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Low prevalence of iron-deficiency anaemia among Inuit preschool children: Nunavut Inuit Child Health Survey, 2007–2008

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Abstract

Objective: To report the prevalence rates and correlates for anaemia, iron deficiency (ID) and iron-deficiency anaemia (IDA) among Inuit preschool-aged children.
Design: A cross-sectional study assessed iron intake, demographic information, medical history, anthropometrics, Hb, ferritin, C-reactive protein and antibodies to *Helicobacter pylori*.
Setting: Sixteen selected Inuit communities in Nunavut Territory, Canada.
Subjects: Inuit (*n* 388) aged 3–5 years randomly recruited from communities.
Results: Anaemia (3–4 years: Hb < 110 g/l; 5 years: Hb < 115 g/l) was prevalent in 16·8 % of children. The prevalence of ID (ferritin < 12 μg/l) was 18·0 % and that of IDA was 5·4 %. When ID was defined as ferritin <10 μg/l, 10·8 % of children were iron deficient and 3·3 % had IDA. In multiple logistic regression, boys were more likely to be iron deficient (OR = 2·28, 95 % CI 1·17, 8·25), but no other risk factor emerged for ID. Three- to 4-year-olds were less likely than 5-year-olds to have anaemia from causes other than ID (OR = 0·11, 95 % CI 0·08, 0·58). Anaemia from other causes was more common among children residing in crowded homes (OR = 2·30, 95 % CI 1·37, 12·31) and those treated for past-year ear infection (OR = 1·35, 95 % CI 1·05, 7·21).
Conclusions: The low prevalence of ID and IDA is encouraging, but efforts are still needed to reduce rates as they continue to be higher than general population rates. Household crowding and infections may contribute to anaemia and warrant further research.

Keywords
Aboriginal
Children
Anaemia
Iron deficiency
Helicobacter pylori

In infants and children, iron-deficiency anaemia (IDA) can have serious health consequences including impaired growth and cognitive development and weakened immune defence[1–3]. Iron deficiency (ID) typically exists in three stages: low iron stores, reduced iron delivery to the tissues and IDA characterized by low Hb and reduced erythrocyte size[4]. The aboriginal people of Canada include three distinct groups: Inuit, First Nations and Métis[5], and there is evidence that the rates of anaemia and ID are higher among aboriginal children than among non-aboriginal children. Recent prevalence estimates for Inuit infants are 36–60 % for ID compared with 33 % for non-aboriginal Canadian infants[6–8]. IDA is thought to affect 26 % of Inuit infants compared with 5 % for non-aboriginal infants[6–8]. Information for the preschool age group of 3–5 years is lacking. Prevalence estimates for Canadian infants and children combined are 24 % for tissue ID and 5 % for anaemia from all causes[9]. Current information on ID and IDA among Canadian Inuit preschoolers, however, is not available.

Studies among Inuit children show that dietary iron intake is most likely adequate[6,10–12]. However, a nutrition transition in the Arctic is rapidly occurring, which warrants ongoing nutritional status assessment and biomarker monitoring. Further, infection with the human pathogen, *Helicobacter pylori*, has been postulated to contribute to ID, although the mechanisms remain unclear[13–23]. *H. pylori* infection is highly prevalent in Arctic populations[6,24,25] and may increase the risk for ID among Inuit children[16]. Therefore, a cross-sectional survey of Inuit preschool children in Nunavut was used to evaluate the prevalence and correlates of anaemia, ID and IDA. The study to date has identified a high prevalence of child food insecurity (56 %) as well as of other indicators of socio-economic disadvantage including household crowding (53·9 %), income support (42·7 %) and living in public housing (69·7 %)[26]. The children are of normal stature[27], but have a high prevalence of overweight based upon Centers for Disease Control and Prevention (CDC)[27] and WHO international standards[26]. Further,

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nearly half of the children consumed traditional Inuit food in the past 24 h and traditional food contributed to nutrient intakes, including iron[28].

