



**Potential Health Impacts Associated with Mould  
in “Leaky Buildings”: A review commissioned  
by the Auckland City Council**

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## KEY POINTS

- Many reviews of the scientific literature indicate associations between mould and damp and respiratory symptoms. The best systematic literature review we identified found clear evidence that exposure to damp and mould (a common problem with leaky buildings) is associated with upper respiratory tract infections, coughs, wheeze, asthma symptoms in sensitized asthmatic persons and hypersensitivity pneumonitis in susceptible persons.
- There is some evidence suggesting that exposure to damp and mould in leaky buildings is associated with the *development* of asthma, but this is still not scientifically certain. There is also uncertainty around exposure to damp and mould and other possible adverse effects eg, on mental health, fatigue and headache.
- The association between mould and damp and respiratory outcomes may be mediated by other factors, such as exposure to house dust mite allergen or bacterial endotoxin (which are also related to dampness).
- Moulds produce a complex mixture of volatile chemicals that are frequently evident as 'mouldy smells'. The volatiles are mixtures of alcohols, esters, aldehyde, various hydrocarbons and aromatics, which can cause nausea and make some people quite ill.
- There is evidence for adverse effects of particular mould species such as *Aspergillus* (a cause of allergic broncho-pulmonary aspergillosis). Also particular mould species seem to be more strongly associated than others with positive allergy skin test results.
- Many aspects of leaky buildings increase perceptions of risk, eg, risks are unknown, or poorly defined and quantified. The risks are unintended, unexpected, unplanned for and largely unseen until major structural damage becomes evident. Organisations, rather than individuals, play the central role as agents of harm and rescue, further undermining individuals' sense of control over their health and assets, and thus their social status.
- Preliminary cost estimates from the health effects of damp and mould from leaky dwellings were estimated in Appendix One to this Report. This work found that the incremental health costs of leaky dwellings, primarily affecting respiratory and mental health conditions through increased mould and damp, are around \$474 million in present day (discounted value) terms. This estimate used a 5% discount rate over a 10-year horizon, and while it is subject to considerable uncertainty, it is probably conservative.

## EXECUTIVE SUMMARY

**Aim:** To detail the up-to-date scientific evidence on the health impacts associated with exposure to mould from “leaky buildings”, with particular reference to damp and mould in homes in the New Zealand setting.

**Methods:** Literature searches were undertaken using a range of search strategies (but particularly of the medical literature using Medline). A specific focus was on reviews, recent intervention studies, and of the New Zealand literature.

**The international scientific evidence:** The most rigorous review to date on indoor mould and health appears to have been by the Institute of Medicine (IOM) of the US National Academies of Science (published in 2004). Key findings included the following:

Sufficient evidence of an association:	<ul style="list-style-type: none"> <li>• Upper respiratory (nasal and throat) tract symptoms</li> <li>• Wheeze</li> <li>• Asthma symptoms in sensitized asthmatic persons</li> <li>• Cough</li> <li>• Hypersensitivity pneumonitis in susceptible persons</li> </ul>
Limited or suggestive evidence of an association:	<ul style="list-style-type: none"> <li>• Lower respiratory illness in otherwise-healthy children</li> </ul>

This IOM review also found similar results for indoor dampness and came to the conclusion that “excessive indoor dampness is a public-health problem”.

The next most detailed review identified was part of a “Towards Healthy Air in Dwellings in Europe” (THADE) Project in 2004. It found that “mould” was one of the major health determinants in dwellings. In particular, it concluded that: “exacerbation of asthma and allergic illness is strongly linked to exposure to allergens such as ...fungi and mould...”.

Another substantive review was for the World Health Organization in 2004. It included data from the WHO LARES study (involving 8 studies in European countries). In addition to respiratory and allergic conditions associated with mould in houses, it also reported associations with poorer mental health (chronic anxiety and depression) and cardiovascular conditions.

The results of the first meta-analysis to quantify the risk of mould and health were available in 2007. Based on 33 peer-reviewed United States studies, the authors estimated that exposure to dampness and mould “raises the risk for various adverse respiratory outcomes by 30–50%”. These outcomes included: upper respiratory tract symptoms, cough, wheeze, current asthma, ever-diagnosed asthma, and asthma development.

With regard to the specific role of fungal cell wall components (glucans), a review in 2005 concluded that the “observational and experimental studies reviewed suggested some association between (1→3)-β-D-glucan exposure, airway inflammation and symptoms, however, results were mixed and specific symptoms and potential underlying inflammatory mechanisms associated with exposure could not be identified.” However all of the seven epidemiological studies relating to glucans in the indoor environment in this review, showed evidence of adverse health effects associated with exposure to glucans.

All the other published recent reviews examined for this Report (n=12 reviews) detailed associations between mould exposure and health problems (particularly respiratory symptoms and asthma). Nevertheless, there are often many methodological limitations with the design and the quality of the available studies (particularly around the accurate measurement of mould and other potentially relevant exposures). Other limitations include the potential for publication bias and possibly the competing interests of some of the authors (given the litigious context of the mould and health topic in North America).

Intervention studies show that reducing dampness and mould in houses is associated with various health improvements. But these intervention studies have not clearly identified the specific benefit of mould removal on these improved health outcomes.

***The burden of mould exposure in New Zealand:*** The New Zealand data from multiple studies is suggestive that mould in New Zealand homes is relatively common (eg, over a third of homes in a national survey). Amongst low-income communities, Pacific communities and tertiary student accommodation, the prevalence of reported visible mould in housing appears to be higher than the national average. The reported problems with mould in New Zealand houses may be higher than in other countries, and there is further preliminary evidence for this when considering fungal (1→3)-β-D-glucans levels in house dust.

***NZ evidence for mould and health:*** The New Zealand data tends to be consistent with the international literature that indicates mould in housing is associated with various adverse health outcomes. But despite the overall pattern there are some studies that show no association and even one that found a significant negative association (for current asthma). It is also plausible that the association between mould and respiratory outcomes is not necessarily causal but is mediated by other factors such as exposure to house dust mite allergen or bacterial endotoxin. More specific aspects are as follows:

- ***Respiratory symptoms:*** A total of six published studies relating to respiratory symptoms and mould exposure in New Zealand were identified. Of these two found a statistically significant positive association with adverse health impacts. Another found this result for the population sampled from Christchurch City, but not for the populations studied in the Hawkes Bay or Wellington. Two publications of the same study population reported positive relationships between mould sensitisation and bronchial hyperresponsiveness. Furthermore,

two New Zealand studies found a positive association between mould and higher levels of dust mite allergen in homes. In contrast to the above, one study found that mould exposure was *negatively* associated with current asthma at a significant level.

- **Asthma severity:** Of studies of asthma severity since 1990, there is evidence from five studies for an association between mould exposure/mould sensitisation and asthma severity. Dunedin cohort study data provides some evidence and combined data for New Zealand and Australia indicates an association between severe (versus mild) asthma with sensitisation to moulds. Similarly, an Auckland study found that a positive skin test for fungal allergens is a significant risk factor for admission to an intensive care unit with an acute attack of asthma. A prospective study found a weak association between days of high basidiospore counts and increased nocturnal waking and reliever medication use among people with asthma. However, it may be that only some age groups are affected (eg, those aged 5-14 years and 15-44 years) given the pattern of peak asthma hospitalisation rates in autumn months when fungal spore levels tend to be highest.
- **Other health outcomes:** One study reported an association between dampness/mould in housing and maternal depression, while another found no association with atopic dermatitis.
- **Intervention study:** The findings from a large intervention study indicated that insulating households resulted in reductions of reported mould and significant improvements in health outcomes (ie, in terms of self-rated health, self-reported wheezing, days off school and work, and visits to general practitioners as well as a trend for fewer hospital admissions for respiratory conditions). The specific role of reduced mould levels in these health outcomes is plausible however it remains unclear given that other factors were involved (eg, particularly the significantly warmer and drier indoor environment in the insulated houses).

**Risk perception issues:** The literature on mould and risk perception is poorly developed and so we applied general risk perception principles to the issue of mould in houses in New Zealand. This work suggested that there are many risk perception principles that could be expected to heighten the public's concerns about the effects of mould on their health.

**Summary:** The available international scientific evidence concerning indoor mould and health is far from conclusive but the best reviews available indicate sufficient evidence for associations between exposure and respiratory symptoms. This and other evidence probably provides sufficient justification for a public health response and individual householder action towards remediation, particularly where household members have established asthma. The New Zealand data from multiple studies are suggestive that mould in New Zealand homes is a relatively common problem. The scientific work in New

Zealand is also consistent with the international literature that indicates that mould in houses may harm health, particularly respiratory health.

***Implications for research:*** While there is sufficient evidence for responding to mould in houses additional research is desirable to better inform the best responses. This Report details the options around: (i) undertaking regular literature reviews; (ii) a descriptive study of the health and concerns of people living in “leaky buildings”; (iii) a case-control study; and (iv) intervention studies (eg, that involve remediation).

## 1) BACKGROUND AND INTRODUCTION

The emphasis to date with “leaky buildings” in New Zealand has been to address the engineering and construction aspects. By contrast, this Report was commissioned by the Auckland City Council to cover the international and national literature of the known health effects of “leaky buildings”. It consists of a systematic review of the medical and public health literature, looking particularly, but not exclusively at the effects of mould on respiratory and other dimensions of health.

To better understand the range of health problems that have been highlighted by affected home-owners, we have also included a brief discussion of risk perception and assessment. This Report also includes an economic estimate of the health-related consequences of leaky dwellings (see Appendix One).

**What is mould?** Visible fungal colonies found indoors are commonly called “mould” or “mildew”. This Report refers to “mould” interchangeably with “fungus”. Specific species and genera of fungi are mentioned where appropriate eg, “Cladosporium”, “Penicillium”, or “Alternaria” species. This Report also discusses “glucans” which are *non-allergenic* water-insoluble structural cell wall components of most fungi. The measurement of these potential markers for mould/fungi exposure has increased substantially in recent years. Of note is that mould is not the only dampness-related microbial agent and mould growth is also usually accompanied by bacterial growth. Also, structural cell wall components of gram-negative bacteria (“endotoxin”) have been shown to cause airway inflammation, and to exacerbate asthma symptoms.

**Non-health aspects of mould and housing:** This review is focused on health issues but it is important to note that citizens, local government and national governments may have other reasons for being concerned about mould and dampness in houses. One of these is the need to prevent damage to building materials and furnishings and hence extend the functional lifetime of these material goods. These are particularly relevant issues for home-owners and for agencies that own social housing stock.

Improved control of mould and dampness may go hand-in-hand with improving insulation levels at the home, town and city level. This can achieve benefits in terms of energy cost savings for consumers, improved national energy security, and reductions in greenhouse gas emissions associated with electricity generation. Energy savings for low-income consumers may contribute to reducing inequalities in New Zealand society (given that this is a government concern).

The odour of mould can be a nuisance that some householders wish to avoid and which can adversely affect the value of their investment in their home. This is particularly relevant in New Zealand where such a large proportion of household wealth is tied up in housing stock.

## 2) METHODOLOGY FOR THIS REVIEW

### *Search strategies*

**Searches for systematic reviews:** The major databases used for such reviews were searched on 31 May 2007: The Cochrane Library (2<sup>nd</sup> quarter 2007); the Database of Abstracts of Reviews of Effectiveness (DARE) (2<sup>nd</sup> quarter 2007); the United States Preventive Services Task Force (USPSTF website); the United States Task Force on Community Preventive Services (TFCPS website); and the Canadian Task Force on Preventive Health Care (CTFPHS website). Search terms included: mould, mold and fungal.

**Medline searches (NZ):** Searches of Medline for the period 1966 to 29 May 2007 were conducted for New Zealand related literature. The following search terms were used: “Zealand and (mould or mold)”; “Zealand and fung\*”; “Zealand and (damp\* or moisture)”; “Zealand and insulat\*”; “Zealand and (home or hous\*)”; “Zealand and (endotoxin or glucan)”; “Zealand and dust”; “Zealand and Cladosporium”; “Zealand and Alternaria”; “Zealand and Penicillium”; “Zealand and Stachybotrys” “Zealand and Zygomycetes”; and “European Community Respiratory Health Survey” (since the latter included New Zealand data). The names of prominent New Zealand-based researchers in the area were also used: “Wickens-K”, “Douwes-J”, “Pearce-N”, “Siebers-R”, “Sears-M” and “Kearns-R”.

**Medline searches (international):** The international literature on mould and health is vast eg, over 3150 articles using the search term in Medline: “(indoor or hous\*) and (mold or mould)”. Therefore the searches were particularly focused on reviews that follow from the period covered by major previous reviews (ie, by the IOM [Institute of Medicine 2004]) ie: for the period 1 January 2004 to 28 May 2007. The following search terms were used: “‘systematic review’ and mold”; “‘systematic review’ and (fung\* or glucan)”; “review and (home or hous\*) and (glucan or mold or fung\*)”; “review and Stachybotrys”; “review and asthma and (glucan or mold)”; “review and mold and (smell or odor)”.

To identify relevant trials the following terms were used: “trial and mold; “trial and damp”; “intervention and damp”; and “intervention and mold”. Also, to identify relevant psychological literature the following terms were used: “mold and (smell or odor) and psych\*”; “mold and (anxiety or depression)”; “risk and perception and mold”; “public and concern and mold”.

**Google Scholar searches:** To identify unpublished reports in the grey literature, the search term used was: “Zealand and mould and housing and (health or asthma or respiratory)” for the period 1 January 2000 to 6 April 2005.

