BMC Public Health

RESEARCH Open Access

Impact of extreme heat on health in Australia: a scoping review

Patrick Amoatey[1*], Zhiwei Xu[5], Chinonso Christian Odebeatu[1], Neha Singh[1,6], Nicholas J. Osborne[1,2,3,4] and Dung Phung[1,4*]

Abstract

Climate change has been recognized as a driver of increased heatwave events in the Asia Pacific region, including Australia. This review systematically retrieved and summarized published evidence on heat-related health impacts in Australia, focusing on heat-associated deaths, morbidity, and vulnerability. We searched PubMed, Scopus, Embase, and Web of Science for heat-health studies published in Australia between 2007 and 2023. A total of 64 articles met the inclusion criteria and were included in our review. Most were epidemiological studies [56 (87.5%)], which accumulatively considered 85 different cause-specific diseases linked to deaths, hospital admissions, emergency department (ED) presentations, and ambulance callouts, while eight studies focused on heat vulnerability index (HVI) assessment. We found strong evidence of increasing risks for heat-associated deaths among individuals with mental/behavioral disorders, cardiovascular diseases, diabetes, and respiratory disease. Evidence supporting an increasing risk for renal/genitourinary-related deaths was limited. The majority of studies reported an increase in heat-associated hospitalization, particularly for patients with renal disease, neurological disease, stroke, mental disorders, diabetes, and ischemic heart disease. Heat-associated ambulance callouts was prominent for patients with cardiovascular and respiratory diseases. This evidence suggests that these heat-related diseases should be used as health indicators for developing and validating HVI in Australia. Most studies did not examine the long-term changes in vulnerability and lacked evaluation with cause-specific health data. Future research must incorporate HVI across diverse climate change scenarios to more accurately inform long-term adaptation measures among vulnerable communities. In addition, research should target nationwide longitudinal heat vulnerability and examine the benefits of using HVI in heatwave action plans.

Keywords Extreme heat, Deaths, Morbidity, Heat vulnerability index, Systematic review, Australia

*Correspondence:
Patrick Amoatey
p.amoatey@uq.edu.au
Dung Phung
d.phung@uq.edu.au
[1]School of Public Health, Faculty of Health, Medicine and Behavioural Sciences (HMBS), The University of Queensland, Brisbane, QLD 4072, Australia
[2]School of Population Health, University of New South Wales, Sydney, NSW 2052, Australia
[3]European Centre for Environment and Human Health (ECEHH), University of Exeter Medical School, Knowledge Spa, Royal Cornwall Hospital, Truro, Cornwall, Exeter TR1 3HD, UK
[4]Queensland Alliance for Environmental Health Sciences, The University of Queensland, Brisbane, Australia
[5]School of Medicine and Dentistry, Griffith University, Gold Coast, Queensland, Australia
[6]Centre for Atmospheric Sciences, Indian Institute of Technology Delhi, Hauz Khas, New Delhi 110016, India

Introduction

Climate change has been recognized as a key driver behind the recent surge in extreme heat events, resulting in increasingly high frequencies and intensities [1, 2]. Asia Pacific regions, including Australian cities, are affected by extreme heat events due to warming and the seasonal effects of El Niño Southern Oscillation [3, 4].

There is a particularly evident threat to Australians with existing health complications, which has been well-documented in literature. Individuals with kidney disease exhibit an increase in risk of heat-associated hospitalization, with a relative risk (RR) of 1.29, 95% [confidence interval (CI): 1.16–1.44] in the Northern Territory [5]. Those diagnosed with respiratory disease had a RR of 1.12 (95% CI: 1.03–1.21) for heatwave-related deaths in Queensland [6]. Increased risk of heatwave-associated out-of-hospital cardiac arrest (RR 1.25 95% CI: 1.04–1.50) was observed in Brisbane [7], and heat-related deaths from diabetes [odds ratio (OR) 1.22 95% CI: 1.03–1.46] was found in Sydney [8]. Extreme heat affects human health via several biological pathways, most of which act to dysregulate systemic blood flow, thus increasing myocardial strain, blood viscosity, and total cholesterol levels to cause coronary and cerebral thrombosis [9, 10]. Inhalation of hot air may trigger high respiratory ventilation rate and bronchoconstriction among healthy people and asthmatic patients [11]. Younger adults may experience high plasma-based kidney biomarkers, particularly creatinine and cystatin C, which increase the risk of kidney complications during extreme heat conditions [12].

Australia's heat-related mortality and morbidity risk is increasing among its cities and States [13–16]. The National Coronial Report shows that 354 Australians were killed by heatwaves from 2000 to 2018 [17]. The 2009 catastrophic heatwave episode caused 20 direct deaths, increased general practice attendance by 65% among older people (>75 years), and increased total cardiac arrests by 280% [18]. A recent report shows that more than 10 million Australians are exposed to hazardous heat. Extreme heat will negatively affect quality of life, with an estimated loss of $ 211 billion in agriculture and labor productivity by the year 2050 [19]. Further estimates from the Australian Climate Council indicate that there will be a $ 571 billion loss in the property market because of extreme climate events, including heatwaves [20].

In 2023, the Australian Department of Health and Aged Care launched a national health and climate strategy for 2024–2028 to help protect the health of the public from extreme climate events (i.e., heatwaves) by building resilient communities. One of the four enablers of the strategy is the prioritization of climate change (extreme heat) and health research to help develop effective adaptation responses [21]. Evidence from heat vulnerability research plays a substantial role in improving health and well-being by fostering climate-resilient health systems and driving sustainable city design.

Although heat research has been well-documented in Australia for the past two decades [22], limited studies have comprehensively compiled or reviewed heat vulnerability studies to inform heat action plans, strategies, and adaptation policies. Therefore, this scoping review aims to comprehensively examine Australia's current heat vulnerability studies.

Therefore, to achieve this objective, the review will address the following four research questions:

(1) What are the trends and distributions of heat-health studies in Australia?
(2) What are the common diseases from heat-related mortality, hospital admissions, emergency department presentations, and ambulance callouts?
(3) What is the current evidence of spatial heat vulnerability assessment index (HVI) studies in Australia?
(4) What are the strengths, limitations, and recommendations for further studies?

Methods

We conducted a systematic scoping review to answer the four research questions. First, we developed a priori protocol for this scoping review and made it available at Open Science Framework OSF (https://osf.io/ewdb9). The protocol was based on Preferred Reporting Items for Systematic Reviews and Meta-Analysis extension for Scoping Reviews (PRISMA-ScR) (Tricco et al., 2018).

Search strategy

We developed a search string and conducted the initial literature search in PubMed, Scopus, Embase, and Web of Science on October 16, 2022, and the final search on December 4, 2023. The search strings were developed following the PECO (populations, exposures, comparators, and outcomes) conceptual framework [23], as shown in Supplementary Table S1. We applied different search fields (such as title, abstract, and keywords) in combination with truncations, wildcards, subject medical headings (MeSH), and terminologies to develop the strings. The typical search terms used were "heatwave" OR "extreme temperature," OR "mortality" OR "morbidity," OR "hospital emergency visits," OR "ambulance dispatch," AND "Australia". The detailed search strategies for all four electronic databases are available in Supplementary Table S2.

Selection criteria

After removing the duplicates, two reviewers (P.A. and N.S.) independently assessed the titles and abstracts of

all identified papers. Studies selected during the title and abstract screening were considered for full-text evaluation. During the screening process (i.e., title and abstract, full text), three-stage inclusive/exclusive criteria were applied. First, we included only full-length epidemiological studies (e.g., case-crossover, time-series) that have applied exposure-response models. Second, we included studies that used heat metrics such as maximum /minimum /mean temperature, heatwave, and excess heat factor (EHF). EHF is a heatwave intensity measure that characterizes heatwaves according to their severities based on a 3-day mean in relation to the 95th percentile of long-term (e.g., past 20 years) and the recent (30 days) mean daily temperature [24]. Third, we included heat vulnerability assessment studies that relied on exposure, sensitivity, and adaptive capacity variables.

Studies on indoor thermal comfort, bushfires, occupational health, sports, toxicology, and accidental burns were not considered. All international multi-country/city studies, systematic reviews, surveys, and interview-based studies were excluded. Studies that met the inclusive/exclusive eligibility criteria progressed to data extraction [25].

Data extraction and analysis

Two reviewers (P.A. and N.S.) conducted data extraction. For heat-health epidemiological studies, the following information was collected: authors, study year, and geographical units (e.g., State level, cities, etc.,). We extracted the heat exposure metric (e.g., EHF), study period, population, the design used, statistical models applied, type of health outcome (e.g., hospital admissions), cause-specific diseases (e.g., Asthma), and the main findings of the study. We extracted data only for heat components from studies that have assessed other climatic variables (rainfall, humidity, air pressure). Where a particular study has employed more than one heat exposure metric, we extracted data for all the metrics. Data was not extracted from studies that combined results from multiple cities, regions, or geographical units. If a study used diverse designs, we extracted results from all designs unless they produced similar outcomes.

For HVI studies, we extracted information about study locations, geographical scale (e.g., cities, States, etc.,), heat exposure, sensitivity, adaptive capacity variables used, the method of HVI construction, and the validation status (e.g., whether the HVI was associated with heat-related mortality and morbidity). All extracted results were presented in the form of tables and bar charts. Any disagreements between the reviewers were resolved via discussion with the last author. Given the nature of the review, we did not assess the quality of individual studies or conduct a meta-analysis based on the PRISMA ScR guidelines.

Results

Study selection processes and characteristics

The literature search generated 696 articles, 687 from scientific databases, and 9 from other sources, primarily manual search. After de-duplication, title and abstract screening, and full-text screening, 64 studies were retained for the systematic scoping review.

The retained articles comprise 56 epidemiological studies and 8 HVI studies (Fig. 1). The publication rate has increased for the past 13 years (2012–2014), indicating a growing research interest in heat-health (Fig. 2).

Description of the study

Supplementary Figure S1 shows the distribution of heat-health studies across Australian States and cities, along with the types of study designs used for different health outcomes. Most studies were conducted in Queensland/Brisbane, and South Australia/Adelaide were mainly case-crossover in design.

Hospital admissions

We identified 81 heat-health epidemiological studies (Supplementary Table S3, Supplementary Figure S1). Of these, 36 studies investigated the effect of extreme temperature exposures on hospital admissions among the general population. The majority of the studies described weather exposure as heatwave ($n=17$), utilized case-crossover ($n=15$) study design, and analyzed the data using Poisson regression models ($n=18$). Most of the hospital admissions studies ($n=27$) were focused on the capital cities, especially in Brisbane ($n=10$) and Adelaide ($n=10$), with fewer studies in Sydney ($n=3$), Melbourne ($n=1$), Perth ($n=1$), Hobart ($n=1$) and Darwin ($n=1$). Only nine hospital admission studies were conducted at the State level; the majority of them were in Queensland ($n=4$), with few studies in South Australia ($n=2$), New South Wales ($n=1$), Western Australia ($n=1$), and Northern Territory ($n=1$).

ED presentations

Twenty of the heat-health epidemiological studies assessed the impact of extreme temperature on ED presentations (Supplementary Table S3, Supplementary Figure S1). The majority of the studies were described in the context of heatwaves ($n=12$) as well as other exposure types, including heat ($n=2$), hot temperature (1), extreme temperature ($n=2$), summer temperature/heatwave ($n=1$), and temperature ($n=2$). Most of these studies determined associations using Poisson regression models ($n=18$) within an ecological study design ($n=11$).