Experimental methods

Setting

This research is part of the Inuit Child Health Survey of preschool children in the Nunavut Territory of Canada, with details of the study design and demographic characteristics described elsewhere[26,28]. Using currently available population census information, we estimated that a sample of ninety to 100 children would provide 90 % power to detect a population prevalence of IDA of 10 (SD 5) %. A sample size of 300 was then desired as a minimum to allow for multivariable analyses and assessment of other correlates. Sixteen of the twenty-five communities in Nunavut were chosen to participate. The communities were selected to represent the three jurisdictional regions within Nunavut, to be geographically dispersed by latitude, and to represent small, middle and large-size communities. From these communities, Inuit children, aged 3–5 years, were randomly selected to participate in the survey using health centre lists of age-appropriate children. Recruiters were instructed to make three attempts to reach caregivers. Written informed consent was obtained from the children's caregivers. The survey was developed by a steering committee consisting of partners from Inuit and community organizations, Nunavut health officials and McGill University and the University of Toronto. Certification of ethical acceptability for research involving human subjects was obtained from the McGill Faculty of Medicine Institutional Review Board. A scientific research licence was obtained from the Nunavut Research Institute.

Anthropometry

For the current report, age- and sex-appropriate BMI *Z*-scores were based upon the 2000 CDC growth reference[29].

Iron status and exposure to H. pylori

Venous or capillary sampling was used to obtain blood samples. When venepuncture was used, 3 ml of blood was collected into sodium heparin Vacutainer® blood tubes (Becton Dickinson, Franklin Lakes, NJ, USA). The Vacutainer tube was inverted gently ten times. One drop of whole blood was dispensed onto Parafilm (Pechiney, Chicago, IL, USA) using a Diff-Safe® blood dispenser (Alpha Scientific Corporation, Southeastern PA, USA). Hb was measured either from this drop or from capillary blood samples using the cyanmethaemoglobin method with a HemoCue™ 201+ portable photometer (HemoCue Inc., Lake Forest, CA, USA). Blood samples were centrifuged within 6 h of collection. Separated plasma was stored at −20°C during fieldwork and at −80°C after completion of data collection.

Ferritin was measured from plasma samples using an autoanalyser (Liason®; DiaSorin, Saluggia, Italy) and a ferritin integral (REF 313551, DiaSorin, Saluggia, Italy). Low, normal and high control samples were tested with each analysis. C-reactive protein (CRP) was measured using a SYNCHRON® autoanalyser (Beckman Coulter, Inc., Brea, CA, USA) and a high-sensitivity CRP (hsCRP) assay at the Montréal Children's General Hospital, Montréal, Canada. Previous exposure to *H. pylori* was assessed using a qualitative ELISA (Pylori Detect IgG; Calbiotech, Spring Valley, CA, USA) for the presence of anti-*H. pylori* IgG antibodies in plasma. Logistical aspects of conducting carbon urea breath tests given the other research priorities precluded assessment of current *H. pylori* infection.

ID was defined as low ferritin (<12 μg/l)[2]. IDA was defined as the presence of low ferritin coupled with low Hb. Other anaemia was defined as low Hb, but normal ferritin (≥12 μg/l). As the cut-off value for low ferritin in children is currently unclear, we also conducted analyses using ferritin <10 μg/l to define ID and IDA[8,30]. The presence of acute inflammation elevates circulating ferritin concentrations; therefore, prevalence estimates for ID and IDA were restricted to children in whom hsCRP was below 8 ng/ml[7]. Analyses were also repeated using a lower hsCRP cut-off of 3 ng/ml[31,32], but as no differences were identified, only results using the 8 ng/ml cut-off are presented.

