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ORIGINAL RESEARCH

Temperature and Humidity Effects on Hospital Morbidity in Darwin, Australia

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Abstract

BACKGROUND Many studies have explored the relationship between temperature and health in the context of a changing climate, but few have considered the effects of humidity, particularly in tropical locations, on human health and well-being. To investigate this potential relationship, this study assessed the main and interacting effects of daily temperature and humidity on hospital admission rates for selected heat-relevant diagnoses in Darwin, Australia.

METHODS Univariate and bivariate Poisson generalized linear models were used to find statistically significant predictors and the admission rates within bins of predictors were compared to explore nonlinear effects.

FINDINGS The analysis indicated that nighttime humidity was the most statistically significant predictor ($P < 0.001$), followed by daytime temperature and average daily humidity ($P < 0.05$). There was no evidence of a significant interaction between them or other predictors. The nighttime humidity effect appeared to be strongly nonlinear: Hot days appeared to have higher admission rates when they were preceded by high nighttime humidity.

CONCLUSIONS From this analysis, we suggest that heat-health policies in tropical regions similar to Darwin need to accommodate the effects of temperature and humidity at different times of day.

KEY WORDS admissions, heat stress, hyperthermia, sleep disruption, tropical

INTRODUCTION

Darwin is the capital city and regional center of the Northern Territory (NT). It is situated on the Timor Sea, on Australia's northern coast. The greater city region is home to approximately 120,586 people, and as such is the densest population of the NT in an otherwise sparsely inhabited region of the country.[1]

Darwin has a tropical climate characterized by low annual temperature variability but high annual humidity variability. This variability can be seen for the 1945 to 2015 period with monthly mean morning (9 AM) conditions varying from 23°C to 30°C, and relative humidity varying from 60% to 83%; whereas afternoon (3 PM) conditions varied from 30°C to 32°C and 37% to 72% relative humidity. Significantly, these seasonal trends in temperature and relative humidity are not in phase, resulting in annual temperature maxima that occur 3 months before annual relative humidity maxima.[2]

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Darwin temperatures have increased by 0.5°C to 1°C over the period 1910 to 2012, and this is projected to increase to up to 4°C in the 21st century, given a "business as usual" Representative Concentration Pathway (RCP) 8.5 emissions scenario.[3] However, some observed aspects of temperature change in Australia's northwest are not replicated in global models,[4] and seasonal temperature changes in the region remain poorly understood.[5]

Warmer air enables the uptake of additional water vapor; the exponential relationship between atmospheric temperature and the water vapor pressure at saturation is known as the Clausius-Clapeyron relationship.[6] Specific humidity—a measure of the water vapor mass of the air[7]—has globally increased "mostly at or above the increase expected from the Clausius-Clapeyron relation … with *high confidence*."[8] Because of this, relative humidity—the ratio of specific humidity to the saturation water vapor mass expected from the Clausius-Clapeyron relation at a given temperature—has stayed approximately constant globally and is expected to continue in the future.[9] However, this global trend masks decreasing relative humidity observations over land surfaces, as relatively slowly warming oceans constrain the water vapor available globally.[8] Annual relative humidity in Darwin may decrease on the order of 1—3% over the 21st century given business as usual, although this may not be distinguishable from internal variability in the region.[9] As constant relative humidity represents a specific humidity increase of approximately 7%/°C of warming,[8] this regional decrease will still represent an increase in specific humidity.

Substantial research, both epidemiologic and physiologic, links extreme temperatures to poor health globally and in Australian towns and cities,[10-12] so these climatic changes noted for northern Australia may have consequences for Darwin's health burden. This research was motivated by recognition of the role that environmental heat plays on the human body.[10] Hyperthermia, a state in which the body's core temperature is elevated and organ function is degraded,[13] is prevented by heat transfer to the external environment; in warmer conditions, this occurs predominantly through evaporation.[12]

Heat flux from sweating is proportional to the vapor pressure differential between skin and the environment.[14,15] Increased specific humidity therefore compromises efforts at thermoregulation.

Relatively few epidemiologic studies have directly investigated the effects of humid heat. One study in Barcelona found that the temperature threshold for increased heart disease mortalities increased approximately 2°C on very humid days.[16] Another study of hospital admissions in 12 US cities found no humidity effects in any patients.[17] A third in Brisbane, Australia, used a suite of biometeorologic indices comprising temperature, humidity, and other factors, but found that they did not produce significantly different results to average daily temperature.[18] Some epidemiologic studies have instead treated humidity as a confounding variable in heat—health relationships.[19] Finally, epidemiologic models featuring temperature and humidity also have been comparatively evaluated in European cities.[20] However, none of these studies were situated in tropical locations featuring higher specific humidity.

