools and methods for tropical microepidemiology: a case-study of malaria clustering in Ethiopia. *Ethiop J Health Dev* 2003;17:1–8.
14. Centers for Disease Control. Famine-affected, refugee, and displaced populations: recommendations for public health issues. *MMWR* 1992; 41:RR13.
15. Cai Y, Feng W. Famine, social disruption, and involuntary fetal loss: evidence from Chinese survey data. *Demography* 2005;42:301–22.
16. Byass P, Berhane Y, Emmelin A, et al. The role of Demographic Surveillance Systems (DSS) in assessing the health of communities: an example from rural Ethiopia. *Public Health* 2002;116:145–50.
17. Fottrell E, Byass P. Identifying humanitarian crises in population surveillance field sites: simple procedures and ethical imperatives. *Public Health*, in press.

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Using high spatial resolution remote sensing for risk mapping of malaria occurrence in the Nouna district, Burkina Faso

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Introduction: Malaria control measures such as early diagnosis and treatment, intermittent treatment of pregnant women, impregnated bed nets, indoor spraying and larval control measures are difficult to target specifically because of imprecise estimates of risk at a small-scale level. Ways of estimating local risks for malaria are therefore important.

Methods: A high-resolution satellite view from the SPOT 5 satellite during 2008 was used to generate a land cover classification in the malaria endemic lowland of North-Western Burkina Faso. For the area of a complete satellite view of 60 × 60 km, a supervised land cover classification was carried out. Ten classes were built and correlated to land cover types known for acting as Anopheles mosquito breeding sites.

Results: According to known correlations of Anopheles larvae presence and surface water-related land cover, cultivated areas in the riverine vicinity of Kossi River were shown to be one of the most favourable sites for Anopheles production. Similar conditions prevail in the South of the study region, where clayey soils and higher precipitations benefit the occurrence of surface water. Besides pools, which are often directly detectable, rice fields and occasionally flooded crops represent most appropriate habitats. On the other hand, forests, elevated regions on porous soils, grasslands and the dryer, sandy soils in the north-western part turned out to deliver fewer mosquito breeding opportunities.

Conclusions: Potential high and low risks for malaria at the village level can be differentiated from satellite data. While much remains to be done in terms of establishing correlations between remotely sensed risks and malaria disease patterns, this is a potentially useful approach which could lead to more focused disease control programmes.

Keywords: *high spatial resolution; remote sensing; malaria; West Africa; Burkina Faso; Anopheles; risk mapping; SPOT 5 satellite*

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There is a widespread consensus on malaria control measures, including early diagnosis and treatment, intermittent treatment of pregnant women, impregnated bed nets, in-door spraying and larval control measures. The policy conundrum, however, is the low and often unfocused coverage with these measures. In the study district of Nouna, Burkina Faso, only 8% of children actually sleep under insecticide treated bed nets (and about 20% of children with fever present at health centres get appropriate treatment in case of

malaria) (1, 2). Neither larval control measures nor indoor spraying is practised.

There are two different policy options to respond to this unfortunate situation in a holo-endemic area. One would propose an overall improvement of the effectiveness of health systems to deliver the control measures to the entire population. This is based on good public health practice and theory which stipulates that when conditions or risk factors are highly prevalent in a population, it is best to offer control

measures to everyone irrespective of the level of risk exposure.

The alternative – unorthodox – approach proposed here is based on the assumption that the financial and logistical constraints of health systems in districts such as the one under study are so formidable that focusing measures on populations at high risk of transmission is justified. No one would challenge a *temporal* focus in an area of highly seasonal transmission. Following this rationale, the recent WHO malaria report (3) suggests distributing bed nets and drugs before the rainy season so that populations have better access during the peak transmission season.

In this paper, we argue that an additional *spatial* focus should be considered. This is based on consistent findings in the study area of very varied malaria incidence rates between even adjacent villages (4). Regions in and close to Sahel are known for very focal and seasonal transmission (5–8). The combination of the advent of low-cost high-resolution remote sensing and reports of different malaria transmission risks based on different surface water quality, size and land cover led us to carry out the current study. The main objective of this paper is to answer the question to which extent remote sensing can validly identify different larval habitats producing different malaria transmission risks. The spatial resolution of sensors is still limited to habitats at least several metres in diameter, and the revisit rate of high-resolution satellites is too low to map dynamic changes.

We are of course aware that, having answered this question, further studies would be needed on

- 1) the statistical associations between remotely sensed risk zones and actual entomological data within them;
- 2) the relationship between both entomological and remotely sensed data, and incident malaria cases and their severity, together with malaria mortality; and
- 3) the evaluation of cost effectiveness of interventions in a target area. These could be raising bed net coverage in high-risk areas, coupled with larval control and indoor spraying. This should be carried out through cluster-randomised and controlled intervention studies.

The next generation of satellites will deliver new dimensions of spatial resolution within the sub-metre range and hence allow detection of even smaller habitats. The more limiting factor will still be the flyover frequency, so even usage of a higher spatial resolution will require temporal modelling of habitat dynamics. New and original approaches on dynamics have been set up for others diseases such as Rift Valley fever in Senegal (9). The predominant percentage of prevalent surface water and related land cover is already detectable with current technology. Risks emerging from small-scale

water agglomerations, e.g. puddles, skid marks, etc. that often do not evaporate completely for periods of several weeks has to be modelled from the implications of their characteristic larvae production, occurrence and duration since they cannot be detected directly via remote sensing.

Materials and methods

The study site lies in the north-western part of Burkina Faso in the Kossi district and correlates to the satellite view of the SPOT 5 satellite from 2008. In the centre of this area, which is 60 × 60 km, the village of Nouna is located at 12° 44' N; 3° 51' W. Most areas in this region lie on an altitude of 150–250 m above the sea level and belong to a Precambrian peneplain. Mean precipitation for Nouna during the last 10 years has been 817 mm per year. The monthly maxima during the rainy season between May and September can reach up to 350 mm. The yearly average temperature of Nouna is 27.8°C.

While some studies (10) have dealt with an extensive collection of ground data for a relatively small study site of few square kilometres, for this study wide parts of a 3,600 km² satellite view were used to map habitats which are known to be appropriate for *Anopheles gambiae* breeding from other studies. A SPOT 5 (Satellite Pour l'Observation de la Terre) satellite image was utilised for this study. Since the study area was visited during late rainy season and the collection of ground truth points had to be close in time to the flyover, a satellite view of 1 September 2008 was programmed. The multispectral image used consists of three bands (red, green and near infrared) and resolution is 2.5 m per pixel. Images were received orthorectified and georeferenced (level 3) in UTM system (zone 30P).

Training zones

During the six-week field phase from August to October 2008, an overall number of 45 ground truth points were taken in different geographic regions within the satellite view in order to produce a classification scheme (Fig. 1).



Fig. 1. Characteristic pool in the vicinity of Nouna. In some parts it is used as brickyard while other parts show lateritic substratum on the ground. (Location: see Fig. 5).

These ground truth points are objects in the terrain that are needed for recognising different land cover in the satellite image. Knowing the location and land cover type of ground truth points in the terrain allows the determination of similar zones in the satellite image. Ground truth points contained rice fields, sorghum, water pools, bare soil, buildings, bush, etc. Most locations were visited contemporaneously with satellite overflight; positions being recorded using GPS handheld receivers (Garmin GPS Map 76s). All ground truth objects were saved as waypoints, and some additionally as polylines using the track recording function of the GPS.

Supervised classification

For analysis of the SPOT image ITTVIS, ENVI image processing software was used. The image was classified by using validation data collected during a six-week field study in 2008. Using the Region of Interest-Tool (ROI) in ENVI, 45 ground truthing points were used for spectral reference. These training signatures were distributed in 15 classes, which were merged later into 10 classes to run the classification. Using three bands (red, green and near infrared), the image was processed using the maximum likelihood calculation for supervised classifications. The maximum likelihood classification assumes that the statistics for each class in each band are normally distributed and calculates the probability that a given pixel belongs to a specific class. During the process each pixel is assigned to the class that has the highest probability; if the highest probability is smaller than a specified threshold, the pixel remains unclassified.

Data analysis

The classified satellite image was saved in ENVI as an ASCII file. The ASCII format was then transformed into a raster file using ArcMaps integrated conversion tools. This procedure allowed keeping the calculated classes in ArcMap in a selectable raster dataset. Classes were renamed and fitted to original colour set. For 30 villages, buffers of 500 m radius were constructed around the centre using ArcMaps buffer wizard. These buffer zones represent the assumed Anopheles mosquito flying range (11, 12). The surface of each class within the radius around each village was calculated. This was performed using the 'zonal histogram' tool in the 'spatial analyst' extension in ArcMap. According to data from the 2008 study, as well as to typical Anopheles presence in different land cover types known from literature, the land cover types have been evaluated (10, 13–23). Since for this region there are no existing studies that deal with absolute numbers of mosquito larvae per habitat per time, a relative risk classification was constructed. Four classes of relative mosquito larvae presence in environmental habitats from low to very high were incremented (see Fig. 2).

No.	Land cover class	Risk level
1	Sandy soil	Low
2	Bare soil	Low
3	Dry vegetation	Low
4	Housing	Medium
5	Forest and bush	Medium
6	Field crops	High
7	Turbid water	High
8	Rice field (submerged/irrigated)	Very high
9	Submerged vegetation	Very high
10	Water covered with vegetation	Very high

Fig. 2. Land cover classes and risk levels according to various literature (10, 13–23).

The percentage of very high and high-risk land cover within the 500 m buffer zone of all villages was compared in a diagram and sorted by percentage (see Fig. 4). On the base of this graduation, two groups of villages were featured, one with a percentage of risk-related land cover lower than 25% of area with high and very high risk, another with more than 25%. This threshold marks at the same time a significant increase in very high risk land cover per village. In ArcMap, villages were redrawn on the satellite image indicating their calculated risk (see Fig. 5).

Results

For the 30 villages included in the local demographic surveillance system, the area of potential habitats with very high risk (submerged and irrigated rice fields, water covered with vegetation and submerged vegetation) and high risk (field crops with clayey soil and turbid water) was calculated for the 500 m buffer zone. The share of total surface accounted for by very high and high-risk habitats within the buffers showed a difference between the lowest and highest by nearly a factor of 20. Some villages (Dembelela) had around 3% of surface within the 500 m buffer covered with very high and high-risk habitats, while it reached up to 60% in the vicinity of other villages (Sere, Tissi). This is shown in Fig. 3.

Villages that already showed a high percentage of high-risk land cover within their 500 m buffer zone also had a higher percentage of very high-risk land cover types (see Fig. 4). Since risk is defined as appropriateness for larvae breeding, which is bound to surface water, the results show that an underlying factor exists that influences the presence or absence of both risk types at the same time.

Villages with similar risks turned out not to be randomly distributed over the survey area but lay together in certain regions. Two zones around villages with elevated risk (risk-related land cover share higher than 25%) and three zones containing villages at lower risk could be separated (see Fig. 5). These zones alternated from South-West to North-East. This remarkable difference in distribution of natural and anthropogenic land cover between regions seems to

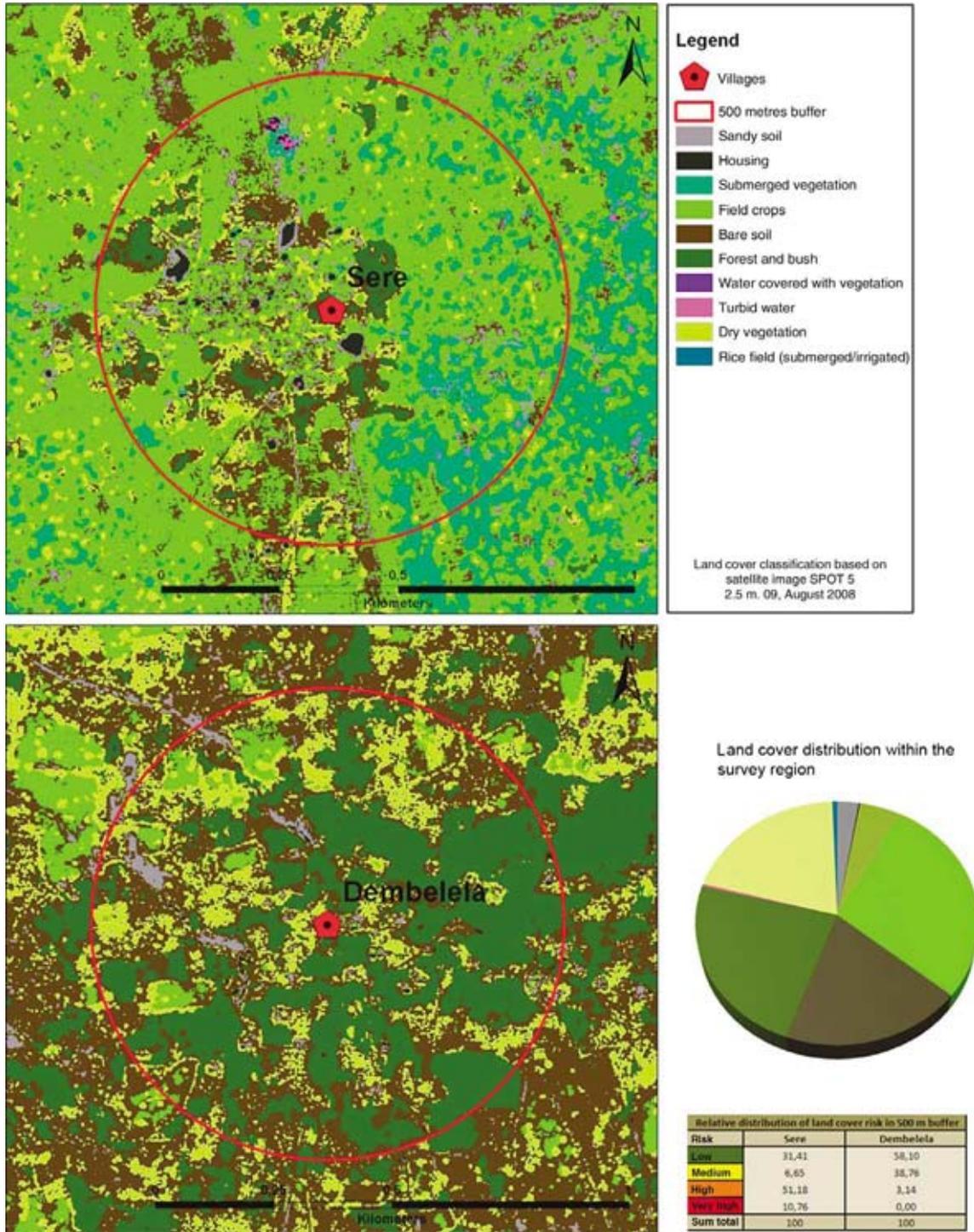


Fig. 3. Land cover distribution in 500 m buffer zone around the villages of Sere (highest risk) and Dembelele (lowest risk). Land cover risk within the survey region (pie diagram, same legend).

have its origin in the natural distribution of geographic and geologic factors. Suitable factors could be the prevailing type of soil and or as additional effect depressions in topography. Those depressions played a role in regional water distribution although only showing height differences of 15 m or less. Since the survey region was relatively

small and risk zones alternated within it, climatic differences do not seem capable of explaining those distributions. Soil types in this region often vary within small areas and show considerable differences in infiltration behaviour and water retention capacity. Some areas have mostly sandy soils, which leads to less environmental water

