

To Supplement or Not to Supplement: Are Inuit Getting Enough Vitamin D?

Fournir ou ne pas fournir de suppléments. Existe-t-il une carence en vitamine D chez les Inuit ?

Peter Frost

Volume 40, Number 2, 2016

URI: <https://id.erudit.org/iderudit/1055442ar>

DOI: <https://doi.org/10.7202/1055442ar>

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Publisher(s)

Centre interuniversitaire d'études et de recherches autochtones (CIÉRA)

ISSN

0701-1008 (print)

1708-5268 (digital)

[Explore this journal](#)

Cite this article

Frost, P. (2016). To Supplement or Not to Supplement: Are Inuit Getting Enough Vitamin D? *Études Inuit Studies*, 40(2), 271–291.
<https://doi.org/10.7202/1055442ar>

Article abstract

Inuit have vitamin D blood levels that generally fall within the range of insufficiency, even when they live on a traditional diet of fish and game meat. Without this vitamin, bones soften and become deformed, a condition called rickets in children and osteomalacia in adults. Until recent times, however, this condition was much rarer among Inuit than among non-Inuit, even when the latter included people living near Inuit communities under similar conditions of climate and housing. This rarity was attributed to extended breastfeeding and a high-meat/low-cereal diet. The situation subsequently reversed, with Inuit becoming more at risk of developing rickets, first in Labrador during the 1920s and later elsewhere. To reduce this excess risk, researchers have recommended vitamin D supplementation, arguing that breast milk has too little vitamin D and that even a traditional diet cannot provide the recommended daily intake. We should ask, however, whether the problem is definitional. Inuit may have lower levels of vitamin D because they need less, having adapted culturally and physiologically to an environment where this vitamin is less easily synthesized in the skin. These adaptations include a diet that enhances calcium bioavailability (by means of β -casein in breast milk, certain unknown substances in meat, and absence of phytic acid), as well as genetic changes that enable vitamin D to be used more efficiently. Although Inuit are today more at risk of developing rickets than are non-Inuit, this excess risk is nonetheless small and seems to have a dietary cause—namely, early weaning and abandonment of a high-meat/low-cereal diet.

To Supplement or Not to Supplement: Are Inuit Getting Enough Vitamin D?

Peter Frost¹

ABSTRACT

Inuit have vitamin D blood levels that generally fall within the range of insufficiency, even when they live on a traditional diet of fish and game meat. Without this vitamin, bones soften and become deformed, a condition called rickets in children and osteomalacia in adults. Until recent times, however, this condition was much rarer among Inuit than among non-Inuit, even when the latter included people living near Inuit communities under similar conditions of climate and housing. This rarity was attributed to extended breastfeeding and a high-meat/low-cereal diet. The situation subsequently reversed, with Inuit becoming more at risk of developing rickets, first in Labrador during the 1920s and later elsewhere. To reduce this excess risk, researchers have recommended vitamin D supplementation, arguing that breast milk has too little vitamin D and that even a traditional diet cannot provide the recommended daily intake. We should ask, however, whether the problem is definitional. Inuit may have lower levels of vitamin D because they need less, having adapted culturally and physiologically to an environment where this vitamin is less easily synthesized in the skin. These adaptations include a diet that enhances calcium bioavailability (by means of β -casein in breast milk, certain unknown substances in meat, and absence of phytic acid), as well as genetic changes that enable vitamin D to be used more efficiently. Although Inuit are today more at risk of developing rickets than are non-Inuit, this excess risk is nonetheless small and seems to have a dietary cause—namely, early weaning and abandonment of a high-meat/low-cereal diet.

RÉSUMÉ

Fournir ou ne pas fournir de suppléments. Existe-t-il une carence en vitamine D chez les Inuit ?

Les Inuit affichent un taux sanguin de vitamine D se situant généralement dans la zone d'insuffisance, même lorsqu'ils mangent une alimentation traditionnelle de poisson et de gibier. Sans cette vitamine, les os se ramollissent et se déforment, condition appelée « rachitisme » pendant l'enfance et « ostéomalacie » à l'âge adulte. Cependant, jusqu'à une période récente, cette condition était beaucoup plus rare chez les Inuit que chez les non-Inuit, même lorsque ces derniers incluaient des personnes vivant tout près des communautés inuit et soumis à des conditions similaires de climat et de logement. On attribuait cette rareté à l'allaitement prolongé et à une alimentation riche en viande et