Only one study was a retrospective cohort using the Rural Acute Hospital Data Register (RAHDaR), which collects comprehensive data on visitations made to the various EDs in Southwestern Victoria, including urgent

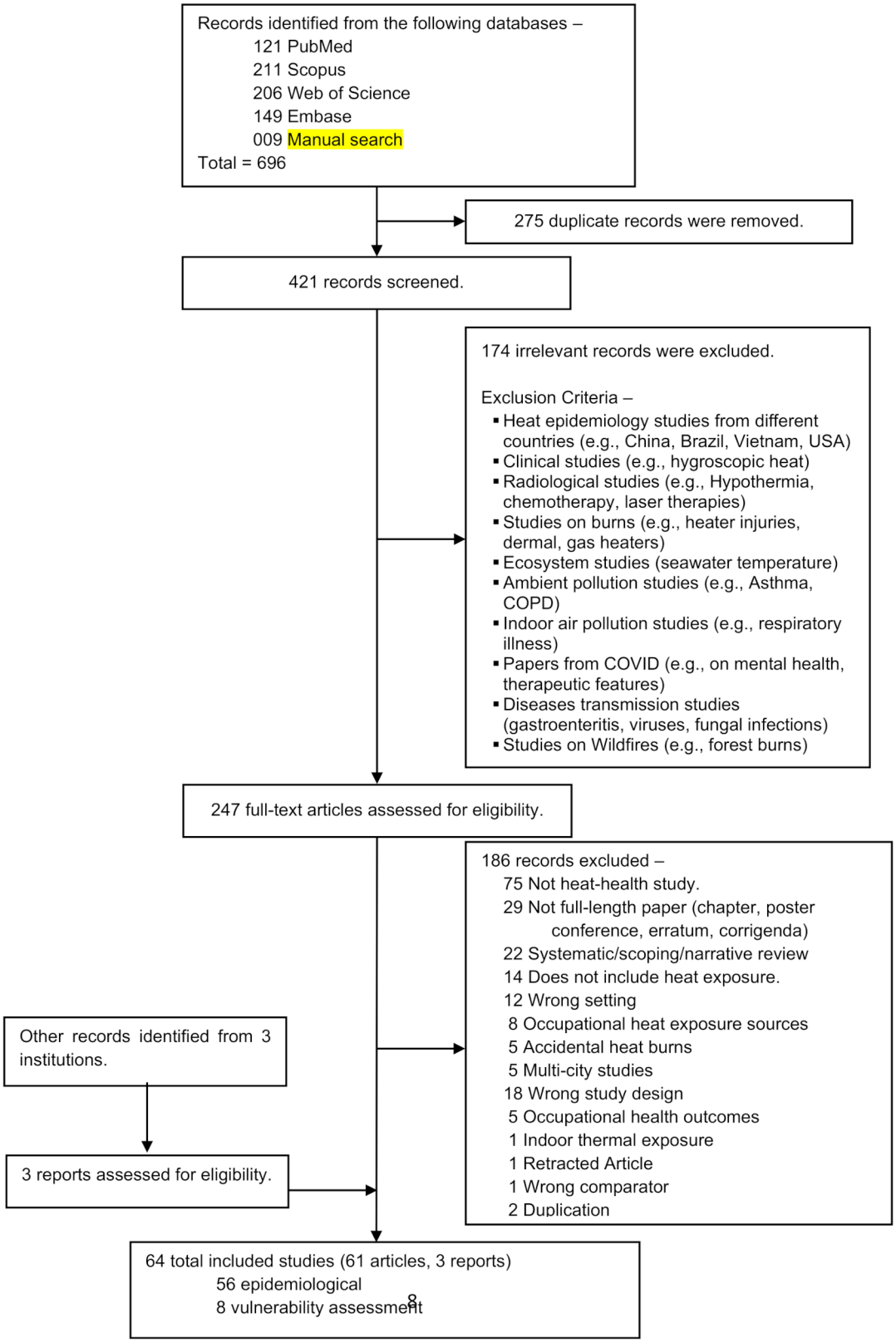

Fig. 1 PRISMA flow chart showing the study selection process

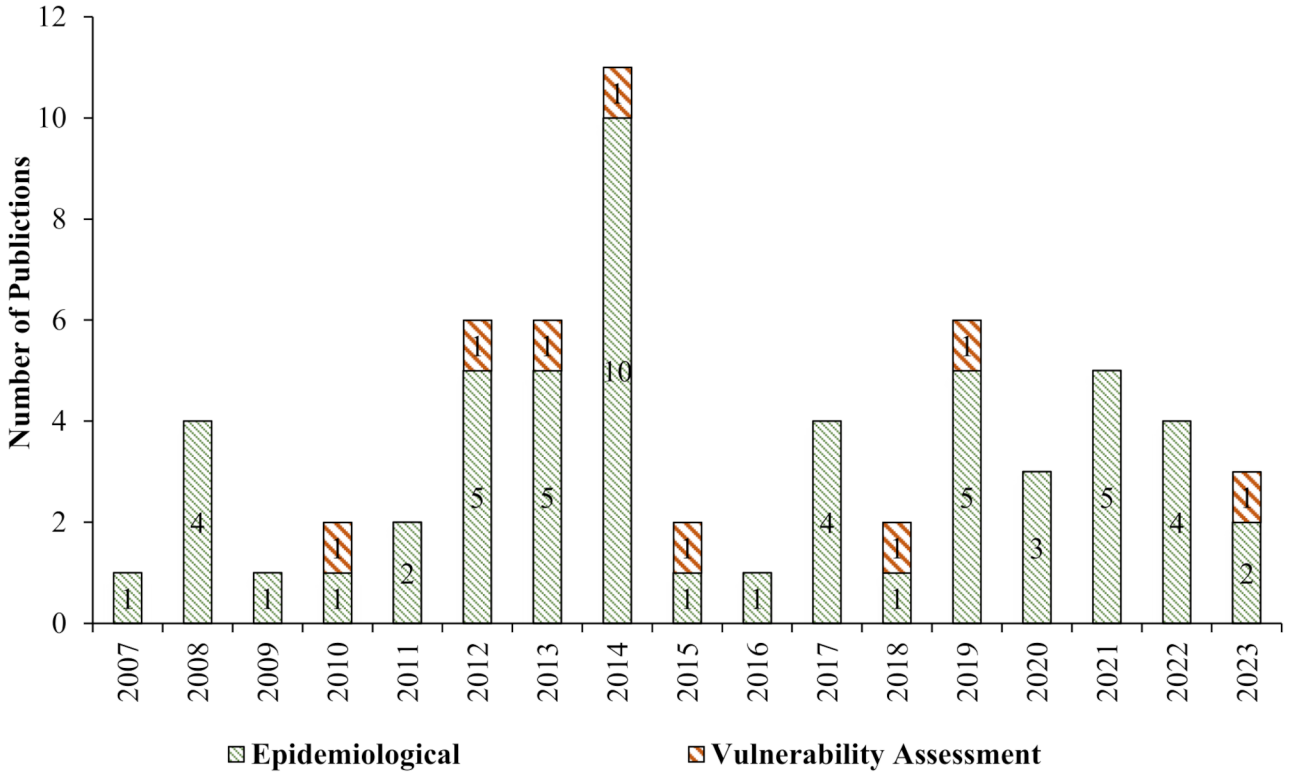

Fig. 2 Different study types published from 2007 to 2023, with the number of heat-health studies ($N = 64$) for each study group indicated by the publication year

care centers [26]. The majority of the ED presentation studies were located in Brisbane ($n = 6$) and Adelaide ($n = 5$); a very small number of the studies were found in Sydney ($n = 1$) and Perth ($n = 1$). All the State-level studies were almost equally distributed, with each state having only one study except Tasmania ($n = 2$) (Supplementary Table S3, Supplementary Figure S1).

Ambulance callouts

Ten Studies investigated the association between extreme temperature and ambulance callouts (Supplementary Table S3, Supplementary Figure S1). The majority of them described the exposure as heatwave ($n = 8$), while others were characterized as heat ($n = 2$) and hot temperature (1). Most studies used a time-series study design ($n = 4$) and a Poisson regression statistical approach ($n = 6$). These six studies were focused only on three major populated capital cities of Australia - Adelaide ($n = 3$), Brisbane ($n = 2$), and Sydney ($n = 1$). Only one State-level study has been carried out in Queensland, South Australia, New South Wales, and Tasmania.

Mortality

Seventeen studies have investigated the link between extreme temperature and mortality in Australia (Supplementary Table S3, Supplementary Figure S1). A greater number of the studies described the association in the context of heatwave ($n = 12$), with a few studies in heat ($n = 3$), extreme temperature ($n = 1$), and summer temperature/heatwave ($n = 1$). The most common study designs used were case-crossover ($n = 7$) followed by ecological study ($n = 4$), time-series ($n = 3$), and case-series ($n = 3$). The statistical approaches used were mostly Poisson regression (8) and conditional logistic regression (5), and only one study used a generalized estimating equation and negative binomial regression. Capital city-specific heat-related mortality studies were primarily conducted in Brisbane ($n = 6$), followed by Adelaide ($n = 4$), with few studies in Sydney ($n = 2$) and Perth ($n = 1$). Limited evidence for heat-mortality association was found at the state level, with only one study conducted in Queensland, Northern Territory, South Australia, and New South Wales (Supplementary Table S3).

Cause-specific health outcomes

As indicated in Supplementary Table S4, we reviewed all the cause-specific heat-associated morbidity and mortality in Australia for (a) hospital admissions, (b) ED presentations, (c) ambulance callouts, and (d) deaths. Supplementary Figure S2 displays the effect estimates [as relative risk (RR), incident rate ratios (IRR), and odds ratio (OR)] of the various heat-related morbidity

and mortality according to disease type, the associated human system, and age categories.

Cause-specific hospital admissions

Under the broad category of hospital admissions, 63 studies have investigated different types of diseases, as shown in Supplementary Table S4. The various diseases from heat-associated hospital admissions could be known by extending hospital admissions, as shown in Supplementary Table S5. Due to the heterogeneity in study designs employed by these studies, the heat-related outcomes from these diseases have been reported as OR, RR, and IRR in Supplementary Figure S2.

Diseases of the genitourinary system Fifteen studies focused on diseases of the genitourinary system comprising seven specific diseases, including acute renal failure (ARF), dialysis (DLS), renal failure (RNF), urolithiasis (URS), urinary tract infections (UTI), and urinary diseases (URD). Among these health outcomes, the most reported disease showing heat-associated hospitalizations was renal disease (RND) [27–35].

A study in Adelaide (1995–2006) found an increased risk of RND [IRR = 1.10 (95% CI: 1.00-1.20)], and ARF [IRR = 1.25 (95% CI: 1.03–1.51)] heat-associated hospitalizations. However, DLS [IRR = 1.01 (95% CI: 0.96–1.05)] was not associated with extreme heat [27]. Earlier research reported a 13% rise in RND hospital admissions from 1995 to 2006, higher than during 1995–2006 [32]. In the same city (Adelaide), there was a marginal increase in RND hospital admissions among the elderly despite the varying frequencies in 2008 [IRR = 1.23 (95% CI: 1.03–1.47)] and 2009 [IRR = 1.48 (95% CI: 1.15–1.88)] heatwave episodes days [31]. Additional factors contributing to hospitalization risk in Adelaide include living alone and low socioeconomic conditions [36]. Residents in Adelaide are shown to be highly susceptible to RND and ARF admissions, including more extended hospital stays [35, 37, 38]. This association was also found among people who either had been diagnosed with RNF[OR = 1.13 (95% CI: 0.89–1.43)] or had underlying RNF conditions [OR = 1.18 (95% CI: 1.09–1.27)] in a regional study in New South Wales [28].

In Queensland, there was an overall decrease in RND hospital admissions from 1995 to 2016. However, males and elderly individuals aged 75 years and older were the most susceptible groups [29, 30]. RNF had a higher attributable fraction compared to UTI [30]. A city-scale analysis in Brisbane also showed an increase in RND admissions [OR = 1.2 (95% CI: 0.38–3.84)] when adjusted for particulate matter with an aerodynamic diameter of 10 micrometers (PM_{10}) [34]. Children in Brisbane showed high susceptibility to RND heat-associated hospital admissions, and caution was given about the importance of improving pediatric healthcare services against extreme temperatures [33]. In Sydney, both single and three-day severe/extreme heat events were associated with increased admissions for RNF [OR = 1.26 (95% CI: 1.16–1.38)] and URS [OR = 1.18 (95% CI: 1.02–1.25)] [8]. Similarly, in Western Australia, severe/extreme heatwave was linked to a 16% increase in RND [IRR = 1.16 (95% CI: 1.05–1.28)] hospital admissions, with the elderly (≥ 60 years) showing the highest vulnerability [39].

Diseases of the circulatory system Eleven studies delved into the effect of extreme heat on hospital admissions from circulatory system diseases. The specific diseases studied included circulatory system disease (CSD), cerebrovascular disease (CBD), acute myocardial infarction (AMI), ischemic heart disease (IHD), heart failure (HTF), stroke, high blood pressure (HBP), and heart diseases (HRD). Two of these studies were conducted at regional levels in New South Wales [28] and Northern Territory [40]. The former study highlighted that people with underlying CSD (12%), including cardiac arrest (14%), and cerebrovascular diseases (11%) [(non-statistically significant)] were at higher risk of hospital admissions after exposure to a 3-day moving average temperature. On the contrary, findings from L Webb, H Bambrick, P Tait, D Green and L Alexander [40] suggested that there were reductions in heat-associated admissions from IHD (63%) and HTF (40%) in older (≥ 65 years) females and males, respectively.