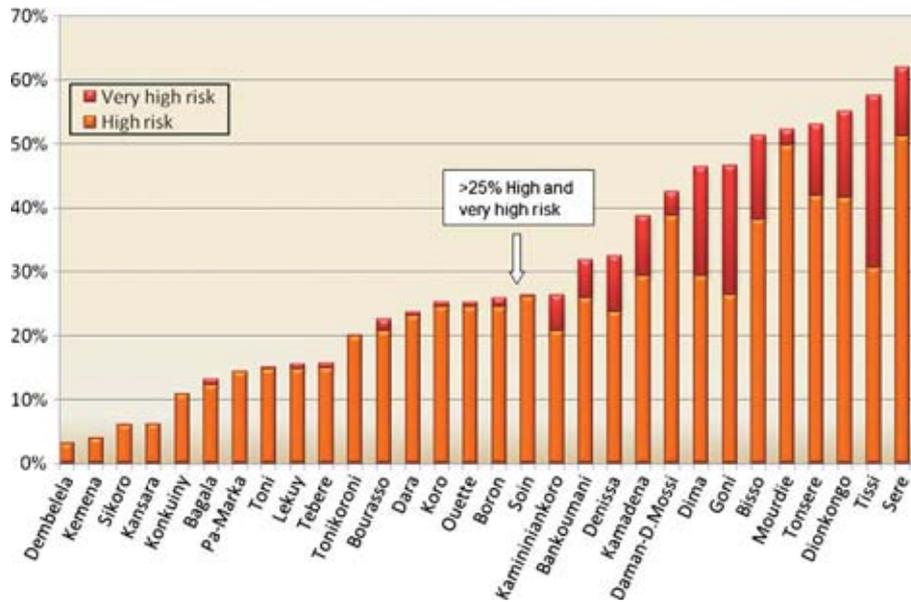


Fig. 4. Percentage of land cover areas with elevated risk for *Anopheles* larvae breeding of total surface in 500 m buffer zones around 30 villages.

reservoirs. Regions with clayey soils often show swampy characteristics during the rainy season and keep water for several days or weeks. Lateritic crusts at the surface are a prevalent type of substratum as well and allow nearly no infiltration but high runoff rates (see Fig. 5).

Discussion

We showed and validated with ground data that high-resolution satellite images can indeed identify small-scale habitats with sizes of only few metres diameter conducive to *Anopheles* larvae development. Micro habitats in the sub-metre scale are not directly detectable at the current state of technology, but need imputation via modelling in further studies. Those micro-habitats mainly play a role within villages where they are close to the population and this mostly during the peak of the rainy season. After more than one week without precipitation they mostly evaporate or are infiltrated and cannot act as productive habitats. Being not directly detectable, these micro-habitats need to be estimated by their average occurrence in typical villages and their occurrence attached to different land cover types. The extremely varied micro-distribution of risks between villages is compatible with the findings of Yé et al. (24), who reported considerable differences in malaria incidence between villages in the region.

The most extensive work on geographical variation of malaria risk in Africa has been made at the continental scale, based on meteorological data and historical ground data from various sites across the continent (25, 26), but using a much coarser resolution and were not useful for malaria control at the district level. At the time those studies were carried out, the current resolution was not

available. Studies mapping *Anopheles* mosquito breeding habitats, transmission or disease, partly with higher resolution, have been made in Africa (8, 22, 25, 27–31) and South and Central America (32–35). Reliable information about vector density and malaria transmission risk is essential for understanding variations in local disease epidemiology and to stratify intervention programmes. The next step is to correlate malaria case data from the demographic surveillance system with the risk modelled by using high-resolution satellite imagery.

We are aware that there is a long and non-linear causal pathway between the number of larvae in a given habitat and the incidence, severity and cause-specific mortality of malaria so we urge for prudence in interpreting our data. Our mapping of villages into two risk categories for malaria transmission is a first step towards exploring the usefulness of targeted control measures. As pointed out in the introduction, our findings need to be connected with entomological and clinical data. On the basis of further results the application of counter measures can be considered. Since risk seems to be focused on certain zones, interventions like bed net distribution and indoor-spraying, but also the use of bacteria produced toxins that selectively kill larvae of certain mosquito species (36) seem to be putative approaches. It will only be after carefully designed intervention studies that any policy implications can be considered.

Conflict of interest and funding

The authors have not received any funding or benefits from industry to conduct this study.

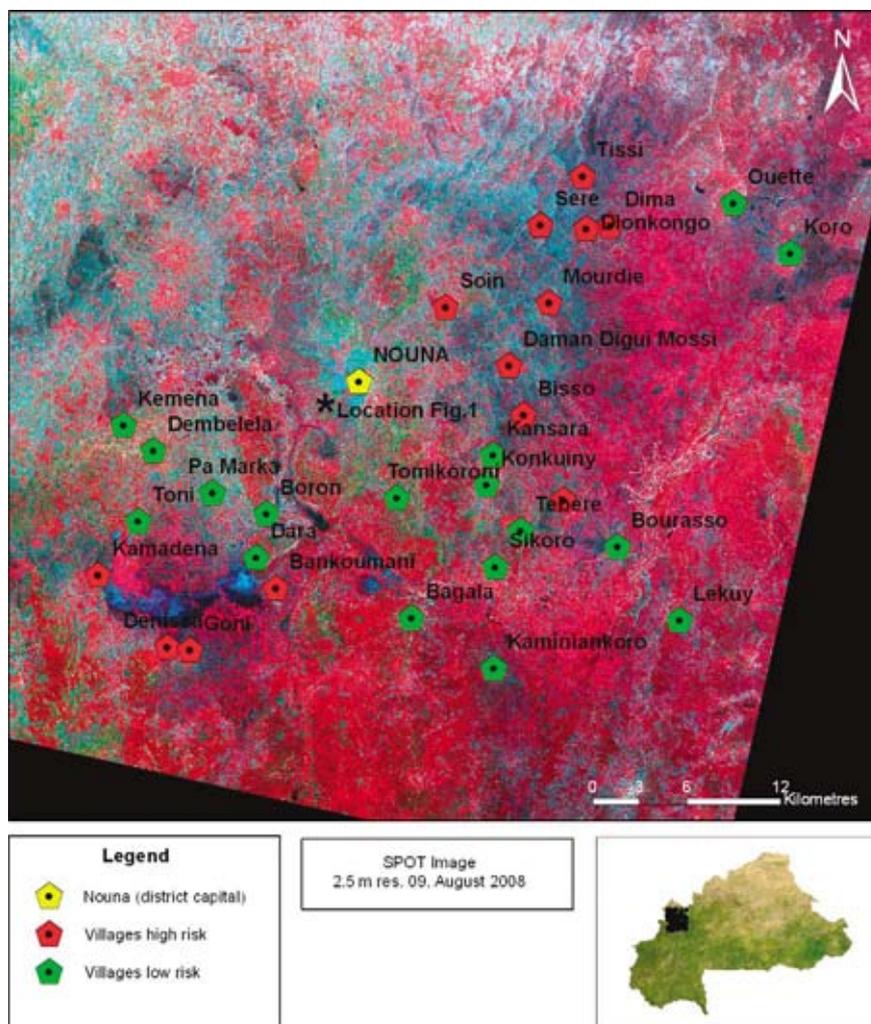


Fig. 5. Villages with similar land cover risk in their 500 m buffer zones. Similar risks show spatial agglomeration in certain zones. Villages with high-risk habitats exceeding the 25% threshold in Fig. 4 have significantly higher land cover with very high risk (red columns in Fig. 4). The asterisk marks the position where Fig. 1 was photographed.

References

- Krause G, Sauerborn R. Comprehensive community effectiveness of health care. A study of malaria treatment in children and adults in rural Burkina Faso. *Ann Trop Paediatr* 2000; 20: 273–82.
- Pfeiffer K, Somé F, Müller O, Sie A, Kouyate B, Haefeli WE, et al. Clinical diagnosis of malaria and the risk of chloroquine self-medication in rural health centres in Burkina Faso. *Trop Med Int Health* 2008; 13: 418–26.
- WHO. World malaria report. Geneva, Switzerland: WHO; 2008.
- Yé Y, Hoshen M, Louis V, Simboro S, Traoré I, Sauerborn R. Housing conditions and *Plasmodium falciparum* infection: protective effect of iron-sheet roofed houses. *Malar J* 2006; 5: 8–15.
- Briet OJT, Dossou-Yovo J, Akodo E, van de Giesen N, Teuscher TM. The relationship between *Anopheles gambiae* density and rice cultivation in the savannah zone and forest zone of Cote d'Ivoire. *Trop Med Int Health* 2003; 8: 439–48.
- Diuk-Wasser MA, Toure MB, Dolo G, Bagayoko M, Sogoba N, Sissoko I, et al. Effect of rice cultivation patterns on malaria vector abundance in rice-growing villages in Mali. *Am J Trop Med Hyg* 2007; 76: 869–74.
- Robert V, Gazin P, Carnevale P. Malaria transmission in three sites surrounding the area of Bobo Dioulasso (Burkina Faso): the savanna, a rice field and the city. *Bull Soc Vec Ecol* 1987; 12: 41–3.
- Thomas CJ, Lindsay SW. Local-scale variation in malaria infection amongst rural Gambian children estimated by satellite remote sensing. *Trans R Soc Trop Med Hyg* 2000; 94: 159–63.
- Vignolles C, Lacaux JP, Tourre YM, Bigeard G, Ndione JA, Lafaye M. Rift Valley fever in a zone potentially occupied by *Aedes vexans* in Senegal: dynamics and risk mapping. *Geospat Health* 2009; 3: 211–20.
- Mutuku FM, Bayoh MN, Hightower AW, Vulule JM, Gimig JE, Mueke JM, et al. A supervised land cover classification of a western Kenya lowland endemic for human malaria: associations of land cover with larval *Anopheles* habitats. *Int J Health Geogr* 2009; 8: 19–32.
- Costantini C, Li SG, DellaTorre A, Sagnon N, Coluzzi M, Taylor CE. Density, survival and dispersal of *Anopheles gambiae* complex mosquitoes in a West African Sudan savanna village. *Med Vet Entomol* 1996; 10: 203–19.

12. Ejercito A, Urbino M. Flight range of gravid and newly emerged Anopheles. *Bull World Health Organ* 1951; 3: 663–71.
13. Charoenpanyanet A, Chen X. Satellite-based modeling of Anopheles mosquito densities on heterogeneous land cover in Western Thailand. *The International Archives of the Photogrammetry, Remote Sensing and Spatial Information Sciences* 2008; 27: 159–64.
14. Gimnig JE, Ombok M, Otieno S, Kaufman MG, Vulule JM, Walker ED. Density-dependent development of *Anopheles gambiae* (Diptera: Culicidae) larvae in artificial habitats. *J Med Entomol* 2002; 39: 162–72.
15. Minakawa N, Mutero CM, Githure JI, Beier JC, Yan GY. Spatial distribution and habitat characterization of Anopheline mosquito larvae in Western Kenya. *Am J Trop Med Hyg* 1999; 61: 1010–6.
16. Minakawa N, Sonye G, Mogi M, Yan G. Habitat characteristics of *Anopheles gambiae* s.s. larvae in a Kenyan highland. *Med Vet Entomol* 2004; 18: 301–5.
17. Minakawa N, Munga S, Atieli F, Mushinzimana E, Zhou G, Githeko AK, et al. Spatial distribution of anopheline larval habitats in Western Kenyan highlands: effects of land cover types and topography. *Am J Trop Med Hyg* 2005; 73: 157–65.
18. Minakawa N, Sonye G, Yan GY. Relationships between occurrence of *Anopheles gambiae* s.l. (Diptera: Culicidae) and size and stability of larval habitats. *J Med Entomol* 2005; 42: 295–300.
19. Mohr KI. Interannual, monthly, and regional variability in the wet season diurnal cycle of precipitation in sub-Saharan Africa. *J Climate* 2004; 17: 2441–53.
20. Munga S, Minakawa N, Zhou GF, Barrack OOJ, Githeko AK, Yan GY. Oviposition site preference and egg hatchability of *Anopheles gambiae*: effects of land cover types. *J Med Entomol* 2005; 42: 993–7.
21. Munga S, Minakawa N, Zhou GF, Mushinzimana E, Barrack OOJ, Githeko AK, et al. Association between land cover and habitat productivity of malaria vectors in western Kenyan highlands. *Am J Trop Med Hyg* 2006; 74: 69–75.
22. Machault V, Gadiaga L, Vignolles C, Jarjaval F, Bouzid S, Sokhna C, et al. Highly focused anopheline breeding sites and malaria transmission in Dakar. *Malar J* 2009; 8: 138–59.
23. Pages F, Texier G, Pradines B, Gadiaga L, Machault V, Jarjaval F, et al. Malaria transmission in Dakar: a two-year survey. *Malar J* 2008; 7: 178–89.
24. Yé Y, Kyobutungi C, Louis VR, Sauerborn R. Micro-epidemiology of *Plasmodium falciparum* malaria: is there any difference in transmission risk between neighbouring villages? *Malar J* 2008; 6.
25. Craig MH, Snow RW, le Sueur D. A climate-based distribution model of malaria transmission in sub-Saharan Africa. *Parasitol Today* 1999; 15: 105–11.
26. Snow RW, Craig MH, Deichmann U, le Sueur D. A preliminary continental risk map for malaria mortality among African children. *Parasitol Today* 1999; 15: 99–104.
27. Diuk-Wasser MA, Bagayoko M, Sogoba N, Dolo G, Toure MB, Traore SF, et al. Mapping rice field anopheline breeding habitats in Mali, West Africa, using Landsat ETM+ sensor data. *Int J Remote Sens* 2004; 25: 359–76.
28. Hay SI, Snow RW, Rogers DJ. Predicting malaria seasons in Kenya using multitemporal meteorological satellite sensor data. *Trans R Soc Trop Med Hyg* 1998; 92: 12–20.
29. Hay SI, Guerra CA, Gething PW, Patil AP, Tatem AJ, Noor AM, et al. A world malaria map: *Plasmodium falciparum* endemicity in 2007. *PLoS Med* 2009; 6: e1000048.
30. Machault V, Orlandi-Pradines E, Michel R, Pages F, Texier G, Pradines B, et al. Remote sensing and malaria risk for military personnel in Africa (Reprinted). *J Trav Med* 2008; 15: 216–20.
31. Machault V. Paludisme urbain et télédétection. Master 2: géographique de la santé. Paris X et Paris XII, 2007, [Urban malaria and remote sensing. Master 2 thesis (health geography) at Paris Universities X and XII in 2007].
32. Beck LR, Rodriguez MH, Dister SW, Rodriguez AD, Rejmankova E, Ulloa A, et al. Remote-sensing as a landscape epidemiologic tool to identify villages at high-risk for malaria transmission. *Am J Trop Med Hyg* 1994; 51: 271–80.
33. Beck LR, Rodriguez MH, Dister SW, Rodriguez AD, Washino RK, Roberts DR, et al. Assessment of a remote sensing-based model for predicting malaria transmission risk in villages of Chiapas, Mexico. *Am J Trop Med Hyg* 1997; 56: 99–106.
34. Rejmankova E, Roberts DR, Pawley A, Manguin S, Polanco J. Predictions of adult Anopheles albimanus densities in villages based on distances to remotely-sensed larval habitats. *Am J Trop Med Hyg* 1995; 53: 482–8.
35. Roberts DR, Paris JF, Manguin S, Harbach RE, Woodruff R, Rejmankova E, et al. Predictions of malaria vector distribution in Belize based on multispectral satellite data. *Am J Trop Med Hyg* 1996; 54: 304–8.
36. Fillinger U, Knols BG, Becker N. Efficacy and efficiency of new *Bacillus thuringiensis* var israelensis and *Bacillus sphaericus* formulations against Afrotropical Anophelines in Western Kenya. *Trop Med Int Health* 2003; 8: 37–47.

