Indoor air quality risk factors for severe lower respiratory tract infections in Inuit infants in Baffin Region, Nunavut: a pilot study

Abstract Inuit infants have extremely high rates of lower respiratory tract infection (LRTI), but the causes for this are unclear. The aims of this study were to assess, in young Inuit children in Baffin Region, Nunavut, the feasibility of an epidemiologic study of the association between indoor air quality (IAQ) and respiratory health; to obtain data on IAQ in their housing; and to identify and classify risk factors for LRTI. Twenty houses in Cape Dorset, Nunavut with children below 2 years of age, were evaluated using a structured housing inspection and measurement of IAQ parameters, and a respiratory health questionnaire was administered. Twenty-five percent of the children had, at some time, been hospitalized for chest illness. Houses were very small, and had a median of six occupants per house. Forty-one percent of the houses had a calculated natural air change rate < 0.35 air changes per hour. NO2 concentrations were within the acceptable range. Smokers were present in at least 90% of the households, and nicotine concentrations exceeded 1.5 μg/m3 in 25% of the dwellings. Particulates were found to be correlated closely with nicotine but not with NO2 concentrations, suggesting that their main source was cigarette smoking rather than leakage from furnaces. Mattress fungal levels were markedly increased, although building fungal concentrations were low. Dust-mites were virtually non-existent. Potential risk factors related to IAQ for viral LRTI in Inuit infants were observed in this study, including reduced air exchange and environmental tobacco smoke exposure.

Practical Implications
Severe lower respiratory tract infection is common in Inuit infants. We found reduced air change rates and high occupancy levels in houses in Cape Dorset, which may increase the risk of respiratory infections. This suggests the measures to promote better ventilation or more housing may be beneficial. Further health benefits may be obtained by reducing bed sharing by infants and greater turnover of mattresses, which were found to have high levels of fungi.

Introduction
The rate of hospitalization for severe lower respiratory tract infection (LRTI), including bronchiolitis and pneumonia caused by respiratory syncytial virus (RSV), in Inuit and Alaska Native children in northern North America appear to be the highest ever reported, with rates of 306 per 1000 infants in Baffin Region, Nunavut and 249 per 1000 in the Yukon-Kuskokwim Delta region of Alaska (Banerji et al., 2001; Karron et al., 1999). Twelve percent of infants with LRTI who were admitted to the Baffin Regional Hospital in
Iqaluit, Nunavut, required intubation and mechanical ventilation, and re-admission rates were high in both regions (Banerji et al., 2001; Karron et al., 1999). Known risk factors for severe bronchiolitis, such as underlying cardiopulmonary disease or prematurity did not appear to be over-represented in these populations (Banerji et al., 2001; Karron et al., 1999). As overcrowding and exposure to environmental tobacco smoke (ETS) are problems in both Inuit populations and children in the developing world, and poverty and malnutrition are likely considerably less extreme in Inuit infants, other causes are needed to explain why severe LRTI appears to be so common in Inuit children (Bulkow et al., 2002; Koch et al., 2002; Orr et al., 2001; Selwyn, 1990).

Inuit and Alaska Native infants also appear to be at increased risk for permanent lung injury following severe LRTI. Alaska Native children have been reported to have an annual incidence of bronchiectasis four times higher than any previously recorded (Fleshman et al., 1968). Bronchiectasis in Yupik Eskimo children in the Yukon-Kuskokwim Delta was preceded by recurrent pneumonia in infancy and early childhood in 91% of cases (Singleton et al., 2000). Chronic lung diseases, including bronchiolitis obliterans and chronic atelectasis appear to be unusually common in native children in northern Canada following adenovirus bronchiolitis (Herbert et al., 1977). Known predisposing conditions for severe LRTI, such as cystic fibrosis and primary ciliary dyskinesia were not observed (Chernick and Kendig, 1990; Culman et al., 1999b; Fleshman et al., 1968). Immune function also appears to be normal, with no abnormalities reported in serum immunoglobulins, mannose-binding lectin, T-cell numbers or T-cell function (Culman et al., 1999a,b; Fleshman et al., 1968; Singleton et al., 2000).

