

INDOOR ENVIRONMENTAL QUALITY IN HOMES OF ASTHMATIC CHILDREN ON THE ELSIPOGTOG RESERVE (NB), CANADA

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ABSTRACT

Objective. To inspect houses and analyze settled dust from 26 homes with asthmatic children in the Elsipogtog Reserve, New Brunswick, for contaminants known to be associated with respiratory symptoms. This pilot observational study was conducted in order to enable larger research into housing and health in Aboriginal communities.

Methods. Twenty-six homes were subject to an informed inspection and settled dust collection from the child's bedroom. The fine dust (< 300 µm) was analyzed for endotoxin, house dust mite and fungal glucan concentrations, as well as for building-associated fungi.

Results. The percentage of homes in this study that had mould damage was slightly higher than in much larger studies in other parts of Canada. Qualitatively, the causes of the water and mould damage were similar to those found in a larger study in nearby PEI. However, in a few cases, the type of damage found in Elsipogtog was at a more advanced stage. House dust mite allergens, endotoxin and fungal glucan concentrations in settled dust included values that have been associated with increased respiratory symptoms. This selection of houses was, on average, cleaner than many homes studied in Canada.

Conclusion. Although the range of mould damage observed in this study was similar to that seen elsewhere in Canada, the underlying causes tended to reflect more serious maintenance problems. A systematic evaluation of mould damage, on a community-wide basis, is a useful process to set priorities for repair. (*Int J Circumpolar Health* 2005;64(1):77-85.)

Keywords: Aboriginal children, asthma, housing, glucan, endotoxin, mould

INTRODUCTION

Studies conducted worldwide have reported that people living in damp homes experience more respiratory symptoms and have elevated rates of asthma (1-3). It has also been reported in many studies that aboriginal children in Canada suffer a much higher prevalence of asthma and respiratory tract infections than the general population (4, 5). A recent survey by the Royal Commission on Aboriginal Peoples of Canada reported chronic asthma, or bronchitis, in over 19% of aboriginal Canadians under the age of fifteen (4). In Canada, many aboriginal communities are in boreal and arctic areas. In these locations, it is difficult for children to go outdoors for 3-6 months of the year, because of cold weather. Opportunities to stay indoors outside the home are generally limited in such areas. Indoor air quality contaminants, such as tobacco smoke, house dust mites and mould and bacterial contaminants, play an important role in asthma development, particularly in children (3).

Children are exposed to two categories of toxic compounds in indoor air: allergens (3) and inflammatory compounds, such as bacterial endotoxin and fungal glucan, both of which are cell wall components (6). Interactions are also possible when there are elevated exposures to both. For example, as noted, house dust mites produce allergens that are known to cause asthma (3). When houses have more versus less endotoxin in dust, dust mite asthmatic patients report more symptoms and use more drugs for symptom relief (7). Fungal glucan in settled

dust is thought to have the same effect and is independently associated with various upper respiratory symptoms in housing (8). Mold growth indoors increases glucan exposure. Studies done in Canada, the United Kingdom and Finland have shown that the area of moisture and mould damage found in homes is a good predictor of the likelihood of respiratory health problems (9-12). Large cohort and prospective studies of home dampness contaminants and health of adults, children and infants have been done, or are nearing completion, in Canada with more than 1000 homes studied in the City of Wallaceburg (Ontario) and in the Province of Prince Edward Island, as well as smaller studies in other locations.

In cooperation with several aboriginal communities, the New Brunswick Lung Association conducts asthma camps for their children. For some children in camp in the year preceding the present study, lung function improvements were studied when the children arrived and departed the camp. This was done under the supervision of physicians at the teaching hospital in Saint John, New Brunswick (Beveridge et al., unpublished data). The homes of a subgroup of these children from Elsipogtog (formerly Big Cove), New Brunswick, were selected for this study of indoor air quality. This study included home inspections and measured some major dampness contaminants according to protocols adapted from research studies conducted in Wallaceburg (13) and an ongoing study in PEI (14). At the time this study was done, no research studies of housing quality had been done in any aboriginal community in Canada.