Dietary intake

A 24 h dietary recall was conducted for each child participant with a non-consecutive day repeat recall conducted for a 20 % subsample. Interviewers were trained using a five-stage, multiple-pass interviewing technique. Food model kits were used to estimate portion sizes. Interviewers were asked about the child's mineral and vitamin supplement use, including frequency and brand information to allow for determination of nutrient content. Each caregiver was asked to complete a past-month qualitative FFQ for their child on traditional Inuit foods and market foods high in iron. Food frequency information was entered using EpiInfo (CDC, Atlanta, GA, USA). The 24 h dietary recall information was entered using CANDAT (Godin London Incorporated, London, Ontario, Canada). Iron intake was obtained using the Canadian Nutrient File (Health Canada 2007) and a database of 2000 additional foods derived from standardized recipes and food labels. All dietary data entries were double verified for errors. Traditional food intake used in univariate analyses was dichotomized based on a frequency of consumption that was greater or less than the median.

The home environment

Interviewers conducted questionnaires for characteristics of the home including the United States Department of Agriculture's eighteen-item Household Food Security Survey Module adapted for the Inuit populations[33,34]. The food security module was scored according to Health

Canada guidelines and detailed methods and findings are described elsewhere[26]. Household crowding was defined as living in a home with greater than the median number of people per household.

Statistical analyses

There was incomplete ascertainment of all variables for all children. Univariate statistical analyses were conducted using all available data (*H. pylori*, *n* 282; hsCRP, *n* 254; Hb, *n* 285; ferritin, *n* 253; iron intake, *n* 374). Weighted prevalence rates with 95 % CI for anaemia, IDA and ID were estimated. Sampling weights were based on the proportion of participating children in each community using the total number of age-appropriate children obtained from health centre lists as the denominator for calculating weights.

Usual iron intake from the 24 h recall was estimated from observed intake using Software for Intake Distribution Estimation (Iowa State University, 1996). Adjustments were made for sequence and day of week of the recall. Within-person variability was estimated using information from the 20 % subsample of repeat recalls. The percentage of children below the age-appropriate Estimated Average Requirement (EAR) for iron was determined[35]. The frequency of consumption of iron-containing traditional and market food was calculated, both for consumers only and for all children. The three outcomes of interest were ID, IDA and other anaemia. Univariate analyses of outcome and exposure variables were performed using a χ^2 test or Fisher's exact test when cell sizes were <10. Relative risks (RR) and 95 % CI were calculated for relevant exposure variables. As sex differences were noted in ID, a *post hoc t* test examined sex differences in dietary iron intake. Multivariable logistic regression was performed to examine independent effects of exposure variables when variables were of borderline significance ($P < 0{\cdot}10$) in univariate analyses; adjusted OR were calculated from regression coefficients. For all analyses, a P value $<0{\cdot}05$ was considered significant. Weighted prevalence rates and dietary adequacy analyses were determined using the SAS statistical software package version 9·1 (SAS Institute, Cary, NC, USA). All other analyses were performed using the STATA statistical software package version 10·0 (StataCorp., College Station, TX, USA).

Differences in Hb levels obtained from capillary and venous blood samples were evaluated to determine the feasibility of combining the data in an overall assessment of anaemia prevalence. Mean Hb concentration in capillary blood samples (114 (SD 12) g/l) was significantly lower than that in venous blood samples (118 (SD 7) g/l, Wilcoxon rank $P < 0{\cdot}01$), perhaps attributed to a dilution effect associated with finger prick sampling. There were, however, no socio-economic differences noted between those who provided a capillary or a venous blood sample, which suggested that anaemia prevalence rates would not be biased by excluding capillary blood samples from the analyses. Thus, analyses involving the Hb reported herein were based solely upon venous blood samples.

Results

Of the 644 homes approached, a total of 537 homes were successfully contacted, of whom seventy-five (11·6 %) refused upon initial contact and seventy-four (13·8 %) cancelled or did not attend their clinic appointment. The overall participation rate was 72·3 % (*n* 388). Venous blood was obtained from 289 of 388 children (74·7 %) and capillary blood samples from seventy-nine of 388 children (20·4 %). Five per cent of the children did not undergo any blood sampling.