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Cooking fuels and the push for cleaner alternatives: a case study from Burkina Faso

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Introduction: More than 95% of the population in Burkina Faso uses some form of solid biomass fuel. When these fuels are burned in traditional, inefficient stoves, pollutant levels within and outside the home can be very high. This can have important consequences for both health and climate change. Thus, the push to switch to cleaner burning fuels is advantageous. However, there are several considerations that need to be taken into account when considering the use and promotion of different fuel types.

Objective: In the setting of the semi-urban area of Nouna, Burkina Faso, we examine the common fuel types used (wood, charcoal and liquid petroleum gas (LPG)) in terms of consumption, energy, availability, air pollution and climate change.

Results and conclusion: Although biomass solid fuel does offer some advantages over LPG, the disadvantages make this option much less desirable. Lower energy efficiencies, higher pollutant emission levels, the associated health consequences and climate change effects favour the choice of LPG over solid biomass fuel use. Further studies specific to Burkina Faso, which are lacking in this region, should also be undertaken in this area to better inform policy decisions.

Keywords: *biomass; fuel; Burkina Faso; air pollution; climate change; wood; liquid petroleum gas*

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Biomass fuels, which include wood, charcoal, crop residues and animal dung, are among some of the most widely used for cooking and heating, particularly in developing countries. Reliance on these materials can lead to numerous economic, environmental, social and health problems. Also, other problems that have already arisen may worsen. For example, increasing levels of biomass harvesting and combustion in response to the energy needs of growing populations can have important impacts on the global carbon cycle and consequently climate change. This growing population also faces the problem of having to invest ever-increasing amounts of time and effort to gather these fuels as resources become scarcer, particularly when harvested non-renewably (1, 2).

From indoor to outdoor air pollution

Air pollution is an international issue because of the transnational movement of pollutants across continents and oceans (3). Local sources usually only comprise part of the concentrations of particulate air pollutants in cities (4). However, an overlooked aspect of air pollution is

indoor exposures. It has been estimated that 80% of the total global exposure to airborne particulate matter occurs indoors in developing nations (5). Indoor air pollution, when vented outside from biomass stoves, can also produce significant local outdoor pollution, particularly in dense urban neighbourhoods (6–8).

The bulk of the global indoor air pollution exposures stem from two sources: environmental tobacco smoke and the combustion of solid biomass fuels for cooking and heating (6). However, the burning of biomass fuels is usually conducted under less than ideal conditions, which leads to the incomplete combustion of this material and the subsequent release of a number of compounds, which can be detrimental to health (7) and the environment. These include carbon dioxide (CO₂), carbon monoxide (CO), methane (CH₄), non-methane hydrocarbons (NMHC), nitric oxide (NO), ammonia (NH₃), particulates and inorganics (9).

Another by-product of incomplete combustion is black carbon or soot. Soot, when released into the atmosphere, blocks and absorbs solar radiation, which can greatly

contribute to solar heating (10). Additionally, soot can mix with other aerosols to produce other products of incomplete combustion (PICs), which, in some cases can multiply associated effects (10, 11). Open biomass burning is estimated to be responsible for approximately 42% of black carbon emissions (12).

Many of the by-products of the incomplete combustion of solid biomass fuels are important in terms of climate change. Therefore, interest in the links between solid biomass fuel use and climate change has been growing (8, 13–15). Importantly, the contribution of biomass solid fuel to greenhouse gas (GHG) emissions and its implications for climate change should not be underestimated (9, 15).

Health consequences of biomass smoke exposure from cooking

A further critical consideration of solid biomass fuel use is the associated health effects. Approximately 1.5 million deaths every year from respiratory infections can be attributed to the environment, including the effects of indoor and outdoor air pollution (16). Acute respiratory infections (ARI) in children are one of the leading causes of infant and child morbidity and mortality (17–19). Detailed studies have found strong correlations between the maternally reported number of hours per day children under two years spent by the fire and the incidence of moderate and severe ARI cases in rural Nepal (20). Open wood-fires were also a significant risk factor for ARI among children under three years in a study in Zimbabwe (21). Additionally, girls in the Gambia under five years of age carried on their mother's backs during indoor cooking were found to have a six times higher risk of ARI than other children in the study, which was more than the risk from exposure to environmental tobacco smoke (22).

Chronic obstructive pulmonary disease is another outcome of exposure to high levels of air pollution (23). For example, studies in Nepal (24) and India (25, 26) found that non-smoking women who cooked on biomass stoves had a higher prevalence of this condition than women who used biomass stoves less. Studies in China and one in Japan have also found associations between biomass fuel use and lung cancer. Thirty-year-old women in Japan cooking with straw or wood had an 80% increased chance of having lung cancer later in life (23). Likewise, a study in China found strong associations between lung cancer and use of biomass-burning stoves (27).

At-risk groups

The health risks posed by biomass smoke exposure are generally borne by women and children. There are very few activities that involve as many person-hours as cooking, which is done in every household every day in most of the world, primarily by women (28). The risks

from biomass smoke exposure are also high in young children as they spend large amounts of time with their mothers (8). Children carried on their mother's backs or lap during cooking are often exposed to emissions from biomass fuel combustion from early infancy (22). Coupled with the fact that children's immune systems are still developing and that they have higher mass-specific inhalation rates than adults, biomass smoke exposure could be an important factor affecting the occurrence of diseases (29).

Addressing the problem

In an effort to address this problem, people have been encouraged to move up the 'energy ladder' and use cleaner burning, more efficient fuels as a way to combat the problems associated with the use of biomass solid fuels (23). The energy ladder is made up of several rungs that represent fuels such as wood, charcoal, gas and electricity. Traditional fuels like dung, crop residues and wood typically occupy the lowest rungs. Charcoal, kerosene, gas and electricity represent the higher rungs, in sequential order (30). The cleanest and most desirable fuel substitutes include kerosene, liquid petroleum gas (LPG) and electricity. Wood typically releases 50 times more particulates, CO, formaldehyde, nitrogen oxides and polycyclic aromatic hydrocarbons during cooking than gas (23).

However, as the energy ladder is ascended and emissions decline, fuel costs increase and the availability of these materials also decreases (30). Household income has been shown to be the most important determinant of the choice of household energy in the developing world. Thus, the use of traditional fuels and poverty is very closely interlinked (31). Halving the number of people worldwide cooking with solid fuels by 2015 through the introduction of LPG would cost approximately 13 billion USD per year and provide an economic benefit of 91 billion USD per year (32), a worthwhile investment. Unfortunately, the prediction is that, in the future, the majority of those using biomass will continue to do so in the short and medium-term. However, shortages in supplies stemming from socio-economic and environmental problems will, at the same time, render this option less feasible (23).

Unfortunately, little information exists as to the consumption, availability and climate change effects of different types of fuel used in Burkina Faso, a country with extremely high levels of biomass use (33, 34). Here, we examine the consumption, energy, availability, air pollution and climate change aspects of some of the most commonly used fuels, such as wood, charcoal and LPG, in Burkina Faso. Within this context, we then discuss some of the beneficial and detrimental aspects associated with the use of these fuels and how these could be used to introduce energy policies in Burkina Faso.

Case study: air pollution in Nouna, Burkina Faso

Burkina Faso

Burkina Faso is a landlocked country located in Western Africa made up of 45 provinces with an area of 274,200 km² (35). Approximately 14.4 million people live in Burkina Faso and the current growth rate is 3% per year since 2006 (36, 37). Eighty-one percent of the population live in rural areas (35). Burkina Faso is one of the poorest African countries with 45% of the population living below the poverty line on approximately 1 USD per day in 2006 (36). The average per capita public expenditure on health care is 5.12 USD, which falls far below the Commission on Macroeconomics and Health's recommended minimum for essential health intervention of 30–40 USD per capita per year in developing economies (38).

Over 95% of Burkinabé households use biomass fuels (32). It has been estimated that over 21,500 deaths every year in Burkina Faso are attributable to exposure to indoor biomass smoke (39). The disability adjusted life years (DALYs) associated with indoor air pollution in Burkina Faso were estimated to be 58 per 1,000 capita per year in 2002 compared to 1.7 DALYs per 1,000 capita per year for outdoor air pollution (39). The burden of disease attributable to solid biomass fuel use in Burkina Faso is approximately 8.5% (34).

Nouna, Burkina Faso

Nouna is a semi-urban town located in the Kossi province in northwest Burkina Faso, approximately 300 km from the capital city of Ouagadougou. There are two seasons: the rainy season runs from June to October and the dry from November to May. Like most of Burkina Faso, Nouna is inhabited mainly by subsistence farmers. Previous studies have indicated that biomass fuel use in Nouna is also very high (>98%) (33). Most households (86%) were found to cook outside in the dry season; however, many preferred cooking in sheltered corners or against walls which tended to concentrate smoke from the fires close to the house (33). Those who did not cook

outside, cooked indoors in kitchens with mud roofs and tiny windows with little ventilation. Over half of the households in the study (58%) exclusively used biomass for cooking while 40% used a mix of biomass and other fuels. Only 2% did not use biomass and cooked exclusively with other fuel types. Additionally, 92% of the households in the study burned biomass solid fuel in traditional, three stone stoves. Combined, these factors resulted in very high pollutant exposures and an increased burden of ARI in children (33).

Wood, charcoal and liquid petroleum gas (LPG)

Consumption

As one of the most widely used fuels in Nouna, wood is an important solid biomass fuel. In a recent study, over 98% of households used wood for cooking (Table 1). Daily and yearly wood consumption in Burkina Faso was slightly lower than the daily (1.79–4.46 kg/capita/day) and yearly (9.3 Tg) wood consumption measured in a study in Zimbabwe (40). However, it must be noted that more recent consumption rates for Burkina Faso could be higher. Differences were also found between rural and urban areas. In rural environments, wood consumption was higher at 1.0 kg/capita/day compared to urban areas (0.6 kg/capita/day) (41). Kituyi et al. (42) also reported differences in firewood consumption rates between rural (2.14 kg/capita/day, weighted average) and urban (0.14 kg/capita/day, weighted average) areas in Kenya.

Charcoal was also reported as being used extensively in Nouna. Not surprisingly, urban areas in Burkina Faso were found to use more charcoal than rural areas (0.030 kg/capita/day and 0.013 kg/capita/day, respectively) (41). In Kenya, the mean weighted average consumption of charcoal was 0.37 kg/capita/day in urban areas and 0.26 kg/capita/day (42).

As far as we are aware, no information exists as to the consumption rates of LPG in Burkina Faso. In the Nouna study, less than 3% of households reported using LPG for cooking. A similar finding was also echoed in Kenya where LPG rates were 0.007 kg/capita/day (42).

Table 1. Type of fuel reported among respondents in Nouna^a

Fuel type ^b	Respondents reporting use (%) ^b	Daily consumption rate (kg/capita/day)	Yearly national consumption
Waste (e.g. crops and dung)	0.9	N/A	N/A
Wood	98.2	1.48 (41)	5.64 Tg (41)
Charcoal	72.9	0.03 (41)	0.13 Tg (41)
Liquid petroleum gas	2.3	N/A	N/A
Electricity	0.0	N/A	500,000 MWh (44)

^aRespondents from the Demographic and Surveillance System (DSS) survey on demographics and health from a catchment area of approximately 1,775 km² covering 74,000 households (43).

^bOut of a sample of 221 respondents. Many households reported using multiple types of fuel. (Yamamoto unpublished, 2009).

Table 2. Energy and efficiency of different fuel types typically used in India (46)

Fuel type	Stove types	Energy (MJ/kg)	Thermal efficiency ^b
Wood	Three stone	15.1–15.5 ^a	0.18–0.29
	Traditional mud		
	Improved vented mud		
	Improved vented ceramic		
	Improved metal		
Charcoal	Angethi ^c	25.7	0.18
LPG	LPG stove	45.8	0.54

^aDepending on wood type (e.g. acacia, eucalyptus and roots).

^bThermal efficiency is a combination of combustion and heat transfer efficiency.

^cGalvanised iron bucket combined with mud/concrete and a grate.

Energy

Biomass contributes between 10 and 12% of the overall energy in the world, although this varies with very low percentages in industrialised countries and values of 50% or more in developing countries (45). As presented in Table 2, a study in India using several stove and fuel combinations (46) found that the energy and efficiency of fuels varies widely. Depending on the type of stove used, conversion efficiencies of biomass fuels typically range between 8 and 18% (45). Energy losses, particularly with traditional stoves like those used in Nouna, are largely in the form of heat and the PICs, including CO, NO and particulates. Additionally, the moisture content of solid biomass fuels can also affect the amount of energy and PICs produced. Air-dried wood has water content between 12 and 20% and a heating value of 13–16 MJ/kg (45). New, freshly harvested wood can have a moisture content of 50% or more, which reduces the heating value to less than 10 MJ/kg (45) and greatly increases the amount of PICs produced. High combustion efficiency, such as found in fuels higher on the energy ladder, may result in lower PICs (15). Taken together, these factors all affect the amount of energy that reaches the cooking pot.

Women in Nouna reported cooking on average 1.6 meals per day during the rainy season. In India, it was estimated that the amount of energy needed to cook one meal was approximately 1 MJ (46). If we use this assumption, as we lack estimates for Burkina Faso, we can estimate that a woman will require 1.6 MJ per day to cook meals for her family. Taking into account the thermal efficiencies listed in Table 2, households would need approximately 0.4–0.6 kg of wood per day just for meals. These crude estimates may even be much higher when other factors such as the size of the family or the type of food cooked are taken into account. Conversely, as the energy and thermal efficiency of LPG is much

greater than that of wood, it is estimated that only 0.06 kg of LPG is required to produce the same amount of energy. Thus, much less fuel is needed to cook the same meals.

Availability

In terms of the exploitable quantity of ligneous fuel, Burkina Faso is estimated to have approximately 4.5 tonnes/capita/year (41). Wood fuel production estimates for Burkina Faso were 9,150,000 m³ in 1994 (47). However, this is probably not evenly distributed, which may lead to shortages and surpluses in different parts of the country. Wood, and particularly charcoal, can be economically transported from rural areas to urban areas (45), which may offset some of these disparities. Nonetheless, biomass fuel is poorly characterised because it is not traded in markets and is generally used or gathered locally (non-commercially) and is therefore not part of national statistics (45).

