Author/s:
Walter, CM;Schneider-Futschik, EK;Lansbury, NL;Sly, PD;Head, BW;Knibbs, LD

Title:
The health impacts of ambient air pollution in Australia: a systematic literature review

Date:
2021-10-01

Citation:
Walter, C. M., Schneider-Futschik, E. K., Lansbury, N. L., Sly, P. D., Head, B. W. & Knibbs, L. D. (2021). The health impacts of ambient air pollution in Australia: a systematic literature review. Internal Medicine Journal, 51 (10), pp.1567-1579. https://doi.org/10.1111/imj.15415.

Walter Clare (Orcid ID: 0000-0002-7340-9689)

The health impacts of ambient air pollution in Australia: A systematic literature review

Clare M. Walter,1,2,3 Elena K. Schneider-Futschik2, Nina L. Hall1, Peter D Sly4,Brian W. Head5 and Luke D. Knibbs1
1School of Public Health, Faculty of Medicine, The University of Queensland, Brisbane, QLD, Australia
2Department of Pharmacology and Therapeutics, School of Biomedical Sciences, Faculty of Medicine, Dentistry and Health Sciences, The University of Melbourne, Melbourne, VIC, Australia
3Australian-German Climate and Energy College, The University of Melbourne, Melbourne, VIC, Australia
4Children's Health and Environment Program, Child Health Research Centre, The University of Queensland, South Brisbane, Qld, Australia.
5School of Political Science and Centre for Policy Futures, The University of Queensland.

c.walter@uq.edu.au, clarem.walter@gmail.com

Abstract

Background: Ambient (outdoor) air pollution is a key risk factor for health, for which effective policy plays an important preventative role. Australian federal and related state air quality standards have historically relied on international evidence for guidance, which may not accurately reflect the Australian context. There has been, however, a large increase in Australian epidemiological studies over recent years.

Aims: To provide an updated systematic literature review of peer-reviewed epidemiological studies that examined the health impacts of outdoor air pollution in Australia, including short- and long-term exposure.

Methods: Following PRISMA guidelines, we conducted a systematic literature review. Broad search terms were applied to two databases (PubMed and Web of Science) and Google Scholar. Quality assessment and risk of bias were assessed using standard metrics. Included studies were summarised by tabulating key study characteristics, grouped by health outcomes.

Results: In total, 72 studies were included in the review. Sixty-four studies (89%) used daily or hourly pollutant concentrations to examine short-term exposure impacts, of which 59 (92%) revealed significant associations with one or more health outcomes, including cardio-respiratory, all-cause mortality or morbidity, and birth outcomes. Eight studies (11%) used annual average pollutant concentrations to investigate long-term exposure finding significant associations with asthma, reduced lung function, atopy and cardio-respiratory mortality across five studies. The remaining three studies found no significant association with asthma, mortality and a range of self-reported diseases, respectively.

Conclusions: Ambient air pollution has substantial health impacts in Australia. The body of domestic evidence has increased markedly since national air quality standards were first set in the 1990s, which could be drawn on by policy-makers when revising the existing standards, or considering new standards.

This is the author manuscript accepted for publication and has undergone full peer review but has not been through the copyediting, typesetting, pagination and proofreading process, which may lead to differences between this version and the Version of Record. Please cite this article as doi: 10.1111/imj.15415

Introduction

Outdoor (ambient) air pollution is the leading environmental cause of premature death globally, with an estimated 4.2 million attributable deaths in 2016 (1). Estimates of attributable deaths in Australia have varied from 2,600 (2) to 4,884 annual premature deaths (3). A recent Australian analysis estimated an annual 2,616 premature deaths attributable to the anthropogenic fraction of fine particulate matter (4), the air pollutant most consistently and robustly associated with adverse human health outcomes. Unlike other leading health risks such as tobacco, alcohol and drug use, air pollution exposure is largely beyond the control of the individual. This places additional importance on effective policy-making to reduce the associated health burden. Standards for the six 'criteria' ambient air pollutants — particulate matter <10 μm, and <2.5 μm in aerodynamic diameter (PM10 and PM2.5, respectively), ozone (O3), nitrogen dioxide (NO2), sulfur dioxide (SO2), carbon monoxide (CO) and lead (Pb) — are set by the National Environmental Protection Measures for Ambient Air Quality (NEPM AAQs). This was first legislated in 1998, with subsequent revisions for particulate matter standards in 2015.

Globally, the past twenty years have seen an upsurge in air pollution epidemiological research that investigates associations for short-term impacts: allergies, asthma, bronchitis, heart attacks, arrhythmias and mortality; and long-term impacts: diabetes, stroke, chronic obstructive pulmonary disease (COPD), lung cancer, ischaemic heart disease (IHD) and lower respiratory infection (5). There is also a mounting body of evidence indicating a wider range of impacts which include: metabolic dysfunction, neurological and psychiatric disease, and adverse pregnancy and developmental outcomes (5).

To date, epidemiological studies investigating PM2.5 have not consistently observed evidence of a 'safe' lower threshold of exposure, below which there are no mortality effects (6, 7). This presents substantial challenges for policy-makers and regulators in the context of outdoor air pollution standards. Further complexities arise from the use of international evidence to guide Australian policy. The paucity of available Australian studies during the development of the NEPM AAQ (in 1998) required the selection of standards and methodologies be guided by landmark North American and European studies. The transferability of risk coefficients from international populations with differing underlying health status and air pollution exposure to the Australian context was debated by the National Environmental Protection Council (NEPC) at the time, as was the inability to account for vulnerable groups with potentially greater susceptibility, such as children and the elderly, and people with existing illness (8). It was noted that these limitations may negatively impact the protection of public health (9).

A 2005 review of the available Australian epidemiological literature, which spanned 1994 – 2002, revealed evidence of cardio-respiratory health effects of outdoor air pollution at levels below applicable standards (10). A key recommendation was for further domestic research to be conducted, the results of which should then be used in combination with international data to develop national standards (10). To our knowledge, subsequent to Howie et al., (2005), there has been just one further peer-reviewed review publication of Australian and New Zealand air pollution research; however, this was undertaken with a focus on exposure assessment techniques rather than health impacts (11).

More recently, international evidence reports the potential for steeper, non-linear exposure-response relationships at lower pollutant concentrations, compared with higher concentrations (6, 7, 12). This raises questions about the transferability of risk estimates

based on studies performed at a given concentration(s) to other locations with lower or higher concentrations, as is often done in the absence of local studies, such as in Australia during the establishment of the NEPM regulations in the late 1990s.

The significant damage of the bushfire season of 2019/2020 has heightened the level of awareness of air pollution impacts in the Australian public and policy-makers. Additionally, for the first time since the NEPM inception, the standards for NO2, SO2 and O3 are under current federal review. Given these topical issues and that numerous Australian studies have been undertaken in the 15 years since the review of Howie et al., — some which include new health outcomes beyond cardio-respiratory impacts— it is timely to perform an updated systematic review.

It is important to periodically consider the relative consistency of Australian and international evidence, in the context of setting air quality guidelines. However, this first requires identifying and assessing all relevant Australian studies, so the scope of our review was restricted to this aspect only. We aimed to perform a systematic literature review to identify and summarise peer-reviewed epidemiological studies of air pollution and health in Australia. Our secondary aims included describing, from the compiled evidence, studies of vulnerable sub-populations, and those that reported health impacts according to a specific pollution source (e.g. traffic emissions vs. bushfires).

2. Methods

We used the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) methodological framework (13).

2.1 Eligibility Criteria

We considered peer-reviewed journal articles of original epidemiological studies of varying design (including cohort, panel, case cross-over, case control, cross sectional, time series and ecological studies). Studies had to be performed in Australia. They also had to examine an exposure-response association between: (a) measured or modelled exposure to one or more criteria ambient air pollutant(s), and (b) one or more physical health outcome(s) in humans, either directly measured, elicited from questionnaire, or using administrative health data. Studies which included self-reported (or parent-reported) health outcome(s) that referred to a formal medical diagnosis of a specific disease were included (e.g. diabetes), while those based on self-reported symptoms not connected with a specific disease (e.g. stuffy nose) were not. No date limitation was applied; the search included all articles published prior to the 1st July 2019.