This study was conducted as an observational pilot study, in order to determine if there existed any major differences in indoor air quality between the homes on the New Brunswick First Nations Reserve of Elsipogtog and homes that had been examined in previous studies in PEI and Wallaceburg (Ontario). The expected outcome of the work was to provide some information on how the houses examined compared to homes in those areas.

METHODS

The Elsipogtog First Nations reserve is located in the northern boreal forest of northern New Brunswick [lat. 46.59017, long. -64.99602] and has a maritime continental climate. The mean annual temperature is 5.1°C (January -8.9°C, July 18.6°C), with average precipitations of 349 cm snow and 865 mm rain.

Approval was sought from the Chief and Band Council (an elected body of, in this case, a Mi'kmaq aboriginal community), their medical advisors and the regional Medical Officer of Health for Health Canada. Ethical approval was obtained from the Institutional Review Board (IRB) of Carleton University. A person from the community contacted each of the fifty-three families that had participated in the asthma camp program. Some families had moved to other houses since the asthma camps and some lived in apartments not appropriate to the assessment planned. In the end, 26 families were accepted in the study. The families signed an informed consent letter ap-

proved by the IRB. Each family received an individual report explaining the results on their home, along with information on how their house compared to the others studied in the community and any recommendations to improve their indoor environmental quality. The Chief and Band Council also received a summary report of the information without identifiers. No honoraria were paid for participation.

A team of trained industrial hygienists and housing inspectors did all of the sample collections and home inspections. For the inspections, they completed survey forms developed for the PEI study (14) and adapted for First Nations homes. Visible mould, areas of water damage, cleaning intensity and other variables, were recorded. Additions to the documentation in this case included administrative information on the ownership of the house. The major difference in process compared to research studies was that the inspections were conducted jointly by Health Canada and Band Council housing inspectors.

Settled dust samples were collected over 1 m² in the asthmatic child's bedroom. This was done using a filter (Midwest Filtration Company, Cincinnati, OH; # FAB0703006PS) fitted to the hose of a canister vacuum cleaner. This sampling protocol was used in the PEI and Wallaceburg studies and has been demonstrated to be quantitative for allergens (15). Samples were transported and stored under air-dry conditions and the dust was sieved to < 300 µm and > 300µm (stainless steel test sieve model # 50, Fisher Scientific, Ottawa) and weighed using a Santorius A120-S 4 balance (0.1 mg).

Twenty-four of the samples had adequate weights of dust < 300 µm that could be further processed. Samples were analyzed for endotoxin, house dust mite allergens and beta 1, 3-D-glucan. If there was insufficient dust weight to provide a sample for all of the analyses, priority of the tests was assigned according to the order above. Endotoxin concentration was analysed using the *Limulus* amoebocyte lysate (LAL)

method, according to the manufacturer's instructions (Associates of Cape Cod; Falmouth, MA). Glucan concentration was analyzed using the Factor G-based LAL assay of Foto et al. (16). The dust mite allergens, Der f1 (*Dermataphagoides farinae*) and Der p1 (*Dermataphaoides pteronyssimus*), were extracted with borate buffer and their concentrations were determined with a monoclonal antibody-based enzyme im-

Table I.

Range and variation of exposure to dust, allergens, endotoxin, glucan and visible mould damage expressed as units/m² (all samples collected over 1 m²)*