Population characteristics

Fifty-three per cent of the participating children were female and the mean age was 4·4 (SD 0·9) years. Daycare was attended by 38·3 % of children. Sixteen per cent of caregivers reported that they gave their children a nutritional supplement containing iron, most commonly multi-vitamin and mineral supplements. The weighted prevalence of exposure to *H. pylori* was high at 46·1 % (95 % CI 40·1, 52·1; Table 1). The median hsCRP concentration was 0·65 ng/ml (25th percentile: 0·2 ng/ml; 75th percentile: 2·2 ng/ml). Overall, fourteen children (5·1 %; 95 % CI 2·2, 8·0) had high hsCRP concentrations of ≥8 ng/ml. These children, along with twenty-one children whose hsCRP status could not be determined, were excluded in determining the prevalence of ID and IDA as well as from univariate and multivariate analyses.

Prevalence of iron deficiency and anaemia

The mean ferritin concentration was 19·1 (SD 10·1) μg/l and the median was 16·6 μg/l (Fig. 1a). Overall, 18·0 % (95 % CI 12·7, 23·3) of children were iron deficient using a ferritin cut-off of 12 μg/l (Table 1). Using a ferritin cut-off of 10 μg/l, the prevalence of ID decreased to 10·9 % (95 % CI 6·5, 15·2; Table 1). IDA was found in 5·4 % (95 % CI 2·3, 8·6) of

Table 1 Prevalence of ID, anaemia, IDA and *Helicobacter pylori* infection among participating children: Nunavut Inuit Child Health Survey, 2007–2008

	n/*N*	Prevalence (%)	95 % CI
Ferritin (<12 μg/l)			
ID	45/238	18·0	12·7, 23·3
IDA†	11/235	5·4	2·3, 8·6
Ferritin (<10 μg/l)			
ID	25/238	10·8	6·5, 15·2
IDA†	7/235	3·3	0·9, 5·8
Anaemia‡	47/285	16·8	12·0, 21·6
H. pylori infection	128/282	46·1	40·1, 52·1

ID, iron deficiency; IDA, iron-deficiency anaemia.
†Presence of anaemia coupled with ID.
‡Hb <110 g/l (in 3–4-year-olds) or <115 g/l (in 5-year-olds).

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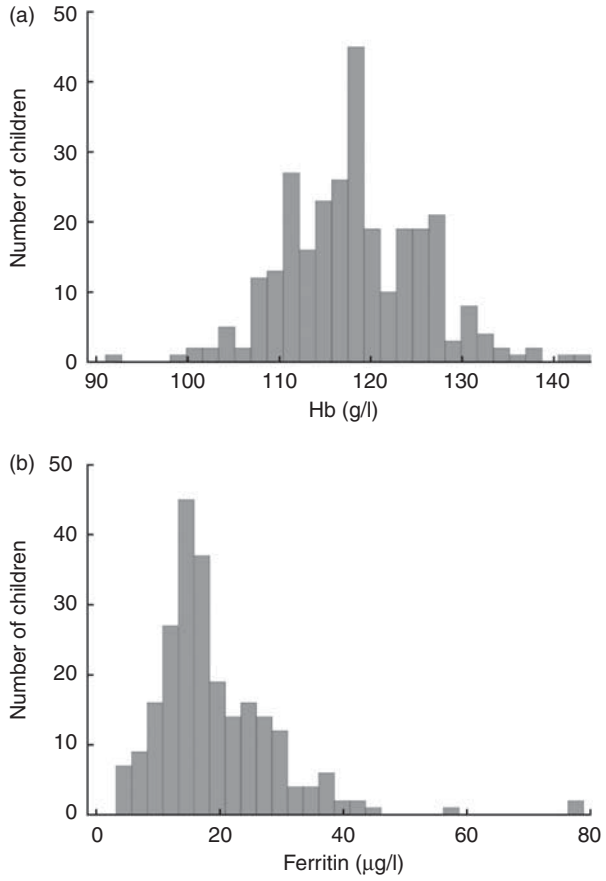