The remaining nine studies examined the effect of extreme heat exposures on several types of CSD-caused admissions across the major Australian capital cities. In Melbourne, for example, episodic extreme heat events increased acute AMI admissions [41]. In Adelaide, a case-series study design highlighted increased IHD admissions among the elderly [31, 32]. A matched case-control study in Adelaide also found increased hospital admissions for heart diseases despite using air conditioning [36]. In Brisbane, XY Wang, AG Barnett, W Yu, G FitzGerald, V Tippett, P Aitken, G Neville, D McRae, K Verrall and S Tong [34] reported a 65% increased odds of stroke admissions from heat-related hospitalizations among those aged 15–64. Still, their previous study showed a non-significant increase in stroke admissions among participants aged 65 years and younger [42]. Z Xu, S Tong, HC Ho, H Lin, H Pan and J Cheng [43] identified that individuals with diabetes are highly susceptible to heat-associated CSD admissions, particularly AMI. LA Wilson, GG Morgan, IC Hanigan, FH Johnston, H Abu-Rayya, R Broome, C Gaskin and B Jalaludin [8] showed a 4% increase in odds of IHD hospitalizations during severe and extreme heat exposures.

Mental and behavioral disorders Eight studies focused on heat-associated hospital admissions for mental and

behavioral disorders, investigating 17 different specific diseases: mainly mental disorders (MDS), depression (DPR), dementia (DEM), all mental and behavioral disorders (MBD), organic including symptomatic, mental disorder (OSMD), mental and behavioral disorders due to psychoactive substance use (MBPS), schizophrenia, schizotypal, and delusional disorders (SSDS), mood disorders (MOD), neurotic, stress-related, and somatic disorders (NSSD), behavioral syndromes associated with psychological disturbances and physical factors (BSPF), disorders of adult personality and behavior (DAPD), mental retardation (MTR), disorders of psychological development (DPD), behavioral and emotional disorders with onset usually occurring in childhood and adolescence (BEDC), psychotic disorder (PSD), senile degeneration of brain (SDB), and senility (SEN).

A comparative study in Adelaide found that heatwave events in 2008 increased MDS-related hospitalizations by 64% in children (5–14 years) and 10% in the elderly (75 + years), while the 2009 event saw decreases of 3% in adults (15–65 years) and 5% in the elderly [31]. An earlier study in the same geographical area (Adelaide) also found an overall increase in MDS hospitalization by 7% across the entire population but a higher risk among the elderly (17%) [32]. S Williams, M Nitschke, T Sullivan, GR Tucker, P Weinstein, DL Pisaniello, KA Parton and P Bi [44] reported a 3% increase in MDS admissions when adjusted for ozone and PM_{10} following a 10℃ increase in maximum temperature. Other studies did not evaluate the specific causes of hospital admissions but found that MDS was among several types of diseases that contributed to an overall increase in the length of hospital stay in Adelaide [38]. An ecological study (1993–2006) also found a 7.3% increase in MDS hospitalization, attributing it to organic diseases, dementia, mood disorders, and senility [14].

A case-control study in Sydney between 1991 and 2009 found 2% increase in odds of overall MDS ([OR = 1.02 (95% CI: 1.00-1.03]) caused hospitalization at 95th percentile temperature exposures. The most susceptible groups were those diagnosed with MTR (27%) and PSD (3%), though the association was not statistically significant [45]. A study assessed the impact of single and continuous (3-day average in a row) heat exposures by employing a case-crossover approach. The results showed a 3% and 9% increase in MDS hospital admissions among all age groups, and those with 65 plus years, respectively [8]. One study in Brisbane examined whether extreme heat (≥ 37 °C for two or more consecutive days) predicted MDS admissions among the population aged 65–74 years and found a non-significant association (OR = 1.50; 95% CI: 0.45–4.99) [34].

Disease of the respiratory system Seven epidemiological studies have assessed respiratory system-caused hospital admissions, focusing on four diseases: all respiratory disease (RSD), chronic obstructive pulmonary diseases (COPD), Asthma, and pneumonia (PNM). These studies were mostly conducted in Adelaide (5), Sydney (1), and New South Wales (1).

The first regional-scale heat-epidemiological study in New South Wales found no association between heat exposure and RSD (including COPD and ASM) as the primary disease diagnosed. There was an increase in admissions for those with underlying health conditions for RSD (14%), COPD (12%), and ASM (57%) for 3-day moving average heat exposure [28]. A case-series study conducted by M Nitschke, GR Tucker, AL Hansen, S Williams, Y Zhang and P Bi [31] found that the 2008 extreme heat event is not a predictor of RSD for all the populations in Adelaide except for 5–14 years and 15–64 years age groups with IRRs of 1.02 (95% CI: 0.71–1.47), and 1.06 (95% CI: 0.77–1.47), respectively. For the subsequent 2009 heatwave event, M Nitschke, GR Tucker, AL Hansen, S Williams, Y Zhang and P Bi [31] later found a 3% increase in RSD admissions among infants (0–4 years). A matched-case control study (43 cases, 25 control) during the same 2009 heatwave event was not significantly associated with RSD, including ASM [36]. The previous evidence (1993–2006) has suggested that only populations within 15–64 years (3%) had high vulnerability to RSD hospitalizations, but the association was not significant [32]. Other studies did not investigate age-specific association but found that both minimum (3%) and maximum (2%) temperatures equally increase RSD admissions, but such association was not statistically significant [44].

Two studies focused on Sydney. The first study when adjusted for heat (95 and 99th percentiles) levels with both ozone and PM_{10}, showed a statistically significant association with RSD [OR = 1.02 (95% CI: 1.01–1.04)] especially 11% increase for pleurisy (PLY), and empyema (EYA), but was protective against ASM ([OR = 0.88 (95% CI: 0.83–0.94)] [45]. The second case-crossover study found that RSD (3%), COPD (6%), and PNM (5%) all showed a similar magnitude of association for 3-day moving average heat exposure. No association was found for ASM [8].

So far, only one study was conducted in Brisbane (1996–2005) but did not find an association between extreme temperature (defined as ≥ 37℃ for two or more consecutive days) and RSD among different age groups. Those within 15–64 years showed the highest vulnerability [OR = 7.7 (95% CI: 0.8–74.9] to RSD admissions, but the association was not statistically significant [34].

Disease of the cardiovascular system Seven studies investigated diseases of the cardiovascular system by

focusing on three main types - chronic heart failure (CHF), cardiac failure (CDF), and all cardiovascular diseases (CVD)- distributed across different geographical scales in Australia. Two of the seven studies were at the State level, particularly in South Australia and Queensland. A retrospective cohort study involving 2961 patients (between 1994 and 2004) in South Australia assessed the impact of hot summer heatwave on heart failure hospitalizations. The study found an increase in 33 cases of CHF-caused admissions [Rate = 33 (95% CI: 30–37)/1000 people] due to extreme summer temperature, revealing that the elderly (≥ 75 years old) were the most at-risk group [46]. The Queensland study (1995–2016) employed a case-crossover design to determine the risk of heat-related CVD admissions. The findings suggest a 10% increase in the risk of heat-associated CVD admissions overall, with a 20% increase observed among those aged 0–59 years; both males and females had an equal risk (10%) of CVD admissions [15].

At the city scale, some studies in Brisbane did not observe any association of extreme heat levels with CVD hospitalization [OR = 1.04 (95% CI: 0.87–1.24] [34]. A similar outcome was also reported in Sydney (1997–2010), where single heat exposure showed a non-significant association with CVD (10%) admissions for all ages, including those aged 65 years and above [8]. In contrast, in the same city (Sydney), a study (1991–2009) found a marginal increase (1%) in the odds of overall CVD hospitalizations [OR = 1.01 (95% CI: 1.00-1.02)]. It should be noted that ozone and PM_{10} were the main confounding variables applied to the model [45]. Only one study was found in Adelaide (1993–2009), and the researchers concluded that extreme heat was not associated with CVD admissions [OR = 1.0 (95% CI: 0.99-1.00)] at a minimum temperature level of 26℃ [44].

Disease of the endocrine, nutritional, and metabolic system Six studies assessed hospitalizations due to diseases of the endocrine, nutritional, and metabolic systems, focusing on diabetes (DBT), and hyperlipidemia (HDM). These studies were primarily conducted in Brisbane ($n = 3$), Sydney ($n = 1$), Adelaide ($n = 1$), and New South Wales ($n = 1$).

A study found no association between hospital admissions and extreme heat events (for 3 days average in a row) among people who have been diagnosed with DBT [OR = 1.13 (95% CI: 0.97–1.31)], and those with underlying DBT [OR = 1.04 (95% CI: 0.98–1.10)] health conditions [28]. Similar findings were reported in another study that accounted for air pollution, such as ozone, PM_{10}, and nitrogen oxide (NO_2) levels [34]. Wilson et al. (2013) reported a positive association between DBT admissions and heat exposure without accounting for air pollution exposure. The intensity of the heatwave can drive DBT admissions. For example, a retrospective cohort study in Brisbane (2005–2015) indicates that admissions increased significantly during moderate (19%, $p = 0.026$) and high (37%, $p = 0.00$) but not low (9%, $p = 0.15$) heat intensity heatwave [47]. A case-control study involving 143 participants with underlying DBT conditions showed a significant increase in DBT hospitalization [36]. Others have also predicted that combining DBT with other diseases can increase the overall length of hospital admissions [38]. Only minimal studies have assessed the indirect effect of heat on hyperlipidemia (HDM). According to Z Xu, S Tong, H Pan and J Cheng [48], individuals with pre-existing HDM [OR = 1.18 (95% CI: 1.07–3.19)] showed a significant increase in stroke-related admissions after adjusting for PM_{10} and NO_2. It was concluded that other environmental factors, such as low urban greenery and income levels, can exacerbate the risk.

Disease of the nervous system Four studies focused explicitly on admissions related to diseases of the nervous system, such as all nervous system disease (NSD), neurological disorders (NUD), and Alzheimer's disease (AZD). The first study applied case-series design across different regions in New South Wales between 1998 and 2006. The researchers identified that individuals having NSD as underlying health conditions were more vulnerable to hospital admissions [OR = 1.12 (95% CI: 1.04–1.14)] during 3-day extreme heat exposure (99th percentile mean temperature). The risk was higher during the first day (10%) of exposure but decreased on subsequent days (6%) [28]. A similar study (matched case-control study) in Adelaide enrolled 143 individuals diagnosed with NSD health issues. The study showed that extreme heat caused a significant increase in NUD hospitalizations among them [36]. Stratification by sex and age groups revealed that females but not males were at increased risk of hospitalizations for AZD. In contrast, males aged 65–74 had a lower risk of AZD admissions than females following heat exposure [14]. Using a retrospective cohort design, Z Xu, S Tong, J Cheng, Y Zhang, N Wang, A Hayixibayi and W Hu [49] revealed that AZD admissions increased significantly during moderate heat intensity and that the risk was very low for people living in high vegetation areas.

Infectious parasitic and cancer diseases Two studies investigated the association between extreme heat and certain infectious and parasitic diseases related to hospital admissions [28, 36]. Both studies used the same health data sources (medical records data) but applied different study designs. The first study in New South Wales applied case-only analysis and found a significant association between extreme heat neoplasm (NPSM) caused admissions [28]. The second study in Adelaide used a matched

case-control approach and found no association with cancer-disease-related hospital admissions [36].

Disease of the digestive system, heat-related illness, injuries, and poisoning We found very limited studies ($n = 1$) on digestive system diseases that comprise only 15% of cases of liver diseases (LVD) in Adelaide. The study found a significant increase (6%, $p = 0.01$) in LVD admissions [36] with exposure to extreme heat. Two studies investigated the extent of increasing hospital admissions because of heat-related illness (HRI). The first study in Sydney found increased heat-associated hospitalization among individuals with HRI and injuries [OR = 12.05 (95% CI: 8.91–16.30)] during the same day of heatwave exposure. The association [OR = 19.47 (95% CI: 11.71–32.36)] was stronger among elderly people (≥ 65 years) [8]. The second study did not evaluate the standalone HRI admissions but found that HRI and other diseases increase the duration of hospital admissions in Adelaide [38].

The only study that investigated admissions caused by injuries, poisoning, and other external consequences found statistically significant heat-associated admissions from fall-related hip fracture (FHF) among the elderly (75–84 years) men [OR = 0.98 (95% CI: 0.96–0.99)] and women [OR = 0.99 (95% CI: 0.98-1.00)] in Sydney [50].

Cause-specific ED presentations

A total of 53 studies assessed different disease-causing ED presentations in the context of extreme temperature exposures in Australia (Supplementary Tables S4-S5). The effects estimates (RR, IRR, OR) of these diseases are presented in Supplementary Figure S2.

Disease of the respiratory system caused ED presentations Out of 53 studies, 10 assessed respiratory system disease-caused ED presentations in five specific diseases: all respiratory diseases (RSD), Asthma, acute upper respiratory infections (ARI), chronic lower respiratory disease (CRI), and chronic obstructive pulmonary diseases (COPD) (Supplementary Table S4).

