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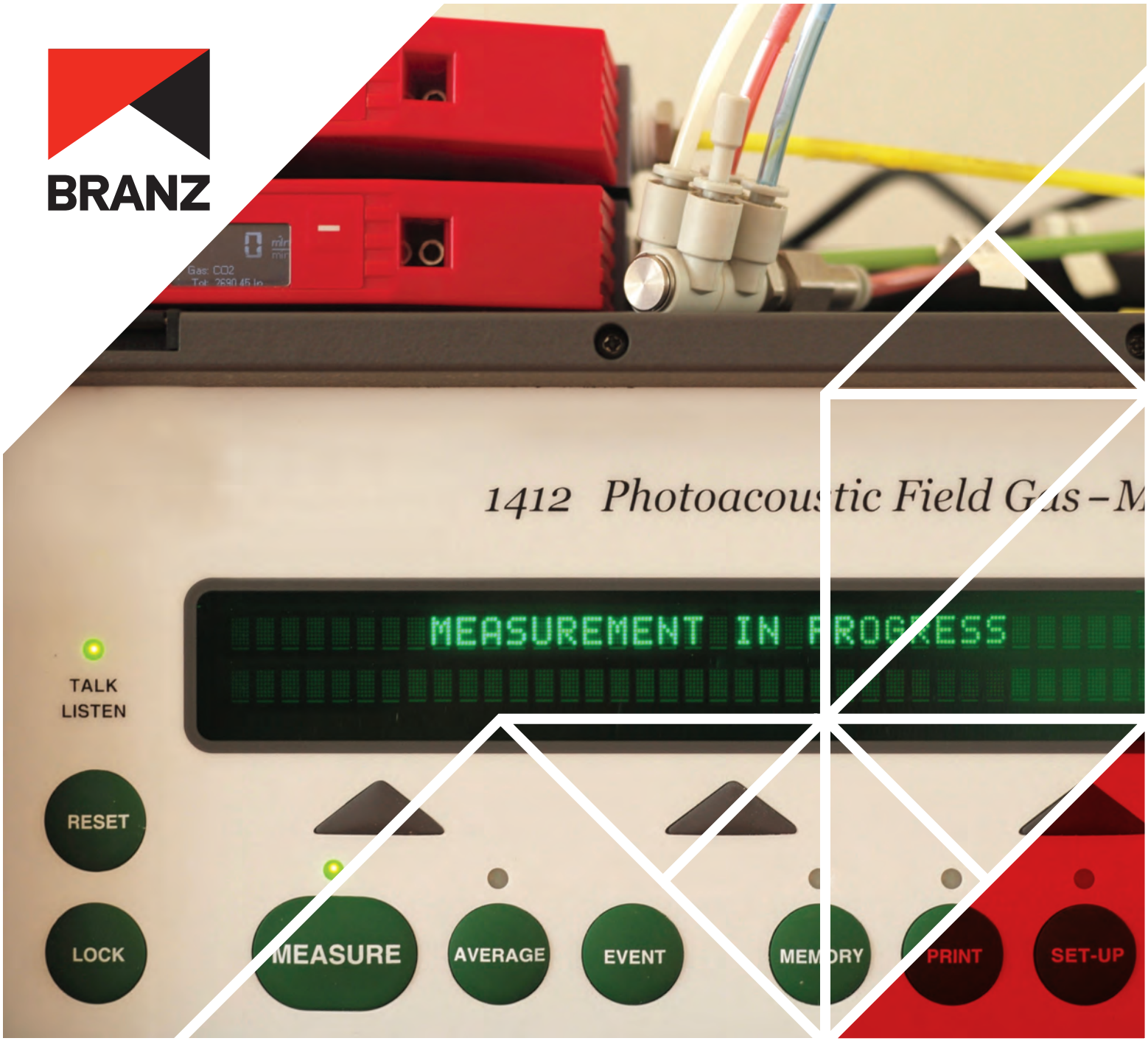

Indoor Air Quality in New Zealand Homes and Schools

A literature review of healthy homes and schools with emphasis on the issues pertinent to New Zealand.

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Contents

List of abbreviations

ACC	Accident Compensation Corporation
ach	air changes per hour
AFRs	alternative flame retardants
ARF	acute rheumatic fever
BFR	brominated flame retardants
BRI	building-related illness
BTEX	benzene, toluene, ethyl benzene, orthe-xylene and meta and para-xylene group
CO	carbon monoxide
CO_2	carbon dioxide
COPD	chronic obstructive pulmonary disease
CPI	Consumers Price Index
DDE	dichlorodiphenyldichloroethylene
DDT	dichlorodiphenyltrichloroethane
EDC	endocrine-disrupting chemicals
ETS	environmental tobacco smoke
HAPINZ	Health and Air Pollution in New Zealand
HEEP	Household Energy End-use Project
HNZC	Housing New Zealand Corporation
HPV	high production volume
HR-PAH	housing-related potentially avoidable hospitalisation
IARC	International Agency for Research on Cancer
IVF	in vitro fertilisation
MFE	Ministry for the Environment
mg/d	milligrams per day
MOH	Ministry of Health
MVOCs	microbial volatile organic compounds
NO_2	nitrogen dioxide
NO_3	nitrate radicals
NOx	nitrogen oxides
OCPs	organochlorine pesticides
OECD	Organisation for Economic Co-operation and Development
OH	hydroxyl radicals
OPEC	Organization of the Petroleum Exporting Countries
OPPs	organophosphate pesticides
PAHs	polycyclic aromatic hydrocarbons
PBDEs	polybrominated diphenyl ethers
PCBs	polychlorinated biphenyls
PM	particulate matter
PM_{10}	particulate matter 10 microns or smaller
$PM_{2.5}$	particulate matter 2.5 microns or smaller
POM	polycyclic organic matter
POP	persistent organic pollutant
PPs	pyrethrin pesticides
PVC	polyvinyl chloride
QV	Quotable Value
RH	relative humidity
RHD	rheumatic heart disease
RIOPA	relationship of indoor, outdoor and personal air
SARS	severe acute respiratory syndrome
SBS	sick building syndrome
SES	socio-economic status
SHS	second-hand smoke
SO_2	sulphur dioxide
SUDI	sudden unexpected death in infancy
SVOCs	semi-volatile organic compounds
TB	tuberculosis
TVOCs	total volatile organic compounds
UFPs	ultrafine particles
USEPA	United States Environmental Protection Agency
UV	ultraviolet
VOCs	volatile organic compounds
WHO	World Health Organization

Introduction

How important is indoor air?

Most urban dwellers spend around 90% of their time indoors (Kostinen et al., 2008) either at home, work or in an educational facility. When air quality is poor, the exposure to pollutants is prolonged, and the occupant's health can be adversely affected.

Indoor air is generally more contaminated than outdoor air by the order of several magnitudes.

The United States Environmental Protection Agency (USEPA) has identified indoor air quality as one of the top five environmental hazards for the Western world. While the USEPA has a mandate to research and disseminate knowledge on indoor air quality, the same level of activity hasn't happened in New Zealand. It can be argued that New Zealand is lagging behind Europe and the USA on its indoor air quality research, public information and uptake programmes and policy.

The poor quality of many New Zealand homes is causal of some significant acute and chronic health issues. Low indoor temperatures and dampness are a common theme in these.

New Zealand is leading the developed world in some of the wrong statistics. New Zealand has:

- the second highest rate of asthma – asthma affects one in four children and costs $4 million per year
- the highest rate of hospitalisations from skin infections – this rate is double that of either Australia or USA and has doubled in the last decade
- one of the worst rates of rheumatic fever, which can be the result of streptococcus throat infections
- the highest rate of excess winter mortality in the Organisation for Economic Co-operation and Development (OECD)
- a very high rate of chronic obstructive pulmonary disease (COPD) with an onset at an average age of 55 years old for Māori and 65 years old for Pākehā
- a high rate of fuel poverty, which is increasing as fuel prices are rising faster than incomes.

Poor indoor air quality of housing, and possibly schools and other settings, is implicated in these factors as discussed in later sections of this review.

One a more positive note, there is robust evidence to show that small improvements in housing have significant health benefits. Installing insulation in ceiling and floor cavities reduces hospitalisations and doctor's visits. Replacing an unflued gas heater or small plug-in electric heater with a heat pump, flued gas heater or wood pellet burner improves asthma and respiratory infections. Moving from private sector to state housing reduces hospitalisations.

Indoor air is a combination of outdoor (ambient) pollutants and pollutants generated within the indoor environment. The outdoor air can be polluted with vehicle emissions, wood smoke, fungi spores and emissions from industrial processes. Ambient air quality is routinely monitored in many New Zealand towns and all cities. Sources are well documented in other forums, including the Ministry for the Environment publications and the HAPINZ study (Kuschel et al., 2012), and are only discussed briefly in this report in the context of outdoor pollutants infiltrating indoor environments.

Indoor pollutants can include chemical emissions such as;

- volatile organic compounds (VOCs) emitted from building materials, finishes and furnishing, such as formaldehyde and benzene
- microbiological contaminants, such as fungi, bacteria and dust mites and their byproducts endotoxins, mycotoxins and beta-glucans
- respirable particulate matter (PM) from wood smoke, vehicle emissions, environmental tobacco smoke (ETS), fungi spores and pollen
- gaseous pollutants such as nitrogen dioxide (NO_2), carbon monoxide (CO) from combustion processes and ozone (O_3).

Moisture is a precursor to high levels of certain indoor pollutants. It will enable fungi and dust mites to grow and will increase the emissions of VOCs from building materials. As such, it can be considered as a contaminant and is discussed later in this report. Carbon dioxide (CO_2) is rarely found at levels that are of risk to health in buildings. However, it is frequently used as a surrogate measure of ventilation effectiveness. The ventilation rate is very important as it affects the dilution of indoor-generated pollutants and moisture as well as the ingress of ambient pollutants into the indoor space.

The home environment is particularly important as most people spend the greatest amount of their time at home, and this is particularly so for vulnerable people such as the very old and the very young.

The early childhood and school environments are also of particular concern as this is where vulnerable children spend the second greatest portion of their time. It has been estimated that many preschoolers are spending more hours of their life in a preschool centre than they do in their home. These environments are drastically under-researched in New Zealand.

In the last 100 years, more than 100,000 new chemicals have been introduced into the environment, many of which have subsequently been shown to be toxic to animals, and some are also harmful to humans (Shu et al., 2014). Three thousand of these 'new' chemicals are known as high production volume (HPV) chemicals. A large proportion of HPVs are incorporated into furniture, building materials and finishes, computers, carpets, perfumes and even food. Less than half of the new

HVP chemicals have been studied in relation to health effects, and less than 8% have had the developmental effects on infants and children investigated (Gorini et al., 2014).

Until recently, regulations on air quality have primarily focused on the air outdoors, but there is increasing effort internationally to quantify and regulate indoor air emissions (Kostinen et al., 2008). This work has progressed rapidly over the past 30 years since the importance of indoor air quality on health has become apparent. However, there are still significant knowledge gaps, and the research and translation into appropriate policy is lagging in New Zealand.

Research on indoor air quality is hampered by complexity. People are frequently simultaneously exposed to a cocktail of thousands of pollutants, often at low doses. The health effects of multiple exposures are unknown, and it is not possible to determine if the health effects from the many permutations of chemicals are additive or synergistic.

This paper is a review of recent research on indoor air quality that is relevant to New Zealand's indoor environments. Where possible, New Zealand research has been presented. Emphasis is placed on gaps in the knowledge in order to identify future research in New Zealand. This paper brings together multidisciplinary research and can provide a starting point for architects, designers, building scientists, property developers, policy makers, public health workers and epidemiologists to find information. For this reason, some of the information may be repeated under different headings.

Life is messy at the frontiers of knowledge. The challenges that we face as a research community in trying to acquire a clear understanding of the environmental health significance of indoor environmental quality is something like assembling a challenging jigsaw puzzle with no finished picture to guide us. (Nazaroff, 2010).

Part one – Health effects

Vulnerable populations

Populations, and indeed individual people, all react differently to air toxins and allergens. Just as a handful of peanuts can be a tasty snack for one person and a mere whiff of peanut odour can be fatal for another, air pollutants will have variable levels of effect on different individuals. A small proportion of people are hypersensitive to air pollution, such as those suffering from chemical hypersensitivity syndrome (also known as multiple chemical sensitivity) or sick building syndrome. Little is known about what causes these conditions (Levy, 2015), and research is difficult because of the non-specific symptoms that are also caused by numerous other conditions. However, it is possible that human rights legislation could be used to argue that buildings should be designed to accommodate people with the disability of chemical sensitivity in the same way design for wheelchair access and visual impairments is required in public buildings.

At the other end of the scale, epidemiologists have long been aware that people whose employment status is 'currently employed' are healthier overall than the general population. This is known as the healthy worker effect and is probably one of the factors contributing to why people in lower socio-economic brackets appear more vulnerable to most health issues, from infections to pollution effects. While being poor is likely to impact health, poor health is also likely to impact socio-economic status towards poverty. It is very difficult to untangle these two inter-related factors, however, it is becoming increasingly clear that poverty is causally related to poorer health (Hodas et al., 2012; Cushing et al., 2015; Brown et al., 2015).

In New Zealand, people of lower socio-economic status are more vulnerable to most health effects. This is known as health inequality. The socio-economic health disparities in New Zealand are particularly strongly pronounced, compared with other similar OECD nations (Howden-Chapman et al., 2011). New Zealand also has a gaping ethnic health disparity, with the burden of ill health disproportionately heavy on Māori and Pacific people (Hales et al., 2012).

Children

The quality of the air that children breathe requires particular attention. Children are considered most vulnerable as they have an underdeveloped or compromised immune system. For this reason, a large number of recent studies have focused on air quality in schools and daycare centres (Fuentes-Leonarte, Tenías & Ballester, 2009) and on the air being breathed by pregnant mothers (Ballester et al., 2010; Farmer et al., 2014). Lower birth weights associated with maternal exposure to many pollutants can relate to poor respiratory outcomes in later life (Farmer et al., 2014).

Children are enormously more vulnerable to air toxins than healthy adults. Children's lungs are larger in relation to their body size, meaning that pollutants can become more concentrated in their

systems (Fuentes-Leonarte, Ballester & Tenías, 2009). They are at particular risk because of multiple converging pathways of effect. Children also breathe more rapidly than adults, meaning that, relative to blood and body volume, children may actually respire more total toxins than adults (Fuentes-Leonarte, Ballester & Tenías, 2009; Esplugues et al., 2010b). Children's organs are still developing, and toxins can interfere with that development (Rauch et al., 2012). For example, human lungs are not fully developed until age 6 (Fuentes-Leonarte, Ballester & Tenías, 2009). Children, especially infants, also spend more time in their home than adults.

There is evidence that children in Western societies are spending less time outdoors. A study from 1984 showed that children spent 18% of time annually (29% in summer) outside (Letz, Ryan & Spengler, 1984), whereas a similar more recent study in southern California (Wu et al., 2007) used activity data for female primary school children of less than 3% annually outside (5% in summer). Such trends mean that indoor exposure has become more significantly important than outdoor exposure (Esplugues et al., 2010b).

As children spend more time at floor level, they are more susceptible to pollutants from or resuspended from flooring. This frequently includes fungi spores, pet allergens, pollen and dust mites and potentially lead and pesticide residues walked in from outdoors. Children are usually closer to the ground, increasing their exposure to those particles that shift between the gas and solid partitions and collect in dust, such as polycyclic aromatic hydrocarbons (PAHs), phthalates and pesticides (Frederiksen et al., 2009; Roca et al., 2014). Children's dust ingestion has been shown to be significantly greater in volume to adults. The daily intake of dust has been estimated to be 100–200 milligrams per day (mg/d) for small children (aged 1–4 years), while the intake for adults is estimated to be around 50 mg/d (Ozkaynak et al., 2011; Frederiksen et al., 2009). Children's play activities also tend to resuspend settled particulate matter (PM), so they expose themselves to a personal cloud of PM.

Compounding the situation further, Telfar-Barnard found in a New Zealand population-based study that those aged under 5 lived in the worst condition housing, between 2000 and 2006, as rated by Quotable Value (QV) valuations and matched against census data, with the very youngest in the very worst condition housing (Telfar-Barnard, 2009)

Perinatal

The developing foetus may be at increased risk from toxic exposures because of its small size, rapid growth and relative inability to detoxify harmful substances (Rauch et al., 2012). Improved understanding of genetic and epigenetic heritance have shown that the father's exposures to environmental pollutants prior to conception and the mother's exposures during pregnancy can impact on the risk profiles for their children right through to adulthood (Gorini et al., 2014; Levy, 2015).

Studies have associated prenatal and perinatal exposures with several health effects including asthma (Clark et al., 2010; Shu et al., 2014), COPD, childhood cancers including neuroblastomas (Heck et al., 2013; Ghosh et al., 2013) and leukaemia (Houot et al., 2015) and neurodevelopmental deficits including autism (Levy, 2015) and attention deficit disorders (Verner et al., 2015; Hoffman et al., 2010).

Nursing infants are likely to experience increased exposure to persistent organic pollutants (POPs) such as polychlorinated biphenyls (PCBs) and dioxin, which, being lipophilic, collect in breast tissue and have been recorded at relatively high levels in New Zealand mothers' breast milk ('t Mannetje et al., 2014). One study has shown that nursing infants' body load of such lipophilic toxins as polybrominated flame retardants (BFRs) are higher than in their mothers (Lunder et al., 2010).

In New Zealand, infants have been found to live in the worst condition houses on average, and it is likely that this relationship would be similar for the pregnancy period also (Telfar-Barnard, 2009). New Zealand has the worst rate of sudden unexpected death in infancy (SUDI) in the industrialised world. This is especially prevalent in Māori families. It is established that exposure to tobacco smoke and bed sharing with adults are risk factors for SUDI. Research hasn't been conducted to investigate other housing-related factors such as low temperature or dampness and SUDI.

Elderly

Ageing leads to decreased immune and lung functioning and predisposition to respiratory infections (Bentayeb et al., 2015). Older people are also more vulnerable to health effects caused by cold and damp living conditions (Howden-Chapman, Signal & Crane, 1999). Poor circulation and arthritis may mean elderly people suffer more in cold temperatures than the general population.

It is estimated that, by 2030, people over 60 years of age will make up 25% of New Zealand's population (Howden-Chapman, Signal & Crane, 1999). With fuel prices rising faster than the Consumers Price Index (CPI), fuel poverty will affect a growing proportion of society every year. However, retirees and others who are on fixed incomes will be hardest hit by rising heating costs. Elderly people feel the cold more, as their circulation can be compromised and they are frequently less active. As the ageing population increases, keeping elderly people healthy by minimising chronic illness will be a priority for New Zealand's health system. It is much more cost-effective for the state and less disruptive for the ageing population for them the live in self-care housing (or ageing in place as it is sometimes known) for as long as possible.

Part of the solution for ageing well necessitates a better understanding of environmental exposures. Few studies have measured indoor air pollution and/or its health effects in elderly people's own housing or residential care homes, especially in New Zealand.

One recent study, which measured NO_2, ozone, PM and formaldehyde in eight nursing homes across Europe, found that, although pollutant levels were moderate and generally below guideline levels,

dose response relationships were found between pollutant concentrations and respiratory function, and this effect increased with increasing age of the participant (Bentayeb et al., 2015).

An investigation of 16 local government state single-bedroom units in Palmerston North found low heater use and low overnight temperatures due to occupants budgeting energy use (Phipps et al., 2014).

Low socio-economic status and ethnic minorities

A number of studies have examined the relationship between air pollution, socio-economic status (SES) and mortality (Laurent et al., 2007; Naess et al., 2007; Pelucchi et al., 2009) or health effects (Morello-Frosch, Pastor & Sadd, 2001; Rauch et al., 2012). There has been general agreement within such research, showing that the ill effects of air pollution are more pronounced for those of lower SES. The two mechanisms that have been hypothesised to account for this are that people of lower SES are exposed to higher levels of pollution and have more co-exposures.

There is substantial evidence to support both these hypotheses (Naess et al., 2007; Hodas et al., 2012; Cushing et al., 2015; Semmens et al., 2015).

A recent exposure study looking at this hypothesis in California, USA, found the odds of living in the suburbs with the highest ambient outdoor pollution were 6.2, 5.8, 1.9, 1.8 and 1.6 times greater for Hispanics, African Americans, Native Americans, Asian/Pacific Islanders and other or multiracial individuals, respectively, than for non-Hispanic whites. Race was a better predictor of exposure than income, with pesticide use and toxic chemical releases being the most unequal (Cushing et al., 2015). Another study from California showed that non-white residents had increased risk of cancer, also related to the exposure profiles of different residential zones (Morello-Frosch, Pastor & Sadd, 2001). Again, the authors showed that, for each income bracket, white citizens had lower exposure profiles, suggesting a double burden of being poor and of minority ethnicity. Another USA study measuring the relationship between organophosphate pesticide exposure and gestational age and weight found the black mothers in the study had higher levels of urinary pesticide metabolites (Rauch et al., 2012). They also hypothesised that black and white mothers in their study may have been differentially exposed. A French study found lower socio-economic families had higher indoor concentrations of formaldehyde than those in higher brackets but lower perchloroethylene (Brown et al., 2015). A study of 96 rural USA homes using wood burners for heating found that lower income was associated with not only higher $PM_{2.5}$ but with a greater proportion of the smallest size fraction, which also poses the greatest health risks (Semmens et al., 2015).

Although exposure differentiation by socio-economic status or ethnicity has not been measured directly in New Zealand, there is evidence to suggest that similar patterns exist here. For example, in New Zealand, a cohort study examining the effects of outdoor air pollution on mortality in Christchurch showed premature mortality in Māori significantly higher than other ethnicities (Hales et al., 2012). While every 10 g/m^3 increase in PM_{10} increased premature mortality by 7% in non-Māori,

the figure was 20% for the same increase in Māori. This result is based on a small number of Māori included in the study. However, the wide disparity is worthy of further research.

Many studies have also found socio-economic and ethnic relationships between common respiratory allergens such as from dust mites, cockroaches and mice (Camacho-Rivera et al., 2014) and mould (Sharpe, Thornton & Osborne, 2014). While the reason for people of lower socio-economic status to be differentially exposed to common indoor and outdoor toxins is not clear (Cushing et al., 2015; Morello-Frosch, Pastor & Sadd, 2001), it is likely a combination of individual behaviours and societal behaviours contribute.

Level of education has been implicated as a risk for increased exposure to some allergens (Camacho-Rivera et al., 2014). A further study examining this relationship found those of lower socio-economic status had increased exposure regardless of their risk perception and ventilation habits (Sharpe et al., 2014). Examination of societal behaviours in Los Angeles found that industries that emit toxins into air are more often situated closer to poorer neighbourhoods and those with more minority ethnic inhabitants (Cushing et al., 2015).

One pollutant that has been clearly demonstrated to be differentiated by ethnicity is tobacco smoke (Ministry of Health, 2014). This has also been linked with adverse health outcomes for exposed children (Trenholm et al., 2012). While recent methods have been highly effective in reducing smoking rates in the population as a whole, certain sectors, including younger Māori women, have been little affected (Ministry of Health, 2014) (see Environmental tobacco smoke).

In terms of dampness-mediated allergens (from mould, cockroaches and dust mites), it is likely that indoor temperature plays a significant role. The producers of these allergens all thrive in an environment with a relative humidity above 60%. A study on housing insulation and health found that retrofitting a small amount of insulation to the subfloor and ceiling of uninsulated New Zealand homes increased the average winter indoor temperature by 1°C, and potentially more importantly, it reduced the exposure to relative humidity above 60% RH by 1.5 hours per day (Howden-Chapman et al., 2008).

A large cohort study that compared the hospitalisation records and housing status for Housing New Zealand Corporation (HNZC) tenants and applicants confirmed that placement of housing applicants into social housing was associated with a significant drop in hospitalisation rates for many health outcomes (Baker et al., 2010). The data suggested that stable social housing contributed to short-term health improvements for this vulnerable population. Applicants tended to have poorer health whilst they were on the HNZC waiting list, with acute hospitalisation rates dropping within their first 6 months as a HNZC tenant. Importantly, housing-related potentially avoidable hospitalisation (HR-PAH) dropped significantly over the following 2 years for long-term tenants but not for unsuccessful applicants. Hospitalisation rates for HNZC tenants dropped by 13.6% for close-contact infectious diseases and 13.1% for circulatory and respiratory diseases.

The concept of fuel poverty is clearly an important mediator in a number of health outcomes, but defining fuel poverty poses some difficulties. An early and frequently used UK definition is that a household that needs to spend more than 10% of its income on heating is living in fuel poverty. A more recent English definition pertains to a low-income, high-costs model, where a household is fuel poor if the energy required to heat their home to the World Health Organization (WHO)-defined minimum of 18°C would leave the household with an income below the poverty line (60%) median (Department of Energy and Climate Change, 2013). This is supported by the observation that low-income households have the triple whammy of the lowest-quality housing, they may be unable to afford the capital cost of an efficient heater and they may be on electricity prepay, which typically has a higher unit rate than paying on account, or use an unflued gas heater, which is an inefficient and expensive prepay option.

Evidence from New Zealand shows that many families that are experiencing fuel poverty self-ration heating (O'Sullivan, Howden-Chapman, & Fougere , 2011) to the extent that they choose between heating or eating. Estimates of the extent of fuel poverty vary. O'Sullivan has estimated this to affect 25% of New Zealand households (O'Sullivan, Howden-Chapman, & Fougere, 2015) with over 60% of respondents to a recent survey, who were using prepay electricity, reporting they shivered while in their home. An assessment prepared for the Ministry of Social Development calculated that 10–14% of New Zealanders live in fuel poverty according to the earlier UK definition, but the figure may be as high as 30% in Dunedin (Lloyd, 2006). Lloyd has stated that fuel poverty has increased since his 2006 report as fuel prices have risen faster than inflation and incomes. The Household Energy End-use Project (HEEP) study found that many families, regardless of income level, do not heat their home to WHO guidelines (Isaacs et al., 2010). Bedrooms are especially too cold, with overnight temperatures down to that of a fridge being measured in many homes.