House	Total dust (mg/m ²)	<300 µm (µg/m ²)	Der f1 (µg/m ²)	Der p1 (µg/m ²)	Endotoxin (µg/m ²)	Glucan (µg/m ²)	Visible mold (m ²)
1	471.4	241.2	0.04	0.09	0.26		0.19
2	604	272.7	n.d.	n.d.	0.82	14.73	0.09
3	1260	660.1	19.64	0.26	0.99	50.17	n.d.
4	81.8	34.6			0.16		0.09
5	3426.8	595.9	13.95	31.46	1.49	166.85	0.46
6	1303.5	694.8	n.d.	n.d.	1.25	69.48	0.46
7	817.6	611.5	2.35	0.09	0.73		2.14
8	1105.4	781.9	0.08	n.d.	1.09	265.85	n.d.
9	169.3	44.9			0.11		n.d.
10	370.3	186.7	n.d.	n.d.	0.32	31.74	0.28
11	813.8	446.9	0.06	5.09	0.98	107.26	n.d.
12	110.2	40			0.07		0.09
13	14						3.90
14	387.8	145.5	0.01	n.d.	0.08		n.d.
15	535	56	0.00	0.06			n.d.
16	768.5	274	0.05	0.08	0.30	27.40	n.d.
17	259.2	51.4	0.02	n.d.			3.81
18	605.2	303.9	0.09	0.39	0.55	63.82	0.09
19	1432	730.2	0.35	0.74	1.10	357.80	0.09
20	1636.7	901.6	2.95	0.04	1.80	60.41	5.95
21	4.1						0.19
22	437.5	259.7	0.01	n.d.	0.78	21.56	0.37
23	412	244.8	0.01	0.03	1.13	10.77	18.58
24	2115.5	1635.7	1.08	0.18	7.36	1078.56	0.93
25	1236.9	403	0.11	0.71	1.29	181.35	0.28
26	1348.7	450	0.09	0.04	0.54	211.50	0.19

* n.d. = below limit of detection; blank values mean that there was insufficient sample and the test was not done

munoassay from Indoor Biotechnologies (Charlottesville, VA), according to the method of Chapman et al. (17). Using the total weight of dust < 300 µm for each home, the amount of each toxin over 1 m² (the collection area) was calculated and is reported as exposure.

Any samples with remaining dust (sixteen total) were cultured for viable fungi and yeasts, by dilution plating on a medium suitable for moderate xerophiles (relating to water activity and appropriate for fungi that grows on most building materials) (15, 18). Each sieved dust sample was diluted two-fold in 0.01% Tween 80 solution and plated in triplicate on both Dichloran-glycerol agar (DG-18; 15) and Littman Oxgall agars (containing benomyl and streptomycin; Oxoid). The plates were incubated at 25°C for 7-10 days. Colonies were then enumerated and transferred to MEA (2% malt extract) and CZ (Czapek-Dox) agar plates for identification (15).

RESULTS

Visible mould was observed in 19 of the 26 homes (Table I). Four homes, or 15% of the sample set, had visible mould on more than 1-2% of the house floor area. This included one home with serious mould damage (18.58 m²) - over three times the next highest value. According to the investigators, most mould damage in these homes was associated with basement leaks, due mainly to improper drainage, improper waterproofing and roof leaks. These were rare in PEI and Wallaceburg (12, 20). The Elsipogtog

homes also had mould due to condensation (e.g. window frames) and "household mould" (bathroom surfaces, refrigerator pans, toilet tanks, etc.) caused by inadequate ventilation. This was fairly common in the other communities, but there were examples in the present study where mould and moisture damage due to condensation was more severe than in the other, larger studies.

Cleanliness, defined as the weight of dust sieved to < 300 µm, was also used as an important variable when considering exposure to allergens. The geometric mean weight of fine dust was 266.9 mg/m² ranging from below the limit of detection (0.1 mg) to 1635.7 mg/m² (Table I).

High levels of dust mite allergens (> 10 µg/g) were measured in three of the homes (one home had both high Der f1 and Der p1). Total dust mite allergen concentration (µg/g) was significantly, but moderately, correlated with mite allergen exposure (µg/m²) by Pearson correlation (0.624, *p* > 0.006).

Median endotoxin concentration was 1800 ng/g (530-4600 ng/g), with a geometric mean of 1940 ng/g. When expressed as exposure, the mean was just over 1000 ng/m² (Table I). There was no correlation between endotoxin concentrations and endotoxin expressed as exposure. Median glucan concentration was 190 µg/g (44-660), with a geometric mean of 172 µg/g, and an average value on an exposure basis ~ 160 µg/m² (Table I). As with endotoxin, there was no correlation between glucan concentrations and glucan expressed as exposure. Results for the contaminant exposures are summarized in Figure 1.

The species spectrum of viable fungi present in settled dust (data not shown) was qualitatively very similar to the spectra found in Wallaceburg (19) and PEI (Miller et al, unpublished data). Given the small sample size, no statistical tests were done.