Concentrations of various pollutants could be elevated in the Far North, possibly because of the relatively tightly sealed housing pattern required for energy efficiency in extreme arctic conditions. Irritant gases have been shown to impair airway defenses, including mucociliary clearance and immune function, and can increase the severity of viral LRTI by inducing chronic inflammation of the respiratory tract epithelium and narrowing of the small airways (Smith et al., 2000). To date, only one study has examined indoor air quality (IAQ) in Canadian arctic housing. It was performed in old housing, much of which was retrofitted or replaced, and health outcomes were not examined (Appin Associates and Yassi, 1991).

The purposes of this pilot study were to characterize indoor air in the houses of Inuit infants, to determine whether significant risk factors for severe LRTI were present, and to assess the feasibility of epidemiologic studies on the association between IAQ and respiratory health in this region. We measured IAQ in a sample of houses of Inuit infants in Baffin Region, Nunavut, and performed a preliminary analysis of the relationship between IAQ and rate of LRTI.

**Methods**

Inuit families including at least one child < 2 years of age, living in Cape Dorset, Nunavut, were eligible for inclusion. Houses were selected by the Nunavut Housing Corporation (NHC) personnel, in conjunction with the Cape Dorset Housing Council. Families, which provided informed consent, received the American Thoracic Society children’s respiratory questionnaire (ATS-DLD-C), slightly modified for use in Nunavut, translated and administered by a program officer (JT) provided by the NHC (Ferris, 1978). A detailed home inspection was carried out by a trained home inspector, also provided by the NHC (BW), using a home inspection protocol developed by the Air Health Effects Division, Health Canada for research in southern Canada, and modified by the NHC for use in Nunavut (Foto et al., 2005). All of these houses are owned and maintained by the NHC, and the home inspector was familiar with the homes, their construction and maintenance. The field study was carried out in the homes of 20 Inuit infants, during extreme cold weather conditions between January and March 2003.

Nitrogen dioxide (NO₂), nicotine, relative humidity (RH) and temperature were monitored simultaneously over 7 days. NO₂ was sampled using two-sided Ogawa™ passive diffusion samplers (Ogawa™ Co, Pompano Beach, FL, USA), and analyzed by ion chromatography (Gilbert et al., 2003). Airborne nicotine was sampled by passive monitors using a sodium bisulfate-treated filter and analyzed by gas chromatography (Hammond and Leaderer, 1987). RH and temperature were recorded using ACR SmartReader Plus data loggers (ACR Systems, Inc., Surrey, Canada). Carbon dioxide (CO₂) was monitored over 24 h using a Yes 206 data logger (YES Environment Technologies Inc., Delta, Canada) and the mean value was used for statistical analysis. Airborne particulate matter was measured over 1 min with a Met One GT-321 laser particle counter (MET One Instruments Inc., Grants Pass, OR, USA). Unfortunately, this device was unable to determine the mass of particles with an aerodynamic diameter < 2.5 μm (PM2.5). The device sampled air over 1 min at a flow of 2.83 l/min and displayed the total number of particles sampled, and particle distribution, by size.

Air leakage was determined using blower door tests, performed using the Natural Resources Canada (NRCan) EnerGuide for Houses rating protocol, using the Canadian General Standards Board (CGSB) 149.10 test protocol (Canadian General Standards Board, 1986). The natural air change rates were determined using the Brookhaven National Laboratories (BNL) AIMS tracer gas technique (Dietz et al., 1982).
Settled dust samples were collected in the mattress where the child normally slept as well as on the living room floor. The floor samples were collected over a 1-m² floor area using a filter (# FAB0703006PS; Midwest Filtration Company, Cincinnati, OH, USA) fitted to the hose of a Shop Vac canister vacuum cleaner (Shop Vac Corp., Williamsport, PA, USA). This sampling protocol has been demonstrated to be quantitative for allergens (Dillon et al., 1996). Samples were transported and stored under air dry conditions and the dust was sieved to <300 and >300 μm (stainless steel test sieve model # 50; Fisher Scientific, Ottawa, Canada) and weighed using Santorius A120-S 4 (0.1 mg) (Data Weighing Systems Inc., Elk Grove, IL, USA).

