

Does forced air heating exacerbate asthma?

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Asthma morbidity increases in the Canadian autumn coincident with the beginning of home heating. To test the hypothesis that forced air heating exacerbates asthma, 51 subjects were randomly assigned to one of two groups: the intervention (off-on₂) group was instructed to keep their home forced air heating off from September 9 to 17 and then to set the thermostat to 22° C from 8 PM to 8 AM between September 18 and 25. The concurrent control (off-off₂) group was instructed to keep their forced air heating off during the entire study period. Forty-eight of the 51 subjects randomized recorded morning peak flows, asthma symptoms, and bronchodilator use during the study period. For the intervention group, results before compared with during home heating, respectively, were as follows: mean nocturnal awakenings 0.36 versus 0.36; any breathing difficulties on awakening 82% versus 73%; mean morning peak flows 406 lpm versus 409 lpm, a difference of 3.3% $\pm$ 6.7% (SEM), $p = 0.6$. There was a 95% probability that the true change in peak flows with forced air was less than 2%. For the control group, the comparable results were as follows: mean nocturnal awakening 0.40 versus 0.47; breathing difficulties on awakening 40% versus 47%; mean morning peak flows 400 lpm versus 399 lpm ($p = 0.9$). The commencement of forced air heating did not exacerbate asthma in this clinical population and is therefore unlikely to be an important problem for most people suffering from asthma. (J ALLERGY CLIN IMMUNOL 1992;90:803-7.)

Key words: Asthma, environmental pollution

Seasonal increases in asthma severity have been documented in several countries.¹⁻¹⁰ Suggested etiologic factors include seasonal variations in pollen, fungi, dust mite antigen, ambient pollution, weather, transmission of viral infections, and commencement of home heating. The role of home heating is the basis of this report.

In Ontario, Canada, hospital admissions for asthma increase approximately 40% during September and October, when most people begin home heating. Forced air heating may render molds, dust, and other allergens airborne by propelling them from heating ducts and by creating air currents throughout the house. Alternatively, the heating of dust particles in the furnace could produce irritating combustion products. Insufficient evidence exists to determine whether forced air heating exacerbates asthma. In a study of 20 children in Vancouver, Canada, 10 were allocated

to receive vinyl mattress and pillow covers, improved bedroom cleaning, and have their forced air heating ducts blocked off.¹¹ After 1 month, these subjects had significantly fewer symptoms and better peak flows than a control group that did not have allergen control carried out. In all cases, pets were to be removed from the home if the child's relevant skin test was positive. The isolated effect of forced air heating was not tested. A large Canadian cross-sectional study of children 5 to 8 years old reported that forced air heating was more common among children with current asthma ($n = 634$) than asymptomatic children ($n = 9,207$), 47% versus 38%, $p < 0.01$.¹² However, when the results were adjusted—for age, race, sex of child, parental education, sex of questionnaire respondent, region of residence, crowding, dampness, gas cooking, and environmental tobacco smoke—the odds ratio was not significant, 1.12 (95% confidence interval 0.88, 1.42).

To clarify this issue, the present study uses an experimental design to test the hypothesis that forced air heating exacerbates asthma.

MATERIAL AND METHODS

Study design

A non-blinded randomized concurrently controlled trial was performed. The intervention was the initiation of forced

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TABLE I. Distributions of host and environmental characteristics at the beginning of the study for the intervention (off₁-on₂) and control (off₁-off₂) groups

Characteristics	Forced air heating exposure group	
	Intervention	Control
Host	(n = 22)	(n = 27)
Age, mean	41	38
range	6-73	7-60
Sex, % male	36	41
Allergies* (%) dust	73	82
molds	55	63
pollens	64	52
animals	64	52
Current smoking (%)	32	35
Regular medications† (%)		
Beta-agonists	73	65
Other bronchodilators	27	27
Inhaled corticosteroids	55	62
Other prednisone	18	4
Cromolyn or nedocromil	9	8
Home environment		
Passive smoke, % exposed	9	15
Furry pets, % present	50	39
Mold or mildew, % present	59	56
Central air conditioning, % used within 2 months	41	67
Furnace fan, % used within 2 months	32	22
Duct cleaning, % within 6 months	32	8

*Defined by a positive response to the question: "Has a doctor ever said that you have one or more allergies (to the following) . . . dust . . . pollen . . . mold . . . animals?"

†Medications missed for less than 4 days during the entire study period.

air home heating, and the primary outcome variable was morning peak flows. Ethical approval was obtained at the institution of the principal investigator.