DISCUSSION

The houses examined in this study were chosen on the basis of previous participation in a health study and cannot be considered representative of the entire community. Nor can this community be seen as completely representative of the entire aboriginal reserve population in Canada. However, the data presented by this study can be used to

describe and highlight some key risk factors for indoor air health.

Overall, very few differences were seen in the Elsipogtog homes with regard to dust contaminant concentrations and exposure, when compared to previous studies in other locations, and none of these were significant for the sample size (Table I, 9-12,14, 20-27). In studies by Hyndman (11), Haverinen et al. (9, 10) and the Canadian studies in Wallaceburg and PEI (12, 20), visible mould and water damage equal to 1-2% of the floor area was the approximate threshold for a detectable increase in the prevalence of respiratory health effects. In Wallaceburg, this threshold was exceeded in 16% of the homes (12, 20), which was not significantly different from the 15% found in Elsipogtog. Although

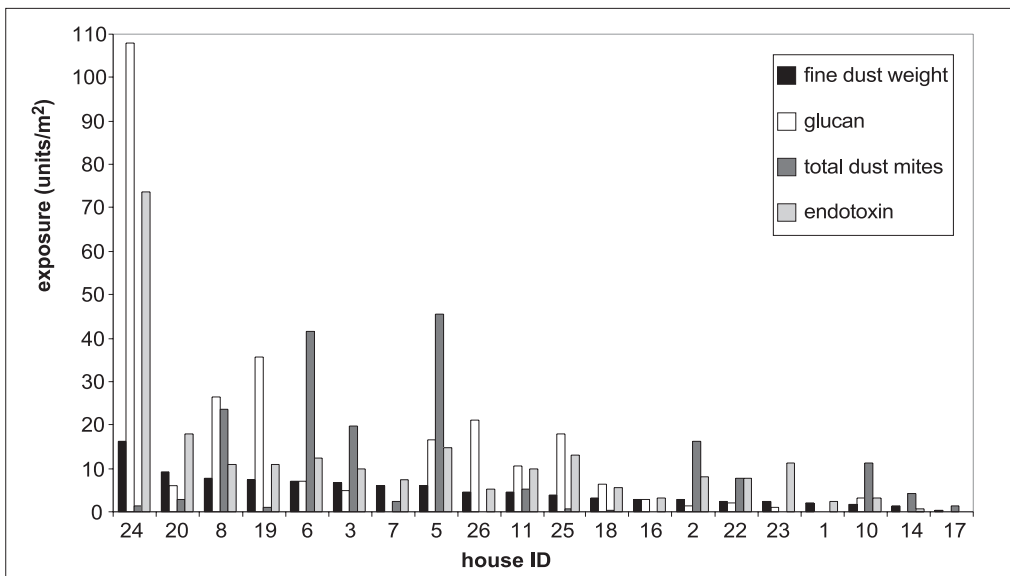


Figure 1. Contaminant burden for each individual house, ranked in order of cleanliness (fine dust weight). Fine dust (< 300 µm) is measured in 10⁻¹ g/m², glucan in 10⁻¹ µg/m², total dust mites (der f1 + der f2) in µg/m² and endotoxin in 10¹ µg/m². None of the contaminants were significantly correlated with one another (p > 0.10). The values for house #24 are 107.96 × 10⁻¹ µg/m² and 73.61 × 10¹ µg/m² for glucan and endotoxin, respectively. Houses 4, 9, 12, 13, 15 and 21 were not included in this figure, as they were missing two, or more, test values.

at least one, and possibly two, of the houses studied showed more mould than reported from ~800 homes in other communities in Canada (12, 20). Noting the small sample in the present study, we found that the range of visible mould damage observed was also similar to those in previous studies including Wallaceburg (0.04-3 m²; 65 homes) (20) and PEI (0.01-6.5 m²; 110 homes) (14). As for sources of mould, the prevalence of mould due to condensation (e.g. window frames) and "household mould" (bathroom surfaces, refrigerator pans, toilet tanks, etc.) in Elsipogtog homes matched that found in both PEI (14) and Wallaceburg (20). However, much of the observed mould damage in Elsipogtog also tended to be due to causes that could progress, if unattended, to possibly hazardous conditions within 1-2 years. This included examples of chronic water leaks, including in the roof and basement, and improper drainage, or waterproofing. As noted, occupants and the Band Council received a report detailing the problems requiring attention from a health perspective (as opposed to cosmetic issues). A major advantage of a quantitative study performed on a community basis is that repair priorities based on health can be established.