A cohort study involving analysis of Rural Acute Hospital Data Register (RAHDaR) between 2017 and 2020 found evidence of a 12% increase in RSD-caused ED presentation during high-heat days (at 95th percentile mean temperature) among rural populations in regional Victoria State [26]. In Adelaide city, a case-series analysis of the 2008 heatwave event found a 3% increase in RSD among all age groups, with higher (21%) among the 65–74 years category. By contrast, RSD decreased (i.e., RR < 1) in the subsequent heatwave event (2009) among the same population [31]. When considering the impact of different heatwave intensities, both low and severe heat cause increased RSD presentations in Adelaide [51].

A time-series study (2000–2008) in Brisbane evaluated the impact of socioeconomic status on RSD-related ED presentations, adjusting for PM_{10} and O_3. Despite the 8% increase in RSD-related ED cases, no association with extreme heat events was found [52]. Between 1996 and 2004, another time-series study in Brisbane applied different heatwave metrics to predict RSD-related presentations. Although RSD was not assessed among various age groups (e.g., elderly), it increased the risk of ED presentations by 14% at the 95th percentile of mean temperature across the population [53]. In a multi-city (Brisbane, Cairns, Longreach, Mackay, Mount Isa, Rockhampton, Toowoomba, Townsville) study, extreme heat was not associated with RSD-related presentations in both rural and urban settings for all age groups [54].

With regards to child health in Brisbane, evidence from two ecological studies indicated a high risk of childhood (≤ 4 years) Asthma, particularly in males [RR = 1.13 (95% CI: 1.02–1.25)] [55], and chronic CRI [RR = 1.13 (95% CI: 1.02–1.38)]] [56] because of extreme temperature.

A case-crossover study (2008–2016) examined the impact of extreme heat on overall RSD, ASM, and COPD in Tasmania. The results showed a similar risk of RSD (4%) and ASM (4%) caused ED presentations but decreased in COPD [OR = 0.97 (95% CI: 0.63–1.05)]. These associations were non-significant [57]. An ecological study based on heatwave vulnerability to RSD-related presentations was also observed in Perth between 2002 and 2009. The study reported no association between heatwave and ED presentations from RSD [58].

Disease of genitourinary system caused ED presentations Ten studies examined the risk of diseases of the genitourinary system on heat-associated ED presentations in Australia (Supplementary Table S4). Among the specific diseases investigated were ARF, RNF, URS, UTI, URD, chronic kidney disease (CKD), lower urinary tract infections (LUTI), pyelonephritis (PHS), renal diseases (RND), and all genitourinary diseases (GUD) (Supplementary Table S4-S5).

Two studies in Adelaide suggested a high vulnerability to heat-related urinary diseases ED presentations. The first ecological study (2003–2014) indicates that a 1℃ increase in temperature is associated with ARF (4%), RNF (3%), CKD (2%), URS (2%), UTIs (0.4%), and LUTIs (0.3%) [59]. Similar results were reported in the same population using a different study design; the use of the excess heat factor (EHF) metric predicted an increase in overall URD, ARF, and URS by 5%, 11%, and 42%, respectively, within lag0-10 days. Under severe heatwave intensity, ARF presentations were further exacerbated by 68% [IRR = 1.67 (95% CI: 1.35–2.07)] [60].

In the same city (Adelaide), a comparative case-series analysis between two (2008 versus 2009 heatwave) major

heatwave episodes indicates an increased risk of all RND-related presentations from the previous (2008: 11%) to the subsequent heatwave event (2009: 39%). The elderly (≥75 years) experienced the highest vulnerability (68%) during the 2009 heatwave episode when compared to children (25%) [31]. In Adelaide, there is evidence of the elderly (≥65 years) showing sensitivity to all RND-caused ED presentation at both minimum and maximum temperature levels of 24% and 30% when compared to all age groups of 13% and 11%, respectively [44]. Also, all RND heat-related presentations can be increased when combined with another disease but are non-significant when assessed as independent health outcomes [51].

The less sensitivity of children to heat-related genitourinary diseases was also supported by another study in Brisbane, where there was a decrease in children (0–14 years) including pediatrics genitourinary diseases related presentation during the two days (lag0-1) of heat exposure and the subsequent days (lag0-21) with RR of 0.91 (95% CI: 0.82–1.02), and 0.92 (95% CI: 0.63–1.35), respectively [56]. In Tasmania (2008–2016), both ARF [OR = 0.93 (95% CI: 0.35–2.52)] and RNF [OR = 0.94 (95% CI: 0.74–1.94)] presentations were decreased among all populations, including children under 5 years of age [57]. A similar effect was observed in Perth (2002–2008), where all RND-related presentations were increased by 12% during heatwave days [58]. At the State level (Queensland), two studies showed a significant increase in all genitourinary systems [RR = 1.05 (95% CI: 1.00-1.09)] [54], and more specifically acute CKD [OR = 2.59 (95% CI: 1.89–3.55)] [61] related ED Presentations at both three days and hourly temperature exposures, respectively.

Mental and behavioral disorders caused ED presentations Five studies assessed mental and behavioral disorders caused by ED Presentations by looking at MDS, organic mental disorder (ORM), and MBD.

A recent ecological study (2014–2017) suggests no significant association between MBD and heatwaves in Adelaide, though there was a rise in presentations within this period [62]. A case-series study demonstrated a consistent increase in MDS-related ED presentations during two consecutive heatwave episodes in Adelaide. The authors reported a higher risk of MDS-related presentations among the elderly (≥75 years) during the 2008 and 2009 heatwaves, at 15% and 18%, respectively, compared to the entire population, which saw increases of 5% and 4%, respectively [31]. In a time-series study (1993–2009) in the same city, heatwave-associated ED presentations from MDB were evident among the ≥65-year age groups for both minimum (24%) and maximum (29%) temperature thresholds. These risks were higher compared to the general population (10%) [44].

Similar to Perth (2002–2009), the risk of MBD presentations was 5% for mean temperature (defined as 35℃ for ≥3 days in a row) but was exacerbated above the minimum (60%) and maximum (43%) temperature thresholds [58]. A case-crossover analysis in Tasmania between 2008 and 2016 showed a decrease in both MBD [OR = 0.95 (95% CI: 0.77–1.18)], and ORM [OR = 0.84 (95% CI: 0.36–1.96)], though the association was not significant [57].

Cardiovascular system caused ED presentations Four studies identified cardiovascular system disease caused ED presentations but focused on only cardiovascular diseases (CVD), as shown in Supplementary Table S4.

The study by Tong et al. (2014) did not find any risk of heat-associated CVD presentations, whereas in their further study, heatwave affected the CVD presentations by 10% and 9% at lag0, and lag1, respectively when adjusted with socioeconomic disadvantage conditions [52].

A case-crossover study in Tasmania (2008–2018) found heatwaves to be a predictor of CVD presentations, particularly atrial fibrillation (AF) = 3%, but the association was non-significant due to a small number of cases [57]. In Perth, an ecological study conducted in 2002–2009 suggests that exposure to temperature (≥35 ℃) for ≥3 days increases the risk of heat-associated CVD presentations by 30% [58].

Endocrine, nutritional, and metabolic system-caused ED presentations Four studies investigated endocrine, nutritional, and metabolic system-caused ED presentations by considering only endocrine, nutritional, and metabolic diseases (ENM) and DBT, as indicated in Supplementary Table S4.

Two studies specifically focused on DBT patients using EHF as an exposure variable. The first study employed Tasmanian Health Service data, finding that there is a 57% non-significant increase in odds [OR = 1.57 (95% CI: 0.82–3.01)] of DPT-related ED presentations during heatwave days [57]. The second study in Adelaide also found similar evidence. There was an increase in the number of DBT-related presentations in heatwave days [12 (95% CI: -15 to 34)], without any statistically significant association [51].

For ENM-related presentation studies, the first study focused on both urban and rural areas of Queensland and concluded that heatwaves affected ENM-related presentations by 18% [RR = 1.18 (95% CI: 1.04–1.34)] among all populations [54]. The last study was conducted among children (0–14 years) living in Brisbane, finding that the risk of those affected by ENM presentations was only 1% during the first two days (lag0-1 day) of heatwave exposure but increased rapidly to 34% in lag0-13 days, and 80% in lag0-21 days [56].

Circulatory system-caused ED presentations Five studies evaluated the effects of circulatory system diseases on heat-related ED presentations by looking at five individual diseases: CSD, IHD, hypertension (HPS), atrial fibrillation (AF), and HTF (Supplementary Table S4).

A retrospective cohort study using RAHDAR data from rural Victoria and at temperature exposure levels ranging between 33.2℃ and 36.8℃ found the risk of CSD-related presentations to be 5–8% [26]. A similar regional study in Queensland did not find any associations with presentations from CSD in both urban and rural localities during heatwave days [54].

A study reported in Adelaide that the risk of IHD-related presentations was reduced [RR = 0.94 (95% CI: 0.81–1.13)] during 2008 heat episodes but was increased by 3% for that of 2009. Those who were 65–74 years (7% of the total population) and 15–64 years (39%), respectively, were the most vulnerable group during the two heatwave events [31]. A more recent study (2014–2017) showed a decrease in the number of IHD-related presentations during heatwave periods [-3 (95% CI: -61 to 53)], except severe heatwave thresholds [2 (95% CI: -35 to 28)]. However, this association was not statistically significant [51]. Evidence from Tasmania based on a case-crossover study (2008–2016) suggests a non-significant increase in risk of CSD heat-related ED presentations, particularly HPS [OR = 1.4 (95% CI: 0.58–3.38)], and AF [OR = 1.03 (95% CI: 0.66–1.60)], except for IHD [OR = 0.79 (95% CI: 0.58–1.07)], which indicated a reduced risk [57].

Nervous system caused ED presentations Three studies examined heat-related ED presentations due to nervous system disease, focusing on two specific diseases–NSD and NUD (Supplementary Table S4). Two of these studies were located in Queensland; the first one identified NSD as a driver of heat-associated presentations specifically among children [RR = 1.06 (95% CI: 1.01–1.13)] in Brisbane [56]. The second study, on the other hand, found the risk of higher NSD presentations among larger populations living in both urban and rural communities [RR = 1.09 (95% CI: 1.02–1.17)] [54]. For the third study conducted in Tasmania, there was no evidence of an association between heat-related ED presentations and NUD [57].

Digestive system caused ED presentations Three studies focused on heat-associated ED presentations of digestive system diseases, primarily assessing digestive system diseases (DSD) and diarrhea (DRH) as the main health outcomes. Most of the studies were focused on children's (0–14 years) health in Brisbane. The first study found heat-associated DSD presentations during the first two (lag0-1) days [RR = 1.07 (95% CI: 0.98–1.17)] of heat exposure, but was reduced slightly in lag0-13 days [RR = 0.99 (95% CI: 0.84–1.18)], with lesser sensitivity observed among younger children (0–4 years) [56].

Evidence from a more detailed study conducted among the same population shows that children who are 1–2 years of age were affected (17%) by bacteria diarrhea (DRH) [RR = 1.17 (95% CI: 1.0- 1.25)] related presentations, and 10% by viral DRH [RR = 1.10 (95% CI: 1.06–1.13)] among all the children within 0–14 years at $p < 0.05$ [63]. In the case of regional Victoria, a retrospective cohort-based study within rural farming communities found a decrease (-6%) in DSD-related presentation during heatwave days compared to non-heatwave days (Adams et al. 2022).

Infectious and parasitic diseases caused ED presentations Two studies that employed ecological design investigated infectious and parasitic diseases (IPD) and intestinal infectious disease-related (IFD) ED presentations in Queensland communities and Brisbane City, respectively (Supplementary Table S4). The first, which evaluated IPD presentations in Queensland (2013–2015), predicted that the risk was higher in rural communities than urban areas [54]. The second study in Brisbane (2003–2009) revealed that increased IFD presentations was a critical health concern in cities. IFD presentation increased by 6% among children (0–14 years) during lag0-1, and by 18% during lag0-13 days, but these increases were not significant [56].

Skin, ear, Musculoskeletal, and heat-related illness caused ED presentations One study examined the effect of three different outcomes, including skin and subcutaneous tissue, all musculoskeletal system and connective tissue, and diseases of the ear and mastoid process on heat-related ED presentation. The study was conducted in Queensland State using ecological design and Queensland health data from 2013 to 2015. The authors found a positive association between these three outcomes (i.e., skin, ear, musculoskeletal), and increased ED presentations in urban areas than in rural settings [54]. In Victoria State, a cohort-based study in rural areas shows substantial reductions (-40%) in ED presentations among patients diagnosed with all musculoskeletal systems and connective tissue during heatwave days [26].

A study examined the relationship between direct heat-related illness and the associated presentations in Adelaide (HRI) through an ecological study design. The authors observed that the HRI patients had many ED presentations during heatwave days [389 (95% CI: 342–427)]. The number of presentations was still higher under both severe [220 (95% CI: 192–241)] and low [198 (95% CI: 164–228)] heatwave intensity thresholds [51].