i. Independent researcher, Québec, Québec. peter_frost61z@globetrotter.qc.ca

pauvre en céréales. C'est par la suite que la situation s'inverse, car les Inuit deviennent plus à risque de développer le rachitisme, d'abord au Labrador pendant les années 1920 et ailleurs ensuite. Pour réduire ce sur-risque, des chercheurs recommandent une supplémentation quotidienne en vitamine D, en soutenant que le lait maternel serait trop pauvre en vitamine D et que même l'alimentation traditionnelle serait insuffisante pour fournir l'apport quotidien recommandé. Mais il faut se demander si cette carence est illusoire. Il se peut que les Inuit affichent un faible taux sanguin de vitamine D parce qu'ils en ont moins besoin, s'étant adaptés tant sur le plan culturel que sur le plan physiologique à un milieu où cette vitamine se synthétise difficilement dans la peau. Ces adaptations incluraient une alimentation qui augmente la biodisponibilité du calcium (grâce à la β -caséine du lait maternel, à certaines substances inconnues dans la viande et à l'absence d'acide phytique), ainsi que des modifications génétiques permettant une meilleure utilisation de la vitamine D. Si, de nos jours, les Inuit sont plus à risque de développer le rachitisme que les non-Inuit, ce sur-risque est néanmoins faible et semble avoir une cause alimentaire, à savoir le sevrage précoce et l'abandon d'une alimentation riche en viande et pauvre en céréales.

“**Y**ou need a vitamin D tablet every day during pregnancy and breastfeeding, no matter what season it is” (Government of Nunavut 2017). This is the advice given to women by the Nunavut Department of Health. Similar advice is offered by Yukon Health and Social Services (Government of Yukon 2017). In a position statement, the Canadian Paediatric Society makes the case for vitamin D supplementation in northern regions:

People in northern regions are particularly vulnerable to deficiency. For example, from October to March in Edmonton, Alberta (52° north), vitamin D₃ production in the skin is almost nonexistent (evidence level II-2). The situation is potentially even worse in the high Arctic, where many Aboriginals live. (Godel 2007: 584)

Are Inuit and other northern peoples getting enough vitamin D? To answer this question, we must understand the tasks that this vitamin performs in the human body and the context in which it performs them; that is, it may be assisted or hindered by other ingested substances and by cultural practices in the family or the community. We must also understand that human bodies differ in the way they metabolize vitamin D. Whereas populations of European descent show a progressive decline in blood levels of vitamin D with increasing latitude, this latitudinal variation disappears when we look at *Homo sapiens* as a whole. Some of the lowest levels exist in people native to the tropics (Hagenau et al. 2009; Frost 2012). Human bodies differ not only in their blood levels of vitamin D but

also in their ways of ensuring that enough remains available. In some populations, this vitamin is stored in large amounts during the summer for use in winter and spring; in others, it is used parsimoniously year-round (Frost 2012). The same physiological problem seems to be resolved in different ways in different populations.

Vitamin D, northern latitudes, and the Inuit

Vitamin D helps the body to absorb bone-building minerals, especially calcium but also magnesium, phosphorus, and zinc, from food passing through the intestines. It is thus essential to the development of strong bones and teeth. Without it, the skeleton softens and can become deformed, a condition called rickets in children and osteomalacia in adults. The skin makes this vitamin from cholesterol when exposed to sunlight, specifically the UVB component (280-315 nm). UVB contributes less to sunlight at northern latitudes because the atmosphere screens more of it out—the sun’s rays enter the atmosphere at a lower angle and follow a longer trajectory before reaching the earth’s surface. Based on rates of biosynthesis calculated from American subjects, the average daily dose of UVB north of 50°N in North America does not meet minimum physiological requirements over the course of a year (Jablonski and Chaplin 2000).

At such latitudes biosynthesis is insufficient, and northern peoples like the Inuit have to rely much more on dietary sources, particularly fatty fish, seal meat, and whale meat (Brunborg et al. 2006). But those sources, too, seem insufficient. According to a study conducted in Nunavut and the Northwest Territories, the median vitamin D intake was 5.13 ± 5.34 µg/day for Inuit women on a traditional diet (≥ 300 g of fish or game meat per day) and 3.5 ± 3.22 µg/day for Inuit women on a non-traditional diet (< 300 g). Both groups received much less than the recommended intake of 15 µg/day (Kolahdooz et al. 2013).