**Searches within online journals:** Each issue of the following six online journals was searched for articles on New Zealand and mould/mold in the

indoor environment: *BMC Pulmonary Medicine*, *Chest*, *Indoor Air*, *Indoor and Built Environment*, *NZ Medical Journal*, and *Thorax*. These searches included “in press” articles that were online as at 28 May 2007.

**Additional methods:** Electronic copies of all the identified reviews relating to dampness/mould and health were searched for the word “Zealand”. Further articles were identified through examining the reference lists of the literature that was identified. A hand search of the abstracts and proceedings of a recent major housing and health conference was undertaken [de Oliveira Fernandes et al 2006a; de Oliveira Fernandes et al 2006b].

### **Assumptions made**

**The relevance of dampness in housing:** Some of the literature in this Report overlaps with exposure to “dampness” and health outcomes. This literature is assumed to be relevant given that dampness is a well-established risk factor for mould growth. Also dampness may support the presence of dust mites which themselves may contribute to mould growth (by providing carbon and nitrogen in their faeces). Also dust mites may be sustained by the presence of mould since these mites are known to consume some fungal species [Van Asselt 1999].

**Fungi in outdoor air:** Some of the information in the New Zealand part of this review relates to airborne fungi exposure that may have occurred outdoors. However, this is assumed to be of some potential relevance as there is evidence that airborne fungi exposure encountered indoors generally parallels that found outdoors [O'Connor et al 2004].

**Methods for assessing mould in houses:** Some of the data cited in this Report relates to mould/damp in housing reported by household residents. The validity of these observations is not well established and for some populations (eg, immigrant communities) there may be issues around understanding what the terms “mould” and “damp” mean. A recent US study reported that families “were highly inaccurate in reporting the presence of mold” [Kercsmar et al 2006]. Social desirability bias may also be relevant in how people respond to such questions. Key reviews also highlight the lack of valid methods to accurately assess mould exposure in epidemiological studies [Kolstad et al 2002; Douwes & Pearce 2003; Institute of Medicine 2004]. The limitations of counting culturable spores in settled dust or in the air, is also described in this literature. Nevertheless, for the purposes of this Report all relevant studies relating to New Zealand are described and not rejected on such methodological grounds. These limitations are further discussed however, in the Discussion Section of this Report.

Furthermore, a focus for this Report was on exposure in the domestic setting and for people without severe immunodeficiency disorders (who are at substantially increased risk of developing pulmonary and systemic fungal infection).

### 3) INTERNATIONAL EVIDENCE ON MOULD EXPOSURE AND HEALTH

#### *Findings of major reviews (the 5 years prior to the 2004 IOM review)*

**IOM 2000 review:** In 2000 the Institute of Medicine (IOM) published a major review of indoor air exposures and asthma [Institute of Medicine 2000]. The findings are summarised in the table below. Of note is that the IOM found “sufficient evidence of an association” between exposure to “fungi or molds” and the exacerbation of asthma in sensitive individuals. However, it concluded that there was insufficient evidence concerning the role of “fungi or molds” in the *development* of asthma.

Also of note is that the IOM identified other indoor agents as having sufficient evidence for causing exacerbations of asthma (eg, cats, cockroaches, house dust mite allergens and secondhand smoke (SHS)). It also stated that there was “sufficient evidence of a causal relationship for the development of asthma” for house dust mite allergens. This issue remains however, an area of on-going controversy scientific controversy. Less controversial is the finding by the IOM that there is “sufficient evidence of an association for the development of asthma” with exposure to SHS in preschool-aged children.

The role of house dust mites is particularly relevant as these are known to feed on some fungal species (and moisture is needed by both mites and fungi to survive indoors). Similarly, the role of cats is of relevance since cat ownership may be a risk factor for mould growth [Bischof et al 2006]. It has also been reported that damp indoor environments can support cockroach and rodent infestations where there is standing water (ie, pooled water) and that excessive moisture may initiate chemical emissions from building materials and furnishings [Institute of Medicine 2004].

Table 1: Summary of findings from the IOM review regarding the association between indoor biologic and chemical exposures and the exacerbation of asthma in sensitive individuals

<b>Biological agents</b>	<b>Chemical Agents</b>
<b><i>Sufficient evidence of a causal relationship</i></b>	
Cat	SHS (in preschool-aged children)**
Cockroach	
House dust mite*	
<b><i>Sufficient evidence of an association</i></b>	
Dog	NO <sub>2</sub> , NO <sub>x</sub> (high-level exposures)
Fungi or moulds	
Rhinovirus	

Biological agents	Chemical Agents
<b><i>Limited or suggestive evidence of an association</i></b>	
Domestic birds	SHS (in school-aged and older children, and in adults)
<i>Chlamydia pneumoniae</i>	
<i>Mycoplasma pneumoniae</i>	Formaldehyde
Respiratory Syncytial Virus (RSV)	Fragrances
<b><i>Inadequate or insufficient evidence to determine whether or not an association exists</i></b>	
Cow and horse	Pesticides
Rodents (as pets or feral animals)	Plasticizers
<i>Chlamydia trachomatis</i>	Volatile organic compounds (VOCs)
Endotoxins	
Houseplants	
Pollen exposure in indoor environments	
Insects other than cockroaches	
<b><i>Limited or suggestive evidence of no association</i></b>	
(No agents met this definition)	

\* The IOM also reported: “sufficient evidence of a causal relationship for the *development* of asthma”.

\*\* The IOM also reported: “sufficient evidence of an association for the *development* of asthma”.

***Mycotoxin review (2000):*** This review specifically considered the health effects of mycotoxins in indoor air [Robbins et al 2000]. It covered animal exposure studies, case reports, and epidemiological studies. It concluded that:

“although evidence was found of a relationship between high levels of inhalation exposure or direct contact to mycotoxin-containing molds or mycotoxins, and demonstrable effects in animals and health effects in humans, the current literature does not provide compelling evidence that exposure at levels expected in most mold-contaminated indoor environments is likely to result in measurable health effects. Even though there is general agreement that active mold growth in indoor environments is unsanitary and must be corrected, the point at which mold contamination becomes a threat to health is unknown.”

***Bornehag et al 2001 review:*** This review identified 590 peer-reviewed articles of which 61 were used in the review [Bornehag et al 2001]. The main finding was that “dampness” in buildings “appears to increase the risk for health effects in the airways, such as cough, wheeze and asthma”. The authors also reported that:

“there also seems to be an association between ‘dampness’ and other symptoms such as tiredness, headache and airways infections. It is

concluded that the evidence for a causal association between 'dampness' and health effects is strong. However, the mechanisms are unknown." .... "Sensitisation to mites may be one but obviously not the only mechanism. Even if the mechanisms are unknown, there is sufficient evidence to take preventive measures against dampness in buildings".

**IEH 2001 review:** This review reported that "although there is consistent evidence of a link between damp and mouldy housing and reports of respiratory symptoms in children, the few epidemiological studies conducted to date show no convincing specific association between exposure to indoor airborne fungi and respiratory disease" [Institute for Environment and Health 2001].

**Systematic review of housing interventions (2001):** This review identified 18 interventions studies [Thomson et al 2001]. Some of these studies found health benefits in terms of self-reported physical and mental health (n=3 studies), improvements in respiratory symptoms and other symptoms (n=4), reduced days lost from school because of asthma (n=1), and other improved health outcomes (n=2). However, the quality of the studies was "generally poor" and this review did not detail any specific data around reductions in mould.

**Kolstad et al review (2002):** This was a high quality review that carefully considered the quality of the identified studies [Kolstad et al 2002]. The key findings were as follows:

- "The analytical studies reviewed provide strong evidence that signs of mold growth or dampness in nonindustrial workplaces or dwellings are associated with asthma as diagnosed by a physician or on the basis of relevant symptoms and with nose, eye, and skin symptoms; fatigue; or headache. However, no consistent associations were found for objective measures of bronchial hyperresponsiveness or nasal mucosal parameters, but the studies were few."
- "In children, asthma also has been related to signs of mold growth or dampness."
- "Mycotoxins have been detected in buildings in which persons have complained about a sick building. However, reported health effects from this exposure have not been disentangled from other possible causes in the indoor environment, and none of the systematic studies reviewed specifically examined mycotoxins."
- "Overall, we conclude that the weight of evidence indicates that exposure to exceptionally high mold levels may cause allergic alveolitis and inhalation fever, and this finding may also pertain to buildings severely damaged by extensive and prolonged mold growth. However, the studies we reviewed provide no evidence that increasing levels of viable mold exposure in nonindustrial work environments or dwellings are related to an increasing occurrence of asthma or to nose, eye, and skin symptoms; fatigue; or headache in the adult population, which may be due to inappropriate or insufficient measures of mold exposure."

**ACOEM 2003 review:** This review was published as a position statement of the American College of Occupational and Environmental Medicine [Hardin et al 2003]. Its findings are not discussed in detail here since there is overlap with the subsequent AAAAI review published in 2006 (see below). Indeed both reviews share an author. Of note is that this review has been criticised as having two authors who are “principals of a defense litigation support corporation” and the other providing expert testimony for the defense in mold litigation [Kilburn et al 2006]. This preceding reference also criticises the methodology of the ACOEM review.

**Review by Fung and Hughson (2003):** This review examined case control studies, cross-sectional surveys, and case reports on fungal exposure and health outcomes [Fung & Hughson 2003]. It concluded that:

“Health effects caused by fungal bioaerosol exposure include allergy, infection, irritation, and toxicity. While the first three categories have well-established mechanisms, there is a lack of dose-response data, and a highly variable degree of individual susceptibility. Specific human toxicity due to inhaled mycotoxins is not well understood, and the likelihood that sufficient mycotoxins are airborne despite visible indoor mold remains unproven and controversial. Excessive moisture is a risk factor for mold proliferation.”

**Asthma and mould/dampness – Douwes & Pearce (2003):** This commentary article provided a review of the literature on indoor mould exposure and asthma [Douwes & Pearce 2003]. The authors concluded that: “In summary, there is consistent evidence that dampness exacerbates preexisting respiratory conditions such as asthma, but it is not clear whether it also causes these conditions.” They also noted various limitations in the evidence:

“Mold exposure has been suggested to play a role, but current knowledge regarding indoor mold exposure and asthma is still very limited because of 1) the fact that specific causal mold components have not yet conclusively been identified, 2) the lack of valid quantitative exposure assessment methods for molds, and 3) the relative lack of prospective cohort studies studying the role of molds using valid exposure assessment methods.”

“Traditionally applied culture methods provide important qualitative information but are only semiquantitative at best. The development of better exposure assessment methods that can practicably be used in epidemiologic studies is therefore a priority.”

The authors also speculate on the potential role of the “hygiene hypothesis” with regard to mould: “It would also be interesting to study whether mold exposure early in life might protect against atopy and asthma, as has been suggested for exposure to bacterial endotoxin.” The evidence for the “hygiene hypothesis” has recently been reviewed [Wong et al 2006].

**Bornehag et al 2004 review:** This review updated a former review (Bornehag et al published in 2001 – detailed above). It concluded that the evidence for a causal association between “dampness” in houses and adverse health effects was “good” (eg, for cough, wheeze and asthma) [Bornehag et al 2004]. But the role of mould/fungi exposure in this relationship was not well clarified at this time relative to other factors associated with dampness such as house dust mites, bacterial endotoxins, and organic chemicals from material degradation processes. Nevertheless, in four of the studies identified in the Bornehag et al review, there were associations between symptoms and exposures to fungal extra-cellular polysaccharides in dust, mould spore levels and glucans. In contrast, there was just one study (actually a New Zealand one) that found that reported mould was negatively associated with current asthma at a significant level, although in the same study there was a 13-fold increased risk of asthma for mould sensitised children [Wickens et al 1999].

**WHO 2004 review:** A review of the evidence on housing and health was undertaken for a WHO Conference in 2004 [Bonney et al 2004]. Key findings were as follows:

“There is increasing evidence that mould growth indoors in damp buildings is an important risk factor for respiratory illness. Mould-related symptoms are likely the result of irritation, allergy or infection.”

“Though in most cases a dose-response relationship could not be derived between the measured concentration of fungi and the registered health problems (Moriske et al, 2003), irritations of the throat and eyes, allergies (most frequently allergic rhinitis), lower respiratory symptoms (dry or productive cough, wheeze) and asthma, as well as increased incidence of respiratory infections have been repeatedly observed. Some studies show a relation between dampness or mould and objective measures of lung function. Apart from respiratory symptoms, depression and the presence of general symptoms like fatigue, headache, dizziness and difficulties in concentration were also reported (Rylander and Etzel, 1999, Moriske et al, 2003).”

The first findings of the WHO pan-European LARES study were also summarised (ie, based on 8 empirical studies in European countries that involved 8519 individual residents in 3373 households). The parts relating to dampness and mould are underlined (for the purposes of this Report):

**Mental health:** “The data gathered from the survey show that people are significantly more depressed and more anxious when they live in a dwelling that: does not offer sufficient protection against external aggressions: noise, vibrations, dampness, moulds, draughts, cold in winter.”

“Besides the allergic and respiratory symptoms, in accordance with the literature data, *fatigue, headache, chronic anxiety and depression* were also significantly associated with mouldy homes.”

**“Asthma and allergies:** As suggested by the literature, available data show that dampness and visible mould growth as assessed by a technician were significantly related to: asthma (ever, attacks in the past year) even when it was diagnosed by a doctor; nasal allergies; eczema.”