The following additional exclusion criteria were applied:

Studies using proxies of pollutant exposure (e.g. distance to source, traffic volumes) were excluded due to the inability to ascribe observed effects to a specific pollutant/(s), and because criteria pollutants are subject to regulation under the NEPM but pollutant proxies are not;

Studies not reporting numerical effect sizes;

Non-original research (i.e., literature reviews) or health impact assessment analyses using effect sizes from other studies to estimate health burden;

Studies of exposure in indoor or occupational settings.

For studies that involved multiple methods and outcomes, only those that matched the inclusion criteria were extracted. For example, if a study considered the link between both pollutant concentration (measured or modelled) and distance to nearest road (proxy measurement), and self-reported symptoms (subjective) and lung function tests

(objective), the extracted results would only include those generated by the measured pollutant concentration and lung function tests.

2.2 Scope and search strategy

Papers indexed in the databases PubMed (14), and Web of Science (15) were considered. Manual screening of references and citations from retrieved articles, along with a Google scholar search, were also undertaken to identify any additional relevant articles not identified by the main searches. All search terms were developed in consultation with a research librarian; search terms are in Supporting information, Appendix SA1.

2.3 Selection

Articles identified were screened by title and abstract. The full text of articles not excluded at this stage was downloaded for further assessment against the exclusion criteria. Each step of the search process was undertaken independently by two authors (CMW and EKSF). When differences arose regarding inclusion, a third author (LDK) was used as a tiebreaker.

2.4 Data extraction and synthesis

Included studies were summarised by extracting exposure characteristics: pollutants, main source, concentrations and whether the exposure was short-term (daily concentrations) or long term (annual concentrations with chronic impact); location, health outcomes, study design, population and duration; and confounders, statistical methods and effect sizes (e.g., relative risk, hazard ratio, odds ratio). As well as numerical findings of individual studies, qualitative description and comparison across the body of research was incorporated, with specific focus on outcomes occurring below current NEPM thresholds. Results were grouped by health endpoints, pollutant sources and identified vulnerable population groups, where relevant.

2.5 Quality assessment

To the authors' knowledge, there is no one quality assessment tool that can be applied across the range of study designs that were included in this review; therefore, three different tools were selected, based on previous validation and applicability to air pollution related epidemiological studies. The Newcastle-Ottawa scale (16) was used to assess cohort and case control studies. The assessment is comprised of eight items grouped into three categories: selection, comparability, and exposure or outcome, with a scale ranging from zero to a maximum of nine (highest quality). Studies scored six or above were included in the review. An adapted version of the Newcastle-Ottawa scale (17) was used for cross-sectional studies: studies scoring seven or more on the ten point scale were included.

Time-series and case-crossover studies were rated with the criteria used by Mustafić and colleagues (18). Studies were scored on three components: health outcome, exposure and adjustment for confounders. The scale ranges from zero to five (highest quality); studies scoring four or higher were included.

Risk of bias was assessed with the Office of Health Assessment and Translation (OHAT) tool by the National Institutes of Environmental Health Sciences National Toxicology Program (19) and the Navigation Guide by the University of California (20). A set of ten questions encompassed: Exposure assessment, outcome assessment and confounding bias and Other Criteria (Selection bias, Attrition/exclusion bias, Selective reporting bias, Conflict of interest, Other source of bias). Each question was graded on a four-point scale, ranging from definitely low, probably low, probably high (or not recorded) and definitely high. Studies which received a "high" or "probably high" risk for key criteria were

removed. The remaining ecological studies were graded using a validated assessment tool developed by Zaza et al., consisting of 24 items designed to rate the study design, methodology, validity, reliability, reporting and risk of bias (21). A point is given if the study meets the criteria for each item. Studies that scored over 70 per cent of applicable points were included in the review.

Quality and risk of bias assessments of all included studies were done separately by two authors (CMW) and (EKSF). Areas of uncertainty or disagreement were referred to a third author (LDK).

Results

3.1 Study selection

Figure 1. Study selection

Figure 1 shows the PRISMA flowchart of our search. In total, 11,669 articles were identified through database searches. An additional 16 papers were identified through Google Scholar and the screening of manual citations from identified key papers. After excluding duplicates, 7,725 papers were assessed against the exclusion criteria by title (7,411 excluded), then abstract (165 excluded), and finally full text (73 excluded). Of the remaining 76 studies, four did not meet the quality assessment or study-level risk of bias assessment. The final review therefore comprised 72 studies analysing the link between ambient air pollution and public health in Australia; a detailed summary of the included studies, including extracted information, is in Supporting information Table S1.

Study characteristics

Publication date range

All but four studies were published during or after 1998, the same year the NEPM AAQs were introduced. Figure 2 displays the frequency of publications for each calendar year.

Figure 2. Year of study publication

Study designs

Twenty-two of the included studies were time series design (31%), followed by 18 case cross-over studies (25%), 12 retrospective cohort studies (17%), six prospective cohort studies (8%) and six cross-sectional studies (8%). The remaining designs included five studies defined as ecological (7%), two case-control studies (3%) and one panel study (1%). Sixty-four (89%) examined short-term impacts, with pollutant exposure averaging measurements in hours or days. Eight studies (11%) examined the chronic impacts of long-term exposure using annual average pollutant concentrations. All included studies accounted for some key covariates, most commonly: temperature, humidity, and temporal trends (Table S1). Studies using comparatively large (8,000 plus) well-defined longitudinal cohorts (22-25) and a recent cross-sectional study (26) included individual level confounder data. All studies examining birth outcomes accounted for a range of potential maternal confounders such as age, smoking status, underlying medical conditions and socio-economic status. Only one study was able to account for individual exposure variability, through the use of personal monitors (27).

Study locations

Most studies were undertaken in major metropolitan centres. Four studies examined state-wide impacts (25, 28-30) and one considered three states (31). The remaining multiple site studies were based in discrete cities, except for one study which considered bushfire impacts in both Melbourne and regional Gippsland (VIC) (32). Two studies combined data from Australian and New Zealand cities (33, 34). Only one study exclusively considered a regional area, investigating the impacts of a large uncontrolled mine fire in the La Trobe Valley (35).

Pollutant sources

Figure 3 shows the number of studies by nominal pollutant source, as reported in each study. Generally, traffic related air pollution (TRAP) is a predominant contributor to ambient air pollution in metropolitan centres; however, unless the main source was specifically referred to as 'traffic' in a study, it was recorded as 'general ambient'. Pollution arising from bushfires, vegetation burns, and planned burns is collectively referred to as landscape fire smoke (LFS). In Darwin, LFS is major source of air pollution during the 'dry' season and was the dominant pollutant source investigated in all Darwin-based studies (36-39). Two studies in Launceston investigated pollution predominantly arising from wood heaters (40, 41).

Figure 3. Pollutant Sources

3.2.5 Pollutant exposure assessment

From the mid-1990s, most states and territories had fixed-site monitor networks of instruments collecting measurements of one or more of total suspended particulates (TSP), PM10, light-scattering particles (i.e., light-scattering particles [BSP] measured by nephelometry), SO2, O3, NO2, while PM2.5 measurements became more widely available from the early 2000s. Advancements in exposure modelling have also occurred. For example, land-use regression modelling (LUR), which combines spatial predictor variables, and sometimes satellite data, to estimate pollutant concentrations for populations regardless of how proximate they are to a monitoring site. Fifty-three (74%) of the studies utilised pollutant measurements from fixed-site monitors. Eighteen (25%) used modelling, nine (13%) of which were done with LUR estimates of NO2. One recent study used portable air quality monitors worn by the study participants, to measure ultrafine particles (<100 nm) (27).

Sixty-one (85%) studies included one or more measurements of particulate matter as PM10, PM2.5, BSP and ultrafine particles. Although the latter two are not criteria pollutants, they were measured in the context of studies measuring multiple pollutants. NO2 was considered in 43 studies (60%), O3 in 33 studies (46%), SO2 in 16 studies (22%) and CO in 14 studies (19%). Of the forty-one studies to examine both PM with one or more gas pollutants, 23 (56%) described the use of two-pollutant or multi-pollutant models to account for the confounding effect of pollutants on each other.

The pollutants assessed and measurement methods are described for each study in Table S1.