This low intake is reflected in low blood levels of 25(OH)D, the main metabolite of vitamin D. In a study of West Greenland Inuit living on a traditional diet (seal or whale meat at least once a week), these levels ranged on average from a high of 52 nmol/l in summer to a low of 42 nmol/l in winter (Rejnmark et al. 2004). Another study found a level of 68 nmol/l in East Greenland Inuit whose diet was over 80% from traditional meat sources; however, seasonal variation was not investigated, apparently because the authors assumed that no biosynthesis took place in the skin during summer (Andersen et al. 2013). In any case, all of these blood levels are considered insufficient by the Canadian Paediatric Society, which sets the bar for insufficiency at ≤ 75 nmol/L and deficiency at < 25 nmol/L (Godel 2007). A recent review of the literature has likewise set the minimum for optimal health at 75 nmol/l (Bischoff-Ferrari 2014).

It seems, then, that most Inuit are not getting enough vitamin D. This is in fact the position of the Nunavut government, as stated by Dr. Geraldine Osborne, currently the deputy chief medical officer of health, in a CBC interview:

About 90 per cent of Nunavut's population is vitamin D deficient, according to preliminary results from *Qanuipitali*, a field study being conducted by Nunavut's Health and Social Services Department. The study, which is still being compiled, also found that only seven per cent of pregnant women in Nunavut's Baffin Island region are getting enough vitamin D. Osborne said that low rate of vitamin D intake among pregnant women is a major factor behind a high rate of rickets—a softening of bones that leads to fractures and deformities—in children in the territory (CBC News 2010; see also El Hayek 2011).

Past and present evidence for vitamin D deficiency among the Inuit

If, on average, vitamin D levels are insufficient even in Inuit living on a traditional diet, a significant proportion of Inuit children should develop rickets. Yet few signs of rickets were absent until well into the twentieth century. There were none in an examination of 374 prehistoric skeletal remains dating from 100 BCE to 1700 CE at Point Hope, Alaska (Dabbs 2011). Between 1903 and 1908, a physician in Okak, Labrador, saw no signs of rickets in Inuit children, although settler children often had the disease (Hanrahan 2008). In 1925, a review article affirmed, without providing a source, that “the Eskimo baby is free from it [rickets], even though he lives in a dark hut, because he is suckled by a mother who consumes great quantities of animal fat and oil” (Craig and Belkin 1925: 542). In 1936–1937, Inuit examined at Angmagssalik, Greenland, showed no signs of rickets (Høygaard 1941). During the mid- to late 1930s, the teeth and jaws of living and dead Greenland Inuit were checked for signs of rickets but none were found (Pedersen 1947). In 1935, an investigation of Inuit in the eastern Canadian Arctic reported “no definite evidence of rickets either in the Straits and Bay or in the more northerly regions (there were 3 suspicious cases at Pangnirtung)” (Rabinowitch 1936: 494). In 1953, a review article stated that “it is generally agreed that rickets and scurvy are unknown [among the Canadian Inuit], the former despite the meagre sunlight and pigmentation of the skin” (Sinclair 1953: 77). These reports are consistent with those from other northern peoples at that time. In the 1930s, a study of Sami children discovered only one case of rickets, in contrast to a much higher rate among children of Finnish and Swedish parents residing in Lapland (Schehk 1936).

Rickets was first reported among Inuit in a 1919–1920 study on the Labrador side of the Strait of Belle Isle; only two cases were found, and the author concluded that “there is very little rickets” (Appleton 1921: 619). The

situation had changed by 1926, when another study found rickets to be absent in Greenland but widespread among Labrador Inuit (Thomas 1927). Between 1972 and 1984, in northern Manitoba, 40 cases (38 Indians and 2 Inuit) were reported among Native children (Haworth and Dilling 1986). Between 2002 and 2004, in Canada as a whole, 104 cases of vitamin D deficiency rickets were reported, 12% of whom were Inuit and another 13% First Nations (Ward et al. 2007). Between 2001 and 2010, the rate of rickets was almost twice as high among Alaskan Native children as among American children in general (Singleton et al. 2015).

Rickets thus appeared first among Labrador Inuit in the 1920s and then much later in other Inuit groups. This increase in prevalence has been attributed to two dietary shifts:

- a shift from extended breastfeeding (i.e., two years or longer after birth) to use of infant formula and early weaning (Craig and Belkin 1925; Hanrahan 2008; Høygaard 1941; Rabinowitch 1936). At later ages, Inuit mothers also used to pre-chew game meat and then feed it to their children. It is unclear when traditional child feeding was abandoned. According to a Nunavut survey conducted in 2007 and 2008, only 16% of Inuit mothers exclusively breastfed their children for more than 6.5 months (McIsaac et al. 2014).
- a shift from a traditional diet of fish and game to store-bought food, particularly cereals (Thomas 1927). Although “country food” is still popular, the reality is that store-bought food now accounts for most of the energy intake of modern Inuit, particularly in younger age groups (Blanchet et al. 2000; Kuhnlein et al. 2004).