Fig. 1 (a) Distribution of Hb concentration values among Inuit preschoolers, where anaemia is defined as Hb below 110 g/l (3–4-year-olds) or 115 g/l (5-year-olds). (b) Distribution of ferritin concentration values among Inuit preschoolers, where ferritin below 10 or 12 μg/l is defined as iron deficiency in children (Nunavut Inuit Child Health Survey, 2007–2008)

children and when the lower ferritin cut-off was used, the prevalence of IDA decreased to 3·3 % (95 % CI 0·9, 5·8).

The mean Hb concentration was 118 g/l (SD 8·0; Fig. 1b). The weighted prevalence of anaemia from all causes was 16·8 % (95 % CI 12·0, 21·6). Among children with anaemia, 4·3 % had moderate-to-severe anaemia (Hb < 100 g/l), whereas 95·7 % had mild anaemia (Hb ≥ 100 g/l, but below age-specific cut-off). ID (ferritin <12 μg/l) explained 31 % of observed anaemia. When ID was defined as ferritin below 10 μg/l, it explained 20 % of the observed anaemia. Thus, 69–80 % of the anaemia observed in Inuit preschoolers was most likely due to causes other than low iron stores.

Dietary iron intake

Iron intake was normally distributed with a mean of 15·6 (SD 11·0) mg/d and median of 13·5 mg/d. Only 0·3 % of children had iron intake below their age-specific EAR (1–3 years: 3·0 mg/d; 4–8 years: 4·1 mg/d). Based on the FFQ, children commonly consumed various iron-rich foods such as caribou meat (84·2 %), ringed seal meat (49·5 %), beef (82·6 %) and breakfast cereals (96·3 %; Table 2).

Correlates of iron deficiency and iron-deficiency anaemia

In univariate and multivariate logistic regressions, no significant correlates emerged for ID or for IDA when using the ferritin cut-off value of 12 μg/l. However, when using the lower ferritin cut-off value (10 μg/l), age and sex were significantly associated with ID (Table 3) but not with IDA (data not presented given the small number of IDA events). In univariate analyses, boys were more likely to be iron deficient than girls (RR = 3·22, 95 % CI 1·33, 7·78), and children aged 3–4 years were more likely to be iron deficient than 5-year-olds (RR = 3·31, 95 % 1·02, 10·71). In a multiple logistic regression model for ID containing age, sex and BMI *Z*-scores, only boys were significantly at risk (adjusted OR = 2·28, 95 % CI 1·17, 8·25). However, there were no significant differences in mean dietary iron intakes between boys (15·5 (SD 5·8) mg/d) and girls (15·6 (SD 6·1) mg/d).

Correlates of other anaemia

Correlates for anaemia from causes other than ID were examined (Table 3). Three- to 4-year-olds were less likely to be anaemic from other causes (5·3 %) than 5-year-olds (19·2 %; RR = 0·28, 95 % CI 0·13, 0·60). Children residing in a home with six or more individuals were more likely to be anaemic (12·3 %) than those living in less crowded homes (4·8 %; RR = 2·56, 95 % CI 1·04, 6·34). Children who were treated for an ear infection in the past 12 months were more likely to be anaemic (14·5 %) than children not requiring treatment for an ear infection (6·6 %; RR = 2·20, 95 % CI 1·01, 4·76). There were no differences in mean BMI *Z*-scores or in hsCRP concentrations between children with and without anaemia from other causes. In a multiple logistic regression model adjusted for BMI *Z*-scores and sex, 3–4-year-olds were less likely to be anaemic relative to 5-year-olds (OR = 0·11, 95 % CI 0·08, 0·58), while household crowding (OR = 2·30, 95 % CI 1·37, 12·31) and past-year treatment for an ear infection (OR = 1·35, 95 % CI 1·05, 7·21) were associated with a significantly elevated risk for having anaemia from other causes.