Humidity effects are better documented in physiologic studies: In one study, study participants who were exercising sweated more when exposed to a controlled increase in humidity, but their sweat evaporation rate plateaued or fell.[21] Heat exposure also causes changes to sleep architecture,[22,23] and humid heat in particular has been documented to compromise the important slow wave sleep and rapid eye movement phases of sleep.[24] The lack of epidemiologic support for humidity worsening heat stress is surprising, given the strong physiologic evidence.

This study aimed to test temperature and humidity relationships to rates of hospital admission among Darwin residents for a selection of diagnoses using daily aggregates of subdaily weather observations, allowing the exploration of diurnally independent contributions from temperature and humidity.

METHODS

For this study, statistical regression models were built using daily temperature and humidity series as predictors and a daily hospital admission count as the health response.

The predictors were daily time series of temperature and humidity. Because Bureau of Meteorology station data does not contain daily aggregates of humidity, subdaily temperature and humidity data from the HadISD station dataset was used instead.[25] Daily maximum, minimum, and mean temperature (T_{max}, T_{min}, T_{mean}) and relative humidity (RH_{max}, RH_{min}, RH_{mean}) were calculated from the subdaily observations. HadISD variables interrogated were temperature and dew point temperature (in degrees Celsius). These were converted to saturated vapor pressure (e_s; Eq. 1) and vapor pressure (e; Eq. 2), which were in turn converted to relative humidity (RH) for this analysis (Eq. 3)[26]:

$$e_s = 6.1078 \cdot exp\left(\frac{17.269T}{T + 237.3}\right) \qquad \text{Eqn. 1}$$

$$e = 6.1078 \cdot exp\left(\frac{17.269T_d}{T_d + 237.3}\right) \qquad \text{Eqn. 2}$$

$$RH = 100 \times \frac{e}{e_s} \qquad \text{Eqn. 3}$$

The health response was a daily admission count running over the period 1993 to 2011, drawn from the admission records of Darwin residents to 5 NT hospitals in the Northern Territory Inpatient Activity dataset. Although some of the hospitals were located outside the admissions cohort, it was assumed that all Darwin residents were exposed to Darwin weather at the time of their admission.

The residential addresses of patients from the 5 hospitals were geocoded by admission dataset's custodian to statistical local areas (SLAs) from the 2006 Australian Statistical Geographical Classification (ASGC)[27]; only residents living in SLAs closer to the Darwin Airport HadISD station than other stations were included as part of the Darwin cohort. Figure 1 shows the boundaries of the SLAs making up this cohort, as well as the Darwin Airport station. SLAs defined in the 1991, 1996, 2001, and 2011 ASGC were also used to build population estimates of the cohort's residence area from Australian censuses, and a stable daily population series was generated by linearly interpolating these estimates, from 80,234 residents at the 1991 census to 122,365 residents at the 2011 census.

Patients' diagnoses were coded using the 9th and 10th editions of the International Classification of Diseases.[28] Admissions were filtered by the principal diagnosis or any secondary diagnoses using criteria developed in personal communications with Peter Tait based on a previous study (2014)[29]: Diagnoses with an existing epidemiologic link to temperature and a physiologic basis that were also prevalent in the NT. A limited number of cases with diagnoses that directly stated heat or sunlight exposure were selected, although these cases represented a negligible fraction of the total, as illustrated by Table 1.

The effects of the 6 predictors on rates of hospital admission for the preselected heat-relevant diagnoses (herein simply admission rates) were studied to look for univariate and bivariate effects, as well as linear and nonlinear effects. First, Poisson generalized linear models (GLMs) were run using a univariate formula to find simple linear relationships:

admission count ~ offset [log(population)] + predictor

Bivariate GLMs were then run to find the linear effects of 2 predictors, with and without an interaction term respectively:

admission count ~ offset [log(population)] + predictor A × predictor B

admission count ~ offset [log(population)] + predictor A + predictor B

To find nonlinear effects, days in the series were binned into fifths—$P_{0\text{-}20}$ for the lowest fifth through to $P_{80\text{-}100}$ for the highest fifth—according to each predictor's quintiles (Table 2), and 95% Poisson confidence intervals were generated for the mean admission rate in each fifth. By testing admission rates between the bins of one predictor within a single bin of a second predictor, nonlinear interactions were considered (e.g., whether T_{mean} affected admission rates specifically on days of high RH_{max}).