Study population

Between 1988 and 1991, patients presenting with physician-diagnosed asthma to emergency departments at the Ottawa General and Ottawa Civic Hospitals Canada, were asked to complete questionnaires about asthma control. For the present study, all those who reported having had forced air heating and had agreed to be contacted by our study group were telephoned. They were asked to attend a group meeting where a detailed explanation of the study procedure was provided, questions were answered, and informed consent was obtained. Subjects were excluded at the initial visit if they could not control their heating (e.g., tenants) or were judged unable to perform peak flows or complete a daily diary satisfactorily. Reported sensitivity to forced air heating was neither an inclusion nor an exclusion criterion.

Characterizing the subjects

During the information and recruitment meetings before the study, subjects were asked to complete questionnaires regarding age, sex, allergies, and indoor exposures to pets,

cigarette smoke, dampness, and molds. Home heating systems were characterized as to the use of furnace fans, central air conditioning, and duct cleaning.

The intervention

During the information and recruitment meeting, subjects were randomly allocated to one of two groups: the intervention (off₁-on₂) group was instructed to keep their forced air heating systems off from September 9 to 17 inclusive (period 1) and then to set their home thermostat to 22° C between 8 PM and 8 AM daily until September 25 (period 2). The concurrent control (off₁-off₂) group was instructed to keep their forced air heating off between September 9 and 25 (periods 1 and 2). At the initial meeting, subjects were asked if they had already begun heating during the autumn season. Whether or not the furnace came on during each study day was recorded in the diary.

Outcome measures

The primary outcome measure was morning peak flows. Subjects were instructed to keep an Assess Peak Flow meter (Healthscan Products Inc., Cedar Grove, N.J.) at the bedside and record the best of three attempts immediately on awakening before the use of asthma medications. Results

TABLE II. Asthma symptoms, peak flows, and bronchodilator use during each time period for both the intervention and control groups

Indicators of asthma severity	Forced air heating exposure group			
	Intervention (n = 22)		Control (n = 26)	
Morning peak flows, liters per minute (SD)	Off ₁ 406 (146)	On ₁ 409 (147)	Off ₁ 400 (173)	Off ₂ 399 (169)
Nocturnal awakenings, mean (SD)	0.36 (0.43)	0.36 (0.44)	0.40 (0.66)	0.47 (0.75)
Morning symptoms, percent	82	73	92	85
Bronchodilator use, mean puffs per day (SD)	3.2 (2.8)	3.1 (2.8)	4.1 (3.2)	4.5 (3.4)

were recorded in a daily asthma diary. Secondary outcome variables included breathing difficulties overnight recorded as 0, none; 1, awakened once by breathing; 2, awakened twice by breathing; 3, awakened three or more times. Breathing difficulties on awakening were recorded as none, slight, moderate, or severe. Daily use of asthma medications was also recorded.

Statistical analysis

A paired one-tailed *t* test was used to assess the difference between mean morning peak flows before and after heating in the intervention group. A nonpaired one-tailed *t* test was used to compare the off₁ versus on₁ difference in the intervention group to the off₁ (September 9 to 17) versus off₂ (September 18 to 25) difference in the control group. The first comparison tested for an association with home heating, whereas the second tested the same association between peak flows and home heating accounting for temporal changes unrelated to heating.

The analysis was verified by a doubly multivariate repeated measures analysis that simultaneously tested for group, time, and a group-by-time interaction.¹³

RESULTS

Fifty-one subjects consented to participate and completed the initial questionnaire. Two subjects said they had mailed in their results, but they were not received. Because of the nature of the blinding, these two subjects could not be classified by study group and were excluded from analysis. Twenty-two of the remaining 49 subjects were randomized to the intervention group and 27 to the control group. One of these 27 who completed the baseline questionnaire (data presented in Table I) did not complete the daily diary (data presented in Table II). Overnight minimum ambient temperatures ranged between 2° and 8° C. Only one subject had commenced forced air heating in the past 6 weeks for approximately 2 to 3 days. In the intervention group, one subject did not think that his fur-

nace came on within the first 3 days of the "on" period. Otherwise furnaces were off and on as anticipated.