The average dust weight of the homes in Elsipogtog (266.9 mg/m²) was low compared to other studies. This is a good sign for health, because, in many studies, cleanliness has been identified as an important housing variable (21-24). Reduced respiratory symptoms have been shown to correspond with houses having > 500 mg/m² dust < 300 µm (24). By comparison, in a recent study of houses in Ottawa (25), many had dust burdens well in excess of this value, as did houses in inner city Seattle

(24). Cleanliness has a significant impact on health (24), due to particulate contaminants (i.e. glucans, endotoxins and dust mites), because exposure is usually due to room activity stirring up fine particulate dust into the air (28), where it can be inhaled. Since exposure to particulate contaminants results from a combination of settled dust load and contaminant concentrations, better correlations with health are obtained when both are known ("exposure"; Table I). This highlights the importance of collecting accurate measurements of settled dust on an area basis.

It was not more difficult to conduct a study of this type on a First Nations reserve, than in PEI, Ottawa, or Wallaceburg. Possible minor improvements were identified in the house inspection forms and improved training opportunities for the industrial hygienist inspectors were noted. The inspection process collected data against approximately 100 questions in a five-page form and additional pages for recording mould and moisture damage on a per room basis. On analysis, much of this information was not relevant to the health issues being studied, but was pertinent to the identification of repairs needed. It may be desirable, for example, to separate the two processes, in order to ensure that each is given proper emphasis. Data analysis was facilitated by a number of specific post-inspection discussions. In some cases, the interpretation of notes and drawings on the forms from a health perspective was not clear. The emphasis for both Health Canada, or, in some cases, Tribal health inspectors and housing inspectors, has been on identifying the remediation needs of the housing. The present study indicated that more training documenting

building damage, or the presence of contaminants, would make the process more efficient.

Based on these experiences, it will be possible to develop additional, more extensive studies on housing and health in other aboriginal communities elsewhere in Canada, as well as to make more informed recommendations on improving the quality of life for the inhabitants.

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REFERENCES

1. National Academy of Sciences. Damp Indoor Spaces and Health. National Academy Press, Washington, DC: 2004.
2. Dales RE, Zwanenburg H, Burnett R, Franklin CA. Respiratory health effects of home dampness and molds among children. *Am J Epidemiol* 1991b; 134:192-199.
3. National Academy of Science. Clearing the air: asthma and indoor air exposures. Washington DC: National Academy of Science Press, 2000.
4. Sin DD, Wells H, Svenson LV, Man SFP. Asthma and COPD Among Aboriginals in Alberta, Canada. *Chest* 2002; 121:1841-1846.
5. Jenkins A, Gyrokos T, Joseph L, et al. Risk factors for hospitalization and infection in Canadian Inuit infants over the first year of life – a pilot study. *Int J of Circumpolar Health* 2004; 61(1): 61-70.
6. Rylander R, Holt RPG. (1→3)-β-D-Glucan and endotoxin modulate immune response to inhaled allergen. *Mediat Inflamm* 1998; 7(2):105-110.
7. Michel O, Kips J, Duchateau F, et al. Severity of asthma is related to endotoxin in house dust. *Am J Resp Crit Care* 1996; 154:1641-1646.
8. Rylander R, Lin RH. (1,3)-beta-D-glucan: relationship to indoor air-related symptoms, allergy and asthma. *Toxicology* 2000; 152:47-52.
9. Haverinen U, Husman T, Vahteristo M, et al. Comparison of two-level and three-level classifications of moisture-damaged dwellings in relation to health effects. *Indoor Air* 2001; 11:192-199.
10. Haverinen U, Vahteristo M, Moschandreas D, Nevalainen A, Husman T, Pekkanen J. Knowledge-based and statistically modeled relationships between residential moisture damage and occupant reported health symptoms. *Atmos Environ* 2003; 37:577-585.
11. Hyndman SJ, Housing dampness and health amongst British Bengalis in East London. *Soc Sci Med* 1990; 30:131-141.
12. Miller JD, Dales RE, White J. Exposure measures for studies of mould and dampness and respiratory health. In: Johanning E (Ed.) *Bioaerosols, fungi and mycotoxins: Health effects, assessment, prevention and control*. Albany: Eastern New York Occupational and Environmental Health Center, 1999; 298-305.
13. Dales RE, Miller JD. Residential fungal contamination and health: microbial cohabitants as covariates. *Environ Health Persp* 1999; 107 (S3):481-483.

