This month’s issue features vitamin K in dairy, breastfeeding and multiple sclerosis, diabetes and dairy, and iron in milk.

**Dairy Products are a Good Dietary Source of Some Types of Vitamin K**

- Dairy products contain multiple forms of vitamin K₂.
- Reduced-fat or fat-free dairy products contain only 5–22% of the vitamin K₂ content of full-fat dairy products.
- Vitamin K₁ and vitamin K₂ are essential for blood coagulation, and vitamin K₂ is also implicated in promoting bone and cardiovascular health.

We all know vitamins are good for us. Some vitamins covet the stage of public awareness and rarely relinquish the limelight. If not consumed in sufficient quantities, they threaten dire health consequences for nonbelievers, children and small noisy dogs. Other vitamins, like vitamin K, are lost in the extrovert vitamin ABCD crowd and struggle to be heard. Their message, however, is equally profound for good health in humans. The paradox is that vitamin deficiency remains for many people a significant health and nutrition issue even in the presence of an abundance of food. Moreover, scientists continue to discover new biological roles of vitamins and new food sources containing different molecular forms of known vitamins, which together cause the regular revision of recommended daily intake levels.

Recent investigations that expanded the known biological roles and molecular forms of vitamin K are a case in point, with several studies highlighting a additional potential benefits to people that may not be realized because of current dietary and nutrition practices [1-3]. A recent study published in *Current Developments in Nutrition* and authored by Fu and colleagues reported that full-fat dairy products contain multiple forms of a poorly characterized form of vitamin K—vitamin K₂—and other studies reveal unexpected roles of this vitamin subtype in maintaining bone and cardiovascular health [1-3].

**What is a Vitamin?**

Scientists discovered most vitamins many decades ago when they noticed that some specific diets consumed routinely by humans and animals caused severe illnesses. The scientists reasoned that food components critical for good health were missing in these diets. They identified these food components (containing vitamins) and added them into the deficient diets, thereby preventing these illnesses. Subsequently, the molecular form of each vitamin was characterized. Vitamins are small organic molecules that cannot be synthesized by the body, and therefore, they must be obtained from external sources such as food or in the case of vitamin D, sunlight. Typically, very low quantities of vitamins are essential for key biochemical reactions that underpin normal cellular metabolism and function. The Nobel Committee recognized the immense importance of vitamins to human health with the award of a total of nine Nobel prizes between 1929 and 1964 [4].

**Functions and Sources of Vitamin K**

The Danish scientist Henrick Dam discovered vitamin K nearly 90 years ago [5]. He noticed that low-fat diets in chickens led to bleeding tendencies that could be prevented when normal levels of fat (containing vitamin K) were restored in the diet. Since then vitamin K has been recognized by nutritionists as a fat-soluble dietary component essential for normal blood coagulation in humans. Dam later discovered that the vitamin K required for blood clotting existed in two related molecular forms, phylloquinone (vitamin K₁) and menaquinone (vitamin K₂). Vitamin K₁ is prevalent in leafy green vegetables and has been well characterized. Vitamin
K₂ is present in meat, eggs, and dairy products, but as Fu and colleagues [3] noted, historically vitamin K₂ has been poorly studied in terms of its functions, multiple structures and abundance in different foods [1, 2]. Additional molecular versions of vitamin K₂ are also produced by microbial fermentation, and these are found in some cheeses and nattō, a Japanese delicacy consisting of fermented soybeans.

Various national health and nutrition agencies throughout the world have produced dietary recommendations of Adequate Intakes of vitamin K. The term Adequate Intakes is used because there is insufficient information available to allow definition of a precise daily recommended intake for humans. Fu and colleagues indicated that menaquinones, the different molecular forms of vitamin K₂, have not been systematically analyzed in US foods nor have menaquinones been included in (total) vitamin K intakes estimated in the US population [3].