Cause-specific ambulance callout

Fifteen studies investigated cause-specific (respiratory, cardiovascular, and nervous system diseases) related ambulance callouts in Australia due to extreme temperature exposure (Supplementary Table S4-S5). The effect measures (as OR, RR, and IRR) of the individual cause-specific outcomes are presented in Supplementary Figure S2.

Respiratory system disease ambulance callout Five studies assessed respiratory system diseases but focused on only all respiratory diseases (RSD). Two of these studies were conducted in Adelaide; both applied case-series study design and investigated whether RSD increases ambulance dispatches. The first study, which used South Australian Ambulance Service Data (1993–2006), reported a non-significant increase in RSD among children (5–14 years) but a reduction among the elderly (≥ 75 years) patients [RR = 0.93 (95% CI: 0.86–1.02)] [32]. The second used the same data source (1993–2009) but focused on two different heatwave episodes: 2008 and 2009 heatwave events. The study reported an overall increase in RSD presentations in 2008 [RR = 1.06 (95% CI: 0.94–1.19)] compared to 2009 [RR = 0.88 (95% CI: 0.77–1.01)]. A higher risk of 32% and 16%, respectively, was observed among the 15–64 years groups, and the risk was reduced (RR < 1) among 75-year patients [31].

Two other studies were conducted in Brisbane using the same 7-year (200–2007) Queensland Ambulance data and the same study design (time series) but different heat definitions. The first study using the daily mean temperature metric found a reduction in RDS-caused dispatches [RR= -1.99 (95% CI: -3.72 to -0.25)] at lag2-15 days [64]. The second study found that maximum temperature exceeding 37℃ increased RDS dispatches by 101.1% (95% CI: 34–202) for all ages, particularly among the 75 + years age groups [163 (95% CI: 33–420)] [65].

The Tasmania study (2008–2019), which assessed the effect of RSD on dispatches during heatwave days based on a case-crossover design, reported a non-significant risk of 5% and 7% during low and severe intensity heatwave days, respectively [66].

Cardiovascular system disease ambulance callout Five studies examined heat-associated ambulance dispatches from cardiovascular system diseases, with three studies focusing on all cardiovascular disease (CVD), while two were on cardiac arrest (CDA). Two time-series studies in Brisbane, all conducted in 2000–2007, found a decrease in CVD dispatches [RR= -1.85 (95% CI: -3.06 to -0.64)] for mean temperature exposure [64], whereas, for a maximum temperature of > 37℃, the risk increased by 29.5% for all ages, and 164% for those within 64–74 years [65]. Although there were null associations in Tasmania (2008–2019), CVD dispatches increased by 5% and 42% during low and extreme heatwave periods, respectively [66].

Two case-series studies in Adelaide have produced contrasting results. The first study found non-statistically significant heat-associated callouts from CDA among 0–4 years children [IRR = 1.21 (95% CI: 0.85–1.74)], but it was reduced among 75 years people [IRR = 0.93 (95% CI: 0.87–0.98)] [32]. The subsequent study found a statistically significant association between CDA-related dispatches during 2008 [RR = 1.10 (95% CI: 1.01–1.2)] and 2009 [RR = 1.13 (95% CI: 1.03–1.23)] heatwave episodes [31].

Nervous system disease ambulance callout Two studies, both case series conducted in Adelaide, have examined the association between nervous system disease and heat-related ambulance callouts by looking specifically at neurological disorders (NUD).

The first study, which focused on the effect of mean daily temperature exposures, did not find any association [RR = 1.00 (95% CI: 0.95–1.04), $p > 0.05$] with NUD dispatches among all populations, but reported a 15% increase among children [RR = 1.15 (95% CI: 0.94–1.41)] [32]. The subsequent study assessed two heatwave episodes: 2008 and 2009 heatwave events. The risk of NUD for heat-related callouts was reported to be 8% and 2%, respectively, among all the patients. The 75 + years (11%) and 65–74 years (35%) patients, respectively, were the most vulnerable groups [31].

Endocrine, genitourinary, and heat-related illness ambulance callout Three different cause-specific diseases, including diabetes (DBT), renal diseases (RND), and Heat-related illness (HRI), were examined to understand their association with heat-associated ambulance dispatches in Tasmania using a case-crossover design. The findings suggest a non-significant increase in the risk of heat-associated dispatch from DBT during low (9%), severe (26%), and extreme (18%) heatwave periods. The risk of RND was found to be 8% and 20% for low and extreme heatwave days, respectively. HRI, on the other hand, increased the risk of ambulance callouts by 9% during low intensity and 43% in severe heatwave periods [66].

Cause-specific mortality

Thirty-three (33) of the studies that satisfied the inclusion criteria focused on cause-specific mortality due to extreme temperature exposure using mixed methods, either ecological, case-crossover, time-series, or case-series (Supplementary Tables S4-S5). The effect size of the various cause-specific health outcomes is shown in Supplementary Figure S2.

Respiratory system disease-related deaths Five studies investigated heat-associated deaths from respiratory system diseases; the studies focused on only respiratory diseases (RSD).

S Tong, XY Wang, G FitzGerald, D McRae, G Neville, V Tippett, P Aitken and K Verrall [53] calculated the risk of RSD heat-associated deaths to be 42% [RR = 1.42 (95% CI: 1.13–1.79), $p < 0.05$] using time-series analysis from 1996 to 2004 in Brisbane at ≥ 95th percentile temperature. At higher exposure (≥ 99th percentile), the risk increases to 70%. When a case-crossover design was applied, there was a non-significant increase in RSD deaths [OR = 1.47 (95% CI: 0.78–2.75)] at ≥ 37℃ maximum temperature after adjusting for air pollutants (PM_{10}, NO_2, O_3) [34]. A recent suburb-level study (2010–2019) in Queensland State showed a statistically significant increase in RSD deaths by 7% across all ages [RR = 1.07 (95% CI: 1.02–1.12)] during heatwave days [6].

In contrast, a case-series study conducted in Adelaide reported that at ≥ 35℃ maximum temperature over ≥ 3 days in a row, there was a decrease in risk of RSD-related mortality [IRR = 0.84 (95% CI: 0.72–0.99)] among older people ≥ 75 years. The risk did not increase across the entire population [IRR = 0.92 (95% CI: 0.80–1.05)] but affected 15- 64-year-old people during the heatwave days [32]. A study in Sydney found that both single-day [OR = 1.14 (95% CI: 1.04–1.24)] and 3-day [OR = 1.08 (95% CI: 0.99–1.18)] heatwave periods (95th percentile maximum temperature) were associated with RSD related deaths. Adult (65 + years) mortality risk increased by 13% and 7%, respectively for the two heatwave exposure periods [8].

Mental and behavioral disorders-related deaths Five studies examined whether extreme heat days are predictive of mental disorders (MDS) related deaths in Australia. The specific disorders investigated were mental and behavioral disorders (MBD), dementia (DEM), cognitive and behavioral disorders due to psychoactive substance use (MBPS), and schizophrenia, schizotypal, and delusional disorders (SSDS).

Two studies were conducted in Adelaide. The first study using case-series design (1993–2004) during heatwave days (≥ 35℃ for ≥ 3 days in a row) showed a non-significant increase in the risk of MDS-related deaths [IRR = 1.04 (95% CI: 0.73–1.47)] Individuals between 15 and 64 years old (24%) were the most vulnerable group [32]. The second study using an ecological design revealed that daily temperature thresholds > 26.7℃ increased the risk of heat-associated deaths from MBD [IRR = 2.39 (95% CI: 1.16–4.92), $p = 0.01$] among the elderly (65–74 years) people. A similar risk was observed for DEM [IRR = 5.06 (95% CI: 1.21–21.23), and SSDS [IRR = 2.08 (95% CI: 1.05–4.14) heat-associated deaths [14].

Two studies, both employing case-crossover design, were conducted in Queensland and reported different findings. The first study (1996–2004) found a non-significant decrease in MDS heat-associated deaths among all the patients [OR = 0.8 (95% CI: 0.21–2.98), except for those aged 75 years and older, where it increased by 5% [34]. The second study (2010–2019) found a significant 9% increase in heat-associated MDS deaths among all patients regardless of age. The risk was exacerbated to 18% during extreme heatwave conditions [6]. The former study accounted for confounders such as air pollution, seasonality, and public holidays, while the latter did not.

In Sydney, a case-crossover study (1997–2007) suggests that both single-day [OR = 1.10 (95% CI: 0.95–1.27), $p > 0.05$] and a 3-day [OR = 1.04 (95% CI: 0.90–1.21)] heatwave periods increased the risk of MDS related deaths. The risk was increased by 7% among the elderly (65 + years) for a single day of heatwave exposure [8].

Endocrine, nutritional, and metabolic systems-related deaths Five studies targeted heat-associated deaths from diseases of the endocrine, nutritional, and metabolic systems by looking specifically at 3 different causes: DBT, HDM, and endocrine and metabolic diseases (EMD) caused deaths due to heatwave.

For a study in Brisbane that used case-crossover design (1996–2004) for a daily maximum temperature of ≥ 37℃ (for ≥ two consecutive days), the authors reported a statistically significant increase in heat-associated deaths from DBT [OR = 9.96 (95% CI: 1.02–96.85) among the older patients (≥ 75 years old) [34]. Another retrospective cohort study (2005–2013) involving 413 patients in Brisbane found evidence of an increase in DBT post-discharge deaths among patients during low (46%, at lag0), medium (64%, at lag0) and high (137%, at lag1) heatwave intensities [47]. In the same city, there is a lack of statistical association between extreme heat and patients with underlying HDM diseases [48]. Similarly, another case-crossover study (2010–2019) in Queensland found no statistical association between extreme heat and EMD-related deaths [6].

In Sydney, a case-crossover study (1997–2007) showed that single-day heatwave events were associated with DBT-related deaths [OR = 1.22 (95% CI: 1.03–1.46)], particularly among the older (> 65 years) patients [OR = 1.26 (95% CI: 1.05–1.51)] [8].

Genitourinary systems-related deaths Four studies evaluated the impact of diseases of the genitourinary system on heat-associated deaths. They investigated RND, RNF, and GUD-related deaths.

A study (1993–2004) applied case-series analysis to understand the impact of extreme temperature on RNF-related deaths in Adelaide. It was reported that exposure to the daily mean temperature of ≥35℃ (in ≥3 consecutive days) did not increase [IRR = 0.84 (95% CI: 0.59–1.19)] RNF-related deaths [32]. For another study (1996–2004) in Brisbane that applied case-crossover design, heatwave exposure increased RND-related deaths by 19%, but the association was non-significant [34]. Similar findings were observed in Sydney, where a study (1997–2007) reported an 18% (not statistically significant) increase in RNF-caused deaths at 95th percentile mean temperature exposure levels [8]. Also, a case-crossover study (2010–2019) did not find any association between GUD-caused deaths and heatwave days in Queensland. Even under extreme, and severe heatwave days, no association was found with GUD-related deaths [6].

Circulatory systems-related deaths Four studies assessed heat-associated deaths from the circulatory system; the three specific diseases investigated were IHD, stroke, and CRD). Two studies examined stroke and IHD deaths during heatwave days.

The first study (1993–2004) in Adelaide, through case-series design, found a statistically significant decrease in stroke-related deaths across all age patients [IRR = 0.84 (95% CI: 0.74–0.96)], with a similar trend observed among those aged ≥75 years [IRR = 0.86 (95% CI: 0.74–0.99)] during heatwave days [32]. The second study (1996–2004), on the other hand, applied a case-crossover design and reported a statistically non-significant association between heatwave and IHD-related deaths [OR = 1.84 (95% CI: 0.31–11.16)] in Brisbane [34].

In Sydney, LA Wilson, GG Morgan, IC Hanigan, FH Johnston, H Abu-Rayya, R Broome, C Gaskin and B Jalaludin [8] found that those affected by heatwave had 7% [OR = 1.07 (95% CI: 1.01–1.12)] and 12% [OR = 1.12 (95% CI: 1.06–1.18)] increase odds of IHD deaths during a single day, and a 3-day heatwave periods, respectively. In contrast, a study (2010–2019) in Queensland, using a case-crossover design, did not find any association between heatwave days and CRD-related deaths [6].

Cardiovascular systems-related deaths Three studies focused on heat-associated deaths from the cardiovascular system, and the specific disease investigated was all cardiovascular diseases (CVD).