The second hypothesised reason for a greater proportion of health effect from air pollution in poorer populations is that other co-exposures such as poorer diet, increased stress and higher levels of smoking cause the relationship observed (Laurent et al., 2007).

Both mechanisms are plausible and likely. Households with low incomes have fewer opportunities to improve their housing. A lack of disposable income will constrain improvements for many owner occupiers, especially in Auckland and other high-cost housing areas. Those living in rental accommodation also face barriers. They risk losing their investment if they improve the rental property or may face an increase in rent if the landlord improves the property.

Research into co-exposures, co-morbidities, health outcomes and indoor air characteristics may be of particular value in New Zealand, where the health inequality gap is particularly wide (Hales et al., 2012; Howden-Chapman et al., 2012; Trenholm et al., 2012).

Health effects related to indoor air quality

Respiratory infections (viral)

The spread of viral infections, including influenza and severe acute respiratory syndrome (SARS), has become an important concern of modern societies. As urban populations become denser and people spend more time indoors, the likelihood of infections reaching epidemic proportions increases, despite better understanding of disease control and prevention.

The role of air transport in viral infections has received less attention in the scientific literature than other routes of exposure, such as direct contact (La Rosa et al., 2013; Nardell, 2015) or other sources of airborne biocontamination such as bacteria and fungi (Roy & Milton, 2004). Until recently, the focus in the literature was predominantly on the spread of contamination directly via droplets across short spaces or transferred via contaminated surfaces (Barker, Stevens & Bloomfield, 2001). During an outbreak of SARS that spread rapidly through an apartment building in Hong Kong in 2003, researchers demonstrated the importance of airborne transmission in the spread of SARS (Li et al., 2005). As a result of this information, some scientists are investigating airborne transmission of common viral contaminants, including rhinoviruses (La Rosa et al., 2013; Li et al., 2007) and even non-respiratory viruses. It has been demonstrated that both toilet flushing and vomiting can aerosolise viral contamination, leading to airborne transmission of non-respiratory (such as gastric) illnesses. This had previously been assumed to be an unlikely means of communication (La Rosa et al., 2013; Li et al., 2005; Nazaroff, 2014). This is leading to rethinking of methods of venting bathrooms and plumbing fixtures.

There are many factors regarding the airborne transmission of viruses that remain inconclusive. The frequency of transmission via air, compared to other pathways, is unclear (Yang, Elankumaran & Marr, 2011) and little is known about the survival period of viruses in the air (Chen et al., 2009; Myatt et al., 2010). The ventilation rate necessary to reduce risk in a given environment is likewise unclear (Li et al., 2007), but reduction has been assumed to be the square of the ventilation rate. Furthermore, very few studies have been conducted in residential settings (Barker et al., 2001; La Rosa et al., 2013). Contrary to survival of bacteria, virus survival in aerosol form and on surfaces is improved by low humidity levels (Myatt et al., 2010). One such study demonstrated the use of humidifiers in reducing viable influenza aerosols and showed a sharp drop-off in virus survival at relative humidity levels over 49%. Other studies have demonstrated lower viral transmission rates with increased humidity (Li et al., 2007). More research is required in this area, including the use of nanotechnology, ultraviolet (UV) light and other technologies to control bioaerosols.

Environmental pollutants, including particulate matter, nitrogen dioxide and tobacco smoke, are consistently associated with increased incidence of respiratory diseases, and it is likely that exposure to indoor and outdoor pollutants increases the susceptibility to viral infections (Zar & Ferkol, 2014).

Those under 5 years of age and over 65 are particularly vulnerable to contracting respiratory infections (Kessaram, Stanley & Baker, 2015).

In New Zealand, between 1999 and 2008, influenza was estimated to have caused between 306 and 401 deaths annually, 86% of which were in those aged over 65 years. Over that period, influenza was responsible for 22% of respiratory and circulatory deaths and 16% of all medical deaths in New Zealand (Kessaram et al., 2015). Another New Zealand study showed a wide ethnic disparity in hospitalisation with H1N1 flu in 2009, with Māori being hospitalised three times more frequently than non-Māori, and Pacific people nearly seven times as often (Baker et al., 2012).

Respiratory infections (bacterial)

New Zealand has unusually high rates of tuberculosis (TB), meningococcal disease, childhood pneumonia, rheumatic fever and skin infections (Baker et al., 2012; Jaine, Baker & Venugopal, 2011). Baker et al. noted that hospitalisations for these illnesses are disproportionately represented in low socio-economic, Māori and Pacific populations. These illnesses are also all associated with overcrowded living conditions (Baker et al., 2000). Acute rheumatic fever (ARF) is an autoimmune response to bacterial infection from *Streptococcus* A. In some children, ARF can lead to permanent damage to heart valves, a chronic health condition known as rheumatic heart disease (RHD) (Marijon et al., 2012). The specific mechanisms that cause some people to develop this immune response to a common bacterial infection remain elusive (Phillips & Osmond, 2014). ARF is almost exclusively a disease of poverty, with 97% of cases occurring in developing nations or in indigenous populations in developed nations worldwide (White et al., 2010). Epidemiology linking ARF with poverty and living conditions dates back to the 1930s (Phillips & Osmond, 2014), and numerous studies have linked ARF with overcrowded housing (Jaine et al., 2011).

Māori and Pacific people in New Zealand have some of the highest rates worldwide of ARF. While ARF rates for Pākehā New Zealanders are low and similar to other developed nations (Baker et al., 2012; Jaine, Baker & Venugopal, 2008), rates for Māori are 22 times that of Pākehā New Zealanders and rates for Pacific people living in New Zealand are 75 times as high. Acute rheumatic fever rates have become more ethnically differentiated since 1980, as non-Māori/Pacific rates have decreased while the incidence rate for those groups has increased, leading to overall similar rates at the population level (Baker et al., 2012; Jaine et al., 2011). Risks for indigenous people living in Pacific Islands may be significantly higher again (Dobson et al., 2012). There is a strong relationship with overcrowding and ARF rates.

In New Zealand, children aged between 5 and 14 are most at risk of developing ARF (Jaine et al., 2011). The fact that ARF seldom occurs in children below the age of 5 and infrequently in children over age 14 has led to speculation that environmental factors in early life or possibly early life infections predispose certain individuals to react to *Streptococcus* A infection with the development of ARF (Phillips & Osmond, 2014).

Asthma

New Zealand has extremely high prevalence rates for asthma (Asher et al., 2001; Global Initiative for Asthma, 2016a). Asthma is estimated to cost the New Zealand economy $800 million annually (Asher et al., 2001).

Currently, 14% of New Zealanders have been diagnosed with the illness (Global Initiative for Asthma, 2016a). There is limited evidence suggesting that New Zealand's asthma rates have decreased somewhat since the 1990s (Gillies et al., 2013; Asher et al., 2008).

A recent report produced for the New Zealand Asthma Foundation cited medicated asthma prevalence over the period 2011–2012 of 19.2% for Māori children compared to 13.8% for European/Other children. Adult rates were 16.7% and 11.4% for Māori and European/Other respectively. Pacific and Asian rates were slightly lower for children, at 13.6% and 11% respectively, and Asian rates for adults were significantly lower at 4.4% compared to Pacific adults at 9.3% (Telfar-Barnard et al., 2015). A study conducted in 2006–2008 found that 3.6% of children in New Zealand had been hospitalised for asthma at least once (Gillies et al., 2013). There are significant ethnic and socio-economic disparities in asthma prevalence, hospitalisations and mortality in New Zealand. Although Māori children, especially boys, have the highest rates of medicated asthma, Pacific children have the highest hospitalisation rates, and Pacific people over 65 have the highest mortality rates.

In 2011, there were 7,400 hospitalisations and 69 deaths from asthma in New Zealand. Asthma deaths were six times higher in the Pacific population and five times higher in the Māori population (Telfar-Barnard et al., 2015).

Asthma can present as either an allergic or non-allergic illness. Asthma developed in childhood is generally allergic asthma and may be associated with other allergies such as eczema or rhinitis (Global Initiative for Asthma, 2016b). No individual causes for developing asthma have been identified, but the current consensus is that interaction between genetic and environmental factors cause asthma (WHO, 2011; Global Initiative for Asthma, 2016b; Kanchongkittiphon et al., 2015) to onset. Genetic factors include a predisposition for allergic sensitisation, sex (male) and obesity. Up to 50% of asthma is thought to be caused by sensitisation to aeroallergens including house dust mite, dog, cat, cockroach, mould and rodent allergens (Gaffin & Phipatanakul, 2009). Furthermore, in children sensitised to these allergens, asthma symptoms are likely to be worse and persist longer than in children not sensitised (Gaffin & Phipatanakul, 2009).

Allergens from dogs, cats and house dust mites have been found in office buildings and libraries, showing that exposure is not restricted to the home environment (Kanchongkittiphon, Gaffin & Phipatanakul, 2014). Other environmental factors related to asthma onset include tobacco smoke as well as outdoor air pollution, paracetamol use and occupational exposures such as wood dust and

flour (Global Initiative for Asthma, 2016a). Phthalates are also emerging as significant risk factors for asthma (Kanchongkittiphon et al., 2015; Shu et al., 2014).

Various factors related to indoor dampness including evidence of rising damp, water stains, condensation, visible mould, mouldy odour and presence of mould spores and fungal components have been associated with asthma exacerbations and new onset asthma in children in epidemiologic studies conducted over the last three decades (Mendell et al., 2011). The qualitative measures describing evidence of dampness (including visible mould and mouldy odour) are consistently associated with asthma exacerbations and new onset asthma, whereas with quantitative measures of mould spores and fungal components, the relationship is unclear (WHO, 2009; Fisk, Lei-Gomez & Mendell, 2007; Mendell et al., 2011).

A recent review and meta-analysis by Sharpe et al. support an association between *Penicillium, Aspergillus, Cladosporium* and *Alternaria* strains of mould spores in relation to asthma exacerbation (Sharpe et al., 2015). These genera are the most common and frequently measured in asthma studies, so this study does not rule out other genera. Microbial volatile organic compounds (MVOCs) are released by moulds and fungi. Several studies have found associations between MVOCs and asthma or allergy (Kim et al., 2007; Elke et al., 1999). A recent update of the Institute of Medicine asthma review *Clearing the air: Asthma and indoor air exposures* (Institute of Medicine, 2000) found that there is sufficient evidence for a causal relationship between dampness or dampness-related agents and asthma in children and for an association in adults and that this relationship is not restricted to those with specific sensitisations (i.e. dust mites or mould) (Kanchongkittiphon et al., 2015).

Combining the results of a number of prospective studies, the World Health Organization reports that children living in homes with signs of dampness have 2.4 times the risk of developing asthma than children in dry homes (WHO, 2011). Another meta-analysis of asthma causation in the US attributed 21% of asthma to damp environments (Mudarri & Fisk, 2007). A modelling study from Finland suggested that achievable improvements in indoor air quality in the home could reduce the national asthma burden by 10% (Rumrich & Hänninen, 2015). It is impossible to rule out humidity itself as the factor related to asthma causation rather than one or more mould species or other allergen, as dampness increases the likelihood and severity of exposure for a number of these, including mould, dust mites, cockroaches and chemicals including formaldehyde and phthalates, all of which have been associated with asthma in epidemiological studies. Conversely, all of these potential allergens or pollutants can be controlled or mitigated by reducing humidity (Sharpe et al., 2014). This means that reducing dampness in homes and schools along with improved ventilation is likely to reduce asthma exacerbations and prevalence significantly, and indeed, interventions aimed at reducing dampness consistently show significantly improved health (Kanchongkittiphon et al., 2015).

Reducing dampness is complex, and research has not fully elucidated the best methodology for remediating dampness issues. Retrofitting insulation into New Zealand homes constructed pre-2004 has been conclusively demonstrated to reduce respiratory illness (Howden-Chapman et al., 2007) as well as doctor's visits and hospitalisations. The savings from avoiding one night in hospital for a respiratory infection is about the same as the one-off capital cost of retrofitting insulation into the subfloor and ceiling of a home, with the health savings accruing year on year.

Intervention studies in the UK have shown that certain energy efficiency renovations can increase indoor pollutants (Hamilton et al., 2015) and increase asthma (Sharpe, Thornton, Nikolaou & Osborne, 2015) due to reduced ventilation.

A number of recent studies have focused on early life exposures in relation to asthma development, as it has become clear that exposures both before and immediately after birth can contribute to health of the individual over their whole life course (Fuentes-Leonarte, Ballester & Tenías, 2009). The same may be true of early life respiratory infections, as the lungs have not developed fully till age 6 years (Fuentes-Leonarte, Ballester & Tenías, 2009).

Karvonen et al. found that moisture damage and visible mould in a child's bedroom or living room during the first 5 months of life were statistically associated with development of asthma by age 6 (Karvonen et al., 2015). Another study showed that concentrations of *Penicillium* were found to be significantly higher in homes with an infant experiencing any wheeze in the first year of life (Rosenbaum et al., 2015).

Asthma epidemiology focusing on pathogenesis in children has recognised two separate populations within the illness. One group have been identified as atopic in early life (before 18 months of age) and tend to have transient wheeze, which has generally disappeared by age 7, while those children who are sensitised later are more likely to have persistent asthma and worse morbidity (Gaffin & Phipatanakul, 2009). How this difference relates to early life exposures remains unclear (Kanchongkittiphon et al., 2015).

Nitrogen dioxide (NO_2), which is a byproduct of combustion, is well established as a respiratory irritant. A study of heating, housing and health where an unflued gas heater or plug-in electric heater was replaced with a non-polluting heater found asthmatic children had significantly less night cough and wheeze, fewer doctor visits and 2.5 fewer days per winter off school from asthmatic symptoms (Howden-Chapman et al., 2008; Free et al., 2010). Real-time monitoring of pollutant levels in homes using an unflued gas heater found that some heaters produced NO_2 that was six times higher than the WHO guideline, and these emissions were found in the asthmatic child's bedroom even when the heater was used in the lounge room (Boulic, 2012).

Allergy

Allergic sensitisation is well correlated with allergen exposure in young and school-age children (Gaffin & Phipatanakul, 2009). Allergic rhinoconjunctivitis (also known as hayfever) and eczema are both associated with dampness and mould, (Mendell et al., 2011) as well as with common indoor bioaerosols or particulates of biological origin, including allergens of dust mites, cockroaches, cats, dogs, rodents and fungi (Mendell et al., 2011; Hulin et al., 2012; Ghosh, Lal & Srivastava, 2015).

Chronic obstructive pulmonary disease

Chronic obstructive pulmonary disease (COPD) (also known as emphysema) is a significant and growing concern in developed nations worldwide. In New Zealand, COPD rates are highest in Māori with onset typically at age 55 for Māori and 65 for non-Māori and are highest for those on fixed incomes. The percentage of people in New Zealand over the age of 65 years is expected to increase from the current rate of 12% to 22% over the next 25 years. Due to the ageing population, COPD is estimated to be the fifth-largest burden to global health worldwide by 2020 (Ko & Hui, 2012). The economic burden of COPD is very high due to people often living a long time with this debilitating condition and requiring significant health sector attention. Finding effective methods to reduce the incidence and morbidity of COPD will become increasingly important.

Resulting from the findings that installing insulation into a home reduces respiratory infections and that a third of COPD exacerbations are the result of a respiratory infection, research is currently under way by the Healthy Housing research group to investigate if a fuel subsidy and the message that "heat is your medicine" could potentially reduce the frequency and severity of COPD exacerbations. Results are expected in 2018.

Cigarette smoking is currently considered the most important cause of COPD, but increasingly, attention is being turned on other exposures including low temperatures, as studies show that, in countries similar to New Zealand, around 25% of COPD may be caused by exposures other than cigarette smoke. Second-hand cigarette smoke and particulate matter have been implicated, as have childhood asthma and acute respiratory illness in childhood and even prenatal exposures (Abramson et al., 2015; Ko & Hui, 2012; Goldizen, Sly & Knibbs, 2015).

Cardiovascular disease

While the link between pulmonary diseases and air pollution is well established, the link with cardiovascular conditions has had less attention in research literature. It is becoming clear, however, especially from ecological studies of pollution and premature mortality, that the effects on cardiovascular health may be as significant as on lung health (Uzoigwe et al., 2013). Pollutants that have been shown to be associated with cardiovascular diseases are mainly combustion products including $PM_{2.5}$, ozone, nitrogen dioxide, sulphur dioxide, cigarette smoke and lead in air. These pollutants are thought to contribute to atherosclerosis (hardening of the arteries), the underlying

condition contributing to most cardiovascular illness, including heart attacks. 75% of those suffering a heart attack die outside the hospital, and for 20% of patients with coronary artery disease, sudden death is the first (and only) manifestation of the illness (Uzoigwe et al., 2013). As treatment is not available to such individuals, prevention becomes the most important strategy.

A New Zealand population-wide study of excess mortality and air pollution found an increase of 6% mortality due to cardiovascular diseases per 10 $\mu g/m^3$ increase in PM_{10} (Hales et al., 2012).

Cancer

The indoor air pollutant most commonly related to cancer is cigarette smoke, including second-hand smoking. Also known as environmental tobacco smoke (ETS), second-hand smoke has been related to lung cancer in a number of studies and is recognised as a group 1 carcinogen by the International Agency for Research on Cancer (IARC). A limited number of studies have looked at childhood exposure to ETS and the later development of respiratory cancer, and results have been conflicting (Goldizen et al., 2015). Very little research has been done on other childhood exposures and adult cancer development. Childhood ETS exposure and traffic exhaust exposure have been associated however with leukaemia and brain tumours in children (Goldizen et al., 2015).

More recently, researchers have looked at the link between other indoor air pollutants and non-respiratory cancers, especially those of childhood. In China's new mega-cities, increased rates of childhood leukaemia have been observed, and a study modelling risk from exposure measurements in homes and offices has suggested a link with formaldehyde (Huang et al., 2013). Another Chinese study, using a case-control design and taking exposure measurements in children's bedrooms, found associations between childhood acute leukaemia and VOCs, NO_2 and renovating or changing the furniture in the previous 5 years (Gao et al., 2014). An American study looking at prenatal exposures and neuroblastoma, a cancer that is usually diagnosed in children under the age of 5, found a link with polycyclic aromatic hydrocarbons (PAHs) (Heck et al., 2013).

Another recent line of research has looked at a link between respiratory cancer and aerosols formed through the high-temperature frying of foods, and IARC has classed this pollutant as group 2A, meaning it probably causes cancer, but there is currently insufficient evidence to be conclusive (Goldizen et al., 2015).

Reproductive illness

A number of endocrine-disrupting chemicals (EDCs) that are common in ambient air, including phthalates, pesticides and flame retardants, have been implicated in reproductive disorders. Research looking at EDCs and health effects is an important new strand of study in the epidemiology of chronic illness.

Building-related illness

There is considerable confusion around the use of the terms building-related illness (BRI) and sick building syndrome (SBS). BRI refers to a diagnosable causal agent in a building such as mould or *Legionella bacteria*. SBS refers to situations where the building is causing illness but the causal agent may not have been identified. These terms are sometimes used interchangeably.

SBS reflects early clinical attitudes towards individuals claiming vague or non-specific neurological symptoms such as headache, nausea, tiredness or dizziness as well as respiratory symptoms such as a cough (Turpin, 2014). The fact that the odour thresholds for most VOCs is orders of magnitude lower than the threshold for irritation effects (Wolkoff, 2013) and that such building-related symptoms are more frequently cited by women than men (Turpin, 2014) were seen as evidence of their psychiatric nature. Numerous studies over the 1980s and 1990s, however, showed that there were associations with claims of SBS and air conditioning, and poorly maintained systems are now seen as a risk factor (Turpin, 2014).

VOCs have frequently been implicated in SBS (Król, Namieśnik & Zabiegała, 2014b). However, they have not been proven to have a causal association (Bernstein et al., 2008) in all situations. This is possibly as a vast cocktail of VOCs are present in indoor air at low levels, with each VOC seldom reaching the threshold level. The concept of total volatile organic compounds (TVOCs) has been proposed but discounted as it is not possible to determine the health effects from a VOC cocktail – are the combinations of VOCs additive or synergistic? Further, it is important to remember that the threshold for irritation is based on normal sensitivity. Certain individuals and vulnerable populations may have a significantly lower irritation threshold than the general population. Research into the variability of physical sensitivity across populations would be valuable to epidemiologists looking at associations between health effects and low-level exposures.

In New Zealand, at least two cases of BRI in office buildings have been recognised and compensated by the Accident Compensation Corporation (ACC). When claims were made by a large number of people claiming effects from the same commercial building, however, one individual claiming effects from a building that apparently does not affect others is less likely to gain recognition or compensation for their claim (W. Glass, personal communication). In one case, compensation was awarded after exposure to high levels of formaldehyde that arose during an office refurbishment. In another case, the causal agent was neither tested for nor identified, but a strong dose response relationship was defined to satisfy the presiding Judge that the building was the cause of the applicant's illness.

Part two – Pollutants

Outdoor air pollution

Exposure studies show that our greatest exposure to outdoor air pollution actually occurs indoors. This is due to duration of time spent indoors and the infiltration of outdoor pollutants into the indoor environment (Hodas et al., 2012; Weschler, 2015). The main gaseous components of anthropogenic (caused by human activity) ambient air pollution are ozone, carbon monoxide, nitrogen dioxide and sulphur dioxide (Levy, 2015). Particulate matter (PM) is another significant pollutant and can include elemental carbon, nitrates, sulphates, sea salt, soil and other organic matter (Hodas et al., 2012). Particulates also provide convenient surfaces for pollutants including polyaromatic hydrocarbons and metal oxides to adhere to, keeping these pollutants airborne and respirable (WHO, 2010).

Outdoor air pollution is a serious health concern globally, especially in urban areas. Outdoor air pollution has recently been classed as a group 1 carcinogen, as has diesel exhaust. Group 1 means there is sufficient evidence to conclude that this pollutant causes cancer (IARC, 2013). In China, which obviously has a very different ambient pollution profile to New Zealand, cancer is now the leading cause of death, and of cancers, lung cancer is the most frequent killer (Zhao et al., 2010). This has been attributed to high levels of smoking and outdoor air pollution. In China, deaths from traffic-related air pollution are now an order of magnitude higher than deaths from traffic accidents (Lelieveld et al., 2015). This demonstrates how serious the impact of outdoor air pollution can become.

Research into the health effects of outdoor air pollution started with broad-scale studies that associated population-scale mortality with particulate matter (PM) both at the PM_{10} scale and $PM_{2.5}$, where PM is used as a marker of pollution that may include many thousands of individual chemical components. These studies consistently show PM is associated with elevated premature death, particularly for cardiovascular (heart attacks and strokes) and respiratory illnesses (Farmer et al., 2014; Chen, Zhao & Weschler, 2012). The same relationship has been shown in studies performed in New Zealand (Hales et al., 2012; Kuschel et al., 2012). People don't generally die from air pollution directly, but the pollution exacerbates or potentially even incites underlying illnesses such as COPD, ischaemic heart disease or respiratory illnesses, which then cause death. This means that it is a relatively easy problem to overlook.

Doctors are trained to ask patients about exposure to tobacco smoke but are less likely to enquire about proximity to a busy traffic route or the patient's use of an unflued gas heater. This lesser-known association means that New Zealand isn't fully accounting the health burden of urban pollution.

Even without anthropogenic sources of pollution, air is replete with a miasma of natural pollutants, including particulate matter from wind erosion of rocks and soils, salt particles from oceans,

thousands of VOCs such as aldehydes, terpenes and alpha pinenes released from plants and fungi, which react with one another or sunlight to create secondary organic pollutants including ozone, and millions of living and dead microbes such as bacteria and viruses.

The Health and Pollution in New Zealand (HAPINZ) study attributed almost half of all air pollution mortality to natural sources of pollution (Kuschel et al., 2012). Since the publication of this report, however, characterisation of outdoor air pollution has continued to advance, and two findings may affect the interpretation of the HAPINZ study. Firstly, recent studies suggest that, although natural sources may account for a significant proportion of the mass of particulate matter in air, those particles are less likely to be dangerous ones causing health effects than the anthropogenic (made as a result of human activity) sources of pollution (Lelieveld et al., 2015; Ostro et al., 2015). The HAPINZ study, which used PM_{10} as the most widely available data, treated all sources as equal in respect to health effects (Kuschel et al., 2012). One of the reasons that anthropogenic sources of PM are now seen as more harmful than natural sources is their generally high contributions of organic or carbon based aerosols (Mauderly & Chow, 2008). More recent studies include characterisation of PM to differentiate the benign from the toxic fractions.

Secondly, studies assessing levels of airborne dust worldwide have found that dust in the southern hemisphere has increased over the last 30 years in very close symmetry with increased land use for agriculture, the authors proposing that lowered river and lake levels due to increased water use is the cause of increased airborne dust (Ginoux et al., 2012). An analysis of the chemical composition of dust from a lake in Iran in which the water level has lowered by 6 metres over the past decade showed particle matter in dust emanation from this source was dominated by sulphate and nitrate ions (representing 58% of total by number of particles) (Gholampour et al., 2015). Nitrate and sulphate particles have been associated with cardiovascular and respiratory illnesses (Ostro et al., 2015).

A recent large analysis of mortality and air pollution which took into account these findings and used advanced modelling combined with satellite data to calculate levels of ozone and $PM_{2.5}$ globally reported that $PM_{2.5}$ pollution caused by agriculture is a significant contributor to premature mortality (Lelieveld et al., 2015). While Lelieveld et al. did not create estimates for New Zealand in their modelling study, the contribution in other developed nations similar to New Zealand, such as USA, Japan, Germany and Russia, from agriculture was found to be responsible for 30–40% of air pollution mortality (Lelieveld et al., 2015), a more significant contributor than transport, except in the USA where it was responsible for approximately similar numbers of pollution-related deaths.