**Multiple Forms of Vitamin K Are Present in Dairy Products**

Fu and colleagues used an extremely powerful and sensitive technique, mass spectrometry, to characterize the various forms of menaquinones and phylloquinone in a range of US dairy products [3]. Detectable but only small quantities of phylloquinone (vitamin K₁) were present in full-fat dairy products. The investigators demonstrated that three molecular forms of menaquinone (different sized versions of vitamin K₂) were present in “appreciable amounts” in full-fat dairy products. Importantly, Fu and colleagues showed that reduced-fat or fat-free dairy products contained only 5–22% of the total vitamin K content of the full-fat dairy products. The investigators concluded that the total vitamin K contents of various dairy products are relatively high and in proportion to the fat content of the product. They speculated that the multiple forms of menaquinones found in full-fat milk may be indirectly derived from microbial fermentation occurring in the specialized ruminant digestive system of the cow.

**Implications**

The study of Fu and colleagues has far-reaching consequences. First, the study will allow national health and nutrition agencies to take into account another rich food source of vitamin K and thereby potentially allow more precision in the recommended total Adequate Intakes level. Second, vitamin K₂ is not only required for normal blood coagulation, but recently has also been implicated in bone and cardiovascular health [1, 2]. These studies demonstrated that vitamin K₂ is associated with inhibition of arterial calcification, inhibition of arterial stiffening and the maintenance of calcium in bone. What is unknown is the level of dietary vitamin K₂ that optimizes these biological functions. Third, the research of Fu and colleagues [3], in addition to a timely review written by Katarzyna Maresz from the International Science and Health Foundation in Krakow [1], highlights difficulties with popular modern diets. They noted the near absence of vitamin K₂ from Western-style “junk” foods and its depletion in fat-free dairy foods. Thus, some people may not be getting sufficient vitamin K₂ in their diets.

Calcium supplementation is widely used by elderly people to aid in the prevention of osteoporosis and its associated increased risk of bone fractures. However, a number of studies that were summarized by Maresz [1] conclude that there is an association with the use of calcium supplements and increased deposits of calcium in blood vessels, which may increase the risk of heart disease. Vitamin K₂ deficiency may hinder removal of excess calcium from soft tissues and blood vessels and its deposition in bone. Thus, an adequate intake of vitamin K₂ may be particularly important for the cardiovascular health of people taking calcium supplements.

There is still considerable additional research required to fully understand the roles of vitamin K in human health. The impacts of this vitamin on human health must also be assessed by health agencies using a background of current dietary trends.

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**Contributed by**

**Dr. Ross Tellam (AM)**
Exclusive Breastfeeding Cuts Multiple Sclerosis Relapse Rates

- It is well established that the rate of multiple sclerosis (MS) relapses decreases during pregnancy.
- Although some studies have also linked fewer relapses to breastfeeding, they have suffered from methodological shortcomings.
- Authors of a large trial in Germany, which guarded against these common problems, recently reported a significant relapse reduction for the first six months after giving birth among women with MS who breastfeed exclusively for at least two months.

As an immunological disease that is usually diagnosed before the age of 40 in about three times as many women as men, multiple sclerosis (MS) affects many individuals who hope to carry a child to term and nurture it thereafter. In the 1950s, experts assumed that pregnancy would be nothing but harmful to these women. Many studies since then have demonstrated that the risk of an MS relapse actually plummets during pregnancy, especially in the third trimester, only to increase again after birth. Recently, a large study in Germany also found that exclusive breastfeeding for at least two months diminishes the odds of relapse for six months after the baby is born [1]. Understanding the immunological changes taking place that mediate these shifting risks may eventually lead to novel treatments.

The symptoms of MS—which vary greatly among patients—result from the immune system mistakenly identifying as foreign the insulating coating that protects nerves, and enables these cells to transmit signals as quickly as they do. The signals, therefore, become disrupted, potentially affecting any part of biology and behavior from speech to bladder control. About 85% of people with MS experience the disease as a series of relapses, whereby their symptoms appear sporadically, lasting for maybe a few weeks during each relapse before fading away again.

The most widely cited study of MS relapses and pregnancy was conducted in the 1990s [2]. It followed 269 pregnancies, from the year before pregnancy to a year after birth, among European women residing in 12 different countries. This set-up allowed the researchers to compare the participants’ relapse rates during different periods of pregnancy with their individual baselines—established during the 12 months before they became pregnant. It uncovered a 70% reduction in disease activity during the third trimester [3].