Spatial analysis work was done in ArcMap 10.2 (Esri, Redlands, CA), while statistical analysis was done in R 3.0.2 (R Foundation for Statistical Computing, Vienna, Austria).

RESULTS

Tables 3 to 5 list the effects sizes and *P* values of predictors in the univariate and bivariate GLMs; effect

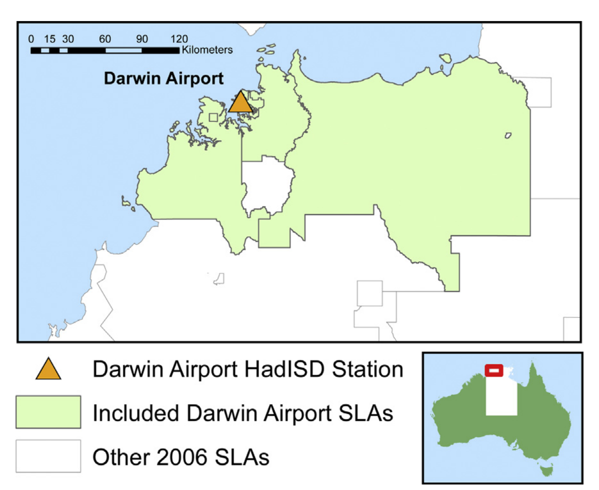

Figure 1. Map of statistical local areas (SLAs), shown in green, associated with the Darwin HadISD station. Other Northern Territory SLAs falling outside the Darwin station catchment are shown in white.

Table 1. Admission Counts, Grouped by ICD-9 and ICD-10 Diagnosis Codes Selected for Aggregation[*]

Condition	Count	ICD-9 Code	ICD-10 Code	Diagnoses
Ischemic heart diseases	5487	410-414	I20-125	PDX
Heart failure	1497	428	I50	PDX
Pneumonia, lower respiratory infections	6689	480-486	J12-J18, J20-J22	PDX
Chronic lower respiratory conditions	3532	491, 492, 494, 496	J40-J44	PDX
Renal failure	693	584-586	N17-N19	PDX
Direct heat	35 (35)	992	T67	PDX, DX2-5
Exposure to sunlight	18 (18)	-	X30	PDX, DX2-5
Total admissions	17,951			

[*] Diagnoses for direct heat and exposure to sunlight are shown before—and, parenthesized, after—ruling out those admitted for other included conditions.

sizes are scaled to show contributions with approximately equal changes in specific humidity. The univariate GLMs showed T_{max} and RH_{max} and RH_{mean} to be statistically significant predictors of hospital admissions (Table 3). Admissions increased 1.74% for every 2°C of T_{max} and 3.73% for every 10 percentage point increase in RH_{max}.

There was no evidence of a statistically significant interaction between temperature and relative humidity (Table 4).

The bivariate GLMs without interactions (Table 5) confirmed the results of the univariate analysis. RH_{max} was very significant, even after accounting for the effects of any temperature predictor, and showed increases in admissions rates of between 3.68% and 4.12% for 10 percentage point increases in RH_{max}. RH_{mean} also continued to be significant after accounting for temperature; admissions rates increased between 1.24% and 2.01% per 10% increase in RH_{mean}. T_{max} remained significant after the effects of RH_{mean} and RH_{min}, increasing admission rates 1.78% for a 2°C increase, but the effect size dropped to 1.54% and became insignificant after accounting for RH_{max}. This suggests that RH_{max} may be a superior predictor to T_{max} in Darwin.

Binning the series by one predictor at a time (Fig. 2) confirmed the importance of T_{max}, RH_{max} and RH_{mean}.

Low RH_{max} (P_{0-20}) showed significantly lower admission rates on high T_{max} days (Fig. 3A) and high T_{mean} days (Fig. 3B). There was limited evidence of RH_{max} effects also occurring on cooler days. Given the results of the bivariate analysis, it seems likely that this coincidence is due to both predictors having nonlinear effects, rather than the presence of an interaction between them.

RH_{mean} also appeared to have a strong nonlinear effect on high T_{max} days (Fig. 4).