At the local level, fuel is available and can be purchased from the Nouna market. A ‘charette’ of 500 kg of wood costs approximately 2,500 CFA (5.62 USD) locally. In comparison, a 100 kg bag of charcoal is 1,500 CFA (3.37 USD) and a 6 kg cylinder of LPG approximately 5,000 CFA (11.23 USD) (Ouédraogo, personal communication). Using the above crude estimates, the approximate cost of cooking 1.6 meals per day over the course of a year in Nouna would be between 730 and 1,092 CFA (1.64–2.45 USD) using wood, 1,895 CFA (4.25 USD) using charcoal and 19,732 CFA (44.28 USD) using LPG. Therefore, LPG is out of reach of many households who survive on less than 72,690 CFA (162.43 USD) per year (48).

Wood can also be gathered for free from nearby sources. However, gathering wood is a time-consuming activity. For example, women in a rural area of Sri Lanka were forced to walk an average of 5.8 km to collect firewood when an irrigation project brought about widespread environmental damage and tree destruction (2). The time expended on this chore alone was 4.7 hours per week. Thus, in an effort to reduce the time demands for fuel collection, women began to carry average loads of 24 kg (2). The consequences of increased gathering time for fuels can be the substitution of inferior fuels, reductions in the amount of wood used and the cooking of fewer meals, which in turn can lead to less income, rest, space and water heating as well as hygiene. Fewer special foods may also be prepared for the ill, pregnant or children and the elderly (49). Nonetheless, biomass fuels are usually available and inexpensive, making them attractive alternatives especially for the rural poor (17, 33, 42, 50, 51).

Both wood and LPG can pose additional hazards to health that also need to be considered. For example, gathering wood fuel can be linked to increased risk of assault or natural hazards (49). Similarly, burns were

Table 3. Emission factors associated with different fuel types (15)

Fuel type	Stove efficiency	CO ₂ (g/kg)	CO (g/kg)	CH ₄ (g/kg)	RPM ^a (g/kg)
Wood	0.20	1,620	99	9.00	2.00
Charcoal	0.30	2,570	210	7.80	1.70
LPG	0.70	3,190	25	0.01	0.10

^aRespirable particulate matter.

responsible for several injuries, particularly among children, from cooking appliances, steam or other gases in a study in Iran. Fatalities were also recorded in the study from fires related to the manipulation of gas equipment for cooking or heating (52).

Pollution

The health effects associated with exposure to biomass pollutants are well known (19, 49, 53). These pollutants stem from the incomplete combustion of wood, charcoal and LPG, which releases several by-products in addition to heat. These include CO₂, CO, CH₄, particulates like black carbon and other organic compounds (Table 3, (15)). Extremely high levels of pollutants can occur with the burning of biomass fuels for cooking. The mean concentration of PM₁₀ measured in the kitchens during the day of 148 households in Nouna was 4.06 mg/m³ (0.020–45.94 mg/m³). The overall PM₁₀ concentrations greatly exceeded the maximum 24 hour limit of <50 µg/m³ recommended by WHO (54). Levels recorded in Nouna kitchens also exceeded those reported in studies in rural India and South Africa, Mexico and Mozambique (31, 55–57).

CO levels were also very high in the kitchens and sleeping rooms of households in Nouna. The mean area concentration of CO in 121 kitchens and sleeping areas combined was 17.02 ppm (0.13–90.27 ppm). These CO concentrations were within the ranges reported by others (57–60). A study by Naeher et al. (61) in Guatemala found that the concentration of CO released from gas stoves, improved stoves and open fires was 1.5, 2.4 and 6.7 ppm, respectively. Similarly, Smith et al. (62) also observed decreasing levels of CO emitted with ascension of the energy ladder. Per meal, combusted wood residues typically release 19 g/MJ-d CO per meal as opposed to LPG, which releases only 1.0 g/MJ-d CO.

Climate change

In terms of the global carbon cycle, biomass combustion emits between 1,800 and 4,700 Tg carbon per year, compared to fossil fuels, which emit 5,700 Tg carbon per year. Biomass combustion therefore has an important role in the global carbon cycle (9). The human consumption of biomass fuels are estimated to represent between 20 and 40% of all biomass combustion globally. It is also estimated that 1–3% of all human-generated global

warming is from the household burning of biomass fuels (15). In terms of specific pollutants, this is a global contribution of between 1–5%, 6–14% and 8–24% of all CO, CH₄ and total non-methane organic compounds, respectively (15). Table 4 presents the estimated mean daily global warming commitments weighted by 20-year global warming potentials (GWPs) from three stone (wood) stoves and charcoal stoves from a study in Kenya. The relative contributions of biomass combustion in West Africa to atmospheric emissions are 46% (CH₄), 42% (CO), 44% (NMHC) and 32% (CO₂) (Table 5; (41)).

It is assumed that biomass fuels, if renewably harvested, are GHG neutral. However, biomass is not merely combusted into CO₂, which is subsequently taken up by vegetation (64). As much of the carbon of biomass is produced as PICs, which have higher GWPs than CO₂, low-efficiency stoves can produce warming if even the biomass is renewably harvested (4, 13, 49, 64, 65). PICs from biomass burning were found to have a GWP 110% that of CO₂, over a 20-year period (15).

In particular, black carbon is an important PIC in terms of climate change as its effects are stronger than climate gases, such as CH₄, chlorofluorocarbons, nitrous oxide and even ground-level ozone (10). Moreover, black carbon may also have an effect on precipitation levels and melting when it falls on ice or snow by reducing reflectivity and increasing the absorption of solar radiation (12, 66). Currently, it is estimated that approximately 15% of the excess radiative forcing and 40% of the net warming occurring is from black carbon (12, 67). It has also been suggested (68) that biomass burning is the largest source of black carbon in India. In Africa, over

Table 4. Mean daily global warming commitments (g C in CO₂ equivalent weighted by 20-year GWPs) from household biomass combustion by stove type in Kenya (14)

	CO ₂	CO	CH ₄	NMHC ^a	Total GHG ^b
Three stone fire	5,450	1,920	701	240	8,310
Charcoal	4,300	3,120	2,201	230	9,850

^aNon-methane hydrocarbons.

^bGreenhouse gases.

Table 5. Emission factors for biomass combustion by fuel type and location (g C or N/kg dry wood)

Location	Fuel type	CO ₂	CO	NO	Source
Zimbabwe	Wood	450	43	0.52	(40)
West Africa	Wood	400	30	1.5	(41, 63)
	Charcoal making and burning	290	55	8.5	(41, 63)

80% of black carbon emissions are estimated to stem from domestic biomass burning (12).

There is also concern about the environment in places where biomass use is the primary source of energy in developing countries (69). Unsustainable fuel wood use may lead to deforestation (40) and desertification (49). As a result, biomass, soil, land and water resources degrade (45). Changes in land cover and flora have also been noted over the last 30 years in Burkina Faso; however, this is thought to be due to intensified grazing pressure and increases in livestock density (70) rather than biomass fuel harvesting.

Discussion

In comparing the relative benefits and drawbacks of biomass and fossil fuels such as LPG, the winner may seem obvious. However, it is important to consider several aspects within the context of Burkina Faso since such a large proportion of the population uses biomass fuels. The switch to cleaner burning fuels is not likely to be quick or even feasible in the near future, given the economic constraints and availability of solid biomass fuels.

There are some advantages associated with biomass use. Biomass can be sustainable for the environment and climate if they are combusted at a high level of efficiency and renewably harvested (45). Also, biomass combustion usually results in low sulphur and nitrogen emissions. This, in turn, produces fewer particles and less acid precipitation. Additionally, other toxics such as mercury, lead, arsenic, fluorine are less in biomass fuels, compared to other fuel types. Ash by-products can also be recycled back to the areas in which biomass was harvested (45).

As a form of stored solar energy, biomass is more reliable and readily available than wind or direct solar energy. Additionally, biomass is not usually affected by energy crises as it is produced, harvested and used locally. Biomass is also widely accepted, provides employment, contributes to infrastructure and promotes conservation (45). Moreover, fuel demand is not usually the primary driver behind deforestation in developing countries (45, 51). Instead, the demand for agricultural land, road building or other land-use changes are largely responsible (45, 51). Significant amounts of wood for domestic use are collected from trees around houses, fields and roads.

These sources are not included in national forest statistics and do not show up on remote sensing surveys. Consequently, if fuel demand seems to exceed local forest growth rates, deforestation may not actually be taking place (49).

Other environmental problems in countries like Burkina Faso are also thought to be attributable to changes in land use rather than the non-renewable harvesting of household fuels, although this may also have an impact. Desertification, for example, may be linked to fuel demand. However, desertification could also be due to climate change, grazing intensification, land-use shifts and industrial fuel harvesting (e.g. forest kilns) (49).

Another consideration is that as rises in energy prices make fossil fuels unaffordable for the poorest households (4, 23), biomass is one of the only available options in Burkina Faso. Economic considerations and not health or environmental aspects are likely dictating households' choice of fuels in these regions. Thus, efforts to introduce more expensive fuels into households will likely not be feasible without considerable subsidies.

Nevertheless, there are several drawbacks in using biomass fuels. Air pollution, particularly indoors, is associated with numerous health effects. One of the most obvious ways to address this issue is to encourage the use of cleaner burning fuels. In moving up the energy ladder, the first step is usually from wood to charcoal or kerosene and the second to LPG (15). Even small movements up the energy ladder will bring some improvements in terms of indoor air pollution exposure (23). If all the current users of biomass (two billion) switched to LPG, the net reduction in exposure would be larger than the current exposure to all fossil fuel emissions (18). Moreover, using liquid or gases with premixed air to achieve high combustion efficiencies could further reduce exposures (64). Another way to reduce air pollution exposures is to use more efficient stoves. Thus, even if cleaner burning fuels are out of the reach of many poor households, introducing more efficient stoves can be a worthwhile long term investment (71–74).

Solid biomass fuel harvesting and combustion can also have important impacts on the global carbon cycle and climate change. As PICs such as black carbon and CH₄ have higher GWPs than CO₂, solid biomass fuels burned inefficiently may not be GHG neutral, even if they are harvested renewably (75). Given the short lifetime of black carbon (weeks), reducing emissions could be a way to mitigate global warming in the near future, address regional climate issues and decrease the retreat of sea ice and glaciers (10, 12). Moreover, Baron et al. (12) argue that controlling black carbon emissions can be a cost-effective means of improving health and alleviating some of the effects of global warming, although this suggestion is not without debate (67, 76).

Furthermore, switching to LPG may not result in an exponential rise in emissions of GHGs. It has been estimated that switching everyone from biomass fuel to LPG would only result in a less than 2% increase in global GHG emissions (18). It has also been argued that petroleum resources are sufficient to supply all household energy needs worldwide. Petroleum supply stresses are predicted to stem from other sectors (4, 18).

There are also good arguments for exploring the development of renewable and sustainable energy as they have the potential to take advantage of local conditions (e.g. sun and wind). Biogas may also be a good alternative as it produces significantly lower concentrations of pollutants (50, 64). Biogas is renewable, has high thermal and combustion efficiencies and low global warming commitments (64). However, there are currently few of these technologies available to replace biomass on the scale needed. Thus, the introduction of LPG in these areas may be more feasible (18).

Clean energy also provides access to education, health care and household resources. Children who do not have to collect biofuels can attend school (4, 62). Switching to cleaner fuels could also free up time for women to engage in income-generating pursuits (4).

The introduction and promotion of cleaner energy for cooking, particularly in developing countries like Burkina Faso, can have several benefits. Encouraging households to switch to LPG would result in the consumption of less fuel per meal and less time spent gathering fuel, which could be expended on other activities such as attending school or participating in microprojects. LPG is available in places such as Nouna, although it is still not within the affordable range of many households, which is an important drawback. Another significant consideration is the availability and affordability of LPG in rural areas. Nonetheless, global supplies of petroleum are estimated to be sufficient to accommodate the needs of domestic users, even if all those currently using biomass switched to LPG (18).

Perhaps one of the strongest arguments for helping households to switch to cleaner fuels is related to pollution and their associated health outcomes. As the burden of disease attributable to biomass use in Burkina Faso is estimated to be 8.5% (34), reductions in exposures are warranted. In particular, at-risk groups such as women and young children who bear the brunt of such exposures could stand to greatly benefit from switches to LPG or other cleaner burning fuels.

Lastly, reductions in biomass solid fuel pollutants have the potential to impact climate change as well. Black carbon and other PICs can have significant impacts in terms of radiative forcing, precipitation and sea ice melting (10, 67). Policies promoting reductions in the levels of PICs may mitigate some of these effects. Thus, the effects of such policies are advantageous for health

and the climate – a little known co-benefit from the perspective of the mitigation debate (77).

Conclusions

Though biomass use has some advantages and is likely to continue in the short to medium-term in Burkina Faso, other energy options should also be explored. From a health, societal and climate change perspective, the burning of biomass solid fuel in inefficient stoves is highly undesirable. Policies encouraging households to move up the energy ladder are warranted and necessary. Also, as limited information specific to Burkina Faso is available, research regarding fuel use and energy consumption patterns and availability is desirable to aid in the development of effective policies.

As clean fuel initiatives will require coordinated efforts, governments and other organisations also need to plan to ensure adequate, reliable provisions and services. Initiatives to encourage households to move up the energy ladder may include stove intervention programmes and subsidised fuel prices. The exploration of sustainable and renewable energies is also a key consideration in clean energy initiatives.

According to Wilkinson et al. (4), the inequity in the access of rich and poor countries to clean fuels is an injustice. As such, providing poor households with affordable access to cleaner alternatives should be paramount. Interventions and policies are key to the successful introduction of cleaner burning fuels among developing countries like Burkina Faso that heavily depend on biomass.

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References

1. World Health Organization. Indoor air pollution from biomass fuel. Report of a WHO Consultation, June 1991. Geneva: World Health Organization; 1992.
2. Awumbila M, Momsen JH. Gender and the environment. Women's time use as a measure of environmental change. *Glob Environ Change* 1995; 5: 337–46.
3. WHO. Health aspects of air pollution with particulate matter, ozone and nitrogen dioxide; 2003. Available from: <http://www.euro.who.int/Document/e79097.pdf> [cited 4 January 2009].
4. Wilkinson P, Smith KR, Joffe M, Haines A. A global perspective on energy: health effects and injustices. *Lancet* 2007; 370: 965–78.