For early observers, the main protective factor was extended breastfeeding:

Among these primitive, carnivorous people there is neither scurvy nor rickets. Children are nursed for four and not infrequently six years; that is, until their teeth are strong enough to eat meat. Both my own observations and the results of most careful questioning of the Danish physicians are definite and convincing on this point. (Thomas 1927: 1560)

The reason, however, for the failure to find rickets at the above-mentioned places appears to be that infants are nursed for as much as two years and more, and the fact that the vitamin D content of seal oil is equal to that of the best cod-liver oil. According to Dr. Urquhart, infants are nursed for very long periods of time amongst the Western Arctic Eskimos also. One of the mothers demonstrated to me that her milk supply was still plentiful, though she was still nursing her child who, according to the interpreter, was now 2½ years old. (Rabinowitch 1936: 494)

It is stated that rickets is practically unknown (1 case seen) amongst Lapp children in spite of the short summer and poor dwellings. They are suckled for periods for about 10.5 [months] to 4 years, with supplements of meat, frozen or dried in the sun during the summer, and butter. In contrast, the children of Finnish and Swedish parents, working at the mines in Lapland, under similar climatic but excellent housing conditions, show a high incidence of rickets. They are suckled for only 3–4 months and are then given undiluted cow's milk. (Schehk 1936: 1395)

One observer also pointed to the traditional Inuit diet of raw meat with no cereals or cereal products:

In Labrador, however, a distressing condition prevails. There the natives have been for many years in somewhat close contact with the influence of civilization, and unfortunately have abandoned their primitive methods of existence. Other factors, however, are at work. Wood is abundant, so they cook their meat. The Moravian missions and the Hudson Bay Company, with the best of intentions, take their furs and sell them provisions—dried potatoes, flour, canned goods, cereals and cereal products—and their fare consists largely of these staples. And Labrador, while not truly arctic, offers very little return for the labor of a gardener, so there is no available source of vegetable food. Consequently, scurvy, rickets and combinations of the two are universal. (Thomas 1927: 1560)

Not until later did reports of rickets come from Inuit farther north and west (Haworth and Dilling 1986; Ward et al. 2007; Singleton et al. 2015). By that time, almost a half-century later, there seems to have been little awareness of the earlier literature. Haworth and Dilling (1986), working with First Nations and Inuit from northern Manitoba, blamed lack of direct sunlight, heavy outdoor clothing, and cultural resistance to cow's milk and vitamin supplements:

There are few hours of direct sunlight in winter in northern Canada, and the low temperatures necessitate heavy outdoor clothing. Even in summer, native infants traditionally are either kept indoors or protected from sun, wind and insects. Dietary sources of vitamin D may have been more available before the acculturation of native people, but now they are very limited. Cow's milk is one of the few good sources of the vitamin, but adult natives drink little of it; in the Island Lake area they drink almost none. Cultural custom, the unavailability of fresh milk and the high incidence of lactose intolerance among adult Indians seem to be the main factors responsible. Despite efforts to encourage pregnant native women to take vitamin supplements, compliance has been poor. (Haworth and Dilling 1986: 240)

While acknowledging that abandonment of the pre-contact traditional diet might be a factor, Haworth and Dilling (1986) argued that “dietary intake of vitamin D is a relatively inefficient and unnatural way of supplying the vitamin.” They also noted that “human milk is an inadequate source of vitamin D.” All of the infants they examined, however, were bottle-fed on cow’s milk that contained 400 IU of vitamin D per quart. Two decades later, Ward et al. (2007) likewise discounted breast milk as a means to prevent rickets:

Breast milk typically contains about 25 IU or less vitamin D per litre, which is insufficient for rickets prevention. Although there is evidence that limited sun exposure may prevent rickets in some breast-fed infants, concern over the health risks of sun exposure have led to the recommendation that all breast-fed infants receive supplemental vitamin D (400 IU/d). It is recommended that breast-fed infants who reside above the 55th latitude in Canada or in areas at lower latitudes that have a high incidence of vitamin D deficiency receive 800 IU/d during the winter months. (Ibid.: 2007: 165)

With the recent study by Singleton et al. (2015) on Alaskan Natives, the medical literature seems to have come full circle, and now cites exclusive breastfeeding as a risk factor: “Risk factors for developing vitamin D deficiency and nutritional rickets in children besides low sun exposure, include exclusive breast-feeding (since breast milk is low in vitamin D)” (Singleton et al. 2015: 821). This conclusion, however, is based on the general vitamin D literature and not on any studies of northern Indigenous Peoples. The authors themselves stated, “In our study [of Alaskan Natives], a history of documented breastfeeding was not associated with an increased risk of rickets/vitamin D deficiency” (2015: 821).

