

Modelling the effects of low indoor temperatures on the lung function of children with asthma

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ABSTRACT

Introduction While many epidemiological studies have shown that low outdoor temperatures are associated with increased rates of hospitalisation and mortality (especially for respiratory or cardiovascular disease), very few studies have looked at the association between indoor temperatures and health. Such studies are clearly warranted, as people have greater exposure to the indoor environment than the outdoor environment.

Objectives To examine the relationship between various metrics of indoor temperature and lung function in children with asthma. Our specific research questions were: (1) In which room of the home is temperature most strongly associated with lung function? (2) Which exposure metric best describes the relationship between indoor temperature and lung function? (3) Over what lag/time period does indoor air temperature affect lung function most strongly?

Methods The Heating Housing and Health Study was a randomised controlled trial that investigated the effect of installing heaters in the homes of children with asthma. This study collected measurements of lung function (daily) and indoor temperature (hourly). Lung function and indoor temperature were measured for 309 children over 12 049 child-days. Statistical models were fitted to identify the best measures and metrics.

Results The strongest association with lung function was found for the severity of exposure to low bedroom temperatures averaged over the preceding periods of 0–7 to 0–12 days. A 1°C increase in temperature was associated with an average increase of 0.010, 0.008, 10.06, 12.06, in our four measures of lung function (peak expiratory flow rate (PEFR) morning, PEFR evening, forced expiratory volume in 1 s (FEV₁) morning and FEV₁ evening).

Conclusions Indoor temperatures have a small, but significant, association with short-term variations in the lung function of children with asthma.

INTRODUCTION

The short-term (0–14 days) health effects of low outdoor temperatures have been well studied in large populations over long time periods.^{1–7} In particular, the association of low outdoor temperatures with respiratory diseases is well established.^{1–3 6} In these outdoor studies, outdoor temperatures are assumed to be a measure of personal exposure to temperature, but indoor exposure is clearly more important, because people spend 80% of their time indoors.^{8 9}

Many factors that are plausibly correlated with low, indoor temperatures; poor-quality housing, damp^{10 11} and mould^{12 13} have been shown to affect the respiratory health of children. Two

randomised community trials (carried out by our research group) have shown an improvement in wheeze and cough symptoms with improved home insulation¹⁴ and improved home heating.¹⁵ Only two studies have examined the direct association between children's respiratory health and indoor temperature. Williamson *et al*¹⁶ reported that the homes of asthmatic children were significantly colder than non-asthmatic controls and Strachan and Sanders¹⁷ reported that temperature was not associated with questionnaire recorded respiratory symptoms. However, these studies were of limited scope and measured temperature and each health outcome on only one occasion per child. Studies of indoor temperature and health restricted in this way to a single (time) measurement for each subject will be heavily confounded by socioeconomic factors, such as poor-quality housing^{11 18} and fuel poverty.¹⁹ Such single measures also cannot provide any information about how long such effects last.

Outdoor studies in, the Netherlands,²⁰ Spain,² London,⁶ Bulgaria⁶ and in 11 US cities²¹ have found that the association between low outdoor temperatures and exacerbation of respiratory disease was best modelled as 'severity' or exposure to low temperatures below a (region-dependent) threshold over a lag of 7–14 days.^{2 6 20}

Metrics other than the simple average such as 'severity' or 'average below a threshold' effect are also plausible in the indoor environment. The outdoor studies suggest that the lag in the effect of exposure may be due to impaired immunological defences,² although the evidence concerning this mechanism is weak.^{20 22} This mechanism is equally plausible in the indoor environment.

Measurement of indoor temperature requires regular monitoring, not only of individual houses, but of different locations in each house. For a low-incidence outcome such as hospitalisation, sufficient indoor temperature measurements are therefore too costly to collect. However, it is possible to examine sensitive subgroups for more common, but less severe outcomes. Children with asthma are particularly sensitive to a wide variety of airway environmental challenges with even small changes having the potential to reduce lung function and increase respiratory symptoms.^{8 23} Little research has been conducted on how lung function of children with asthma changes with indoor temperatures.

Exposure to low temperatures could reduce their lung function either directly by reflex bronchoconstriction²⁴ or by increased water loss in less humid, colder air²⁵ or indirectly by impairing immunological defences to respiratory infections such as

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viruses.²⁶ If exposure to low indoor temperatures reduces lung function directly, these temperature effects on lung function would manifest quickly. Whereas, if impaired immunity to viruses, for example, is the more powerful mechanism, there will be a lag between the exposure and the reduction in lung function. Research suggests that virus-induced asthma symptoms are apparent 3 days after exposure to and infection by the virus.²⁷ The viruses themselves have longer survivability in cold environments.²⁸

The data analysed come from the Housing Heating and Health Study, a randomised controlled trial that investigated the effect of installing heaters in the homes of New Zealand children with asthma (see Description of the New Zealand Climate, New Zealand Fact File, online supplementary appendix 1). The study found that installing heaters raised the average indoor temperature and reduced the children's respiratory symptoms.¹⁵ Written consent included a description of the i-button temperature monitors and participants were told that the heaters would be assigned randomly and not removed after the study. The initial analysis of the study was restricted to analysis of changes in temperature due to the intervention.¹⁵ In this paper, we use the detailed repeated measurement data to examine the relationship between different metrics of exposure to indoor temperature and respiratory health. In particular, we seek the most appropriate summary exposure measure that can be derived from the hourly indoor temperature measurements.