The first study (1997–2007) in Sydney revealed that both a single-day [OR = 1.06 (95% CI: 1.02–1.12)] and a 3-day [OR = 1.11 (95% CI: 1.06–1.17)] heatwave increases the risk of death from CVD ($p < 0.05$) by 6% and 11%, respectively across all age groups, the similar magnitude of risk was also found among ≥65 years patients [8]. The second study (1996–2004) in Brisbane found a statistically significant increase in heat-associated deaths from CVD ([OR = 1.89 (95% CI: 1.44–2.48)]. The authors highlighted that heat-related death was still evident despite good acclimatization of the population to heatwaves [34]. The third study was also conducted in Brisbane but applied a time-series design and reported a statistically significant association between heatwave (≥95th percentile mean temperature) and an increase in CVD-caused deaths [RR = 1.37 (95% CI: 1.24–1.51)] among all the age groups [53].

Nervous systems-related deaths Two studies assessed heat-related deaths from the disease of the nervous system, with a focus on NSD and AZD.

Xu et al. (2019c) examined heat-associated deaths from AZD under different heatwave intensity thresholds in Brisbane. The authors reported that medium heatwave intensity (95th percentile for ≥2 consecutive days) thresholds showed a statistically significant increase in heat-related deaths from AZD by 269% (95% CI: 76-665%) [49]. Similarly, In Queensland (2010–2019), the use of matched case-crossover analysis demonstrated a 10% increase in NSD-related deaths during heatwave days [6].

Malformation, infectious, skin, musculoskeletal, and perinatal deaths The effect of the heatwave on different health outcomes was investigated in Queensland using match case-crossover analysis of cause-specific mortality data from the Australian Bureau of Statistics between 2010 and 2019 [6].

The study did not find any association between heatwave days and an increase in deaths from congenital diseases (CND) and infectious and parasitic diseases (IPD). The study also found that exposure to heat waves was not associated with skin diseases (SKD) but all musculoskeletal diseases (MSK) and perinatal diseases (PRD) related deaths [6]. In contrast, there was evidence of an increased risk of heatwave-associated deaths from neoplasm (NPSM) [RR = 1.05 (95% CI: 1.03–1.08)]. Similarly, NPSM deaths also increased by 12% under extreme heat ([RR = 1.12 (95% CI: 1.08–1.17)] intensity thresholds [6].

Heat vulnerability assessment

Eight studies assessed Australia's heat vulnerability index (HVI) in the context of exposure, sensitivity, and adaptive capacity components as shown in Supplementary Table S6. All the studies constructed HVI based on heat exposure (e.g., temperature, impervious surface), sensitivity (e.g., age, income), and adaptive capacity (e.g., home ownership, emergency services) variables for each component. No study has validated the HVI with health

outcome data, either morbidity or mortality data (Supplementary Table S6).

From spatial scale standpoint, majority (87%) of the studies constructed HVI at city scale [67–73], only one study was focused at State level [74], while none of the eight studies assessed heat vulnerability from national scale.

W Zhang, P McManus and E Duncan [67] characterized spatial variability of HVI in Sydney at statistical area level 2 (SA2) geographical unit by using one satellite-based heat exposure data (land surface temperature), nine sensitivity variables (infants, elderly, ethnicity, low education, language barriers, new arrivals, low-income, disability, and isolated people), and four adaptive capacity variables (traffic convenience, internet access, proximity to vegetation, and proximity to water bodies). HVI was constructed through a novel raster-based subdividing indicators (RSI) approach. The authors did provide the importance of using RIS for HVI mapping, considering the increasing growth of the use of spatial data for urban designs in recent years.

A El-Zein and FN Tonmoy [68] developed a similar HVI for Sydney in the local government area (LGA), which is bigger than SA2. The researchers used six heat exposure variables. Three of them (maximum temperature, minimum temperature, days with temperature > 30℃) were direct heat variables, while the other three (impervious surface, population density, road density) were indirect heat variables. The study then developed HVI by including four sensitivity variables (elderly [≥ 65 years], elderly [≥ 65 years and living alone], children [≤ 4 years], [multi-unit housing dwellers]), and 12 adaptive capacity variables (people finishing year 12 education, people who speak other languages different from English, median home loans repayment, home ownership, internet access, median household income, ratio of assets to liabilities, per capita business rates to local council, per capita community service expenses to local council, per capita expenses on environment and health, people that needs financial support). Correlation and multi-criteria ranking were the main data analytical approaches for constructing the HVI.

M Loughnan, N Nicholls and NJ Tapper [70] developed HVI for Melbourne by situating the study to a finer geographical scale known as postal area (POA), which is smaller than LGAs and SA2s. Four sensitivity variables (single-person households, age [elderly of ≥ 65 years and children of < 0–4 years], disability, the burden of chronic diseases) and four adaptive capacity variables (age care facility and nursing homes, ethnicity, socioeconomic status derived from socioeconomic index for areas [SEIFA], and density of non-single dwellings) were used to develop the HVI. Mixed and simple methodological approaches, including correlation, linear regression, and an unweighted approach, were applied to develop the HVI.

Q Sun, J Hurley, M Amati, A Saunders, J Arundel, B Boruff and P Caccetta [72] focused on the HVI mapping in Melbourne but at statistical area level 1 (SA1), which is a much smaller geography than SA2 and LGAs but bigger than POAs. The authors employed one exposure variable (land surface temperature) and six sensitivity variables, of which two were environmental based (number of roads, vegetation cover), and four were sociodemographic (population density, elderly of ≥ 65 years, children of ≤ 4 years, persons that need care support). The researchers developed HVI by combining the heat exposure data with two adaptive capacity variables in the form of indices such as the index of relative socioeconomic disadvantage [IRSD] and the index of education and occupation [IEO]); these indices were developed from a wide range of socioeconomic variables (e.g., income level, housing, occupational status, education, housing). Correlation, linear regression, and unweighted were the most common approaches used to construct the HVI.

Three studies [69, 71, 73] mapped HVI at the POA level for the same eight capital cities (Canberra, Melbourne, Brisbane, Sydney, Hobart, Perth, Darwin, and Adelaide) of Australia. The first two studies both used two exposure variables (land surface temperature, population density), three sensitivity variables (single-person households, age [elderly with ˃ 65 years, and children of 0–4 years], disability), and six adaptive capacity variables (age care facilities, ethnicity, non-single dwellings, vegetation cover, access emergency services, socioeconomic status) to construct the HVI but with different methodological approaches. M Loughnan, N Tapper and T Loughnan [41] used correlation analysis and linear regression. M Loughnan, N Tapper, T Phan, K Lynch and J McInnes [71] also used the same methods but improved the estimates by including principal component analysis (PCA). S Wang, QC Sun, X Huang, Y Tao, C Dong, S Das and Y Liu [73] similarly applied PCA analysis using two exposure variables (land surface temperature, population density) and 17 demographic, socioeconomic, and health variables without any of the variables linked to adaptive capacity (Supplementary Table S6).

Discussion

This scoping review has noted six major findings. (i) We observed compelling evidence of increased risk of heat-associated deaths for those with mental and behavioral disorders, as well as for those with history of cardiovascular disease. Moderate evidence was found to support an increased risk of heat-associated deaths for those diagnosed with respiratory disease or diabetes. Studies on heat and renal or genitourinary diseases are limited, (ii) A large number of studies reported an increase in

heat-associated hospital admissions for those with renal, diabetes, neurological, stroke, mental disorders, and ischemic heart disease, (iii) Individuals with renal, neurological, cardiovascular, or respiratory diseases did not exhibit strong associations with heat-related ambulance callout, (iv) Elderly individuals (65+) were at higher risk of heat-related deaths and morbidity, (v) Regarding HVI, studies were only focused on Australian cities, with heat vulnerability predicted based on socioeconomic and health data from single-year census estimates, and (vi) Studies investigating the longitudinal effect of heat vulnerability is sparse, as is the case for Australian-based HVI studies that incorporate mortality and morbidity data.

Across the heat-health epidemiological studies that fulfilled our inclusion criteria, we identified 85 different cause-specific diseases, presenting concurrently with heat-events, for hospital admissions, ED presentations, ambulance callouts, and mortality. Importantly, these diseases are non-exhaustive compared to the total number of diseases (1,529) that cause death in Australia [75]. These epidemiological studies differ in outcomes due to the heterogeneity of heat exposure metrics used (e.g., EHF, mean temperature, maximum temperature) are limited to specific study designs (mainly ecological), and are heavily focused on cities such as Brisbane and Adelaide.

Studies that employ ecological design usually assess health outcomes at the population level, by aggregating health data, rather than at the individual level. Although such a study design may help in understanding the broader public health trends and patterns in health outcomes [76], this design lacks specificity and is prone to ecological fallacy, where the association observed at the population level does not necessarily hold at the individual level. Future studies might consider complementing ecological designs with individual-level analyses using longitudinal data. This will enable a more nuanced understanding of potential risk factors for increased heat-related mortality and morbidity.

Studies using case-crossover design were mainly focused on a few cities in Australia making it difficult to compare risk in non-urban areas that have high socioeconomic disadvantaged groups [77]. For example, in the Northern Territory of Australia, there is an increasing risk of heat-associated deaths despite improvements in infrastructural technologies (i.e., adaptive capacity), suggesting high population sensitivity to extreme heat [77]. Evaluating the impact of heat on a wider geographical unit, such as the inner (remote) and outer regional areas where most Indigenous people live, will improve the understanding of heat-related risk to inform evidence-based adaptation strategies in the future.

Assessing heat-related deaths and morbidity in smaller geographical units such as SA2s/SA1s is important because finer spatial-scaled analysis may offer more localized insights into patterns and trends to tailor more effective heatwave adaptation programs. However, it is important to note that the population in these spatial units is much smaller. Therefore, such data must be treated with caution due to ethical concerns related to the potential identifiability of individuals. Nevertheless, with this high-resolution data, communities in these areas can more easily interact and participate in local heatwave intervention programs [78], with tailored plans based on homogenous characteristics. In the United States, for example, neighborhoods with a high risk of heat-related deaths [79] have been able to develop heat intervention programs, such as tree planting/shading and adaptation measures (e.g., building connections with culture/ancestors) to help build resilience to extreme heat [80]. This emphasizes the importance of assessing heat-related outcomes in smaller areas (neighborhoods) to help design efficient intervention programs.

Most of the studies reviewed have benefited from using highly accurate observational temperature data, thanks to the availability of networks of weather stations across cities in Australia [81]. This accessibility to temperature datasets could make it easy for researchers to adopt a common method of heat exposure, such as excess heat factor (EHF), thereby improving accuracy and reducing discrepancies in heat-health study outcomes. Evidence from international studies (e.g., in Europe) has shown that EHF is the most impactful heatwave metric used in predicting heat-related health outcomes [82–84]. The use of modeled temperature data for heat studies is emerging in Australia [66, 85]; while this data can help predict future heat events, these models tend to underestimate and overestimate minimum and maximum temperatures [86].

The recent Australian National Health and Climate Strategy (NHCS) has recognized extreme heat as an important hazard of national concern and encouraged the urgent need to build a climate-resilient public health system, through climate science research, to help protect the health and well-being of Australians [21]. However, the health risk assessment (e.g., HVI map) part of the strategy, particularly at the national scale, is lacking [21, 72, 73]. Our scoping review found that all eight HVI studies focused only on the major cities, making it challenging to know the extent of heat vulnerability across the non-urban parts of Australia, especially the remote and regional areas. Therefore, developing national HVI mapping that includes these different geographical units will provide a better understanding of the heat vulnerability in Australia.

Also, the HVI studies in Australia, like other studies in the literature [87, 88], developed HVIs by utilizing sociodemographic and health data from single-year census data (e.g., 2016 census) without comparing it to data

from remaining consecutive census years to understand how HVI changes over time within the same geographical unit (e.g., SA2). This warrants longitudinal heat vulnerability assessment in Australia and evaluation of the reliability of HVI in predicting health outcomes because it is difficult for policymakers to rely on a single-year HVI mapping for long-term policy applications.

Studies suggest that the risk of heat-associated death and morbidity depends on the intensity of the heatwave [6, 89]. However, current HVI studies failed to assess vulnerability according to heatwave intensity thresholds (i.e., low, severe, and extreme) [24]. This is important because specific sensitive populations (e.g., those with comorbidities) can still be affected by heat-related deaths even when exposed to low heat intensities. Also, CE Reid, K Mann Jennifer, R Alfasso, B English Paul, C King Galatea, A Lincoln Rebecca, G Margolis Helene, J Rubado Dan, E Sabato Joseph, L West Nancy, et al. [90] have concluded that HVI was associated with respiratory and renal diseases hospital admissions, but it remains unclear which heatwave intensity influenced the association. Thus, HVI should be able to indicate the risk of deaths and morbidity (e.g., hospitalizations) based on the population's susceptibility to heatwave intensity levels.