**Other respiratory conditions:** The report also described a significantly increased risk for “chronic bronchitis diagnosed by a doctor among people living in *damp* or mouldy houses”. Upper respiratory tract illness during the last year was also significantly associated with mould growth in the home.

**Other conditions:** “The odds ratios of *cerebral stroke, heart attack and hypertension* adjusted to age, sex, socioeconomic status, city, smoking and marital status indicated significantly increased risk associated with mouldy homes but these results require further confirmation because other studies did not mention (if investigated at all) such effects. A possible link may be explained by the common link with depression.”

Unfortunately more detailed housing-related results from the LARES-survey have yet to be published in the peer-reviewed literature (based on the 3 June Medline search for this Report).

#### ***Institute of Medicine review 2004***

The most rigorous review performed to date on dampness and mould and human health appears to be that of the Institute of Medicine (IOM) in the United States [Institute of Medicine 2004]. This was published in 2004 and is the review which appears to be the most detailed identified for this Report (at over 340 pages long). It also appears to have used the most rigorous system for grading the scientific evidence. The findings are summarised in the table below.

This review looked at such general exposures as “damp indoor environments” and “presence of mold or other agents in damp indoor environments”. This was probably appropriate given the state-of-the-science regarding the multiple specific potential chemical and biological hazards involved (eg, mould, dust mites, bacterial endotoxins, chemicals associated with material decomposition etc). Nevertheless, this is also a limitation when specifically considering what the specific role of mould exposure may be in particular health effects.

Table 2: Summary of IOM findings regarding the association between health outcomes and exposure to damp indoor environments or the presence of mould (shared categorisations are in italics)

<b>Level of evidence</b>	<b>Exposure to damp indoor environments*</b>	<b>Presence of mould or other agents in damp indoor environments*</b>
Sufficient evidence of a causal relationship	<i>No outcomes met this definition</i>	<i>No outcomes met this definition</i>
Sufficient evidence of an association	<i>Upper respiratory (nasal and throat) tract symptoms</i> <i>Wheeze</i> <i>Cough</i> <i>Asthma symptoms in sensitized asthmatic persons</i>	<i>Upper respiratory (nasal and throat) tract symptoms</i> <i>Wheeze</i> <i>Asthma symptoms in sensitized asthmatic persons</i> <i>Cough</i> <i>Hypersensitivity pneumonitis in susceptible persons**</i>
Limited or suggestive evidence of an association	<i>Dyspnoea (shortness of breath) Asthma development</i> <i>Lower respiratory illness in otherwise-healthy children</i>	<i>Lower respiratory illness in otherwise-healthy children</i>
Inadequate or insufficient evidence to determine whether an association exists	<i>Airflow obstruction (in otherwise-healthy persons)</i> <i>Skin symptoms</i> <i>Mucous membrane irritation syndrome</i> <i>Gastrointestinal tract problems</i> <i>Chronic obstructive pulmonary disease</i> <i>Fatigue</i> <i>Inhalation fevers (non-occupational exposures)</i> <i>Neuropsychiatric symptoms</i> <i>Lower respiratory illness in otherwise-healthy adults</i> <i>Cancer</i> <i>Acute idiopathic pulmonary haemorrhage in infants***</i> <i>Reproductive effects</i> <i>Rheumatologic and other immune diseases</i>	<i>Dyspnoea (shortness of breath)</i> <i>Skin symptoms</i> <i>Airflow obstruction (in otherwise-healthy persons)</i> <i>Asthma development</i> <i>Mucous membrane irritation syndrome</i> <i>Gastrointestinal tract problems</i> <i>Chronic obstructive pulmonary disease</i> <i>Fatigue</i> <i>Inhalation fevers (non-occupational exposures)</i> <i>Neuropsychiatric symptoms</i> <i>Lower respiratory illness in otherwise-healthy adults</i> <i>Cancer</i> <i>Rheumatologic and other immune diseases</i> <i>Reproductive effects</i> <i>Acute idiopathic pulmonary haemorrhage in infants***</i>

\* These conclusions are not applicable to immuno-compromised persons, who are at increased risk for fungal colonisation or opportunistic infections.

\*\* For mold or bacteria in damp indoor environments. Hypersensitivity pneumonitis is a relatively rare immune-mediated condition in susceptible persons.

\*\*\* This particular condition has had substantive media coverage in the US with regard to cases in which an alleged association was reported for exposure to indoor *Stachybotrys* (in Cleveland Ohio, USA).

### ***Recent reviews (post the IOM review)***

***European review on indoor air and health (2004):*** This 97 page review was part of a “Towards Healthy Air in Dwellings in Europe” (THADE) Project [Franchi et al 2004; Franchi et al 2006]. It found that “mould” was one of the major health determinants in dwellings (along with tobacco smoke, dust mites, pet allergens, cockroaches, pollen, and various chemical exposures). In particular, it concluded that “exacerbation of asthma and allergic illness is strongly linked to exposure to allergens such as ...fungi and mould...”. The journal article summarising this review cited a recent large Italian study [Simoni et al 2005], to suggest that the avoidance of mould/dampness exposure at home would: “abate wheeze by 6%, asthma or cough/phlegm by 7%, rhinoconjunctivitis by 4% in children, asthma by 6% and wheeze by 4% in adolescents”. Other statements of note (from full report [Franchi et al 2004]) included:

- “Inhalation of high concentrations of mould spores containing these toxins [ie, mycotoxins] may deleteriously affect respiratory health. Mycotoxin-induced effects on the immune system could reduce resistance to other micro-organisms perhaps resulting in chronic health problems.”
- “Lastly, fungi produce a complex mixture of volatiles that are frequently evident as ‘mouldy smells’. The volatiles are mixtures of alcohols, esters, aldehyde, various hydrocarbons and aromatics, which can cause nausea and make some subjects quite ill.”

***Review on glucans (2005):*** This review was published in 2005 by a New Zealand-based researcher who specifically examined the role of (1→3)- $\beta$ -D-glucan in the development of respiratory symptoms associated with indoor fungal exposure [Douwes 2005]. This reviewer concluded that the “observational and experimental studies reviewed suggested some association between (1→3)- $\beta$ -D-glucan exposure, airway inflammation and symptoms, however, results were mixed and specific symptoms and potential underlying inflammatory mechanisms associated with exposure could not be identified”. The results were particularly mixed for the occupational studies, the human challenge studies and the animal studies. However, all of the seven epidemiological studies relating to glucans in the indoor environment showed evidence of being associated with adverse health effects (with six of these relating to adverse symptoms or adverse lung function).

***Review on asthma and the indoor environment (2005):*** This review included a section on indoor mould [Richardson et al 2005]. However, it did not advance on reviewing new literature since the IOM report (eg, it cited no publications for 2005 and only two for 2004).

**The AAAAI review (2006) on mould:** A review published in February 2006 covered the medical effects of mould exposure as an *American Academy of Allergy, Asthma and Immunology* (AAAAI) “position paper” [Bush et al 2006]. The conclusions of this review are summarised in the table below. This review triggered substantial correspondence in the journal in which it was published. This correspondence:

- Noted that two of the five authors of this review “earn a substantial income testifying against patients in mold-related litigation” [Marinkovich 2006]. Others suspected that the position paper might “represent an agenda for the defense” [Lieberman et al 2006]. The lack of transparency, lack of rigour and lack of disclosure were criticised by others [Kilburn et al 2006; Shoemaker et al 2006].
- Criticised the review statements concerning mycotoxins and argued for *Stachybotrys chartarum* posing a plausible risk to health [Straus & Wilson 2006; Strickland 2006]. It was also noted that mycotoxins from this mold have been detected in blood and urine of exposed persons [Goldstein 2006].
- Criticised the review statement concerning the condition “chronic rhinosinusitis” and argued for a role played by mould exposure in this inflammation [Ponikau & Sherris 2006].
- Criticised the review on other methodological grounds eg, [Kilburn et al 2006].

Of note, however was that many of these comments came from those who also had declared competing interests as expert witnesses in mould litigation cases in the United States. In response to these comments, two of the original authors stated that their approach to the issue of mycotoxins may be “overly conservative” [Wood & Bush 2006].

Table 3: Summary of relevant conclusions from the review by Bush et al (2006) for an AAAAI Position Paper on mould and health\*

Health outcomes	Conclusions
Allergy and asthma	<p><b>Evidence for a relationship:</b></p> <ul style="list-style-type: none"> <li>• “Atopic patients (those with allergic asthma, allergic rhinitis, and atopic dermatitis) commonly have IgE antibodies to molds as part of polysensitization.</li> <li>• Allergic responses to inhaled mold antigens are a recognized factor in lower airway disease (ie, asthma).”</li> </ul> <p><b>Inconclusive evidence or evidence for no relationship:</b></p> <ul style="list-style-type: none"> <li>• “Currently available studies do not conclusively prove that exposure to outdoor airborne molds plays a role in allergic rhinitis, and studies on the contribution of indoor molds to upper airway allergy are even less compelling.</li> <li>• Exposure to airborne molds is not recognized as a contributing factor in atopic dermatitis.</li> <li>• Exposure to airborne molds is not recognized as a cause of urticaria, angioedema, or anaphylaxis.”</li> </ul>
Allergic broncho-pulmonary aspergillosis (ABPA)*	<p><b>Evidence for a relationship:</b></p> <ul style="list-style-type: none"> <li>• “ABPA and allergic fungal sinusitis are manifestations of significant hypersensitivity to fungi, particularly <i>Aspergillus</i> species.”*</li> </ul> <p><b>Inconclusive evidence:</b></p> <ul style="list-style-type: none"> <li>• “Data supporting the role of fungi in CRS [chronic rhinosinusitis] are lacking at this time.”</li> </ul> <p>* NB: ABPA is a well-recognised clinical entity affecting individuals with asthma or cystic fibrosis. Exposure to <i>A fumigatus</i> can occur from indoor, as well as outdoor, sources.</p>
Toxicity caused by inhalation	<p><b>Inconclusive evidence:</b></p> <ul style="list-style-type: none"> <li>• “The occurrence of mold-related toxicity (mycotoxicosis) from exposure to inhaled mycotoxins in nonoccupational settings is not supported by the current data, and its occurrence is improbable.”</li> </ul>
Irritant effects of mould exposure (from volatile organic compounds & particulates (eg spores))	<p><b>Inconclusive evidence:</b></p> <ul style="list-style-type: none"> <li>• “The occurrence of mold-related irritant reactions from exposure to fungal irritants in nonoccupational settings are theoretically possible, although unlikely to occur in the general population given exposure and dose considerations.</li> <li>• Such irritant effects would produce transient symptoms-signs related to the mucus membranes of the eyes and upper and lower respiratory tracts but would not be expected to manifest in other organs or in a systemic fashion.</li> <li>• Further information about thresholds for irritant reactions in at-risk populations is needed to better define the role of molds, mold products, and other potential irritants in such individuals.”</li> </ul>
Immune dysfunction	<ul style="list-style-type: none"> <li>• “Exposure to molds and their products does not induce a state of immune dysregulation (eg, immunodeficiency or autoimmunity).”</li> </ul>

Health outcomes	Conclusions
<b><i>Outcomes with low or no relevance to indoor mould exposure</i></b>	
Hyper-sensitivity pneumonitis (HP)	<p><b><i>Evidence for a relationship:</i></b></p> <ul style="list-style-type: none"> <li>• “HP is an uncommon but important disease that can occur as a result of mold exposure, particularly in occupational settings with high levels of exposure.”</li> </ul> <p>* NB: Hypersensitivity pneumonitis (also known as extrinsic allergic alveolitis), is a disease that exists in acute, subacute, and chronic forms (albeit with considerable overlap). This disease is mainly associated with occupational settings or unusual circumstances (eg, raising pigeons as a hobby).</p>
Infection	<p><b><i>Evidence for a relationship:</i></b></p> <ul style="list-style-type: none"> <li>• “Common superficial fungal infections are determined by local changes in the skin barrier, resident microflora, or both.</li> <li>• A very limited number of aggressive fungal pathogens can be acquired through specific outdoor exposures.</li> <li>• Host factors, rather than environmental exposure, are the main determinant of opportunistic fungal infection.”</li> </ul>

\* This summary excludes the conclusions for toxic effects of mould ingestion.

***Review of fungi and severe asthma (2006):*** This review examined the association between fungi and severe asthma [Denning et al 2006]. It concluded that “there is current evidence to demonstrate a close association between fungal sensitisation and asthma severity. Whether such an association is causal remains to be confirmed...”

***Meta-analysis of dampness/mould and health (2007):*** This review was the first to systematically undertake a meta-analysis to quantify the health risk associated with mould exposure [Fisk et al 2007; Mudarri & Fisk 2007]. From 33 peer-reviewed United States studies, the authors estimated that exposure to dampness and mould “raises the risk for various adverse respiratory outcomes by 30–50%” (see the Table below). These data were then used to estimate the proportion of US current asthma cases that are attributable to dampness and mould exposure at 21% (95% confidence interval 12–29%).

Table 4: Summary health risks for dampness and mould in United States houses [Fisk et al 2007; Mudarri & Fisk 2007].