Our aim was to determine how the short-term effects of temperature can best be modelled to show maximum association with changes in lung function of children with asthma. Our specific research questions were:

1. What is the most important location to measure the effect of indoor temperature on the child's respiratory health: the bedroom, the living room or a combination of the two?
2. Is the effect of indoor temperature on respiratory health best determined by the 'severity' of exposure (average exposure to low temperatures below a threshold), the 'duration' of exposure (length of exposure to low temperatures below a threshold) or a simple average of temperature?
3. Over what time period or lag from 0 to 14 days does indoor temperature most strongly affect lung function?

METHODS

In this analysis, our approach is to find an appropriate daily summary measure of the hourly multilocation temperature measurements that best correlates with the lung function measures in children with asthma. A data reduction of this sort is necessary to analyse the effect of the intervention (the provision of a heater) on health outcomes through the mediating variable of temperature. This necessitates a data mining exercise that carries the inevitable drawbacks of multiple testing as we investigate a very large number of potential data summaries and models. We have therefore taken care to construct measures that are informed by the relevant literature. We considered an overall average measure as performed previously indoors,¹⁶ severity and duration measures as suggested by the outdoor studies^{2,6} and a more speculative difference in temperature approach.²⁹ For all these models we examined a range of time periods and thresholds, as performed in the outdoor studies.⁵

Study population

In total, 409 New Zealand children aged 6–12 years with doctor-diagnosed asthma and inefficient home heaters, such as 2 kW plug in electric heaters, unflued gas heaters or no heaters were enrolled in the Housing Heating and Health study.¹⁵ One

child with asthma was studied in each household. The children were visited every 4 weeks from 1 June until 7 October 2008 (128 days), the visits were staggered and the children were each in the study for approximately 100 days. Half the households were randomised to receive a new heater >6 kW before the winter of 2006 and the control group were given new heaters at the end of the study.

Measurements

Lung function was measured each morning and evening daily throughout the winter of 2006. The children were given and instructed in the use of Piko meters (nSpire Health, Longmont, Colorado, USA), which were used to self-record forced expiratory volume 1 s (FEV₁) peak expiratory flow rate (PEFR) and an indicator of the validity of the blow (as displayed by the meter) when providing five blows every morning and evening. The highest valid FEV₁ and PEFR from each set of five blows were used as outcome measures in this study. We report the results for four separate lung function outcomes (PEFR morning, PEFR evening, FEV₁ morning and FEV₁ evening). Separate morning and evening outcome measures are necessary for the purposes of this analysis, as there is a large natural diurnal variation in lung function.²³ Temperature meters were used to record the temperature every hour in the living room and the child's bedroom.

Hourly individual measurements

We measured temperature every hour in two locations: the child's bedroom and the living room: that is, there is a temperature measurement T_{idhrss} for each child i on day d at hour h in location r (bedroom or living room). Using the pairs of temperature measurements at each hour we defined four hourly exposure measures. The first two are simply the temperature in the bedroom $X_{idh}^B = T_{idh}(\text{bed})$ and the temperature in the living room $X_{idh}^L = T_{idh}(\text{living})$. The third measure we refer to as 'Assumed Personal Exposure' (APE) X_{idh}^A , which we define as the bedroom temperature from 21:00 until 8:00 and the living room temperature between 16:00 and 20:00, to reflect an assumed location of the child at each hour. Data between 8:00 and 16:00 were discarded when using this exposure measure. In addition, we hypothesised that change in temperature overnight could be an important exposure.²⁹ Therefore, we defined a fourth hourly exposure measurement X_{idh}^D which is the 'difference exposure'. For each night, we first extracted the maximum of the living room temperature at 20:00 and the temperature in the bedroom between 21:00 and 8:00. We then took the hourly measure as the difference between this maximum value and the hourly bedroom measure. The measurements at 20:00 were taken in the living room to capture an exposure driven by moving from a warm living room to a cold bedroom. Daytime temperatures were discarded when using this exposure measure.

Aggregation of hourly measurements to daily measurements

Our exposure data were measured hourly, but each of the four lung function outcomes were measured daily. We therefore aggregated the hourly exposure measurements into daily measures. For each of the four types of hourly exposure measurement X_{idh} we aggregated the observations in three different ways (over the hours for which that measure is defined) to create a daily exposure measure.

For any given observation period R of length n_R hours, let X_{idh} be the exposure measure for child i on day d at hour

h. The daily exposure to low temperatures is calculated in three ways:

1. The *duration* of exposure to low temperatures below some threshold is defined as the proportion of hours per day of exposure less than a threshold H :

$$D_{id} = \frac{1}{n_R} \sum_{h \in R} I[X_{idh} < H]$$

where $I[X_{idh} < H] = 1$ if $X_{idh} < H$ and is zero otherwise. We calculated this daily measure for temperatures H from 10°C to 18°C for bedroom, living room and APE. The difference exposure is similarly defined except exposure is the duration of the contrast *above* the threshold H , and H was varied from 1°C to 10°C.