DISCUSSION

The analyses indicated that maximum temperature, maximum relative humidity, and mean relative humidity are significant predictors of hospital admission rates in Darwin. This was an unexpected result for 2 reasons. The first is the presence of any sort of humidity effect, as studies addressed here previously found no humidity effect in other locations. The second reason is that maximum temperature and maximum relative humidity occur at opposing times of day: Maximum temperature occurs in midafternoon,

Table 2. Closed and Half-open Intervals Specifying Range of Each Predictor Fifth Used to Bin the Time Series[*]

Bin	Temperature Predictors (°C)[†]			Relative Humidity Predictors (p.p.)[‡]		
	T_{max}	T_{min}	T_{mean}	RH_{max}	RH_{min}	RH_{mean}
P_{0-20}	[23.0, 30.1]	[12.8, 21.0]	[17.9, 25.4]	[26.6, 84.5]	[7.95, 31.3]	[20.6, 62.2]
P_{20-40}	(30.1, 31.1]	(21.0, 23.5]	(25.4, 26.9]	(84.5, 89.2]	(31.3, 42.9]	(62.2, 68.7]
P_{40-60}	(31.1, 32.0]	(23.5, 24.7]	(26.9, 28.0]	(89.2, 94.1]	(42.9, 52.6]	(68.7, 74.1]
P_{60-80}	(32.0, 33.0]	(24.7, 25.8]	(28.0, 28.9]	(94.1, 95.9]	(52.6, 62.5]	(74.1, 80.6]
P_{80-100}	(33.0, 37.4]	(25.8, 31.7]	(28.9, 31.7]	(95.9, 100]	(62.5, 94.7]	(80.6, 98.6]

[*] Parentheses specify an open half; brackets specify a closed half.
[†] Temperature predictors are in units of °C.
[‡] Relative humidity predictors are in units of percentage points.

Table 3. Effect Sizes (Expressed as Percentage Change in the Admission Count Given a 2°C Change in Temperature or a 10 Percentage Point Change in Relative Humidity) and *P* Values (statistical significance) of Univariate Poisson generalized linear models

Predictor*	Effect Size (%)	*P* Value
T_{max}	1.74	0.049
T_{min}	−0.19	0.800
T_{mean}	0.13	0.863
RH_{max}	3.73	<0.001
RH_{min}	0.02	0.968
RH_{mean}	1.21	0.049

* Predictors, top to bottom, are maximum, minimum and mean temperature (T_{max}, T_{min}, T_{mean}) and relative humidity (RH_{max}, RH_{min}, RH_{mean}). Statistically significant predictors are shaded in dark grey ($P < 0.001$) and light grey ($P < 0.05$).

whereas maximum relative humidity occurs overnight, in the early morning. This may not necessarily mean that temperature and humidity act on human health at different times of day; it may simply be characteristic of Darwin's climate, and mean relative humidity was also statistically significant. On the other hand, the statistical significance of mean relative humidity may merely be maximum relative humidity, which it partially captures.

A large body of literature links heat stress to sleep disruption: High overnight temperatures are associated with reduced sleep quality in people of all ages, but especially the elderly.[22,23] In particular, "Humid heat exposure further increases wakefulness, decreases [rapid eye movement] and [slow wave sleep], and excessively suppresses the decrease in [core body temperature]."[30] Sleep disruption is, in turn, associated with immediate respiratory[31] and cardiovascular[32] problems. Because maximum relative humidity occurs early in the morning, hospital admission counts in the models investigated here would likely represent admissions following this peak; this is somewhat less likely for maximum temperature, which occurs later in the day. A comparison of health outcomes across tropical, subtropical, and extratropical locations, where temperature and humidity variances change, would help to make the underlying mechanisms of heat stress clear: Epidemiologic studies situated in the tropics are virtually nonexistent, although a collection of studies used survey data from approximately 40,000 Thai workers to make inferences about occupational heat stress effects.[33-36]

Nighttime humidity effects in Darwin appeared to be strongly nonlinear. Days with less than 84.5% nighttime humidity were associated with significantly

Table 4. Effect Sizes (Expressed as Percentage Change in the Admission Count Given a 2°C Change in Temperature or a 10 Percentage Point Change in Relative Humidity) and *P* Values (Statistical Significance) of Bivariate Poisson generalized linear models predictors