Health outcomes

Respiratory and cardiovascular impacts were the most investigated health outcomes (Figure 4). Most studies analysed short-term impacts of air pollutants, with investigations of long-term impacts limited to the few studies using longitudinal analyses or cross-sectional analyses with cohort studies (22, 23, 25, 26, 42-44) and one ecological study (45) (Table S1). The cardio-respiratory effects of single pollutants were generally found to persist in multi-pollutant models (45-51); however, some attenuation of effect was noted, particularly for PM when NO2 was included in the model. (47, 48, 52-55). The respiratory impacts of NO2 remained strong and unaltered in multi-pollutant models (26, 34, 48) and across different statistical models (56). Due to the substantial heterogeneity of methods and outcomes, a meta-analysis was not attempted.

Figure 4. Health outcomes

Short-term respiratory effects

Ambient air pollution was consistently associated with short-term respiratory impacts. Thirty-nine studies (54%) examined short-term respiratory impacts (Table S1), of which 36 (92%) reported one or more statistically significant adverse associations. The remaining three studies found no asthma associations for particulate matter generated by a dust storm (57), LFS (58), or SO2 from an industrial point source (59). PM was the most frequently reported pollutant, and was included either individually or in combination with other pollutants in 37 studies (95%). Effects were generally greatest on the day of exposure (lag 0) (50, 60, 61). Chronic obstructive pulmonary disease (COPD) associations were mixed, with non-significant positive associations for general ambient PM (48) and strong significant associations for LFS PM (50, 62). Twenty of 26 (77%) studies found significant associations for asthma, with differences noted across sources. Significant associations for a range of respiratory infections were also revealed in children (34, 63, 64).

3.3.2 Long-term respiratory effects

Seven studies (10%) investigated long-term respiratory impacts with mixed results (Table S1). Five studies used modelled annual NO2 concentration, revealing significant associations for atopy (42), asthma and reduced lung function in children (26), and a sub-group of adults who carried of a variant genotype for the endogenous anti-oxidant glutathione(23). A significant association for severe asthma exacerbations was also demonstrated in a small cohort of thunderstorm asthma patients (44). No associations were found for self-reported asthma and COPD in a cohort of adult woman (43). Long-term exposure to PM2.5 and NO2 were not associated with pneumonia or COPD, but weak positive associations were observed for asthma and all respiratory diseases (25). A significant association with cardio-respiratory mortality was demonstrated for increases in annual average SO2 concentrations, but was not present for NO2 and O3 (45).

3.3.3 Cardiovascular effects

Twenty-seven studies (38%) examined cardiovascular associations, with 18 (67%) finding significant associations, predominantly with particulate matter (17 studies) and nitrogen dioxide (6 studies) (Table S1). Impacts were greatest in the first 48 hours after exposure (54, 65). Cardiovascular hospital admissions related to LFS were generally non-significant (37-39, 62); however, significant associations were revealed between LFS and out of hospital cardiac arrests (OHCAs) in the first 48 hours after exposure (28, 66), and ischaemic heart disease (IHD) at lagged intervals of 2 – 3 days (28, 62). A recent Tasmanian study identified a lower PM2.5 threshold for heart failure of 4μg/m3 (30). Two

long-term exposure studies included cardiovascular endpoints, finding: a significant association between SO2 and 'cardio-respiratory mortality' (described in section 3.3.2) (45); and no associations between modelled NO2 and self-reported heart disease (43).

3.3.4 All-cause mortality and morbidity

All–cause mortality and morbidity were consistently associated with short-term PM exposures (11 studies), with the largest effects noted 24 hours post exposure (lag 1) (50, 65, 67) (Table S1). There was less consistency across the other pollutants, with some studies revealing significant associations between short-term O3 and NO2 and all-cause mortality (54, 65), and a strong association between sulfur dioxide and mortality during Sydney summers (68). Long-term exposures to NO2 and PM2.5 were associated with large, but non-significant, increased risk of mortality (22).

3.3.5 Adverse birth outcomes

Eleven studies examined pre-natal exposure impacts, with mixed outcomes varying according to season of conception, pollutants considered, proximity to fixed site monitor, and windows of exposure (Table S1). Significant positive associations were found across the range of pollutants for preterm birth and/or reduced intra-uterine growth (5 studies), and congenital abnormalities after second trimester exposure to PM10 (69). The remaining studies found weak, inconsistent or no associations for congenital defects (70), preterm birth (35, 71) and reduced foetal growth (35, 72).

A consistent trend noted across all nine studies to examine trimester-specific impacts of gases (predominantly NO2) was for lower risk estimates, and in some cases even negative (protective) associations, for exposures in the first trimester. This increased to positive and significant risk effects for exposures in the second or third trimester.

Exposure misclassification was a limitation across all studies; stratified risk estimates were significantly higher for study sub-populations in closer proximity to static air pollution monitors (70, 71, 73) and 'stay at home' mothers (74).

Impacts below NEPM thresholds

Forty-seven (94%) of the 50 studies to examine diffuse ambient pollution unrelated to a specific pollution event found one or more significant associations below the applicable NEPM AAQ standards. Nearly half of these studies (22 studies) investigated respiratory endpoints, finding the most consistent associations with NO2 (12 studies). Sixteen of the eighteen studies (89%) to examine cardiovascular endpoints found one or more significant associations, predominantly with PM2.5 (13 studies) and NO2 (8 studies). In the elderly, CO was associated with the highest cardiovascular risk estimates (33, 75).

3.5 Source-specific pollution

The evidence presented suggests source-specific variances in magnitude and temporality of pollution impacts. Risk estimates for respiratory outcomes were consistently high in LFS studies (38, 39, 62), with direct comparisons between general background PM and LFS PM revealing higher respiratory risk estimates for the latter (50, 60, 61). Conversely, the evidence for LFS cardiovascular impacts was weaker by comparison to general ambient PM. Several studies observed highest mortality risk effects at a lagged interval of one day post-exposure (lag 1) for ambient and LFS PM (50, 65, 67), which was found to change to three days (lag 3) for PM generated by dust storms (67). Age-stratified studies revealed differing groups, with heightened sensitivities contingent on pollution source (Table S1).

3.6 Vulnerable groups

Age, gender, Indigenous status, socio-economic status, and the underlying presence of asthma or atopy were identified as factors contributing to increased vulnerability (Table S1).

Age was the most investigated vulnerability, with studies stratifying risks for children (15 studies), elderly (20 studies), and other studies limiting investigations to children (7 studies) or elderly (1 study).

Significant associations were consistently present between TRAP or general ambient pollution and childhood asthma, with higher risk estimates when compared to adults (48, 55, 57, 76). An early study observed children's asthma was most strongly associated with NO2 (48), which was supported by subsequent studies (Table S1), although some differences were observed across age sub-sets of children, with some larger respiratory risk estimates for CO and SO2 (34, 47). Increased hourly or daily NO2, with IQR increases ranging from 2.81ppb to 9.5ppb, were associated with a 3 – 14 per cent increase in asthma hospital attendances (34, 47, 77), which in one study increased to 70 per cent when further stratified to children 0 – 4 years (77). The 0-4 year age group was consistently identified as the most vulnerable to respiratory impacts, by studies that stratified risks across children's age groups (34, 47, 77). A cross-sectional study of school children across 12 cities found that an IQR (4ppb) increase in annual NO2 was significantly associated with a 24 per cent and 54 per cent greater odds of current (i.e. prevalent) asthma, for fixed-site and LUR model exposure assessments respectively. A significant association was revealed for airways inflammation and reductions in some lung function tests. Higher point estimates for airways inflammation and lung function reductions were noted for school place exposures (compared to residential), although only when using the fixed-site assessment (26).Conversely, age stratified LFS studies revealed higher asthma impacts in adults (29, 50, 62), with one study finding a six-fold higher risk estimate for the 15 – 64 year age group, compared to < 15 years (62).

Mortality and cardiovascular impacts were consistently higher in the elderly (≥ 65 years) (52, 78). Further age stratification in two studies identified the 65 – 74-year age group as most vulnerable to ozone associated OHCAs (66), and PM associated cardiovascular hospital admissions (79). In both studies, the risk estimates decreased at 75 years and over. There was less evidence of increased respiratory impacts for elderly exposed to general ambient pollution, although two studies noted significantly higher respiratory morbidities associated with NO2 (76) and PM (50) in the elderly.