Outcome	Number of studies	Odds ratio (95% CI)
Upper respiratory tract symptoms	13	1.70 (1.44 – 2.00)
Cough	18	1.67 (1.49 – 1.86)
Wheeze	22	1.50 (1.38 – 1.64)
Current asthma	10	1.56 (1.30 – 1.86)
Ever diagnosed asthma	8	1.37 (1.23 – 1.53)
Asthma development	4	1.34 (0.86 – 2.10)

This review [Mudarri & Fisk 2007], also encompassed the topic of dampness/mould in schools and offices. It included 14 studies of schools and eight studies of offices and institutional buildings. The study designs for offices included cross-sectional studies, two case-control studies, and a blinded crossover intervention study. All of these studies of offices found one or more statistically significant association between dampness or mould and adverse respiratory or other health effects. In summarising this review the authors stated:

“The evidence supporting associations of dampness or mold in offices and institutional buildings with respiratory or other health effects of occupants is reasonably robust.” .... “Overall, there is good reason to believe that the results found in offices and institutional buildings reflect an underlying causal relationship between dampness and mold exposures and the reported health outcomes.

The school studies were methodologically weaker as they usually included only a small number of buildings and often had only a small number of subjects. Yet “despite these weaknesses, the overall results indicate that adverse health outcomes are likely to be elevated among occupants of damp and moldy schools. Many of the studies found that damp or moldy schools, or molds and bacteria in floor dust were significant risk factors for a variety of health outcomes.” Of note however was that one of these school studies reported an *inverse* finding of *improved* health with dampness or mould. Also of interest was that the school studies that controlled for numerous potential confounders still found statistically significant health risks.

With regard to the school studies the authors concluded that:

“taken in isolation, the schools literature is non-conclusive. However, the consistency of findings from these school-based studies with the findings from homes, offices, and other buildings strengthens the case for adverse health effects in damp and moldy schools.”

These findings for offices and schools are not entirely applicable to the situation with dampness and mould in domestic dwellings. Nevertheless, they are consistent with the other scientific literature for health effects for dampness and mould in domestic settings.

### ***Evidence from recent housing intervention studies (since 2005)***

***New Zealand trial:*** The best designed and largest housing intervention study (a randomised community trial) that included data on mould was the one undertaken in New Zealand [Howden-Chapman et al 2007]. However, as discussed elsewhere in this Report, the improvements in health that were detected may have been due to other factors and not the reduction in exposure to indoor mould.

***South Wales trial:*** This was a randomised controlled trial that was conducted to determine if asthma improves when indoor mould is removed [Burr et al 2007]. In the intervention group (with 95 participants in 68 intervention houses) indoor mould was removed, fungicide was applied, and a fan was installed in the loft. In the control group (87 participants in 63 control houses), the intervention was delayed for 12 months. At six months, the intervention group showed significant improvements in (i) wheeze affecting activities; (ii) perceived improvement of breathing; and (iii) perceived reduction in medication. By 12 months the intervention group showed significantly greater reductions than the controls in preventer and reliever use, and improved more for rhinitis and rhinoconjunctivitis. The authors concluded that “although there was no objective evidence of benefit, symptoms of asthma and rhinitis improved and medication use declined following removal of indoor mould. It is unlikely that this was entirely a placebo effect.”

***Cleveland Ohio home remediation study:*** Another randomised trial undertook home remediation aimed at moisture sources in homes with children with asthma [Kercsmar et al 2006]. The remediation group had a significant decrease in symptom days ( $p = 0.004$ , intent to treat). “In the post-remediation period, the remediation group had a lower rate of exacerbations compared with control asthmatics (as treated: 1 of 29 vs. 11 of 33, respectively,  $p = 0.003$ ; intent to treat: 28.1% and 10.0%, respectively,  $p = 0.11$ ).” The authors concluded that “Construction remediation aimed at the root cause of moisture sources and combined with a medical/behavioral intervention significantly reduces symptom days and health care use for asthmatic children who live in homes with a documented mold problem.” Nevertheless, as well as significantly reducing “mold scores”, this intervention significantly reduced endotoxin levels and so it is not clear what the benefit was likely to be attributed to. Also the study was fairly small ( $n=62$  children).

***Scottish intervention study:*** A recent intervention study in Scotland reported reduced problems with dampness/condensation and improved self-reported health status [Thomson et al 2007]. The intervention involved replacing ex-council owned housing stock that was reported to have problems of damp and mould, with newly built housing in the same locality. However, the study was

limited as there were only 50 households in the intervention group and 50 in the control group and specific mould-related data were not recorded.

**Northern Ireland intervention study:** A fuel poverty intervention in Northern Ireland involved energy efficiency measures and installing central heating systems [Shortt & Rugkasa 2007]. There were reductions in mould reported and also significant reductions in various health complaints (including mean number of illnesses per head). However, the number of households was small (54 in the intervention group, 46 in the control group).

**German study:** A study of homes with mould included remediation and individuals leaving the affected homes [Kopf et al 2006]. It was reported that absence from the home led to better health in 60% of people (33/55). After removing the mould, 27% reported improved health (10/38). However, there was no control group and the full report has not yet been published in a peer-reviewed journal.

#### 4) SPECIFIC TYPES OF MOULD AND HEALTH RISKS

**Stachybotrys:** In the United States there has been substantive media coverage with regard to cases in which an alleged association was reported for exposure to the indoor mould *Stachybotrys* and the condition “acute idiopathic pulmonary haemorrhage in infants” (particularly for cases in Cleveland Ohio, USA).

The Institute of Medicine (IOM) reviewed this evidence and considered that for both “damp indoor environments” and for “mould or other agents in damp indoor environments” there was “inadequate or insufficient evidence to determine whether an association exists” for the condition “acute idiopathic pulmonary haemorrhage in infants” [Institute of Medicine 2004]. Similarly, it considered that there was inadequate or insufficient evidence for all of the following other non-respiratory conditions: skin symptoms, mucous membrane irritation syndrome, gastrointestinal tract problems, fatigue, inhalation fevers (non-occupational exposures), neuropsychiatric symptoms, cancer, rheumatologic and other immune diseases, and reproductive effects.

Another review concluded similarly to the IOM [Bush et al 2006]. However this review was heavily criticised and some of the critics argued for *Stachybotrys chartarum* posing a plausible risk to health [Straus & Wilson 2006; Strickland 2006]. It was also noted by critics of this review that mycotoxins from this mould have been detected in blood and urine of exposed persons [Goldstein 2006]. In response to various criticisms, two of the original authors of the Bush et al review stated that their approach to the issue of mycotoxins may be “overly conservative” [Wood & Bush 2006].

**Aspergillus:** One of the reviews on mould and health identified in our literature review [Bush et al 2006] stated that there was evidence that *Aspergillus* species were associated with allergic broncho-pulmonary aspergillosis (ABPA). That is: “ABPA and allergic fungal sinusitis are

manifestations of significant hypersensitivity to fungi, particularly *Aspergillus* species.” Of note is that exposure to *A fumigatus* can occur from indoor, as well as outdoor sources. Sensitivity to *A fumigatus* has also been detailed in various New Zealand studies [Burrows et al 1995b] [Burrows et al 1995a] and has been found (in the form of extracellular polysaccharides) in New Zealand homes [Douwes et al 2006a].

***Other mould species that produce mycotoxins:*** Mycotoxins are produced by a wide range of moulds and it is scientifically well established that ingested mycotoxins can cause illness and death in humans and animals [Franchi et al 2004].

One review specifically considered the health effects of mycotoxins in indoor air [Robbins et al 2000]. It covered animal exposure studies, case reports, and epidemiological studies. It concluded that:

“although evidence was found of a relationship between high levels of inhalation exposure or direct contact to mycotoxin-containing molds or mycotoxins, and demonstrable effects in animals and health effects in humans, the current literature does not provide compelling evidence that exposure at levels expected in most mold-contaminated indoor environments is likely to result in measurable health effects. Even though there is general agreement that active mold growth in indoor environments is unsanitary and must be corrected, the point at which mold contamination becomes a threat to health is unknown.”

On the issue of mycotoxins, another high quality review that carefully considered the quality of the identified studies [Robbins et al 2000; Kolstad et al 2002] reported that: “Mycotoxins have been detected in buildings in which persons have complained about a sick building. However, reported health effects from this exposure have not been disentangled from other possible causes in the indoor environment, and none of the systematic studies reviewed specifically examined mycotoxins.”

Another review published in 2003 [Fung & Hughson 2003], concluded that: “Specific human toxicity due to inhaled mycotoxins is not well understood, and the likelihood that sufficient mycotoxins are airborne despite visible indoor mold remains unproven and controversial.”

Another review stated that: “The occurrence of mold-related toxicity (mycotoxicosis) from exposure to inhaled mycotoxins in nonoccupational settings is not supported by the current data, and its occurrence is improbable” [Bush et al 2006].

### ***Summary of specific types of mould and health risks***

There are many limitations in the quality of the evidence around mould exposure and various health outcomes, nevertheless, there is evidence for particular adverse effects of *Aspergillus* (ie, allergic broncho-pulmonary

aspergillosis), and particular mould species seem to feature more than others in being associated with positive allergy skin test results. However, other adverse effects (eg, from *Stachybotrys*) are not adequately supported by the currently available scientific evidence. Also the evidence that mould species that generate detectable mycotoxins produce additional harm to human health from these mycotoxins is not scientifically established.

These conclusions are based on our examination of recent reviews and it is plausible that individual studies (that have not yet been included in the review literature), may support other conclusions.

## 5) BURDEN OF MOULD IN NEW ZEALAND AND SPECIFIC HEALTH RISKS

New Zealand as a country is noted for its high level of dampness in housing, which probably reflects high outdoor humidity, relatively poor housing stock, inadequate insulation and inadequate heating [Isaacs & Donn 1993; Schluter et al 2000; Isaacs et al 2004]. A national telephone survey found that mould in one or more of rooms was reported for 35% of the houses [Howden-Chapman et al 2005b]. Of the houses where mould was reported, 48% had it in bathrooms and 47% in the master bedroom. Additional results from this national survey are summarised below:

- **Geographic/climatic factors:** The national survey found that mould was reported significantly more often in houses in the northern half of the North Island. The results were also suggestive that higher locality temperature and humidity were associated with an increased risk of mould (but these associations were not at statistically significant levels).
- **Housing condition, size and mould:** It was found that there was significantly increased risk of reported mould in older houses, those in poorer condition, in houses that got little sun in winter and in those with three or more bedrooms. In the multivariate analysis, the factors that continued to be significantly associated with mould were older houses, those in poorer condition and those lacking in sun exposure.
- **Insulation protects against mould:** There was significantly less mould reported in well-insulated houses (results from the multivariate analysis). Of note was that many respondents did not know the insulation status of the house (ie, 24% for insulation in the outside walls, 20% for floors and 13% for ceiling). The lack of insulation and its association with reported mould is likely to be due to the effect of insulation in increasing average indoor temperature. Another reason may be that people in colder houses behave differently in terms of making more extensive use of water-generating unflued gas heaters or restricting ventilation more (eg, opening windows less). Other New Zealand work has found that the use of insulation is associated with lower dust mite allergen levels [Wickens et al 2001]. Similarly, floor

insulation, and building orientation (north facing living rooms) have been associated with lower endotoxin levels in New Zealand homes [Wickens et al 2003]. Endotoxin in house dust has been shown to be a risk factor for wheezing and skin symptoms in New Zealand infants – eg, [Gillespie et al 2006].

- **Resident numbers and mould:** The risk of mould being reported increased with both the total number of residents in the house and the number of those under age 18 years. This finding for total residents persisted in the multivariate analysis and was apparent for each additional person to the house. The number of residents per bedroom (a proxy for house size) was also related to reported mould.
- **Living practices and mould:** Specific practices that were significantly associated with reported mould were a high frequency of having baths, frequent shower use, and frequent clothes washing. The pattern of results for the various methods for drying clothes inside in winter was also suggestive of increased risk (but not at a statistically significant level). It is very plausible that such factors influence mould growth by increasing humidity (eg, as found in other overseas studies of mould [Wan & Li 1999]). Increased humidity may also increase endotoxin levels in New Zealand homes according to another study [Wickens et al 2003]. No specific form of heating showed any clear association with reported mould but there was a significantly increased risk of mould when multiple forms of heating were used (ie, including gas heating, electricity and open/closed fire being used to heat the sitting room in winter).
- **Responses to control dampness and mould:** Use of extractor fans was reported by 47% of respondents in the national survey, followed by 43% using range-hoods, and 23% using a dehumidifier. The risk of reported mould was slightly lower in houses where range-hoods and extractor fans were used (but not at statistically significant levels). In contrast, houses with mould were significantly far more likely to report using dehumidifiers. Of the respondents in houses with mould, 85% reported specific activities designed to control mould. These were: use of commercial cleaners (32%), cleaning/washing (28%), ventilation (15%), use of a dehumidifier (13%), wiping up moisture/ drying (12%), room heating (9%), use of an extractor fan (3%) and use of insulation (1%).
- **Views of respondents on the causes of mould:** Of the respondents in houses with mould, the most common explanation for mould was “dampness” or “moisture” (44%). This was followed by condensation (23%), lack of sun (22%), lack of ventilation (19%), cold (12%), poor insulation (12%), southern orientation of the house (11%), humidity (5%), and leaks (2%). There were 5% of respondents who “didn’t know” and 12% who gave various other reasons.

**Findings from an intervention study:** Another study, that involved 1350 households from seven low-income communities around New Zealand, identified mould as a frequent problem [Howden-Chapman et al 2007]. Based on self reporting, 18% were in “poor” or “very poor” condition, 89% had condensation, and 75% had mould. Building inspectors reported even worse conditions, with 53% of the houses in the 140 random sub-sample being in “poor” or “very poor” condition and 81% with some mould. Furthermore, the inspectors reported “large patches” of mould in 24% of houses.