A			B			AB Interaction	
Predictor	Effect Size (%)*	*P* value	Predictor	Effect Size (%)*	*P* value	Effect Size (%)*	*P* Value
T_{max}	−7.18	0.332	RH_{max}	−10.97	0.368	0.05	0.238
T_{max}	2.11	0.376	RH_{min}	1.26	0.868	< −0.01	0.883
T_{max}	−2.65	0.572	RH_{mean}	−8.26	0.404	0.03	0.339
T_{min}	0.39	0.930	RH_{max}	5.80	0.294	−0.01	0.765
T_{min}	−0.40	0.791	RH_{min}	0.06	0.989	< 0.01	0.971
T_{min}	0.53	0.857	RH_{mean}	5.05	0.318	−0.01	0.547
T_{mean}	−3.04	0.613	RH_{max}	0.19	0.983	0.01	0.683
T_{mean}	0.59	0.782	RH_{min}	1.51	0.822	−0.01	0.819
T_{mean}	−0.99	0.813	RH_{mean}	0.46	0.955	< 0.01	0.911
RH_{max}	−10.97	0.368	T_{max}	−7.18	0.332	0.05	0.238
RH_{max}	5.80	0.294	T_{min}	0.39	0.930	−0.01	0.765
RH_{max}	0.19	0.983	T_{mean}	−3.04	0.613	0.01	0.683
RH_{min}	1.26	0.868	T_{max}	2.11	0.376	< −0.01	0.883
RH_{min}	0.06	0.989	T_{min}	−0.40	0.791	< 0.01	0.971
RH_{min}	1.51	0.822	T_{mean}	0.59	0.782	−0.01	0.819
RH_{mean}	−8.26	0.404	T_{max}	−2.65	0.572	0.03	0.339
RH_{mean}	5.05	0.318	T_{min}	0.53	0.857	−0.01	0.547
RH_{mean}	0.46	0.955	T_{mean}	−0.99	0.813	< 0.01	0.911

* Effect sizes of the interaction terms are given for a 1°C change in the temperature predictor and a 1 percentage point change in the relative humidity predictor.

Table 5. **Effect Sizes (Expressed as Percentage Change in the Admission Count Given a 2°C Change in Temperature or a 10 Percentage Point Change in Relative Humidity) and *P* Values (Statistical Significance) bivariate Poisson generalized linear models predictors with no interaction terms**[*]

A			B		
Predictor	Effect Size (%)	p-value	Predictor	Effect Size (%)	p-value
RH_{mean}	1.24	0.045	T_{max}	1.78	0.045
RH_{mean}	2.01	0.008	T_{min}	−1.19	0.066
RH_{mean}	1.39	0.038	T_{mean}	−0.53	0.504
RH_{min}	0.14	0.762	T_{max}	1.78	0.047
RH_{min}	0.22	0.717	T_{min}	−0.35	0.610
RH_{min}	−0.02	0.969	T_{mean}	0.14	0.864
RH_{max}	3.68	< 0.001	T_{max}	1.54	0.084
RH_{max}	4.12	< 0.001	T_{min}	−0.92	0.096
RH_{max}	3.89	< 0.001	T_{mean}	−0.61	0.414
T_{mean}	−0.61	0.414	RH_{max}	3.89	<0.001
T_{mean}	0.14	0.864	RH_{min}	−0.02	0.969
T_{mean}	−0.53	0.504	RH_{mean}	1.39	0.038
T_{min}	−0.92	0.096	RH_{max}	4.12	<0.001
T_{min}	−0.35	0.610	RH_{min}	0.22	0.717
T_{min}	−1.19	0.066	RH_{mean}	2.01	0.008
T_{max}	1.54	0.084	RH_{max}	3.68	<0.001
T_{max}	1.78	0.047	RH_{min}	0.14	0.762
T_{max}	1.78	0.045	RH_{mean}	1.24	0.045

[*] Statistically significant predictors are shaded in dark grey ($P < 0.001$), medium grey ($P < 0.01$), and light grey ($P < 0.05$).

lower admission rates (Figs. 2, 3); this could be because nighttime humidity has a strongly nonlinear relationship, a threshold relationship or simply due to the width of that particular bin. Daytime temperature and mean relative humidity exhibited more evenly distribution bins (Table 2), and Figure 4 shows a monotonic increase in admission rates on hot days with mean relative humidity, suggesting that the bin width could be the cause of this ostensible nonlinearity.

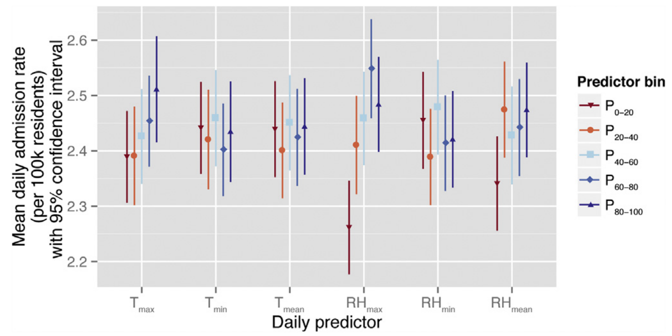

Figure 2. **Mean admission rates (points) and 95% confidence intervals (lines) for the fifths of maximum, minimum, and mean temperature (T_{max}, T_{min}, T_{mean}), from coldest to warmest, and relative humidity (RH_{max}, RH_{min}, RH_{mean}), from driest to wettest.**