Discussion

The present study is the first to report population-level prevalence estimates of ID and IDA for Inuit preschoolers in the Nunavut Territory, Canada. National prevalence estimates for Canadian preschoolers are currently not available. However, in comparison with American preschoolers, Inuit preschoolers have a higher prevalence of ID and IDA. In the USA, ID affects 4·5 % of children aged 3–5 years and IDA is found among 0·5 %[19], whereas in the present study 10·8–18·0 % were iron deficient and 3·3–5·4 % had IDA, depending upon the cut-offs used. Natives of Alaska[16] as well as Canadian Inuit infants[6,7,9] have been observed to have higher rates of ID than the

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Table 2 Frequency of consumption of traditional and market food sources of iron among Inuit children aged 3–5 years: Nunavut Inuit Child Health Survey, 2007–2008

	Percentage of 3–5-year-olds who consumed the food in the past month		Average number of days consumed in a month	
	%	*n*/*N*	Consumers only	All children
Traditional foods				
Caribou meat (dried, cooked, raw)	84·2	320/380	11·8	10·0
Fish, all types	65·3	248/380	7·1	4·6
Ringed seal meat	49·5	188/380	4·3	2·2
Clams/mussels from the land	15·5	59/380	3·2	0·5
Goose	14·7	45/307†	2·9	0·4
Ringed seal liver	11·6	44/380	3·8	0·4
Duck, all types	10·8	41/380	2·2	0·2
Walrus meat	8·6	29/336†	2·0	0·2
Musk-ox meat	6·0	8/133†	1·7	0·1
Caribou liver	5·0	19/380	5·4	0·3
Beluga meat	4·5	17/380	2·5	0·1
Ptarmigan and spruce hen	3·7	14/380	2·3	0·1
Narwhal meat	2·9	11/380	6·0	0·2
Market foods				
Cereal, all types	96·3	366/380	28·9	27·8
Beef, all types	82·9	315/380	10·4	8·5
Poultry, all types	82·6	314/380	8·5	7·0
Pork, all types	69·7	265/380	8·0	5·6

†Owing to the limited geographical availability of this food, *N* is reduced because certain communities were not asked about this traditional food item.

general population[6,19]. The prevalence rates determined from the present study would most likely be defined as mild according to WHO thresholds for population-level ID[36]. However, improvements in iron status are possible given that rates of deficiency continue to be higher among Inuit preschoolers compared with preschoolers of the general US population.

Iron intake in this population is most likely adequate as only 0·3 % of children had intake below the EAR. In addition, breakfast cereals, many of which are iron-fortified, and beef and caribou were frequently consumed in this population. Among the same study population, only 0·1 % had intake below the EAR for vitamin C[28], suggesting that low iron bioavailability due to low vitamin C intake is unlikely. While over-reporting of portion sizes on 24 h dietary recalls is possible, the energy intake reported in the present study is similar to that observed in the Canadian Community Health Survey[37]. Dietary iron intake levels are also similar in the present study in comparison with others in Inuit, Métis and American children[11,12,38,39].

The finding that more boys than girls were iron deficient is difficult to explain since there are no sex differences in iron requirements in the preschool age group. It has recently been shown that obesity and being overweight are associated with greater risk of ID, perhaps due to low diet quality, increased iron requirement due to higher blood volume as well as decreased iron absorption induced by chronic low-grade inflammation[40–44]. In another analysis of the Inuit Child Health Survey, boys were noted to have higher BMI-for-age *Z*-scores than girls[27]. However, we found no significant associations between ID and BMI *Z*-scores, and BMI *Z*-scores did not alter the relationship between sex and ID in logistic regression. It is possible that the association between BMI *Z*-scores and ID is relevant to Inuit preschoolers, but our study was not powered to detect this perhaps weak or moderate association.