Findings on the rate of MS relapses during breastfeeding, however, have been less readily accepted. This is because research in the area has tended to struggle with two issues that could muddy the results. The first problem is one of self-selection: women who feel well enough to breastfeed are reasonably likely to be those less frequently troubled by relapses anyway, so a finding that breastfeeding is associated with lower rates of relapses might be an artifact of these women’s disease being less severe. The second problem has been that most studies have not distinguished women who exclusively breastfeed from women who breastfeed while supplementing their infants’ diet with other foods. This distinction matters a great deal for the immunological changes that take place in a woman’s body.

The recent study in Germany overcame both of these problems. Researchers can’t—and shouldn’t—tell women whether to breastfeed or not, but they can monitor and compare the symptoms of those who decide either way. (Indeed there was no relationship between pre-pregnancy disease severity and whether or not women in this study breastfed.) The study also evaluated women who breastfed exclusively for a minimum of two months separately from non-exclusive breast feeders. In all, it followed 201 women over four-and-a-half years from before they become pregnant, interviewing them regularly about their symptoms, the drugs they were taking, their pregnancies and breastfeeding habits.

Out of these 201 women, 120 intended to feed their infant their own milk without supplementation for two months or more. Among this group, 24% experienced a relapse in the six months after their child was born. That proportion is significantly lower than the 38% of women who relapsed among those who either did not breastfeed at all or breastfed with supplementation. The authors considered these effects large enough to conclude that “exclusive breastfeeding acts like a MS treatment with a natural end date.”
But what explains the differences? The reason that distinguishing exclusive and non-exclusive breastfeeding is thought to be so important is that introducing regular formula or solid food into an infant’s diet prompts hormonal changes in a woman’s body that prepare it to restart ovulation. Getting one’s period back after having a baby is associated with a rise in a pro-inflammatory signaling molecule called tumor necrosis factor-alpha (TNF-alpha). Although the details of the mechanisms involved are not entirely clear, and other immune factors are likely involved, at least one other study has linked lactational amenorrhea (not having periods while breastfeeding) with a decrease in the CD4 cells that produce TNF-alpha [4]. The suppression of CD4 cells that occurs during pregnancy—as part of a suite of changes that prevent a women’s body rejecting a fetus—is also thought to explain the quelling of MS-relapse risk at that time.

As the authors of a review on the topic have noted, plenty more research needs to be done to tease out the immunological details of these shifting risks, including more completely identifying the pro-inflammatory molecules involved [3]. That will be necessary before this line of inquiry can lead to new drug targets, which is certainly possible in the future. Until then, the message from the German study is clear: exclusive breastfeeding is not only best for the infant but also for a new mother with MS. And that’s especially good news for women who struggle with a condition for which the best medications are already extremely costly, and seem to be getting ever more so each year [5]. Although breastfeeding isn’t free—it costs time and energy—it should improve the symptoms for those who choose and are able to do it, without straining their bank accounts.


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**Glycemic Status, Type of Dairy and Risk of Diabetes**

- Studies of the association between dairy consumption and type 2 diabetes have had widely varying results, making it hard to draw any unequivocal conclusions.
- A new study finds that the association between dairy intake and prediabetes or diabetes varies by dairy type and glycemic status.
- The results suggest that differences in baseline glycemic status might underlie the variability in the association between dairy consumption and diabetes.

Diabetes is a major public health problem that affects hundreds of millions of people worldwide [1]. Diet is known to influence the risk of diabetes, and researchers have been trying to understand how dietary changes could help prevent type 2 diabetes and other cardiometabolic diseases [2]. “My research program has a long history of investigating the relationship between diet and cardiometabolic risk,” says Paul F. Jacques, program director of nutritional epidemiology and senior scientist at the Jean Mayer USDA Human Nutrition Research Center on Aging at Tufts University.

While researching how different foods influence cardiometabolic risk, Jacques became interested in studying the effects of dairy. “As dairy has long been considered to be part of a healthy diet based largely on its nutrient density, we were interested in determining if the healthy nutrient profiles of different dairy foods translated into a healthier metabolic profile and lower risks of cardiometabolic disease,” he says.