Heatwave intensity and its impact on death are expected to increase in the future because of climate change [91]. Developing HVI based on future temperature changes is necessary to accurately forecast communities that may be vulnerable to heatwaves in the coming decades. However, the current HVI studies have not been projected under different climate change scenarios. HVI mappings must be projected according to varying levels of greenhouse gas emissions representative concentration pathways (RCP), such as RCP 2.6 (low emissions), RCP 4.5 (medium emissions), and RCP 8.5 (high emissions). These HVI projections will help design long-term heatwave adaptation strategies among susceptible Australian communities. It will also help evaluate if Australia's NHCS reduces heatwave-related health outcomes.

Summary of gaps and future perspectives

- Most epidemiological studies are skewed to larger spatial units such as cities, leaving smaller areas understudied. Future research must focus on these smaller areas because it is easy to develop effective intervention programs (e.g., heat campaigns) compared to the larger cities [92]. However, it should be noted that specific study designs (e.g., case time series) will be assumed ecological since small areas depend on aggregated data. For smaller data sizes, non-significant results with wide confidence intervals are likely to occur compared to the regional studies.
- The strong evidence of increasing risk of mental/behavioral disorders, cardiovascular deaths from heat, and hospitalizations from cardiometabolic (diabetes, stroke, and heart disease), neurological, and renal diseases suggest that in Australia, these heat-related health outcomes should be used as indicators for HVI development. In addition, HVI developed in Australia should evaluate the performance or the reliability of HVI with these health outcomes because of their stronger association with heat.
- For long-term climate-health policies, it is challenging to assess vulnerability by relying on socioeconomic and health indicators derived from a single census year (e.g., 2006 census estimates) without considering whether heat vulnerability will change for the next consecutive years. Future research should assess longitudinal heat vulnerability levels across Australia by employing data from multiple census years and evaluate the performance of the HVI with heat-associated mortality and morbidity data.
- The socioeconomic (e.g., poverty) and climatic factors (e.g., heatwave intensity types) driving vulnerability could also be examined to help develop effective targeted adaptation programs.
- Although all eight HVI studies suggested increased vulnerability in Australia, particularly within the capital cities, including rural areas, which may be more vulnerable to heat-related deaths and morbidity, future studies will provide a realistic overall vulnerability situation in Australia [93].
- The HVI developed by previous studies may be useful in identifying highly vulnerable areas for mitigation. To date, it is still unknown whether the HVI maps could be integrated into the Australian national health and climate strategy. To address this gap, future research should explore qualitative interviews among climate-health practitioners across relevant stakeholders (e.g., State health departments) to discuss the effectiveness of integrating HVI maps into heat action plans.
- Considering the long-term impact of climate change in Australia, HVI mappings must be projected according to different climate change scenarios to help forecast the effects of future climate-induced heatwaves.

Conclusion

Extreme heat-related health risks have become an important public health issue in Australia because of the warming climate. However, the previous systematic reviews lack a focus on all health outcomes and often fail to account for the human vulnerability assessment aspect

of heat exposure. In this systematic review, we compiled and analyzed the trends in heat-health studies in Australia by considering mortality and morbidity from different disease types, geographical scales, and spatial HVI assessment (including exposure, sensitivity, and adaptive capacity indicators). We found adequate evidence to indicate an increasing risk of heat-related deaths for those with history of mental/behavioral disorders or cardiovascular disease during extreme temperatures. Extreme heat triggers a high risk of hospitalizations, particularly for individuals with renal disease, neurological disease, stroke, ischemic heart disease, mental disorders, and diabetes. This suggests that these are heat-health outcomes that should be employed for HVI development and validation in Australia.

We found that the current heat vulnerability index assessment research was limited to only Australian cities, leaving non-urban/remote areas understudied and without adequate understaning of how heat vulnerability changes over time. We also identify demand for fine-scaled spatial heat-health studies for the development of tailored intervention at a small area scale. Future studies can construct nationwide longitudinal heat vulnerability and evaluate the performance of the HVI with mortality and morbidity data. In addition, the projection of HVI under different climate change scenarios based on greenhouse gas emission levels can help design adaptation strategies.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12889-025-21677-9.

Supplementary Material 1

Acknowledgements
We acknowledge the University of Queensland (UQ)'s Graduate School for providing PhD scholarships to PA. Thanks to Max Tyler for providing the English language edits.

Author contributions
PA: conceptualization, screening, methodology, data extraction, visualization, write-up, review, editing, ZX: supervision, review, editing, CCO: review, editing, NS: screening, data extraction, visualization, NO: supervision, methodology, review, editing, DP: conceptualization, methodology, supervision, review, editing.

Funding
Not applicable.

Data availability
All the data supporting the findings of this study are available within the paper, and its supplementary materials.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent of publication
Not applicable.

Competing interests
The authors declare no competing interests.

Received: 15 July 2024 / Accepted: 29 January 2025
Published online: 07 February 2025

References

1. Perkins-Kirkpatrick SE, Lewis SC. Increasing trends in regional heatwaves. Nat Commun. 2020;11(1):3357.
2. Thompson V, Mitchell D, Hegerl GC, Collins M, Leach NJ, Slingo JM. The most at-risk regions in the world for high-impact heatwaves. Nat Commun. 2023;14(1):2152.
3. Reddy PJ, Perkins-Kirkpatrick SE, Sharples JJ. Intensifying Australian heatwave trends and their sensitivity to Observational Data. Earths Future. 2021;9(4):e2020EF001924.
4. Eggeling J, Gao C, An D, Cruz-Cano R, He H, Zhang L, Wang Y-C, Sapkota A. Spatiotemporal link between El Niño Southern Oscillation (ENSO), extreme heat, and thermal stress in the Asia–Pacific region. Sci Rep. 2024;14(1):7448.
5. Talukder MR, Islam MT, Mathew S, Perry C, Phung D, Rutherford S, Cass A. The effect of ambient temperatures on hospital admissions for kidney diseases in Central Australia. Environ Res. 2024;259:119502.
6. Franklin RC, Mason HM, King JC, Peden AE, Nairn J, Miller L, Watt K, FitzGerald G. Heatwaves and mortality in Queensland 2010–2019: implications for a homogenous state-wide approach. Int J Biometeorol 2023.
7. Doan TN, Wilson D, Rashford S, Bosley E. Ambient temperatures, heatwaves and out-of-hospital cardiac arrest in Brisbane, Australia. Occup Environ Med. 2021;78(5):349.
8. Wilson LA, Morgan GG, Hanigan IC, Johnston FH, Abu-Rayya H, Broome R, Gaskin C, Jalaludin B. The impact of heat on mortality and morbidity in the Greater Metropolitan Sydney Region: a case crossover analysis. Environ Health. 2013;12:98.
9. Keatinge WR, Coleshaw SRK, Easton JC, Cotter F, Mattock MB, Chelliah R. Increased platelet and red cell counts, blood viscosity, and plasma cholesterol levels during heat stress, and mortality from coronary and cerebral thrombosis. Am J Med. 1986;81(5):795–800.
10. Chaseling GK, Iglesies-Grau J, Juneau M, Nigam A, Kaiser D, Gagnon D. Extreme Heat and Cardiovascular Health: what a Cardiovascular Health Professional should know. Can J Cardiol. 2021;37(11):1828–36.
11. Andersen ZJ, Vicedo-Cabrera AM, Hoffmann B, Melén E. Climate change and respiratory disease: clinical guidance for healthcare professionals. Breathe. 2023;19(2):220222.
12. McKenna ZJ, Atkins WC, Foster J, Belval LN, Watso JC, Jarrard CP, Crandall CG. Kidney function biomarkers during Extreme Heat exposure in Young and older adults. JAMA. 2024;332(4):333–5.
13. Xu Z, Hu X, Tong S, Cheng J. Heat and risk of acute kidney injury: an hourly-level case-crossover study in queensland, Australia. Environ Res. 2020;182:109058.
14. Hansen A, Bi P, Nitschke M, Ryan P, Pisaniello D, Tucker G. The Effect of Heat Waves on Mental Health in a temperate Australian City. Environ Health Perspect. 2008;116(10):1369–75.
15. Lu P, Xia G, Zhao Q, Xu R, Li S, Guo Y. Temporal trends of the association between ambient temperature and hospitalisations for cardiovascular diseases in Queensland, Australia from 1995 to 2016: a time-stratified case-crossover study. PLoS Med. 2020;17(7):e1003176.
16. Li S, Chen G, Jaakkola JJK, Williams G, Guo Y. Temporal change in the impacts of ambient temperature on preterm birth and stillbirth: Brisbane, 1994–2013. Sci Total Environ. 2018;634:579–85.
17. Coates L, van Leeuwen J, Browning S, Gissing A, Bratchell J, Avci A. Heatwave fatalities in Australia, 2001–2018: an analysis of coronial records. Int J Disaster Risk Reduct. 2022;67:102671.
18. Department of Human Services. January 2009 heatwave in Victoria: an assessment of health impacts. Victorian Government Department of Human Services; 2009.
19. 38% of Australians. face high risk from extreme heat [https://kpmg.com/au/en/home/media/press-releases/2024/01/38--of-australians-face-high-risk-from-extreme-heat.html#:~:text=Economic cost%3A Rising%20temperatures%20are,due%20to%20heatwaves%20by%202061.].