2. The *severity* of exposure to low temperatures S_{id} is the mean number of degree-hours below a chosen threshold temperature H :

$$S_{id} = \frac{1}{n_R} \sum_{h \in R} \max[H - X_{idh}, 0]$$

We calculated this daily measure for temperatures of H from 6°C to 14°C for bedroom, living room and APE. The difference exposure is similarly defined except exposure is the average contrast *above* the threshold H , and H was varied from 1°C to 10°C.

3. We also calculated the simple *average* exposure:

$$A_{id} = \frac{1}{n_R} \sum_{h \in R} X_{idh}$$

The ranges of threshold temperatures were chosen to capture the best predictive models.

Time period

It is possible that the impact of temperature on lung function takes longer than 1 day to produce a measureable change. Therefore, we averaged the daily exposure measures over a range of time periods preceding each lung function measurement. The outdoor models mostly used a lagged exposure window where, for example, the exposure at the 3-day to 7-day lag is the average exposure measured between the third and seventh days before the outcome event. However, we used a full average over all of the preceding L days, (ie, where L is 7 we average the chosen daily exposure measure over all the 7 days preceding the outcome). This average was chosen because we felt this was more biologically plausible than the window method used in the outdoor studies. In order to ensure that the exposure precedes the outcome, we only used the data until 8:00 for day 0.

Figure 1 shows, for illustrative purposes, the data for a single child collected during the study and include the health outcome and various bedroom temperature measurements. The PEFR

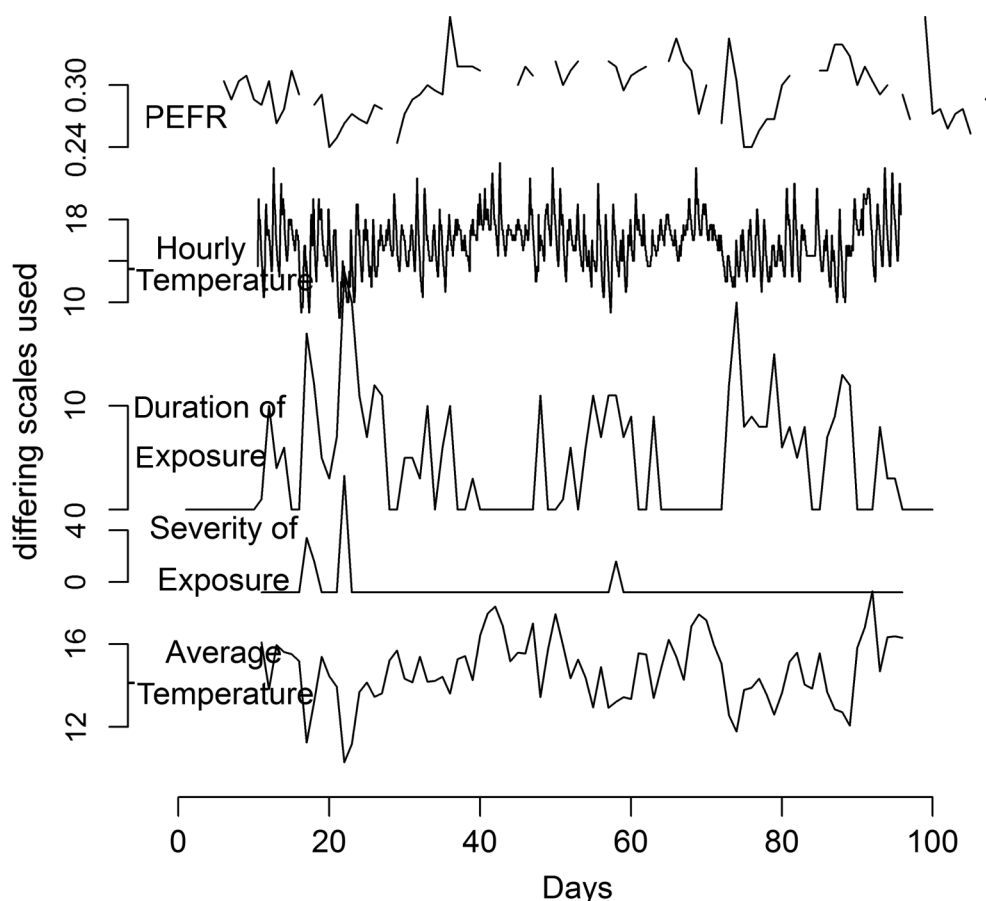


Figure 1 The hourly temperature is measured in the bedroom $T_{idh(\text{bed})}$; the *duration* D_{id}^B of hours in the day that the bedroom temperature was less than 14°C; the *severity* S_{id}^B is the weighted average of bedroom temperature for time spent below 10°C; and the *average* daily bedroom temperature A_{id}^B .

line is the PEFR morning measurement from each day, where this was recorded. The gaps indicate unrecorded days. The hourly temperature is the temperature in the child's bedroom recorded every hour. The duration of exposure D_{id} line is recorded daily and is the number of hours when the temperature was below $H=14^{\circ}\text{C}$. It is zero when the temperature did not fall below 14°C . The severity score S_{id} is 0, except where the temperature dips below a fixed threshold (in this case $H=10^{\circ}\text{C}$). The average daily temperature A_{id} is the average of all temperatures recorded over the day.