**A study of glucans/fungi in homes:** A recent New Zealand study has examined dust in 32 homes in Wellington [Douwes et al 2006a]. These homes were ones where there was at least one 6-14-year-old child present. The residents of approximately 20% (n=6) of the studied homes reported the presence of surface mould in the living room and more than 40% (n=14) of the homes had previously been affected by water damage. There were two homes that had damp spots. In particular, the study found that fungal (1→3)- $\beta$ -D-glucans levels were higher in comparison to previous comparable European studies. Nevertheless, the levels of extracellular polysaccharides from the fungi *Aspergillus* and *Penicillium* (EPS-Asp/Pen) were comparable to these previous studies. Glucan (as well as bacterial endotoxin) levels were higher in homes with self-reported water damage (borderline statistically significant;  $p < 0.10$ ). Other findings of note included a positive association ( $p < 0.10$ ) for dust mite and a combination of self-reported mould, dampness and water damage. EPS levels were higher in homes where residents indicated the presence of mould spots on the wall (but not at a statistically significant level).

**Other NZ studies:** The *Auckland Birthweight Collaborative Study* is a case-control study of risk factors for small for gestational age babies [Purvis et al 2005]. It has collected data from parental reports on household environment conditions. For the homes of European children in Auckland a report from this study described 27.3% (150/550) of homes having mould on the ceilings or walls. Similarly, 21.2% (117/552) reported a damp home.

Higher levels were reported in another study that found that 37% of Pacific Island respondents reported that their homes had “dampness/mould problems” [Butler et al 2003]. These data were gathered as part of the Pacific Islands Families (PIF) Study in which 1376 mothers were interviewed when their infants (all born in South Auckland) were six weeks old.

A study in Auckland and Christchurch also collected housing data but did not precisely detail the data on dampness in the published article [Kearns et al 1991]. Nevertheless, it reported the surprising finding that coldness and dampness were more often identified as problems in Auckland than in Christchurch. The authors suggested that this pattern may have been due to the Auckland dwellings having fewer heaters.

A Dunedin survey of 91 student houses and flats was carried out to establish base-line thermal conditions in what was considered to be a “poor housing

sector” [Shannon et al 2003]. A total of 72% of the student tenants reported that they experienced mould or damp in the house.

***Mould in furnishings:*** Furnishings may also be a source of exposure to mould inside New Zealand houses. One preliminary New Zealand study found that synthetic pillows contain about fourfold higher total amounts of  $\beta$ -(1,3)-glucan than feather pillows [Siebers et al 2006]. This is consistent with work elsewhere that suggests that pillows are a substantial source of fungi [Woodcock et al 2006].

***On-going studies:*** Other studies are collecting data on mould in New Zealand housing eg, for the New Zealand Asthma and Allergy Cohort Study [Epton et al 2007]. The mould related results have yet to be published, though reported mould data has been controlled for in a study of endotoxin [Gillespie et al 2006].

***Summary – the burden of mould exposure in New Zealand:*** In summary, the New Zealand data from multiple studies is suggestive that mould in New Zealand homes is relatively common (eg, over a third of homes in a national survey). Amongst low-income communities, Pacific communities and tertiary student accommodation, the prevalence of reported visible mould in housing appears to be higher than the national average. The reported problems with mould in New Zealand houses may be higher than in other countries, and there is further preliminary evidence for this when considering fungal (1 $\rightarrow$ 3)- $\beta$ -D-glucans levels in house dust.

## 6) NEW ZEALAND EVIDENCE ON MOULD AND HEALTH

### ***Descriptive studies***

***Mould associated with respiratory symptoms:*** The earliest work of note that relates to New Zealand and was identified in this review was published in 1981. This study found that reported exposure to damp housing by parents was associated with higher prevalence of lower respiratory tract symptoms among Pacific infants in the Hutt Valley ( $p < 0.01$ ) [Kerr 1981].

Another study relating to the Pacific population found an association between reported damp/mouldy housing and maternal asthma [Butler et al 2003]. In multivariate analyses that controlled for many factors (maternal age, ethnicity, education, marital status, birthplace, number of years lived in New Zealand, and household income) damp/mouldy housing was significantly associated with maternal asthma (adjusted OR = 1.82; 95% CI = 1.18–2.83).

As detailed above in the international review by Bornehag et al, there was included in this review a New Zealand study that found that reported mould was *negatively* associated with current asthma at a significant level [Wickens

et al 1999]. That is, this study found that reported mould was associated with a *lower* occurrence of asthma. This finding was in contrast with most other studies in this review. However, in this same study, mould sensitisation was strongly and significantly associated with asthma (a 13-fold increased risk of asthma for mould sensitised children).

Of the three New Zealand centres in a multi-country study [Zock et al 2002], one centre had a statistically significant association between reported mould exposure and current asthma (ie, Christchurch City, odds ratio (OR) >2). In the Hawkes Bay centre the risk was raised (OR = 1.3) but not to a statistically significant level and in the Wellington centre the risk was not significantly raised at all (OR = 1.0). Other New Zealand data on specific IgE (to the mould *Cladosporium herbarum*) were collected in this survey (for the three centres listed above). However, this specific IgE data was not detailed in the resulting publications (ie, just the total IgE levels were described) [Burney et al 1997; Svanes et al 1999].

Also of note was a New Zealand study which found that a “history of condensation or mould” in the university student accommodation was associated with higher levels of house dust mite allergen [McRae et al 2002]. Reported or observed damp or mould on living room floors or walls also tended to be associated with higher levels of dust mite allergen in living rooms in a Wellington study [Wickens et al 2001]. However, this was only statistically significant for visible damp on living room walls. The issue of dust mites is particularly relevant since these mites are known to consume some fungal species (see the *Methods Section*).

Studies on a birth cohort of New Zealand children (born in Dunedin) have included relevant data on fungal allergies. In this group positive allergy skin tests (including for the mould *Aspergillus* as one of the four skin tests) were associated with bronchial hyperresponsiveness [Burrows et al 1995b]. There were statistically significant relationships for each of the moulds *Aspergillus fumigatus*, *Alternaria*, *Cladosporium* but not for *Penicillium* [Burrows et al 1995a]. However, in the multivariate analysis only the results for *Aspergillus fumigatus* were statistically significant (along with those for dust mite allergen, cat allergen and dog allergen). At age 13 years, 6% of the cohort were reported to have positive skin tests to the mould *Alternaria* and this rose to 13% at age 21 years [Sears et al 2002].

**Asthma severity:** Earlier New Zealand work reported that for a third of asthmatic patients in a small sample (n=35) there was a relationship between asthma symptoms (or peak expiratory flow rate) and the presence of particular fungal spores in the home environment [Holst et al 1983]. Other work has also suggested a possible role of basidiospore exposure in sensitisation and asthma hospitalisations [Hasnain et al 1985a; Hasnain et al 1985b]. However, the latter was just a based on similar seasonal patterns of exposure and hospitalisations.

When considering the Dunedin cohort study members admitted to hospital for asthma and wheeze, it was found that atopy at age 13 years and 21 years

were statistically significant risk factors (along with total number of positive allergy tests at these two years). However the various moulds (*Aspergillus fumigatus*, *Alternaria*, *Cladosporium* and *Penicillium*) were only four out of the 11 allergens tested (and comprising the composite measures for atopy) [Rasmussen et al 2002].

Also of note is subsequent work that found that only some age groups in New Zealand have peak asthma hospitalisation rates in autumn months when fungal spore levels tend to be highest (eg, those aged 5-14 years and 15-44 years) with the rest having peak rates in winter months [Kimbell-Dunn et al 2000].

A prospective study investigated a population of mild to moderate asthmatic subjects in Blenheim with regard to weather and airborne fungal spore and pollen counts [Epton et al 1997]. It found a weak association between days of high basidiospore counts and increased nocturnal waking and reliever medication use. Nevertheless, it concluded that the impact of aeroallergens on asthma symptoms in this population are relatively small. However, a subsequent Auckland study found that 54% of patients admitted to the intensive care unit (ICU) with a severe attack of asthma had a positive skin test for one or more fungal allergens compared with 30% of patients in each of the other comparison groups ( $p = 0.005$ ) [Black et al 2000]. In contrast, these ICU patients were no more likely to have positive skin tests for the grass mix, cat dander, or house-dust mite than the other patients. These authors concluded that a positive skin test for fungal allergens is a risk factor for admission to an ICU with an acute attack of asthma.

There are also New Zealand data as part of a large international study into mould sensitisation and asthma severity by Zureik et al [Zureik et al 2002]. The combined New Zealand and Australian data were consistent with the overall pattern for a strong association of severe (versus mild) asthma with sensitisation to moulds (either *Alternaria alternata* or *Cladosporium herbarum*, or both). However, the odds ratio for just the New Zealand and Australia data combined was not at a statistically significant level (OR = 1.45, 95%CI = 0.69 to 3.05).

**Mental health:** Only one New Zealand study has examined a mental health outcome with the presence of dampness/mould in housing (the above mentioned Pacific Islands Families (PIF) Study [Butler et al 2003]). In multivariate analyses that controlled for many factors (maternal age, ethnicity, education, marital status, birthplace, number of years lived in New Zealand, and household income) damp/mouldy housing was significantly associated with probable maternal depression ( $p < 0.05$ ; adjusted OR = 1.40; 95% CI = 1.02–1.91).

**Atopic dermatitis:** A study of infants in Auckland found that atopic dermatitis at 3.5 years of age was not associated with damp or mould on the ceilings or walls [Purvis et al 2005]. In this study atopic dermatitis was largely related to a personal and a parental history of atopic disease as well as the duration of breastfeeding.

### ***Evidence from intervention studies in New Zealand***

One of the world's largest housing intervention studies has recently been conducted in the New Zealand setting [Howden-Chapman et al 2005a; Howden-Chapman et al 2007]. This study aimed to determine whether insulating existing houses increases indoor temperatures and improves occupants' health and wellbeing. It used a community-based, cluster, single-blinded randomised design and involved seven low-income communities (n= 1350 households containing 4407 participants). The study found that after insulation the odds of insulated households reporting mould decreased significantly (OR=0.24, 0.18 to 0.32; p<0.0001). Similarly there were significant declines in reports of condensation and non-condensation dampness (Table 5 below). In terms of health outcomes, insulating existing houses resulted in significant improvements (ie, in terms of self-rated health, self-reported wheezing, days off school and work, and visits to general practitioners as well as a trend for fewer hospital admissions for respiratory conditions). Nevertheless, the role (if any) of reduced mould levels in these health outcomes remains unclear given that other factors were involved (eg, particularly the significantly warmer and drier indoor environment in the insulated houses). Further analysis of aspects of this study are still underway (eg, changes in reported "musty smell" and mould mass and speciation).

Table 5: Selected results for a New Zealand-based trial of insulating houses [Howden-Chapman et al 2007] (Values are number/total number of households for self reported house condition)

<b>Outcome measure</b>	<b>Before intervention</b>		<b>After intervention</b>		<b>Odds ratio (95% CI)</b>	
	<b>Intervention group</b>	<b>Control group</b>	<b>Intervention group</b>	<b>Control group</b>	<b>Un-adjusted</b>	<b>Adjusted</b>
Any mould	71.5% (364/509)	72.3% (362/501)	37.5% (191/509)	68.5% (343/501)	0.28 (0.21 to 0.36) p<0.0001	0.24 (0.18 to 0.32) p<0.0001
Condensation	90.5% (487/538)	90.8% (486/535)	59.1% (318/538)	89.7% (480/535)	0.17 (0.12 to 0.23) p<0.0001	0.16 (0.11 to 0.22) p<0.0001
Non-condensation dampness	64.4% (334/519)	62.8% (324/516)	30.1% (156/519)	65.7% (339/516)	0.23 (0.18 to 0.30) p<0.001	0.18 (0.13 to 0.24) p<0.0001

## **Summary of the NZ evidence**

**Respiratory symptoms:** A total of six published studies relating to respiratory symptoms and mould exposure/sensitisation in New Zealand were identified. Of these two found a statistically significant positive association with adverse health impacts [Kerr 1981; Butler et al 2003]. Another found this result for the population sampled from Christchurch City, but not for the populations studied in the Hawkes Bay or Wellington [Zock et al 2002]. Two publications of the same study population reported positive relationships between mould sensitisation and bronchial hyperresponsiveness [Burrows et al 1995a; Burrows et al 1995b]. Furthermore, two New Zealand studies found a positive association between mould and higher levels of dust mite allergen in student accommodation and homes [Wickens et al 2001; McRae et al 2002]. In contrast to the above, one study found that reported mould exposure was *negatively* associated with current asthma at a significant level [Wickens et al 1999].

**Asthma severity:** Of recent studies of asthma severity since 1990, there is evidence from five studies for an association between mould exposure/mould sensitisation and asthma severity. Dunedin cohort study data provides some evidence and combined data for New Zealand and Australia indicates an association between severe (versus mild) asthma with sensitisation to moulds [Zureik et al 2002]. Similarly, an Auckland study found that a positive skin test for fungal allergens is a significant risk factor for admission to an intensive care unit with an acute attack of asthma [Black et al 2000]. A prospective study in Blenheim [Epton et al 1997] found a weak association between days of high basidiospore counts and increased nocturnal waking and reliever medication use among people with asthma. However, it may be that only some age groups are affected (eg, those aged 5-14 years and 15-44 years) given the pattern of peak asthma hospitalisation rates in autumn months when fungal spore levels tend to be highest [Kimbell-Dunn et al 2000].