The dust was analyzed for endotoxin, house dust-mite allergens, 1,3-β-D-glucan and viable fungi (limited by the amount of dust available). If there was insufficient sample to perform all the analyses, they were performed in the above-given order. Endotoxin was analyzed by the limulus amoebocyte lysate (LAL) method according to the manufacturer’s instructions (Associates of Cape Cod, Falmouth, MA, USA). Glucan was analyzed by the Factor G-based LAL assay of Foto et al. (2004). The dust-mite allergens Der f 1 (Dermatophagoides farinae) and Der p 1 (Dermatophagoides pteronyssimus) were extracted with borate buffer and determined with a monoclonal-based enzyme immunoassay from Indoor Biotechnologies (Charlottesville, VA, USA) according to the method of Chapman et al. (1987). Measurement of 1,3-β-D-glucan concentration and microscopic examination of other dust-mite species was also carried out (details available on request).

Culturable (viable) fungi and yeasts were determined by dilution plating on media suitable for fungi and yeasts. The sieved dust samples were diluted in 0.01% Tween 80 solution and plated in triplicate on DG-18 agar (Difco; BD Diagnostics, Sparks, MD, USA) and Littmanan Oxgall agar (containing benomyl and streptomycin; Oxoid Ltd., Basingstoke, UK) in triplicate in two dilutions (Dillon et al., 1996). The plates were incubated at 25°C for 7–10 days, colonies were enumerated and the fungal colonies transferred to malt extract agar and Czapek-Dox agar plates for identification (Difco) (Dillon et al., 1996).

The study was conducted in consultation with two local Inuit associations, the Qikiqtani Inuit Association (Iqaluit, Nunavut) and the Nunavut Tunngavik Inc (Iqaluit, Nunavut), as well as the Mayor, town council, and the local housing council in Cape Dorset. Ethical approval was obtained through the Research Ethics Board at the Children’s Hospital of Eastern Ontario and a Nunavut Research Institute License was obtained from the Nunavut Research Institute (Iqaluit, Nunavut).

Data were analyzed using Pearson’s correlation analysis and logistic regression (SPSS, version 11). A probability level of <0.05 was considered statistically significant.

Results

The 20 houses included in the study comprised approximately one-third of dwellings with children under 2 years of age in Cape Dorset, based on census data indicating that there were 30 births in Cape Dorset in 1996 (Statistics Canada, 2002). The 20 infants and children had a median age of 13 months (range 2–25 months). Nine (45%) of the infants were male. Five (25%) of the infants had been hospitalized for chest illness before 2 years of age, and these infants had a total of 13 hospitalizations for chest illness between them (Table 1).

Houses were single-story, and were raised above ground level. These houses were small in terms of living space area (46–130 m²), and lacked basements that add to the total volume of southern Canadian homes. The mean indoor volume was 233 m³. In contrast, the volume of most small houses in southern Canada is 350–400 m³, and is 400–600 m³ for medium houses (D. Fugler, pers. comm.). Seventy-five percent of the

Table 1 Health outcomes (n = 20)

<table>
<thead>
<tr>
<th>Health outcome</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hospitalization for chest illness before 2 years of age</td>
<td>5</td>
<td>25</td>
</tr>
<tr>
<td>Number hospitalized twice</td>
<td>2</td>
<td>10</td>
</tr>
<tr>
<td>Number hospitalized three times</td>
<td>3</td>
<td>15</td>
</tr>
<tr>
<td>Hospitalization in a southern pediatric hospital (Montreal or Ottawa) before 2 years of age</td>
<td>2</td>
<td>10</td>
</tr>
<tr>
<td>Number hospitalized outside Nunavut once</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Number hospitalized outside Nunavut twice</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Bronchiolitis before 2 years of age</td>
<td>7</td>
<td>35</td>
</tr>
<tr>
<td>Pneumonia before 2 years of age</td>
<td>4</td>
<td>20</td>
</tr>
</tbody>
</table>

Fig. 1 Distribution of number of occupants in studied houses in Cape Dorset, Nunavut
houses had five to six rooms, excluding bathroom(s). Occupancy rates for the houses in the study were high, with a median of 6 persons/house (mean 5.9 persons/house) and a range of 2–12 persons/house (Figure 1). In addition to the index infant, there was an average of one additional child <4 years of age in the house. The median airflow rate per person was 5.6 l/s/person, and was below 7 l/s/person, the current American Society of Heating, Refrigerating and Air-Conditioning Engineers Inc. (ASHRAE) standard, in 10 of 17 (59%) of the dwellings (Table 2) (American Society of Heating Refrigerating and Air Conditioning Engineers Inc. 1997). Because the houses were very small, calculated air change rates tended to be high, with a mean of 0.67 air changes per hour. However, seven of 17 houses (41%) had a calculated natural air change rate below the recommended standard of 0.35 air changes per hour (Shaw 2003). The median CO₂ concentration was 1205 ppm. This is above the target CO₂ concentration of 1000, often used as an indicator of adequate ventilation (Enmet Canada Ltd., 2002).