Thus, since the 1980s, the medical literature has argued that Inuit, particularly children and pregnant women, need vitamin D supplements. This argument is based on the presumed inadequacy of natural sources:

- Inuit cannot make enough vitamin D on their own by exposing their skin to sunlight because the sun’s rays have too little UVB most of the time (Singleton et al. 2015). Heavy clothing further blocks biosynthesis (Haworth and Dilling 1986).
- Inuit cannot get enough vitamin D from their diet. Traditional country food is richer in vitamin D, but even that source cannot provide the recommended daily intake (Kolahdooz et al. 2013). The situation is worsened by breastfeeding, which is a poor source of vitamin D (Ward et al. 2007; Singleton et al. 2015; Streym et al. 2016).

Yet there was a time in the recent past when Inuit rarely developed rickets, even though they were exposed to the same low-UVB sunlight, wore the same heavy

clothing, were breastfed much longer and in a higher proportion than is the case today, and ate country food now judged to be an insufficient source of vitamin D. This rarity of rickets is well documented in the medical literature of the early to mid-twentieth century, but that literature is not referenced in recent studies on vitamin D and the Inuit. Current researchers either are unaware of this earlier literature or feel that it does not meet modern standards of scientific evidence. Because earlier authors could not measure vitamin D in the food or in the blood, their mode of enquiry was often subjective (e.g., personal observation and questioning of local physicians). Rickets, however, is not a subjective state and was reliably diagnosed even a century ago. Its absence among Inuit until recent times weakens the case for vitamin D supplementation.

In sum, we can conclude that rickets in Canada is today more common among Inuit than among non-Inuit, whereas the reverse used to be true. This conclusion should be treated with some caution because earlier observers were comparing the situation among Inuit with the very high incidence of rickets that prevailed throughout the Western world until the postwar era. The lower incidence among Inuit was attributed at that time to extended breastfeeding and a diet of fish and game. Recent medical researchers have ignored these possible factors, noting that breast milk is low in vitamin D and that the vitamin D content of a traditional meat-based diet, though higher than that of store-bought food, cannot supply the recommended daily intake. These facts have apparently convinced policy makers that it is not enough to promote breastfeeding and country food: vitamin D supplementation is also needed.

How serious is this problem among Inuit today?

Before proceeding with mass supplementation, we should ask whether this kind of intervention is warranted. Is the problem sufficiently serious? It seems so at first glance. In 2010, a field study for Nunavut Health and Social Services concluded that about 90% of Nunavut's population was vitamin D deficient (CBC News 2010). The picture changes, however, when we ask how many Inuit suffer harmful effects, notably rickets in children and osteomalacia in adults. In Alaska, Native children are hospitalized for rickets at an annual rate of 0.00223%, versus 0.00123% for American children in general (Singleton et al. 2015). Both of these rates are very low by historical standards. As recently as 1943, 12 to 14% of British infants were diagnosed with rickets, according to a British Paediatric Association survey (Stewart et al. 1964). Rickets is today classified as a rare disease in most Western populations, and the Inuit are no exception.

To be fair, rickets is not the only harmful effect attributed to vitamin D deficiency or insufficiency, many others having in fact been cited in the literature (see, e.g., Godel 2007). Only one of them, a higher risk of respiratory infection, has been studied among Inuit in relation to vitamin D. When 388 Inuit preschool children were examined in Nunavut, no association was found between respiratory

infections and vitamin D deficiency (< 25nmol/L) or vitamin D insufficiency (25 nmol/L to 75 nmol/L) (Tse et al. 2016). While all possible effects of vitamin D deficiency or insufficiency should be investigated, it would be surprising to find that some of them are common when the main one, rickets, is so rare. The problem may thus be definitional. We may be applying the terms “deficient” or “insufficient” to people who in reality are neither.

The need for vitamin D can vary with diet

When Nunavut Health and Social Services concluded that about 90% of Nunavut’s population was vitamin D deficient, it was using Canadian Paediatric Society norms, which in turn are based on data from southern Canada, the United States, and Great Britain. These norms reflect the physiological needs of populations whose diet is not necessarily the same as that of Inuit in Nunavut. This point is important because the body’s need for vitamin D is reduced considerably by three aspects of the traditional Inuit diet: extended breastfeeding, high meat consumption, and low cereal consumption.