In addition, the present study used a 24 h dietary recall and a 20 % random subsample repeat recall, which does not provide enough information to estimate nutrient intake for individuals. As such, nutritional ID or anaemia cannot be ruled out. However, since meat and dietary iron intake was high in the population overall, nutritional causes are unlikely to explain the observed ID and anaemia found among preschoolers.

The 45·4 % prevalence of *H. pylori* exposure observed in the present study is high and consistent with other studies of Canadian First Nations and Inuit and Alaskan native children[6,16,24,25,45]. In contrast, lower rates of *H. pylori* exposure have been reported (5·5–7·1 %) for American and Canadian children[19,46]. We found no association between ID and *H. pylori* exposure among Inuit preschoolers. The present study is limited in that *H. pylori* infection assessment was related to previous exposure[47–49], thereby precluding comparisons with studies evaluating current infection. However, in various epidemiological studies and case reports in which *H. pylori* was shown to be independently associated with iron status, the association emerged primarily among older children[16,19,20–23,50]. For example, among the natives of Alaska, *H. pylori* was independently associated with ID for children aged 9 years and above, but not in younger age groups[16]. A causal relationship between *H. pylori* and iron status has yet to be established, but

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Table 3 Univariate analyses for correlates of iron deficiency (ferritin <10 μg/l) and anaemia from other causes: Nunavut Inuit Child Health Survey, 2007–2008

	Iron deficiency				Other anaemia			
	%	*n*/*N*	RR	95 % CI	%	*n*/*N*	RR	95 % CI
Age group (years)								
3–4	13·1	22/164	3·31	1·02, 10·71*	5·3	10/189	0·28	0·13, 0·60***
5	4·1	3/74			19·2	14/73		
Sex								
Male	16·1	19/118	3·22	1·33, 7·78*	6·5	8/123	1·77	0·79, 3·99
Female	5·0	6/120			11·5	16/139		
Helicobacter pylori exposure								
Positive	7·8	8/102	0·60	0·27, 1·33	8·4	10/119	0·82	0·38, 1·77
Negative	13·1	17/130			10·3	14/136		
Household size								
≥6	11·3	13/115	1·19	0·56, 2·55	12·3	16/130	2·56	1·04, 6·34*
<6	9·5	11/116			4·8	6/125		
Child food insecurity								
Yes	9·4	12/128	0·77	0·36, 1·63	9·1	13/143	1·23	0·53, 2·86
No	12·2	12/98			7·4	8/108		
Iron supplement								
Yes	8·6	3/35	0·80	0·25, 2·53	9·5	4/42	1·13	0·40, 3·16
No	10·8	21/195			8·5	18/213		
Attends daycare								
No	13·0	18/138	0·48	0·20, 1·16	10·3	16/155	0·76	0·34, 1·71
Yes	6·3	6/96			7·8	8/102		
Income support								
No	12·0	14/117	0·77	0·36, 1·65	7·0	9/129	1·41	0·62, 3·23
Yes	9·2	10/109			9·8	12/122		
Housing type								
Public	11·8	19/161	1·16	0·46, 2·94	7·4	13/176	0·61	0·26, 1·46
Private	10·2	5/49			12·1	7/58		
Extended family contact								
Daily/often	10·1	20/199	0·96	0·35, 2·64	8·1	18/221	0·53	0·22, 1·25
Sometimes/never	10·5	4/38			15·4	6/39		
Traditional food intake (times/month)								
≥19	8·9	11/124	0·75	0·35, 1·61	6·8	9/133	0·56	0·26, 1·24
<19	11·8	13/11			12·0	15/125		
Treated for ear infection (past 12 months)								
Yes	10·7	8/75	1·10	0·49, 2·47	14·5	12/83	2·20	1·01, 4·76*
No	9·7	15/154			6·6	11/167		
Breathing/respiratory illness (past 12 months)								
Yes	8·3	9/108	0·7	0·32, 1·54	10·4	12/115	1·24	0·58, 2·64
No	11·9	15/126			8·5	12/142		