Household temperature levels were high, with a mean of 23.7°C (range 19.0–28.8°C). RH was generally low, with a mean value of 24.6%; most houses had levels between 15% and 30%. In contrast, in houses in Saskatoon, Saskatchewan, the mean temperature was 20°C and the mean relative humidity was 35% in winter (Dumont, 1995). Mean RH was significantly inversely related to airflow (Pearson’s coefficient  \( r = -0.52, P = 0.034 \) ) and was positively associated with mean CO₂ concentration (\( r = 0.58, P = 0.012 \) ). Mean temperature was positively associated with the number of occupants of the home (\( r = 0.56, P = 0.011 \) ) and with air change rate (\( r = 0.50, P = 0.040 \) ).

Maternal smoking data were available for eight cases. Of the six (75%) of these mothers that smoked, five (62.5%) smoked inside the house. Smokers were said to be present in at least 18 (90%) of households (see below), and smoking indoors by individuals other than the mother happened in 14 (70%) of households. Fourteen (70%) of the households also reported smoking by individuals other than the mother in the infant’s bedroom. In two houses where the total number of smokers was not reported but other smokers were reported to be present, the number of smokers was conservatively estimated at one per household. Households with smokers had a mean of 2.3 (median 2.0) smokers per home (range 1–5). The mean nicotine concentration was 0.84 μg/m³ and the highest value recorded was 4.04 μg/m³. Nicotine concentrations exceeded 1.5 μg/m³ in five houses in the study (25%). The mean value was similar to that of published data from the eastern US, where nicotine concentrations in the houses of smokers averaged approximately 1–1.5 μg/m³ (Hammond, 1999). Two samplers located in a non-smoking area recorded concentrations of ‘not detected’ and 0.05 μg/m³, respectively. Using all available data, nicotine concentrations were found to be weakly associated with the reported number of smokers (\( r = 0.326, P = 0.161 \) ). However, the house with the second-highest nicotine concentration reported no occupants who smoked. It was felt that either the occupants had provided incorrect information, they had visitors who smoked, or there was a reading error, and this house was excluded from subsequent analyses. With this house excluded, nicotine concentrations were strongly correlated with the number of smokers in the house (\( r = 0.667, P < 0.002 \) ) (Figure 2). Nicotine concentrations could not be meaningfully compared between houses with and without smokers, as only one house clearly did not have smokers; the nicotine concentration in this house was 0.058 μg/m³. Nicotine concentrations were not significantly correlated with airflow (\( r = 0.30, P = 0.245 \) ) or air change rates (\( r = 0.11, P = 0.686 \) ).

### Table 2 Indoor air quality parameters in houses in Cape Dorset

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean</th>
<th>Median</th>
<th>Minimum</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calculated airflow (l/s)</td>
<td>17</td>
<td>33.6</td>
<td>6.3</td>
<td>117</td>
</tr>
<tr>
<td>Airflow rate per person (l/s/person)</td>
<td>17</td>
<td>5.6</td>
<td>1.9</td>
<td>19.5</td>
</tr>
<tr>
<td>Calculated air change rates (per hour)</td>
<td>17</td>
<td>0.67</td>
<td>0.13</td>
<td>2.24</td>
</tr>
<tr>
<td>CO₂ (ppm)</td>
<td>18</td>
<td>1205</td>
<td>1201</td>
<td>1792</td>
</tr>
<tr>
<td>Mean household temperature (°C)</td>
<td>20</td>
<td>23.9</td>
<td>23.7</td>
<td>29.2</td>
</tr>
<tr>
<td>Relative humidity (%)</td>
<td>20</td>
<td>23.3</td>
<td>12.9</td>
<td>43.9</td>
</tr>
<tr>
<td>NO₂ (ppb)</td>
<td>20</td>
<td>3.7</td>
<td>5.6</td>
<td>14.7</td>
</tr>
<tr>
<td>Nicotine (μg/m³)</td>
<td>20</td>
<td>0.28</td>
<td>N.D.</td>
<td>4.04</td>
</tr>
</tbody>
</table>

N.D., not detected.