**Other health outcomes:** One study reported an association between dampness/mould in housing and maternal depression [Butler et al 2003], while another found no association with atopic dermatitis [Purvis et al 2005].

**Intervention study:** The findings from a large intervention study indicated that insulating households resulted in reductions of reported mould and significant improvements in health outcomes (ie, in terms of self-rated health, self-reported wheezing, days off school and work, and visits to general practitioners as well as a trend for fewer hospital admissions for respiratory conditions) [Howden-Chapman et al 2007]. The specific role of reduced mould levels in these health outcomes is plausible however it remains unclear given that other factors were involved (eg, particularly the significantly warmer and drier indoor environment in the insulated houses).

**Overall summary:** The New Zealand data tends to be consistent with the international literature that indicates mould in housing is associated with various adverse health outcomes. But despite the overall pattern there are

some studies that show no association and even one that found a significant negative association (for current asthma). It is also plausible that the association between mould and respiratory outcomes is not necessarily causal but is mediated by other factors such as exposure to house dust mite allergen or bacterial endotoxin.

## 7) DISCUSSION OF THE LIMITATIONS AROUND THE EVIDENCE

**Limitations of this review:** The multi-disciplinary nature of the housing and health field means that some studies will not be indexed in Medline or picked up by the reviews identified in the literature searches undertaken for this Report. A more thorough review could have used a wider range of search engines and a wider search period. Nevertheless, it seems likely that all of the particularly key New Zealand studies and the most substantive recent reviews have been identified in with the search strategy detailed in the *Methods Section*. Future reviews could undertake more extensive searches for recent single studies which have the highest quality (ie, controlled trials involving mould remediation and longitudinal studies that accurately assess mould exposure and health outcomes).

**Limitations with study design:** Most of the studies reported in the reviews in this Report were cross-sectional. There is a shortage of both intervention studies (with appropriate control groups), and other analytic studies such as case-control studies and longitudinal studies (eg, to assess if health outcomes arise after mould exposure). Nevertheless, the end of Section 3 does describe some recent intervention studies and some of the reviews have actually included longitudinal studies, case control studies and even one case crossover study. Some recent high quality analytical studies have yet to be included in reviews eg, [Pekkanen et al 2007]. This case-control study showed that only moisture damage in the main living areas or in the child's bedroom was associated with asthma. Moreover, the toxicity of different microbes is modified by differences in the materials upon which they live and in their growth conditions indoors, so that apart from the difficult of knowing which microbes to measure, their growth in different building materials is also needed.

**Limitations with measurement of relevant exposures in studies:** As detailed in the *Methods Section* there are various potential limitations with assessing exposure data based on self-report (including understanding of what mould is and also social desirability bias). Also most of the commonly used methods of detecting mould in household dust have limitations [Kolstad et al 2002; Douwes & Pearce 2003; Institute of Medicine 2004].

Other limitations around exposure measurement that need to be considered include:

- The complex inter-relationships between mould and dust mites (as discussed in the Section on New Zealand evidence) still need further

clarification. Dust mites and their associated allergens may be an important confounding factor in the relationship between mould and adverse health outcomes.

- There are associations between mould and bacterial endotoxins [Institute of Medicine 2004] but these need further clarification. As with dust mites, endotoxins may be a confounding factor in the relationship between mould and particular adverse health outcomes (eg, asthma exacerbation). An additional complexity is the general weight of evidence that suggests that endotoxin exposures may actually *protect* against the *development* of atopy and asthma [Wong et al 2006]. Indeed, there is one recent Dutch study that provides support for this hypothesis for exposure to mould (ie, extracellular polysaccharides from the genera *Penicillium* and *Aspergillus* and doctor-diagnosed asthma and persistent wheeze) [Douwes et al 2006b].
- Studies on mould in housing and health outcomes may not adequately consider other potential biological confounders associated with respiratory symptoms (see Table 1). In particular these include the level of exposure to insect allergens (eg, cockroaches) and to animal dander (eg, pets and rodents).
- Many studies on mould focus on visible mould on walls and ceilings. However, furnishings such as pillows may be an important source of exposure [Siebers et al 2006; Woodcock et al 2006]. Also, organic waste storage in houses appears to increase both dust endotoxin and fungal extracellular polysaccharides [Herr et al 2004].
- Studies on mould in housing and health outcomes may not adequately consider other potential toxicological exposures. These include the level of indoor smoking in low-income households, use of forms of heating that produce toxic emissions (eg, unflued gas heaters [Brown et al 2004]), the use of potentially toxic cleaning agents by people attempting mould remediation, the release of toxic compounds from building materials associated with mould-induced building damage, and exposure to other compounds associated with respiratory symptoms (see Table 1). Cigarette smoke has been found to contain high levels of endotoxins [Larsson et al 2004] and tobacco contains large amounts of other bacterial and fungal compounds [Szponar et al 2006].

**Limitations with reviews:** The United States scene appears to be highly litigious with regard to mould and health issues. This may be impacting on what is, or is not, included in the scientific literature (see the debate around the position statements of the AAAAI above). Furthermore, none of the reviews identified in this Report has attempted to assess publication bias.

Even rigorous systematic reviews can have methodological problems. For example, major problems were identified in 29% of Cochrane systematic reviews published in 1998 [Olsen et al 2001]. Also a flawed Cochrane systematic review had to be retracted after being published in 2004 [Rada 2005]. The methodology of certain Cochrane systematic reviews is also debated (eg, [Walsh 2007]).

**Deficits in basic biological knowledge:** A substantial proportion of >1 million fungal species thought to exist remain even to be speciated or subjected to antigenic scrutiny. It is quite conceivable that unknown but particularly toxic or allergenic fungal species may be present in household settings.

To address the above listed problems, there is a need for further research. Possible research-based responses are detailed in the following section.

## 8) RESEARCH NEEDS TO CLARIFY THE HEALTH ISSUES

Given the list of limitations in the previous section there is a need for the relevant authorities in New Zealand to consider additional research. The following options should be considered, none of which are mutually exclusive.

1. Regularly **updating literature reviews** with regard to dampness, mould and health. The literature from European scientists may have some advantages over that from North American – given the possible distortions associated with the high level of legal activity associated with mould and health issues in the United States. At least one large literature review has yet to be published in full ie, [Lovik et al 2006].
2. Undertaking a **descriptive study of the health conditions and concerns** of 75 people living in “leaky houses” and 25 people living in structurally sound houses in New Zealand, in order to identify the range of health effects that people attribute to these “leaky buildings”. This medical assessment could be important for two reasons: (i) it would show the complainants that their concerns were being taken seriously; and (ii) it would help to identify any health effects and concerns that have not been identified in the literature. For example, what we usually think of as “at risk” should extend beyond physical health. Psychological well-being, family health and communities are all exposed when some home owners perceive their homes as “polluted”.
3. Undertaking a **case-control study** that includes people living in “leaky houses” and matched community controls who are living in non-leaky housing. This would be important because it would help to identify which health outcomes reported by people could legitimately be associated with “leaky buildings”. There would be a need in such a study to accurately assess levels of exposure to mould and damp, and collect other relevant socio-demographic and exposure data (eg, on smoking, use of unflued gas heaters etc).
4. Undertaking carefully controlled **intervention studies** in New Zealand that address the study design and measurement issues that have been raised (see the above discussion). Some New Zealand work is progressing but ideally more should be done considering the relatively

poor quality of housing stock in this country and the high humidity environment. Such intervention studies could be specifically related to remediating “leaky buildings” and seeing whether this has an impact on the occupants’ health (ie, it could include before-and-after health assessments and ideally use control groups as well).

5. A study of the **effectiveness of remediation strategies** for restoring a leaky home, such as remediation of the leaky envelope ie replacing deteriorated materials and making the home weathertight, compared to remediation of the structure using practices to contain any disturber fungal material and HEPA vacuuming and thorough cleaning of the home to remove fungal material.

## 9) RISK PERCEPTION AROUND MOULD IN HOUSES

In New Zealand, it was found in a national mould survey that a majority of respondents with mould in their houses took measures to control mould (eg, use of chemical cleaners) [Howden-Chapman et al 2005b]. This suggests that the presence of mould is a cause of concern for many New Zealanders. Also, the finding that houses with mould were significantly more likely to report using dehumidifiers may suggest that these are specifically being used to address perceived problems with mould. Similarly, the association of reported mould with multiple different forms of heating might reflect attempts by residents to deal with mould.

The literature search undertaken for this Report also did not identify specific risk perception literature relating to indoor mould. Indeed, the THADE Report [Franchi et al 2006], stated that:

**“The public-at-large is unaware of the negative effects of poor IAQ [indoor air quality].** The public-at-large is more conscious of the negative effects of bad outdoor air quality, particularly as regards traffic pollution. The impact of IAQ is very high because we spend almost all our time indoors (dwellings, workplaces, schools, means of transportation, etc.). Moreover, the public is not aware that various substances found in homes (cleaning products, furniture components, paint etc.) and in material used to build their houses are dangerous, ‘hidden’ sources of pollution.”

Despite this statement it is possible that in particular settings public concern around mould is elevated. Indeed, the “leaky building” issue in New Zealand has had a relatively high media profile (eg, 287 news items on the *NZ Herald* website for the term “leaky building” and 143 for “leaky home” – as of 3 June 2007). Information exchange has been relatively unrestricted, which has highlighted a number of different organisational solutions.

To further explore the issue of public risk perception of mould we considered some risk perception principles that may impact on public concern about “leaky buildings” (adapted from other risk perception work in: [Howden-Chapman & Chapman 1998] [Wilson 1994] [Sandman 1990] [Slovic 1987]).

Work on acceptable risks has consistently shown that the public *perceives* threats to their health from situations over which they have little control. When organisations rather than individuals, play the central role as agents of harm and rescue, this further undermines individuals’ sense of control [Clarke 1989].

Moreover, studies of risk assessment that have looked at risk from the point of view of the *public* facing risks, suggests that an important element in any development of distrust of officials and organisations, is the extent to which the aggrieved see themselves and their responses as being seen by official organisations as part of the hazard, rather than as people who have borne the unintended, but negative consequences of public policy.

The more significant the uncertainties, the more this magnifies the perception of risk.

Another significant factor is that technological advances allow substances to be measured in ever-smaller quantities and this may magnify the perceived risk.

A number of case studies have shown that contrary to the risk assessment literature, risk evaluation is not fundamentally a rational, scientific enterprise and that the solution often depends on the outcomes of inter-organisational struggles, which may not reflect the public interest [Clarke 1989].

Organisations are often active parties, pursuing their own interests by shaping both the form and content of controversies over what constitutes acceptable risk, which like most important public issues is fundamentally about political values and public choice. Risk assessments can be seen as claims to legitimacy that are directed at other organisations. The process of accepting risk involves: problem definition, assessing consequences, ordering alternatives, constructing acceptable risk assessments and accepting the risks. (The sequence of research studies proposed in the previous section would help to establish these parameters.)

The table below details some of the specific issues for the New Zealand context. Some of these issues would appear to increase public concern about mould as a threat to health, others may tend to reduce it and for some the impact is unclear in the current New Zealand context.

Table 6: Some specific risk perception issues relating to mould in homes

<b>Risk perception principle</b>	<b>Comment on relevance to mould in homes (NZ perspective)</b>
<b><i>Likely to increase public concerns regarding mould and health</i></b>	
Control with the individual vs control with society	<p>There is much less public concern over risks in which individuals still remain in control compared with ones where exposure is involuntary (possibly by an order of magnitude). This may apply to some extent for:</p> <ul style="list-style-type: none"> <li>• Individuals in rental accommodation who have limited capacity to remediate house design problems or existing mould problems.</li> <li>• Individuals who blame (correctly or otherwise) other agencies or individuals for mould problems (eg, landlords, architects, builders, building inspectors, local government agencies, central government).</li> </ul>
Not dreaded vs dreaded	<p>Risks associated with “dreaded disease” outcomes attract far more concern. This would apply to some extent where individuals believe or know that mould is associated with:</p> <ul style="list-style-type: none"> <li>• Severe asthma (for which there is indeed some evidence).</li> <li>• Chronic diseases and cancer (eg, concerns about mycotoxins – some of which may have some biological plausibility).</li> <li>• Concerns about serious disease (eg, acute idiopathic pulmonary haemorrhage in infants reported in the media in relation to <i>Stachybotrys</i>).</li> </ul>
Knowable vs unknowable/ unknown	<p>Unknown or poorly defined and quantified risks tend to be perceived as “riskier”. This concern is magnified if there is a public perception of a scientific controversy. This may be the case for mould given that there are many unknowns concerning the health effects and some level of controversy (eg, over the risks of mycotoxins). Aspects of the mould risk (eg, spores or other microscopic components) are also invisible to the human eye and hard to people to understand.</p>
Non-memorable vs memorable	<p>Risks that are associated with personal experiences that are “memorable” tend to increase levels of concern. The “leaking building” situation in New Zealand may have added a memorable element. So may have media reports concerning mould damaged buildings in the wake of Hurricane Katrina in New Orleans.</p>
Diffuse in space and time vs static or focused	<p>Risks that are diffuse in both space and time are of less concern than those that are focused. Visible indoor mould is generally a focused risk.</p>
<b><i>Unclear impact on public concerns regarding mould and health</i></b>	
Trustworthy vs not trustworthy	<p>The perception in information sources as “trustworthy” can reduce public concern over risk. This may mean that statements by university departments and trusted researchers/doctors/scientists are considered credible and can contribute to perceived risk reduction. However, in some societies (perhaps including New Zealand) there are low levels of public trust in “experts” and the public is suspicious of statements by government officials. This may possibly apply to statements around mould in New Zealand in the wake of the “leaking building” issue.</p>

<b>Risk perception principle</b>	<b>Comment on relevance to mould in homes (NZ perspective)</b>
Courtesy and caring vs arrogance and defensiveness	Evidence of arrogance and defensiveness by authorities can be perceived by the public as evidence about lack of concern about risk. Some people in New Zealand may have these negative perceptions in the wake of the “leaking building” issue.
Fair vs unfair	If the distribution of benefits and risks of an exposure are similar over the community the risk tends to be of less public concern. In the case of mould the health risk is focused on those in defective or poor quality housing. Some householders may also consider it is unfair if they pay rent to landlords but do not get adequate living facilities in return.
<b><i>Likely to decrease public concerns regarding mould and health</i></b>	
Natural vs human-made	Exposure to a naturally occurring agent is generally associated with a lower perception of risk than for human-made agents. This will tend to lower concerns about mould – except where people perceive the fundamental problem to be a building design one or other human failure (eg, a plumbing failure leading to water damage). Also where mould decomposition of building materials is substantial, people may become concerned about the “human-made chemicals released”.
Familiar vs exotic	There is less public concern about familiar than unfamiliar risks. Household mould may be generally conceived of as familiar – except where it changes in scales and becomes very noticeable (eg, large black areas appear on a wall). Again, the “leaking building” situation in New Zealand may have added a new and unusual element to public thinking around mould.