5. Ezzati M, Kammen DM. The health impacts of exposure to indoor air pollution from solid fuels in developing countries: knowledge, gaps, and data needs. *Environ Health Perspect* 2002; 110: 1057–68.
6. Smith KR, Mehta S. The burden of disease from indoor air pollution in developing countries: comparison of estimates. *Int J Hyg Environ Health* 2003; 206: 279–89.
7. Smith KR. Biomass cookstoves in global perspectives: energy, health, and global warming, in indoor air pollution from biomass fuel. Working papers from a WHO consultation. Geneva: World Health Organization; 1991.
8. Smith KR. National burden of disease in India from indoor air pollution. *Proc Natl Acad Sci USA* 2000; 97: 13286–93.
9. Crutzen PJ, Andreae MO. Biomass burning in the tropics: impact on atmospheric chemistry and biogeochemical cycles. *Science* 1990; 250: 1669–78.
10. Ramanathan V, Carmichael G. Global and regional climate changes due to black carbon. *Nat Geosci* 2008; 1: 221–7.
11. Service RF. Study fingers soot as a major player in global warming. *Science* 2008; 319: 1745.
12. Baron RE, Montgomery WD, Tuladhar SD. An analysis of black carbon mitigation as a response to climate change. Copenhagen: Copenhagen Consensus Center; 2009.
13. Andreae MO, editor. Biomass burning: its history, use and distribution and its impact on environmental quality and global climate. Cambridge, MA: MIT Press; 1991.
14. Bailis R, Ezzati M, Kammen DM. Greenhouse gas implications of household energy technology in Kenya. *Environ Sci Technol* 2003; 37: 2051–9.
15. Smith KR. Health, energy, and greenhouse-gas impacts of biomass combustion in household stoves. *Energy Sust Develop* 1994; 1: 23–9.
16. Prüss-Üstün A, Corvalán C. How much disease burden can be prevented by environmental interventions? *Epidemiology* 2007; 18: 167–78.
17. Emmelin A, Wall S. Indoor air pollution: a poverty-related cause of mortality among the children of the world. *Chest* 2007; 132: 1615–23.
18. Smith KR. In praise of petroleum? *Science* 2002; 298: 1847.
19. von Schirnding Y, Bruce N, Smith KR, Ballard-Tremeeer G, Ezzati M, Lvovsky K. Addressing the impact of household energy and indoor air pollution on the health of the poor – implications for policy action and intervention measures, Working Group 5 (Improving the health outcomes of the poor); 2002. Available from: <http://www.who.int/indoorair/publications/impact/en/index.html> [cited 1 April 2009].
20. Pandey MR, Smith KR, Boleij JSM, Wafula EM. Indoor air-pollution in developing-countries and acute respiratory-infection in children. *Lancet* 1989; 1: 427–9.
21. Collings DA, Sithole SD, Martin KS. Indoor woodsmoke pollution causing lower respiratory-disease in children. *Trop Dr* 1990; 20: 151–5.
22. Armstrong JRM, Campbell H. Indoor air-pollution exposure and lower respiratory-infections in young Gambian children. *Int J Epidemiol* 1991; 20: 424–9.
23. WHO. Indoor air pollution from biomass fuel. Report of a WHO Consultation, June 1991. Geneva: WHO; 1992.
24. Pandey MR. Domestic smoke pollution and chronic bronchitis in a rural community of the Hill Region of Nepal. *Thorax* 1984; 39: 337–9.
25. Behera D, Jindal SK. Respiratory symptoms in Indian women using domestic cooking fuels. *Chest* 1991; 100: 385–8.
26. Malik SK. Exposure to domestic cooking fuels and chronic bronchitis. *Indian J Chest Dis Allied Sci* 1985; 27: 171–4.
27. Hong CJ. Health aspects of domestic use of biomass and coal in China. Indoor air pollution from biomass fuel. Geneva: World Health Organization; 1991, pp. 43–77.
28. Smith KR. Fuel combustion, air-pollution exposure, and health – the situation in developing-countries. *Ann Rev Energ Environ* 1993; 18: 529–66.
29. USEPA. Mid-atlantic risk assessment; 2004. Available from: <http://www.epa.gov/reg3hwmd/risk/human/index.htm> [cited 28 August 2009].
30. Reddy AKN, Reddy BS. Substitution of energy carriers for cooking in Bangalore. *Energy* 1994; 19: 561–71.
31. Balakrishnan K, Sambandam S, Ramaswamy P, Mehta S, Smith KR. Exposure assessment for respirable particulates associated with household fuel use in rural districts of Andhra Pradesh, India. *J Exp Anal Environ Epidemiol* 2004; 14: S14–25.
32. WHO. Fuel for life. Household energy and health; 2006. Available from: <http://www.who.int/indoorair/publications/fuel-for-life/en/index.html> [cited 9 March 2009].
33. Akunne AF. Assessing the adverse health effect of biomass using DALYs as outcome measure: a field study from Burkina Faso. Heidelberg, Germany: University of Heidelberg; 2006.
34. WHO. Indoor air pollution: national burden of disease estimates; 2007. Available from: <http://www.who.int/indoorair/publications/nationalburden/en/index.html> [cited 9 March 2009].
35. Central Intelligence A. Burkina Faso; 2005. Available from: <https://www.cia.gov/library/publications/the-world-factbook/geos/uv.html> [cited 6 December 2005].
36. UNPF. Population, health and socio-economic indicators/policy development; 2006. Available from: <http://www.unfpa.org/worldwide/countryprofiles/burkinafaso.html> [cited 12 June 2005].
37. WHO. World malaria report 2008; 2008. Available from: <http://apps.who.int/malaria/wmr2008/malaria2008.pdf> [cited 4 January 2009].
38. Sachs JD, Ahluwalia IJ, Amoako KY, Aninat E, Cohen D, Diabre Z, et al. Macroeconomics and health: investing in health for economic development; 2001. Available from: <http://whqlibdoc.who.int/publications/2001/924154550x.pdf> [cited 9 March 2009].
39. WHO. Estimated deaths and DALYs attributable to selected environmental risk factors, by WHO member state 2002. Geneva: World Health Organization; 2007.
40. Ludwig J, Marufu LT, Huber B, Andreae MO, Helas G. Domestic combustion of biomass fuels in developing countries: a major source of atmospheric pollutants. *J Atmos Chem* 2003; 44: 23–37.
41. Brocard D, Lacaux JP, Eva H. Domestic biomass combustion and associated atmospheric emissions in West Africa. *Glob Biogeochem Cyc* 1998; 12: 127–39.
42. Kituyi E, Marufu L, Huber B, Wandiga SO, Jumba IO, Andreae MO, et al. Biofuel consumption rates and patterns in Kenya. *Biomass Bioenergy* 2001; 20: 83–99.
43. Yé Y, Sanou A, Gbangou A, Kouyaté B. Nouna demographic surveillance system Burkina Faso; 1999. Available from: http://www.indepth-network.org/dss_site_profiles/nounadss.pdf [cited 3 October 2006].
44. Energy Information Administration. 2009. Available from: http://tonto.eia.doe.gov/country/country_energy_data.cfm?fi ps=UV [cited 3 September 2009].
45. Kaltschmitt M, Thrän D, Smith KR. Renewable energy from biomass. Burlington, MA: Academic Press/Elsevier; 2002.
46. Smith KR, Kishore VVN, Lata K, Joshi V, Zhang J, et al. Greenhouse gases from small-scale combustion devices in developing countries: phase IIa; 2000. Available from: <http://www.epa.gov/nrmrl/pubs/600r00052/600R00052.pdf> [cited 9 March 2009].

47. FAO. Forest products 1983–1994. Rome: Food and Agriculture Organization; 1996.
48. IMF. Burkina Faso. Poverty reduction strategy paper; 2000. Available from: <http://www.imf.org/external/np/prsp/2000/bfa/01/> [cited 9 March 2009].
49. Holdren JP, Smith KR, Kjellstrom T, Streets D, Wang X, Fischer S, editors. Energy, the environment and health. New York: United Nations Development Programme; 2000.
50. Haines A, Smith KR, Anderson D, Epstein PR, McMichael AJ, Roberts I, et al. Policies for accelerating access to clean energy, improving health, advancing development, and mitigating climate change. *Lancet* 2007; 370: 1264–81.
51. Kituyi E, Marufu L, Huber B, Wandiga SO, Jumba IO, Andreae MO, et al. Biofuel availability and domestic use patterns in Kenya. *Biomass Bioenergy* 2001; 20: 71–82.
52. Rezapur-Shahkolai F, Naghavi M, Shokouhi M, Laflamme L. Unintentional injuries in the rural population of Twiserkan, Iran: a cross-sectional study on their incidence, characteristics and preventability. *BMC Public Health* 2008; 8: 269.
53. Smith KR, Mehta S, Feuz M. The global burden of disease from indoor air pollution: results from comparative risk assessment. Proceedings of the 9th international conference on indoor air quality and climate. *Indoor Air* 2002. June 30–July 5 2002. Monterey, CA: Vol. 4: 10–19.
54. WHO. Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide; 2006. Available from: http://whqlibdoc.who.int/hq/2006/WHO_SDE_PHE_OEH_06.02_eng.pdf [cited 4 January 2009].
55. Brauer M, Bartlett K, RegaladoPineda J, PerezPadilla R. Assessment of particulate concentrations from domestic biomass combustion in rural Mexico. *Environ Sci Technol* 1996; 30: 104–9.
56. Ellegård A. Cooking fuel smoke and respiratory symptoms among women in low-income areas in Maputo. *Environ Health Perspec* 1996; 104: 980–5.
57. Röllin HB, Mathee A, Bruce N, Levin J, von Schirnding YER. Comparison of indoor air quality in electrified and un-electrified dwellings in rural South Africa. *Indoor Air* 2004; 14: 208–16.
58. Boleij JSM, Ruigewaard P, Hoek F, Thairu H, Wafula E, Onyango F, et al. Domestic air-pollution from biomass burning in Kenya. *Atmos Environ* 1989; 23: 1677–81.
59. Bruce N, McCracken J, Albalak R, Schei M, Smith KR, Lopez V, et al. Impact of improved stoves, house construction and child location on levels of indoor air pollution exposure in young Guatemalan children. *J Expo Anal Environ Epidemiol* 2004; 14: S26–33.
60. Saksena S, Singh PB, Prasad RK, Prasad R, Malhotra P, Joshi V, et al. Exposure of infants to outdoor and indoor air pollution in low-income urban areas – a case study of Delhi. *J Expo Anal Environ Epidemiol* 2003; 13: 219–30.
61. Naeher LP, Leaderer BP, Smith KR. Particulate matter and carbon monoxide in highland Guatemala: indoor and outdoor levels from traditional and improved wood stoves and gas stoves. *Ind Air – Int J Ind Air Qual Clim* 2000; 10: 200–5.
62. Smith KR, Rogers J, Cowlin SC. Household fuels and ill health in developing countries: what improvements can be brought by LP gas? Paris: World LP Gas Association and Intermediate Technology Development Group; 2005.
63. Brocard D, Lacaux C, Lacaux JP, Kouadio G, Youboué V. Emissions from the combustion of biofuels in Western Africa. In: Levine JS, ed. Biomass burning and global change. Vol. 1. Cambridge, MA: MIT Press; 1996, pp. 350–60.
64. Smith KR, Uma R, Kishore VVN, Zhang J, Joshi V, Khalil MAK. Greenhouse implications of household stoves: an analysis for India. *Ann Rev Energ Environ* 2000; 25: 741–63.
65. Kambis AD, Levine JS. Biomass burning and the production of carbon dioxide: a numerical study. In: Levine JS, ed. Biomass burning and global change. Vol. 1. Cambridge, MA: MIT Press; 1996, pp. 170–7.
66. Menon S, Hansen J, Nazarenko L, Luo Y. Climate effects of black carbon aerosols in China and India. *Science* 2002; 297: 2250–3.
67. Kandlikar M, Reynolds COC, Grieshop AP. A perspective paper on black carbon mitigation as a response to climate change. Copenhagen: Copenhagen Consensus Center; 2009.
68. Venkataraman C, Habib G, Eiguren-Fernandez A, Miguel AH, Friedlander SK. Residential biofuels in South Asia: carbonaceous aerosol emissions and climate impacts. *Science* 2005; 307: 1454–6.
69. Marufu L, Ludwig J, Andreae MO, Meixner FX, Helas G. Domestic biomass burning in rural and urban Zimbabwe – part A. *Biomass Bioenergy* 1997; 12: 53–68.
70. Wittig R, König K, Schmidt M, Szarynski J. A study of climate change and anthropogenic impacts in West Africa. *Environ Sci Pollut Res Int* 2007; 14: 182–9.
71. Smith KR, Dutta K, Chengappa C, Gusain PPS, Masera O, Berrueta V, et al. Monitoring and evaluation of improved biomass cookstove programs for indoor air quality and stove performance: conclusions from the household energy and health project. *Energy Sust Develop* 2007; 11: 5–18.
72. Dutta K, Naumoff Shields K, Edwards R, Smith KR. Impact of improved biomass cookstoves on indoor air quality near Pune, India. *Energy Sust Develop* 2007; 11: 19–32.
73. Chengappa C, Edwards R, Bajpai R, Naumoff Shields K, Smith KR. Impact of improved cookstoves on indoor air quality in the Bundelkhand region in India. *Energy Sust Develop* 2007; 11: 33–44.
74. Boy E, Bruce N, Smith KR, Hernandez R. Fuel efficiency of an improved wood-burning stove in rural Guatemala: implications for health, environment, and development. *Energy Sust Develop* 2000; 4: 23–31.
75. UNEP. Energy and air pollution; 2006. Available from: <http://www.unep.org/geo/yearbook/yb2006/> [cited 9 March 2009].
76. Bond TC, Sun H. Can reducing black carbon emissions counteract global warming? *Environ Sci Technol* 2005; 39: 5921–6.
77. Smith KR, Haigler E. Co-benefits of climate mitigation and health protection in energy systems: scoping methods. *Annu Rev Public Health* 2008; 29: 11–25.

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Climate change influences infectious diseases both in the Arctic and the tropics: joining the dots

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Climate change is incontestably a phenomenon of global causes and impacts. However, as much as the contribution of different regions and countries to climate change differs, as much differ the impacts. This paper examines the current and potential impact of climate change on infectious diseases in regions that could not be more different: the Arctic and the tropics (The Arctic is the area north of the Arctic Circle (66.6°N), while the tropics lie between the Tropic of Cancer (23.4°N) and the Tropic of Capricorn (23.4°S)) (Fig. 1). Despite obvious differences in environmental and socio-economic contexts, there are commonalities between these areas, both in the mechanisms through which climate change influences disease transmission and in the adaptation responses health systems can and

should mount. We hope that the lessons in this comparison can be distilled both by policy makers and researchers in both regions.

The purpose of this article is ‘to join the dots’ and thus stimulate discussion. Inevitably, the different dots (issues) themselves cannot be elaborated on in detail here. For this, we refer the interested reader to a wide-ranging list of references.

Climate-sensitive infectious diseases in the Arctic

Of all regions in the world, the Arctic is particularly affected by global warming. The proportional increase in surface air temperature has been twice the average global increase. Importantly, warming since 1980 has been strongest in the wintertime at 1°C per decade. It comes as no surprise that the sea-ice cover has decreased by 10% during this period (1). Warming is projected to an increase of 2–9°C by the end of the century, a much higher increase than the projected global mean of 1.1–6.4°C (1). Thus, models project a substantial decrease in snow and sea-ice cover over most of the Arctic by the end of the 21st century (2). An already noticeable change was the opening of both the Northwest and the Northeast passages for traffic in 2008. The Barent Sea has been ice-free and open during summer for the past four years.

The health effects of these changes in the far North, particularly in the distribution and incidence of infectious diseases, have received little attention so far both in the published literature (3, 4) and in the media. Climate change will affect the distribution of infectious diseases directly as well as indirectly.

Disruptions in the operation of water supply and sewage facilities increase the risks for intestinal infections through the spread of water and food-borne infections.