Dairy is a staple of the US diet, with US dietary guidelines recommending two to three servings per day [3,4]. In addition, dairy products are a major source of dietary saturated fats, although there is evidence that saturated fats from dairy may be less harmful than those from other foods [5]. Many studies have investigated the association between dairy intake and the risk of type 2 diabetes, but the results have been variable, making it hard to draw any firm conclusions [6-17].
"We initiated our work with dairy about eight years ago, focusing first on its relationship with weight maintenance and blood pressure control," says Jacques. "During that time, evidence that certain dairy foods affected the risk of type 2 diabetes was accumulating," he says. "Based on the mixed evidence from those studies and work we were doing with other foods that suggested risk factors for diabetes might depend on an individual’s glycemic status, such as normal blood glucose, insulin resistance, prediabetes, or diabetes, we thought it would help sort out some of the confusion in the existing literature to examine the role of glycemic status and individual types of dairy as risk factors for diabetes,” says Jacques.

In a new study, Jacques and his colleagues found that the association between dairy intake and diabetes varied by dairy product and glycemic status [18]. The dairy products associated with reduced risk of prediabetes in those with a normal baseline glycemic status were different from the dairy products associated with reduced risk of diabetes in those with an impaired baseline glycemic status. The findings suggest that differences in baseline glycemic status might underlie the variability in the association between dairy intake and diabetes.

“I would consider the main implications of this work to be in the potential importance of glycemic status and the types of dairy together in determining the possible benefits of dairy on diabetes risk,” says Jacques. “As our study was the first to truly consider the simultaneous role of both glycemic status and dairy type, it is probably too early to consider recommendations, but it is our hope that this will stimulate additional research on these determinants of metabolic risk,” he says.

Most previous studies did not look into the association between dairy intake and the impaired glucose or hyperglycemic stages preceding type 2 diabetes. One previous study found that total dairy, excluding cheese, was associated with lower odds of hyperglycemia but did not find a significant association with type 2 diabetes [12].

In the new study, Jacques and his colleagues examined the relation between consumption of dairy and milk-based products and the long-term risk of prediabetes among healthy individuals, as well as the risk of type 2 diabetes among individuals with prediabetes. They also examined the effects of baseline glycemic status on the association between dairy intake and risk of type 2 diabetes. The researchers used food-frequency questionnaires to assess total dairy and milk-based product consumption across a mean of 12 years of follow-up among 2809 participants from the Framingham Heart Study Offspring Cohort.

Jacques and his colleagues found that total, high-fat and low-fat dairy intakes were inversely associated with the risk of developing prediabetes or diabetes among those with a normal glycemic state. There was no such protective association for yogurt intake or total or low-fat dairy intake among those with an impaired baseline glycemic state. However, cheese intake was inversely associated with type 2 diabetes in these participants. Neither cheese nor cream and butter was associated with the risk of prediabetes among those with normal glycemic status at baseline.

The study concludes that the association between dairy intake and type 2 diabetes varies according to the dairy product and baseline glycemic status in middle-aged US participants. The findings suggest that differences in underlying glycemic status may explain the varying results of previous studies and that future research should take into account the glycemic status of participants. Other studies have shown that preexisting metabolic states can influence how the body handles different foods [19-22].

The mechanisms by which different dairy products might interact with glycemic status to influence the risk of diabetes are unclear, and that’s something Jacques plans to explore in the future. “The next project that we are undertaking will compare hundreds of metabolic products found in the blood of high and low consumers of different dairy products to try to determine possible mechanisms by which different dairy products might affect metabolic health,” he says.

Do Breastfed Infants Need Extra Iron?

Human breast milk contains only a small amount of iron, but full-term human infants are born with a substantial supply of iron stored in their liver.

There is debate among nutritionists and clinicians as to whether exclusively breastfed infants require iron supplementation and when to begin supplementation with iron-fortified foods such as cereal.

Iron is not water-soluble, making it critical for policy makers to weigh the risks of potential iron deficiency with the risks of providing more iron than needed, particularly in iron-replete populations.

Breast milk is considered the gold standard for human infant nutrition. But at some point, even “white gold” cannot suffice as the only source of nutrition, and infants must begin to take in complementary foods to support their growth and development. When that point is, however, remains a matter of debate centered largely on the availability of one particular micronutrient—iron.