Extrapolations of these results for future warming require caution for at least 2 reasons. First, confidence in northern Australian temperature and humidity projections is still limited, especially with regard to seasonal changes.[4] Finer model resolutions have enhanced modeling outcomes in southeast Australia,[37] but the same improvements may not necessarily be possible in northwest Australia; better theoretical understanding of the mechanisms in the northwest may be required. Second, heat-health models based on different indices or statistical models may diverge as the climate changes; some, such as Wet Bulb Globe Temperature[38] and physiological equivalent temperature,[39] are developed on the basis on entirely different methodologies. Heat-health models with robust physiologic explanations of these phenomena are required to reliably extrapolate results.

CONCLUSIONS

This analysis looked at the effects of 6 daily temperature and humidity predictors on hospital admission rates in 5 hospitals in the NT. Using univariate and bivariate Poisson GLM, significant increases in hospital admission rates were found with daytime temperature, nighttime humidity, and average daily humidity. There was no evidence of a significant interaction between them. The effect of nighttime humidity appeared to be strongly nonlinear; high nighttime humidity was associated with increased admission rates on hot days.

These results have implications for heat-related health policies in northern Australia. The results presented here showed that at different times of day temperature and humidity act differently on the health of people living in the NT. Health providers in climatically similar locations may benefit from considering temperature and humidity separately.

These results are preliminary in some ways, and there is room for further research in this area to expand its scope. More complex models of heat stress, such as those employing heat stress indices, were not investigated, and methods of correcting predictors for cyclical effects, such as seasonal or weekly cycles, were avoided in favor of a simpler analysis of temperature and humidity relationships. Furthermore, the study did not account for many socioeconomic or behavioral factors that may influence heat-health, including age, sex, Indigenous status, country of birth, or employment status. The results are limited to Darwin, Australia; comparison with a variety of climatic regions, including tropical and extratropical population centers, is an ideal topic for further research.

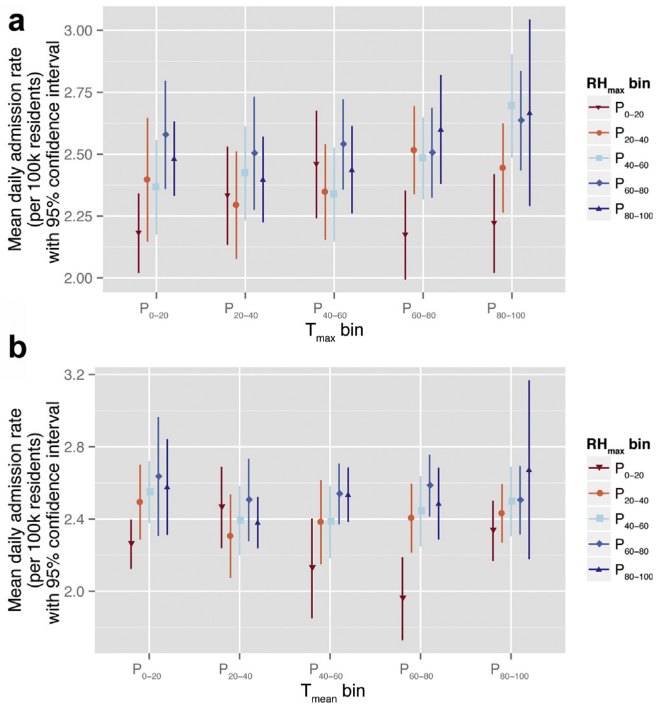

Figure 3. Mean admission rates (points) and 95% confidence intervals (lines) for the fifths of RH_{max} within fifths of (a) T_{max} and (b) T_{mean}.

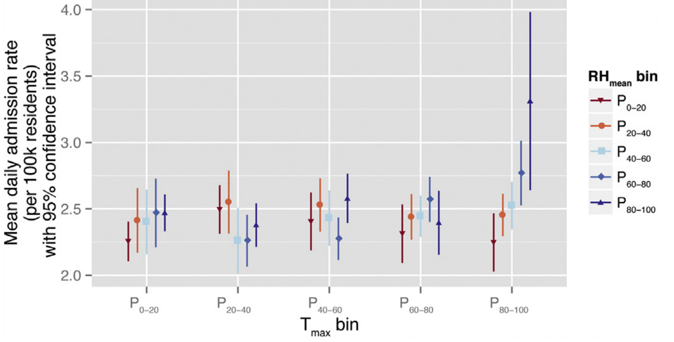

Figure 4. Mean admission rates (points) and 95% confidence intervals (lines) for the fifths of RH_{mean} within fifths of T_{max}.