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**Indoor air quality in Nunavut housing**

![Fig. 2 Relationship between the total number of smokers in the home and nicotine concentration. Note the outlier, where no smokers were reported in the home, but where the measured nicotine concentration was 3.76 μg/m³](image-url)
The median NO\textsubscript{2} concentration was 3.7 parts per billion (ppb), and the maximum value observed was 14.7 ppb (Table 2). All the NO\textsubscript{2} concentrations measured were below the current residential IAQ guideline of 50 ppb (Environmental Health, 1989). Sixteen of 20 homes sampled were single-family dwellings heated with a furnace using low-sulphur arctic diesel as the fuel source (Northern News Services Online, 2003). The remaining four houses were townhouses with furnaces located as a separate entity. There was no statistically significant difference in NO\textsubscript{2} concentration between houses with a furnace inside (median 3.7 ppb) than in those without (median 5.9 ppb).

Houses had an average of 167 872 particles (standard deviation 161 045 particles) per cubic foot (1 min sample). On average, 72% of the particles were <0.3 \textmu m in diameter and the particle distribution was consistent with tobacco smoke or diesel exhaust. All particle sizes, and total particle counts were found to be correlated strongly with nicotine concentration (r = 0.62–0.76, P < 0.001–0.008). Particles <0.3, and between 0.3 and 0.5 \textmu m in diameter were also found to correlate well with the number of occupants in the home (r = 0.58, P = 0.015, and r = 0.51, P = 0.036, respectively). No particle size, or the total particle count, correlated with NO\textsubscript{2} concentration (r < 0.48, P > 0.05).

The amount of dust in Cape Dorset houses was similar to or less than that in houses in southern Canada, and the weight distribution of settled floor dust was similar to results from studies from the Big Cove (First Nations) Reserve in New Brunswick, and the Prince Edward Island (PEI) Infant Health Study (D. Miller, unpubl. data) (Dales and Miller, 1999). The median living room dust weight was 0.67 g for the >300 \textmu m fraction, and 0.29 g for the <300 \textmu m fraction. However, mattress dust weights were at least 10 times higher than maritime reference values, with a mean weight of 0.59 g. Bacterial lipopolysaccharide endotoxin levels were approximately twice the level seen in other studies, with a median concentration of 2500 (mean 3218, range 1500–7400) ng/g dust (D. Miller, unpubl. data).

Settled dust 1,3-\beta-D-glucan concentrations were elevated compared with those of previous studies, ranging from 63 to 494 \mu g/g dust, and this was due to extremely high levels of yeasts, with few building-associated molds detected. In a few houses, duplicate samples were obtained, and these were averaged for statistical analysis, except for one house where one duplicate sample had a concentration of 0, which was dropped. The median 1,3-\beta-D-glucan concentration was 217 \mu g/g in settled living room dust and 210 \mu g/g in mattress dust (<300 \textmu m fraction). No significant associations were found between settled dust 1,3-\beta-D-glucan and concentrations of fungi, endotoxin, relative humidity, temperature, or air change rates (P > 0.07).

The mean concentration of fungi in living-room dust samples was 700 267 colony-forming units (CFU)/g. Yeasts were present in living-room dust in all 15 houses with samples available, and were the commonest fungus isolated in nine of 15 living rooms (60%). Penicillium was present in 10 of 15 houses (67%). Aspergillus was present in six houses, and Mucor was present in five houses. The average concentration of yeast in living-room dust samples was 386 261 CFU/g. The concentration of yeast in living-room dust was not significantly associated with the number of occupants, air change rates, relative humidity, mean temperature, or endotoxin levels (P > 0.42).

The median concentration of fungi in mattress dust samples in the eight homes with samples available was 2 783 373 CFU/g (range 43 032–12 975 000 CFU/g). Penicillium was present in six of seven mattress dust samples (86%). Mucor and Aspergillus species were each present in two bedrooms. The number of fungal colonies in mattress and living-room dust samples were highly correlated (r = 0.76, P = 0.028), but fungal concentration in mattress dust samples was not significantly associated with the number of individuals sharing the bed, relative humidity, mean temperature, or endotoxin levels (P > 0.13), although the sample size was very small. Yeasts were present in mattress dust in all samples available, and were the commonest fungus isolated in six of seven cases (86%).