Randomization achieved comparability on host characteristics (Table I) except for prednisone use. The percentage difference appeared large (18% in the intervention group vs 4% in the control group), but the absolute difference was small (4 vs 1 subject) and because of this were not significant at *p* < 0.05 with use of a Chi-square statistic. Home environmental characteristics differed by more than 15% for the use of central air conditioning, which was more frequent in the control group, and for duct cleaning within 6 months before the study, which was greater in the intervention group. Morning peak flows were similar between the intervention and control groups when forced air heating was off (Fig. 1 and Table II). In the intervention group, the average difference between the off₁ and on₁ periods was an improvement of 3.3 lpm ± 6.7 (SE), *p* = 0.6. There was a 95% probability that the true change in mean peak flow with forced air heating was less than 8 lpm or 2% of the mean peak flows. The difference between the off₁ and on₁ periods in the intervention group (3.3 lpm) was not statistically different from the difference between the off₁ and off₂ periods in the control group (−0.9 lpm). The omnibus test from the repeated measures analysis of variance (ANOVA) confirmed these results; there was no group-by-time interaction. Mean nocturnal awakenings, morning symptoms, and bronchodilator were also not significantly different, consistent with the peak flow measurements.

To investigate the presence of host or environmental characteristics that might have influenced the response to forced air heating, changes in peak flow between the off and on periods were stratified by the following variables: heating ducts cleaned within 3 to 6 months; physician-diagnosed allergies to mold, dust, or ani-

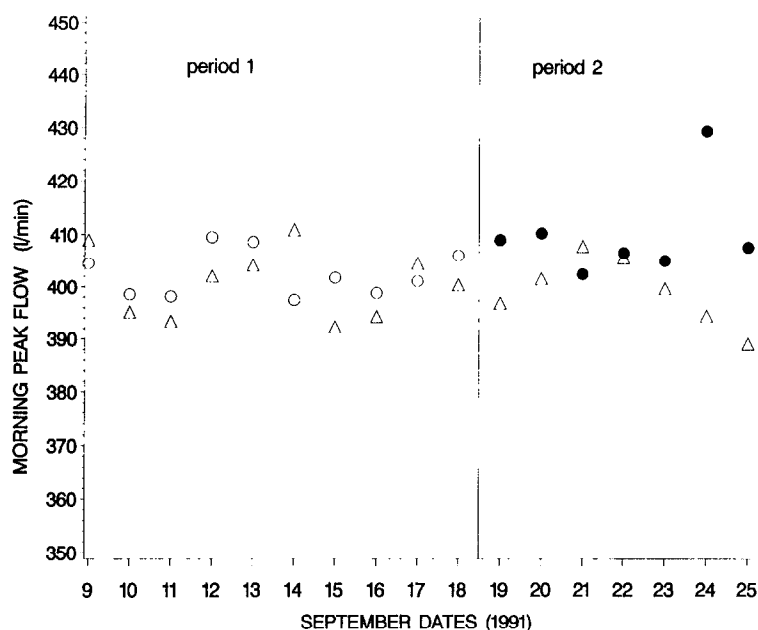


FIG. 1. Daily mean peak expiratory flows recorded on awakening each morning. ○ represents mean values for group 1 when furnace was off; ● represents group 1 when furnace was on; △ represents group 2 (furnace is always off).

mals; the presence of an allergy to animals plus a pet in the house; and furnace fan and air conditioning operating during the last 2 months. Sample sizes were too small to accurately assess the differences in many of these variables, but no trends were seen that would support the hypothesis that forced air heating exacerbates asthma. For example, of 16 patients in the intervention group who reported allergies to dust, peak flows were 422 lpm during the off period and 431 during the on period. Respective values for the control group were 379 lpm and 378 lpm.

DISCUSSION

Forced air heating could theoretically exacerbate asthma by increasing airborne concentrations of antigens from dust mites, molds, or pets. Alternatively, respiratory irritants could be produced by the combustion of dust particles on the furnace heating plate. However, the commencement of forced air heating was not observed to exacerbate asthma in our study population. Therefore methodologic issues that could have reduced the observed estimate of effect must be considered.

Two potentially important biases were prevented by the study design: (1) Assigning exposure prevented an artifactual relation whereby changes in asthma symptoms would prompt subjects to alter their home heating. (2) The concurrent control group accounted for changes in asthma severity temporally related to, but not caused by, home heating. The sensitivity of this

study to detect an association between asthma and home heating was enhanced by several features: First, responsive subjects were chosen. They had visited the emergency department within 24 months, awakened because of breathing on average every third night, and used an average of four bronchodilator inhalations daily. Second, subjects were potentially exposed to home heating on 7 consecutive nights. Exposure was verified both by ambient temperatures (which were low enough to trigger the thermostat) and by the daily diary question: whether they believed the furnace came on during previous nights. Third, to enhance the ability to detect changes caused by nocturnal exposure, overnight and morning symptoms and morning peak flows were assessed. Fourth, the study had enough power to detect peak flow changes greater than 2%. Fifth, analysis of potentially susceptible subgroups, that is, those with dust and mold allergies, revealed no trends to suggest an association. Thus we conclude that forced air heating does not significantly aggravate asthma symptoms in this clinical population. Theoretically, forced air heating could exacerbate asthma, but it is sufficiently infrequent to be detected either by this experimental design or by a previous large cross-sectional design in a general population.¹² Dust mites are concentrated in sofas, carpets, and beds.¹⁴ Carpet vacuuming produces large increases in airborne dust mite antigen that returns to low levels within 20 minutes after the disturbance.¹⁵ Although dust mites may exacerbate asthma, the dis-