Extended breastfeeding

One component of breast milk is β -casein, which helps to keep calcium soluble during digestion and contributes greatly to the high bioavailability of this element in breast milk (Lönnerdal 2003). β -casein thus reduces the body’s need for vitamin D, whose main function is to assist the absorption of calcium, magnesium, phosphorus, and zinc from food passing through the intestines. Breast milk may be low in vitamin D because less is needed to produce the same effect.

High meat consumption

A British study found that meat decreases the risk of rickets and osteomalacia independently of its vitamin D content. This inverse association with meat eating (grams ingested per day) seems to be curvilinear, the risk of rickets not falling any further at meat intakes of 60 g daily. Meat eating seems to reduce this risk more effectively in women than in children. No one has yet identified the meat component that produces this effect (Dunnigan et al. 2005).

Low cereal consumption

The same British study found that fibre independently increases the risk of rickets and osteomalacia (Dunnigan et al. 2005). The causal factor seems to be phytic acid, which is found in the hulls of nuts, seeds, and grains, and is most commonly ingested from store-bought bread. Phytic acid binds to minerals to form insoluble precipitates, thereby depleting the body’s usable supply of calcium and other essential minerals. The body needs more vitamin D because less calcium is available in a usable form.

The association between this disease risk and cereal consumption has been shown in controlled animal and human studies. When puppies were fed an oatmeal diet, they developed rickets, the severity of which was in proportion to the phytic acid content of their diet (Harrison and Mellanby 1939). Similarly, human subjects absorbed less calcium, magnesium, phosphorus, and potassium on a 92% flour diet than on a 69% flour diet (McCance and Widdowson 1942a). Absorption of these elements was inversely proportional to the percentage of phytic acid in their flour diet (McCance and Widdowson 1942b).

The need for vitamin D can vary because of physiological differences

The body's vitamin D requirements have been estimated overwhelmingly from subjects of European descent, and yet, as the National Academy of Medicine, USA, notes, "there may be some biological differences in the way in which different ethnic/racial groups respond to calcium and vitamin D" (Ross et al. 2011: 347). Similarly, Hagenau et al. (2009) attribute differences in vitamin D levels not only to cultural practices (sun avoidance, diet, etc.) but also to genetic adaptations:

However, genetic adaptation apart from skin colour may also influence vitamin D metabolism. Inuits appear to have developed an enhanced renal conversion of 25(OH)D to 1,25(OH)₂D improving the utilization of available 25(OH)D. In contrast, Asian Indians have developed (or maintained) an increased renal 24,25(OH)₂D-hydroxylase activity facilitating the production of the inactive 24,25(OH)₂D at the expense of 1,25(OH)₂D. (Ibid.: 138; see also Rejnmark et al. 2004)

Modern humans entered northeastern Asia and Beringia about 20,000 to 15,000 years ago (Hoffecker, and Elias 2007: 18). If we assume a mean generation time of 25 years, the Inuit and their ancestors have lived at high northern latitudes for 600 to 800 generations. During that time their bodies have adapted to the cold in various ways: shorter limbs in relation to the trunk to conserve body heat (Yeats 2011; Foster and Collard 2013); shorter and stockier fingers for the same purpose (Lazenby and Smashnuk 1999; Betti et al. 2015); faster recovery of peripheral body temperature after cold exposure (Andersen et al. 1963; Livingstone et al. 1978); smaller maxillary sinus to reduce exposure to cold air (Shea 1977); and higher increases in basal metabolic rate in response to severe cold (Leonard et al. 2002). Inuit bodies have also adapted to a diet high in polyunsaturated fatty acids (Fumagalli et al. 2015), low in carbohydrates, and high in protein (Draper 1977). Finally, the association between body fat and risk of heart disease is weaker in Inuit than in Euro-Canadians (Young 1996; Smith et al. 2009). The cardioprotective effect seems to be due to higher HDL levels, which in turn are associated with an allele at the *CPT1A* gene (Lemas et al. 2012).

In addition to adapting to the cold and a high-meat diet, Inuit bodies have adapted to a low-UVB environment where vitamin D cannot easily be made in the skin. We have seen that Inuit have adapted to these circumstances through extended breastfeeding and a diet that is optimal for calcium, magnesium, phosphorus, and zinc absorption. Have they also adapted physiologically?

There is good evidence that they have, by using vitamin D and calcium more efficiently. According to a study of Nunavik children, Inuit need less vitamin D because their bodies need less calcium.