It may be that some of the PM classified as natural in New Zealand outdoor air pollution calculations needs to be redefined as anthropogenic. Agriculture can also release other pollutants into air, including pesticides, sulphates and nitrates from fertiliser and PM from burn-off. Currently, there is no information available in New Zealand about the volume of air pollution produced by the primary industries, and this is a major gap in the science of air quality in this country (Ministry for the

Environment and Statistics New Zealand, 2014) (see Pesticides and Fine particulate matter). This topic has a strong research programme both within New Zealand and internationally. Instrumentation advances and multidisciplinary study designs are opening up further new areas for investigation.

Relationship of indoor and outdoor air pollution

A large-scale study from the US looking at the relationship of indoor, outdoor and personal air (RIOPA) took measurements in 212 homes in various localities across the US and found the median contribution to indoor $PM_{2.5}$ from outdoor sources was 56% (Qing et al., 2005). These findings are similar to a study from Santiago, Chile, which found approximately 50% of indoor $PM_{2.5}$ came from outdoor sources (Barraza et al., 2014). The contribution of outdoor pollution to indoor air quality may need re-examination in light of recent work, demonstrating that the finest fraction of PM, also known as ultrafine particles (UFPs) and representing particles .01 µm in diameter and smaller, are more efficient at infiltrating indoor environments from the outdoors than larger fractions (El Orch, Stephens & Waring, 2014), which is important as these fractions are also most likely to cause adverse health effects (see Ultrafine particles). A detailed analysis of VOCs from an industrialised urban centre in Edmonton, Canada, found the outdoor contribution of VOCs to indoor concentrations at around 30% (Bari et al., 2015). The smaller contribution compared to PM is due to the larger number of indoor sources of VOCs.

The intensive pollutant monitoring study nested within the Heating, Housing and Health study, found that homes located near a major roadway had significant levels of indoor NO_2 at peak traffic periods (Boulic, 2012), and NIWA research has found homes located within 700 metres of a roadway to experience high levels of NO_2 and PM (G. Coulson, personal communication, 2015). The evidence that ambient pollutants readily migrate into New Zealand housing challenges the widely reported advice that people are protected from ambient pollution if they stay indoors. This advice, which is often given where there is an acute ambient pollution situation such as a fire in a building with asbestos, may provide a false sense of security. This topic is worthy of further research.

The use of air conditioning to mitigate the impact of PM infiltration has been investigated. Hodas et al. used outdoor exposure data from three different urban regions in the US with data showing the prevalence of air conditioning in homes to demonstrate that increased frequency of air conditioning was related to decreased mortality estimates at the large scale (Hodas et al., 2012). The same study looked at the issue of socio-economic inequalities of exposure and concluded that those at the lower end of the socio-economic scale had increased infiltration rates due to poorer housing condition and being more likely to live close to areas of high road traffic, both of which contribute to higher indoor air exposure to outdoor (Hodas et al., 2012).

These factors are relevant to the New Zealand situation where housing quality has been shown to reflect socio-economic inequalities (Telfar-Barnard, 2009).

An unexpected but pleasing result of the solar heated ventilation in classrooms project was that PM levels were significantly lower in classrooms operating a roof-mounted solar air heater than outdoors and half that of the control classrooms (Boulic et al., 2016).

Chemical pollutants – gas phase

Carbon monoxide

Carbon monoxide (CO) is a colourless, odourless gas produced in combustion reactions both indoors and outdoors. As its presence is undetectable visually or by smell, people are vulnerable to accidental poisoning. CO binds to haemoglobin in the blood 200 times more readily than oxygen, thereby starving the body of oxygen (Levy, 2015). This causes the transportation of oxygen to tissues to be dramatically reduced. Once inside a human body, the half-life of CO is about 4–5 hours. CO levels primarily affect the heart and lungs and will antagonise pre-existing conditions such as angina. High CO levels are fatal. However, lower levels will also cause headaches and fatigue and gastric and flu-like symptoms. Mental and physical functioning is impaired – a person with acute exposure to CO can appear drunk. The affected person can be on the point of collapse without realising it. Mild physical exertion after exposure to CO can prove fatal.

CO has the potential to remain an invisible threat, as non-specific symptoms and the short half-life in the body may frequently lead to misdiagnosis (Robertson & Cohn, 2014; Levy, 2015), which in turn may lead to patients being sent back to the dangerous conditions that made them seek attention none the wiser (Robertson & Cohn, 2014).

The effects of CO may also go under-recognised by exposure scientists and epidemiologists. In his comprehensive review of CO health effects, Levy pointed out that CO may be the link in studies that have found associations between autism and autism spectrum disorder and traffic emissions, as carbon monoxide can cross the placental barrier, affecting neurodevelopment in utero (Levy, 2015). Carbon monoxide exposures are virtually always highly correlated with other combustion-related toxic exposures, especially nitrous oxides, particulates and VOCs. Few studies have looked at the effects of low-level chronic carbon monoxide exposure (Levy, 2015), but there is evidence of low birth weight, cardiovascular illness and mortality (WHO, 2010) as well as dementia (Chang et al., 2014) associated with chronic low-level CO exposure.

Carbon monoxide is one of the most commonly found and widely distributed indoor pollutants and a major component of vehicle emissions, cigarette smoke and gas cooking or combustion-sourced heating (Levy, 2015). It is found in homes with internal garages. Indoor levels are typically as high as outdoor levels. Unflued gas heaters, tobacco smoking and vehicle emissions from attached garages are the primary indoor sources of CO.

In the UK, more children die from CO poisoning than any other form of poisoning. Carbon monoxide poisoning is the leading cause of poisoning death, both accidental and intentional (WHO, 2010; Peiris-John, Kool & Ameratunga, 2014). Installation of CO detectors (similar to a smoke detector) are compulsory in some countries but are seldom used in New Zealand.

Accidental poisonings are more common after power cuts due to weather events or disasters (Chen et al., 2013) when people decide to burn fuel indoors in unventilated conditions, leading to the dangerous build-up of CO. One study looked at the short-term health effects of CO immediately after Hurricane Sandy in New York State, USA. This study found 437 hospitalisations for CO poisoning in the 2 weeks after the storm – 311 of those were due to a large fire in an apartment complex. Of the rest, 29% were due to grilling indoors and 17% to inappropriate generator placement (Chen et al., 2013).

Low-level, chronic carbon monoxide exposure may occur in buildings where gas is used for heating or cooking as a result of incomplete combustion. Gas fires or stoves with blocked or dirty ducts, inadequate installation or inappropriate use of gas fires that use radiant stones may cause persistent levels of CO in indoor air and even typical use of gas stoves has been shown to increase indoor CO (Wallace, 2000).

There are typically several CO accidental deaths or poisoning stories in the New Zealand media every year, most of which occur in the home and typically low-income housing. A systematic review of this data does not appear to have been undertaken. The intensive real-time pollutant monitoring undertaken during the New Zealand Heating, Housing and Health study found levels of CO in homes operating an unflued gas heater that were below the guideline values, but nevertheless of considerable concern (Phipps et al., 2007).

Nitrogen dioxide

Traffic pollution is the major outdoor source of nitrogen dioxide (NO_2) in the US and many other countries (Zhang & Samet, 2015).

Indoors, NO_2 is commonly released from the combustion of gas energy sources (Fuentes-Leonarte, Ballester & Tenías, 2009) and cigarette smoking (WHO, 2010). Simons et al. found the use of a gas stove for cooking was associated with higher NO_2 (Simons et al., 2007).

A New Zealand study showed that NO_2 level in homes with a gas source are on average three times higher than homes without gas (Gillespie-Bennett et al., 2008). This study looked at the release of NO_2 by unflued gas heaters and found NO_2 levels in the living room approximately four times higher and three times higher in bedrooms in homes using unflued gas heating compared with homes using an electric heat pump, wood pellet burner or flued gas heater (Gillespie-Bennett et al., 2008).

Also in this study, Boulic et al. found that NO_2 levels increased rapidly as soon as the unflued gas heater was turned on and could exceed the WHO-recommended level several fold within minutes of the unflued gas heater being operated. Exceedances were measured in a bedroom even when the heater was located in the lounge room (Boulic, 2012). This study found that replacing an unflued gas heater or small electric heater with a non-polluting heater (either a heat pump, wood pellet burner or flued gas heater) significantly reduced night-time coughing, night-time wheezing, GP visits for respiratory infections and days absent from school (2.5 days per winter) for asthmatic children (Howden-Chapman et al., 2008).

Meta-analysis has shown that similar indoor increases in NO_2 as those found by Gillespie-Bennett et al. (increase of 28 $\mu g/m^3$) were associated with a 20% increase in lower respiratory illness in children (WHO, 2010).

A further and unexpected finding from this study was the evidence of NO_2 measured indoors during peak traffic times, even where there was no indoor source. This showed the permeability of the typical New Zealand home to ambient pollutants and suggests that the widely reported advice that people are protected from ambient pollution if they stay indoors may provide a false sense of security.

Many schools have traditionally relied on gas heaters, especially in large areas such as the school hall. Several studies have looked at NO_2 exposure in schools and have found elevated NO_2 related to the use of gas for heating is associated with coughs and respiratory symptoms in exposed children (Kanchongkittiphon et al., 2014). Nitrogen dioxide exposures are generally higher in winter due to increased combustion sources and reduced photochemical reactions (Vardoulakis & Heaviside, 2012).

Elevated indoor nitrogen dioxide exposures have been linked with increased asthma exacerbations (Kanchongkittiphon et al., 2014), enhancement of airway responses to airborne allergens (Gillespie-Bennett et al., 2008) and increased risk of snoring in children by 4.5 times (Zhang, 2004). At the population level, outdoor NO_2 measurements have been associated with lower birth weight (Ballester et al., 2010), increased respiratory illness (Zhang & Samet, 2015), and dementia (Chang et al., 2014).

Sulphur dioxide

Sulphur dioxide (SO_2)is a respiratory irritant produced from the burning of fossil fuels so is primarily present indoors as a result of infiltration of outdoor air (Uzoigwe et al., 2013), but it is also produced indoors from the use of gas appliances (Jones et al., 2000). Coal and diesel combustion give off significantly more sulphur dioxide than petrol combustion, and this pollutant is also given off from metal smelting processes (Uzoigwe et al., 2013). Sulphur dioxide may react with PM to produce sulphates. Sulphate in air is present almost exclusively in particles of less than 2.1 µm in diameter, meaning current assessments of air pollution in New Zealand based on PM_{10} are unlikely to sufficiently account for the volume of sulphate in outdoor air pollution (Jones et al., 2000).

Sulphur dioxide in New Zealand has been regularly monitored at only nine sites nationally. At three out of the nine monitoring sites, SO_2 exceeded WHO short-term standards, but not the New Zealand standards, on 13, 54 and 69 days in 2012 (Ministry for the Environment and Statistics New Zealand, 2014). The sites associated with these exceedances were close to shipping and industrial sites. Sulphur dioxide along with nitrogen dioxide and PM are the most common industrial pollutants in New Zealand (Ministry for the Environment and Statistics New Zealand, 2014).

Sulphur dioxide in air has been associated with increased blood viscosity and may be an independent trigger for ischaemic heart disease (Uzoigwe et al., 2013). The irritant nature of SO_2 also means it can exacerbate respiratory conditions and increase risk of bacterial respiratory infections (Ministry for the Environment and Statistics New Zealand, 2014).

Ozone

Ozone (O_3) is a secondary pollutant, meaning it is produced by chemical reaction of primary pollutants, namely the reaction between VOCs or NO_x pollution with sunlight, in what is known as a photochemical reaction (reaction driven by sunlight). Ozone is the primary constituent of photochemical smog (Zhang & Samet, 2015). As ozone is dependent largely on the amount of these other chemicals in air, which in turn are driven to a significant extent by traffic and other combustion pollution, if in the future these sources of pollution remain the same and climate change increases sunlight hours, this will drive increases in ozone pollution. If both combustion pollution and sunlight hours increase, ozone pollution could increase dramatically (Vardoulakis & Heaviside, 2012). Traffic emissions are generally understood to be the primary cause of ozone pollution in outdoor air (Zhang & Samet, 2015), and it is gaining attention as a possible cause of modern diseases of asthma, eczema and other allergies, which were essentially unknown in pre-industrial times (Ionescu et al., 2015; Behrendt et al., 2014).

Ozone is present in indoor air, mainly as a result of infiltration from outdoors, although it is also produced in small amounts by electronic equipment. Squalene, a lipid component of human skin, reacts with ozone, significantly reducing ozone in indoor air (Weschler, 2015). The health impact of these reactions is currently unknown.

Ozone, like particles, is an air pollutant for which there is no indication of a threshold concentration below which health effects are unlikely (Streeton, 1997). However, unlike particles, the WHO has established a specific air quality guideline concentration for ozone. More than any other air pollutant, there is considerable variation in air quality guidelines/standards for ozone because of the complexities involved in reducing ambient concentrations. In New Zealand, a relatively 'pure' approach has been taken, and air quality standards for ozone of 150 $\mu g/m^3$, 1-hour average, and 100 $\mu g/m^3$, 8-hour average, have been established (Fisher et al., 2007).

Ozone can be a byproduct of products sold as air cleaners. Some negative ion generators produce ozone. Ozone is also marketed as a treatment to remediate buildings with high fungi infestations. These are both currently unregulated.

Volatile organic compounds (VOCs)

This chemical group consists of many thousands of individual organic chemicals released into ambient air by many products and processes including combustion and off-gassing from building materials. VOCs are found at higher concentrations indoors than outdoors, indicating that they are predominantly produced by indoor activities and processes (Delgado-Saborit et al., 2011; Villanueva et al., 2015).

A recent review of exposure studies from Germany found that overall VOC levels indoors had decreased over the past 20 years. Pooled exposure data from a number of epidemiological studies conducted in one German city, Leipzig, from 1994 to 2008 showed a reduction in alkanes and aromatics but no change in the levels of aldehydes, terpenes or chlorinated hydrocarbons measured. The authors attributed this reduced exposure to a reduction in smoking indoors and the introduction of low-VOC paints and building products (Herbarth & Matysik, 2013).

Some chemicals found in indoor environments have reduced as a result of tighter regulation and policy since the 1950s (formaldehyde and some other VOCs, PAHs, tri and tetrachloroethylene, chlorinated pesticides and PCBs). Others have increased and remain high (phthalates, flame retardants, synthetic musks, terpenoids including alpha pinene and limonene, synthetic pyrethroids and triclosan) (Weschler, 2009).

No such measurements have been made in New Zealand. Several companies make claims of low emissions coatings (such as paints), and there has been a significant reduction in smoking indoors, so indoor VOC levels may reflect this same reduction.

VOCs have received a vast amount of attention in health research literature. One reason for this is the enormous increase in VOC production and use since mid last century in cleaning products and building materials and the temporal relationship between this increased exposure and increases in the prevalence of asthma and allergies in the developed world. Despite many studies conducted on the topic, whether or not VOCs cause or exacerbate asthma and allergies remains inconclusive (Nurmatov et al., 2015). While a number of observational studies have found a relationship between respiratory health effects and activities related to VOC exposure, such as cleaning, painting or laying new carpet, prospective studies measuring the actual exposure to specific chemicals have found no consistent relationship (Mendell, 2007; Nurmatov et al., 2015).

VOCs have not been ruled out in relation to asthma causation, however (Nurmatov et al., 2015). A recent review found that exposure assessment was seldom comprehensive in such studies, with

most studies using static ambient rather than personal exposure and most exposure collection not being conducted over an extended period (Nurmatov et al., 2015). Despite the lack of specific and conclusive evidence of a relationship with respiratory health effects, it is generally agreed that VOC exposure causes a range of illnesses and reactions, especially related to eye and naso-respiratory complaints (Wolkoff, 2003; Kostinen et al., 2008). Between 50–300 different VOCs may be found in any body of indoor air at measurable levels (Bernstein et al., 2008), such complexity contributing to the difficulty of finding associations with health effects but also contributing to the ongoing agreement that VOC exposure does impact health (Bernstein et al., 2008).

More recently, the role of ambient VOCs in relation to cancer causation and particularly childhood leukaemia has received attention. While IARC has recognised the role of benzene, and more recently formaldehyde, in cancer causation (IARC, 2012), Gao et al. conducted a case-control study, finding significant associations with their measurements of both benzene and formaldehyde as well as several other VOCs in the homes of newly diagnosed childhood leukaemia cases. Other VOCs statistically associated with acute childhood leukaemia cases included styrene, carbon tetrachloride, methyl isobutyl ketone, butyl acetate and butyl alcohol as well as nitrous oxide (Gao et al., 2014).

A number of VOCs have been established to be carcinogenic at high exposure levels including benzene and formaldehyde, both of which are found in domestic indoor air at levels warranting concern in developed countries (Kostinen et al., 2008). The WHO and the European Union have initiated steps to reduce indoor VOC pollution including placing formaldehyde, benzene and naphthalene in their top five indoor pollutants to work to reduce (Kostinen et al., 2008; WHO, 2010).

The most commonly found indoor VOCs are aromatic solvents benzene, toluene, ethylbenzene and monomers of xylene, known collectively as the BTEX group (Esplugues et al., 2010a). These solvents are common in tobacco smoke, cleaning products and consumer products such as air fresheners and deodorisers.

Materials that give off VOCs include paint, varnish, fabrics, cleaning products, polish, cosmetics, air fresheners, deodorisers, PVC wall and floor coverings and furnishings (Nurmatov et al., 2015). Bari et al. conducted a comprehensive analysis of VOCs in indoor and outdoor air in Edmonton, a large industrial city in Alberta, Canada (Bari et al., 2015). They measured 193 VOCs in indoor and outdoor air in 50 houses in Edmonton, Alberta, and attempted to define sources for all measured VOCs. This study attributed 44% of indoor air VOCs in their sample to household products including general cleaning products, furniture and nail polish. A further 8.4% was attributed to deodorisers, 2.4% to perfumed products and 3.6% more from off-gassing from tap water and use of bleach. In this sample, building materials including paints and flooring materials accounted for 11% of the total VOCs found. Esplueges et al. found a dose-response relationship between BTEX measured indoors and frequency of traffic passing outside the house (Esplugues et al., 2010a).

Natural sources of VOCs include some microbial varieties (MVOCs), which are produced by mould and bacteria (Hung et al., 2015; Elke et al., 1999). MVOCs include many alcohols, aldehydes and esters, some of which are specific to microbial sources, but many of which are also characteristic of anthropogenic sources such as building materials and cleaning products (Kim et al., 2007). These differences have not been well characterised, so as yet there are no useful indicator MVOCs that can be used to indicate the presence of mould (Kim et al., 2007; Elke et al., 1999). This could be a useful, though complex, area of research, as the volatility of such substances means they can travel through building materials, so such indicators could potentially be used to test for the presence of hidden mould and fungi in building structures (Elke et al., 1999; Hung et al., 2015).

MVOCs are not generally thought to contribute significantly to total indoor VOC exposures. H however, there is a lot more that needs to be known around MVOCs (Kim et al., 2007). Indeed, little is known on the health effects from MVOCs, especially as each such chemical reacts differently in the body. Further, the proportion of total VOC exposure from each individual component is not necessarily a useful measure in relation to health effects. Some researchers have implicated MVOCs and BRI. Plants also give off VOCs, such as isoprene and alpha and beta pinenes as a byproduct of photosynthesis (Bari et al., 2015 Hung et al., 2015) and floral perfumes. These are more common (a larger contributor to total VOCs in general) than MVOCs and were assessed in Edmonton by Bari et al., and such biogenic sources were calculated to comprise up to 10% of the total outdoor VOC load, at 10.8 g/m^3 (Bari et al., 2015).

Plants also absorb VOCs. Dela Cruz et al. examined a number of plants, measuring their ability to remove toluene from the atmosphere, and found the most efficient to be able to absorb 66.5 µg/m^3/ hour (Dela Cruz et al., 2014). Other studies have shown similar effects with formaldehyde and ethyl benzene (Mahnert, Moissl-Eichinger & Berg, 2015). However, the number of plants required can be excessive, and source control is considered a more effective control strategy.

Another recent focus of research is the effect of human occupants on indoor pollutant levels (Nazaroff & Goldstein, 2015). Occupancy of a building reduces chemicals of low vapour pressure from the atmosphere two orders of magnitude (100–900 times) faster than happens in unoccupied buildings (Weschler, 2015). VOCs and other chemicals react with skin oils, significantly reducing their concentration in air, but the impact on humans of these chemical reactions is currently unclear (Weschler, 2015).

Evidence is emerging that exposure to some anthropogenic pollutants (produced through human activity) at typical ambient levels may reduce immunity and lung function in otherwise healthy individuals (Carlsten & Georas, 2014). This evidence points to two important factors of indoor air science. Firstly, there may be no safe levels for certain pollutants, such as combustion and traffic pollutants (WHO, 2006), and secondly, if exposure to some pollutants occurs at a child's developmental stage where immune functions are still developing, such exposures may lead to

conditioning of the immune system, meaning an increased risk of adverse health effects throughout life (Phillips & Osmond, 2014).

Research into endocrine-disrupting chemicals (EDCs), including phthalates, pesticides and flame retardants, has increasingly shown a connection between low exposures in early life and the later development of chronic health conditions including obesity and diabetes (Gore et al., 2015) and neurodevelopmental disorders including attention deficit disorders (Verner et al., 2015). Furthermore, while many of these chemicals were previously thought to be ingested primarily through the diet, recent research has highlighted the importance of dust ingestion, which it now seems may be just as significant an exposure route for phthalates, pesticides and flame retardants (Wu et al., 2007).

Formaldehyde

Formaldehyde can build up in indoor environments to harmful levels and has received some attention in the literature. Indoor exposure accounts for 99% of formaldehyde exposure (Bruinen de Bruin et al., 2008). Formaldehyde is used in most pressed wood products such as plywood, chipboard and medium-density fibreboard (MDF). It is also present in most urethane coatings and building foams (fillers). Formaldehyde is a component of tobacco smoke, and it is also produced as a result of ozone reacting with terpenes, both of which are common in indoor air, however the size of contribution from this pathway is unclear (WHO, 2010). Formaldehyde is one of the most studied exposures in indoor epidemiology (Hulin et al., 2012). Formaldehyde has been measured in relation to asthma exacerbation or onset, and it is thought that exposure to formaldehyde may increase sensitivity to other allergens such as dust mites (Mendell, 2007). Overall, the relationship between formaldehyde exposure and respiratory illness is currently considered minor. The WHO attribute less than 1% of all wheeze illness to formaldehyde (WHO, 2011).

Formaldehyde has been identified as a carcinogen. The International Agency for Research on Cancer (IARC) has recognised formaldehyde as a cause of nasopharyngeal cancer and leukaemia (IARC, 2012). In China, over the past decade, the unprecedented level of construction has led to millions of new homes in mega-cities, which has coincided with substantial increases in lung cancer and childhood leukaemia. Several studies have made the link between formaldehyde in newly constructed or refurbished homes and leukaemia, suggesting a causal relationship, although this has not yet been confirmed by prospective epidemiological studies (Tang et al., 2009; Huang et al., 2013; Gao et al., 2014).

Huang et al. measured formaldehyde levels in 383 homes and 451 offices, all of which had been remodelled within the previous 12 months. They found levels of formaldehyde in 85% of homes and 67% of offices exceeded guidelines (Huang et al., 2013). Mean formaldehyde in homes was 131 $\mu g/m^3$ and 85 $\mu g/m^3$ in the offices. These levels were significantly higher than WHO guidelines limits of 30

µg/m^3. Other studies from China that measured formaldehyde in homes that had not been recently remodelled measured mean levels of formaldehyde at 22 µg/m^3 and 30 µg/m^3 (Huang et al., 2013).

European pooled data estimates have found mean domestic indoor formaldehyde levels around 26 µg/m^3
(Kostinen et al., 2008), which is very close to its estimated no effect level of 30 µg/m^3, and similarly high levels were measured in US homes (Bruinen de Bruin et al., 2008). The European Union has classed formaldehyde in its top five indoor pollutants of concern for European residents and has recommended measures to reduce formaldehyde levels in indoor air (Kostinen et al., 2008).

Guideline values for formaldehyde exposure published by the New Zealand Government are 100 µg/m^3 for 30 minutes. There are no long-term formaldehyde exposure guidelines. There has been little research on VOC levels in New Zealand homes or schools. This is an area ripe for research.

Benzene

Benzene is ubiquitous in ambient air, although 90% of this is produced from anthropogenic processes (Kostinen et al., 2008; Saloman, 2013). Benzene has been commonly found in indoor air, and higher concentrations are associated with cigarette smoking, having an attached garage and living close to a petrol station, while smaller contributions may come from building materials including paints and adhesives and cleaning products (WHO, 2010). Personal exposure analysis has shown that benzene is also closely related to exposure during transportation or commuting, which accounted for 29.4% of average personal exposures (Bruinen de Bruin et al., 2008).

Benzene is a component of petrol, and while levels in petrol have been regulated and reduced substantially in New Zealand since the 1990s – guidelines initially reduced formulations to 4%, then, in 2006, to 1% (Ministry for the Environment, 2004) – traffic emissions and domestic fuel burning remain the largest contributors to benzene exposure, although this is highly differential throughout the country, and in some localities, industry is the greatest contributor (Saloman, 2013).

IARC has listed benzene in their group 1 for carcinogenicity, meaning substantial proof of a causal relationship. Internationally, studies have found both benzene exposure and childhood leukaemia associated with living close to a petrol station or mechanical workshop (Gao et al., 2014). Attention is turning to the relationship between benzene and ischaemic heart disease, although the evidence remains scarce for this association (Bard et al., 2014). Benzene is in the European Union's top five indoor pollutants of concern for European residents (Kostinen et al., 2008).