Many of the above issues fit with the “leaky building” situation in New Zealand. The risks were unintended, unexpected, unplanned for, and in the case of mould in wall cavities, largely unseen until major structural damage becomes evident. Moreover, the measurement of mould and mycotoxins (toxins produced by some moulds), which is the detectable evidence of “leaky buildings”, has become more sophisticated and therefore more technologically worrying.

As most of these modern homes involved in the “leaking buildings” issue were privately owned, “leaky buildings” may have also undermined the perceived benefits of home-ownership, autonomy, safety, privacy and, in the present climate, capital gains. The uncertainties around liability and remediation may also have undermined householders’ sense of control, and this may be one of the indirect path-ways to causing mental health impacts among the affected householders.

As risk is a relative concept, it is appropriate to ask *whose* risks are being considered by the different organisations involved in the “leaky buildings”. When it comes to the possible policy responses (health protection, compensation, remediation etc), it is important to establish what, exactly, are the risks being mitigated. Unless agreement on these matters among affected parties can establish an “acceptably safe level” of mould exposure, there will be no predictable end to the amount of resources the project would require

before the houses are usable. There are therefore “opportunity costs” from eradicating all mould from “leaky buildings”, because the amount of resources available for other housing problems is diminished.

## **10) RECOMMENDATIONS**

1. Given the rapidly evolving international literature on mould/damp and health, it is recommended that the ACC and other agencies commission regular reviews (eg, every two to three years) to stay abreast of new knowledge in this area.
2. It is also recommended that any future detailed study of housing and health in the Auckland area include work on mould exposure (mould speciation and possibly indoor mycotoxin levels) and detailed health assessment.
3. The health impacts of remediating leaking buildings should be evaluated in order to refine the cost/benefit calculations included in this report.

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### ***Competing interests***

Two of the authors (NW, PH-C) have previously been involved in contract work on housing and health issues for the New Zealand Ministry of Health. However, none of the authors have ever undertaken contract work relating to commercial agencies involved in housing and health issues and have never acted as expert witnesses on housing-related issues.

## **Glossary and acronyms**

*Biological contaminants.* Living organisms (eg, viruses, bacteria, fungi, and mammal and bird antigens) and derivatives that can be inhaled and can cause allergic reactions, respiratory disorders, hypersensitivity diseases, and infectious diseases. Also referred to as ‘microbiologicals’ or ‘microbials.’\*

*Building-related illness (BRI).* Diagnosable illness whose symptoms can be identified and whose cause can be directly attributed to airborne building pollutants (eg, Legionnaire’s disease, hypersensitivity pneumonitis).\*

*Confidence intervals (CI).* A confidence interval is a range of values that has a high probability of containing the parameter being estimated. The 95% confidence interval is constructed in such a way that 95% of such intervals will contain the parameter.\*

*Endotoxin.* A cell wall component of gram-negative bacteria that has been shown to cause airway inflammation.

*EPS.* Extracellular polysaccharides (from fungi).

*Fungi.* The unicellular or multicellular eukaryotic organisms embracing a large group of microflora including moulds, mildews, yeasts. Few fungi actually cause infectious diseases; most health effects are associated with allergic responses to antigenic material or toxic effects from mycotoxins.\*

*Glucans:* These are *non-allergenic* water-insoluble structural cell wall components of most fungi. They are measured in some studies of indoor mould.

*Indoor air quality (IAQ).* The characteristics of the indoor climate of a building, including the gaseous composition, temperature, relative humidity and airborne contaminant levels.\*

*Mould.* Visible fungal colonies found indoors are commonly called “mould” or “mildew”. This Report refers to “mould” interchangeably with “fungi” (see *Fungi*). Specific species and genera of fungi are mentioned where appropriate eg, “Cladosporium”, “Penicillium”, or “Alternaria” species. Others have defined mould more specifically as “a fungal infection that causes disintegration of substance”.

*Mycotoxins.* The toxins that can be produced by some moulds.

*OR.* Odds ratio.

*SHS.* Second-hand smoke (also known as environmental tobacco smoke (ETS) or passive smoke).

*Sick building syndrome (SBS).* Term sometimes used to describe situations in which building occupants experience acute health and/or comfort effects that appear to be linked to time spent in a particular building, but where no specific illness or cause can be identified. The complaints may be localised in a particular room or zone, or may be spread throughout the building.\* (See also *BRI*).

*Volatile organic compounds (VOCs).* Compounds that evaporate from the many housekeeping, maintenance, and building products made with organic chemicals. These compounds are released from products that are being used and that are in storage. In sufficient quantities, VOCs can cause eye, nose, and throat irritations, headaches, dizziness,

visual disorders, memory impairment; some are known to cause cancer in animals; some are suspected of causing, or are known to cause, cancer in humans. At present, not much is known about what health effects occur at the levels of VOCs typically found in public and commercial buildings.\*

Note: \* This definition is from the THADE Report [Franchi et al 2006].

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**APPENDIX 1**

**THE INCREMENTAL COSTS OF HEALTH-RELATED CONSEQUENCES  
OF LEAKY DWELLINGS: A PRELIMINARY ECONOMIC ESTIMATE**

**A review commissioned by the Auckland City Council**

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# The incremental costs of health-related consequences of leaky dwellings: a preliminary economic estimate

## Aim

The aim of this analysis is to complement the accompanying work by Wilson et al on the epidemiology of leaky dwellings, by providing a first approximation estimate of the main incremental costs of the health-related consequences of leaky homes. This analysis does not examine the nature of the health consequences; rather, the focus is on translating the health impact estimates into approximate economic values through setting out a method and its assumptions, and applying that method, placing first-approximation dollar values on the health consequences estimated by Wilson et al.

This study does not include non-health costs of leaky dwellings, such as higher energy costs arising from the need to mitigate dampness by additional heating, and higher emissions of carbon dioxide likely to be associated with additional heating in a proportion of dwellings.

## Methodological approach and concepts

Critical elements in estimating the incremental costs of the health-related consequences of leaky homes include:

- The size of the affected population (population at risk)
- The incremental incidence of health impacts (e.g. respiratory morbidity) attributable to the problem (in this case, leaky dwellings)
- The cost per case of the various health impacts (e.g. the cost of respiratory consequences, cost of days off work, etc.).

Incremental incidence is the higher incidence of a condition attributable to the leaky condition of the dwelling. Thus, incremental incidence is tied to the type of health impact under consideration. It is possible that the incremental incidence of a condition is zero, e.g. the incremental incidence of lung cancer may be zero if leaky homes have no impact on this condition. Incremental incidence is also often described in relative risk terms (e.g. odds ratio of a condition for those in leaky homes compared with those in 'normal' homes).

The cost per case should include both direct costs, such as costs of health care, and indirect costs, such as lost production and mental health impacts such as stress costs. The overall costs are the product of these three factors (population size, incremental incidence and costs), i.e.

Cost = Affected population size *times* incremental incidence of health impacts *times* cost per case of these impacts.

If the 'relative risk' (or odds ratio) of a condition associated with damp and mould is  $r$  ( $r$  greater than 1), and the baseline incidence (whole population) of the condition is say  $b$ , then the incremental incidence,  $i$ , is:

$$i = b(r-1)$$

For example, if  $r=1.2$ , and  $b=0.3$ , then  $i=0.06$

This formulation is similar to that set out in Landrigan et al (2002):

"Costs = Disease rate *times* EAF *times* Population size *times* Cost per case

where EAF represents the environmentally attributable fraction; "Disease rate" refers to the incidence of the disease; "Cost per case" refers to discounted lifetime expenditures attributable to a particular disease including direct costs of health care, costs of rehabilitation, and lost productivity; and "Population size" refers to the size of the population at risk. Clearly, there is a close correspondence with the formulation set out above (in box), given that incremental (attributable) incidence corresponds to disease rate times EAF. If, for example,  $EAF=0.2$  and (as before)  $b=0.3$ , then the incremental incidence,  $i=0.06$ .

## Applying the method

### Estimating the affected population size

#### *Population in damp or mouldy dwellings*

The population of interest is the population exposed to leaky homes with significant dampness or mould. Howden-Chapman et al (2005) note that 35% of respondents in a random sample of New Zealand households reported mould in one or more rooms, with houses in the northern half of the North Island reporting mould significantly more often. While this proportion may seem high, Mudarri and Fisk (2007), in a study of US households, report that the population weighted average prevalence of dampness or mould from a number of studies was 47%, and concluded that "approximately half or almost half of residents of housing units in the United States have a substantially higher risk of experiencing adverse respiratory related health effects because of their exposure to dampness and/or mold in their homes."

An approximate estimate of the relevant population with houses affected by mould or damp could thus be 35% of the total New Zealand stock, or 515,000. The proportion may be higher in the North Island and lower in the South Island.

However, some of these dwellings may suffer from rising damp, or damp/mould due to causes other than 'leaky fabric', for example, damp from damaged spouting. For the present study, a particular estimate is needed for the number of dwellings suffering from damp/mould *due to leaking* caused by construction method or material deficiencies. For the purposes of a first round estimation of impacts, we use in this study the estimate provided by Cosgriff (pers. comm., 2007) of 30,000 damp or mouldy dwellings of this nature.

#### *Population with asthma*

Holt and Beasley (2002) provide a range of estimates of the proportion of the general population suffered from asthma. For example, they report (their Table 7) a prevalence of wheeze in New Zealand among younger adults of 24 to 27%. However, for present purposes, we focus on the proportions who are suffering from severe asthma *and* being hospitalised. This is a much smaller number, 4 per 1000 for children and 1.8 per 1000 for adults (their Table 19). Its use undoubtedly leads to an underestimate of the true cost of asthma, and thus represents very much a lower bound number.

#### *Other respiratory diseases*

No data are readily available to allow estimation of the rates of other respiratory disease in New Zealand, such as chronic obstructive pulmonary disease (COPD), and cough, wheeze and other upper respiratory tract infections. However some indications are provided by Holt and Beasley (2002: 25) for COPD. They note that, in terms of years lost to disability (using 1996 data), COPD is responsible for 73% as much loss as asthma (12418 years lost vs 17059, respectively). Pneumonia does not register on the top 10 list of causes of years lost to disability.

#### *Population with mental health conditions*

A mid-1990s study, the Christchurch Psychiatric Epidemiology Study (CPES), found that 28% of adults in the general population experienced a diagnosable mental disorder in the past six months (Bushnell, 2007). We assume here that this applies for the whole population, i.e. not just adult, and represents the relevant base prevalence of mental health conditions which might be exacerbated by damp or mouldy housing.

### **Estimating incremental incidence**

As set out in the accompanying paper by Wilson et al, leaky housing conditions can be expected to increase the extent of various forms of respiratory morbidity, including asthma. As Landrigan et al (2002: 723) note, asthmatic episodes are the result of complex interactions among genetic predisposition, respiratory infection, climate change, the indoor environment at home and at school, secondhand cigarette smoke, and ambient air pollution.

Dwelling leakiness, and consequent dampness and mould, is thus one of the potential causative or precipitating agents of asthma.

### *Asthma*

The assessment by Wilson et al notes that there is evidence, e.g. from the IOM (2004) study and the THADE project (2004), that leaky dwellings with indoor mould are associated with increased symptoms in sensitized asthmatic persons. Wilson et al note that Mudarri and Fisk (2007), reporting on 33 peer-reviewed United States studies, estimate that exposure to dampness and mould “raises the risk for various adverse respiratory outcomes by 30–50%”. As a result, Mudarri and Fisk estimate, the proportion of current asthma cases attributable to dampness and mould exposure in housing in the US is 21%. We take this as the incremental incidence estimate. In the absence of better data, we assume that this incremental risk (conservatively, 20%) applies in New Zealand.