Statistical methods

The outcome measures were continuous variables (FEV₁ morning, FEV₁ evening, PEFR morning and PEFR evening) for up to 110 days/child. To capture this repeated measurement for each child, we used a random effects structure with hierarchical linear mixed effects model³⁰ to relate each of the health outcomes to each lagged exposure measure with a random effect term for each child. For each health outcome, we fitted models with the four different types of hourly measure (bedroom, living room APE and difference), three different daily aggregates (severity, duration and average), and 15 different averaging periods $L=0-14$ days. Models were compared to identify those with the best fit based on the Akaike Information Criterion (AIC).³¹ When comparing two models a difference of three or more in the AIC indicates that the model with the lower score fits the data significantly better. The mean level of the outcome over all time periods was included as an

explanatory variable for each child, removing the effects of time-invariant child-level explanatory variables (such as age, sex and whether the child was in the intervention or control arm) on the model fit. The models with morning outcomes were adjusted for reliever use during the night (yes/no) and the NO₂ level. The evening models were adjusted for the number of reliever puffs taken during the day, the number of preventer puffs taken during the day and the NO₂ level. The NO₂ level that was used in the adjustment is the log of the level recorded in the 4-week time period during which the outcome is measured.^{32 33} We assumed that all data missing was missing completely at random. All models were fitted using the R software (V2.15.1).³⁴

RESULTS

There were 409 households in the study, with a total of 1.27 million hourly temperature measurements (620 000 in the living room). There were 286 children and 9194 child-days, where we had NO₂, asthma medication use, at least 10 days of lung function measures and the previous 14 days' temperature measurements (figure 2).

The 286 children were aged between 7 and 13, with a mean age of 10.4 years, 59% were boys, 34% were Māori (indigenous New Zealanders), 17% were of Pacific Island ethnicity, 54% had a family history of asthma and 19% had a smoker living in the home. Heights were only available for 203 children and these ranged from 111 to 176 cm with a mean height of 142 cm. The

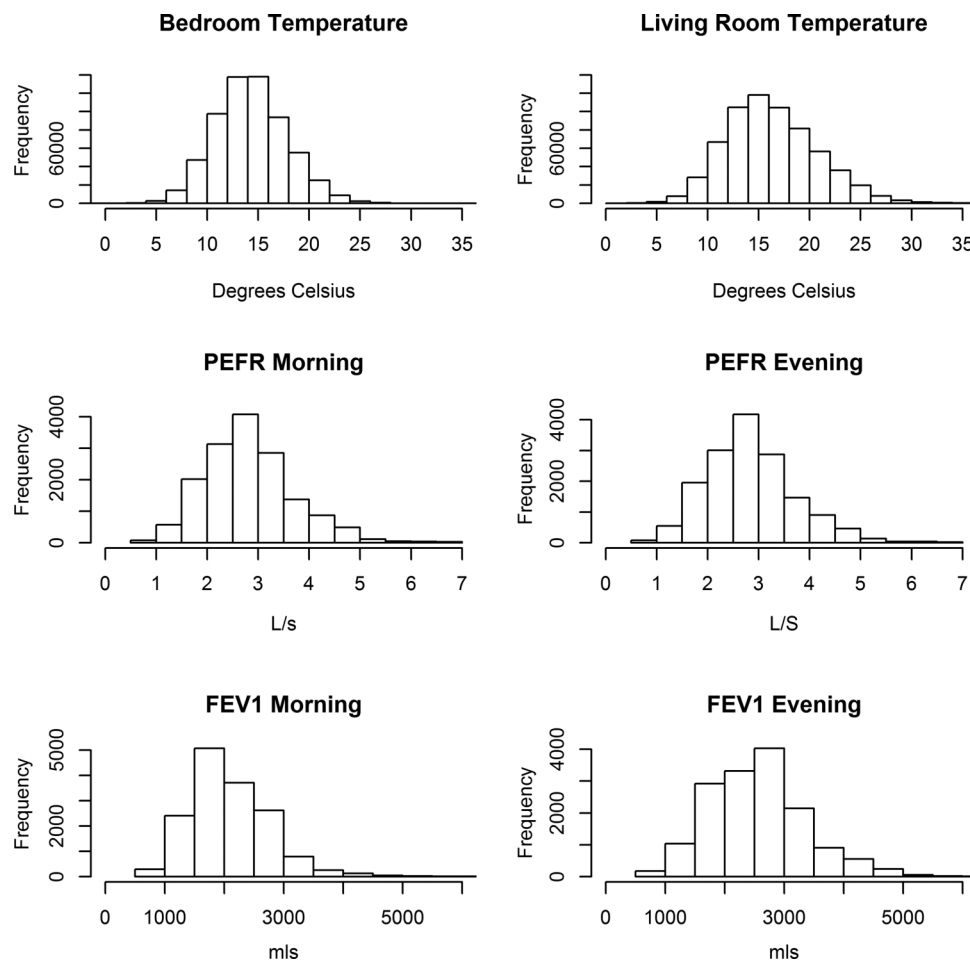


Figure 2 The temperature and lung function measures.