14. Foto M, Vrijmoed LLP, Miller JD, Ruest K, Lawton M, Dales RE. A comparison of airborne ergosterol, glucan and Air-O-Cell data in relation to physical assessments of mould damage and some other parameters. *Indoor Air* 2004 (in press).
15. Dillon HK, Heinsohn PA, Miller JD. Field guide for the determination of biological contaminants in environmental samples. Fairfax, VA: American Industrial Hygiene Association, 1996.
16. Foto M, Plett J, Berghout J, Miller JD. Modification of the *Limulus* Amebocyte Lysate assay for the analysis of glucan in indoor environments. *Anal Bioanal Chem* 2004 379:156-162.
17. Chapman MD, Heymann PW, Wilkins SR, Brown MJ, Platts-Mills TA. Monoclonal immunoassays for major dust mite (*Dermatophagoides*) allergens, Der p1 and Der f1, and quantitative analysis of the allergen content of mite and house dust extracts. *J Allergy Clin Immunol* 1987; 80:84-94.
18. Health Canada Fungal Contamination in Public Buildings: Health Effects and Investigation Methods. Health Canada, Ottawa. 2004; (http://www.hc-sc.gc.ca/hecs-sesc/air_quality/pdf/fungal_contamination.pdf).
19. Miller JD, Day JH. Indoor mould exposure: epidemiology, consequences and immunotherapy. *Can J Allergy & Clinical Immunology* 1997; 2:25-32.
20. Lawton MD, Dales RE, White J. The influence of house characteristics in a Canadian community on microbiological contamination. *Indoor Air* 1998; 8:2-11.
21. Brugge D, Vallarino J, Ascolillo L, Osgoode ND, Steinbach S, Spengler J. Comparison of multiple environmental factors for asthmatic children in public housing. *Indoor Air* 2003; 13:18-27.
22. Gehring U, Douwes J, Doekes G, et al. β -(1 \rightarrow 3)-glucan in house dust of German homes: housing characteristics, occupant behaviour, and relations with endotoxins, allergens and molds. *Environ Health Persp* 2001; 109:139-144.
23. Roberts JW, Clifford WS, Glass G, Hummer PG. Reducing dust, lead, dust mites, bacteria and fungi in carpets by vacuuming. *Arch Environ Con Tox* 1999; 36:477-484.
24. Krieger JW, Takaro TK, Song L and Weaver M. The Seattle-King County Healthy Homes Project: A randomized, controlled trial of a community health worker intervention to decrease exposure to indoor asthma triggers among low-income children. *Am J Public Health* 2004 (in press).
25. Dugandzic R, Miller JD, Judek S, Frescura AM. Exposure measures of indoor and outdoor-derived contaminants in some urban and rural homes in Ottawa, Canada. (submitted).
26. Vijay HM, Dales RE, Kumar V, Abebe M. Prevalence of Dust Mite and Cat Allergen in Prince Edward Island (PEI), a Canadian Province With a High Frequency of Reported Asthma. *J Allergy Clin Immunol* 2001; 107: abstract 83.
27. Heinrich J, Gehring U, Douwes J, Richter K, Koch A, Bischof W, Fahlbusch B, Kinne RW, Wichmann HE. Pets and vermin are associated with high endotoxin levels in house dust. *Clin Exp Allergy* 2001; 31:1839-1845.
28. Ferro AR, Kopperud RJ, Hildemann LM. Source strengths for indoor human activities that resuspend particulate matter. *Environ Sci Technology* 2004; 38:1759-1764.

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