### *Other respiratory diseases*

Without adequate prevalence rate data for other respiratory diseases, specifically COPD, cough, wheeze and other upper respiratory tract infections, we cannot estimate the cost of increased incidence of these conditions. As noted before, however, omission of the costs of these conditions will lead to a significant underestimation of the overall costs of health conditions exacerbated by damp or mouldy dwellings.

For reference, Mudarri and Fisk (2007) and WHO (2006:15) provide evidence on the incremental incidence of some of these conditions. Table 1 below shows the odds ratio data (note that the odds ratios for other respiratory symptoms are generally marginally higher than for asthma). It could be assumed that the incremental incidence for the other diseases listed below is approximately the same as that applying to asthma, i.e. the incremental incidence may be around 20%.

Table 1: Summary of health risks for dampness and mould in United States houses [Fisk et al 2007; Mudarri & Fisk 2007].

<b>Outcome</b>	<b>Number of studies</b>	<b>Odds ratio (95% CI)</b>
Upper respiratory tract symptoms	13	1.70 (1.44 – 2.00)
Cough	18	1.67 (1.49 – 1.86)
Wheeze	22	1.50 (1.38 – 1.64)
Current asthma	10	1.56 (1.30 – 1.86)
Ever diagnosed asthma	8	1.37 (1.23 – 1.53)
Asthma development	4	1.34 (0.86 – 2.10)

### *Other health conditions*

There is growing evidence of the *mental health impacts* of dampness and mould that can be inferred from evidence on houses with structural problems, including water leaks from outside and leaks from inside the structure. Weich and Lewis (1998), from a study of 10,000 UK adults, found an odds ratio of 1.4 for common mental disorders among those in housing with structural problems.

This sort of empirical evidence comes from studies of people in substandard housing moving to improved housing or their homes being retrofitted. There is almost no direct evidence on the impact on mental health of people who assumed they were in good quality housing but discovered that their housing was substandard. Anecdotal reports (Easton, 2007) suggest that this change in status could be highly psychologically stressful.

## **Estimating cost elements (cost per case)**

### *Introduction*

The costs of the various health consequences include the costs of:

- treatment (including hospitalisation)
- days off work
- days off school and other educational costs arising where children living in cold and damp homes have their learning impaired
- increased judicial and police services if cold and damp homes lead to increased likelihood of certain criminal behaviour (Gilbertson et al, 2006: 14)
- pain, suffering and stress to the individual/family members caused by the illness
- rehabilitation
- lost leisure time.

Some of these costs, e.g. the costs of days off work and school, will not vary by health condition/ consequence; i.e. the cost of a day off is the same whether the day off is caused by asthma or by cough/wheeze, for example. But some will vary, e.g. costs of treatment of pneumonia will differ from the costs of asthma treatment; and costs of suffering such as stress will vary by condition.

Another conceptual point is that health consequences may, in some more extreme cases, become ongoing. Jaakola et al (2002) point to the presence of mould as associated with the *development* of asthma. This is supported by Mudarri and Fisk (2007) who point to four studies associating asthma development with mould and dampness. On the other hand, as Wilson et al note in respect to the study by Douwes and Pearce (2003), “there is consistent evidence that dampness exacerbates pre-existing respiratory conditions such as asthma, but it is not clear whether it also causes these conditions.” It is possible, then, that in some cases asthma continues even after environmental exposures are terminated, but more research is needed to

quantify any ongoing consequences of limited-period exposure. In the current study, it is assumed that all health consequences are 'current' (i.e. future cost streams due to ongoing conditions precipitated by dampness and mould are ignored); thus, costs estimated here may well understate the real costs of total health impacts.

Literature, especially New Zealand literature, on cost elements is scarce, and while some papers discuss approaches to estimating costs (e.g. Davidson, 2007; McChesney et al, 2006), few provide useable estimates, or provide estimates of treatment cost for only some conditions (e.g. Asher and Byrnes, 2006). Thus, a comprehensive set of cost estimates is not available. Some elements of cost cannot, of course, be readily valued; examples are costs of pain and suffering, and lost leisure time. Thus, as noted by the US Environmental Protection Agency (1999: H15), estimates of health costs that omit these elements will underestimate total health costs.

Once estimated and aggregated, cost streams have to be assessed over the horizon of interest. For example, under a 'business as usual' base case assumption, the dampness and mould in leaky homes will continue to cause health consequences over the horizon. By contrast, if a policy measure is introduced to eliminate the leakiness problem, then the future health cost streams can be assumed to cease.

The relevant horizon is assumed here to be 10 years, on the basis that, in the absence of policy action to remedy the dampness and mould problems, other non-health concerns may lead to remediation of the dwellings after a decade or so. This is an optimistic assumption; it might also be argued that under 'business as usual' the health costs of damp and mouldy housing might continue to be incurred for a longer period. In any case, making this assumption implies a conservative estimation of future health costs.

The discount rate taken to be appropriate here is 5% real, i.e. future health costs are discounted to the present at a real discount rate of 5%, with a sensitivity range tested, of 3% to 7%. This is consistent with the international literature, e.g. Landrigan et al (2002: 723) use a discount rate of 3%; US EPA (1999: H32) use 5%, with a range of 3 - 7% (see also NCEDR, 2004, and New Zealand papers: Chapman et al, 2007; Young 2002).

### *Costs of treatment for asthma*

The costs of asthma vary depending on whether the condition involves hospitalisation or not; that is, impacts can be categorised into overnight (inpatient) admissions and day (outpatient) visits. Typically, the cost to the household associated with a hospitalisation is ignored, unless captured partly in cost of days off work or school. Estimates for the costs of asthma admissions were used (for children, adults and older people) based on data for 1994/95 with prices updated using a component of the producer price index to align all price data to 2006 (Holt and Beasley 2002; New Zealand Health Information Service 1999; Statistics New Zealand 2007); this gives an approximate average cost per asthma admission (i.e. average across age

groups) of \$1,904 in 2006.<sup>1</sup> Holt and Beasley (p. 36) also give guidance as to the relative size of inpatient and outpatient admission costs for asthma: they report the ratio as about 5.7 to 1; that is, outpatient costs add another 17.6% to inpatient costs of asthma.

#### *Costs of other respiratory conditions*

Examples of other respiratory conditions are pneumonia and COPD. We are not aware of a cost per case estimate for COPD. Asher and Byrnes (2006: 38) provide data for the estimated cost of pneumonia treatment for children: an average hospital admission cost around \$1,500 while an average outpatient visit cost around \$200.

#### *Costs of non-respiratory conditions*

The largest part of the cost of poor mental health (depression, anxiety disorders, etc.) arises because of the sheer number of people affected. One estimate is that worldwide, mental health conditions account for about 28% of years lived with disability (Mental Health Commission, 1998: 6), and this is supported by New Zealand survey work by Bushnell (2007). Bushnell's estimate of the proportion of the general population suffering from depression and anxiety is around 28%. In addition, there is a small additional cost due to some cases of mental health conditions proceeding to suicide.

To estimate the cost per case of mental health conditions, we draw on a preliminary study by the British economist Richard Layard (Layard et al 2006). In particular, we use that study's estimate of the cost of mental illness that could be averted by treatment, i.e. the benefit of treatment. Layard et al's estimate is around NZ\$12,343 per case, excluding fiscal benefits which may be in the nature of transfers; we use a more conservative estimate of half this for New Zealand (\$6,171), to allow for a lower overall cost structure.

Data on suicide costs arising from mental health conditions are drawn from O'Dea and Tucker (2005), who note (p.29) that around 70 per cent of suicides are estimated to be related to depression. They also note that, in all the suicide studies, direct costs were massively dominated by indirect costs, and that typically the latter were of the order of \$400,000 per suicide and upwards. We make the assumption that depression is the *cause* of only 35% of the suicides in New Zealand (i.e. conservatively, half of the cases that are *associated with* depression). We can then estimate the suicide consequence of depression in annual cost terms.

In New Zealand, there were 460 suicides in 2002. The economic cost of these 460 suicides in 2002 was around \$206 million (including the cost of lost production). We use O'Dea and Tucker's estimate of the cost of depression per case of suicide as around \$448,000. The incremental level of suicide is estimated as around *one case per year*.

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<sup>1</sup> The producers price index input series for health and community services was used for cost updating.

### *Cost of days off work*

The cost per day for days off work can be proxied using the estimated cost of forgone earnings, on average and, conservatively, discounting this by some proportion (say 33 %) to allow for the offsetting gain from avoiding the disutility of work, and workers' ability to make up work after a day off, or have a co-worker make up. It is assumed that the value of lost production (approximated by the gross daily wage rate) is the relevant yardstick, rather than an estimate of the direct cost to the worker of a day off (the latter could be less, for example where the day off is covered by sick leave). Data on average daily wage rates for New Zealand workers can be taken from Statistics New Zealand (Statistics New Zealand 2005 and ongoing). For example, based on those wage rates, and with the adjustments mentioned above, a value (cost) of \$99 per day in 2002 dollars can be placed on a day off work. For comparison, the US Environmental Protection Agency (1999) uses an estimate of US\$83 in 1992 dollars for a day of lost earnings (making no deduction for the disutility of work). The New Zealand value of \$99 per day in 2002 dollars is equivalent to \$113 in 2006 (using earnings series from Statistics New Zealand 2006).<sup>2</sup>

The number of days off work caused by increased dampness and mould due to leaky homes is not known. However, one way in which it can be roughly proxied is on the basis of the measured impacts of better housing insulation in the study reported in Chapman et al (2007: Table 4), on the assumption that retrofitting a standard insulation package may have approximately the same (positive) impact effect as dampness and mould have a (negative) effect. That study reported 102 fewer days off work per 1000 adults, per year.

### *Costs of days off school*

A similar approach applies to days off school: the insulation cost-benefit study provides an estimate of the incremental effect and costs per day off are estimated based on teenager labour earnings, discounted appropriately. Chapman et al (2007: Table 3) estimate days off school as 512 per 1000 children, and 1316 per 1000 teenagers.

## **Pulling together the estimates**

1. The final step in making an overall preliminary cost estimate for New Zealand for the health consequences of leaky dwellings is to bring the elements identified above together.
2. *Incremental asthma costs*
3. The estimate is limited to the costs of incremental asthma admissions to hospital, plus outpatient asthma treatment costs, and omits pharmaceutical and other costs of incremental treatment of asthma. On

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<sup>2</sup> Ordinary time earnings (male and female) increased 14.5% between 2002 and 2006; hence the estimated cost of a day off work increased from \$99 to around \$113.

this basis, incremental costs are estimated at around \$866,000 per year (in 2006 dollars).

4. To put this estimate into perspective, Holt and Beasley's estimate of the total direct costs of asthma (all causes) was around \$125 million per year in the late 1990s. However, they did not limit their estimate to severe cases that were hospitalised; they included pharmaceutical, out-of-pocket patient costs and other costs in this overall asthma cost estimate; and their estimate is a total for *all causes* of asthma, not just asthma caused by particular housing conditions.

5.

#### 6. *Incremental mental health costs*

The major cost element in the mental health area arises from the incremental costs of conditions such as depression and anxiety, which are already widespread in the population and expected to be exacerbated by damp and mouldy housing conditions. These are estimated at \$58,957,000 per year, a very large cost by any standard.

The incremental cost of suicides (about one per year) arising from mental health conditions due to damp/mouldy dwellings is estimated at \$471,000 per year. Clearly, this is only a small addition to the major 'general' mental health effect above.

#### *Days off work and school*

The cost of days off work and school could only be very roughly approximated due to a lack of good estimates for the incremental impact on health and hence absenteeism of damp or mouldy dwellings. The estimate made here is \$654,000 for incremental days off work and \$427,000 for days off school, per year.

#### 1. *Total costs per year*

2. Adding the various cost elements, the total cost due to asthma hospitalisations and outpatient costs, mental health, days off work and days off school due to damp/mouldy housing is estimated at around **\$61.4 million per year** for New Zealand as a whole. This estimate is in 2006 dollars. It is very likely to be a highly conservative estimate, since it omits a number of possible health impacts of damp or mouldy housing, such as COPD, and deliberately makes conservative assumptions about estimated costs.

3.

#### 4. *Present discounted value over a horizon of 10 years*

5. Taking a horizon of 10 years as suggested above, the total present value of costs over this horizon can be estimated for various discount rates. As

discussed above, there is a good case for placing most weight on the estimate made using a 5% discount rate (in bold in Table 1 below).

6.

7. Table 1: Summary of the preliminary incremental cost estimates for the health impact of leaky dwellings

1.	1. Present discounted value of incremental health costs of damp/mouldy dwellings, over a 10-year horizon		
1. Discount rate	2. 3%	<b>1. 5%</b>	1. 7%
1. Present value <sup>1</sup>	3. \$523.5 million	<b>2. \$473.9 million</b>	2. \$431.1 million

1. 1: Based on an annual value of costs of \$61.4 million (2006 dollars)

## Discussion

This study has provided only preliminary cost estimates for the health impact of leaky dwellings. Numerous simplifying assumptions have been made, and a number of elements of potential health cost have been omitted for lack of data. In almost all cases, where an assumption has been made, a conservative estimate was used. The results shown in Table 1 above should be treated with considerable caution, and as a conservative estimate of the true level of costs, for these reasons.

What stands out in terms of total cost is the significance of mental health conditions that may be exacerbated by damp or mouldy housing. Further work to check assumptions here would be worthwhile, especially considering that cost per case estimates from the UK have been used, for want of better local estimates of cost impacts. In other words, further research on the impact of mould and damp on mental health in New Zealand circumstances may be the most critical element for future economic analyses.

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