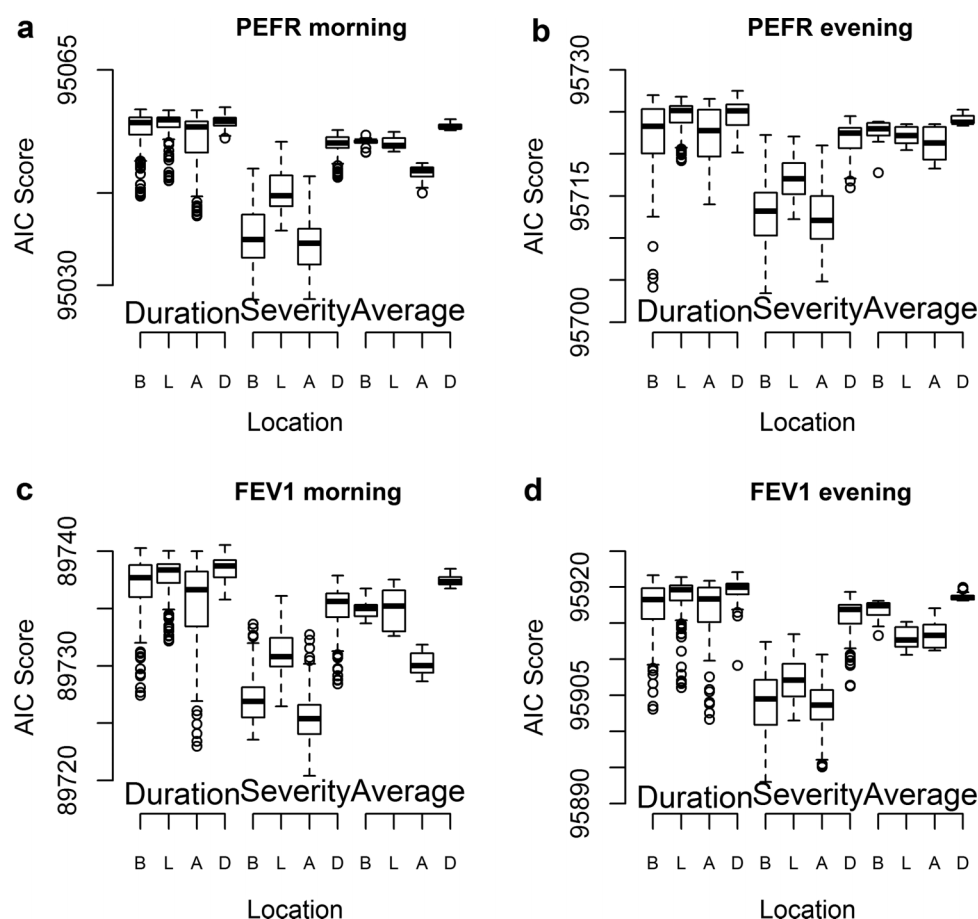


Figure 3 (a–d) Boxplots of the Akaike Information Criterion (AIC) scores for all models of peak expiratory flow rate (PEFR) morning with duration, severity and average for exposure to bedroom (B), living room (L), assumed personal exposure (A) and difference (D). For each of the four outcomes PEFR morning, PEFR evening, FEV1 morning and FEV1 evening, there are 135 models (9 temperature thresholds by 15 time periods for the averaging window) for duration and severity by each hourly measurement, and 15 models (one for each time lag) for the average exposure by each hourly measurement. A lower AIC score indicates a better fit.

mean bedroom temperature was 14.4°C and the mean living room temperature was 16.53°C.

Figure 3a–d show the boxplots of the AIC scores of each fitted model. AIC is a measure of the relative quality of a statistical model for a given set of data, with lower AIC scores indicating a better model fit. In figure 3 it is clear that the median for severity has significantly lower AIC and therefore greater association with all our measures of lung function, than either duration or average exposure. Similarly, for the four sets of hourly individual measurements (B/L/A/D) the difference measure (D) is the worst (highest AIC) at explaining lung function, while all living room (L) models are significantly worse than the best bedroom (B), and APE (A) models, both of which gave similar results. In 45% of models, temperature was significantly associated with lung function at the 0.1 significance level, in 38% of the models at the 0.05 level, in 26% at the 0.01 level in 12% at the 0.001 level and 1.2% at 10^{-5} level.