Virtually, no dust-mites were detected in Nunavut mattress dust samples. Only one of 18 houses (6%) had mattress dust-mite antigen levels greater than the limits of detection; this house had Der f 1 and Der p 1 concentrations of 0.07 and 0.08 \mu g/g dust, respectively. In contrast, in Boston, Massachusetts, the average bed dust concentrations for Der f 1 and Der p 1 were approximately 1 and 1–10 \mu g/g dust, respectively (Chew et al., 1999). Microscopic examination of the mattress dust failed to detect dust-mite species other than D. farinae and D. pteronyssinus. Infants often shared their bed with other individuals, with this occurring in 45% of households. Among infants who shared their bed, a median of 2 (median 1.7, range 1–3) other individuals were present. Households in the community likely keep mattresses for extended periods of time (B. Wortman, pers. comm.).

Associations between IAQ and respiratory symptoms were limited by the small sample size, and only exploratory analyses were performed. To avoid issues of multiple comparisons, univariate logistic regression was carried out only using key respiratory health variables, and performed using composite indices of related health outcomes. No significant relationships were found using logistic regression (data not presented). In univariate testing, no IAQ variables were associated with an increased risk of LRTI or hospitalization (P > 0.12). Cough between colds was significantly associated with total mold colony counts in
living-room floor settled dust (1,711,778 vs. 511,316 CFU/g, \( P = 0.039 \)). Increased calculated airflow and calculated air change rates appeared to be associated with wheeze between colds (70.7 vs. 28 l/s, \( P = 0.006 \), and 1.26 vs. 0.5 air changes per hour, \( P = 0.023 \), respectively), and wheezing most days (calculated airflow 47.4 vs. 20.9 l/s, \( P = 0.02 \)). Wheeze between colds was also associated with increased total particulate counts [323,393 vs. 103,071 particles/cubic foot (1-min sample), \( P = 0.014 \)] and with increased mean relative humidity (20.7 vs. 31.7%, \( P = 0.003 \)).

**Discussion**

This pilot study identified several factors which may increase the incidence and/or severity of severe LRTI in Inuit infants. A significant proportion of houses had reduced air change rates. ETS exposure was nearly universal. Mattress fungal levels were markedly increased, although building fungal concentrations were low. Dust-mites were virtually non-existent. Neither evidence of spillage from heating devices, nor other combustion sources resulting in significant elevation of indoor NO\(_2\) levels were observed. Measured IAQ indices were not associated with LRTI, although this result must be regarded as preliminary, given the small sample size. The relationship between smoking and respiratory health outcomes could not be assessed, as smokers were present in virtually all households. Twenty-five percent of our small sample of infants in Cape Dorset, had been hospitalized for a chest illness in the first 2 years of life consistent with larger studies from the Baffin Region (Banerji et al., 2001). Wheeze was common, although this may have been partly due to nasal secretions causing noisy breathing called ‘wheeze’ by parents (Elphick et al., 2001).

Approximately one-half of houses in this study had relatively low air change rates, and reduced airflow rates per person, despite the very small volume of these dwellings. The ASHRAE standard suggests an air change rate of 1 m\(^3\)/min per person of outdoor air per person, or approximately 7–8 l/s/person to keep indoor concentrations of CO\(_2\) in the acceptable range of under 1000 ppm (American Society of Heating Refrigerating and Air Conditioning Engineers Inc, 1997). The reduced air change rates may be particularly problematic, given the very high occupancy rate of these extremely small houses. The houses in our study had a mean of 5.9 inhabitants. Hospitalized infants from Baffin Region were also reported to live in a household with an average of 6.4 people, including 3.4 adults and 3.0 children (Banerji et al., 2001). Sharing a bedroom with additional children was associated with an increased risk of LRTI in Sisimiut, Greenland (Koch et al. 2003). The one previous study which examined IAQ in northern Canadian housing was conducted by the Canada Mortgage and Housing Corporation (CMHC), and assessed 55 houses in 1989–1990 in seven communities in present-day Nunavut and NWT. The houses had minimal exhaust equipment, consisting primarily of low-capacity kitchen and bathroom exhaust fans, which were often used intermittently because of noise. Some houses also had passive vents, consisting of piping. Air change rates were as low as 0.042 air changes per hour, with approximately half of the houses having air change rates below 0.30/h. Spot testing revealed that 25 of 54 houses (44%) had levels of CO\(_2\) above the ventilation target level of 1000 ppm. CO\(_2\) varied inversely with air change rates (Appin Associates and Yassi, 1991). Similarly, we found air change rates below 0.35 air changes per hour in 41% of the houses tested. The average CO\(_2\) concentration was 1205 ppm, which is not markedly elevated but which was higher than the 1000 ppm target concentration (Appin Associates and Yassi, 1991). Our measurements of home air tightness may have been underestimates, as all house openings were not sealed, and as the EnerGuide testing procedure does not record fresh air ducts connected for the furnace or central exhaust systems. However, this would not affect the natural air change rates used in the data analysis, which were obtained using the tracer gas technique.