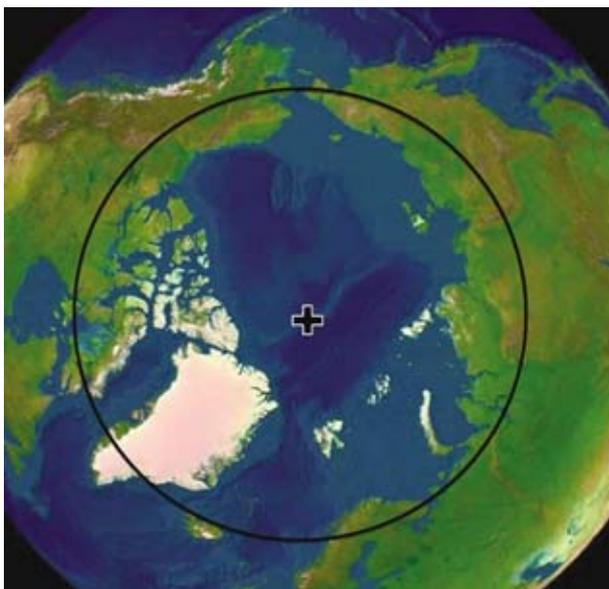


Fig. 1. The Arctic, looking down to the North Pole.

Some recent examples include a 30-fold increase in hepatitis A in one Russian region after flooding. Also, reduced access to clean water may increase skin infections, many of which are caused by multi-resistant bacteria.

As the boreal forest extends north, areas for certain animals such as foxes carrying rabies or echinococcosis and beavers carrying *Giardia intestinalis* will expand.

Another effect of warmer average temperatures and changes in the relative length of seasons is the changing pattern in vector-borne diseases. Insects that exist today in the south of the region transmit different *rickettsia* species such as Mediterranean spotted fever and Q-fever, and viruses such as West Nile virus. These and the Chikungunya virus, recently established in Europe, could potentially move northwards, although this is less likely, as temperatures needed are higher than most models predict. However, the transmitting vector can change, as has occurred with the transmission of Dengue virus and Chikungunya virus from *Aedes aegyptii* to *Aedes albopictus*, an insect that thrives in colder temperatures.

Ticks transmit both Lyme disease (5) and tick-borne encephalitis (TBE) (6), see Box 1. While the incidence of TBE has halved since the late 1990s in Russia, the incidence rate tripled in Arkhangelsk Oblast, in the North, during the same period. In the 1980s, there were relatively few cases of TBE in the Russian Federation, including 300

cases per year in Arkhangelsk Oblast, but rising temperatures from 2001 increased incidence to 2,300 cases per year in 2007, a 10-fold increase within a decade.

Climate-sensitive infectious diseases in the tropics

There have been several excellent recent reviews on this topic, to which we refer the reader (13–18). Table 1 compares the major climate-sensitive infectious diseases in the tropics with those in the Arctic. The list is neither exhaustive nor exclusive. Furthermore, the listed diseases are not necessarily *limited* in their prevalence to the tropics or the Arctic.

Commonalities between climate-sensitive infectious diseases in the Arctic and the tropics

A large proportion of the populations in both these extreme climate zones share the characteristic of living in close proximity to their ecosystems, in fact living on them (3, 19). This makes these populations highly vulnerable to the health effects of climate change.

We consider here five main common characteristics and requirements, respectively, regarding climate-sensitive infectious diseases:

- 1) Exposure to new patterns of climate-sensitive infectious diseases.
- 2) Disease surveillance and early warning systems.

Box 1. Tick-borne encephalitis (TBE) in Sweden.

TBE was first discovered in Sweden in the 1950s and the reported annual numbers of cases have increased ever since. Up to 1979 approximately 25 cases were reported each year. During the 1980s around 40 cases were reported annually and during the 1990s the number rose to 60–80 (7). In the last 10 years the incidence increased further to about 140 cases annually. About 224 cases were reported in 2008 (8). All cases of encephalitis admitted in Stockholm County have been serologically tested for TBE since the late 1950s (9). Viral meningo-encephalitis has been a notifiable disease in Sweden since 2004. TBE causes more than half of the cases of viral meningo-encephalitis reported to the Swedish Institute for Infectious Disease Control (SMI), with most of the patients contracting TBE in Sweden. Only a few patients were infected in other European countries (8).

Most cases occur in the central-eastern parts of the country, particularly in the archipelagos surrounding Stockholm (10). In the eastern parts of the country the risk areas have remained unchanged (7). In the 1990s new TBE-endemic areas became established around lake Vänern and lake Vättern, mainly close to the water where the climate is milder (7). Other areas where the disease seems to be emerging is in the south (Skåne and Blekinge) and in the west, the Bohuslän and Dalsland areas (10). During the last few years, additional foci have appeared. The virus has spread north along the east coast and cases have been reported from Dalarna in central Sweden, and Bohuslän on the west coast (10).

Climatic factors such as mild winters and early arrival of spring are thought to have contributed to the spread of the tick *Ixodes ricinus* further north and to an increase in tick density (10, 11). Other factors contributing to the increase in TBE may include increasing host animal populations such as roe deer, and more people spending time in endemic areas due to an increase in summer cottages (12). In addition, an increased awareness of the disease amongst health care workers and the general population leads to a higher number of diagnosed cases (7, 9). The cause of the increase in TBE in Sweden is most likely multi-factorial.

During the last ten years SMI has diagnosed more than 20 cases with serologically verified TBE despite complete active vaccination against the disease (8). The majority of these cases were diagnosed in 2007. The diagnosis of acute TBE is hard to establish using only a single serum among patients with prior vaccination. For these patients complementary laboratory tests are needed.

Table 1. Climate-sensitive infectious diseases in Arctic's and tropics by type of pathogen

Infective agents	Climate-sensitive diseases	
	Tropics	Arctic
Parasite	Malaria Leishmaniasis Schistosomiasis Trypanosomiasis	Giardiasis Cryptosporidiosis Echinococcosis multilocularis Toxoplasmosis
Bacteria	Meningococcal meningitis	Lyme borreliosis Relapsing fever Tularaemia <i>Vibrio parahemolyticus</i> infections Brucellosis Multi-resistant <i>Staphylococcus aureus</i> <i>Haemophilus influenzae</i> , <i>Streptococcus pneumoniae</i> and <i>Mycobacterium tuberculosis</i>
Rickettsia	Rocky mountain spotted fever, South African tick typhus, Queensland tick typhus and more	Mediterranean spotted fever
Virus	Dengue fever Yellow fever Rift Valley fever West Nile fever Hantavirus cardio-pulmonary syndrome Ross river virus fever Chikungunya Crimean-Congo haemorrhagic fever	Nephropathia epidemica (Puumala virus) Tick-borne encephalitis Russian summer and spring encephalitis West Nile fever Chikungunya Dengue fever Rabies Hepatitis A

- 3) Health system preparedness.
- 4) Enhanced global efforts towards developing drugs and vaccines.
- 5) Common challenges for research.

Exposure to new patterns of climate-sensitive infectious diseases

The pathways leading from the presence of pathogens, vectors and host animals to manifestations of infectious disease in humans are non-linear and complex. Apart from climate change, they are influenced by human immune response, human behaviour (particularly regarding land use (20, 21)), the quality of social and health systems, the development of drug resistance and many more. They all have a strong influence on whether or not diseases will manifest in populations and how they will spread.

With some caveats, it is generally true that some pathogens such as salmonella respond *directly* to higher temperatures in terms of their proliferation. Vector-borne diseases react *indirectly* to changes in temperature and humidity, as insect vectors and their associated pathogens undergo possibly accelerated life

cycles as ambient temperature increases (up to a certain point). They, in turn, frequently depend on host vertebrates, which have their own climate-dependent migration patterns. We would therefore expect some of these diseases to expand pole-wards and upwards in terms of altitude.

Certain vectors have a particular potential to act in joining the dots between the south and the north. The species mentioned below exist today in southern Europe:

- Mosquitoes (*Anopheles*, *Culex* and *Aedes*) can give rise to malaria (different species), West Nile virus, Dengue virus and Chikungunya virus, respectively.
- Ticks (*Ixodes ricinus*, *Dermacentor reticulatus*, *Hyalomma marginatum* and *Rhipicephalus sanguineus*) transmit *Borrelia burgdorferi* (leading to Lyme borreliosis), TBE virus (TBE-encephalitis), *Tularaemia franciscella* (tularaemia), *Coxiella burnetii* (Q-fever), Bunyaviridae viruses (Crimean-Congo haemorrhagic fever), *Rickettsia conorii* (Mediterranean-spotted fever).

- Sandflies (Phlebotominae) transmit *Leishmania donovani* and *Leishmania infantum*.

Autochthonous (locally transmitted) cases of most of these infectious diseases have been reported in Europe.

Changing spatial range of diseases

Higher latitudes. The last case of indigenous malaria in Sweden was reported during the 1930s. Even if other parameters, such as socio-economic situations and well-performing health systems, influence the epidemiology of these infections, higher temperatures could - in a longer-term perspective - move the northern limits of these infections as locally transmitted malaria cases occurring in Italy have shown. West Nile virus has expanded to Canada, and the northward movement of TBE is described in Box 1.

A particularly instructive case, albeit from a veterinary infectious disease, is Blue Tongue, a frequently fatal disease of cattle, goats, sheep and deer. Between 1998 and 2005, this insect-borne disease (spread by the biting midge *Culicoides imicola*) spread northwards in Europe. In 2006, it reached central and northern Europe including Switzerland, the Netherlands, Belgium and Germany. The spread of the disease could be explained by the basic reproductive number R_0 , which is temperature-dependent (22, 23). In 2007, Blue Tongue disease

appeared for the first time in Britain. Gubbins et al. (23) reported the greatest risk of spread in Britain (R_0 between 2 and 4) in a temperature range from 15 to 25°C.

Higher altitudes. The case of highland malaria in Africa has been hotly debated in the literature (24–27). The emerging consensus (28) seems to reflect many factors influencing the upward move of malaria transmission in the Kenyan highlands (Fig. 2), but shows that climate change is among them (29).

Changing temporal pattern of diseases

Changing *seasonal* patterns of infectious diseases have been reported for many climate-sensitive infectious diseases including malaria (30, 31), tularaemia (32) and TBE (33).

Not surprisingly, *inter-annual variability of climate*, too, has been shown to have an influence on infectious diseases: increases in the incidence of malaria, dengue fever, Rift Valley fever, hantavirus infections, cholera and other diseases have been associated with the *El-Nino-Southern Oscillation* (ENSO). For an overview, see Kovats et al. (34) and Anyamba et al. (35).

The few studies on possible relationships between infectious diseases and the *North Atlantic Oscillation* (NAO) have been inconclusive so far. Palo (36) performed

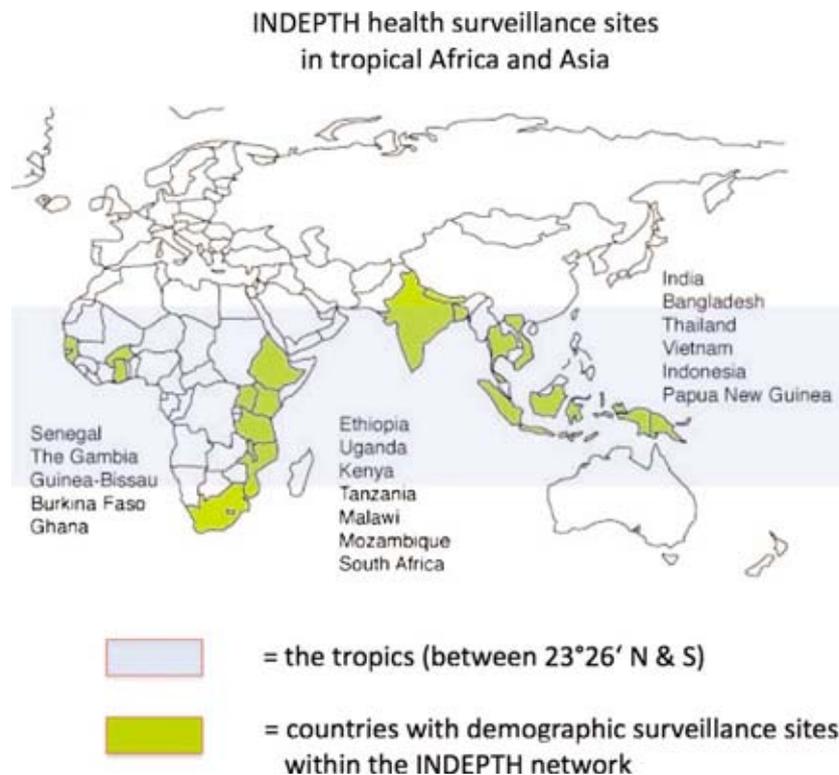


Fig. 2. The world, showing the tropics (blue zone) and countries containing INDEPTH health and demographic surveillance sites (yellow). Source: www.indepth-network.org

Note: There is one INDEPTH site in Latin America, in Nicaragua (not shown on the map).

time-series analyses between the frequency of cases of nephropathia epidemica and the NAO index and did not find a significant association. Hubalek (37) found an association between the NAO index and some infectious diseases such as toxoplasmosis. Intriguingly, Lyme Borreliosis and TBE showed no relationship at all. More research is certainly needed in this arena.

In conclusion, in spite of the extremely different ecosystems in the Arctic and the tropics, the nature of transmission frequently via vectors, generates similarities between climate-sensitive diseases in both areas. This is illustrated in Table 2 using the example of tularaemia and malaria.

Disease surveillance and early warning systems

Given the dynamics and the complexity of climate-sensitive infectious diseases, particularly those transmitted by mosquitoes or rodents, both regions need to develop and sustain surveillance and early warning systems. While a global early warning system, such as the Global Alert and Response Network (GOARN), which was developed by the World Health Organization (38), is impressive, it cannot achieve the task of monitoring the imperceptible extension of infectious diseases into new areas. We discuss below some specific issues pertaining to the Arctic and the tropics.

The Arctic

As populations are relatively small and scattered over a large area, it makes sense to develop a region-specific surveillance system to detect significant trends in infectious diseases. Linking existing national monitoring systems is the basis for appropriate and coordinated actions. Standardising laboratory methods and clinical surveillance definitions across borders facilitates comparing and analysing regional epidemiological data.

An example of such a network is the 'International Circumpolar Surveillance System for Emerging Infectious Diseases' (39). This network links hospital and

public health laboratories for the purposes of monitoring invasive bacterial diseases and tuberculosis in Arctic populations (39). It is planned to extend this surveillance network to include climate-sensitive infections.

The tropics

There are three main weaknesses in most developing countries with regard to disease reporting: Firstly, health services 'see' only a fraction of those suffering from diseases, typically less than one fifth. Those who do visit health services are certainly not representative of the entire population. It is therefore quite possible to miss an increase in cases, if those occur in remote (geographically barred) or in poor (financially barred) populations. Secondly, incomplete reporting may generate fluctuations in case numbers, which are reporting artefacts, rather than actual changes in case frequencies. Thirdly, case verification is difficult and lengthy, given the dearth of reference laboratories.

Population-based health surveillance on a national scale (40) would be ideally implemented using a random sample of sentinel sites. This is however quite expensive and out of reach for many low-income countries.