Fisher et al. estimated 47 New Zealand cancer deaths in 2001 were attributable to benzene exposure. The authors further broke down the causes into approximately half due to vehicle emissions, half to domestic emissions and a small amount (3%) to industrial emissions (Fisher et al.,

2007). These estimates have been questioned (Kuschel et al., 2012), however estimates have not been recalculated and remain difficult due to the differential levels of exposure in different locations (Saloman, 2013) and the sporadic nature of benzene monitoring. In 2010, the Ministry for the Environment reduced guidelines for exposure to benzene by 70% from 10 $\mu g/m^3$ to 3.6 $\mu g/m^3$ as an annual average exposure (Ministry for the Environment, 2002), while monitoring has measured levels 40% above this at Auckland urban monitoring sites (Ministry for the Environment and Statistics New Zealand, 2014).

Semi-volatile organic compounds

A subgroup of VOCs, semi-volatile organic compounds (SVOCs), may switch between the gas and solid partitions and therefore condense on surfaces, including house dust, and so may be found in both gas and solid phase. The major contributors to SVOCs are pesticides, phthalates, polybrominated diphenyl ethers (PBDEs) and other brominated flame retardants (BFRs), polycyclic aromatic hydrocarbons (PAHs), alkylphenols, phenols, parabens and perfluorinated compounds (PFCs) (Blanchard et al., 2014).

SVOCs cannot remain suspended indefinitely and so deposit on surfaces, remaining mobile as they frequently attach to dust particles, which may be remobilised through human activity (see Dust). Many common indoor materials such as wallboard and fabrics can reabsorb gas-phase SVOCs, eventually becoming sources for redistribution of these highly mobile chemicals (Weschler & Nazaroff, 2008).

Many SVOCs were previously understood to have a primary exposure route via diet. However, recent research has highlighted that inhalation of indoor air and ingestion of dust are significant routes of exposure for many SVOCs, including pesticides and phthalates (Blanchard et al., 2014), polybrominated diphenyl ethers (PBDEs) (Coakley et al., 2013; Lim et al., 2014) and even polychlorinated biphenyls (Harrad et al., 2009), therefore infants and toddlers are at risk of higher exposures to these substances than adults and older children because of frequent hand to mouth behaviours and because their breathing zone is closer to the floor. Król et al. measured PBDEs in ingested dust in toddlers and adults and found that toddlers consumed at least five times the PBDEs of adults (Król, Namieśnik & Zabiegała, 2014a). Studies have shown that levels of SVOCs in dust are higher in older houses and buildings (Whitehead et al., 2014a).

Polycyclic aromatic hydrocarbons

Polycylic aromatic hydrocarbons (PAHs) are a common type of SVOC and the most common and studied forms of polycyclic organic matter (POM) (WHO, 2010). PAHs all contain two or more rings of six carbon-hydrogen pairs, known as benzene rings. Lower molecular weight PAHs may exist in vapour form in ambient air, while the larger molecules (five or more benzene rings) will generally

be bound to solids, including airborne particles and dust. Intermediate sized molecules partition between solid and vapour phases, disattaching and reattaching to surfaces and particles depending on climatic conditions. Particle-bound PAHs are considered very hazardous to human health (WHO, 2010). Smaller particles have more surface area to volume so are likely to be relatively more contaminated with these dangerous pollutants (WHO, 2010). A positive feedback loop exists in that the smaller the particle, the further into our anatomy it can travel (see Ultrafine particles) and the more concentrated in pollutants it is likely to be. PAHs are frequently carcinogenic (WHO, 2010).

PAHs are formed in high-temperature combustion of carbonaceous materials and as such are associated with traffic fumes, cigarette smoking and wood, coal or oil smoke (WHO, 2010). Wallace et al. conducted detailed continuous monitoring of PAHs in one house for 16 months to determine sources of PAHs amongst other pollutants. They found that neighbourhood wood burning and morning rush hour traffic were the most significant contributors, along with a citronella candle (Wallace, 2013). A study conducted in Krakow, Poland, aimed to assess the relative contribution of indoor and outdoor sources to indoor PAHs, concluding that outdoor sources were more significant, even when there was a smoker in the house (Choi & Spengler, 2014).

Flame retardants

Flame retardants are chemicals that have saved many New Zealand lives due to their ability to slow down the spread of fires (Besis & Samara, 2012). The introduction of many synthetic materials, which were often much more flammable than traditional materials, over the 20th century led to governments worldwide regulating on the flammability of certain products including fabrics. The earliest flame retardants were polychlorinated biphenyls (PCBs) and went out of production in the 1970s after discovery that they were causing health effects in workers, combined with the recognition of their accumulation in the environment. They were widely used in elastic sealants and ceiling tiles, much of which may still exist in New Zealand homes, and recent evidence suggests that inhalation of indoor air and ingestion of indoor dust may continue to comprise a significant exposure route (Harrad et al., 2009).

PCBs have been classed as persistent organic pollutants (POPs) with health effects similar to dioxins, acting as endocrine disrupters and causing developmental issues. In 2013, IARC classed PCBs as group 1 carcinogens (IARC, 2015). PCBs have been measured in Wellington, New Zealand, and although low (range 13–680 ng/g, median 46 ng/g of house dust), they were nevertheless found at measurable levels despite three decades passing since their use in household products was discontinued (Harrad et al., 2009).

PCB flame retardants were widely replaced with polybrominated diphenyl ethers (PBDEs), which were used in large quantities in plastics, textiles, cars, building materials and computers because of their ability to prevent the production of flammable gases (Besis & Samara, 2012). This has led to

a wide exposure, which is in turn leading to increasing literature on adverse health effects (Besis & Samara, 2012). PBDEs are an example of additive BFRs, meaning when they are added to plastics and foams, they mix but don't form chemical bonds. This increases the likelihood of PBDEs leaching out of products compared to reactive BFRs (Kefeni, Okonkwo & Botha, 2014; Besis & Samara, 2012). The wide distribution of these products and their chemical stability mean PBDEs have already become ubiquitous in the environment and are now also considered POPs. The most frequently found PBDE in homes is BDE-209, making up 37–92% of total PBDEs found (Besis & Samara, 2012).

PBDEs are chemically similar to thyroid hormones and can therefore act as endocrine disruptors. PBDEs have been recently found to have neurodevelopmental effects in children exposed in utero (as measured in cord blood), and several studies have linked them with the development of attention deficit disorders (Hoffman et al., 2010; Verner et al., 2015), and they may also affect sexual development, with one study showing cryptorchidism in newborn boys associated with PBDEs in their mother's breast milk (Besis & Samara, 2012).

There is no apparent drop-off in release of PBDEs from products that contain them, and they continue to release contaminants into air until they are removed (Besis & Samara, 2012). Dust is recognised as one of the primary exposure pathways to PBDEs (Besis & Samara, 2012; Coakley et al., 2013) in the population, which means children and especially toddlers have the highest exposures (see Semi-volatile organic compounds). The most sensitive populations are thought to be pregnant mothers, developing foetuses and infants. Breastfeeding infants are also particularly at risk, as PBDEs accumulate in breast tissue (Besis & Samara 2012). High levels have been measured in breast milk in New Zealand ('t Mannetje et al., 2014), and one study found the body load in nursing infants to be higher than their mothers' (Lunder et al., 2010).

Concern over the toxicity of PBDEs accompanied by high measured exposure levels, especially in the United States, led to penta and octa-PBDEs being voluntarily phased out in the US in 2004 and banned in Europe in 2005. The European Union later banned all PBDEs, and in 2009, they were recognised as persistent organic pollutants by the Stockholm Convention, meaning that the body is unable to efficiently metabolise them, leading to bioaccumulation. New Zealand ratified the Stockholm Convention on persistent organic pollutants in December 2004 (Latimer & Keet, 2013), obliging the New Zealand Government to eliminate the use and reduce the release of PBDEs into the environment.

PBDEs have been measured in New Zealand house dust and found at similar levels to those reported from studies in European homes, which are below USEPA exposure limits and an order of magnitude lower than levels measured in US and Canadian homes (Harrad et al., 2008; Coakley et al., 2013). While levels of these contaminants remain low in New Zealand, safe exposures for developing children have not been established. Furthermore, combined effects with other chemical exposures are generally unknown and an important reason to monitor exposures in our population (Knez, 2013).

Restrictions on the use of PBDEs have seen the introduction of alternative flame retardants (AFRs) including organophosphate flame retardants, hexabromocyclododecanes and other novel brominated flame retardants. Hexabromocyclododecanes were listed as POPs on the Stockholm Convention list in 2013 (Kajiwara & Takigami, 2013), and like PCBs and PBDEs before them, little is known about the health effects of these newly introduced chemicals (Ali et al., 2012). Some measurements of novel flame retardants have been undertaken on samples collected in 2007, which found that levels were generally low in New Zealand compared to the small amount of data available internationally. However, these results should be interpreted with caution because of the low numbers and the fact that the chemicals currently in use may have changed since the samples were taken in 2007.

It is interesting to note that flame retardants are not imported directly into New Zealand (Ali et al., 2012). The New Zealand Government does not regulate on the flammability of electronics or furnishings, so flame retardants that reach New Zealand are the result of regulations in the country of manufacture or another exporting nation. Consequently, it is difficult to find out how much of these and other chemicals exist in New Zealand. The Environmental Protection Authority only requires notification of the importation of raw chemicals, and there is no requirement to notify authorities of any chemical that is present in an already manufactured imported item unless it is on the hazardous chemicals list (Ali et al., 2012).

Phthalates

Phthalates are commonly found in indoor air and dust (Bornehag & Nanberg, 2010). They are also known as plasticisers as they are added to plastics to increase flexibility. In products made with PVC, depending on the formulation, they may account for 15–60% of the formulation by weight. PVC flooring commonly has at least 30% phthalates by weight, whereas PVC water piping which is much more rigid, is not made with phthalates. Phthalates are also used as solvents in dyes (Kim et al., 2013) and as detergents in personal care products (Weschler, 2009) as well as in food packaging, cosmetics and clothing (Shu et al., 2014). Four million tonnes of phthalates are consumed globally each year (Lyche et al., 2009). Phthalates added to PVC products are covalently (weakly) bound, so as with additive PBDEs, they are readily released into ambient air. Their increasingly widespread use since the 1920s combined with this volatility has led to them now being ubiquitous in the environment, having been found frequently in freshwater, seawater and in wild animals (Bornehag & Nanberg, 2010; Annamalai & Namasivayam, 2015). Unlike PBDEs and some organopesticides, phthalates are not persistent in the environment and are broken down (metabolised).

Phthalates are considered endocrine-disrupting chemicals (EDCs) (Kim et al., 2013; Lyche et al., 2009; Bornehag & Nanberg, 2010). They can cross the placenta to directly affect foetal development (Lyche et al., 2009). Exposure can affect reproductive development (Lyche et al., 2009), neurodevelopment (Ejaredar et al., 2015) and possibly cause asthma (Bornehag & Nanberg, 2010; Shu et al., 2014; Kim et al., 2007), type 2 diabetes (Chevalier & Fénichel, 2015; Song et al., 2015) and cancer (Gaspar et al., 2014).

A risk assessment study prepared for the New Zealand Ministry of Health found that New Zealand children are at risk of health effects from the ingestion of phthalates in house dust (Ashworth & Cressey, 2014). Their analysis showed the likelihood of wide distribution of toys that did not meet international guidelines due to a lack regulations and monitoring here. Monitoring conducted in Europe suggests that readily available consumer products, including toys, frequently exceed guideline levels of phthalates (0.1% by weight) (Ashworth & Cressey, 2014). An exposure study from the United States of early childhood centres found phthalate levels were generally higher than recommended limits, and for 25% of the children participating, levels were an order of magnitude higher than reproductive risk levels. The author suggested dermal uptake of gas-phase phthalates was the most significant route of exposure followed by inhalation (Gaspar et al., 2014).

Pesticides

Several types of pesticides, including organochlorine pesticides (OCPs), organophosphate pesticides (OPPs) and pyrethrin pesticides (PPs) have been linked with health effects. Evidence is pointing to neurodevelopmental and sexual development disorders being associated with pesticide exposures in utero (Muñoz-Quezada et al., 2013; Engel et al., 2015). OCPs, similar to PCBs and PBDEs, are lipophilic and persistent and accumulate in the body (Gorini et al., 2014). Organochlorine pesticides have been banned, but this persistent nature means they are still found in exposure studies. For example, Dichlorodiphenyldichloroethylene (DDE), a metabolite of DDT, was found at measurable levels in non-occupationally exposed New Zealanders more than four decades after the restriction of their use (Bates et al., 2004). Recent studies have suggested a link between prenatal exposure to pesticides and congenital heart defects, but the evidence is not conclusive (Gorini et al., 2014). Links have been suggested between pesticide exposure and childhood leukaemia (Bailey et al., 2015) as well as neurobehavioural disorders including autism spectrum disorder (Muñoz-Quezada et al., 2013). Improved understanding of genetic and epigenetic heritance have shown that a father's pesticide exposures prior to pregnancy and mother's exposures during pregnancy can impact on the risk profiles for their children right through to adulthood (Gorini et al., 2014). Pesticides are of particular concern as many can cross the blood brain barrier and the placental barrier (Gorini et al., 2014; Muñoz-Quezada et al., 2013). Recognition of this has led to increased attention of parental exposures and children's health outcomes (Bailey et al., 2015). As well as health effects resulting from exposure to agricultural pesticides, there is also a link between household pesticides and childhood leukaemia. A number of case-control studies have been conducted, and a recent meta-analysis conducted by the International Agency for Research on Cancer confirmed the association (Bailey et al., 2015; IARC, 1991).

Recent studies have found pesticides in house dust in the US (Whitehead et al., 2014b) and France (Blanchard et al., 2014). While pesticide residues on fruit and vegetables are monitored regularly, similar surveys have not been undertaken in ambient air or house dust in New Zealand. Whitehead

et al. found that levels of organochlorine pesticides in US house dust were correlated with house age (older houses tended to have higher levels), while pyrethrin pesticides were not (Whitehead et al., 2014b). The levels of pyrethrin in homes with automated insecticide dispensers has not been measured. This could be an interesting line of investigation.

Other endocrine-disrupting chemicals

There is a trend of increasing human exposure to endocrine-disrupting hormones (Yuan et al., 2015). Bisphenol A and triclosan are other chemicals commonly used indoors that have an endocrine-disruption capability. Both are generally thought to be primarily ingested via diet, though there is increasing evidence of dermal uptake, and inhalation is a possible exposure pathway, especially for triclosan, which is often a component of air fresheners and therefore frequently aerosolised (Yuan et al., 2015). There is very little research on indoor air exposure to either of these chemicals, but one recent study suggested a link between exposure via any pathway and failed implantation in in vitro fertilisation (IVF) treatment (Yuan et al., 2015).

Pollutants – solid phase

Particulate matter in air

Although particulate matter (PM) is mostly produced outdoors, studies have shown that we are actually exposed to more outdoor pollutants while indoors. This is due to spending such a large proportion of our time inside and outdoor air infiltrating indoors (Hodas et al., 2012).

The World Health Organization have found that there is no threshold level below which PM in air is safe (WHO, 2006). This means that, even at very low levels of exposure, health is being compromised, and susceptibility to illness is increased. While it would be ideal to eliminate particles, the reality is that many are of natural occurrence, such as a volcanic eruption, and can't be controlled. However, it is highly advisable to reduce manmade sources of particles such as combustion and to filter indoor air to reduce exposures.

The first generation of particle science studies quantified particles by weight. This method grossly underestimates fine and ultrafine particles, which have the greatest adverse health effects. The second generation quantified particles by size fraction, with ultrafine particles only being included in the more sophisticated studies. There are technology implications for monitoring very fine particles, as the instruments are costly and difficult to calibrate. However, with pure quantification, all particles within a size were considered to have the same adverse health effects, regardless of their toxicity. The third generation of particle science is the characterisation of particles. This method requires very sophisticated and hugely expensive equipment but provides very useful data including source apportionment. The analogy is being able to not only count the number of felines in a room but to differentiate the tigers from the tabbies.

Respirable particulate matter

Early studies of New Zealand's ambient air quality showed New Zealand to have relatively high levels of PM_{10} (suspended particles of 10 microns across or smaller) pollution during winter, especially in the South Island where levels frequently exceeded maximum exposure levels recommended by international authorities (Fisher et al., 2007; Kuschel et al., 2012). Source attribution showed that most of this particulate matter was due to the burning of solid fuel for domestic heating.

A smaller but still significant component is due to transport emissions. Agricultural sources of PM require reassessment (see Outdoor air pollution). Industrial sources of particulate matter vary by location but average around 10% of the total volume, slightly less than the average of 12% due to open burning (Kuschel et al., 2012).

In September 2005, the National Standards for Exposure to Air Quality (NESAQ) to PM_{10} were introduced. This saw the introduction of urban air monitoring stations throughout the main centres of New Zealand (although several centres had been monitoring air pollution previously). Since 2006, new regulations have led to significant improvements in petrol formulations (both the benzene and sulphur content of petrol have been regulated and significantly reduced) and improvements in fuel efficiency in both cars and solid fuel heaters as well as other technological improvements. These improvements have, in turn, led to significant reductions in the release of pollution into air. PM_{10} reduced by 8% between 2006 and 2012, and estimates suggest a 14% reduction in premature mortality from an estimated 1,031 deaths annually from anthropogenic sources (Ministry for the Environment and Statistics New Zealand, 2014).

Since the introduction of regulations, North Island emissions of PM_{10} and other common pollutants have generally remained below NESAQ limits (Ministry for the Environment and Statistics New Zealand, 2014; Mitchell, 2012). Some South Island towns' emissions profiles, however, are primarily influenced by wood burning for home heating (Scott, 2012) and consistently exceeded limits, frequently reaching double and occasionally triple the 24-hour limit (Scott, 2012; Mallet, 2014). This will be of significance with new enforcement of regulations in force in 2016 specifying a maximum of three exceedances annually. It is unlikely that these will be achieved in South Island urban centres. Christchurch, for example, needs a 47% reduction in domestic emissions to meet the 2020 target of one exceedance per year (Mallet, 2012). This estimate does not take into account any effect climate change may have on the meteorology of the area (Mallet, 2012). While it is recognised that one positive impact of the Christchurch earthquakes has been an increased attrition rate of older, more polluting fuel burners, this effect is not enough to reach the reduction levels needed to meet targets (Mallet, 2012).

Fine particulate matter

In recent years, much work internationally has gone into characterising PM to better understand the specific components that pose the greatest health risks. This work has shown that particulate matter

2.5 microns or smaller ($PM_{2.5}$) is able to deposit deeper into the respiratory system than PM_{10}, meaning it has increased risk of causing health issues (Ostro et al., 2015; Hodas et al., 2012).

Other work has assessed the relative carcinogenicity of $PM_{2.5}$, finding that $PM_{2.5}$ measured in a retirement home and a school dormitory had almost twice the level of trace metalloids and carcinogenic PAHs bound to it as the PM_{10} measured in the same environments (Hassanvand et al., 2015).

Household characteristics associated with higher $PM_{2.5}$ include smoking indoors and the presence of a cat or dog, while higher PM_{10} was associated with urban location, location on an arterial road and use of a gas stove (Simons et al., 2007). Cooking, especially frying and roasting, was found to be a strong source of particles between the size of 0.3 and 10 microns in a study that took continuous measurements in one US house over 16 months, as was burning candles and incense (Wallace, 2000).

Monitoring of particulate matter in New Zealand performed over the last decade, particularly prior to 2006, has generally not included measurements of $PM_{2.5}$ (Ministry for the Environment and Statistics New Zealand, 2014). Where these levels were measured in Christchurch during 2013, measurements showed that $PM_{2.5}$ exceeded NESAQ limits 22 times, while PM_{10} only exceeded limits 15 times over the same period (Mallet, 2014). This suggests that new limits, yet to be enforced, may not sufficiently regulate $PM_{2.5}$, the fraction most important to adverse health outcomes (Mallet, 2014). In order to create better assessment schedules, regular monitoring of $PM_{2.5}$ is essential. Recent analysis using modelling of $PM_{2.5}$ caused by agriculture has shown that it is a major contributor to PM mortality risks (Lelieveld et al., 2015).

Ultrafine particles

Ultrafine particles (UFPs) are defined as particles with a diameter of 0.1 µm or smaller (Spilak et al., 2014). This partition of particulates is the smallest contribution to total PM by mass but the largest contribution by number (Diapouli, Chaloulakou & Spyrellis, 2007). In urban areas such as Brisbane, UFPs may represent 80% of the particles in ambient air (Jamriska et al., 1999). This means that regulations based on PM_{10} or even $PM_{2.5}$ are unlikely to restrict UFPs sufficiently.

Following the trend of the smaller the particle size the larger the health effect, UFPs seem likely to be the most dangerous to health as the particles are small enough to cross cell walls and to enter directly into the bloodstream and organs after respiration (Ostro et al., 2015; Ham & Kleeman, 2011). This changes the impact of their effects, drastically increasing the likely risk of health effects compared to larger particles, although epidemiological studies on the specific health effects of UFPs are not yet available (Kumar, Verma & Srivastava, 2013).

Ultrafine particles are produced almost exclusively through combustion processes, so natural sources of PM such as salt, soil or rock particles are unlikely to contribute to this fraction. Many of the carcinogenic components of combustion reactions, however, are found in this submicron fraction

of particles, including toxic metals and PAHs (Diapouli et al., 2007). A detailed and large-scale study from the USA found wood smoke was the greatest contributor to urban UFPs, followed by traffic exhaust (Ostro et al., 2015). Indoor sources include smoking, cooking (especially frying and baking), the use of gas appliances, hot surfaces, burning candles and fireplaces (Spilak et al., 2014). While fireplaces have been improved significantly over past decades, recent studies have shown that even new, low-emission wood stoves contribute to indoor UFPs (Salthammer et al., 2014).

A study conducted in schools in Athens found indoor levels of UFPs ranged between 50–90% of outdoor levels if there were no indoor sources (Diapouli et al., 2007). These results are consistent with a number of other studies (Qing et al., 2005; Barraza et al., 2014). When there are indoor sources, indoor UFPs are approximately double outdoor levels on average (Qing et al., 2005; Diapouli et al., 2007; Barraza et al., 2014). A study conducted in New Zealand schools found levels in classrooms where half the outdoor levels when additional ventilation was installed in the classrooms (Boulic, 2012).

Fine particles are more effective at entering buildings from outdoors than larger particles (Jones et al., 2000), which is related to the fact that they have lower deposition rates so are less likely stick to the edges of cracks and openings in a building's structure, and once airborne, they remain so for a longer period of time compared to larger particles. UFPs can remain suspended almost indefinitely, and deposition rates are virtually unaffected by gravity, Brownian motion being the main driver of UFP deposition (Spilak et al., 2014). UFP levels measured indoors are highly correlated with activity, the presence of pets and hard floors (Spilak et al., 2014; Diapouli et al., 2007). These associations are likely to be a reflection of UFPs' resistance to deposition rather than these being indoors sources of UFP.

In New Zealand, no research has been reported on indoor levels of UFPs, most probably due to the expense and specialisation of the equipment required to measure UFPs .

Arsenic

Arsenic in New Zealand air is present largely due to the burning of timber treated with copper chromium arsenate (CCA) preservatives and certain industrial processes (Ministry for the Environment and Statistics New Zealand, 2014). In some localities, this can become a significant health issue, such as in Wainuiomata, where air monitoring in 2012 showed ambient levels that exceeded New Zealand Government guidelines (Ministry for the Environment and Statistics New Zealand, 2014). Arsenic targets lungs, heart and nervous system, kidneys and liver and can lead to the development of cancer (Ministry for the Environment and Statistics New Zealand, 2014; Naujokas et al., 2013).

Lead

Lead poisoning can cause brain damage, heart damage and, in some cases, can be fatal. Small children are most at risk from consuming particles with lead due to their activities involving hand to mouth contact with soil and paint chips and their play at ground level. They also have the double whammy of being most susceptible to the effects of lead poisoning, which can include learning and memory difficulties. Children and adults with lead poisoning can experience stomach pains, sleeping difficulties, constipation and weakness. Lead poisoning is a serious hazard for children and causes significant cognitive and behavioral impairment for life.

Lead used to be commonly found in ambient air, as it was a component in petrol and paint. New Zealand petrol has been lead-free since 1996 (Ministry for the Environment and Statistics New Zealand, 2014), and since then, virtually no air monitoring has been conducted, but ambient outdoor levels are presumed to be well below guidelines (Ministry for the Environment and Statistics New Zealand, 2014). Homes near industrial sources or homes that have exposed lead paint may have significant concentrations of the pollutant in their outdoor environs.

Lead used to be a major ingredient in paint, used for both indoor and outdoor surfaces. Most homes and schools constructed prior to 1965 have high levels of lead from the lead-based paints. Lead was especially prevalent in paint manufactured pre-1945. New Zealand was one of the last countries to phase out lead-based paint. While repainting can cover the lead paint and limit exposure, renovations or old peeling paint present a well known health risk.

An important pathway for lead is when it is carried into the home on shoes and clothes from outdoor soils. Elevated concentrations of lead, as a result of corroding lead-based paint, have been frequently detected in house dust in New Zealand between 2007 and 2010 (Keet, 2011). Data indicated that the elevated concentrations of lead were potentially associated with lead sources in and outside the house. Based on the measurements of lead in painted surfaces, it was found that lead-based paint was the major source of lead.

Lead particles are relatively heavy and do not travel far via air from their source. This means that lead may be concentrated in small areas, and broad-scale monitoring may not be relevant to actual exposures. The heaviness is relevant in the indoor environment as lead particles will not remain suspended for long and will tend to accumulate in dust on or near the floor, meaning that infants and toddlers may have significantly higher exposures to this toxin than older people. Lead may persist in soils for many years, so even if an industrial source is no longer producing lead fumes, exposure may still be occurring in nearby homes and public spaces. Monitoring of exposure to small children near industrial sources, past and present, and in houses with lead paint would be valuable. A preliminary cost-benefit analysis of social costs of lead exposure in New Zealand was conducted in an MBA thesis by Keet (2015), which found there are significant social costs of lead and further monitoring was required.