Recommended elemental calcium intakes for North American children are 800 mg/d for those 4 to 8 years old and 1300 mg/d for those 9 years of age and older. With a traditional diet, Inuit children in northern Canada ingest only 20 mg of elemental calcium per day. After presumably adapting to this constraint over millennia, the Inuit are now adopting a more southern “market” diet, with appreciably higher calcium intakes. (Sellers et al. 2003)

The study found that the children excreted excess calcium at an unusually high rate despite low blood calcium levels. They seemed to be absorbing calcium more efficiently from their diet, in part because their vitamin D receptor (*bb* genotype) is associated with more efficient intestinal calcium absorption (Sellers et al. 2003).

A study of Greenland adults has described two other genetic adaptations. First, Inuit have a lower set-point for calcium-regulated release of parathyroid hormone. Second, Inuit convert vitamin D at a higher rate from its common form, 25(OH)D, to its most active form, 1,25-dihydroxyvitamin D: “Due to a low endogenous 25OHD production in Greenlanders ... evolution may have selected individuals with a relatively high 25-hydroxyvitamin D1 α -hydroxylase activity” (Rejnmark et al. 2004: 261). The conversion rate may be even higher during prenatal and neonatal life, when bone growth is most critical. An Inuvik study found that Inuit and native Indian newborns had 25(OH)D levels that were only 67% of their mothers’ levels. Yet they showed no clinical evidence of vitamin D deficiency, and, more surprisingly, their blood calcium levels were significantly higher than their mothers’ levels (Waiters, Godel, and Basu 1999).

To varying degrees, similar genetic adaptations may exist in Amerindian peoples, who originally lived at high latitudes, and in many cases still do. This may explain the apparent paradox of Amerindian women having lower levels of 25(OH)D than Euro-American women while having higher bone mass until menopause (Perry et al. 1998; Weiler et al. 2007).

Should there be mass supplementation?

More research is always needed, but current knowledge does not justify encouraging Inuit to take vitamin D supplements. First, we find little evidence of vitamin D deficiency if we look for the expected harmful effects, such as rickets,

osteomalacia, and risk of respiratory infection. One can counter that Inuit have a rate of rickets almost twice that of non-Inuit, but in both cases the rate is very low. There may be other harmful effects, but none have been shown to date.

Second, dietary supplementation is not without its own risks. Unlike vitamin C, vitamin D is not water-soluble, and excess amounts will not be excreted in urine; it is fat-soluble, and any excess will tend to accumulate (DeLuca 1978: 114). In addition, it acts more like a hormone than a vitamin, regulating not only uptake of calcium, magnesium, phosphorus, and zinc but also many other aspects of human physiology in ways still poorly understood (DeLuca 1978: 81-114; Tuohimaa 2009; Tuohimaa et al. 2009). In theory, such regulation should be optimal within a certain range of concentrations, with adverse effects increasing as one moves beyond this range in either direction (i.e., a U-shaped response curve).

There is broad agreement that a U-shaped response curve describes the relationship between vitamin D blood levels and various disease risks (Shroff et al. 2010). A Swedish study found that the total mortality rate was about 50% higher among men in the lowest 10% of blood levels (< 46 nmol/L) and the highest 5% (> 98 nmol/L) than among men in between (Michaëlsson et al. 2010). According to a Finnish study, the risk of prostate cancer increased below 40 nmol/L and above 60 nmol/L (Tuohimaa 2008; Tuohimaa et al. 2009). In women from the United States, Finland, and China, mortality for seven types of cancer (endometrial, esophageal, gastric, kidney, non-Hodgkin's lymphoma, pancreatic, ovarian) increased below 45 nmol/L and above 124 nmol/L (Helzlsouer et al. 2010). Another transnational study reported a higher risk of pancreatic cancer above 100 nmol/L (Stolzenberg-Solomon et al. 2010). The Framingham Heart Study concluded that cardiovascular disease risk increased below 50 nmol/L and above 62.5 nmol/L, while the NHANES III found higher mortality from all causes above 122.5 nmol/L (Davis 2009). Finally, a Danish study found mortality from all causes to be lowest at 50 to 60 nmol/L, with higher mortality below 10 nmol/L and above 140 nmol/L (Durup et al. 2012). A similar response curve was found for risk of death from cardiovascular disease, which was lowest at 70 nmol/L and highest at 12.5 nmol/L and 125 nmol/L (Durup et al. 2015).

Perhaps most worrisome, studies on mice have shown a U-shaped response curve for the aging process; that is, premature aging is associated with both too little and too much vitamin D (Tuohimaa 2009; Tuohimaa et al. 2009). Human evidence is limited. During the early years of vitamin D supplementation after the Second World War, very high doses were administered to East German children, who subsequently fell victim to hypercalcemia, early aging, cardiovascular complications, and early death (Tuohimaa 2009).