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REFERENCES

1. 2011 Census QuickStats: Greater Darwin. Australian Bureau of Statistics. Available at: http://www.censusdata.abs.gov.au/census_services/getproduct/census/2011/quickstat/7GDAR?opendocument&navpos=220; 2013. Accessed June 15, 2015.
2. Climate statistics for Australian locations. Australian Bureau of Meteorology. Available at: http://www.bom.gov.au/climate/averages/tables/cw_014015.shtml; 2015. Accessed June 15, 2015.
3. Reisinger A, Kitching R, Chiew F, et al. Australasia. In: Field CB, Barros VR, Dokken DJ, et al., eds.

Climate Change 2014: Impacts, Adaptation, and Vulnerability. Part B: Regional Aspects. Contribution of Working Group II to the Fifth Assessment Report of the Intergovernmental Panel on Climate Change. Cambridge: Cambridge University Press; 2014:1371–438.

4. Alexander LV, Arblaster JM. Assessing trends in observed and modelled climate extremes over Australia in relation to future projections. Int J Climatol 2009;29:417–35.
5. Alexander LV, Hope P, Collins D, Trewin B, Lynch A, Nicholls N. Trends in Australia's climate means and extremes: a global context. Aust Meteorol Mag 2007;56:1–18.
6. Planton S. Annex III: Glossary. In: Stocker T, Qin D, Plattner GK, et al., eds. Climate Change 2013: The Physical Science Basis. Contribution of Working Group I to the Fifth Assessment Report of the Intergovernmental Panel on Climate Change. Cambridge, United Kingdom: Cambridge University Press; 2013: 1447–66.
7. Guide to Meteorological Instruments and Methods of Observation 2008. 7th edVol. Geneva: World Meteorological Organization. Available at: http://www.wmo.int/pages/prog/www/IMOP/CIMO-Guide.html; 2008. Accessed 2015-02-05.
8. Hartmann DJ, Klein Tank AMG, Rusticucci M, et al. Observations: atmosphere and surface. In: Stocker T, Qin D, Plattner GK, et al., eds. Climate Change 2013: The Physical Science Basis. Contribution of Working Group I to the Fifth Assessment Report of the Intergovernmental Panel on Climate Change. Cambridge, United Kingdom: Cambridge University Press; 2013:159–254.
9. Collins M, Knutti R, Arblaster J, et al. Long-term climate change: projections, commitments and irreversibility. In: Stocker T, Qin D, Plattner G-K, et al., eds. Climate Change 2013: The Physical Science Basis. Contribution of Working Group I to the Fifth Assessment Report of the Intergovernmental Panel on Climate Change. Cambridge, United Kingdom: Cambridge University Press; 2013: 1029–136.
10. Bi P, Williams S, Loughnan M, et al. The effects of extreme heat on human mortality and morbidity in Australia: implications for public health. Asia Pac J Public Health 2011;23: 27S–36S.
11. Nitschke M, Tucker GR, Hansen AL, Williams S, Zhang Y, Bi P. Impact of two recent extreme heat episodes on morbidity and mortality in Adelaide, South Australia: a case-series analysis. Environ Health 2011;10:42–50.
12. Loughnan M, Nicholls N, Tapper N. Mortality-temperature thresholds for ten major population centres in rural Victoria, Australia. Health Place 2010;16:1287–90.
13. Kosaka M, Yamane M, Ogai R, Kato T, Ohnishi N, Simon E. Human body temperature regulation in extremely stressful environment: epidemiology and pathophysiology of heat stroke. J Therm Biol 2004;29:495–501.
14. Nadel ER. Control of sweating rate while exercising in the heat. Med Sci Sports 1979;11:31–5.
15. Steadman RG. The assessment of sultriness. Part I: a temperature-humidity index based on human physiology and cothing science. J Appl Meteorol 1979;18:861–73.
16. Saez M, Sunyer J, Tobias A, Ballester F, Anto JM. Ischaemic heart disease mortality and weather temperature in Barcelona, Spain. Eur J Public Health 2000;10:58–63.
17. Schwartz J, Samet JM, Patz JA. Hospital admissions for heart disease. The effects of temperature and humidity. Epidemiology 2013;15:755–61.
18. Vaneckova P, Neville G, Tippett V, Aitken P, FitzGerald G, Tong S. Do biometeorological indices improve modeling outcomes of heat-related mortality? J Appl Meteorol Climatol 2011;50:1165–76.