Environmental tobacco smoke

Tobacco smoking indoors has reduced dramatically in New Zealand over the past two decades due to various public health strategies, and there is no doubt that this has had significant positive impact on New Zealanders' exposure to indoor environmental tobacco smoke (ETS). However, it remains an important contributor to ill health in certain sectors of society, especially in Māori, Pacific and low socio-economic groups (Ministry of Health, 2014). Tobacco control and improving the housing stock have been identified as the two most important factors in improving respiratory illness in New Zealand (Kirby, 2015).

Although tobacco smoking and second-hand exposure to tobacco smoke have reduced significantly in New Zealand over the past 10 years (Ministry of Health, 2014), ETS exposure remains one of the most significant causes of pollution exposure in New Zealand homes (Kirby, 2015). Tobacco smoke releases numerous pollutants into indoor air, including benzene, formaldehyde, PAHs, carbon monoxide and particulates including UFPs (Hulin et al., 2012). Tobacco smoke exposure is the leading cause of preventable death worldwide (Bernstein et al., 2008). A meta-analysis has shown children with ETS exposure had double the risk of wheeze compared with children without ETS exposure (Bernstein et al., 2008), and studies into asthma causation have suggested ETS is related to a 40–200% increased risk (Bernstein et al., 2008).

Tobacco smoke has been implicated as a modifier of other indoor air exposures, especially biological ones. One recent study measured the relationship between endotoxin exposure and other indoor pollutants on asthma exacerbations. Although no overall association was found between asthma outcomes and endotoxin, when there was a co-exposure with airborne nicotine, endotoxin was associated with worse asthma symptoms. The opposite effect was reported for co-exposure with high relative NO_2, in the presence of which endotoxin was protective, and with low NO_2, endotoxin was associated with worse asthma symptoms. (Matsui, 2014).

A Finnish study reported a synergistic interaction between parental history of asthma, second-hand smoke (SHS) exposure and risk of adult-onset asthma in a population-based incident case-control study. Specifically, the odds ratios for asthma were 1.97 for SHS exposure and 2.64 for parental asthma but 12.69 for their joint effect (Lajunen, Jaakkola & Jaakkola, 2013).

Another study from Brittany, an area of France with a similar climate to New Zealand, showed that regular smoking inside was associated with increased indoor fungi of the *Aspergillus* genera (Dallongeville et al., 2015). Smoking indoors has also been associated with higher endotoxin than found in non-smoking houses (Chen et al., 2012). The mechanism to explain this association of tobacco smoke with increased biological exposures is currently unexplained. A study to investigate if tobacco smoke indoors is related to live bacteria or bacterial fragments such as endotoxin would be valuable and could help explain the aforementioned mechanism.

Although, not directly explored in the literature, there is some evidence to suggest a link between ETS exposure and bacterial skin infections (Bergmann & Ring, 2014; Keten et al., 2015; Mishra et al., 2015; Claessen et al., 2015). This could be another interesting investigation, as New Zealand has double the rate of hospitalisations for skin infections than either Australia or the USA.

According to data from the Ministry of Health, 45,000 children and 106,000 adults were exposed to SHS in their homes in New Zealand in 2012/13 (Ministry of Health, 2014). SHS exposure is highly differentiated by socio-economic status and ethnicity, with over 10% of children in the most deprived neighbourhoods exposed in their homes, which is double the national average of 5% of children nationwide. Approximately 9% of Māori children and 6.4% of Pacific children are exposed at home (Ministry of Health, 2014).

Overall exposure at home to second-hand tobacco smoke has approximately halved since 2006/07 (Ministry of Health, 2014). These reductions are due to a concerted campaign of legislation, increased tobacco taxes and education, alongside cessation support, which have seen New Zealand cited as a world leader in tobacco smoke exposure reduction (Farmer et al., 2014). Residual exposure may need different approaches in order to further improve these results, as some groups within the population have seen little change over this period. In the general population, there are more male smokers than female smokers. However, in the Māori population, this trend is reversed, with significantly higher rates of smoking among females – 42% compared with 37% of Māori males. This same reversal of gender trend is seen among 20–24-year-old New Zealanders, with higher rates for female than male current smokers in this age group.

These factors may be related to the fact that children under 15 are significantly more likely to be exposed to SHS at home than adult non-smoking New Zealanders (5% versus 3.7%). A reduction in smoking rates among Māori women and young non-Māori New Zealand women may therefore have a more significant beneficial effect on the health of the population by reducing SHS exposures in children than a reduction in the population as a whole. A New Zealand study assessing predictors for smoking found that parental smoking was a significant predictor for teenagers taking up smoking (Scragg, Laugesen & Robinson, 2003).

A study assessing predictors of smoking indoors in homes with young children in a rural German population found that smoking indoors was significantly more likely in homes where children did not attend early childhood education centres and in homes that had no private outdoor area (Ulbricht et al., 2014). Replication of this research in New Zealand could be useful in helping design interventions that effectively target vulnerable populations. Including sheltered outdoor smoking areas may need to considered in medium-density housing designs to encourage outdoor smoking.

Biological contaminants

Indoor dampness and mould are known to be associated with airways inflammation, nasal congestion, wheezing, chest tightness, coughing and throat irritation. Prolonged exposure to high levels of indoor dampness and mould are associated with reduced lung function and chronic health problems such as asthma. Those who already suffer from asthma and allergies are more likely to have more severe symptoms when exposed. According to the World Health Organization, a considerable proportion of the world's 300 million cases of childhood asthma is attributable to exposure to indoor dampness and mould.

In New Zealand, the prevalence of indoor dampness and mould is high compared to other Western societies, and this may in part play a role in the high prevalence of asthma in New Zealand, where it is known to affect one in four children and one in five adults. Indoor air, in any particular building, can be thought of as a microbiome (Nazaroff, 2014; Lax et al., 2014), with numerous environmental characteristics and conditions contributing to a home's or building's unique microbial signature. For example, high indoor humidity, which itself is related to structural factors such as ventilation, house size, construction, indoor sources and heating, predicts the rate of proliferation of fungi and the survival rate of aerosolised viruses and bacteria (Nazaroff, 2014).

High indoor humidity has also been shown to increase the release of certain chemical compounds from building components and furnishings, including formaldehyde and phthalates (Mendell, 2007; Bornehag et al., 2005). These chemicals, in turn, may impact on the survival or proliferation attributes of biological components of indoor air, although this has been understood. Kim et al. found MVOCs were associated with airborne phthalates in schools in Sweden but not with airborne mould spores (Kim et al., 2007).

Measurements have shown that human inhabitants are the primary vector in bacterial communities within buildings. Lax et al. showed that humans' microbial variation predicted that of their homes, and when humans moved house, the new house's microbial community quickly adapted to align with that of their previous dwelling (Lax et al., 2014).

Bioaerosols, which are defined as any suspended particles in air that are of biological origin, are typically highly concentrated, both indoors and out. One study that took personal air samples from 81 teachers over a 24-hour period found total bacterial aerosol numbers in the range of 10,000 to 100,000 per cubic metre and fungal aerosols that averaged close to 10,000 per cubic metre (Toivola, Nevalainen & Alm, 2004). Only a small fraction were viable cells. The bulk were dead cells or fragments, which nevertheless may cause health effects via allergic responses (Douwes et al., 2003).

During the flu season, virus levels have been measured indoors at 5,800–37,000 genome copies per cubic metre (Chen et al., 2009), suggesting a veritable soup of biologic material in our breathing zones. While fungal (mould) spores and fragments are generally higher outdoors, bacterial

components are usually higher indoors (Nazaroff, 2014; Chen & Zhao, 2011), suggesting that occupants are the major source of bacteria in homes. Another study that conducted comprehensive measurements of many taxa of bacteria and fungi and compared indoor to outdoor levels to identify source also found that most fungi indoors were more prevalent outdoors than indoors, suggesting they enter from the outdoor environment, and they also found that bacteria were predicted by occupants, including dogs, cats and interestingly, a slight but consistent variation in the type of bacteria by gender of occupants (Barberán et al., 2015).

An innovative study into the pathogenesis of asthma analysed (along with fungi and bacteria) yeasts, which are unicellular forms of fungi, and found that those children who had higher yeast exposure at age 2–5 months had less asthma or wheeze at age 13 (Behbod et al., 2015). This protective effect of yeast was only found in measurements of floor dust and not the air measurements, and the authors suggest a possible differential reaction via ingestion versus breathing. Nevertheless, assessing the source of such yeasts could prove valuable. Other illnesses known to be caused by bacteria or fungi found in the home environment include legionnaires' disease, hypersensitivity pneumonitis and skin conditions (Turpin, 2014).

Eliminating all biological artifacts in the indoor air is not ideal, as epidemiological studies have shown that overcrowded, unhygienic living conditions and the presence of bacterial endotoxin are associated with lower prevalence of allergic conditions including eczema, hayfever and asthma (Douwes et al., 2006; WHO, 2009). This is known as the hygiene hypothesis in that humans need some exposure to biological contaminants in order to stimulate their immune system, especially in the first 5 years of life. However, if an individual's immune system becomes overloaded, they can experience chronic asthma, eczema or allergies. Significant improvements in asthma severity can be gained from spending time in a very dry and hence mould-free respiratory hospital in the Swiss Alps (El Margoushy, 2013).

On the other hand, dampness is consistently associated with poorer respiratory health outcomes, and the mechanism for this is generally agreed to be higher exposure to moulds and bacteria, which thrive in damp conditions, along with other dampness-mediated microbial exposures including dust mite and cockroach allergens.

In terms of controlling bioaerosols in domestic or indoor air, there are two methods of achieving this. The first is to reduce their proliferation. Moisture is the only limiting agent for mould growth, and controlling mould is best achieved by controlling indoor humidity levels (reducing dampness). The second is through ventilation, whether natural or mechanical. One important factor to bear in mind regarding mechanical ventilation is the hygiene of the filters. Air conditioning units are designed to be regularly maintained, and their filters need to be changed regularly to avoid microbial matter being introduced into the indoor air from dirty filters. Most manufacturers recommend filters are cleaned or changed monthly. Furthermore, filters only eliminate a fraction of the PM contamination

in air, and multiple passes of contaminated air through filters may be needed to significantly reduce their concentration (Hodas et al., 2012) (see Ventilation). The same comments on filters apply to heat pumps, even though these recirculate the indoor air and don't provide ventilation.

Mould

Fungal infections kill 1 million people around the world every year, which is more deaths than caused by either breast cancer or malaria. However, fungi exposures can slide under the radar. Fungi are very tough, and they are difficult to kill once established in the indoor environment. They are also difficult to treat if they become established inside the human body, and they can manipulate the immune system and thus prevent them being recognised. It is estimated that fungi infections are severely underdiagnosed. There are no vaccines and limited treatments.

There are more than 5 million types of fungi, but little is known about the health effects of most species. Three main groups of fungi cause the most deaths. Some species of the *Aspergillus* family affect the lungs, with some species being able to grow in lung tissue. There are limited treatments for aspergilloses. *Cryptococcus* species mainly attack brain tissue. Other species have varying degrees of toxigenic, carcinogenic or allergenic effects.

There is strong evidence of links between asthma, allergies and respiratory illness and damp living conditions, visible mould or signs of water damage (Mendell et al., 2011; WHO, 2009). The mechanism causing these associations has been assumed to be caused by an allergic sensitivity or a non-allergic immunologic response to mould fragments such as glucans or mould spores (Douwes et al., 2003). However, evidence directly linking quantitative mould measurements and health effects remains tenuous, which is in part due to the fact that quantifying mould in indoor environments presents difficulties (see Exposure assessments) (Mendell et al., 2011; Chen et al., 2014a; Kanchongkittiphon et al., 2015).

A recent review and meta-analysis by Sharpe et al. support an association between *Penicillium, Aspergillus, Cladosporium* and *Alternaria* strains of mould and asthma exacerbation (Sharpe et al., 2015). These genera are the most frequently measured in asthma studies, so this study does not rule out other genera (Crawford et al., 2015).

MVOCs are released by moulds. A few studies have found associations between MVOCs and asthma or allergy (Kim et al., 2007; Elke et al., 1999). However, Kim et al. also found that MVOCs were not associated with indoor airborne mould spores. This relationship is in need of clarification (Kim et al., 2007) but is presumably as not all species produce MVOC metabolites and MVOCs as well as that sporulation is only produced under certain conditions.

MVOCs may also be important in relation to mouldy odour, which has been associated with reduced lung function in healthy non-asthmatic adults (Hernberg et al., 2014). This relationship between MVOCs and mouldy odour has not been assessed directly.

Recently, in response to the difficulty of making quantitative associations between mould particles and health effects (Mendell et al., 2011), studies have focused on validating the relationship between visible mould, mouldy odour, water or flood damage and measurements of airborne and dustborne fungal allergens. These studies generally find positive associations between signs of dampness and mould, and mould measurements. Ceylan et al. found the number of occupants in a home to be positively associated with number of airborne mould spores (Ceylan et al., 2013).

Several studies have found *Penicillium* is the mould genus most strongly associated with indoor visible mould (Crawford et al., 2015; Dallongeville et al., 2015). Higher indoor relative humidity (RH) is also most closely associated with airborne *Penicillium* levels, while mouldy odour was most closely associated with total airborne fungi (Crawford et al., 2015; Quansah et al., 2012). Aguiar et al. found that *Penicillium* species were the most prevalent indoor moulds in winter in measurements taken in an old persons' residential facility, while in summer, species of the genus *Cladosporium* were the most prevalent (Aguiar et al., 2014). This is in line with Dallongeville et al., who showed increased open bedroom window time was associated with decreased *Aspergillus* and increased *Cladosporium* (Dallongeville et al., 2015).

While *Penicillium* species have been associated most often with visible indoor mould (Crawford et al., 2015; Dallongeville et al., 2015) and higher indoor RH (Crawford et al., 2015), studies measuring several genera point to *Cladosporium* having the strongest effect on lung function and asthma exacerbations (Chen et al., 2014a; Behbod et al., 2013; Sharpe et al., 2015). This is important because Cladosporium is associated more strongly with outdoor air than indoor air (Crawford et al., 2015; Dallongeville et al., 2015). Furthermore, Crawford et al. found one of the strongest predictors of indoor fungi was outdoor levels of fungi, suggesting epidemiological studies assessing the health effects of moulds should pay more attention to season and outdoor fungal levels.

Further attention to the differences in relation to health effects between genera of mould would also be valuable. For a detailed analysis of moulds, yeasts and bacteria and their determinants in indoor air see, Crawford et al. (2015). One finding of interest of this study is that non-sporulating (not forming spores) species of mould were prevalent in the indoor environment, an important consideration in relation to fungal colony growth as a method of calculating fungi prevalence, as the standard methods will not account for these species (Pounder et al., 2007). Mould species may be non-sporulating because conditions are not conducive to sporulation (Kung'u, 2004) or because they never form spores, in which case, they are known as sterile fungi. This means they are under-represented if culture methods of analysis are used in an investigation – this can lead to a false negative.

Another interesting finding from Crawford et al. was the effect of snow, which suppresses levels of outdoor fungi when it is in the form of sitting snow, but when there are periods of thawing and periodic snowfall, ratios between indoor and outdoor mould may fluctuate widely (Crawford et al.,

2015). The study of Crawford et al. focused on low-income urban households in New York, USA. Replicating these methods in different environments would be informative. Another interesting analysis of microbes in indoor dust has been conducted by Barberán et al. (2015).

Dallongeville et al. found regular indoor smoking to be associated with increased moulds of the *Aspergillus* genera (Dallongeville et al., 2015). Understanding the cause of this association could be valuable as *Aspergillus* fungi can attack lung tissue.

An evaluation of various antifungal agents used for mould decontamination compared two industrial disinfectants, 70% ethanol, vinegar and tea-tree oil for efficiency in inhibiting fungal growth. This study found tea-tree oil the most efficient while 70% ethanol and vinegar were the least efficient (Rogawansamy et al., 2015).

A recent review of studies assessing modifiable indoor environment factors and their association with elevated indoor fungal measurements reported numerous conflicting findings, highlighting the complexity of factors that may impact increased fungal exposures and their assessment (Sharpe et al., 2014). Sharpe et al. concluded that behaviour changes can be as effective as modifying the built environment in terms of reducing mould exposure for occupants.

Endotoxin

Endotoxin has presented a particularly complex relationship with health effects in epidemiological studies. Endotoxin is a component of certain gram-negative bacteria and is frequently found in house dust and in schools and workplaces (Wickens et al., 2003; Douwes et al., 2003). Exposure to high levels of endotoxin is known to exacerbate asthma symptoms (Thorne et al., 2015; Kanchongkittiphon et al., 2015). However, other research suggests that endotoxin has a protective effect over sensitisation to allergens (Douwes et al., 2006; Doreswamy & Peden, 2011; Kanchongkittiphon et al., 2015).

Endotoxin is found at particularly high levels on farms and has been proposed as the explanatory factor in the remarkably low levels of asthma and allergy found amongst children brought up on farms (Douwes et al., 2006). Endotoxin, a lipopolysaccharide, acts as a chemical trigger for the body's innate immune response (Chen et al., 2012). In occupational settings, high levels of endotoxin are known to trigger asthma in adults (Doreswamy & Peden, 2011). Endotoxin has been studied in association with asthma onset in numerous studies looking at children and infants where results have been conflicting, some studies showing that endotoxin is a predictor of asthma or wheeze (Rabinovitch et al., 2005; Thorne et al., 2015) with others showing a distinct protective effect from exposure to high levels during infancy (Gereda et al., 2000; Douwes et al., 2006).

A multi-centre study based in Europe was used to assess indoor endotoxin prevalence according to house characteristics and geographical location and showed that subtropical localities had

consistently higher mean endotoxin, while the colder climates had the lowest mean endotoxin (Chen et al., 2012). Higher levels of occupancy, a house older than 30 years and visible mould were also associated with higher endotoxin as was smoking indoors (Chen et al., 2012). For a comprehensive assessment of house characteristics in New Zealand homes associated with endotoxin, see Wickens et al. (2003).

A study by Matsui et al. showed a complex interaction between endotoxin, ETS and NO_2 exposures in the home, where endotoxin was protective for asthma in combination with some exposures and a risk factor when in combination with other exposures (Matsui et al., 2013). Further research into interactive effects with endotoxin and other common indoor exposures may help with understanding why evidence surrounding endotoxin exposure and respiratory health remains conflicting.

Rabinovitch et al. took daily personal exposure measurements for endotoxin exposure in asthmatic children in urban USA and found that these were positively associated with asthma severity scores and evening FEV1 but not with morning FEV1. This suggests that respiratory symptoms were worsening during the course of the day and improving overnight. Monitoring was conducted over 24 hours, with personal monitors being left switched on next to the bed at night (Rabinovitch et al., 2005). It is as if the immune system was reacting differently to endotoxin during sleep than while awake. Although the function of sleep remains unclear (Ibarra-Coronado et al., 2015), it has been shown that sleep is induced by cytokines, constituents of the immune system (Majde & Krueger, 2005). Furthermore, there is evidence that the expression of immune cells and modulators, such as neurotransmitters, hormones and cytokines, is modulated during sleep (Ibarra-Coronado et al., 2015).

Research into the potential differences between waking and sleeping immune functioning could prove valuable. If immunity during sleep switched into a calibration mode, where exposures while asleep that predicted waking exposures would see the least adverse health effects, this could explain results found by Jacobs et al., who found that higher school exposure to endotoxin was positively associated with non-atopic asthma while home exposure to endotoxin was inversely associated with asthma (Jacobs et al., 2013).

According to Gereda et al., "Reductions in allergen sensitisation and atopic disease have been found in children of farmers, children with pigs, dogs, or cats in their homes, children raised in day-care from an early age, and children of large families. In all of these environmental settings, gram-negative bacteria, and their cell-wall component endotoxin, may exist in abundance." (Gereda et al., 2000). In all of these settings, also, it is arguable that individuals will sleep with higher levels of endotoxin than the general population.

There are well established guidelines for remediating mould from buildings. A leading document is the guidelines produced by the American Industrial Hygiene Association (Prezant et al., 2008).

However, an Australian guideline has been produced (Kemp & Neumeister-Kemp, 2010), and there are several European guidelines. These guidelines define rigorous testing, remediation and clearance testing procedures. However, there is anecdotal evidence that internationally accepted procedures for mould remediation are ignored in New Zealand. This can expose both workers and building occupants to unacceptably high levels of mould. Research into the practices of mould remediation within the New Zealand context is required.

Pets and pest animals

Dallongeville et al. found that dust mite allergen was associated with season, being found at higher concentrations in colder seasons, (Dallongeville et al., 2015), but the same effect was not found for dog or cat allergens. This may be an artifact of ventilation practices, as dust mites are unlikely to cause people to open windows in winter, while dogs and cats will. Dog and cat allergens are highly predicted by a presence of those animals in the house. These allergens are still found in houses without a dog or cat as well as in workplaces and schools (at approximately 25–30% of the level found in houses with pets (Wickens et al., 1997a; Dallongeville et al., 2015).

Dust mites

Dust mites received much research attention between 1990 and 2010 in asthma epidemiology studies. Dust mite allergen prevalence in bedding (an environment in which people spend a considerable amount of their lives and in very close proximity to their breathing zone) and the sensitisation of many asthmatics provided hope that this factor would be the most significant one in asthma pathogenesis and could offer an effective strategy for reduction in asthma worldwide (Institute of Medicine, 2000).

These hopes were supported by evidence of dose-response relationships between dust mite allergen and health effects (Institute of Medicine, 2000), because asthma was found to be exacerbated by dust mite allergen even in individuals not sensitised (allergic) (Mendell, 2007) and because sensitisation to dust mites was shown to predict increased medication use (Wang et al., 2009). Since these earlier results, a number of confounding factors have emerged, which have had the effect of reducing the relative importance of dust mites in asthma epidemiology but not of removing the association (Kanchongkittiphon et al., 2015).

It appears that allergens in bedding are not as important in terms of illness as previously thought. A highly cited Cochrane review of bedding intervention studies to reduce dust mites and allergens, which showed morning peak flow spirometry as the most frequent outcome measure within the review, found no clinical benefit from dust mite reduction interventions (Gøtzsche & Johansen, 2008). Other evaluations of bedding interventions have also found that bedding seems relatively minor to asthma conditions (van Strien et al., 2003; Gehring et al., 2012; van Boven, 2014; Wright & Phipatanakul, 2014). For example, an intervention placing mite-proof mattress coverings on

children's and parents' beds in a birth cohort followed for 8 years showed non-significant increases in wheeze, asthma, allergic sensitisation and sensitisation for dust mite allergen for the intervention group compared to the control group, who received a cotton placebo mattress covering (Gehring et al., 2012).

Research is currently being conducted by the Otago Medical School on the effects of bedding and bedroom heating on newborn infants.

Following the hypothesis from above (see Endotoxin) that the immune system may function differently during sleep, such results may be expected if immunity during sleep switched into a calibration mode, where exposures while asleep that predicted waking exposures would see the least adverse health effects.

This could explain why studies of asthma and dust mite exposure consistently fail to find a relationship between dust mite levels in the home and asthma severity (Smith et al., 2011). A study from New Zealand that appears to support this hypothesis was conducted by Smith et al., who conducted skin prick tests using allergen derived from the participants' own bedding and found that, out of 29 house dust mite-sensitive participants in their study, 25 showed no allergic response to dust from their own bedding. Out of the 29 participants, 22 had current symptoms of allergy, and this group was found to have higher dust mite levels in their bedding, but this had no effect on their allergic response to their own bedding dust (Smith et al., 2011).

A review from the Wellington Asthma Research Group compared bedding types and found that feather pillows and covers may be protective for allergy compared with synthetic pillows and covers and hypothesised that the cause could be the tighter weave used in feather pillow coverings, which they showed in a simple experiment was impervious to dust mite penetration. Such tighter weave fabric is also used in some newer synthetic pillows, and this would be a useful point to follow up (Siebers & Crane, 2011).

Research has shown that dust mite proliferation is effectively restricted in houses where mean daily RH is 50% or lower, even if RH rises significantly higher for up to 8 hours of the day (Arlian, Neal & Vyszenski-Moher, 1999). New Zealand has among the highest levels of dust mites in houses and bedding in the world, and it is likely that climate is related to this given New Zealand's high, year-round relative humidity (Arlian et al., 1999).

It is worth noting that the climate preferred by dust mites is also ideal for many mould species, and it is plausible that mould spores provide a food source for dust mites. Both mould and dust mites proliferate in high humidity environments – controlling moisture is the limiting agent.

Dust mites only require a high relative humidity for 1 hour per day to stay fully hydrated, and males will shelter a breeding female to prevent her from becoming dehydrated.

Cockroaches

Cockroaches, like dust mites, prefer damp living conditions and cannot survive in dry indoor conditions. For this reason, the presence of cockroaches is highly correlated with the presence of mould, especially *Penicillium*, and with high indoor RH (Crawford et al., 2015). Sensitisation to cockroach allergen has been associated with increased asthma morbidity (Wang et al., 2009), and studies have shown that inner-city children in the US sensitised and exposed to cockroach allergen have the highest morbidity (worst symptoms) (Gaffin & Phipatanakul, 2009). Cockroach allergen has also been associated with increased general allergen sensitisation (Kass et al., 2009). Pest control systems that rely on chemical controls may introduce other pollutants into the indoor environment (Kass et al., 2009) (see Pesticides) and be less permanent than eradication via humidity control.