For PEFR morning and evening outcomes the best fitting model was using the bedroom severity measure, whereas for FEV₁ morning the best fitting model was for severity with APE. Next we examine just these models, by varying the severity threshold temperature and the numbers of days over which the exposure measure was averaged (figure 4). The models using 9°C, 10°C or 12°C as a threshold and an averaging window of

6–11 days in length had the lowest AIC scores. The best individual models for each outcome are shown in table 1.

In table 1 we show the final model selected for each of the four health outcomes. For three of the outcomes, the variable with the strongest association was severity of exposure measured in the bedroom at a lag of 6–9 days. For FEV₁, morning severity of exposure (as measured by APE with an 11-day averaging window) had a marginally better association than bedroom temperature measured over the same period.

The estimated model parameters show that for every 1°C increase in temperatures below the threshold of 9°C we would expect PEFR to improve by 0.010 L/s in the morning and 0.008 L/s in the evening. We would also expect FEV₁ to improve by 10.06 mL for every 1°C increase in temperatures below 12°C in the morning and 12.06 mL in the evening for every 1°C increase in temperatures below 10°C.

DISCUSSION

Bedroom exposure was found to have a stronger association with asthmatic children's lung function than living room exposure. From the bedroom measurements, the measures with the greatest association with lung function were severity or the average exposure below a threshold. The best threshold was in the range 9–11°C. 87% of the models that used the severity of exposure to lagged bedroom temperature were significant at $p=0.05$, but only 15% of

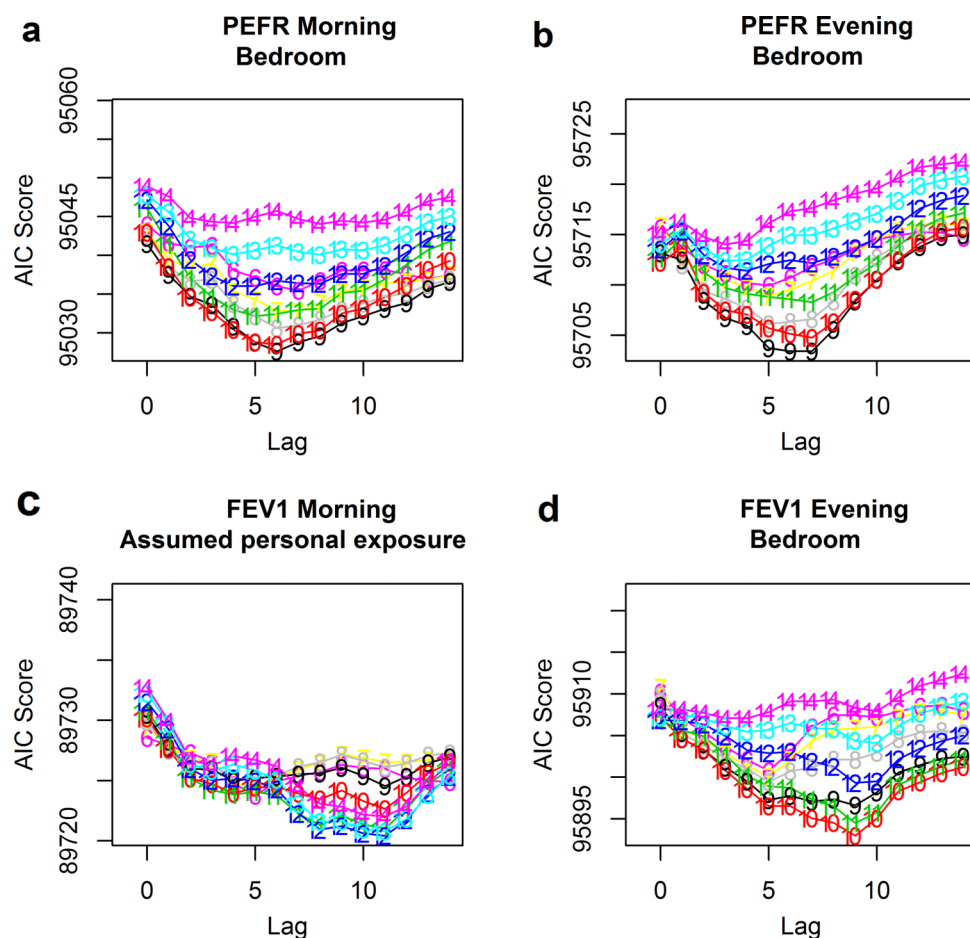


Figure 4 (a–d) Akaike Information Criterion score for models predicating peak expiratory flow rate each outcome by severity of exposure with the length of the averaging window of 0–14 days and cut-off temperature of 6–14°C. The numbers on each curve indicate the threshold temperature that was used.

those that used any of the ‘average’ measures were significant at this level. Outdoor studies have reported that similar severity metrics give the strongest association with lung function. However, in the outdoor studies the threshold varied considerably by the method used and in the regions studied. For example, some thresholds used were 5.25°C in London,⁶ –0.46°C in Sofia,⁶ 16.5°C in the Netherlands²⁰ and 18°C in the Eurowinter⁷ study. Allowing for the differences in methodology, our threshold values of 9–11°C are within the wide range of values used in the outdoor studies.