Nine studies considered gender specific effects. Several studies noted higher risks in males for mortality (40), all-case morbidity (80), OHCAs (28, 51, 66) and acute coronary syndrome (63). Higher asthma impacts were also noted in male children exposed to TRAP (77). One study observed greater risks in females for LFS asthma (same day) and IHD (lag2-3)(28).

Pre-existing conditions that increased the likelihood or severity of respiratory impacts included atopy (27), asthma (25, 81) and carriers of genetic polymorphisms for Glutathione S-transferase (GST) (23).

Three studies stratified risks for Indigenous Australian participants, identifying higher risks for general ambient pollution and emergency department admissions (EDA) (80), and for LFS associated respiratory admissions (37, 38) and lagged associations with IHD (37) and respiratory infections (38). In some cases, the disparity of impacts was large: a borderline association between LFS PM10 and respiratory ED admissions increased from 8 to 17 per cent for the Indigenous subpopulation (37). Pneumonia admissions three days post

exposure (lag3) was 15 per cent for Indigenous Australians, yet no association was observed in the overall population (38).
Only one study stratified results according to socio-economic status, finding a stronger association between short-term pollutant exposure and EDA admissions in the lower SES study population (80).

4. Discussion

Our review has provided an update of Australian air pollution literature, which has not been systematically reviewed and presented in a peer-reviewed publication for 15 years. We identified 72 studies that examined the relationship between outdoor exposure to one or more criteria pollutants and an objectively measured physical health outcome. The publication dates spanned from 1987 to 2019. Investigated health outcomes were predominantly cardio-respiratory endpoints associated with short-term exposures in metropolitan regions. There was a small overlap (10 studies) with the only comparable peer-reviewed publication, by Howie et al., (2005) indicating most of the literature in this review (62/72 studies) is collated as a single body of evidence for the first time. The evidence was consistent with the previous review, which also found positive associations between short-term ambient air pollution exposure and cardio-respiratory health outcomes that persisted at concentrations below current NEPM AAQ thresholds. The subsequent increase in number and methodological diversity of studies, and the qualitative trend for increasingly sophisticated methods, enabled the inclusion of gene-environment interactions and biomarkers. This provided additional insights of the impacts and underlying susceptibilities of various sub-populations. Birth outcomes were reviewed for the first time, and revealed mixed results: with six of the nine studies that examined pre-term birth or reduced intrauterine growth revealed positive associations. Globally, an estimated 82 per cent of disease burden attributable to air pollution stems from chronic diseases related to long-term exposures (5), yet this review identified only eight Australian studies (11%) that investigated long-term exposure, five of which revealed adverse associations with one or more health outcomes.
Overall, the Australian literature was consistent with international findings, in terms of the relative importance of different pollutants, or pollutant mixtures, on health. However, there are some differences that merit consideration. Several Australian studies investigating short-term exposures to general ambient PM2.5 revealed cardiovascular risk estimates that were higher than comparable landmark North American and European studies (51, 53, 82). Long-term impacts are only recently emerging from Australian literature. Only one study was identified that examined chronic PM2.5 exposure and mortality, revealing a point estimate nearly tenfold higher than the landmark American Cancer Society Study (83); however, the confidence intervals were wide and non-significant (22). These findings signpost, along with recent international work suggesting steeper cardio-respiratory exposure-response relationships at lower pollution concentrations, this is a topic suited to further investigation in Australia.
NO2, a surrogate for TRAP, was consistently associated with significant respiratory impacts across studies with different designs, locations, measurement techniques and statistical approaches. Respiratory effects were not attenuated in multi-pollutant models, demonstrating potential independent effects of NO2 alone or due to correlation with non-criteria TRAP pollutants (e.g. ultrafine particles, black carbon). The associations were present for small incremental increases in short-term exposures, occurring at background

1-hour average concentrations, ranging from 15.7 - 23.2 ppb (34). A 4ppb increase in annual exposure, at an average annual background concentration of 8.8ppb, was significantly associated with a very large increased risk of in asthma for children (26). These findings are relevant to the current NEPM review of NO2 standards, as they demonstrate evidence of asthma and lung damage occurring at concentrations well below the proposed new standards of 19ppb and 90ppb for annual and worst hour respectively. The NEPM methodologies stipulate that in setting health-based standards, consideration should be given to sensitive groups. In recognition of Australia's high prevalence of childhood asthma, particular attention should be given to this condition (8).

These findings are also relevant for major roads infrastructure. A recently published Australian guideline (84) suggested that development and planning decisions should be based on long-term PM2.5 exposure and premature mortality in > 30 years, using the risk co-efficient from the landmark American Cancer Society study (83). Based on the evidence presented in this review, we believe risk assessments for road infrastructure projects could be expanded to include more exposure-outcome pairs that are more relevant to that context (e.g. children's respiratory morbidity endpoints associated with NO2).

A key finding of our review was the observed trend of differences in magnitude and temporality of health impacts, along with vulnerable groups contingent on pollution source. Broadly, TRAP exerted greater respiratory impacts on children, while for LFS pollution, the impacts increased in magnitude with age. Immediate cardiac impacts were more apparent for ambient PM exposure and traffic pollution, while IHD associated with PMLFS manifested two to three days post exposure. The elevated acute cardiac impacts of bushfire and wood-heater pollution in males, along with the greater IHD and asthma impacts in females, may implicate hormones play some role in the underlying pathological mechanisms. A greater understanding of their role may be valuable in preparing for future pollution events, such as bushfires and dust storms. This would enable targeted education, and the ability to anticipate the likely shifts in resourcing requirements of the health care system.

Suggestions for future research

While some evidence indicated adverse birth outcomes, such as pre-term birth, and reduced intra-uterine growth (73, 74, 85-87), overall the birth outcomes were heterogeneous and it was not possible to draw firm conclusions. Further research is required to better characterise the range of neo-natal impacts and identify specific exposure windows of heightened risk within the pregnancy.

Long-term exposure impacts contribute to the bulk of air pollution related disease and death, yet thus far, chronic exposures are underrepresented in the domestic literature. Further research is required to better understand the impacts of long-term air pollution exposure in Australia, including expanding the range of considered health endpoints to include the causal outcomes of lung cancer and diabetes.

There is a significant knowledge gap pertaining to regional communities who are exposed to the emissions of coal-fired power stations. While these communities tend to be small, creating difficulty in undertaking a sufficiently powered epidemiological study, they are exposed to the largest source of anthropogenic particulate pollution in Australia (88) and have been a long-documented cause of concern (89). Excepting a large uncontrolled mine fire (35), this review failed to identify any studies that considered this source.

Finally, Australia has produced landmark studies identifying source-specific impacts of LFS (50, 61). As LFS becomes an increasing concern with warming temperatures, Australia is uniquely placed to build on the existing evidence presented in this review providing valuable information for preventative and emergency health responses.

Strengths and limitations

Our review was rigorously undertaken following the standard PRISMA protocol. The screening of each database, study selection and quality assessment of studies was independently undertaken by two authors CMW and EKSF. All included studies controlled for some potential confounders, most commonly: temperature, humidity, temporal trends and respiratory infections. Individual level confounders were accounted for in studies examining birth outcomes and prospective cohort studies. A common theme for all studies, excepting one which utilised personal monitors (27), was the potential inaccuracies of exposure assessments, which were unable to account for intra-urban variability. Over two thirds of the studies included in this review used fixed site monitors, and noted the limitations in capturing spatial variability of population exposure. This resulted in a likely downward bias (i.e., attenuation) of associations. Due to the high level of heterogeneity in exposure measurements and reporting of health impacts across the included studies, a meta-analysis was not undertaken. The included studies ranged in design and size, with one quarter being cohort design and of modest size by international comparison. The exclusion of proxy exposure measurements and subjective health measurements, such as questionnaires, resulted in the omission of several otherwise well-conducted studies that were relevant to the remit of our review.