20. Compound costs. How Climate Change is Damaging Australia's Economy [https://www.climatecouncil.org.au/wp-content/uploads/2019/05/costs-of-climate-change-report-v2.pdf]
21. National Health and Climate Strategy. [https://www.health.gov.au/our-work/national-health-and-climate-strategy]
22. Mason H, King C, Peden JE, Franklin AC. Systematic review of the impact of heatwaves on health service demand in Australia. BMC Health Serv Res. 2022;22(1):960.
23. Morgan RL, Whaley P, Thayer KA, Schünemann HJ. Identifying the PECO: a framework for formulating good questions to explore the association of environmental and other exposures with health outcomes. Environ Int. 2018;121:1027–31.
24. Nairn JR, Fawcett RJB. The excess heat factor: a Metric for Heatwave Intensity and its use in classifying heatwave severity. Int J Environ Res Public Health 2015, 12(1).
25. Tricco AC, Lillie E, Zarin W, O'Brien KK, Colquhoun H, Levac D, Moher D, Peters MDJ, Horsley T, Weeks L, et al. PRISMA Extension for scoping reviews (PRISMA-ScR): Checklist and Explanation. Ann Intern Med. 2018;169(7):467–73.
26. Adams J, Brumby S, Kloot K, Baker T, Mohebbi M. High-heat days and presentations to Emergency Departments in Regional Victoria, Australia. Int J Environ Res Public Health 2022, 19(4).
27. Hansen A, Bi P, Ryan P, Nitschke M, Pisaniello D, Tucker G. The Effect of heatwaves on Hospital admissions for renal disease in Adelaide, South Australia. EPIDEMIOLOGY. 2008;19(6):S105–6.
28. Khalaj B, Lloyd G, Sheppeard V, Dear K. The health impacts of heat waves in five regions of New South Wales, Australia: a case-only analysis. Int Arch Occup Environ Health. 2010;83(7):833–42.
29. Lu P, Miao J, Feng S, Green D, Lim YH, Gao X, Li S, Guo Y. Temporal variations of the association between summer season heat exposure and hospitalizations for renal diseases in Queensland, Australia, 1995–2016. Environ Res Lett 2022, 17(6).
30. Lu P, Xia G, Zhao Q, Green D, Lim YH, Li S, Guo Y. Attributable risks of hospitalizations for urologic diseases due to heat exposure in Queensland, Australia, 1995–2016. Int J Epidemiol. 2022;51(1):144–54.
31. 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.
32. Nitschke M, Tucker GR, Bi P. Morbidity and mortality during heatwaves in metropolitan Adelaide. Med J Aust. 2007;187(11–12):662–5.
33. Wang XY, Barnett A, Guo YM, Yu WW, Shen XM, Tong SL. Increased risk of emergency hospital admissions for children with renal diseases during heatwaves in Brisbane, Australia. World J Pediatr. 2014;10(4):330–5.
34. Wang XY, Barnett AG, Yu W, FitzGerald G, Tippett V, Aitken P, Neville G, McRae D, Verrall K, Tong S. The impact of heatwaves on mortality and emergency hospital admissions from non-external causes in Brisbane, Australia. Occup Environ Med. 2012;69(3):163–9.
35. Hansen AL, Bi P, Ryan P, Nitschke M, Pisaniello D, Tucker G. The effect of heat waves on hospital admissions for renal disease in a temperate city of Australia. Int J Epidemiol. 2008;37(6):1359–65.
36. Zhang Y, Nitschke M, Krackowizer A, Dear K, Pisaniello D, Weinstein P, Tucker G, Shakib S, Bi P. Risk factors of direct heat-related hospital admissions during the 2009 heatwave in Adelaide, Australia: a matched case-control study. BMJ Open. 2016;6(6):e010666.
37. Zhang Y, Nitschke M, Bi P. Risk factors for direct heat-related hospitalization during the 2009 Adelaide heatwave: a case crossover study. Sci Total Environ. 2013;442:1–5.
38. Wondmagegn BY, Xiang J, Dear K, Williams S, Hansen A, Pisaniello D, Nitschke M, Nairn J, Scalley B, Xiao A, et al. Increasing impacts of temperature on hospital admissions, length of stay, and related healthcare costs in the context of climate change in Adelaide, South Australia. Sci Total Environ. 2021;773:145656.
39. Xiao J, Spicer T, Jian L, Yun GY, Shao C, Nairn J, Fawcett RJB, Robertson A, Weeramanthri TS. Variation in population vulnerability to heat wave in Western Australia. *Frontiers in Public Health* 2017, 5(APR).
40. 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(2):1942–59.
41. Loughnan M, Tapper N, Loughnan T. The impact of unseasonably warm spring temperatures on acute myocardial infarction hospital admissions in Melbourne, Australia: a city with a temperate climate. J Environ Public Health. 2014;2014:483785.
42. Wang XY, Barnett AG, Hu W, Tong S. Temperature variation and emergency hospital admissions for stroke in Brisbane, Australia, 1996–2005. Int J Biometeorol. 2009;53(6):535–41.
43. Xu Z, Tong S, Ho HC, Lin H, Pan H, Cheng J. Associations of heat and cold with hospitalizations and post-discharge deaths due to acute myocardial infarction: what is the role of pre-existing diabetes? Int J Epidemiol. 2022;51(1):134–43.
44. Williams S, Nitschke M, Sullivan T, Tucker GR, Weinstein P, Pisaniello DL, Parton KA, Bi P. Heat and health in Adelaide, South Australia: assessment of heat thresholds and temperature relationships. Sci Total Environ. 2012;414:126–33.
45. Vaneckova P, Bambrick H. Cause-specific hospital admissions on hot days in Sydney, Australia. PLoS ONE. 2013;8(2):e55459.
46. Inglis SC, Clark RA, Shakib S, Wong DT, Molaee P, Wilkinson D, Stewart S. Hot summers and heart failure: seasonal variations in morbidity and mortality in Australian heart failure patients (1994–2005). Eur J Heart Fail. 2008;10(6):540–9.
47. Xu Z, Tong S, Cheng J, Crooks JL, Xiang H, Li X, Huang C, Hu W. Heatwaves and diabetes in Brisbane, Australia: a population-based retrospective cohort study. Int J Epidemiol. 2019;48(4):1091–100.
48. Xu Z, Tong S, Pan H, Cheng J. Associations of extreme temperatures with hospitalizations and post-discharge deaths for stroke: what is the role of pre-existing hyperlipidemia? Environ Res. 2021;193:110391.
49. Xu Z, Tong S, Cheng J, Zhang Y, Wang N, Hayixibayi A, Hu W. Heatwaves, hospitalizations for Alzheimer's disease, and postdischarge deaths: a population-based cohort study. Environ Res. 2019;178:108714.
50. Turner RM, Hayen A, Dunsmuir WT, Finch CF. Air temperature and the incidence of fall-related hip fracture hospitalisations in older people. Osteoporos Int. 2011;22(4):1183–9.
51. Wondmagegn BY, Xiang JJ, Dear K, Williams S, Hansen A, Pisaniello D, Nitschke M, Nairn J, Scalley B, Varghese BM, et al. Impact of heatwave intensity using excess heat factor on emergency department presentations and related healthcare costs in Adelaide, South Australia. SCIENCE OF THE TOTAL ENVIRONMENT; 2021. p. 781.
52. Toloo GS, Guo Y, Turner L, Qi X, Aitken P, Tong S. Socio-demographic vulnerability to heatwave impacts in Brisbane, Australia: a time series analysis. Aust N Z J Public Health. 2014;38(5):430–5.
53. Tong S, Wang XY, FitzGerald G, McRae D, Neville G, Tippett V, Aitken P, Verrall K. Development of health risk-based metrics for defining a heatwave: a time series study in Brisbane, Australia. BMC Public Health. 2014;14:435.
54. Xu Z, FitzGerald G, Guo Y, Jalaludin B, Tong S. Assessing heatwave impacts on cause-specific emergency department visits in urban and rural communities of Queensland, Australia. Environ Res. 2019;168:414–9.
55. Xu Z, Huang C, Hu W, Turner LR, Su H, Tong S. Extreme temperatures and emergency department admissions for childhood asthma in Brisbane, Australia. Occup Environ Med. 2013;70(10):730–5.
56. Xu Z, Hu W, Su H, Turner LR, Ye X, Wang J, Tong S. Extreme temperatures and paediatric emergency department admissions. J Epidemiol Community Health. 2014;68(4):304–11.
57. Campbell SL, Remenyi TA, Williamson GJ, White CJ, Johnston FH. The Value of Local Heatwave Impact Assessment: A Case-Crossover Analysis of Hospital Emergency Department Presentations in Tasmania, Australia. *Int J Environ Res Public Health* 2019, 16(19).
58. Williams S, Nitschke M, Weinstein P, Pisaniello DL, Parton KA, Bi P. The impact of summer temperatures and heatwaves on mortality and morbidity in Perth, Australia 1994–2008. Environ Int. 2012;40:33–8.
59. Borg M, Bi P, Nitschke M, Williams S, McDonald S. The impact of daily temperature on renal disease incidence: an ecological study. Environ Health. 2017;16(1):114.
60. Borg M, Nitschke M, Williams S, McDonald S, Nairn J, Bi P. Using the excess heat factor to indicate heatwave-related urinary disease: a case study in Adelaide, South Australia. Int J Biometeorol. 2019;63(4):435–47.
61. Xu ZW, Hu XX, Tong SL, Cheng J. Heat and risk of acute kidney injury: an hourly-level case-crossover study in queensland, Australia. Environ Res 2020, 182.
62. Wondmagegn BY, Xiang J, Dear K, Williams S, Hansen A, Pisaniello D, Nitschke M, Nairn J, Scalley B, Varghese BM, et al. Impact of heatwave intensity using excess heat factor on emergency department presentations and related healthcare costs in Adelaide, South Australia. Sci Total Environ. 2021;781:146815.
63. Xu Z, Liu Y, Ma Z, Sam Toloo G, Hu W, Tong S. Assessment of the temperature effect on childhood diarrhea using satellite imagery. Sci Rep. 2014;4:5389.

64. Turner LR, Connell D, Tong SL. Exposure to hot and cold temperatures and ambulance attendances in Brisbane, Australia: a time-series study. BMJ OPEN 2012, 2(4).
65. Turner LR, Connell D, Tong S. The effect of heat waves on ambulance attendances in Brisbane, Australia. Prehosp Disaster Med. 2013;28(5):482–7.
66. Campbell SL, Remenyi T, Williamson GJ, Rollins D, White CJ, Johnston FH. Ambulance dispatches and heatwaves in Tasmania, Australia: a case-crossover analysis. Environ Res 2021, 202.
67. Zhang W, McManus P, Duncan E. A raster-based Subdividing Indicator to Map Urban Heat vulnerability: a Case Study in Sydney, Australia. In: Int J Environ Res Public Health 15; 2018.
68. El-Zein A, Tonmoy FN. Assessment of vulnerability to climate change using a multi-criteria outranking approach with application to heat stress in Sydney. Ecol Ind. 2015;48:207–17.
69. Loughnan EM, Tapper J, Phan N, McInnes TA. Can a spatial index of heat-related vulnerability predict emergency service demand in Australian capital cities? Int J Emerg Serv. 2014;3(1):6–33.
70. Loughnan M, Nicholls N, Tapper NJ. Mapping Heat Health Risks in Urban Areas. Int J Popul Res. 2012;2012:518687.
71. Loughnan M, Tapper N, Phan T, Lynch K, McInnes J. A spatial vulnerability analysis of urban populations during extreme heat events in Australian. capital cities Final Report; 2013.
72. Sun Q, Hurley J, Amati M, Saunders A, Arundel J, Boruff B, Caccetta P. Urban Vegetation, Urban Heat Islands and Heat Vulnerability Assessment in Melbourne, 2018. In. Melbourne, Australia: Department of Environment, Land, Water and Planning, Victorian Government;; 2019.
73. Wang S, Sun QC, Huang X, Tao Y, Dong C, Das S, Liu Y. Health-integrated heat risk assessment in Australian cities. Environ Impact Assess Rev. 2023;102:107176.
74. Choy DL, Baum S, Serrao-Neumann S, Crick F, Sanò M, Harman B. Climate Change vulnerability in South East Queensland: a spatial and sectoral Assessment. In.; 2010.
75. Causes of Death. Australia [https://www.abs.gov.au/statistics/health/causes-death/causes-death-australia/2020#revisions-to-causes-of-death]
76. Greenland S. Ecologic versus individual-level sources of bias in ecologic estimates of contextual health effects. Int J Epidemiol. 2001;30(6):1343–50.
77. Quilty S, Jupurrurla NF, Lal A, Matthews V, Gasparrini A, Hope P, Brearley M, Ebi KL. The relative value of sociocultural and infrastructural adaptations to heat in a very hot climate in northern Australia: a case time series of heat-associated mortality. Lancet Planet Health. 2023;7(8):e684–93.
78. Zhang W, McManus P, Duncan E. A raster-based Subdividing Indicator to Map Urban Heat vulnerability: a Case Study in Sydney, Australia. Int J Environ Res Public Health 2018, 15(11).
79. Putnam H, Hondula DM, Urban A, Berisha V, Iñiguez P, Roach M. It's not the heat, it's the vulnerability: attribution of the 2016 spike in heat-associated deaths in Maricopa County, Arizona. Environ Res Lett. 2018;13(9):094022.
80. Guardaro M, Messerschmidt M, Hondula DM, Grimm NB, Redman CL. Building community heat action plans story by story: a three neighborhood case study. Cities. 2020;107:102886.
81. Weather Station Directory. [http://www.bom.gov.au/climate/data/stations/]
82. Kanti FS, Alari A, Chaix B, Benmarhnia T. Comparison of various heat waves definitions and the burden of heat-related mortality in France: implications for existing early warning systems. Environ Res. 2022;215:114359.
83. Oliveira A, Lopes A, Soares A. Excess heat factor climatology, trends, and exposure across European functional urban areas. Weather Clim Extremes. 2022;36:100455.
84. Scalley BD, Spicer T, Jian L, Xiao J, Nairn J, Robertson A, Weeramanthri T. Responding to heatwave intensity: excess heat factor is a superior predictor of health service utilisation and a trigger for heatwave plans. Aust N Z J Public Health. 2015;39(6):582–7.
85. Su CH, Eizenberg N, Steinle P, Jakob D, Fox-Hughes P, White CJ, Rennie S, Franklin C, Dharssi I, Zhu H. BARRA v1.0: the Bureau of Meteorology Atmospheric high-resolution Regional Reanalysis for Australia. Geosci Model Dev. 2019;12(5):2049–68.
86. Alexander LV, Arblaster JM. Historical and projected trends in temperature and precipitation extremes in Australia in observations and CMIP5. Weather Clim Extremes. 2017;15:34–56.
87. Li F, Yigitcanlar T, Nepal M, Thanh KN, Dur F. Understanding Urban Heat Vulnerability Assessment methods: a PRISMA Review. In: Energies 15; 2022.
88. Cheng W, Li D, Liu Z, Brown RD. Approaches for identifying heat-vulnerable populations and locations: a systematic review. Sci Total Environ. 2021;799:149417.
89. Xu Z, Hockey R, McElwee P, Waller M, Dobson A. Accuracy of death certifications of diabetes, dementia and cancer in Australia: a population-based cohort study. BMC Public Health. 2022;22(1):902.
90. Reid CE, Mann Jennifer K, Alfasso R, English Paul B, King Galatea C, Lincoln Rebecca A, Margolis Helene G, Rubado Dan J, Sabato Joseph E, West Nancy L, et al. Evaluation of a heat vulnerability index on abnormally hot days: an Environmental Public Health Tracking Study. Environ Health Perspect. 2012;120(5):715–20.
91. Khatana SAM, Eberly LA, Nathan AS, Groeneveld PW. Projected change in the burden of excess Cardiovascular deaths Associated with Extreme Heat by Midcentury (2036–2065) in the Contiguous United States. Circulation. 2023;148(20):1559–69.
92. Lee K, Brown RD. Effects of Urban Landscape and Sociodemographic Characteristics on Heat-Related Health Using Emergency Medical Service incidents. In: Int J Environ Res Public Health 19; 2022.
93. Jegasothy E, McGuire R, Nairn J, Fawcett R, Scalley B. Extreme climatic conditions and health service utilisation across rural and metropolitan New South Wales. Int J Biometeorol. 2017;61(8):1359–70.

Publisher's note