Rodents

Like cockroaches, mouse allergen has been associated with increased risk of sensitisation to allergens in general (Kass et al., 2009). In sensitised children, mouse allergen exposure is strongly correlated with increased asthma morbidity (Kanchongkittiphon et al., 2014).

Cats

Having a pet cat in the home has been found to be associated with higher measurements of PM (Simons et al., 2007), indoor mould species (Dallongeville et al., 2015; Sharpe et al., 2014) and endotoxin (Doreswamy & Peden, 2011), although this association with endotoxin is less consistent than the association with dogs (Wickens et al., 2003; Doreswamy & Peden, 2011). Exposure to cat allergen in infancy has also been shown to be strongly correlated with atopy at age 6 in a large multi-centre birth cohort study (Gaffin & Phipatanakul, 2009). Cat allergen is frequently found in public places and houses, even those without pet cats in residence (Wickens et al., 1997b, Dallongeville et al., 2015). Cat ownership is high in New Zealand compared to other similar countries at around 60% of households (Wickens et al., 1997b).

Dogs

While it is clear that, in sensitised children, exposure to dog allergens in the home can exacerbate asthma symptoms, there is suggestive evidence that, even in adults and non-sensitised individuals, dog allergen at home can exacerbate asthma (Kanchongkittiphon et al., 2015). Presence of a dog has been associated with decreased Penicillium and increased Cladosporium (Sharpe et al., 2014). However, given that similar associations are found for opening windows (see Mould), this association may be confounded by ventilation habits. Dogs are a predictor of higher endotoxin in homes.

Exposure assessments

Indoor air pollution exposure assessments are complex, and many pollutants lack universally accepted measurement techniques. Studies are often not comparable due to differing

methodologies. For example, a meta-analysis of PM exposure by Morawksa et al. (2013) found that, out of eight studies that measured PM both indoors and out, only two used the same lower size limit for particles to describe PM concentrations, limiting comparability.

Many studies rely on proxies instead of actual measurement, such as proximity to pollution sources (with or without modelling) or use of polluting appliances (for example, presence of a gas stove to represent NO_2 exposure) to reduce project costs. While proxy studies are relatively low cost and highly feasible, they do have limitations on the conclusions that can be drawn from the data. Improved accessibility of existing data sets (such as anonymised hospitalisation records, household energy use and ambient air pollution) is enabling relationships between building characteristics and health to be explored more cost-effectively.

In the last decade, more attention has been paid to quantitative assessment of indoor air pollutants (Hulin et al., 2012), and there has been significant progress in building detailed characterisations of indoor air quality (Turpin et al., 2007; Bari et al., 2015; Bruinen de Bruin et al., 2008; Esplugues et al., 2010a). However, studies conducted overseas need to be tested under New Zealand construction typologies before they can be adopted.

Assessment is complex, not only because there are virtually always more pollutants present in any indoor environment than can practically be measured, some (or even many) of which may be interacting with one another, but also because of the complexity of human behaviour. Exposure is sometimes mistakenly assumed to be the same as pollutant concentration (Morawksa et al., 2013). This is only true when using personal sampling methods (breathing zone air sampling). Researchers comparing static measurements in homes or workplaces with personal exposures have found such measurements do not predict individual exposure, even when static measurements are taken at a variety of locations the individual visits and use time-weighted evaluation methods (Toivola et al., 2004; Rabinovitch et al., 2005). So it appears that we may live out our lives within our own 'personal cloud' of pollutants of generally higher concentration than ambient levels (Toivola et al., 2004; Rabinovitch et al., 2005). Semmens et al. demonstrated this effect in their study of $PM_{2.5}$ in homes with a wood burner. When measured levels were restricted only to time periods when children were at home, levels were 4 $\mu g/m^3$ higher on average than the 48-hour average levels (Semmens et al., 2015).

The lack of understanding concerning the physics of indoor microenvironments means that exposure studies may often be confounded, and false conclusions can be drawn. For example, exposure assessment of dust mites, fungi and endotoxin conducted around two decades ago clearly showed that pollutant levels of these allergens were significantly higher in carpets and rugs than on hard floors (Wickens et al., 1997b). This was an expected association, and it was assumed that carpets were unhealthy compared with hard floors due to their moisture-retention capacity. However, health effects studies consistently failed to show increased symptoms associated with carpets or rugs. Through the use of personal exposure measurements, it became clear that, although carpets and rugs are

significant reservoirs of allergens and other pollutants, their physical nature means they are also good at holding on to these pollutants. In contrast, hard floors easily relinquish PM into air, especially as a result of activity or movement. While hard floors harbour far lower concentrations, they are associated with more airborne dust and pollution than carpets and therefore higher exposures.

This relationship of exposure concentration with human activity is becoming more recognised as an important aspect in exposure assessment. The studies of Morawksa et al. (2013) and Toivola et al. (2014) showed that the higher exposures found in personal measurements may be related to human activity, in that people are perhaps more likely to be in close proximity to high-exposure events such as peak-hour traffic exposures, cooking exposures, smoking and lighting a fire, and of course, movement can resuspend deposited pollutants from floors and surfaces, increasing the risk of breathing them in (Morawksa et al., 2013; Toivola et al., 2014).

Another example of our lack of understanding of the indoor environment may be responsible for another confusing but consistent finding in exposure studies. Studies looking at allergens and comparing bedding, floor dust and airborne allergens find floor dust in either lounges or bedrooms is generally more closely related to health effects than exposures measured in either air or bedding (Douwes et al., 2006), which cannot yet be fully explained. One under-recognised resource is the wealth of indoor exposure studies that now exist, including in New Zealand homes. While many of these pieces of research found few relationships with the hypothesised health effects under study, a researcher looking simply for relationships between exposure findings may well generate valuable hypotheses regarding the physics of indoor microenvironments.

Indoor air exposure research is also benefiting from technological advances and especially from discovery of less-expensive exposure assessment techniques, which can make broad-scale assessment more feasible. More work to reduce exposure assessment costs would be highly valued. Recent work from the Wellington Asthma Research Group (Otago University) has conducted validations of electrostatic impaction of mould on readily available dust cloths (Shorter et al., 2015). The researchers compared samples with both researcher inspections and self-report of mould and moisture damage and found significant correlation between the methods. This methodology shows much promise in solving several difficulties in exposure assessment of bioaerosols. Firstly, the cloths can be left for a long sampling period with no extra cost. This is important, as many exposure studies using more traditional methods must rely on 'snapshot' exposure samples due to the difficulties and cost of using and analysing multiple short-term samples. Secondly, the cloths require no expert knowledge to set up or interpret, and thirdly, they are cheap, meaning each home or room can be sampled using multiple cloths to minimise random error.

Researchers from Massey University are developing low-cost, temperature, relative humidity and CO_2 monitoring instruments. NIWA is developing a low-cost PM sampler. Advances in instrumentation may be a key to permitting more environments to be investigated.

Interpretation of results also requires caution The risks of misinterpretation are illustrated in a meta-analysis of NO_2 exposure by Lin, Brunekreef and Gehring (2013). The results of this study showed that asthma was more clearly associated with gas cooking (a behaviour that produces NO_2) than with actual NO_2 measurements, while wheeze showed the opposite effect – a stronger association with NO_2 measurements than with gas cooking. This fact, recognised by the authors, raises questions. Is NO_2 the sole culprit? Are there co-exposures and possibly synergistic effects? Is the effect even due to NO_2 at all or is it simply acting as a surrogate for a related exposure such as PM? (Lin et al., 2013).

In another example of the need for caution when interpreting exposure assessment results, researchers focusing on the validity of using questionnaire data to model SHS exposure found parents were significantly less likely than children to report smoking inside the home or car. This was followed by biochemical testing that indicated that the children's reporting was more accurate (Glover et al., 2013). This has implications for future studies relying on self-reporting by children and/or their caregivers.

Recent work examining effect modification in the presence of more than one risk factor shows promise. For example, the relationship between genetic predisposition for asthma combined with SHS exposure has been shown to increase odds of adult-onset asthma from an odds ratio of around 2 for each of the factors individually to close to 13 for those individuals who had both exposures (Lajunen et al., 2013).

Another study measured the relationship between endotoxin exposure and other indoor pollutants on asthma exacerbations, Although no overall association was found between asthma outcomes and endotoxin, when there was a co-exposure with airborne nicotine, endotoxin was associated with worse asthma symptoms, but the opposite effect was reported for co-exposure with high relative NO_2, in the presence of which endotoxin was protective, and with low NO_2, endotoxin was associated with worse asthma symptoms (Matsui et al., 2013). These results show that complex interactions may confound results when studying the health effects of individual air pollutants.

One airborne toxin may even mediate the effects of another. For example, Matsui et al. (2014) conducted a study on the effects of endotoxins on asthma and found no statistically significant differences in asthma outcomes. However, when analysing NO_2 and endotoxin exposures separately, there was evidence of a protective effect of endotoxin in the presence of airborne nicotine but no protective effect in the presence of NO_2 (Matsui, 2014). Research into immune response looking at the differences between single-exposure immune responses and responses to multiple exposures suggest synergistic effects (where combined exposure is enormously more damaging than would be expected from a cumulative effect). However, this area of research is not yet well developed due to complexities in experimental design (Carlsten & Georas, 2014).

Spatial modelling of outdoor air exposures has become increasingly refined over recent decades and is now a valuable tool in assessing outdoor exposures, which, as discussed earlier, are an important aspect of indoor exposures. However, the above-mentioned lack of physical understanding of indoor microenvironments means there is much work to be done before modelling techniques can be usefully used to predict indoor exposures.

Some useful findings for broad-scale exposure studies using modelling include a relationship found by Telfar-Barnard between house condition and age of inhabitants when matching New Zealand QV house condition data with census data. House condition was best for those aged 15–19 and those aged 65, with those aged under 5 and around 30 years old occupying houses of the worst condition. This suggests New Zealand studies looking at associations between house condition and health effects should adjust for age of the occupants in the data analysis (Telfar-Barnard, 2009). Hodas et al. found that people at the lower end of the socio-economic spectrum lived in houses that were less airtight than people with higher incomes, another factor that could be included in exposure models (Hodas, 2012).

For some interesting reading of the use of spatial modelling in epidemiological research, see Gilliland et al. (2005), Little et al. (2012), Kloog et al. (2012), Fournier, Glorennec & Bonvallot (2014), Taylor et al. (2014), Ostro et al. (2015) and Lelieveld et al. (2015). For an exposure model for individual exposure, see Wu et al. (2005).

Part three – Building characteristics

Indoor air physics

Buildings are an extremely complex thermodynamic system (Trethowen, 1976). While many models are available for calculating exposures given physical characteristics of the environment and the pollutant(s), these examples of engineering are dependent upon the physical models from which they draw their predictive value. Physical models of atmospheric characteristics are useful at large scales, such as the outdoor environment, but the complexities of indoor physical characteristics, which can affect air, moisture and temperature transport through what are essentially a fairly predictable yet complex agglomeration of microenvironments that reflect the typical home, are not yet defined to a sufficient degree to allow modelling inside the home to be useful.

In 2009, the World Health Organization published their *Guidelines for indoor air quality: Dampness and mould* (WHO, 2009). Among these guidelines was the following: "Management of moisture requires proper control of temperatures and ventilation to avoid excess humidity, condensation on surfaces and excess moisture in materials. Ventilation should be distributed effectively throughout spaces, and stagnant air zones should be avoided." Stagnant zones are important microclimates

within the built environment that are likely to impact overall indoor air quality in terms of pollutant concentration and dampness but have received little attention (Licina et al., 2014).

Compared to outside air, inside air has a much higher surface to volume ratio (estimated average 4.6 square metres per cubic metre of air) (Licina et al., 2014). These surfaces can increase the concentration of pollution in several ways. They can create physical barriers to ventilation, causing pollutants to pool indoors, they may release pollutants themselves such as formaldehyde or phthalates, which are chemical components of their structure, and they also may provide a 'sink effect', allowing pollutants such as SVOCs and PAHs to adhere to them to be released into the air at another time (Karimi et al., 2015).

As well as these building characteristics, human behaviour can also increase pollutant concentrations. Cooking, showering, cleaning and breathing all release moisture, and unless adequately ventilated, this will lead to conditions conducive to the proliferation of moulds and bacteria. Cooking also releases significant levels of particulates, including UFPs (Ostro et al., 2015) into the air, as do gas or wood fires and stoves, while cleaning frequently releases a wide variety of VOCs (Bari et al., 2015). For these reasons, indoor air is generally more concentrated in pollutants, and introducing outdoor air is usually the best way of reducing indoor contaminants.

This relationship may not hold true in built-up urban areas, however, where outdoor air pollution levels are high, and filtering outdoor air may be preferable. As more people move into the inner city, it may be useful to consider the physics of air movement during building design in order to minimise the infiltration of pollutants into living spaces. Little work has been done on transport of pollution through urban geographies, although design features that minimise infiltration of particles and other pollution could have a powerful impact on indoor air quality in urban environments (Kumar et al., 2013).

A common assumption is that, if indoor to outdoor ratios of a given pollutant exceed unity, there must be indoor sources of that pollutant (Stranger, Potgieter-Vermaak & Van Grieken, 2008; Diapouli et al., 2007). While this is reasonable, most of the time, it is also plausible that, given certain environmental and pollutant parameters, the increased surface areas indoors could promote a pooling effect that could lead to higher levels indoors than outdoors from a continuous outdoor source.

The relationship between human activity and building physics is also critical to predicting indoor air quality. Early studies appeared to suggest that carpeted floors led to increased risk of damp-related exposures (Wickens et al., 1997b; 2003). Later studies found the opposite effect (Dellongueville et al., 2015). What has become apparent since these earlier studies is that carpets hold dust more efficiently than hard floors, meaning that a larger amount will be found when using the standard exposure assessment method of vacuuming the same floor area in each house (for example, 1 m^2),

but this fact is not related to airborne levels. The fact that hard floors are inefficient at holding on to dust means that dust from hard floors is resuspended and recirculated far more efficiently compared with carpeted floors, so in fact, human exposure will generally be greater with hard floors than carpet (Spilak et al., 2013).

The interactions between physics and human activity are also demonstrated in a study by Spilak et al., who found higher UFPs in houses two or three streets further from busy roads than those houses closest (Spilak et al., 2013). The authors hypothesised that people living closest to roads opened their windows less frequently (possibly due to traffic noise), which was shown to be true in a different study by Van Renterghem and Bottledooren in a study in Ghent, Belgium (Van Renterghem & Bottledooren, 2012).

Other interesting work with a focus on the physics of human interactions with their domestic surroundings include vertical pathogen gradients. Khare and Marr (2015) assessed the vertical gradient of virus particles created as a result of resuspension from floors due to walking. They showed that concentrations at 1 metre above floor height were 40% higher than at 2 metres. This is one of many factors suggesting risk of air pollution-related health effects is greater for children than adults (see Vulnerable populations and Children) (Khare & Marr, 2015). Licina et al. demonstrated that a human convective boundary layer can strongly influence exposure to pollutants originating at floor level, funnelling them upwards towards our breathing zone (Licina et al., 2014).

Several studies have shown that dust measurements are more closely associated with health effects than sampling of air (Douwes et al., 2006; van Strien et al., 2004; Thorne et al., 2009; Chen et al., 2012; van Boven, 2014; Wright & Phipatanakul, 2014; Kanchongkittiphon et al., 2015). This could be an artifact of the fact that air measurements are often brief snapshots while floor dust has collected over a longer period, meaning floor dust could be more representative of past exposures. Clarification of this point would be helpful in understanding the mechanisms of disease pathogenesis. Another interesting finding has been that bedding and bedrooms seem less important to human exposure than has been generally assumed (Smith et al., 2011; Kanchongkittiphon et al., 2015).

A recent update of the Institute of Medicine review on asthma discussed this issue in relation to cockroach allergen. The author pointed out that those studies that took bedroom floor dust samples found consistently positive association with asthma severity in the presence of cockroach allergen, while results from studies taking samples directly from bedding or air showed inconsistent results (Kanchongkittiphon et al., 2015).

Another study that showed exposure in bedrooms was less important than exposures elsewhere in the house looked at the relationship between vinyl floors (a proxy for phthalate exposure) and asthma incidence (Shu et al., 2014). Regression analysis showed that PVC flooring in bedrooms

was associated with asthma incidence within the large cohort of Swedish children. Interestingly, however, the association was stronger when the PVC flooring was in the parents' room than when in the child's own room. The authors recorded parental floor covering as an indication of prenatal exposures, and it is certainly true that phthalate exposure during pregnancy could be causally related to the measured effects of increased asthma incidence. Another interpretation could be that exposures during sleep do not cause the same immune response as exposures while awake. Chen et al. showed that having a bed in the living room increased levels of mattress endotoxin by 40% compared with mattresses in bedrooms (Chen et al., 2012). Perhaps less activity in bedrooms is a factor, or are bedrooms perhaps more frequently ventilated? The study of indoor physics of microenvironments needs to be coupled with human interaction with those microenvironments.

Heating is an important driver of the indoor climate in homes and other buildings. Different heater types have been shown to distribute their warmth differently through buildings, and this is likely to impact air quality. Gas heating is related to moisture transport through floors and ceilings more strongly than other heating types (Taptiklis et al., 2011). The Energy Efficiency and Conservation Authority have reported that the most frequently asked question to their help desk is for advice on heater selection. Independent consumer advice could be very useful. Some parameters assigned to assess quality of heat, for instance, distribution (how does the heater distribute the warmed air), humidity and mixability (how well does the heated air mix with ambient air), could assist occupants in choosing the best heating system for their built environment.

Dust

Dust is an important aspect of indoor air quality, not only when it becomes airborne itself, but also because it frequently harbours SVOCs, which release into air when they partition to the gas phase under conditions of increased temperature or low vapour pressure (Weschler & Nazaroff, 2008). Many SVOCs were previously understood to have a primary exposure route via diet. However, recent research has highlighted the fact that inhalation of indoor air, dermal exposure and ingestion of dust are significant routes of exposure for many SVOCs, including pesticides, phthalates (Blanchard et al., 2014) and polybrominated diphenyl ethers (PBDEs) (Coakley et al., 2013; Lim et al., 2014) and even polychlorinated biphenyls (PCBs) (Harrad et al., 2009).

Therefore, infants and toddlers are at risk of higher exposures to these substances than adults and older children because of frequent hand to mouth behaviours and because their breathing zone is closer to the floor. Król et al. measured PBDEs in ingested dust in toddlers and adults and found that toddlers consumed at least five times the PBDEs of adults (Król et al., 2014a). Studies have shown that levels of SVOCs in dust are higher in older houses and buildings (Whitehead et al., 2014a).

The daily intake of dust has been estimated to be 100–200 mg/d for small children (aged 1–4 years) while the intake for adults is estimated to be around 50 mg/d (Sippola, Sextro & Thatcher, 2014).

Sippola et al. investigated the transport of dust into the house from outdoors via foot transport and demonstrated that toxins of outdoor origin can be spread throughout the house in just a few hours (Sippola et al., 2014).

Indoor air chemistry

Indoor air is full of organic compounds, including VOCs and PAHs, which react readily with oxidising agents also in air. Such chemistry is driven primarily by three oxidising agents in indoor air – ozone (O_3), hydroxyl radicals (OH) and nitrate radicals (NO_3) (Nazaroff & Goldstein, 2015; Gligorovski, Wortham & Kleffmann, 2014; Waring & Wells, 2015). Ozone is formed outdoors from UV rays reacting with aerosols. Although ozone levels are generally higher outdoors, exposures are usually higher from indoor sources, due to the fact that so much more time is spent indoors (Weschler, 2006). Recently, the popularity of ozonolysis air cleaning units has become a public health concern in the US, and in a house where such a machine is used, ozone indoors will be significantly higher than outdoors (Fadeyi, 2015). Although ozonolysis is effective at sterilising the air of biocontaminants, it may have an equally harmful effect on humans as it does on airborne bacteria (Nazaroff & Goldstein, 2015).

This is of particular concern given other recent findings on the relationship between ozone in air and human skin lipids (Weschler, 2015). It has been demonstrated that human skin reacts strongly with ozone in the atmosphere to the extent that occupation of a previously unoccupied room can reduce ozone in air by 60% or more (Weschler, 2015). Research is needed to determine the impact of dermal ozone exposure and health effects, including skin infections (Nazaroff & Goldstein, 2015), and it may be that data already exists to examine this relationship, for example, Gillespie et al. measured 'rash' in a large New Zealand birth cohort, and data could possibly be matched with modelled ozone exposure assessments for the same period (Gillespie et al., 2006). Ozone fogging is frequently used to kill off mould, and the health effects and clearance testing need further research.

Nitrate radicals are formed by a reaction between ozone and nitrous oxide (Nazaroff & Goldstein, 2015). Nitrate radicals react quickly with terpenes, including common cleaning product constituents alpha pinene and d-limonene, to produce secondary organic aerosols such as alkyl nitrates. While ozone chemistry indoors has received some attention in recent years, little research has been done to characterise the impact of hydroxyl and nitrate radicals on indoor air quality (Nazaroff & Goldstein, 2015).

Human occupants have physical and chemical effects on their surroundings. Although it is well established that human bioeffluents impact strongly on air quality, very little work has been done to chemically measure and characterise the effects of human occupancy on indoor air (Nazaroff & Goldstein, 2015) (see Biological contaminants).

Various surfaces, especially fabrics, have been shown to act as chemical sinks or reservoirs where

airborne chemicals may adhere to or be adsorbed by surface chemistry to be released in the same form or chemically altered under different conditions of temperature and pressure (Karimi et al., 2015). This effect is found particularly frequently in SVOCs, which include PAHs, phthalates, flame retardants and pesticides. These chemicals pass back and forth between solid and gas partitions so are likely to deposit in solid form and be released upon returning to the gas phase. This effect has been measured in relation to flame retardants, phthalates and tobacco smoke, and findings show that surfaces including fabrics can continue releasing adsorbed chemicals for years after the original exposure. For example, under laboratory conditions, fabrics exposed to tobacco smoke continued to give off tobacco smoke-derived chemicals for 18 months after the last exposure to tobacco smoke (Bahl et al., 2014), while another study in homes found no drop-off in the level of PBDEs released by furnishings containing them (Besis & Samara, 2012).

Neighbourhood characteristics

A recent global study of mortality and ambient air pollution estimated 3.3 million premature deaths caused by air pollution in 2010 worldwide, of which 2 million were urban dwellers (Lelieveld et al., 2015). Population density in cities is constantly increasing, and pollution density is likely to increase at the same time, meaning more people at higher risk.

New Zealand has not felt the impact of outdoor air pollution to the same extent as many other nations due to the relatively sparse population. However, even in New Zealand cities, outdoor air pollution is causing unnecessary mortality and morbidity (Fisher et al., 2007; Hales et al., 1999; Kuschel et al., 2012), and this will almost certainly increase in future years as pollution sources continue to increase and as people live in higher-density housing.

A USA study assessing indoor pollutants and house characteristic differences in the homes of asthmatic children compared urban versus suburban location and found a wide disparity in risk factors and health outcomes. Inner-city homes had lower prevalence of dust mite, dog and cat allergens but higher levels of PM, NO_2 and ozone, as well as higher levels of mouse and cockroach allergens and higher frequency of disrepair (Simons et al., 2007). Coleman et al. focused on differences in indoor exposures between urban and rural populations, also finding lower levels of cockroach and mouse allergens in rural populations, despite finding that those allergens were in fact widespread in rural populations, just at lower levels (Coleman et al., 2014).

As yet, there has been little research on how urban geographies can impact on infiltration rates (Taylor et al., 2014), but results available suggest that urban characteristics can have significant effects on indoor/outdoor pollutant ratios. For example, when measuring UFPs in residential areas of Copenhagen, Spilak et al. found that homes further from trafficked roads had higher mean UFPs (Spilak et al., 2013). The authors hypothesised that individuals living closer to highly trafficked roads were less likely to leave their windows open because of traffic noise, an effect demonstrated by Van

Renterghem and Bottledooren in a study in Ghent, Belgium (Van Renterghem & Bottledooren, 2012). Another study undertaken in London used modelling to demonstrate that ratios of indoor/outdoor $PM_{2.5}$ were higher in outer London than in the innermost suburbs. The authors identified the higher prevalence of flats compared to semi-detached or detached dwellings in the outer suburbs, leading to lower infiltration rates, was likely responsible for this apparent inversion of effect (Taylor et al., 2014).

Traffic

Traffic exhaust fumes release carbon monoxide, various VOCs including benzene, nitrogen dioxide, sulphur dioxide and particulate matter. Some proportion of these pollutants will react with UV light to produce ozone, a secondary pollutant and the primary constituent of photochemical smog (Zhang & Samet, 2015). Traffic pollution is understood to be the primary cause of ozone pollution (Behrendt et al., 2014) and photochemical smog (Zhang & Samet, 2015). Traffic pollution has been associated with cardiovascular disease, cardio-cerebral illnesses including stroke, ischaemic heart disease and, more recently, asthma (Uzoigwe et al., 2013; Farmer et al., 2014; Fuentes-Leonarte, Ballester & Tenías, 2009; Behrendt et al., 2014). In 2012, diesel and outdoor air pollution were recognised by the International Agency for Research on Cancer (IARC) as human carcinogens (Carlsten & Georas, 2014).

Traffic pollution in New Zealand has received some attention over the past two decades. A modelling study commissioned by the Ministry for the Environment, the Ministry of Transport and the Health Research Council, published in 2007, estimated around 900 premature adult deaths annually attributable to traffic emissions pollution and its health effects (Fisher et al., 2007). This study was updated in 2012, this time also including funding from the New Zealand Transport Agency, which found around half the level of premature death related to traffic pollution. While there have been real reductions in traffic pollution over this period due to regulations on petrol formulations and improved fuel efficiency, much of this difference in findings is due to differing study designs between the two studies. These differences include the fact the second study only looked at PM_{10} and not NO_2, benzene and carbon monoxide, which had been included in mortality estimates in the earlier study, that it used data from regional air-quality monitoring instead of land-use regression modelling as the Fisher study had and that it estimated a higher proportion of PM_{10} as attributable to natural sources (Kuschel et al., 2012).