Conclusion

This review provides an overdue update, at a time when air pollution is front and centre of the political and public eye. We have collated a substantial body of evidence for short-term exposures and cardio-respiratory disease, which has accumulated to a point where policymakers could now begin to draw on Australian studies as part of a weight of evidence approach to inform Australian policy and standards. In general, more Australian studies on long-term exposures are needed to be useful to policy-makers. Outdoor air pollution is a significant contributor to the burden of disease and premature death in Australia, and while some of this burden is created by pollution events such as bushfires and dust storms, everyday exposures in low concentration settings to traffic, wood heaters and general ambient pollution also play an important role. There are vulnerable groups who have greater susceptibilities to the health effects of air pollution. Current health risk assessment methodologies may not adequately meet key NEPM AAQ objectives of adequate protection and the adoption of the precautionary principle. There are apparent differences in the magnitude and range of health impacts across different pollutant sources, which may be beneficial in formulating preventative strategies aimed at reducing the health burden of outdoor air pollution in Australia.

Declaration of competing interest

The authors declare they have no actual or potential conflicts of interest

Acknowledgements

The authors want to thank Scott Mcintyre, specialist public health librarian at the University of Queensland, and Raymond Soo (NAB) for their technical support.
CMW is supported by an Australian Government Research Training Program Scholarship from the University of Queensland.

E.K.S.-F. supported by a School of Biomedical Science ECR Grant. E.K.S.-F. is a Research Fellow of the Australian National Health and Medical Research Council (NHMRC).
PDS is a Senior Principal Research Fellow of the Australian National Health and Medical Research Council (NHMRC).

Supporting information

Additional supporting information may be found in the online version of this article at the publisher's website:

Figure SA1. Database search strategies
Table S1. Summary of studies

References

1. WHO. Ambient (outdoor) air pollution. 2018. Report No.: WHO CC BY-NC-SA 3.0 IGO; 2018.
2. AIHW. Australian Burden of Disease Study: impact and causes of illness and death in Australia 2015. In: Welfare AIoHa, editor. Canberra: Australian Federal Government; 2019.
3. Global Burden of Disease Study (GBD 2017). Burden by Risk 1990 - 2017. [Internet]. Institute for Health Metrics and Evaluation (IHME). 2018.
4. Hanigan IC, Broome RA, Chaston TB, Cope M, Dennekamp M, Heyworth JS, et al. Avoidable Mortality Attributable to Anthropogenic Fine Particulate Matter (PM2. 5) in Australia. International journal of environmental research and public health. 2021;18(1):254.
5. HEI. State of the Global Air. Boston, MA: Health Effects Institute; 2019. Report No.: ISSN 2578-6873.
6. Di Q, Wang Y, Zanobetti A, Wang Y, Koutrakis P, Choirat C, et al. Air Pollution and Mortality in the Medicare Population. The New England journal of medicine. 2017;376(26):2513-22.
7. Pinault L, Tjepkema M, Crouse DL, Weichenthal S, van Donkelaar A, Martin RV, et al. Risk estimates of mortality attributed to low concentrations of ambient fine particulate matter in the Canadian community health survey cohort. Environmental Health. 2016;15(1):18.
8. NEPC. Methodology for setting air quality standards in Australia. Part A. Canberra: Federal Government; 2011.
9. Sullivan R, Beudeker B. New directions in air quality impact assessment in Australia. Clean Air and Environmental Quality. 1999;33(3):32.

10. Howie J, Tong S, Verrall K, Gerber R, Wolff R. Air pollution and cardiopulmonary diseases in Australia: A review of epidemiological evidence. Environmental Health. 2005;5(1):23.
11. Kingham S, Dorset W. Assessment of exposure approaches in air pollution and health research in Australia and New Zealand. 2011.
12. Shi L, Zanobetti A, Kloog I, Coull BA, Koutrakis P, Melly SJ, et al. Low-Concentration PM2.5 and Mortality: Estimating Acute and Chronic Effects in a Population-Based Study. Environmental health perspectives. 2015;124(1).
13. Liberati A, Altman DG, Tetzlaff J, Mulrow C, Gøtzsche PC, Ioannidis JP, et al. The PRISMA statement for reporting systematic reviews and meta-analyses of studies that evaluate health care interventions: explanation and elaboration. Journal of clinical epidemiology. 2009;62(10):e1-e34.
14. National Library of M. PubMed. US.: National Centre for Biotechnology Information US [Internet]. 1951.
15. Web of Science [Internet]. Institute for Scientific Information. 2001.
16. Wells G, Shea B, O'Connell D, Peterson J, Welch V, Losos M, et al. The Newcastle-Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in meta-analyses. Ottawa, Canada: Ottawa Hospital Research Institute. 2013.
17. Modesti PA, Reboldi G, Cappuccio FP, Agyemang C, Remuzzi G, Rapi S, et al. Panethnic differences in blood pressure in Europe: a systematic review and meta-analysis. PloS one. 2016;11(1):e0147601.
18. Mustafić H, Jabre P, Caussin C, Murad MH, Escolano S, Tafflet M, et al. Main air pollutants and myocardial infarction: a systematic review and meta-analysis. Jama. 2012;307(7):713-21.
19. Rooney AA, Boyles AL, Wolfe MS, Bucher JR, Thayer KA. Systematic review and evidence integration for literature-based environmental health science assessments. Environmental health perspectives. 2014;122(7):711-8.
20. Woodruff TJ, Sutton P. The Navigation Guide systematic review methodology: a rigorous and transparent method for translating environmental health science into better health outcomes. Environmental health perspectives. 2014;122(10):1007-14.
21. Zaza S, Wright-De Agüero LK, Briss PA, Truman BI, Hopkins DP, Hennessy MH, et al. Data collection instrument and procedure for systematic reviews in the Guide to Community Preventive Services. American journal of preventive medicine. 2000;18(1):44-74.
22. Hanigan IC, Rolfe MI, Knibbs LD, Salimi F, Cowie CT, Heyworth J, et al. All-cause mortality and long-term exposure to low level air pollution in the '45 and up study' cohort, Sydney, Australia, 2006-2015. Environment international. 2019;126:762-70.
23. Bowatte G, Erbas B, Lodge CJ, Knibbs LD, Gurrin LC, Marks GB, et al. Traffic-related air pollution exposure over a 5-year period is associated with increased risk of asthma and poor lung function in middle age. European Respiratory Journal. 2017;50(4).
24. Bowatte G, Lodge CJ, Knibbs LD, Erbas B, Perret JL, Jalaludin B, et al. Traffic related air pollution and development and persistence of asthma and low lung function. Environment international. 2018;113:170-6.
25. Salimi F, Morgan G, Rolfe M, Samoli E, Cowie CT, Hanigan I, et al. Long-term exposure to low concentrations of air pollutants and hospitalisation for respiratory diseases: A prospective cohort study in Australia. Environment international. 2018;121:415-20.

26. Knibbs LD, de Waterman AMC, Toelle BG, Guo YM, Denison L, Jalaludin B, et al. The Australian Child Health and Air Pollution Study (ACHAPS): A national population-based cross-sectional study of long-term exposure to outdoor air pollution, asthma, and lung function. Environment international. 2018;120:394-403.

27. Clifford S, Mazaheri M, Salimi F, Ezz WN, Yeganeh B, Low-Choy S, et al. Effects of exposure to ambient ultrafine particles on respiratory health and systemic inflammation in children. Environment international. 2018;114:167-80.

28. Haikerwal A, Akram M, Del Monaco A, Smith K, Sim MR, Meyer M, et al. Impact of Fine Particulate Matter (PM2.5) Exposure During Wildfires on Cardiovascular Health Outcomes. Journal of the American Heart Association. 2015;4(7).

29. Haikerwal A, Akram M, Sim MR, Meyer M, Abramson MJ, Dennekamp M. Fine particulate matter (PM2.5) exposure during a prolonged wildfire period and emergency department visits for asthma. Respirology (Carlton, Vic). 2016;21(1):88-94.

30. Huynh QL, Blizzard CL, Marwick TH, Negishi K. Association of ambient particulate matter with heart failure incidence and all-cause readmissions in Tasmania: an observational study. BMJ open. 2018;8(5).

31. Johnson AL, Dipnall JF, Dennekamp M, Williamson GJ, Gao CX, Carroll MTC, et al. Fine particulate matter exposure and medication dispensing during and after a coal mine fire: A time series analysis from the Hazelwood Health Study. Environmental Pollution. 2019;246:1027-35.

32. Tham R, Erbas B, Akram M, Dennekamp M, Abramson MJ. The impact of smoke on respiratory hospital outcomes during the 2002-2003 bushfire season, Victoria, Australia. Respirology (Carlton, Vic). 2009;14(1):69-75.