Another possibility would be to use existing population-based surveillance systems, such as the INDEPTH network (www.indepth-network.org) as a tool for the surveillance of climate-sensitive infectious diseases in the tropics. This network currently covers about two million people in the tropics under demographic and health surveillance in some 30 sites, mainly in Africa and Asia (see Fig. 2). Ascertainment of cause of death is standardised across sites and as valid as possible in the absence of facility-based biomedical exams. Many of these research centres include high quality laboratories, which could be included in systematic surveillance and early warning systems. Although certainly not strictly representative of their countries' *general* population, these sites provide population-based data which is unbiased by the different barriers associated with health care utilisation. In addition, they are certainly faster and more valid in their case ascertainment than the average local health service.

Table 2. Common effects of climate change on disease transmission, taking the examples of tularaemia and malaria

Common effects on diseases transmission	Tularaemia	Malaria
Incidence sensitive to inter-annual climate changes	NAO	ENSO (El Nino)
Length of transmission period	Increased	Increased
Expansion of geographical coverage	Yes	Yes, both latitudinal and altitudinal
Human land-use important	Yes	Yes

Source: Rydén et al. (32).

Health system preparedness

Particularly in low-income countries in the tropics, under-performing health services must be strengthened to meet the additional challenge of network to include climate-sensitive infectious diseases. In the language of climate policy, this would be called a 'no regrets' strategy, something '... whose benefits equal or exceed their costs to society, excluding the benefits of avoided climate change' (41). Hence, strengthening health systems is a sensible policy even in the absence of climate change.

Priority action should be considered in the following areas:

- Protection of the infrastructure, particularly communications networks, against extreme weather events such as floods, storms and heat waves.
- Training health staff about diseases which might be expected to occur or increase and the local measures that should be taken. This applies, for example, as much to Lyme disease in northern Sweden as to malaria in the latitudinal and altitudinal fringes of its current distribution.
- Establishing decentralised stocks of drugs, vaccines and equipment for preventing or treating climate-sensitive diseases.
- Establishing fast routine reporting systems for climate-sensitive infectious diseases.

Part of preparedness pertains to the active cooperation of health sector staff with colleagues from other relevant sectors, such as forestry, agriculture and meteorology. The need for such inter-sectoral cooperation is obvious, as the control of climate-sensitive infectious diseases, particularly those borne by vectors, strongly depends on information and action from these sectors. Table 3 illustrates the common characteristics of adaptation measures, using the example of two otherwise unrelated diseases, tularaemia in the Arctic and malaria in the tropics.

Enhanced global efforts towards developing drugs and vaccines for climate-sensitive infectious diseases

The development of vaccines against dengue fever and malaria is an important global research priority *per se*. The importance is accentuated by the projected increase in the burden of these diseases burden due to climate change. In principle, the need for vaccine development arises also in the case of Lyme disease (42). The debate

Table 3. Common adaptation mechanisms, illustrated by two climate-sensitive diseases: tularaemia and malaria.

Common adaptation	Tularaemia	Malaria
Early warning systems based on meteorology	In place (e.g. in Sweden)	At a research stage
Early warning systems based on case detection	Active and passive	Passive
Surveillance possible	Yes	Yes
Preparedness of health services	Moderate	Low
Involvement of other sectors (forestry, agriculture and infrastructure)	Yes	Beginning

Source: Rydén et al. (32).

should be re-opened in view of its anticipated spread northwards into the Arctic.

Drug treatment of malaria currently hinges primarily on one single type of drug, the artemisinin derivatives. Given that we have evidence of *in-vitro* markers of resistance, particularly in south-east Asia (43), clinical resistance is imminent (44). The need for research and development of anti-malarial drugs is highlighted in view of potential effects of climate change on malaria transmission.

The same holds for drugs against other tropical climate-sensitive diseases, such as leishmaniasis and trypanosomiasis against which we urgently need new, less toxic and more effective drugs.

Common challenges for research

Surprisingly, research output on the links between climate and infectious diseases is limited for both regions, particularly as far as intervention-oriented research is concerned.

In both regions, researchers essentially face a similar agenda:

- 1) The attribution of changes in infectious diseases to climate change.

This almost invariably involves modelling techniques, linking regional climate models or at least time-series of meteorological data to models of infectious diseases.

For example, the Arctic is a region characterised by complex and incompletely understood climate processes, making climate modelling for this region challenging. Local climate data need to be coupled to large-scale data flows. There is a rapid development in the field, but, so far, regional models have not been used for the Arctic Climate Impact Assessment (ACIA).

- 2) The validation of early warning and surveillance systems for climate-sensitive infectious diseases.
- 3) The development and evaluation of evidence-based adaptation strategies, their protective effectiveness and costs.
- 4) The identification of most vulnerable population sub-groups and subsequent targeting of adaptation interventions to them.

For example, there are 40 different ethnic groups in northern Russia. These minorities speak different languages as well as the language of the nation they live in and they have their own customs and cultures. Adapting the research methodology used according to these differences is important and needs special consideration.

- 5) Research training.

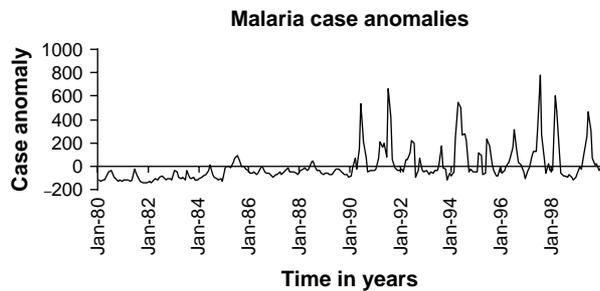


Fig. 3. Time trend for malaria in the Kenyan highlands. Source: Ref. (29).

Few health researchers are comfortable with modelling infectious diseases, let alone with using climate models and linking them to disease data (45). Many of the required techniques, such as time series analysis and analysis of remote sensing data are new, particularly to public health researchers and epidemiologists. Techniques for working with multi-sectoral data sets, e.g. from meteorology and agriculture, need to be learnt. While we need to raise interest for this issue within the global research community, a special effort in training and capacity building is needed in low income countries, where climate effects will be first and most strongly felt (46).

Conclusions

The task of understanding the nexus between climate change and infectious diseases is not made easier by the complex simultaneous ecological, social and health service influences, as the debate on highland malaria illustrates (28) (Fig. 3).

The transition from knowledge to knowledge-based actions is another challenge. The key issue is: What degree of confidence or certainty do we need in order to act? The United Nations Framework Convention on Climate Change has clearly stated that action is needed in the presence of uncertainty if the potential adverse effects are severe and irreversible ('Precautionary Principle'¹ (47)).

In spite of obvious contrasts in geography and the respective biospheres, these challenges are common to researchers and policy makers both in the Arctic and in the tropics. Many lessons can be shared. Joining the dots essentially means connecting the people who work around these dots across different parts of the globe.

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References

1. IPCC. Fourth assessment report. Climate change: I. The scientific basis. Cambridge: Cambridge University Press; 2007.
2. Katsov VM, Källén E. Future climate change: modeling and scenarios for the Arctic. In: Arctic climate impact assessment. Cambridge: Cambridge University Press; 2005, pp. 99–150.
3. Parkinson A, Evengård B. Climate change, its impact on human health in the Arctic and the public health response to threats of emerging infectious diseases. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2075
4. Parkinson AJ, Butler JC. Potential impact on climate change on infectious disease emergence in the Arctic. *Int J Circumpolar Health* 2005; 64: 478–86.
5. Jaenson TG, Eisen L, Comstedt P, Mejlon HA, Lindgren E, Bergström S, et al. Risk indicators for the tick *Ixodes ricinus* and *Borrelia burgdorferi* Ticks *eri sensu lato* in Sweden. *Med Vet Entomol* 2009; 23: 226–37.
6. Randolph SE. Evidence that climate change has caused 'emergence' of tick-borne diseases in Europe? *Int J Med Microbiol* 2004; 293: 5–15.
7. Haglund M. Occurrence of TBE in areas previously considered being non-endemic: Scandinavian data generate an international study by the International Scientific Working Group for TBE (ISW-TBE). *Int J Med Microbiol* 2002; 33: 50–4.
8. Smittskyddsinsitute. Sjukdomsinformation om Tick Borne Encephalitis (TBE) (Disease information on TBE); 2009. Available from: <http://www.smittskyddsinstitutet.se/sjukdomar/tbe/> [cited 21 September 2009].
9. Lindgren E, Gustafson R. Tick-borne encephalitis in Sweden and climate change. *Lancet* 2001; 358: 16–8.
10. Fält J, Lundgren Å, Alsterlund R, Carlsson B, Eliasson I, Haglund M, et al. Tick-borne encephalitis (TBE) in Skåne, southern Sweden: a new TBE endemic region? *Scand J Infect Dis* 2006; 38: 800–4.
11. Lindgren E, Tällekliint L, Polfeldt T. Impact of climatic change on the northern latitude limit and population density of the disease-transmitting European tick *Ixodes ricinus*. *Environ Health Perspect* 2000; 108: 119–23.
12. Randolph SE. Tick-borne encephalitis virus, ticks and humans: short-term and long-term dynamics. *Curr Opin Infect Dis* 2008; 462, 467.
13. Jaenisch T, Patz J. Assessment of associations between climate and infectious diseases. *Global Change Human Health* 2002; 3: 67–72.
14. Epstein P. Detecting the infectious diseases consequences of climate change and extreme weather events. In: Martens P, McMichael AJ, eds. *Environmental change, climate and health – issues and research methods*. Cambridge: Cambridge University Press; 2002, pp. 172–96.
15. Sutherst RW. Global change and human vulnerability to vector-borne diseases. *Clin Microbiol Rev* 2004; 17: 136–73.
16. Cardenas R, Sandoval CM, Rodriguez-Morales AJ, Vivas P. Zoonoses and climate variability. *Ann NY Acad Sci* 2008; 1149: 326–30.
17. Sauerborn R, Louis V, editors. *Global environmental change and infectious diseases: impacts and adaptation strategies*. Heidelberg: Springer Verlag; 2009 (forthcoming).
18. Zhang Y, Bi P, Hiller JE. Climate change and the transmission of vector-borne diseases: a review. *Asia Pac J Public Health* 2008; 20: 64–76.
19. Grassl H, Schubert R, Epiney A, Kulesa M, Luther J, Nuscheler F, et al. Fighting poverty through environmental

- policy. London and Sterling: German Advisory Council for Global Change (WBGU, Berlin), Earthscan; 2005.
20. Vittor AY, Gilman RH, Tielsch J, Glass G, Shields T, Sánchez-Lozano W, et al. The effect of deforestation on the human-biting rate of *Anopheles darlingi*, the primary vector of *falciparum* malaria in the Peruvian Amazon. *Am J Trop Med Hyg* 2006; 74: 3–11.
 21. Afrane YA, Little TJ, Lawson BW, Githeko AK, Yan G. Deforestation and vectorial capacity of *Anopheles gambiae* Giles mosquitoes in malaria transmission, Kenya. *Emerg Infect Dis* 2008; 14: 1533–8.
 22. Racloz V, Venter G, Griot C, Stärk KDC. Estimating the temporal and spatial risk of bluetongue related to the incursion of infected vectors into Switzerland. *BMC Veter Res* 2008; 4: 60–8.
 23. Gubbins S, Carpenter S, Baylis M, Wood JLN, Mellor PS. Assessing the risk of bluetongue to UK livestock: uncertainty and sensitivity analyses of a temperature-dependent model for the basic reproduction number. *J R Soc Interface* 2008; 5: 363–71.
 24. Hay SI, Rogers DJ, Randolph SE, Stern DI, Cox J, Shanks GD, et al. Hot topic or hot air? Climate change and malaria resurgence in East African highlands. *Trends Parasitol* 2002; 18: 530–4.
 25. Bouma MJ. Methodological problems and amendments to demonstrate effects of temperature on the epidemiology of malaria. A new perspective on the highland epidemics in Madagascar, 1972–89. *Trans R Soc Trop Med Hyg* 2003; 97: 133–9.
 26. Zhou G, Minakawa N, Githeko AK, Yan G. Association between climate variability and malaria epidemics in the East African highlands. *Proc Natl Acad Sci USA* 2004; 101: 2375–80.
 27. Minakawa N, Omukunda E, Zhou G, Githeko A, Yan G. Malaria vector productivity in relation to the highland environment in Kenya. *Am J Trop Med Hyg* 2006; 75: 448–53.
 28. Pascual M, Cazelles B, Bouma MJ, Chaves LF, Koelle K. Shifting patterns: malaria dynamics and rainfall variability in an African highland. *Proc Biol Sci* 2008; 275: 123–32.
 29. Githeko A. African highland malaria. In: Sauerborn R, Louis V, eds. *Global environmental change and infectious diseases: impacts and adaptation strategies*. Heidelberg: Springer Verlag; 2009 (forthcoming).
 30. Yé Y, Sauerborn R, Simboro S, Hoshen M. Using modeling to assess malaria infection risk during the dry season on a local scale in an endemic area of rural Burkina Faso. *Ann Trop Med Parasitol* 2007; 101: 375–89.
 31. Yé Y, Hoshen M, Kyobutungi C, Louis VR, Sauerborn R. Local scale prediction of *Plasmodium falciparum* malaria transmission in an endemic region: a meteorologically based dynamic model. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.1923
 32. Rydén P, Sjöstedt A, Johansson A. Effects of climate change on tularemia activity in Sweden. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2063
 33. Gray JS, Dautel H, Estrada-Peña A, Kahl O, Lindgren E. Effects of climate change on ticks and tick-borne diseases in Europe. *Interdiscip Perspect Infect Dis* 2009; 593232.
 34. Kovats RS, Bouma MJ, Hajat S, Worrall E, Haines A. El Niño and health. *Lancet* 2003; 362: 1481–9.
 35. Anyamba A, Chretien JP, Small J, Tucker CJ, Linthicum KJ. Developing global climate anomalies suggest potential disease risks for 2006–2007. *Int J Health Geogr* 2006; 5: 60–8.
 36. Palo RT. Time series analysis performed on nephropathia epidemica in humans of northern Sweden in relation to bank vole population dynamic and the NAO index. *Zoonoses Public Health* 2009; 56: 150–6.
 37. Hubalek Z. North Atlantic weather oscillation and human infectious diseases in the Czech Republic, 1951–2003. *Eur J Epidemiol* 2005; 20: 263–70.
 38. WHO. Global alert and response network (GOARN); 2009. Available from: <http://www.who.int/csr/outbreaknetwork/en/> [cited 28 September 2009].
 39. Parkinson AJ, Bruce M, Zultz T. International circumpolar surveillance, and Arctic network for surveillance of infectious diseases. *Emerg Infect Dis* 2008; 14: 18–24.
 40. Ling HY, Rocklöv J, Ng N, Sauerborn R, Siang CT, Yin PF. Climate variability and increase in intensity and magnitude of dengue incidence in Singapore. *Global Health Action* 2009, in press.
 41. IPCC. Fourth assessment report. Climate change: III. Mitigation of climate change. Cambridge: Cambridge University Press; 2007.
 42. NIAID. Lyme disease – the facts and the challenge. NIH publication No. 08-7045; 2008. Available from: <http://www3.niaid.nih.gov/topics/lymeDisease/PDF/LymeDisease.pdf> [cited 14 June 2009].
 43. Jambou R, Legrand E, Niang M, Khim N, Lim P, Volney B, et al. Resistance of *Plasmodium falciparum* field isolates to *in-vitro* artemether and point mutations of the SERCA-type PfATPase6. *Lancet* 2005; 366: 1960–3.
 44. Noedl H, Socheat D, Satimai W. Artemisinin-resistant malaria in Asia. *N Engl J Med* 2009; 30: 540–1.
 45. Sauerborn R. Global environmental change-an agenda for research and teaching in public health – invited editorial. *Scand J Public Health* 2007; 35: 561–3.
 46. Sauerborn R, Kjellstrom T, Nilsson M. Health as a crucial driver for climate policy. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.2104
 47. UN Conference on Environment and Development. Rio declaration on environment and development; 1992. Available from: <http://www.unep.org/Documents.Multilingual/Default.asp?documentID=78&articleID=1163> [cited 14 June 2009].