In Auckland in 2012, benzene was recorded at levels 42% higher than guideline levels of 3.6 g/m^3 (Ministry for the Environment and Statistics New Zealand, 2014). This measurement from urban monitoring close to busy roads shows that, despite reduced levels of benzene in petrol formulations (regulations now permit no more than 1%) and the increased fuel efficiency that has seen overall reductions in traffic emissions over the past decade (see Outdoor air pollution), benzene remains a pollutant of concern in urban areas. Benzene is increasingly being recognised as an important environmental pollutant of concern and is associated with cancers, including childhood cancers, and nervous system health effects (WHO, 2010; Tchepel et al., 2014).

Living close to heavily trafficked roads has been found in studies to be associated with numerous health effects, including asthma (Clark et al., 2010), childhood cancers (Ghosh et al., 2013), autism development (Levy, 2015) and pre-term birth (Llop et al., 2010). Esplueges et al. found a dose-response relationship between BTEX measured indoors and frequency of traffic passing outside the house (Esplugues et al., 2010a), confirming that indoor air close to major roads can be significantly impacted by traffic emissions. Results for PM levels close to roads show a more complex relationship.

Particulate matter associated with fresh combustion sources, including unseasoned wood and traffic exhaust, has been found to be more toxic than other sources of PM. Furthermore, Hodas et al. found that fresh combustion particles may have higher penetration rates indoors, compared with other aerosols due to lower deposition rates (Hodas et al., 2012).

Impact of greenery

Outdoor trees and shrubs impact on indoor air quality in a number of ways. Trees can reduce wind, reducing air infiltration rates, which in turn reduces energy costs for heating (Millward & Sabir, 2010). Transpiration, the process of water evaporating from leaves after moving up the plant from its roots, lowers the temperature in the canopy, so trees can also reduce energy costs for cooling. This is particularly relevant in densely populated urban areas prone to urban heat island effects (Millward & Sabir, 2010).

Urban greenery also has the capability of reducing pollution concentrations through both chemical and physical processes. Particles adhere to leaf surfaces, and even small trees and shrubs have been shown to be remarkably efficient at collecting particles at a wide range of sizes from PM_{10} to submicron (Maher et al., 2013). One study estimated an average of 10 grams per square metre of leaf cover per year, leading to a potential total US reduction of air pollution of 711,000 tonnes annually (Nowak, Crane & Stevens, 2006). However, this factor varies widely according to species and climate conditions.

Another innovative study that temporarily placed immature birch trees between the road and houses in the United Kingdom showed a 50% reduction in PM_{10} in the ground floor roadside interiors. Micrography of leaves from this same study also showed high levels of impacted submicron-sized particles (Maher et al., 2013). This suggests that the way trees are distributed relative to domiciles and pollution sources may significantly increase or reduce their efficiency in pollution reduction in relation to indoor air.

Trees have also been implicated as important vectors in the outdoor chemistry of nitrogen oxides and ozone. Leaves take up NO_2, reducing ambient concentrations, but in certain circumstances may increase NO_2 within the canopy. Tree canopies have been shown to have increased levels of NO_2 and decreased levels of O_3 relative to nearby ambient levels. Harris and Manning postulate that

high ambient ozone promotes oxidation of NO released from soils to NO_2 in the canopy at a rate faster than leaves can absorb it (Harris & Manning, 2010). Complicating this relationship further, the release of VOCs by plants, such as alkyl aldehydes, alpha pinenes and isoprene, can increase the concentration of ozone through photolysis (reactions with sunlight).

Like dust mites, indoor plants gained attention during the earlier phases of asthma epidemiology as potential sources of allergens. Evidence has been conflicting as to whether or not indoor plants constitute significant contribution to pathogenic micro-organisms in indoor air, but a consensus has emerged that any contribution is insignificant except in relation to immunocompromised persons (Prussin & Marr, 2015; Torpy et al., 2012). Indoor plants have been demonstrated to impact on indoor RH, although this contribution appears relatively minor, and it is likely that any contribution to indoor fungal concentrations from houseplants is directly related to the volume of water and frequency of watering (Torpy et al., 2012).

Meanwhile, a number of studies have looked into possible health benefits of indoor plants. Plants are known to both emit and absorb VOCs in the atmosphere. Some studies have shown that certain common indoor plants are remarkably efficient at reducing concentrations of toxic VOCs in indoor air including benzene, toluene ethyl benzene, xylene and formaldehyde (Dela Cruz et al., 2014; Sriprapat et al., 2014; Mahnert et al., 2015).

Plants have also been shown to present psychosocial benefits to humans both indoors (Wolverton, 1986) and at the neighbourhood level (Zhang et al., 2015). Recognition of these various beneficial factors has led to a proliferation of roof gardens in many urban centres, and evaluations of typical roof gardens have demonstrated a significant reduction in urban air pollution can be achieved by harnessing this natural ability of plants and combining it with significant areas of flat roof space in cities (Yang, Yu & Gong, 2008).

Schools

New Zealand children are legally required to spend at least 6 hours a day at school, mostly indoors. Duration in the school environment can be longer for children enrolled in before-school or after-school care programmes. This makes the school, in terms of environmental exposures, the second most important environment after children's homes. The energy budgets for New Zealand schools have been capped at 2010 levels, which can lead to behaviours by the caretakers/teachers that will underventilate classrooms during cooler weather conditions.

The recognition that toxic pollutants can have a more serious and long-lasting impact on children and infants and impact their ability to develop into healthy adults (Fuentes-Leonarte, Ballester & Tenías, 2009) has led to the recent creation of a significant body of work on health-related exposures in schools (Jacobs et al., 2014; Morgan, Wilson & Chuang, 2014; Sofuoglu et al., 2011;

Stranger et al., 2008; Chen et al., 2014b; Diapouli et al., 2007; Dadvand et al., 2013). Many of these studies have focused on the relationship between school exposures and asthma. One cross-sectional study found a relationship between the measured level of fungal spores in classrooms and current asthma diagnosis and also found symptoms reduced during weekends and holidays (Chen et al., 2014b). Another study showed that children at schools with high levels of mouse allergen had increased asthma-related days off school (Kanchongkittiphon., 2014).

Ferreira and Cardoso (2014) looked at the air quality in 51 schools in Portugal, finding that concerning concentration of CO_2 indicated poor ventilation and a problematic learning environment. CO_2 is produced as a result of breathing and was therefore measured as an indicator of ventilation level rather than as a toxic pollutant. High levels of CO_2, however, reduce oxygen availability in air and can impact significantly on cognitive function and therefore learning (Ferreira & Cardoso, 2014).

Teachers in New Zealand schools typically don't open windows during winter. Most schools are naturally ventilated and dependent on opening of windows, consequently CO_2 levels frequently exceed the 800 ppm guideline value (Bassett & Gibson, 1999; McIntosh, 2011; Boulic et al. 2014; Wang, 2016) and often by a very large margin.

A study from a large city in Belgium found that $PM_{2.5}$ and BTEX concentrations were higher in classrooms than in ambient outdoor air (Stranger et al., 2008), and another study in urban schools in Turkey calculated that a third of their sample were exposed to benzene at carcinogenic risk threshold levels and 50% to carcinogenic risk threshold levels of formaldehyde (Sofouglu et al., 2011). Rivas et al. also measured indicators of traffic pollution in schools and found classrooms levels approximately 1.2 times the average background urban levels (Rivas et al., 2014). Jacobs et al. also measured higher levels of endotoxin in schools than in homes of the same cohort of children (Jacobs et al., 2013).

The Schools Indoor Pollution and Health Observatory Network in Europe (SINPHONIE) study (Csobod et al., 2014) found poor ventilation was a widespread issue in schools. This led to a number of indoor air quality problems, and children were exposed to high levels of indoor pollutants. Health impacts included respiratory disturbances and reduced nasal patency (Csobod et al., 2014). The study provided evidence that exposure to endotoxins in schools exceeded the levels found in homes. It is plausible that the same is also true for other microbial agents. This study also found that 25% of the school children were exposed to benzene whilst at school at more than 5 μgm^3, which is the limit set for management of the excess lifetime risk for leukaemia, and more than 60% of the school children were exposed to formaldehyde whilst at school at more than 10 μgm^3, which is the long-term indoor guideline value set by the Agence Francaise de Sécurité Sanitaire de l'Environnement et du Travail (AFSSET).

The SINPHONIE study has produced a long list of knowledge gaps and suggested research. These include indoor air quality audits in schools and medical surveillance and health screening of school children and school staff. These would be fruitful avenues for research in New Zealand also.

Schools are crowded, and people are highly active in them, compared with offices, which may impact on exposures due to increased resuspension (Toivola et al., 2004; Stranger et al., 2008). Schools with more trees in and around their boundaries have been found to have lower traffic-related pollution than other schools (Dadvand et al., 2015). Schools in New Zealand are frequently located on arterial routes.

New Zealand schools have not been extensively examined in terms of pollutants and health effects, other than the studies by Basset and Gibson, 1999; Cutler-Welsh, 2006; McIntosh, 2011; Boulic et al., 2016; and Wang et al., 2016. All New Zealand studies to date found high to very high levels of carbon dioxide, low indoor temperatures and high RH levels during winter conditions. Boulic's (2012) study found PM_{10} levels in classrooms retrofitted with a solar heater/ventilation unit were half the level of a matched control classroom without the ventilation unit operated. The use of a solar air heater ventilation unit significantly improved CO_2 and bacteria levels.

Preschools

The air quality in New Zealand preschools has be drastically under-researched in New Zealand, apart from two postgraduate student research projects that are currently under way – one based at Otago University and one based at Massey University. There are significant complexities and sensitivities conducting research around preschool-aged children, which can hamper the research logistics. These are not insurmountable.

However, balancing the complexities is the urgent need for research in this area. Preschoolers are most at risk from exposure to pollutants. Some children, especially those from lower SES families, are spending up to 12 hours per day in the preschool environment so they have long periods of exposure. Preschools are frequently located along busy arterial routes, which will expose the children to high levels of traffic pollution and noise. The density of children in many preschools is very high, and operation budgets for energy, cleaning and maintenance can be tight, meaning the indoor environments can be substandard.

Important areas for attention include dampness-related exposures in schools and preschools from nitrogen dioxide, bacteria and viruses; ventilation, VOCs and traffic pollution exposure in urban schools and preschools; and phthalate exposure, particularly in preschools.

Noise levels in some New Zealand preschools have been measured at industrial hazard levels. More research is required to determine how widespread this is and monitor the benefits from adding acoustic absorption materials.

Aged care facilities

The housing conditions of elderly, especially lower SES elderly, needs to be investigated in New Zealand. There are several potential factors that could make housing for elderly worse than for other populations. Firstly, most elderly are on fixed incomes and can struggle to afford home heating, home maintenance or home improvements. Many elderly are frightened to open windows even when they are at home for fears of personal safety. This can lead to chronic underventilation. The ageing demographic and housing unaffordability will mean more elderly people will require social housing. The logistics and conditions needed for comfortable ageing in place require further research.

Elderly people are more vulnerable to health effects from air pollution than the general population, but little attention has yet been paid to exposure profiles of the elderly. One recent study measured indoor air quality in 50 European homes for the elderly, finding that, although levels were generally below exposure guidelines, the respiratory health of inhabitants was nevertheless impacted. COPD was significantly associated with formaldehyde levels above the median, and wheeze was associated with $PM_{0.1}$ (Bentayeb et al., 2015). The same authors also found relationship between increased (but still low) levels of PM_{10} and bronchitis symptoms (Bentayeb et al., 2010).

A consideration for the design of houses, and especially those for the elderly, is the BTEX group of VOCs as well as carbon monoxide. These are consistently found at higher levels in homes with an attached garage (Delgado-Saborit et al., 2011). Amongst health effects of these chemicals is an increased risk of accidental poisoning (Peiris-John et al., 2014) and dementia (Chang et al., 2014), and more recently, a link has been found between benzene and myocardial infarction (Bard et al., 2014).

Homes in general and especially for the elderly should perhaps not be built such that garage exposures can enter the dwelling. Research examining this relationship could also prove interesting.

Indoor air quality in workplaces

Indoor air quality in workplaces is covered in this report as a brief aside. Workplace exposures to airborne pollutants in New Zealand are a frequent and significant cause of adverse health effects. Many of the pollutants described in this review are found at particularly high levels in industrial and commercial situations, such as benzene in road building and construction and formaldehyde in plywood, joinery and furniture restoration as well as in funeral services. VOCs and NO_2 are common in many industrial situations, and VOCs are particularly common in hospitals and painting. SO_2 exposure is related to milk powder production and metal smelting. PM_{10}, $PM_{2.5}$ and UFPs are common exposures in mining, saw milling and food preparation including meat working, food processing, baking and cooking (Ostro, 2015; Spilak et al., 2014). Arsenic exposure is related to building, metal working, mining and smelting. Carbon monoxide is common in all combustion processes, and high

levels have often been associated with generating electricity using diesel. Endotoxin levels are particularly high in farming, composting and mushroom growing (Douwes et al., 2003; Chen et al., 2011). Of course, industries use many other chemical pollutants not covered in this report.

Several common workplace pollutants have the potential to cause neurodegenerative illnesses including dementia. These chemicals include formaldehyde, tetrachloroethylene, carbon monoxide and pesticides. Recently, pesticide use in New Zealand has been hypothesised as a possible cause of depression in New Zealand farmers (Douwes, 2014).

Office workers' exposures are likely to be similar to the household exposures described in this review, except for the fact that air conditioning can potentially introduce pollution from outdoors, especially if the intake is poorly situated, and microbial pollution can also build up within air conditioning systems leading to BRI (Turpin, 2014). Teachers' exposures are examined above (see Schools and Preschools).

New Zealand houses

There are currently around 1.5 million occupied dwellings in New Zealand and around 200,000 unoccupied dwellings (Statistics New Zealand, 2015). New Zealand's building stock is highly diverse in terms of construction styles (Amitrano et al., 2014), although 80% of the building stock are stand-alone houses and the majority are single storey (Bengtsson, Hargreaves & Page, 2007). Beacon Pathway has developed a set of 10 different New Zealand house typologies by which the housing stock can be described on a consistent set of parameters designed to assess them for suitability for retrofitting (Ryan, Burgess & Easton, 2008). These house typologies could be developed further to increase the value of this work.

Leaky buildings

Recent calculations estimate there are 100,000 leaky homes in New Zealand (Norman & Page, 2014) constituting approximately 6% of the current housing stock (Statistics New Zealand, 2015). There are also numerous leaky schools. The health impact of leaky buildings has not been quantitatively assessed and is not likely feasible due to ethical concerns. However, there is general consensus that numerous New Zealanders have had their health negatively impacted by this massive failure in design and build. The cause of leaky buildings has been addressed in other reports and is outside the scope of this review. However, there is no doubt that, in terms of improving the overall quality of New Zealand's building stock, leaky buildings have been a major step backwards.

The building industry in New Zealand drives over 40% of all the capital produced in this country (Page & Norman, 2014). It would therefore seem that the provision of useful information regarding build quality to decision makers (designers, private owners and government regulators) is critical for New Zealand's economic success.

Housing condition

An unequal distribution of social determinants, including housing condition as well as income, education and employment, is fundamental to driving health inequalities in New Zealand (Blakely & Simmers, 2011) and elsewhere (Urbanos-Garrido, 2012). New Zealand has more extreme health disparities than other nations with similar levels of development (Howden-Chapman & Chapman, 2012), and these disparities have been increasing over recent years (Baker et al., 2012). Although the New Zealand Government has committed to working to reduce health inequalities, "political barriers remain, including vested interests at all levels of the sector and constant restructuring across the sector" (Ministry of Health, 2004).

A recent study from Spain aimed to quantify the contribution of housing condition to health inequalities and estimated 7–14% of the burden of health inequality could be attributed to housing condition (Urbanos-Garrido, 2012). It is likely that the proportion in New Zealand would be even higher than in Spain due to New Zealand's climate and construction styles (Howden-Chapman & Chapman, 2012; Thomson et al., 2013).

In New Zealand, housing condition over the entire building stock is not good (Buckett, Jones & Marston, 2012). Telfar-Barnard examined house condition in New Zealand by using QV data, which found mean condition at 0.05 (average) on a scale from -1 to 1 (Telfar-Barnard, 2009). This finding is similar to findings from BRANZ's regular house condition survey, the most recent of which, conducted in 2010, found mean overall condition of a sample of 550 New Zealand houses moderate (Buckett et al., 2012).

New Zealand has unusually high rates of many infectious illnesses linked in epidemiological studies with a poor housing condition, including tuberculosis (TB), meningococcal disease, childhood pneumonia, rheumatic fever and skin infections (Baker et al., 2012; Jaine et al., 2011). Baker et al. noted that hospitalisations for these illnesses are disproportionately represented in low socio-economic, Māori and Pacific populations. They are also all associated with overcrowded living conditions (Baker et al., 2000; Jaine et al., 2011). The mechanism by which these illnesses are related to housing condition is not clear. However, the high prevalence of these illnesses, especially meningococcal illness and rheumatic fever in children and infants, has led to increased attention on housing condition in New Zealand and its relationship with health of late. Although the specific links between housing quality, illness and indoor air quality remain opaque, air quality is likely to be a factor within these associations.

Housing condition is a very broad and perhaps unhelpful term. In epidemiological studies, it is often used to refer to the condition of the structure as well as its suitability for the occupants (such as overcrowding or availability of facilities) and also to the congeniality and lack of pollution in the neighbourhood. Perhaps separating out these three factors would be preferable to housing condition as a measure and would help elucidate more detail in the relationship. In terms of the structural

factors of housing condition, one factor is maintenance. Almost 87% of households with incomes below $20,000 require immediate repairs at an average cost of $5,800. This obviously poses a problem for repairing these houses due to affordability constraints (Page & Curtis, 2013).

Dampness

The relationship between damp housing and poor health has been explored in many studies and reviews internationally, and the link with poorer respiratory health is well established (Fisk et al., 2007; Bornehag et al., 2005; Institute of Medicine, 2004; WHO, 2009). Other health effects have also been associated with damp housing. The large analysis and review of European housing and health status (LARES), which assessed the health of 8,519 inhabitants of 3,373 dwellings in eight European cities, found that those living in damp housing had 70% increased risk of reporting poor health and found statistically significant associations between damp housing and a wide range of illness, including asthma, bronchitis, arthritis, anxiety or depression, migraine, cold and diarrhoea (WHO, 2007).

Recently, a large population-based birth cohort from Germany, LISAplus, demonstrated increased odds of sleep problems in children living in damp homes (Tiesler et al., 2015). This is important because sleep is essential for maintaining a healthy immune system, and continuously interrupted sleep increases the risk for inflammatory illnesses and cancer (Geiger, Fagundes & Siegel, 2015).

Mould growth is dependent on moisture, and moulds proliferate at RH of 70% or higher (Fisk et al., 2007). In New Zealand, outdoor air frequently reaches this level of humidity (MetService, n.d.), consequently homes and buildings need to be designed and managed to keep out dampness from outside. Our activities also produce moisture, and we contribute to increasing relative humidity indoors through breathing, cooking and washing. In winter, New Zealand bedrooms often have higher relative humidity than living rooms, and bedrooms may frequently reach relative humidity levels over 90% at night, providing ideal conditions for mould growth (Boulic, Hosie & Phipps, 2010).

Indoor dampness can also increase the rate of release of other pollutants from building materials, including formaldehyde and phthalates (Mendell., 2007; Bornehag et al., 2005).

Damp air takes more energy to heat, thus increasing the impact of fuel poverty (WHO, 2009) and contributing to colder indoor conditions.

BRANZ conducts regular surveys of newly built houses, and these surveys continue to find problems with the quality of newly built homes in New Zealand (Page, 2015). The most recent of these surveys found the most frequently measured defect by their inspectors as well as the most frequently cited building issue for builders was flashings around penetrations in the cladding materials (such as doors and windows) (Page, 2015). Inspectors found flashings were frequently poorly constructed, and builders frequently cited insufficient flashings detail in building plans. As a

primary waterproofing element, this high frequency of flashing defects and problems suggests that dampness issues continue to be built into our homes.

Further, the Weathertightness, Air Quality and Ventilation Engineering (WAVE) study conducted by BRANZ found houses constructed in the most recent decade were more airtight than homes built during other periods. Airtightness has reached the point where ventilation via infiltration may be insufficient, and mechanical ventilation may be required.

In 2009, the World Health Organization published *Guidelines for indoor air quality: Dampness and mould*. It is worth reiterating three of these guidelines here:

> Well-designed, well-constructed, well-maintained building envelopes are critical to the prevention and control of excess moisture and microbial growth, as they prevent thermal bridges and the entry of liquid or vapour-phase water. (WHO, 2009, p. xv)

In terms of these three critical factors for avoiding dampness and mould in housing, evidence suggests we are performing poorly in all three areas (see Leaky buildings, Housing condition and Building maintenance).

> Building owners are responsible for providing a healthy workplace or living environment free of excess moisture and mould, by ensuring proper building construction and maintenance. The occupants are responsible for managing the use of water, heating, ventilation and appliances in a manner that does not lead to dampness and mould growth. Local recommendations for different climatic regions should be updated to control dampness-mediated microbial growth in buildings and to ensure desirable indoor air quality. (WHO, 2009, p. xv)

Without regulations on private landlords on issues such as home condition or maintenance, there is little surprise that many people in lower socio-economic strata live in unhealthy conditions in New Zealand with very little legal power over their circumstances.

> Dampness and mould may be particularly prevalent in poorly maintained housing for low-income people. Remediation of the conditions that lead to adverse exposure should be given priority to prevent an additional contribution to poor health in populations who are already living with an increased burden of disease. (WHO, 2009. p. xv)

The government's responsibility to ensure good-quality housing for New Zealanders has come under intense debate of recent years, but the position of the WHO is clear.

Heating

Two intrinsic building characteristics define the indoor temperature – insolation (sunlight reaching directly into the interior) and thermal resistance of the structure as a whole. When these two factors

are low, indoor temperatures will be similar to ambient outdoor temperatures, but if either thermal resistance or insolation are high, indoor temperatures will tend to be significantly higher than outdoor ambient temperatures, even without additional heating (Trethowen, 1976).

Insolation or insulation were not important considerations in the design of many buildings and homes in New Zealand, particularly prior to the 1970s (Ryan et al., 2008). More recently, passive house design has identified the value of using thermal sinks such as dark-coloured floors and walls in interiors to further increase the benefits of intrinsic heating components in energy-efficient buildings (Jaques & McNeil, 2013).

New Zealand has a temperate maritime climate, marked by relatively small differences between summer and winter mean temperature (most of New Zealand has annual mean temperature differences of 8–11°C with the maximum mean difference of 14°C seen in inland Otago) and high relative humidity (mean RH is often 65–85%), with western parts of the country tending to the highest humidity and inland South Island regions the lowest (MetService, n.d.).

Other countries with similar climates include some areas of the United Kingdom and Japan. In these countries, construction styles are very different (Thomson et al., 2013), and the use of conjoined housing and central heating in these countries impacts strongly on the thermal qualities of the housing stock.

New Zealand homes can be more difficult and expensive to heat due to the use of timber-framed construction styles along with poor insulation levels and the predominance of stand-alone housing compared to other similar-climate countries (Howden-Chapman et al., 2011). New Zealand has high excess winter mortality, estimated to be around 1,600 excess winter deaths per year (Howden-Chapman et al., 2012). Fuel poverty and cold indoor temperatures have been linked to this excess mortality (Howden-Chapman et al., 2012).

Cold temperatures affect the body by causing vasoconstriction and increased blood viscosity. These symptoms increase strain on the heart, which is why most of excess winter mortality is due to cardiovascular conditions, about a third due to influenza and respiratory conditions and a smaller amount due to the increased air pollution common in winter (WHO, 2011).

The relationship between cold and illness in children was demonstrated in a study conducted in Christchurch, New Zealand, as 25% of children admitted to hospital for lower respiratory tract illness came from houses that did not use any heating in winter (Trenholm et al., 2012). This figure compares with a population-wide figure of only 5% of households that don't heat their homes in winter (Isaacs et al., 2010).

Only about 5% of New Zealand homes have central heating, and most New Zealanders only heat one room. Two-thirds of New Zealand households never heat bedrooms, and a significant proportion

don't use any heating in their house at all throughout the year, even in the coldest regions of the country (Isaacs et al., 2010). House age, insulation and heater type impact the mean winter temperature in homes, with older houses, open fires and portable gas and electric heaters all associated with lower mean winter indoor temperatures (Isaacs et al., 2010) as well as lower levels of insulation (Howden-Chapman et al., 2007).

Several intervention studies from New Zealand and the UK have found improved health as a result of increased mean indoor temperature (Howden-Chapman et al., 2007; Lloyd, 2006; Walker et al., 2009; Free et al., 2010). Heater types in New Zealand are varied, as are house designs and indeed lifestyles.

Wood burners and fireplaces

It is clear from research conducted on outdoor air pollution in New Zealand that the use of fireplaces for heating is paradoxically both a significant contributor to ambient air pollution and warm homes. A recent report commissioned by the Ministry for the Environment concluded that emissions from home heating were the most important cause of outdoor air pollution in New Zealand (Ministry for the Environment and Statistics New Zealand, 2014). Unfortunately, this report based its conclusions entirely on monitoring of PM_{10}, the least important fraction of PM to health. Evidence from other research shows that repeating the research and including smaller, more health-relevant fractions of PM could produce significantly different results (Mallet, 2014).