Reduced ventilation rates and outdoor air supply, estimated using indoor CO\(_2\) concentrations, has been associated with enhanced airborne transmission of common respiratory viral infections in experimental models, and with increased quantities of airborne rhinovirus in southern office buildings (Myatt et al., 2004; Rudnick and Milton, 2003). While most respiratory pathogens are not normally spread by airborne droplets, these data suggest that this could occur when ventilation rates are sufficiently low (Myatt et al., 2004). Reduced indoor ventilation rates are strongly associated with increased risk of tuberculosis (TB) in healthcare workers in a hospital setting (Menzies et al., 2000). The incidence of TB was markedly higher in NWT than in southern Canada (Health Canada, 2001; Nguyen et al., 2003). TB incidence has been shown to be positively associated with housing density in Canadian First Nations communities (Clark et al., 2002).

Low-sulphur arctic diesel is used for heating in Nunavut (Northern News Services Online, 2003). Particulate matter from diesel exhaust emissions increased air inflammation and RSV gene expression in mice infected with RSV (Harrod et al., 2003). However, the uniformly low NO\(_2\) concentrations measured in houses in this study and lack of correlation between the indoor NO\(_2\) concentrations and particulate counts suggest that spillage of combustion products of diesel used for heating was not occurring. The earlier CMHC study in the NWT also did not find evidence of combustion spillage (Appin Associates and Yassi, 1991). While indoor exposure to relatively low...
concentrations of NO\textsubscript{2} have been associated with more severe exacerbations triggered by respiratory viruses in children with asthma, NO\textsubscript{2} concentrations in our study were well below recommended guidelines (Chauhan et al., 2003; Environmental Health Directorate Health Protection Branch, 1989).

Environmental tobacco smoke exposure has been reported to increase the risk of LRTI and hospitalization for LRTI in infants in temperate climates and in Sisimiut, Greenland (Koch et al., 2003; Li et al., 1999; McConnachie and Roghmann, 1986; Nafstad et al., 1997). Smokers were reported to be present in 90\% of the homes of infants and children, and, based on nicotine concentrations, probably in 95\%. The close correlation between indoor particulate numbers and nicotine concentrations, and the particle distribution suggests that particulates were mainly generated from tobacco products. Despite the reduced air change rates in some of the houses, average measured nicotine concentrations were similar to values obtained in the homes of smokers in the continental US (Hammond 1999). However, the average relative humidity and temperature values were quite different from those in more temperate climates, and studies are required to determine whether this could have affected the passive nicotine samplers. The prevalence of smoking in the homes of infants and young children in northern Canada is much higher than is reported for Canada as a whole, where 25\% of children under 12 years of age were exposed to ETS at home (Health Canada, 2001). Jenkins et al. (2004) reported that 85\% of Inuit infants in a birth cohort from Iqaluit, Nunavut were exposed to ETS at home. The previous CMHC study found indoor particulate levels in NWT housing to be high, with a maximum level of 167 \mu g/m\textsuperscript{3}, and 23 of 30 houses (77\%) having levels above the acceptable range of 40 \mu g/m\textsuperscript{3}, which was felt to be due to cigarette smoking, as indoor soapstone carving was infrequent, although animal fur preparation as a contributor could not be excluded (Appin Associates and Yassi, 1991). Pulmonary inflammation increases with particulate matter exposure in alveolar epithelial cells infected with adenovirus (Fuji et al., 2003; Saldiva et al., 2002). We were unable to compare our measurements of particle numbers with published acceptable standards. We observed a positive relationship between total particulates and chronic wheezing.