We found that lung function had the strongest association with temperature metrics defined as the rolling average over a time period of 7–12 days. In general, the outdoor studies averaged temperature exposure over the previous day, at lag 1, at lag 1–3 days, at lag 4–6 days and at lag 7–14 days previously. Of

these, the exposure at lag 7–14 days had the greatest association with health outcomes in most studies.^{2 6 20} When we examined our data with the lags used in the outdoor studies, the 4-day to 6-day lag had the greatest association. However, none of the models with this window gave a better fit than our models using an averaging window of full length between 0–7 and 0–12 days. Our slightly shorter preferred lag could be due to the less severe nature of our outcome (decrease in lung function) compared with theirs (hospitalisation or mortality).

We found that the changes in temperature are associated with small changes in lung function. These changes in lung function are similar to those reported by Donaldson *et al*³⁵ who showed a small increase in lung function for patients with chronic obstructive pulmonary disease, when there was a rise in indoor

Table 1 Model with the greatest association for each measure of lung function

Outcome	Time of measurement	Aggregator	Hourly measurement	Threshold (°C)	Length of averaging window	Average effect of constant 1°C increase in temperature	p Value
PEFR	Morning	Severity	Bedroom	9	6	0.010 L/s	10 ^{–8}
PEFR	Evening	Severity	Bedroom	9	7	0.008 L/s	10 ^{–6}
FEV ₁	Morning	Severity	Assumed personal exposure	12	11	10.06 mL	10 ^{–6}
FEV ₁	Evening	Severity	Bedroom	10	9	12.06 mL	10 ^{–9}

FEV₁, forced expiratory volume in 1s; PEFR, peak expiratory flow rate

bedroom temperature. The WHO noted that “improvement of the indoor climate of dwellings is recognised as an efficient means of secondary prevention of acute respiratory infection.”²²

All models were adjusted for NO₂ and medication use. NO₂ was significant at $p = 0.05$ in less than 5% of the model. This is probably due to the fact that relative to temperature measured every hour, NO₂ is poorly measured with just one NO₂ value for every 4 weeks. Removing the adjustment for NO₂ did not change the results in any important way.

The children were not given any specific instruction on asthma medication use during the study. However, the use of reliever during the night and the number of puffs taken during the day was significantly negatively associated with lung function ($p < 10^{-6}$ in all models). This indicates that reliever use was greater on days with lower lung function and the reliever use did not on average return lung function to the mean by the time the measurement was made. Both measures of reliever use were weakly, though significantly, correlated with exposure to low temperature (r ranged from 0.03 to 0.16) with the severity measure at no lag having the most significant correlation ($p < 10^{-16}$). This could potentially explain why we do not see much short-term effect of temperature, with the children using relievers more when they were cold.

Limitations

It is of course possible to adjust for multiple testing using Bonferroni adjustment which would yield a cut-off p value of 10^{-5} . At this level 56 models are significant including the four presented in table 1; 55 of the significant models used the severity aggregator at lags and thresholds similar to those in table 1. Bonferroni's method assumes a degree of independence in the tests, which is not the case here. Even though there is a significant association at this high level of significance, this study is a hypothesis-generating data mining exercise, and future studies are needed to confirm our findings. The study consisted of an exhaustive search of the most plausible models for the association between lung function and indoor temperature. We have reported the best fitting of a large number of models, and thus the effect size and significance are likely to be overstated. However, as airway function and indoor temperatures have not been reported before, an exploratory approach was appropriate as an initial step. Our primary goal was to describe which metric of indoor temperature had the strongest association with lung function, rather than specifically examine the size of the effect.

A limitation of the study is the lack of exact personal temperature exposure. Personal temperature exposure will of course be a combination of indoor home temperature, outdoor temperature and other temperature exposures such as car and school. Outdoor temperature is most frequently used as a proxy of personal exposure in research as it is both readily available and covers wide area such as cities.⁷ However, recent research has shown that at colder temperatures, outdoor temperature is only weakly associated with indoor temperature. Therefore, research suggests “relating outdoor weather to human health should take into consideration how well outdoor conditions serve as indicators of indoor or personal weather exposure.”³⁶ When considering the implications of exposure to temperature, indoor exposure is more important, as it is the easiest to change and has the longest duration of exposure.⁹

Implications

Better metrics for measuring indoor temperature exposure will help guide estimates of health benefits that can be realised from improving the indoor environment. Using the current or previous day's

average temperature is not optimal for explaining the relationship between indoor temperature and lung function. Instead, we suggest measuring indoor temperature over a longer period and subject to a local threshold similar to those used for outdoor studies.

Key Messages

- ▶ Small changes in indoor temperature are associated with small changes in the lung function of asthmatic children.
- ▶ Exposure to temperatures below 12°C had the greatest effect on lung function.
- ▶ These effects were detectable at lags of up to 14 days.
- ▶ Bedroom temperature had a greater association with lung function than Living room temperature.