Human milk is very low in iron, usually containing between 0.3–0.4 mg/L [1-4]. To compensate for this low dietary intake, human infants are born with significant liver stores of iron that accumulate mainly during the third trimester of gestation. Many researchers propose that the combination of stored iron in the liver and highly bioavailable milk iron are sufficient to facilitate optimal growth and development until infants reach six months of age [1, 2, 5-9], whereas others believe the scientific evidence supports an earlier introduction of iron-fortified foods or supplements at four months of age [3, 4, 10, 11]. Although it seems like a simple solution to just err on the side of avoiding iron deficiency in early infancy, more is not...
necessarily better; the risks of not consuming enough iron must be carefully weighed against the risks of providing infants with too much iron when deciding if and when breastfed infants need extra iron.

**Iron in Our Diet and Our Bodies**

Iron deficiency (ID) is the most prevalent nutritional deficiency in the world, and is especially prevalent in children and women because of their higher iron requirements (the former because of growth, the latter because of reproduction and menstruation) [1, 2, 12]. Because infancy also is a period of rapid growth, ID in infants has become a public health concern.

Iron is the fourth most abundant element on earth—so why are so many people deficient? Iron is a tricky mineral to absorb, and it is estimated that humans only absorb between 5–35% of the iron available in a food [13]. Heme iron, which is the type of iron found in animal tissues, is associated with greater iron absorption than non-heme iron from plant sources. In addition, many foods can inhibit the body’s ability to absorb iron, such as calcium (which is itself a tricky mineral to absorb). However, iron found in breast milk has high bioavailability, and infants are able to absorb up to 50% of the iron ingested in breast milk [1].

Iron plays multiple roles within the body but is probably best known for its association with red blood cells. Red blood cells have proteins called hemoglobin, which are composed of four polypeptide chains. Each chain contains an iron atom responsible for binding oxygen. As red blood cells move throughout the body, the iron on the hemoglobin chains (referred to as heme iron, hence why it is found in animal tissues) is essential for delivering oxygen to cells throughout the body, and in binding carbon dioxide and returning it to the lungs. Moreover, iron is required for manufacturing of red blood cells. Oxygen may be essential for life, but oxygen delivery is completely dependent on the availability of iron.

Most of the body’s iron is bound to hemoglobin (approximately 65%) and iron hemoglobin stores are conserved to the detriment of other cells in the body [13]. Thus, in periods of iron depletion and deficiency, it is possible to have normal hemoglobin levels but be iron deficient. Indeed, by the time the hemoglobin is affected (referred to as iron deficiency anemia, or IDA), an individual is likely to have been iron deficient for some time.

Because of the role of iron in growth and development, ID in infants and children is associated with poor development of cognition, gross and fine motor skills, and even social and emotional development [1, 2]. These risks are even greater when ID has progressed to IDA, and both conditions are associated with increases in morbidity [13].

On the other side of the coin are the potential risks that come with consuming too much iron. Some micronutrients, such as vitamin C, are water soluble. You can consume 300% of your daily value of vitamin C without concern because your body will simply excrete the excess in urine. This is not the case for iron; the body has no pathway to excrete excess iron, and even moderate excess has the potential to interfere with normal physiological processes [1, 2, 13]. Excess iron can affect the body’s stores of other essential minerals, (including copper and zinc), increase oxidative stress (because iron is a pro-oxidative element), and slow growth (in both weight and height) in infants and children [1, 2]. Moreover, excess iron may increase the frequency or severity of infections, particularly those that affect the gastrointestinal tract [2].