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Climate change and population health in Africa: where are the scientists?

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Despite a growing awareness of Africans' vulnerability to climate change, there is relatively little empirical evidence published about the effects of climate on population health in Africa. This review brings together some of the generalised predictions about the potential continent-wide effects of climate change with examples of the relatively few locally documented population studies in which climate change and health interact. Although ecologically determined diseases such as malaria are obvious candidates for susceptibility to climate change, wider health effects also need to be considered, particularly among populations where adequacy of food and water supplies may already be marginal.

Keywords: *climate change; Africa; population health; vulnerability; research*

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Despite increasing media coverage around the theme of African peoples' potential vulnerability to actual and predicted changes in climate at the global level, detailed scientific evidence remains scanty. Much existing work has looked at the effects of climate on specific diseases, without making clear connections to overall changes in population health. A simple search of the PubMed database, undertaken on 26 June 2009, using the keywords 'health', 'Africa' and 'climate change', revealed the number of hits shown in Fig. 1. Despite almost two million scientific citations matching one or another of these terms, the intersection between all three contained a mere 31 citations (0.002%), some of which were brief comments or related to veterinary health. Even though this was a somewhat simplistic search, deliberately intended not to include disease-specific studies which did not explore more general implications for population health, the paucity of results in the intersection is striking. Nevertheless, the oldest of these 31 citations was published as far back as 1991 (1) and one therefore has to ask questions about what has or has not happened in terms of progressing the scientific understanding of climate change and population health in Africa over the past two decades.

The aim of this review article is to highlight some of the important points that emerge from the relatively scant literature on climate change and health in Africa, with a view to both identifying some of the key issues that have been documented and revealing some of the possible gaps where further scientific research is urgently needed.

The literature on climate change and health in Africa

Ramin and McMichael (2), acknowledging the paucity of evidence-based results, present five hypothetical case studies illustrating what they believe to be the major issues. These are concerned with the interaction between HIV and rain-fed food production capacity; impact of drought on child nutrition; disease impact of floods; changing patterns of malaria transmission; and the impact of natural disasters. However, these but serve as examples of scenarios which are all too rarely documented in detail with a basis in reliable data.

Much more attention has been given to global and regional estimates, using multiple data sources and models, of possible changes in climate and, in some cases, their potential health consequences. The Stern Report (3) paints a gloomy picture for Africa, concluding that 'The poorest will be hit earliest and most severely. In many developing countries, even small amounts of warming will lead to declines in agricultural production because crops are already close to critical temperature thresholds. The human consequences will be most serious and widespread in sub-Saharan Africa, where millions more will die from malnutrition, diarrhoea, malaria and dengue fever, unless effective control measures are in place'. This is also reflected in the recent UCL Lancet Commission report (4), which suggests that the highest regional burden of climate change is likely to be borne by sub-Saharan Africa, with 34% of the global disability adjusted life years (DALYs) attributable to climate

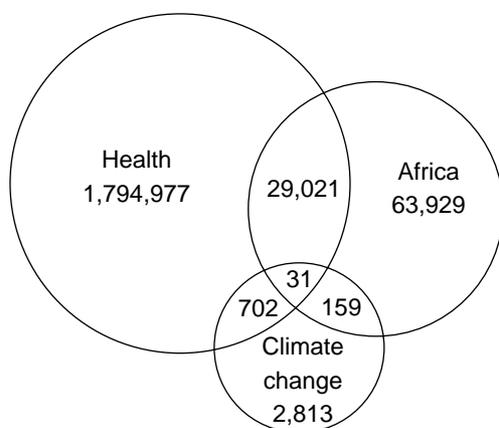


Fig. 1. Results of a PubMed search for 'health', 'Africa' and 'climate change' (26 June 2009) showing the intersections between the three terms (total citations 1,891,632).

change located there. Since sub-Saharan Africa only contains 11% of the world's population (5), this reflects a three-fold population-based risk for adverse effects of climate change among Africans compared with the global population.

A review of likely impacts of climate change on food insecurity (6) concludes that current estimates of climate change will probably lead to greater risk of hunger, particularly in Africa, but without going on to consider the likely population health impacts of such hunger. Changes in land use, either forced by climate change or in response to environmental pressures, may also impact on food security (7). Infectious diseases tend to be considered first when contemplating the effects of climate change on health (1), but these may not be the only or even the major factor; impacts such as the physiological effects of heat may be equally important (8), particularly on the African continent where large proportions of people are regularly exposed to extremes of high temperature.

A review of cholera seasonality globally from 1974 to 2005 (9) suggests that climate change may well lead to less predictable patterns of cholera outbreaks; within Africa this is likely to be seen more in northern and western areas rather than in the equatorial zone. Detailed analyses of diarrhoeal disease patterns in Benin (10) have established expected relationships between risks of diarrhoea and lack of good-quality water supplies. These authors go on to suggest that the consequences of future climate change scenarios, leading to large-scale population displacements, may be predicated on resource considerations, such as water supply, at migratory destinations.

Whilst changes in climate may have impacts on distributions of vector-borne diseases as a direct consequence of ecological pressure on vectors, it is also important to take into account changes in host factors, such as exposure and immunity, in considering health

consequences (11). Furthermore, the disease manifestations of longer-term epidemiological changes driven by climate may be masked by adaptational mechanisms in human populations (12).

Malaria is probably the most documented single disease in relation to climate change. Part of this seems to derive from concerns that the global range of malaria may be extended as territories outside current endemic zones become wetter and warmer, rather than concerns about Africa *per se*, even if these concerns may in some cases have been overstated (13). Detailed climatic analyses suggest that patterns of malaria transmission in Africa may well change as a result of climate changes (14). These changes are modulated by altitude, and the interactions between climate, altitude and vector behaviour (15). The general notion that climate is changing, alongside observations of changes in malaria disease patterns, do not, however, necessarily constitute a cause and effect relationship between climate change and malaria (16). The importance of carrying through climate modelling into vector dynamics modelling has been stressed (17). Some predictions suggest that the main effect of climate change on malaria in Africa will be geographic shifts of transmission within the continent, rather than overall changes (18).

Examples of local impacts on overall population health in Africa as a consequence of climate change are relatively rare, not least because of the relative scarcity of detailed health data from the African continent in the first place (19). After a series of debilitating rainfall failures in northern Ethiopia, a microdam construction programme was initiated in the 1990s to harvest rain water. As well as being a response to climate variability, these new reservoirs generated their own microclimates, often including nearby villages and one consequence was a seven-fold increase in childhood malaria incidence near the dams (20). In one district of Burkina Faso, detailed connections have been made between temperature, rainfall, mosquitoes and malaria, resulting in a local-level model relating climate to malaria transmission (21). In central Ethiopia, links have been made between erratic seasonal patterns of rainfall and disease epidemics which gave rise to a 2.5-fold increase in local overall population mortality over a two-year period (22).

Discussion

Scientific evidence connecting climate change and population health in Africa remains scanty, both because of the lack of clear understanding of the progress and nature of climate change, and the widespread lack of community-based health data. By contrast, the heatwave experienced in western Europe in 2003 was immediately identified as an unusual meteorological event and shortly afterwards analyses of resultant excess mortality were available, using data from routine systems (23).

Although the dominance of malaria in the literature linking climate change and health in Africa might lead one to suppose that it is the major issue, in fact other effects that are relatively under-documented may turn out to have numerically greater impacts as time passes. It may simply be the case that malaria has been the most researched issue in relation to climate thus far. However, if predictions of changing epidemic patterns of malaria within Africa are borne out (18), then the effects of new or increased endemicity among relatively less-immune populations could be severe, affecting all sectors within populations.

Apart from situations where interventions for other purposes facilitate the investigation of climate impacts (such as the Ethiopian microdams (20)), most research into the effects of climate change on population health has to essentially follow a process of making retrospective connections between meteorological data and health outcomes. Population groups cannot be randomised to different kinds of natural climate change, and hence drawing causal inferences between observed climatic variations and disease patterns is difficult to achieve with certainty. Settings where prospective longitudinal population surveillance is in place, such as Indepth Network sites (24), are likely to be particularly valuable for analysing the population effects of climate change in Africa, since it is usually too late to collect health outcome data once a particular climate phenomenon has been observed. Routine collection of meteorological data at such sites should be regarded as normative in the current era of climate change, as well as routinely georeferencing population health data in order to make connections with external meteorological data (25). Even within such surveillance sites, prompt routine review procedures for incoming data are very important if major health-related events at the population level that might be climate-linked are to be spotted promptly (26).

Perhaps the lack of potential for experimental study design in relating climate change to health in Africa, as well as the practical difficulties of obtaining the necessary data, have led to the relative lack of detailed scientific output in the intersection between health and climate change in Africa. The relatively weak research infrastructure in many African settings is a contributory problem, but, more importantly, cross-disciplinary work (between climatology and health in this instance) is not common in Africa and needs to be actively promoted. Expertise in applying global climate models regionally is very scarce in Africa (27), against the inevitable background of the world's most sophisticated climatological research centres being northern-based. Thus stronger links and capacity-building collaborations need to be forged between these global resources in the North and African health and climate research groups, if meaningful

and useful scientific connections are going to be made between climate change and health in Africa.

Despite the dearth of research on the ground in Africa so far, it seems clear from major global-level considerations of climate change and their possible effects on population health (3, 4) that, yet again, Africa and her people stand to come out worst from the likely changes in the world's weather patterns. This has to be seen against a background of food and water security which is already at marginal levels in many places. This therefore constitutes a major challenge to governments in terms of future policy-making, and hence to universities and other research centres for generating the necessary evidence base. Thus there are compelling reasons to promote and facilitate good quality and detailed research on climate change and population health in Africa, in much larger quantities than heretofore.

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References

1. Shope R. Global climate change and infectious diseases. *Environ Health Perspect* 1991; 96: 171-4.
2. Ramin BM, McMichael AJ. Climate change and health in sub-Saharan Africa: a case-based perspective. *EcoHealth* 2009. DOI: 10.1007/s10393-009-0222-4.
3. Stern N. *The economics of climate change*. Cambridge, UK: Cambridge University Press. Available from: http://www.hm-treasury.gov.uk/independent_reviews/stern_review_economics_climate_change/sternreview_index.cfm; 2007 [cited 1 August 2009].
4. Costello A, Abbas M, Allen A, Ball S, Bell S, Bellamy R, et al. Managing the health effects of climate change: Lancet and University College London Institute for Global Health Commission. *Lancet* 2009; 373: 1693-733.
5. Unicef. *The state of the world's children 2007*. New York: Unicef. Available from: http://www.unicef.org/publications/files/The_State_of_the_Worlds_Children_2007_e.pdf; 2006 [cited 1 August 2009].
6. Parry M, Rosenzweig C, Livermore M. Climate change, global food supply and risk of hunger. *Philos Trans R Soc B* 2005; 360: 2125-38.
7. Patz JA, Olson SH, Uejio C, Gibbs HK. Disease emergence from global climate and land use change. *Med Clin North Am* 2008; 92: 1473-91.
8. Kjellstrom T. Climate change, direct heat exposure, health and well-being in low and middle-income countries. *Global Health Action* 2009. DOI: 10.3402/gha.v2i0.1958.
9. Emch M, Feldacker C, Islam MS, Ali M. Seasonality of cholera from 1974 to 2005: a review of global patterns. *Int J Health Geogr* 2009; 7: 31.

10. Pande S, Keyzer M, Arouna A, Sonneveld BJGS. Addressing diarrhoea prevalence in the West African middle belt: social and geographic dimensions in a case study for Benin. *Int J Health Geogr* 2008; 7: 17.
11. Patz JA, Reisen WK. Immunology, climate change and vector-borne diseases. *Trends Immunol* 2001; 22: 171–2.
12. Githeko AK, Lindsay SW, Confalonieri UE, Patz JA. Climate change and vector-borne diseases: a regional analysis. *Bull World Health Organ* 2000; 78: 1136–47.
13. Rogers DJ, Randolph SE. The global spread of malaria in a future, warmer world. *Science* 2000; 289: 1763–6.
14. Tanser FC, Sharp B, le Sueur D. Potential effect of climate change on malaria transmission in Africa. *Lancet* 2003; 362: 1792–8.
15. Lindsay SW, Martens WJM. Malaria in the African highlands: past, present and future. *Bull World Health Organ* 1996; 76: 33–45.
16. Hay SI, Cox J, Rogers DJ, Randolph SE, Stern DI, Shanks GD, et al. Climate change and the resurgence of malaria in the East African highlands. *Nature* 2002; 415: 905–9.
17. Pascual M, Ahumada JA, Chaves LF, Rodo X, Bouma M. Malaria resurgence in the East African highlands: temperature trends revisited. *Proc Natl Acad Sci* 2006; 103: 5829–34.
18. Peterson AT. Shifting suitability for malaria vectors across Africa with warming climates. *BMC Infect Dis* 2009; 9: 59.
19. Byass P. The unequal world of health data. *PLoS Med* 2009, in press.
20. Ghebreyesus TA, Haile M, Witten KH, Getachew A, Yohannes AM, Yohannes M, et al. Incidence of malaria among children living near dams in northern Ethiopia: community-based incidence survey. *BMJ* 1999; 319: 663–6.
21. Yé Y, Sankoh OA, Kouyaté B, Sauerborn R. Environmental factors and malaria transmission risk: modelling the risk in a holoendemic area of Burkina Faso. Surrey, UK: Ashgate; 2008.
22. Emmelin A, Fantahun M, Berhane Y, Wall S, Byass P. Vulnerability to episodes of extreme weather: Butajira, Ethiopia, 1998–1999. *Global Health Action* 2008. DOI: 10.3402/gha.v1i0.1829.
23. Conti S, Meli P, Minelli G, Solimini R, Toccaceli V, Vichi M, et al. Epidemiologic study of mortality during the summer 2003 heat wave in Italy. *Environ Res* 2005; 98: 390–9.
24. Evans T, AbouZahr C. INDEPTH @ 10: celebrate the past and illuminate the future. *Global Health Action* 2008. DOI: 10.3402/gha.v1i0.1899.
25. Aron JL. Barriers to use of geospatial data for adaptation to climate change and variability: case studies in public health. *Geosp Health* 2006; 1: 11–6.
26. Fottrell E, Byass P. Identifying humanitarian crises in population surveillance field sites: simple procedures and ethical imperatives. *Public Health* 2009; 123: 151–5.
27. Pierce DW, Barnett TP, Santer BD, Glecker PJ. Selecting global climate models for regional climate change studies. *Proc Natl Acad Sci* 2009; 106: 8441–6.

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