turbance created by forced air heating may not change the inhaled concentration enough to be clinically relevant. Indoor molds may be found in particularly high concentration in areas of water damage or condensation.¹⁶ Reports of indoor dampness and molds by household residents have been associated with increased respiratory symptoms and asthma, but objective measurements have often failed to confirm this association.¹⁷ Similar to dust mites, mold spores increase greatly with household activity,¹⁸ but forced air heating may not have the same effect.

In summary, although many potential mechanisms exist whereby forced air heating could provoke asthma, empirical observation fails to support this. Reasons include lack of allergenicity or insufficient concentration of forced air heating contaminants. The implications are that subjects suffering from asthma should not generally be dissuaded from forced air heating, and second, that forced air heating is unlikely to account for the autumn increase in asthma admissions.

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REFERENCES

1. Salvaggio J, Seabury J, Schoenhardt EA. New Orleans asthma. V. Relationship between Charity Hospital admission asthma rates, semiquantitative pollen and fungal spore counts, and total particulate aerometric sampling data. *J ALLERGY CLIN IMMUNOL* 1971;48:96-114.
2. Goldstein IF. Weather patterns and asthma epidemics in New York City and New Orleans, U.S.A. *Int J Biometeorol* 1980;24:329-39.
3. Weiss KB. Seasonal trends in U.S. asthma hospitalizations and mortality. *JAMA* 1990;263:2323-8.
4. Murray AB, Ferguson AC, Morrison B. The seasonal variation of allergic respiratory symptoms induced by house dust mites. *Ann Allergy* 1980;45:347-50.
5. Mao Y, Semenciw R, Morrison H, Wigle DT. Seasonality in epidemics of asthma mortality and hospital admission rates, Ontario, 1976-86. *Can J Public Health* 1990;81:226-8.
6. Bates DV, Baker-Anderson M, Sizto R. Asthma attack periodicity: a study of hospital emergency visits in Vancouver. *Environ Res* 1990;51:51-70.
7. Khot A, Burn R, Evans N, Lenney C, Lenney W. Seasonal variation and time trends in childhood asthma in England and Wales 1975-81. *BMJ* 1984;289:235-7.
8. Knot A, Burn R. Seasonal variation and time trends of deaths from asthma in England and Wales 1960-82. *BMJ* 1984;289:233-4.
9. Ayres JG. Trends in asthma and hay fever in general practice in the United Kingdom 1976-83. *Thorax* 1986;41:111-6.
10. Pedersen PA, Weeke ER. Seasonal variation of asthma and allergic rhinitis. Consultation pattern in general practice related to pollen and spore counts and to five indicators of air pollution. *Allergy* 1984;39:165-70.
11. Murray AB, Ferguson AC. Dust-free bedrooms of asthmatic children with house dust mite allergy: a controlled trial. *Pediatrics* 1983;71:418-22.
12. Dekker C, Dales R, Bartlett S, Brunekreef B, Zwanenburg H. Childhood asthma and the indoor environment. *Chest* 1991;100:922-6.
13. Schultz RW, Gessaroli ME. The analysis of repeated measured designs involving multiple dependent variables. *Res Q Exerc Sport* 1987;58:132-49.
14. Platts-Mills TA, Chapman MD. Dust mites: immunology, allergic disease, and environmental control. *J ALLERGY CLIN IMMUNOL* 1987;80:755-75.
15. de Blay F, Heymann PW, Chapman MD, Platts-Mills TAE. Airborne dust mite allergens: comparison of group II allergens with group I mite allergen and cat-allergen *Fel d 1*. *J ALLERGY CLIN IMMUNOL* 1991;88:919-26.
16. Burge H. Bioaerosols: prevalence and health effects in the indoor environment. *J ALLERGY CLIN IMMUNOL* 1990;86:687-701.
17. Strachan DP, Flannigan B, McCabe EM, McGarry F. Quantification of airborne moulds in the homes of children with and without wheeze. *Thorax* 1990;45:382-7.
18. Hunter CA, Grant C, Flannigan B, Bravery AF. Mould in buildings: the air spora of domestic dwellings. *Int J Biodegradation* 1988;24:81-101.

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