In theory, this U-shaped response curve should shift leftward if the body uses vitamin D more efficiently, as seems to be the case with Inuit. Their zone of optimum health should therefore cover a lower range of vitamin D levels. If

this reasoning holds true, Inuit should begin to suffer toxic effects at levels that are too low to harm Euro-Canadians.

Because of the above findings, the World Health Organization (WHO) has revised its vitamin D guidelines, stating that “Vitamin D supplementation is not recommended for pregnant women to improve maternal and perinatal outcomes” (WHO 2018b) As for children and infants, “further research is needed before specific recommendations can be made” (WHO 2018a, 2018c). In the United States, the National Academy of Medicine, USA (formerly the Institute of Medicine), has likewise joined the camp of vitamin D skeptics:

Of great concern recently have been the reports of widespread vitamin D deficiency in the North American population. Based on this committee’s work and as discussed below, the concern is not well founded. In fact, the cut-point values used to define deficiency, or as some have suggested, “insufficiency,” have not been established systematically using data from studies of good quality. (Ross et al. 2011: 480)

Although the committee’s decisions for the ULs [tolerable upper intake levels] made use of emerging data concerning a U-shaped (or perhaps reverse-J-shaped) curve for risk, which suggested adverse effects at levels much lower than those associated with hypervitaminosis D, the lack of data on the safety of higher intakes of vitamin D when used chronically is very concerning. (*Ibid.*: 486)

A foolproof mechanism, until vitamin D tablets came along

Vitamin D cannot rise to toxic levels if obtained only from a traditional diet or from biosynthesis in the skin. Levels in the body are normally kept within a narrow range, apparently by some kind of homeostatic mechanism. This mechanism was postulated by Holick (1985), and its existence has been confirmed in a Finnish study. In response to a standardized dose of UVB, vitamin D levels rose if they had been lower than 90 nmol/L and fell if they had been higher than 90 nmol/L (Karppinen et al. 2017). The body seems to respond to an excess of vitamin D by synthesizing less in the skin and by storing more in fatty tissues.

This homeostatic mechanism worked quite well until it became possible to provide high doses of vitamin D in an easily ingestible form. Today, we can immerse human tissues in concentrations of vitamin D that previously were difficult to achieve and even harder to maintain. This is a radical change, and it is all the more radical for bodies that previously were accustomed to low-level concentrations. Homeostasis can no longer be maintained if high doses are taken daily. The self-stabilizing mechanism is overwhelmed.

There is some evidence that Inuit bodies react adversely to concentrations of vitamin D that are considered safe by North American and European standards. A recent study of Greenland Inuit found a positive linear association between blood levels of vitamin D and risk factors for type 2 diabetes, *i.e.*, higher glucose levels and lower beta-cell function (Nielsen et al. 2016). This dose-response curve seems to hold true even at concentrations as low as 40 nmol/L, a level considered very insufficient for Euro-Canadians.

Conclusion

Traditionally, Inuit coped with vitamin D scarcity through certain dietary practices: extended breastfeeding of children for two years or longer, and a high-meat and low-cereal diet. They consumed not only the few sources of vitamin D available (seal meat, whale meat, fatty fish) but also substances that helped them to use vitamin D efficiently (β -casein in breast milk, unknown substances in meat, foods low in phytic acid).

Inuit also coped through certain genetic adaptations: receptors that bind more tightly to the vitamin D molecule; a lower set-point for calcium-regulated release of parathyroid hormone; and conversion of vitamin D at a higher rate from its common form to its most active form. There may be others, including, perhaps, higher levels of β -casein in breast milk. All of these genetic adaptations arose through a recurring evolutionary process in our species: gene-culture coevolution. When humans enter a new environment, they adapt by pushing the bounds of their phenotypic plasticity—they do the most with what they have already. They adapt culturally. There is then natural selection for genetic variants that can stabilize the new pattern of adaptation. A new phenotype thus ends up becoming a new genotype (Laland, Odling-Smee, and Myles 2010). This kind of evolutionary process has been shown by a recent genome study of the Greenland Inuit, whose marine diet has apparently selected for genetic variants that help their bodies to digest and use polyunsaturated fatty acids (Fumagalli et al. 2015).

Today, Inuit are entering an environment where vitamin D no longer has to be in short supply. By ingesting tablets and other dietary supplements, it is now possible for them to bathe their body tissues in concentrations of vitamin D that they and their ancestors have not experienced for tens of thousands of years. Unlike previous environmental challenges, however, this one is human-made and intentional. The intention, though good, is based on the flawed assumption that human bodies universally share the same need for vitamin D.

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