33. Barnett AG, Williams GM, Schwartz J, Best TL, Neller AH, Petroeschevsky AL, et al. The effects of air pollution on hospitalizations for cardiovascular disease in elderly people in Australian and New Zealand cities. Environmental health perspectives. 2006;114(7):1018-23.

34. Barnett AG, Williams GM, Schwartz J, Neller AH, Best TL, Petroeschevsky AL, et al. Air pollution and child respiratory health - A case-crossover study in Australia and new Zealand. American Journal of Respiratory and Critical Care Medicine. 2005;171(11):1272-8.

35. Melody SM, Ford J, Wills K, Venn A, Johnston FH. Maternal exposure to fine particulate matter from a coal mine fire and birth outcomes in Victoria, Australia. Environment international. 2019;127:233-42.

36. Johnston FH, Kavanagh AM, Bowman D, Scott RK. Exposure to bushfire smoke and asthma: an ecological study. Medical Journal of Australia. 2002;176(11):535-+.

37. Johnston FH, Bailie RS, Pilotto LS, Hanigan IC. Ambient biomass smoke and cardio-respiratory hospital admissions in Darwin, Australia. BMC public health. 2007;7:240.

38. Hanigan IC, Johnston FH, Morgan GG. Vegetation fire smoke, indigenous status and cardio-respiratory hospital admissions in Darwin, Australia, 1996-2005: a time-series study. Environmental health : a global access science source. 2008;7:42.

39. Crabbe H. Risk of respiratory and cardiovascular hospitalisation with exposure to bushfire particulates: new evidence from Darwin, Australia. Environmental geochemistry and health. 2012;34(6):697-709.

40. Johnston FH, Hanigan IC, Henderson SB, Morgan GG. Evaluation of interventions to reduce air pollution from biomass smoke on mortality in Launceston, Australia: retrospective analysis of daily mortality, 1994-2007. BMJ (Clinical research ed). 2013;346:e8446.

41. Meszaros D, Markos J, FitzGerald DG, Walters EH, Wood-Baker R. An observational study of PM10 and hospital admissions for acute exacerbations of chronic respiratory disease in Tasmania, Australia 1992-2002. Bmj Open Respiratory Research. 2015;2(1).

42. Bowatte G, Lodge CJ, Knibbs LD, Lowe AJ, Erbas B, Dennekamp M, et al. Traffic-related air pollution exposure is associated with allergic sensitization, asthma, and poor lung function in middle age. The Journal of allergy and clinical immunology. 2017;139(1):122-9.e1.

43. Lazarevic N, Dobson AJ, Barnett AG, Knibbs LD. Long-term ambient air pollution exposure and self-reported morbidity in the Australian Longitudinal Study on Women's Health: a cross-sectional study. BMJ open. 2015;5(10):e008714.

44. Lai VWY, Bowatte G, Knibbs LD, Rangamuwa K, Young A, Dharmage S, et al. Residential NO2 exposure is associated with urgent healthcare use in a thunderstorm asthma cohort. Asia Pacific Allergy. 2018;8(4).

45. Wang XY, Hu W, Tong S. Long-term exposure to gaseous air pollutants and cardio-respiratory mortality in Brisbane, Australia. Geospatial health. 2009;3(2):257-63.

46. Jalaludin BB, O'Toole BI, Leeder SR. Acute effects of urban ambient air pollution on respiratory symptoms, asthma medication use, and doctor visits for asthma in a cohort of Australian children. Environ Res. 2004;95(1):32-42.

47. Jalaludin B, Khalaj B, Sheppeard V, Morgan G. Air pollution and ED visits for asthma in Australian children: a case-crossover analysis. International archives of occupational and environmental health. 2008;81(8):967-74.

48. Morgan G, Corbett S, Wlodarczyk J. Air pollution and hospital admissions in Sydney, Australia, 1990 to 1994. American journal of public health. 1998;88(12):1761-6.

49. Petroeschevsky A, Simpson RW, Thalib L, Rutherford S. Associations between outdoor air pollution and hospital admissions in Brisbane, Australia. Archives of environmental health. 2001;56(1):37-52.

50. Morgan G, Sheppeard V, Khalaj B, Ayyar A, Lincoln D, Jalaludin B, et al. Effects of bushfire smoke on daily mortality and hospital admissions in Sydney, Australia. Epidemiology (Cambridge, Mass). 2010;21(1):47-55.

51. Dennekamp M, Akram M, Abramson MJ, Tonkin A, Sim MR, Fridman M, et al. Outdoor air pollution as a trigger for out-of-hospital cardiac arrests. Epidemiology (Cambridge, Mass). 2010;21(4):494-500.

52. Simpson R, Denison L, Petroeschevsky A, Thalib L, Williams G. Effects of ambient particle pollution on daily mortality in Melbourne, 1991-1996. Journal of exposure analysis and environmental epidemiology. 2000;10(5):488-96.

53. Simpson R, Williams G, Petroeschevsky A, Best T, Morgan G, Denison L, et al. The short-term effects of air pollution on hospital admissions in four Australian cities. Australian and New Zealand journal of public health. 2005;29(3):213-21.

54. Simpson R, Williams G, Petroeschevsky A, Best T, Morgan G, Denison L, et al. The short-term effects of air pollution on daily mortality in four Australian cities. Australian and New Zealand journal of public health. 2005;29(3):205-12.

55. Chen K, Glonek G, Hansen A, Williams S, Tuke J, Salter A, et al. The effects of air pollution on asthma hospital admissions in Adelaide, South Australia, 2003-2013: time-series and case-crossover analyses. Clinical and experimental allergy : journal of the British Society for Allergy and Clinical Immunology. 2016;46(11):1416-30.

56. Erbas B, Hyndman RJ. Sensitivity of the estimated air pollution-respiratory admissions relationship to statistical model choice. International journal of environmental health research. 2005;15(6):437-48.
57. Rutherford S, Clark E, McTainsh G, Simpson R, Mitchell C. Characteristics of rural dust events shown to impact on asthma severity in Brisbane, Australia. International journal of biometeorology. 1999;42(4):217-25.
58. Smith MA, Jalaludin B, Byles JE, Lim L, Leeder SR. Asthma presentations to emergency departments in western Sydney during the January 1994 Bushfires. International journal of epidemiology. 1996;25(6):1227-36.
59. Hunt TB, Holman CD. Asthma hospitalisation in relation to sulphur dioxide atmospheric contamination in the Kwinana industrial area of Western Australia. Community health studies. 1987;11(3):197-201.
60. Chen L, Verrall K, Tong S. Air particulate pollution due to bushfires and respiratory hospital admissions in Brisbane, Australia. International journal of environmental health research. 2006;16(3):181-91.
61. Salimi F, Henderson SB, Morgan GG, Jalaludin B, Johnston FH. Ambient particulate matter, landscape fire smoke, and emergency ambulance dispatches in Sydney, Australia. Environment international. 2017;99:208-12.
62. Johnston FH, Purdie S, Jalaludin B, Martin KL, Henderson SB, Morgan GG. Air pollution events from forest fires and emergency department attendances in Sydney, Australia 1996-2007: a case-crossover analysis. Environmental health : a global access science source. 2014;13:105.
63. Johnston FH, Salimi F, Williamson GJ, Henderson SB, Yao JY, Dennekamp M, et al. Ambient Particulate Matter and Paramedic Assessments of Acute Diabetic, Cardiovascular, and Respiratory Conditions. Epidemiology (Cambridge, Mass). 2019;30(1):11-9.
64. Xu Z, Hu W, Williams G, Clements AC, Kan H, Tong S. Air pollution, temperature and pediatric influenza in Brisbane, Australia. Environment international. 2013;59:384-8.
65. Morgan GE, Corbett S, Wlodarczyk J, Lewis P. Air pollution and daily mortality in Sydney, Australia, 1989 through 1993. American journal of public health. 1998;88(5):759-64.
66. Dennekamp M, Straney LD, Erbas B, Abramson MJ, Keywood M, Smith K, et al. Forest Fire Smoke Exposures and Out-of-Hospital Cardiac Arrests in Melbourne, Australia: A Case-Crossover Study. Environmental health perspectives. 2015;123(10):959-64.
67. Johnston F, Hanigan I, Henderson S, Morgan G, Bowman D. Extreme air pollution events from bushfires and dust storms and their association with mortality in Sydney, Australia 1994-2007. Environ Res. 2011;111(6):811-6.
68. Hu W, Mengersen K, McMichael A, Tong S. Temperature, air pollution and total mortality during summers in Sydney, 1994-2004. International journal of biometeorology. 2008;52(7):689-96.
69. Hansen C. Ambient Air Pollution and Congenital Anomalies in Brisbane, Australia: Should we be Concerned? Environmental Health. 2006;6(2):11.
70. Hansen CA, Barnett AG, Jalaludin BB, Morgan GG. Ambient air pollution and birth defects in brisbane, australia. PloS one. 2009;4(4):e5408.
71. Jalaludin B, Mannes T, Morgan G, Lincoln D, Sheppeard V, Corbett S. Impact of ambient air pollution on gestational age is modified by season in Sydney, Australia. Environmental Health. 2007;6.