19. Huang C, Barnett AG, Wang X, Tong S. The impact of temperature on years of life lost in Brisbane, Australia. Nat Clim Chang 2012;2:265–70.
20. Rodopoulou S, Samoli E, Analitis A, et al. Searching for the best modeling specification for assessing the effects of temperature and humidity on health: a time series analysis in three European cities [e-pub ahead of print]. *Int J Biometeorol.* http://dx.doi.org/10.1007/s00484-015-0965-2. 2015.
21. Alber-Wallerström B, Holmér I. Efficiency of sweat evaporation in unacclimatized man working in a hot humid environment. Eur J Appl Physiol Occup Physiol 1985;54:480–7.
22. Ohnaka T, Tochihara Y, Kanda K. Body movements of the elderly during sleep and thermal conditions in bedrooms in summer. J Physiol Anthropol 1995;14:89–93.
23. Ohnaka T, Takeshita J. Upper limit of thermal comfort zone in bedrooms for falling into a deep sleep as determined by body movements during sleep. In: Tochihara Y, Ohnaka T, eds. Environmental Ergonomics - The Ergonomics of Human Comfort, Health, and Performance in the Thermal Environment. Oxford: Elsevier; 2005:121–6.
24. Okamoto-Mizuno K, Mizuno K, Michie S, Maeda A, Iizuka S. Effects of humid heat exposure on human sleep stages and body temperature. Sleep 1999;22:767–73.
25. Dunn RJH, Willett KM, Thorne PW, et al. HadISD: a quality-controlled global synoptic report database for selected variables at long-term stations from 1973–2011. Clim Past 2012;8: 1649–79.
26. Murray FW. On the computation of saturation vapor pressure. J Appl Meteorol 1967;6:203–4.
27. 1259.0.30.002-Statistical Geography - Australian Standard Geographical Classification (ASGC), Digital Boundaries. Australian Bureau of Statistics; 2006. Available at: http://www.abs.gov.au/AUSSTATS/abs@.nsf/Lookup/1259.0.30.002Main+Features12006; 2011. Accessed April 16, 2014.
28. International Statistical Classification of Diseases and Related Health Problems. Tenth Revision. 2nd ed. Geneva, Switzerland: World Health Organization; 2004.
29. Webb L, Bambrick H, Tait P, Green D, Alexander L. Effect of ambient temperature on Australian Northern Territory public hospital admissions for cardiovascular disease among Indigenous and non-Indigenous populations. Int J Environ Res Public Health 2014;11: 1942–59.
30. Okamoto-Mizuno K, Mizuno K. Effects of thermal environment on sleep and circadian rhythm. J Physiol Anthropol 2012;31:14–22.
31. Gabor JY, Cooper AB, Hanly PJ. Sleep disruption in the intensive care unit. Curr Opin Crit Care 2001;7: 21–7.
32. Kloner RA. Natural and unnatural triggers of myocardial infarction. Prog Cardiovasc Dis 2006;48: 285–300.
33. Tawatsupa B, Lim LL-Y, Kjellstrom T, Seubsman S-A, Sleigh A; the Thai Cohort Study Team. The association between overall health, psychological distress, and occupational heat stress among a large national cohort of 40, 913 Thai workers. Glob Health Action 2010;3:1–10.
34. Tawatsupa B, Lim LLY, Kjellstrom T, Seubsman S, Sleigh A; the Thai Cohort Study Team. Association between occupational heat stress and kidney disease among 37 816 workers in the Thai Cohort Study (TCS). J Epidemiol 2012;22:251–60.
35. Tawatsupa B, Yiengprugsawan V, Kjellstrom T, Seubsman S-A, Sleigh A. Heat stress, health and well-being: findings from a large national cohort of Thai adults. BMJ Open 2012;2:e001396.
36. Tawatsupa B, Yiengprugsawan V, Kjellstrom T, Berecki-gisolf J, Seubsman S, Sleigh A. Association between heat stress and occupational injury among Thai workers: findings of the Thai cohort study. Ind Health 2013;51:34–46.

37. Evans JP, McCabe MF. Effect of model resolution on a regional climate model simulation over southeast Australia. Clim Res 2013;56:131–45.

38. American College of Sports Medicine. Prevention of thermal injuries during distance running. Med Sci Sports Exerc 1984;16:9–14. 17.

39. Höppe P. The physiological equivalent temperature—a universal index for the biometeorological assessment of the thermal environment. Int J Biometeorol 1999;43:71–5.