Recent NESAQ air quality regulations may also not be sufficiently addressing the health impact due to this same fact (Mallet, 2014). Particles in wood smoke have been shown to peak in numbers between 0.15 µm and 0.04 µm. In other words, wood smoke has more UFPs than PM_{10} (Semmens et al., 2015). Another finding from this study was that well aged wood was associated with lower numbers of UFPs than newer wood, and it is possible that this fact was linked to the finding that lower-income brackets had significantly higher $PM_{2.5}$ and UFPs than higher-income bracket homes (Semmens et al., 2015). Replication of this work in a sample of New Zealand homes would be valuable.

The HEEP study found only homes using a wood burner were heated to the WHO minimum temperature of 18°C (Isaacs et al., 2010). A similar result was found by the Housing, Heating and Health study, which found that some households, even when given a non-polluting heater free of charge, failed to heat their home to 18°C (Howden-Chapman et al., 2008), primarily due to concerns of receiving a high energy bill. Some families preferred to use their old wood burner rather than the new heater as they were able to collect free fuel or could prepurchase wood and budget the consumption of this fuel. More research is required on the relationship between fuel costs and ability to budget for fuel and heating requirements.

Since 1996, the use of solid fuel for heating has reduced significantly, although 38% of New Zealand homes (594,000 homes) still use a fireplace, down from 62% (788,000 homes) in 1996. As of 2012,

49,000 New Zealand homes were still heated by burning coal, the smoke from which is known to be significantly more harmful to health due to higher levels of PAHs and other carcinogens than untreated wood smoke (Kuschel et al., 2012). South Island homes have a higher prevalence of solid fuel heaters than North Island homes (Ministry for the Environment and Statistics New Zealand, 2014).

Environment Canterbury has put in place regulations to address the problem of outdoor air pollution caused by burning solid fuel for heating, which is particularly serious in that area. As of April 2013, fireplaces older than 15 years can no longer be used, and no new wood burners, including newer low-emissions burners, can be installed in new or existing homes. As of 2015, ultra-low emissions burners were allowed to be installed, and a proposal under review is to ban the use of all burners except ultra-low emissions burners as of 2019 (Environment Canterbury, 2015). Regulations also include the requirements to use dry, well seasoned wood and not to produce visible smoke except for brief periods (Environment Canterbury, 2015).

An important consideration that has not yet been assessed in New Zealand is the contribution of PM directly to the indoor environment from wood burners and fireplaces. Research on modern wood burners from other countries suggests that even lower-emissions wood burners may release unhealthy levels of $PM_{2.5}$ and UFPs into indoor air (Salthammer et al., 2014; Semmens et al., 2015). Semmens et al. (2015) showed that levels in US homes using enclosed wood burners for heating frequently exceeded WHO guideline limits. The authors also showed that peaks in indoor $PM_{2.5}$ did not occur in the same time periods as peaks in outdoor $PM_{2.5}$. While indoor $PM_{2.5}$ peaked between 6pm and 10pm, the typical time period of use, outdoor levels peaked between 10pm and 2am, suggesting that indoor $PM_{2.5}$ was not caused by infiltration of outdoor pollution but from use of the wood burner (Semmens et al., 2015).

Another study from Germany showed that an atmospheric inversion, a common winter-time meteorological occurrence in Christchurch and other New Zealand localities, can reduce the flow of air through the wood burner, significantly increasing the release of PM and CO indoors during the ignition phase of operation (Salthammer et al., 2014). While attention has been paid to wood burner use and its contribution to outdoor air pollution in New Zealand, especially in the Canterbury region, the role of pollution from wood burners directly into indoor air has not been assessed.

Insulation

The vast majority of New Zealand homes built prior to 1978 had no or minimal insulation. Minimum insulation standards were first brought into law in 1978, when newly built houses had to have a thin layer of ceiling insulation installed. In the 1990s, the insulation standards were upgraded to include wall insulation in all newly built housing, and in 2008, this was again updated to include floor insulation. Double glazing has only been required since 2004, and most homes built prior to the 2004 revision to the insulation standard have substandard insulation.

New Zealand's older housing stock has been considerably improved as a direct result of research into the relationship between level of insulation in the home and health (Howden-Chapman et al., 2007). This study found a cost-benefit ratio of 3:1, which led to government subsidies for improving insulation. Adding a small amount of insulation had a significant improvement in occupant health, Including reduced medications, reduced GP visits and reduced hospitalisations. One night's stay in hospital for respiratory infection is roughly the cost of insulating a home.

In 2011, Christchurch (post-earthquakes) faced a critical shortage of hospital beds and, with a view to keeping people well and out of hospital, instigated a plan to insulate the homes of frequent users of the health system. The programme initially focused on people who had been admitted to Christchurch hospital at least twice in the previous year. The programme was so successful at reducing admissions from this 'high user' group that it was extended in 2013 to families with children under 17 who were at risk or families with newborn babies or people who were over 65 years of age. Under this scheme, 1,500 homes were insulated and 450 heaters were provided.

The costs were covered by a partnership with Canterbury District Health Board (DHB), with the DHB's share being $1.7 million. A review of the programme showed a 29.2% reduction in hospital bed days from 900 high-risk patients, which was a saving of $1 million to the DHB. Other savings will accrue over the 40-year life of the insulation, from benefits to other household members and reduction in other community healthcare (Canterbury District Health Board, 2016). Similar schemes are being picked up in Gisborne, with reportedly similar results.

Insulation upgrades have been widely taken up across the nation. However, there are still several hundred thousand homes without insulation or with inadequate insulation. These subsidies have disproportionately been taken up in owner-occupied dwellings, and the improvement in the New Zealand rental housing stock has been negligible (Community Energy Action, 2012). However, the benefits will reside with the homes for at least 40 years, and as owner-occupied homes can become rental accommodation, this distinction can be arbitrary.

Recent law changes have stipulated that, from 2019, New Zealand rental homes must have ceiling and floor insulation. The government estimates that these regulations will apply to 180,000 currently uninsulated homes (New Zealand Government, 2015). Low infrared emissivity coatings have been demonstrated to reduce winter heating requirements by 12.5% (Marino et al., 2015) and should be assessed as a potential gap filler in houses where retrofitting insulation is not practical. Identifying solutions for retrofitting insulation into wall cavities that don't create moisture bridges is a critical issue for improving New Zealand's housing stock. Research is recommended in this area.

The health and indoor air quality benefits (less condensation, mould and dust mites) from a higher than Code insulation standard have not been researched yet.

Ventilation

Ventilation can refer to both active ventilation – opening windows and doors or use of mechanical ventilation (extraction fan or mechanical air supply) – as well as passive ventilation – air infiltration through the building structure, via passive ventilation details such as weepholes and trickle vents and through construction joints or defects in the structure.

Ventilation is important for two main purposes – reducing the concentration of indoor air pollutants and bioeffluents as well as reducing the build-up of moisture.

There is a complex relationship between ventilation, energy efficiency and indoor air quality. New Zealand's maritime climate means outdoor air is warm and moist (see Heating), limiting the moisture take-up capacity of outdoor air (Bassett, 1985). In winter, this means the moisture removal property of ventilation is relative to the temperature difference from indoors to out (Bassett, 1985). Trethowen calculated the difference in required ventilation between heated and unheated rooms to have the same effect in reducing relative humidity and found that, when outdoor temperatures are low (5^{o}C), a continuously heated room will be three times as effective at reducing relative humidity compared with an unheated room for the same number of air changes per hour (Trethowen, 1972). In other words, a cold house needs to be three times more ventilated to reach the same lower relative humidity.

The opposite is true in regards to air pollutants released from construction materials, furnishings and so on. The warmer the air, the faster most pollutants are released from surfaces (Mendell, 2007), although this effect is likely to be less extreme than the relationship with humidity.

Infiltration trends vary considerably through New Zealand's housing stock, due primarily to the construction style. The oldest houses had the leakiest construction styles, including floorboards without tongue and groove detailing, weatherboard cladding and interior sarking all having inevitable construction gaps leading to ample opportunity for air infiltration. Since those earliest designs, many construction features have reduced air infiltration rates in newer homes, including tongue and groove floorboards, sheet flooring, concrete slab floors, sheet lining of interiors and exteriors, aluminium joinery, wraps under claddings and insulation. Masonry veneer (brick) cladding has been found not to reduce air infiltration compared to claddings such as weatherboards (Bassett, 1985). Building complexity has also been found to relate to air infiltration rates, increasing with complexity of the building's shape (Bassett, 1985). These factors have seen mean air infiltration rates reduce from an average of 19 air changes per hour (ach) at 50 pascals (Pa) of air pressure in pre-1960s homes to 4.5 ach @ 50 Pa in houses built since 2000 (Overton, 2013).

A concerted effort to design houses to minimise air infiltration came about in the 1970s as a result of the Organization of the Petroleum Exporting Countries (OPEC) oil crisis and resulting drive towards energy efficiency. In New Zealand, there are still many old houses that are too draughty to heat

efficiently, while many new houses may be becoming too airtight for the occupants to effectively maintain healthy air quality without some form of mechanical ventilation (Overton, 2013).

Another significant change over this timeframe that has not been investigated in relation to ventilation is the fact that many households are empty during work hours, and consequently windows are shut all day. Ventilation designs and standards in New Zealand are based on the assumption of windows being used as a primary aspect of ventilation (Overton, 2013). However, with houses more commonly empty of occupants during the day, it may be that relying on people to ventilate their houses through opening windows is no longer sufficient for reducing pollutants and humidity to healthy levels. Trickle vents in aluminium window joinery as a primary source of ventilation may not be sufficient for reducing harmful chemicals indoors (Howieson, 2014).

Another change is that of window styles. With a significant move away from timber joinery to aluminium joinery, window styles in New Zealand have changed significantly. Small fanlight windows, which are common in older houses, are less common in newer houses. Aluminium joinery is a thermal bridge and thus creates a site for condensation to form and mould to grow. It may increase the humidity on the back face of curtains and increase mould growth on curtains. Research is required in this area.

Research into ventilation behaviours according to style of window has recently been commenced by Massey University. Factors that contribute to ventilation behaviours may include:

- noise levels outside
- living close to a busy street
- perception of personal security – age and gender of the occupants, height of windows above ground level
- owning a pet
- whether or not the house is occupied during the day.

More research is required to understand ventilation behaviours especially in light of evidence that New Zealand homes are becoming more airtight and mechanical ventilation may be required.

Studies measuring the relationship between ventilation rates in offices and schools and the wellbeing of occupants have found higher ventilation rates are associated with reduced absences and symptoms of BRI (Fisk, 2013; Wargocki, Wyon & Fanger, 2004). Other studies have also found higher ventilation rates associated with increased performance on computerised English tests in schools (Bakó-Biró et al., 2012) and higher productivity in offices (Seppänen, Fisk & Lei, 2006; Hamilton et al., 2015). Improved filtration in air conditioning systems has also been associated with improvements in health and performance (Fisk, 2013). Few studies have directly measured the health effects of using dirty filters in forced air ventilation systems, although one study measured reported BRI symptoms and productivity in a call centre. When the air conditioning system was used

with dirty air filters, symptoms increased and productivity decreased, while the reverse associations were observed when filters were replaced with clean ones (Wargocki et al., 2004).

Several intervention studies in homes have looked at filtering air and the effect on children's asthma. A large study nested in the third US National Health and Nutrition Examination Survey (NHANES III) looked at the association between asthma and the use of gas stoves. This study found that, in homes using gas stoves for cooking, children whose parents reported using ventilation while cooking had significantly reduced odds of asthma, wheeze and bronchitis than homes where gas stoves were used without ventilation (Kile et al., 2014). This is likely due to the respiratory irritant effects of nitrogen dioxide.

With the rapid transition to the use of heat pumps in New Zealand homes over the past two decades, evaluation of these changes could be valuable. Topics worth investigating are, whether heat pumps are being cleaned and maintained as frequently as recommended, whether heat pumps are being used instead of ventilation (as it is often erroneously assumed they ventilate the indoor air) and water ingress resulting from installations.

Building maintenance

Home maintenance perhaps suffers the scientific disability of being 'common sense'. Maintenance is frequently implicated in health literature in relation to indoor dampness and mould and indeed is enshrined in the 2009 WHO guidelines on dampness and mould (WHO, 2009) but has not been assessed in direct relationship with health effects. Studies that have looked directly for an association between house maintenance and dampness or mould consistently find a positive association (Platt et al., 1989; Howden-Chapman et al., 2005; Taptiklis et al., 2011).

In the US, Rosenbaum et al. looked at home characteristics and mould, including housekeeping, cockroaches, pets, leaks, house type and season, and found that the two strongest predictors of mould presence in homes were low income and house built prior to 1955 (Rosenbaum et al., 2015). Page and Curtis also found that almost 87% of households with incomes below $20,000 were living in homes that required immediate repairs at an average cost of $5,800 (Page & Curtis, 2013).

Rental houses in New Zealand have been demonstrated to have significantly poorer maintenance than owner-occupied homes in the latest house condition survey (Buckett et al., 2012). This survey found almost half of the rental houses (44%) were in poor overall condition, compared with 25% of owner-occupied houses. Overall, 34% of New Zealand houses were in poor condition. The 2005 house condition survey reported that 34% of houses with timber weatherboard cladding had visible paint deterioration (Clark, Jones & Page, 2005).

According to a study on the life cycle of New Zealand building components (Mithraratne & Vale, 2004), exterior paint can be expected to remain efficient in terms of waterproofing for as little as

8 years. With two-thirds of New Zealand houses relying on paint finishes for waterproofing of their exterior (Buckett et al., 2012), this is a potentially significant problem. Massey University and BRANZ are currently conducting trials to assess the impact of maintenance of painted claddings on indoor air conditions.

Decisions made or actions undertaken or not undertaken during the design and construction phase of any building may impact on the burden of maintenance required to keep the building in good condition. Research by BRANZ (Page, 2015) has found that 81% of owners of new-build houses have to call builders back to remedy one or more defects in their brand-new home. Of these defects, plumbing or electrical were most common. Plumbing leaks are frequently the cause of dampness problems in homes, especially due to the fact that plumbing tends to be hidden, therefore problems can persist a long time prior to detection. Forty-eight percent of new homes had plumbing defects needing remediation and 46% electrical faults. This was followed by interior claddings including plaster and interior paint faults at around 30% of new homes requiring defect repair. Plumbing and electrical faults may not be immediately apparent, and the three council inspections that domestic buildings must pass to be compliant are not intended to inspect every aspect of a build, so there are likely to be residual quality issues in many new homes being built in New Zealand, which increases the burden of ongoing maintenance of the home (Page, 2015).

In the UK, 65% of the total annual building work conducted was for maintenance or repairs to existing buildings, while in New Zealand, the proportion was only 50% (Page & Curtis, 2013). The authors suggested this could be related to difference in relative age of the housing stock and that cultural behaviours towards home maintenance may be more relevant to the difference in these figures. A recent study has shown that New Zealanders over 65 years of age tend to live in better-maintained houses than the general population, although this may be confounded by the fact that they also tend to live in newer houses (Jaques et al., 2015) and that this age group are more likely to own their own homes. Clarification of the relationships between maintenance behaviours, cultural attitudes and home ownership may point to cultural attitudes towards maintenance that are demonstrably healthier than others.

Energy efficiency

The International Energy Agency ventilation code suggests air exchange rates of 7 air changes per hour or less for energy efficiency purposes are required to maintain indoor air quality. A study of New Zealand homes by Bassett surveyed air change rates and found many older homes (pre 1970s) had higher rates than 7 air changes per hour – some up to four times as draughty as this recommended maximum (Bassett, 1985).

Some government-led efforts have been undertaken to encourage energy efficiency improvements to buildings in the UK. However, results from evaluations on both the health outcomes and

improvements to indoor air quality in such schemes have been conflicting (Howieson, 2014). Intervention studies showing increased energy efficiency associated with fewer health effects (Howden Chapman et al., 2007; Howden-Chapman et al., 2008; Ahrentzen, Erickson & Fonseca, 2015; Colton et al., 2014), while others show the opposite effect, including increased asthma (Sharpe, Thornton, Nikolaou & Osborne, 2015) and health effects (Milner et al., 2014).

Reduced ventilation has been suggested as the mechanism by which energy efficiency improvements may increase health effects by leading to increased build-up of harmful indoor pollutants (Sharpe, Thornton, Nikolaou & Osborne, 2015; Howieson, 2014). Housing improvement, especially of the current building stock, is a complex problem, and part of the difficulty in assessing energy efficiency improvements is that interventions frequently include a number of changes that occur simultaneously, making it difficult to ascribe particular outcomes to particular causes. Lloyd, Bishop and Callau (2007) conducted a stepped energy retrofit of state houses in Dunedin and found the standard Housing New Zealand programme was ineffective in Dunedin's climate and households were exposed to temperature less than 12^{o}C for nearly half the day in winter (Lloyd et al., 2007).

One large review of housing intervention studies published recently included 39 separate interventions and concluded that those that aim to improve thermal comfort appear to improve health, while results of other interventions are less clear (Thomson et al., 2013).

It has been noted that landlords are frequently reluctant to improve the energy efficiency of their rental homes (Ambrose, 2015), and it is likely that the lack of clear understanding (Xiong et al., 2015) demonstrated in energy efficiency research is a contributing factor. Therefore, it is important to keep working on this complex area of indoor environment research.

Passive house design has contributed valuable understanding to energy efficiency ideals in new buildings (Ionescu et al., 2015). Next steps could include extrapolating this learning to assess the viability of using passive heating techniques for deep retrofits of existing houses.

Climate change impact

Ilacqua et al. (2015) modelled potential changes to infiltration rates due to rising temperatures from climate change, concluding that, while peak summer infiltration rates may increase, overall, there would be an average decline in infiltration rates by approximately 5%. These effects would impact pollution profiles indoors, possibly increasing damp-related winter exposures when infiltration was reduced while increasing any infiltration of outdoor air pollutants during summer (Ilacqua et al., 2015).

Outdoor air pollution is also likely to increase due to climate change, as photochemical reactions producing smog will increase with either an increase in emissions or sunlight. If both sunlight and emissions increase, outdoor air pollution could increase dramatically (Vardoulakis et al., 2015).

Furthermore, outdoor air pollution is also predicted to increase in New Zealand due to increased forest fires. Forest fires could increase in frequency by more than 50% in eastern parts of the country (Bengtsson et al., 2007) – an increase that, if it does eventuate, is likely to lead to measurable increases in poorer health.

Another likely effect of climate change is increased flooding (Bengtsson et al., 2007; Institute of Medicine, 2011), which can impact health via the indoor environment by causing water damage in buildings. Amongst the dampness-related factors that are consistently associated with increased respiratory health effects are water damage and water stains (Institute of Medicine, 2004; WHO, 2009; Fisk et al., 2007; Kanchongkittiphon et al., 2015) (see Asthma). Again, if increased flooding does increase the prevalence of water damage in housing, this is very likely to result in increases in negative respiratory health outcomes including asthma.

Conclusions

New Zealanders experience an excessive rate of asthma, COPD, rheumatic fever, hospitalisations from skin infections, excess winter mortality and other potentially avoidable conditions. Poor-quality housing is implicated in many of these.

Several intervention studies have shown that small improvements to housing, such as heating and insulation, can have significant improvements in health and that these are cost-effective.

However, the research in this area is the tip of the iceberg, and more can be done to achieve housing and schools that protect the health and wellbeing of the occupants. There are particular gaps in understanding the issues specific to New Zealand and specific to the under-researched settings of schools, early childcare and housing for the elderly.

The: Towards Healthy Air in Dwellings in Europe (THADE) study concluded that there is a lack of evidence-based information on the appropriate measures for preventing adverse health effects caused by indoor air pollution (Franchi et al., 2006). This lack of information is one of the reasons for the global dearth of indoor air regulations and not the lack of a perceived problem (Fuentes-Leonarte, Ballester & Tenías, 2009). Indoor air science presents serious difficulties for the researcher, mainly due to the heterogeneity of pollutants, which makes it particularly difficult in epidemiological studies to eliminate confounders.

The WHO *Guidelines for indoor air quality: Dampness and mould* (WHO, 2009) made specific mention of envelope design and ventilation as means to control moisture and hence mould. The need to control moisture is consistently made throughout much of the literature. This is obviously a challenge in New Zealand's maritime climate. More studies investigating the incidence of mould and other biological agents is required as a means to reduce moisture, dampness and mould. Greater understanding of the health effects of dampness and biological agents is also recommended.

Outdoor air pollution in New Zealand has reduced by up to 14% since 2001 (Ministry for the Environment and Statistics New Zealand, 2014) due to concerted efforts to measure, regulate and reduce pollutants at source. However, even with the recent research focus, there is more to learn about New Zealand's ambient air pollution, especially characterisation and source (both mineral and biological) apportionment of PM. There is also a lot that needs to be learned about the migration of outdoor pollutants into indoor spaces and safe means to provide ventilation in areas where outdoor environment is polluted or noisy.

While monitoring of PM_{10} has seen significant advances in New Zealand in recent years, there is more research and monitoring required of the $PM_{2.5}$ fraction. It is clear that the effect of PM_{10} on human health is minimal compared with smaller particles. While some centres have already been conducting PM monitoring (Fisher et al., 2007; Mitchell, 2012), consistency of monitoring practices throughout New Zealand is important for making accurate reports of current pollution and modelling

future scenarios (Ministry for the Environment and Statistics New Zealand, 2014) as well as research into the health effects.

The relationship between outdoor air pollution and the migration of these pollutants indoors is an issue that is largely unknown. As cities become more densely populated and people are living closer to roads and industrial areas, there is a need to measure and characterise indoor concentrations of pollutants that have originated outdoors. While it is assumed that the home has a protective effect, this is unlikely to be the reality.

Also relating to outdoor air pollution, findings from urban schools in Europe show a concerning trend of pollution components including $PM_{2.5}$, benzene and formaldehyde consistently measured at higher levels in classrooms than in ambient outdoor air (Stranger et al., 2008; Sofouglu et al., 2011; Rivas et al., 2014). Children's increased vulnerability to such toxins means that such a relationship could represent a significant preventable burden of ill health in future years. Improved understanding of indoor air pollutants inside New Zealand classrooms is highly desirable.

Many measurements of indoor air and dust pollutants have been taken and assessed usually only in terms of the determinants of those pollutants and impact on health effects. Using measurements from epidemiological studies to generate hypotheses regarding the movement and behaviour of pollutants in the domestic setting may prove useful.

Fertility and diabetes are important health concerns in New Zealand, so more work is needed characterising New Zealanders' exposure to such endocrine-disrupting chemicals as flame retardants, pesticides and phthalates
indoors in New Zealand. This work should include workplaces and schools as well as the domestic environment.

Evidence is growing to support the view that indoor air contaminants may be important in the pathogenesis of childhood cancers. Case control studies examining exposure in newly diagnosed child cancer cases and cohorts including measurements in the homes of pregnant mothers will be valuable to identifying pollutants of concern. Outdoor exposures may remain more stable over time than indoor exposures, so for these exposures, retrospective study designs may be valuable.

More studies characterising indoor exposures such as VOCs in New Zealand homes and schools, especially new builds and renovations, would be valuable. Of particular interest is buildings located in noisy neighbourhoods, as there is evidence that occupants are not opening windows, hence ventilation is compromised.

In order to study a wide variety of contaminants at one time, it is necessary for a study to have large numbers of participants. With New Zealand's population a limiting factor to the size of many studies, particularly in the case of rare health outcomes such as childhood cancer, collaborating with partners in other countries will improve the value of such work.

There is a need for improved technology transfer and independent advice to be made readily accessible to property owners and policy makers. There is currently a vast number of companies marketing 'solutions' for a healthy home – these lack evidence of proof of the claims. Several city and district councils in New Zealand provide independent advice on home improvement to their populations, and this is a valuable service that should be maintained and expanded, rather than contracted as has recently occurred in one city.

Independent advice is invaluable for creating government regulations to protect the most vulnerable in our communities. The value of independent versus commercial advice can be compared between insulation and ventilation advice in New Zealand. While insulation has received attention from independent research bodies in this country, there is now sufficient understanding to allow government to regulate on this specific issue (New Zealand Government, 2015), while ventilation and heating have generated a proliferation of industry advice but little independent attention.

The production of $PM_{2.5}$ and UFPs indoors is an important new area of discussion (Ostro et al., 2015; Polidori et al., 2006; Spilak et al., 2014). While cigarette smoking, candles, incense and open fireplaces are significant contributors to indoor $PM_{2.5}$ and UFPs (Spilak et al., 2013; Salthammer et al., 2014), a surprising recent finding is that cooking may be the most significant contributor to indoor PM in many homes and even an important contributor to outdoor PM (Ostro et al., 2015; Ham & Kleeman, 2011).

Ventilation has become exponentially more important over the last two or three decades, as newer buildings become more airtight and coincidentally more VOC-emitting materials are used in construction and furnishing materials. New Zealand's current practices for ventilating homes may not be sufficient to provide healthy comfortable homes. Although regulations are in place to reduce outdoor air pollution in New Zealand, with urban intensification and high-density housing increasing, there is the risk of a large health problem being built into our housing stock if consideration is not taken of infiltration of outdoor air pollution in urban areas at the design phase.

As mechanical ventilation is likely to become increasingly used for achieving indoor air quality, work to develop 'smart ventilation' that can monitor and respond to the specific needs of the home or school may be required. Energy efficiency is very important for schools and many homes.

Research into combined effects of multiple pollutants has been explored in very few studies in New Zealand or internationally (Phipps & Warnes, 2007; Carlsten & Georas, 2014), but this approach shows promise, especially in regards to asthma epidemiology. Of course, this approach presents difficulties in that increasing the number of factors to be assessed usually necessitates an increase in the number of study participants for the sake of statistical power and therefore the costs of the research. Inventive and collaborative strategies may be necessary to identify useful study populations. For example, the use of natural experiments could prove fruitful and cost-effective.

This review has identified key aspects of indoor air quality as it affects New Zealand homes and schools with the intent to generate interest and help identify areas for future research. It is intended to be a living review that is added to as new research is published.

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