72. Hansen C, Neller A, Williams G, Simpson R. Low levels of ambient air pollution during pregnancy and fetal growth among term neonates in Brisbane, Australia. Environmental Research. 2007;103(3):383-9.
73. Mannes T, Jalaludin B, Morgan G, Lincoln D, Sheppeard V, Corbett S. Impact of ambient air pollution on birth weight in Sydney, Australia. Occupational and environmental medicine. 2005;62(8):524-30.
74. Pereira G, Cook AG, Haggar F, Bower C, Nassar N. Locally derived traffic-related air pollution and fetal growth restriction: a retrospective cohort study. Occupational and environmental medicine. 2012;69(11):815-22.
75. Jalaludin B, Morgan G, Lincoln D, Sheppeard V, Simpson R, Corbett S. Associations between ambient air pollution and daily emergency department attendances for cardiovascular disease in the elderly (65+ years), Sydney, Australia. Journal of exposure science & environmental epidemiology. 2006;16(3):225-37.
76. Hinwood AL, De Klerk N, Rodriguez C, Jacoby P, Runnion T, Rye P, et al. The relationship between changes in daily air pollution and hospitalizations in Perth, Australia 1992-1998: a case-crossover study. International journal of environmental health research. 2006;16(1):27-46.
77. Pereira G, Cook A, de Vos A, Holman CDJ. A case-crossover analysis of traffic-related air pollution and emergency department presentations for asthma in Perth, Western Australia. Medical Journal of Australia. 2010;193(9):511-4.
78. Simpson RW, Williams G, Petroeschevsky A, Morgan G, Rutherford S. Associations between outdoor air pollution and daily mortality in Brisbane, Australia. Archives of environmental health. 1997;52(6):442-54.
79. Hansen A, Bi P, Nitschke M, Pisaniello D, Ryan P, Sullivan T, et al. Particulate air pollution and cardiorespiratory hospital admissions in a temperate Australian city: A case-crossover analysis. The Science of the total environment. 2012;416:48-52.
80. Patel D, Jian L, Xiao JG, Jansz J, Yun G, Robertson A. Joint effect of heatwaves and air quality on emergency department attendances for vulnerable population in Perth, Western Australia, 2006 to 2015. Environmental Research. 2019;174:80-7.
81. Jalaludin BB, Chey T, O'Toole BI, Smith WT, Capon AG, Leeder SR. Acute effects of low levels of ambient ozone on peak expiratory flow rate in a cohort of Australian children. International journal of epidemiology. 2000;29(3):549-57.
82. Straney L, Finn J, Dennekamp M, Bremner A, Tonkin A, Jacobs I. Evaluating the impact of air pollution on the incidence of out-of-hospital cardiac arrest in the Perth Metropolitan Region: 2000-2010. Journal of epidemiology and community health. 2014;68(1):6-12.
83. Krewski D, Jerrett M, Burnett RT, Ma R, Hughes E, Shi Y, et al. Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality: Health Effects Institute Boston, MA; 2009.
84. Capon A, Wright J. An Australian incremental guideline for particulate matter (PM2. 5) to assist in development and planning decisions. Public Health Research & Practice. 2019;29(4).
85. Chen G, Guo Y, Abramson MJ, Williams G, Li S. Exposure to low concentrations of air pollutants and adverse birth outcomes in Brisbane, Australia, 2003-2013. Science of the Total Environment. 2018;622:721-6.
86. Li SS, Guo YM, Williams G. Acute Impact of Hourly Ambient Air Pollution on Preterm Birth. Environmental health perspectives. 2016;124(10):1623-9.

87. Pereira G, Nassar N, Cook A, Bower C. Traffic emissions are associated with reduced fetal growth in areas of Perth, Western Australia: an application of the AusRoads dispersion model. Australian and New Zealand journal of public health. 2011;35(5):451-8.
88. The National Pollutant Inventory [Internet]. 2019 [cited 1st December 2019].
89. Higginbotham N, Freeman S, Connor L, Albrecht G. Environmental injustice and air pollution in coal affected communities, Hunter Valley, Australia. Health and Place. 2010;16(2):259-66.

Abstract

Background: Ambient (outdoor) air pollution is a key risk factor for health, for which effective policy plays an important preventative role. Australian federal and related state air quality standards have historically relied on international evidence for guidance, which may not accurately reflect the Australian context. There has been, however, a large increase in Australian epidemiological studies over recent years.

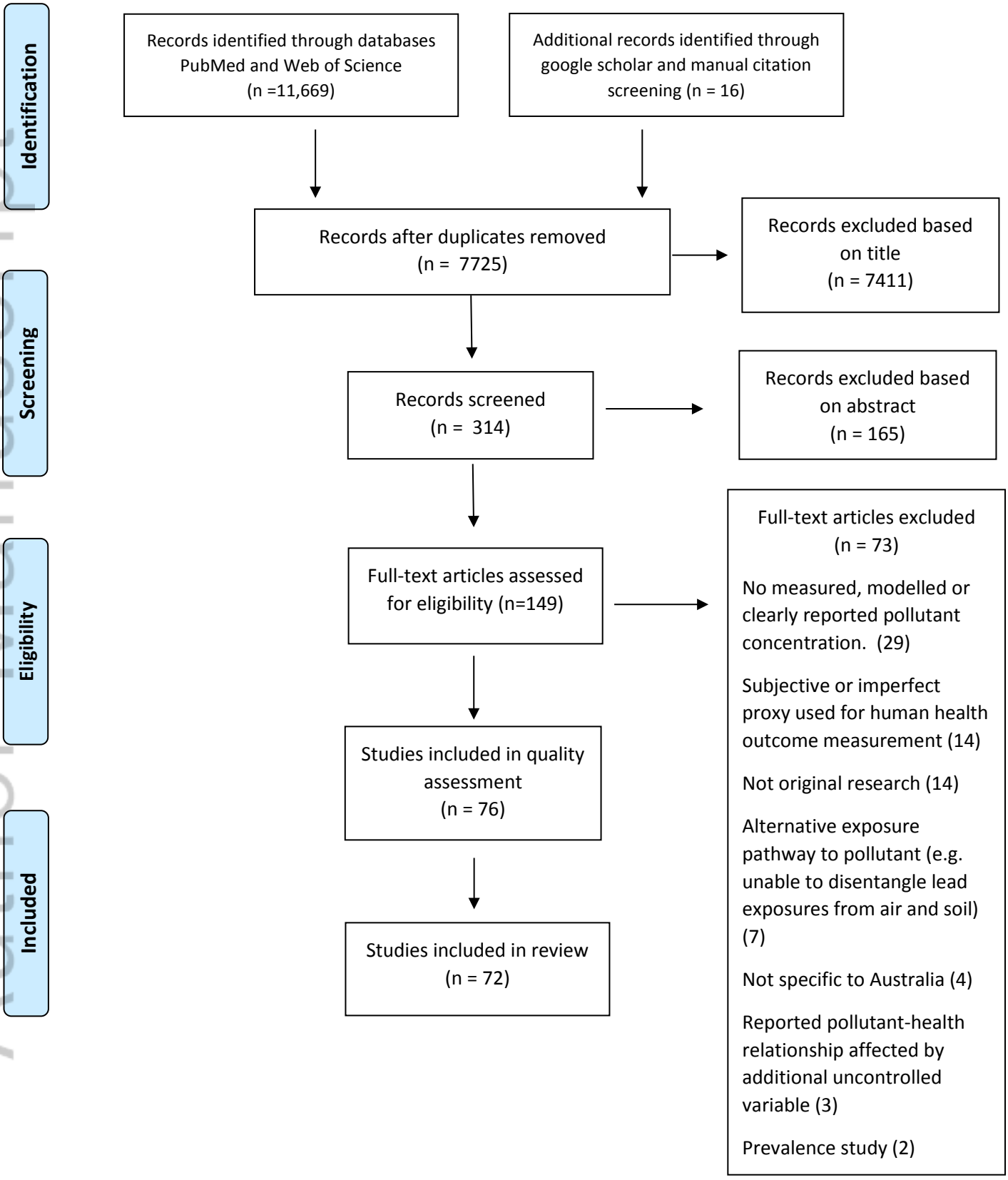
Aims: To provide an updated systematic literature review of peer-reviewed epidemiological studies that examined the health impacts of outdoor air pollution in Australia, including short- and long-term exposure.