What is already known on this subject

- ▶ Low outdoor temperatures are associated with poorer respiratory health outcomes. This association is strongest over lags of up to 14 days and to temperatures below a locally dependent threshold.

What this study adds

- ▶ This study shows that for asthmatic children, the association of indoor temperature and lung function is greatest when temperature is measured over the preceding 12 days with a threshold temperature of 9–12°C.

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Contributors NP carried out the study, analysed the data, wrote the paper and approved the final version of the manuscript. He is the guarantor. RA, MK, PH-C, JC and MC conceptualised and designed the study and along with all authors participated in the analysis and interpretation of the data, revised the manuscript critically for important intellectual content and approved the version to be published.

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Competing interests None.

Patient consent Obtained.

Ethics approval Ethical approval for the heating study was obtained from the University of Otago and the regional ethics boards of each of the five centres.

Provenance and peer review Not commissioned; externally peer reviewed.

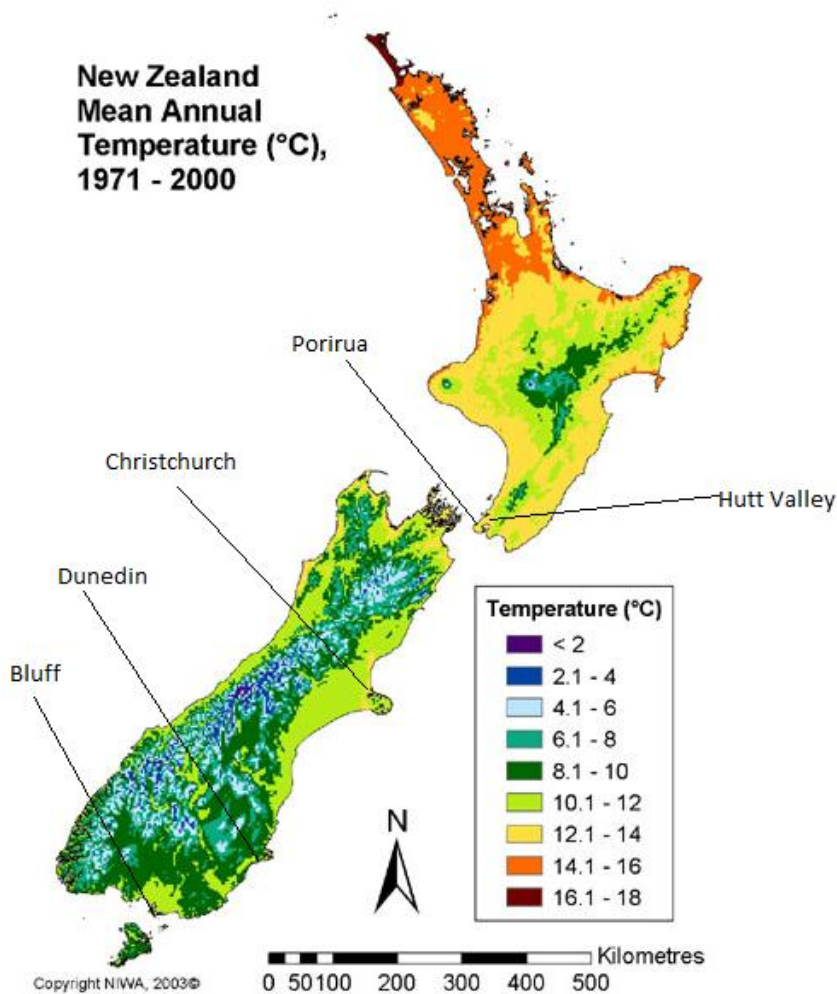
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Appendix 1

New Zealand Fact file.



Mean Daily Minimum Air Temperature 1971-2000 (°C) in study centres

Centre	June	July	August	September
Porirua*	7.2	6.3	6.7	7.9
Hutt Valley*	7.2	6.3	6.7	7.9
Christchurch	2.3	1.9	3.2	5.2
Dunedin	4	3.1	4.2	5.9
Bluff	1.9	1	2.2	4

*Porirua and The Hutt Valley are in the same region and hence have the same temperature data.

Map and Data courtesy of NIWA (National institute of Water and Atmosphere) New Zealand,

The New Zealand climate is a temperate maritime one (a little bit warmer) but similar to the British isles, or the North-eastern coast of the United States. Also because it is in the southern

hemisphere winter is June, July and August and temperatures are generally higher in the north.

New Zealand is largely temperate; in the study area it is slightly warmer and windier in the lower North Island study areas of Porirua and the Hutt Valley than the cooler temperate climates in the South Island centres of Christchurch, Bluff and Dunedin.

Most areas of New Zealand have between 600 and 1600 mm of rainfall, spread throughout the year with a dry period during the summer. Over the in the lower north island winter is the wettest season, whereas for much of the South Island of New Zealand, winter is the season of least rainfall.

Mean annual temperatures range from 10°C in the south to 16°C in the north of New Zealand. The coldest month is usually July and the warmest month is usually January or February. In New Zealand generally there are relatively small variations between summer and winter temperatures.