Household temperatures in Cape Dorset dwellings were high, and RH levels were low. The earlier CMHC study in the NWT also found that most houses had an RH below 30\% (Appin Associates and Yassi, 1991). It is unclear whether adverse health effects are linked to relatively dry ambient air conditions. Low RH may promote persistence of infective aerosols as an aerosol, as hydration and subsequent settling of infectious aerosols is impeded (Arundel et al., 1986). Influenza virus survives better at low RH levels, but most respiratory viruses survive better at higher humidity levels (Arundel et al., 1986; Buckland and Tyrrell, 1962; Ijaz et al., 1985; Karim et al., 1985). The inverse relationship between chronic wheezing air change rates was likely the result of a type II error related to the small sample size, as wheeze between colds was also associated with increased humidity, and airflow and humidity were found to be negatively related in both our study and in the earlier CMHC study (Appin Associates and Yassi, 1991).

Exposure to high levels of fungi and reported home dampness has been associated with an increased risk of LRTI in infants <1 year of age and bronchitis in children (Dales et al., 1991; Stark et al., 2003; Verhoeff et al., 1995). Little information is available on fungi in northern housing. While we found a relatively higher amount of fungal glucan in the settled dust compared with other studies, this was not associated with elevated viable counts of building-associated fungi. The fungi found in the present study were remarkable for the relative absence of phylloplane fungi such as \textit{Alternaria} and \textit{Cladosporium}. The remaining molds were either food-associated fungi or species of penicillia more associated with food than damp building materials (Flannigan and Miller, 2001). By elimination, the majority of glucan found was from the yeasts in the floor and mattress dust. In contrast, the glucan in the air of homes in PEI is predominantly from filamentous fungi (Foto et al., 2004). Total fungal counts, comprising both yeasts and filamentous fungi in mattresses were notably elevated, likely reflecting prolonged use of these mattresses, evidently often by multiple individuals simultaneously. Fungal glucan are generated by both yeasts and filamentous fungi, and the mean values of glucan in settled dust were unexpectedly higher than those in studies conducted in Ottawa and in the Elsipogtog Reserve in New Brunswick, Canada (Berghout et al., 2005). We observed an association between living-room mold counts and chronic cough. Sampling of airborne fungi in the homes of 38 children in Sør-Varanger, a sub-arctic community on the far northern tip of Norway, revealed that the commonest fungus was \textit{Penicillium}, present in 89\% of homes, and \textit{Alternaria} was not detected. Allergy to mold in schoolchildren living in Greenland is rare, with only 1.2\% of children found to be sensitized to \textit{Cladosporium herbarum} (Krause et al., 2002).

Elevated family room dust endotoxin levels have been associated with wheezing episodes in children in the first year of life (Park et al., 2001). Living room endotoxin levels were moderately elevated. We did not test indoor concentrations of furred animal allergens, as previous northern Canadian studies had indicated that the Inuit do not usually keep dogs or cats indoors (Hemmelgarn and Ernst, 1997).

We found virtually no dust-mites. Dust-mites were detectable in only 6\% of mattresses, and the only

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to reduce the exposure of children to ETS is needed. Functional in extreme cold winter conditions (Appin or reducing indoor relative humidity, while being ventilation without markedly increasing heating costs ventilation systems in the Arctics to achieve improved additional housing and the development of better problem, including promoting the construction of public health approaches which may reduce this extremely common. This pilot study suggests a number infectious aerosols, rather than contact. ETS exposure was reduced air change rates, which may increase the risk not observed. A significant proportion of houses had perturbations in IAQ, such as spillage from heating devices or marked elevation in indoor NO2 levels were not observed. A significant proportion of houses had reduced air change rates, which may increase the risk of respiratory viral infections spreading through infectious aerosols, rather than contact. ETS exposure was extremely common. This pilot study suggests a number of public health approaches which may reduce this problem, including promoting the construction of additional housing and the development of better ventilation systems in the Arctics to achieve improved ventilation without markedly increasing heating costs or reducing indoor relative humidity, while being functional in extreme cold winter conditions (Appin Associates and Yassi, 1991). Further public education to reduce the exposure of children to ETS is needed (D'Souza 2003). Health education regarding replacement of old mattresses, and use of separate cribs for infants should be considered. We confirmed that the measurement of IAQ is feasible in this region. Further epidemiologic research is therefore planned to examine the prevalence of reduced ventilation in a larger sample of current Inuit housing in Nunavut. We also plan to examine and evaluate more effective ventilation systems for housing in the high Arctic.

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