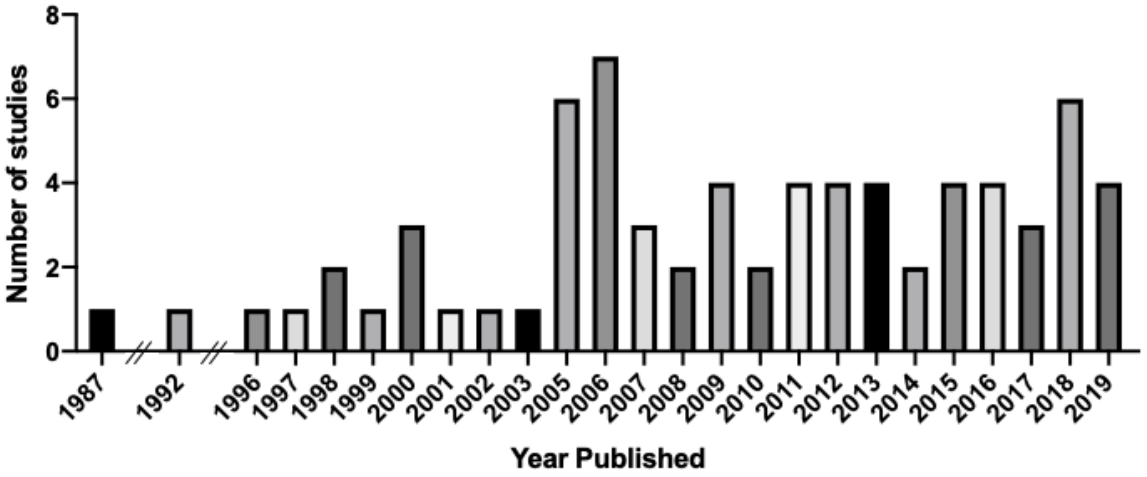
Methods: Following PRISMA guidelines, we conducted a systematic literature review. Broad search terms were applied to two databases (PubMed and Web of Science) and Google Scholar. Quality assessment and risk of bias were assessed using standard metrics. Included studies were summarised by tabulating key study characteristics, grouped by health outcomes.

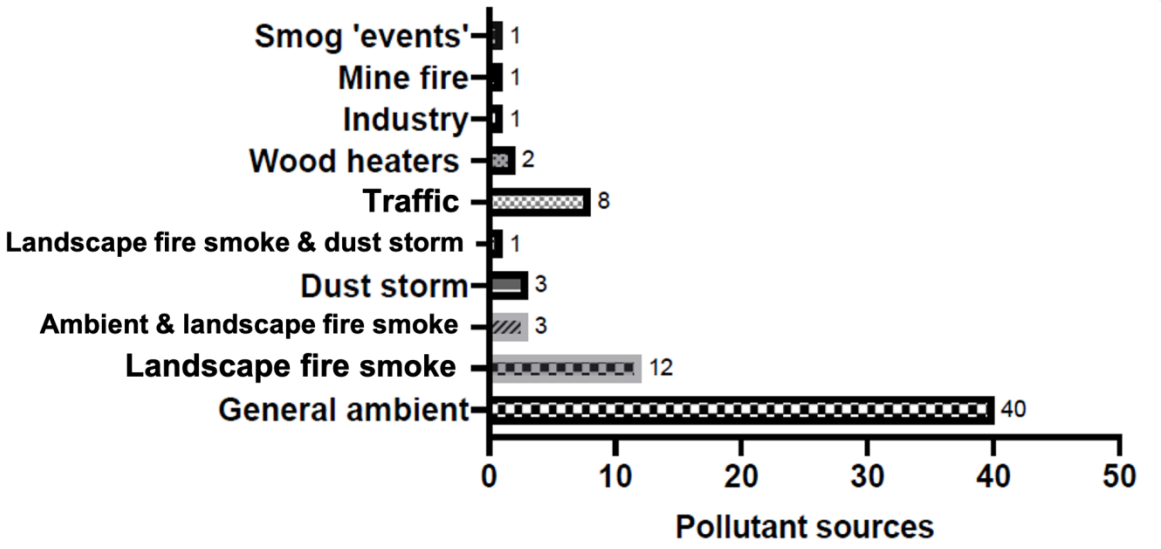
Results: In total, 72 studies were included in the review. Sixty-four studies (89%) used daily or hourly pollutant concentrations to examine short-term exposure impacts, of which 59 (92%) revealed significant associations with one or more health outcomes, including cardio-respiratory, all-cause mortality or morbidity, and birth outcomes. Eight studies (11%) used annual average pollutant concentrations to investigate long-term exposure finding significant associations with asthma, reduced lung function, atopy and cardio-respiratory mortality across five studies. The remaining three studies found no significant association with asthma, mortality and a range of self-reported diseases, respectively.

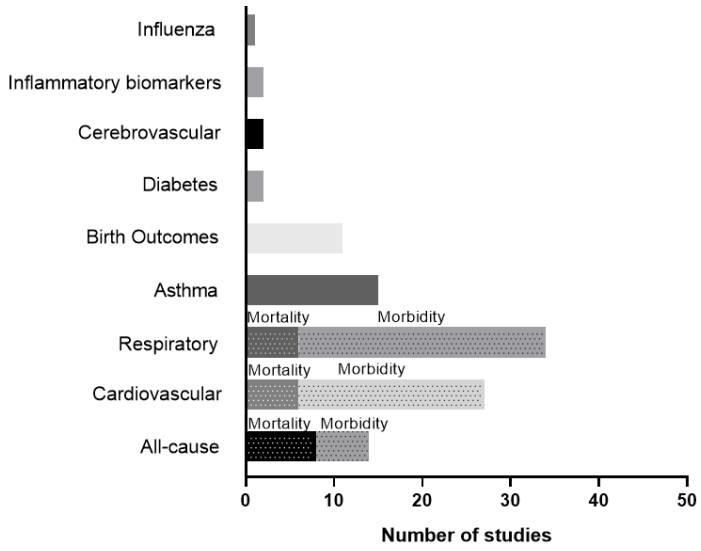
Conclusions: Ambient air pollution has substantial health impacts in Australia. The body of domestic evidence has increased markedly since national air quality standards were first set in the 1990s, which could be drawn on by policy-makers when revising the existing standards, or considering new standards.

Figure 1. Study selection









The health impacts of ambient air pollution in Australia: A systematic literature review

Clare M. Walter,[1,2,3] Elena K. Schneider-Futschik[2], Nina L. Hall[1,] Peter D Sly[4],Brian W. Head[5] and Luke D. Knibbs[1]

[1]School of Public Health, Faculty of Medicine, The University of Queensland, Brisbane, QLD, Australia
[2]Department of Pharmacology and Therapeutics, School of Biomedical Sciences, Faculty of Medicine, Dentistry and Health Sciences, The University of Melbourne, Melbourne, VIC, Australia
[3]Australian-German Climate and Energy College, The University of Melbourne, Melbourne, VIC, Australia
[4]Children's Health and Environment Program, Child Health Research Centre, The University of Queensland, South Brisbane, Qld, Australia.
[5]School of Political Science and Centre for Policy Futures, The University of Queensland.

c.walter@uq.edu.au, clarem.walter@gmail.com