In adults, iron levels are regulated in the intestines at the time of absorption [1, 13]. Individuals that are iron-replete (i.e., have adequate iron stores) absorb less iron than those with low iron status [1, 13]. However, Lönnerdal [1] reports that these mechanisms of iron homeostasis are not fully functional in infants. Results of several studies on iron supplementation of infants less than 12 months of age suggest they may be better at increasing iron absorption when iron status is low than decreasing absorption when iron status is sufficient [1]. From an evolutionary perspective, this is a very interesting finding. That infants are better at dealing with too little compared with sufficient or too much iron suggests low iron availability during early infancy may have been the rule rather than the exception (more on this later!). Thus, Lönnerdal [1, 2] warns that supplementing infants that are iron-replete with iron or over-supplementing iron-deficient infants can result in poor health and developmental outcomes.
Iron Supplementation in Breastfed Infants

It seems paradoxical to provide nutritional supplements to exclusively breastfed infants—doesn’t human milk provide infants with all of the required macro- and micronutrients in exactly the right concentrations? When it comes to iron, human milk on its own is definitely not optimal, providing approximately 0.4 mg/L. But human milk is not the only source of iron to newborns and young infants. During the final trimester of gestation, fetuses accumulate large liver stores of iron. The combination of milk iron (which is highly bioavailable) and iron stored in the liver is sufficient to support the growth and developmental needs of infants but only for a finite amount of time. When do these stores run out?

Unfortunately, there is not a consensus among nutritionists and clinicians on when exclusively breastfed infants require external sources of iron. Currently, the American Academy of Pediatrics (AAP) Committee on Nutrition [11] recommends supplementing breastfeeding infants with iron beginning at four months of age, whereas the AAP Committee on Breastfeeding [9] along with the World Health Organization (WHO) [8] do not recommend iron supplementation (and introduction of solid foods) until 6 months of age. The AAP Committee on Nutrition [11] make their recommendations based on a small number of papers [e.g., 4] that demonstrate that the proportion of infants with ID and IDA increases between four months and six months of age, as well as a positive association between iron supplementation of breastfed infants at four months and improved iron status and psychomotor scores. However, Lönnertdal and colleagues [1, 2, 5], along with the AAP Committee on Breastfeeding, argue that there is still insufficient evidence to suggest a need to provide external iron sources (i.e., iron drops or earlier introduction of iron-fortified cereal) before infants reach six months of age. They emphasize that we currently do not know whether iron supplements lead to improvements in growth, decreases in growth, or no effect at all [5]. Whereas Friel [4] found improved growth with iron supplementation, Lönnerdal and colleagues found less growth in height and head circumference in iron-supplemented infants [5].

It is critical to recognize that exclusively breastfed infants are not a homogenous group. Although on somewhat opposite sides of the debate, nutritional scientists Friel [3] and Lönnertdal [1] both agree that there are certainly some infants who would greatly benefit from iron supplementation prior to six months of age, including infants born prematurely, multiples, and infants whose mothers have severe anemia [1–4, 10]. However, blanket policy guidelines for all infants from diverse ecological and economic settings could put some infants at risk for ID (and IDA) or others for excess iron.

Not Enough or Just Right?

Adding another perspective to the debate on iron supplementation is evolutionary anthropologist EA Quinn [6]. Quinn argues against the view that low milk iron and low iron stores are pathological and in need of intervention. Instead, Quinn proposes that low milk iron and lower milk iron stores may actually provide an adaptive advantage to infants that are transitioning to solid foods. Infants are immunologically naïve, and the transition to non-milk foods means the first introduction of food-borne pathogens, including bacteria and parasites. Pathogens, like people, need iron for growth and replication. If infant iron stores are low at the time of complementary food introduction, the availability of iron for pathogens is also low, which could function to decrease the frequency and severity of infections [6].

Moreover, Quinn [6] argues that by supplementing infants with iron too early, or by adding more iron to infant formulas than is available from human milk, we may inadvertently be promoting the growth of pathogenic, iron-requiring bacteria in infant gut microbiomes. The types of bacteria that initially colonize the infant gut may act as seeds and select for future generations of bacteria involved in immune function and metabolism regulation. Thus, excess iron during early infancy has the potential to negatively impact health during infancy and throughout the lifespan [1].

Indeed, many of the differences in health outcomes between formula-fed and breastfed infants that are usually attributed to the lack of immune factors in formula may be explained, at least in part, by different intakes of dietary iron [1, 6].

Taken together, Quinn’s [6] evolutionary perspective on low milk iron and declining iron stores and Lönnertdal’s [2] findings on the potential risks for consuming too much iron suggest that policies about when to supplement with iron-fortified food (and how much iron should be used) should consider both the potential benefits and risks based on particular infant attributes. When it comes to iron, more is not always better.


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