*$P < 0·05$; ***$P < 0·001$.

possible mechanisms involve bacterial damage to gastric glandular tissue and competition for iron in the stomach[13,15,18,51–53]. In addition to the limitations explained above, perhaps the young age of the study group is relevant in explaining the lack of association between iron status and previous *H. pylori* exposure.

Although 10·8–18·0 % prevalence of mild ID warrants continued public health attention, it is reassuring that the prevalence of IDA, the most severe form of ID, is low. However, apart from sex, we found no significant correlates of iron status in Inuit preschoolers in Nunavut. Iron absorption is one relevant issue that was not explored in the present study. It has been postulated that the Inuit may have adapted to excessive and deleterious iron intake through lowered iron absorption, which may now have implications for low iron stores given the nutrition transition away from iron-rich traditional foods[54]. However, little evidence is currently available to support or refute this hypothesis[16,54].

Finally, in the present study, anaemia from all causes was found in 16·8 % of Inuit children and only 20–31 % of this anaemia was explained by low iron status. Other studies in children and infants have shown similar results in which only a portion of the observed anaemia is explained by ID[55–57]. Dietary causes of anaemia other than low iron intake include deficiencies in vitamin A, folate, vitamin B_{12} and riboflavin[58]. However, again, given the high meat and cereal intake noted, most of these micronutrient deficiencies are unlikely. Vitamin A deficiency could be evaluated in future research given that an earlier study found that young Inuit adults had a greater likelihood of having a retinol activity equivalent falling below the EAR than older Inuit adults, attributed to the changing pattern of traditional food consumption by age[59]. However, in analyses of the present study population, the majority of children had a vitamin A intake above the EAR[28].

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Anaemia not attributable to ID could be related to acute inflammation. In acute infection, inflammation reduces erythrocyte half-life and the acute phase response blocks iron export proteins trapping iron inside cells[60–62]. Interestingly, we found that children who required treatment for an ear infection in the past year, when compared to those who did not, had a significantly greater risk for being anaemic. Ear and respiratory infections are common among Inuit children[63,64]. One hypothesis is that recurring infections during childhood may result in mild anaemia, although the reverse may also occur, where anaemia may increase the risk of infection[65,66]. We also found that household crowding significantly predicted anaemia. Household crowding is a relevant health indicator for young children who spend the majority of their time at home with increased risk for person-to-person spread of infections[63,67–69].

Limitations

Owing to the rarity of IDA in this population, our study was underpowered to detect significant correlates. The cross-sectional design prevents exploration of causal relationships between correlates and ID, IDA and anaemia.

Conclusion

Inuit children aged 3–5 years have higher rates of ID, IDA and anaemia than non-aboriginal children. The rates observed, however, were not excessively high and do not warrant immediate intervention. The present study also revealed that ID explains only 20–31 % of low Hb and that anaemia was generally mild. The role of household crowding, history of infections and age need to be evaluated for their role in exacerbating anaemia. The study found that Inuit preschoolers have iron intake levels that are most likely adequate for their age, which is an important positive finding for children likely to be exposed to rapid nutrition transition in the Arctic. Health promotion efforts are best placed at encouraging beneficial dietary behaviours that already exist in Nunavut's communities and in extending these beneficial behaviours in efforts targeting high-risk women of reproductive age and infants. Caregivers should be commended for feeding preschoolers many different iron-rich foods, especially iron-rich traditional meats, and encouraged and supported to continue these practices while reducing intake of foods that are less nutrient-rich but contribute to high energy intake.

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