Mechanistic links between vitamin deficiencies and diabetes mellitus: a review

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ABSTRACT
Vitamin deficiencies are suspected of causing the increasing prevalence of diabetes mellitus. However, this subject has not received adequate attention. This study reviews and communicates the plausible links between vitamin deficiencies and DM. Relevant articles were retrieved from reputable academic databases and the current information revealed that vitamins of primary importance in the pathogenesis of DM are vitamins A, C, D, E, and B-vitamins. Vitamin A enhances beta-cell formation and glucose metabolism. B-vitamins lower homocysteine levels and thus prevent oxidative stress, endothelial dysfunction, β-cell dysfunction, and insulin resistance. Vitamin C reduces free radicals and enhances superoxide dismutase, glutathione, and blood glucose circulation. Vitamin D prevents autoimmunity and promotes cellular and systemic uptake insulin. Vitamin E reduces free radicals and C – reactive proteins, prevents lipid peroxidation, and improves insulin function. These vitamin deficiencies can result in beta-cell abnormalities, insulin resistance, and insulin insufficiency. The major causes of vitamin deficiencies are low dietary supplementation, malabsorption, diseases, and lifestyle. Fortunately, dietary or therapeutic administration of these vitamins can reverse or delay the mentioned pathologies, resulting in improved diabetic conditions. Therefore, people are advised to consider vitamin intervention in the prevention and treatment of diabetic cases having a vitamin deficiency etiology.

Introduction
The term diabetes mellitus (DM) describes several distinct metabolic disorders characterized by high blood glucose (hyperglycemia) [1]. Diabetes mellitus was previously tagged a disease associated with ‘sweet urine’ and high muscle loss [2]. The initial notion stemmed from the sweet taste of the urine of diabetics due to the leakage of glucose accumulated in the blood into the urine. Type 1 diabetes mellitus (T1DM) and type 2 diabetes mellitus (T2DM) are the two most common subtypes of DM[1]. Most often, T1DM begins when the immune system dysfunctionally destroys the pancreatic β-cells that produce insulin and causes an accumulation of glucose in the blood [3]. The main features of T2DM are insufficient insulin, or insulin resistance – a condition in which the body cells fail to utilize insulin [4]. Other forms of DM include gestational diabetes

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mellitus, maturity-onset diabetes of the young (MODY), and neonatal diabetes mellitus [3,5,6]. Major symptoms of DM include thirst, polyuria, blurred vision, emaciation, and genital yeast infections [1].

The burden and mortality of DM are high [7]. Of the 463 million adults with DM in 2019, almost 4.2 million deaths were recorded [8]. Over 1 million children and teens also lived with DM (mainly T1DM) in the mentioned year [8]. Additionally, more than 20 million newborns were affected by DM in 2019 [8]. In 2019, a minimum of USD 760 billion was spent globally on DM health care, accounting for 10% of the annual budget for adults [8]. Overall, the global burden of DM has increased significantly in recent decades and will continue unabated for a long time, unless urgent measures are put in place [7]. According to the International Diabetes Federation (IDF), DM prevalence could rise to 700 million by 2045 [8].

The cause of DM and its rising prevalence remain unclear. Several etiologies have been linked to DM, including genetic and epigenetic predispositions, environmental factors, and lifestyle changes [9]. Relatively recently, antioxidant deficiencies, including antioxidant vitamins and other vitamins, have also been linked with the rising burden of the disease [10]. Studies show that diabetics generate abnormally high reactive oxygen species and weak antioxidant activities, resulting in high oxidative damage [11]. However, the mechanisms by which vitamin deficiencies cause DM are not clear. Therefore, this study was carried out to review and communicate the correlation between vitamin deficiencies and DM.

**Link between vitamin deficiency and diabetes mellitus**

Deficiencies in certain vitamins can cause several physiological and metabolic malfunctions and predispose to DM. These physiological malfunctions include pancreatic β-cell dysfunction, B-cell death, reduced islet cell populations, defective tyrosine kinase activity, and oxidative stress [12]. Others include decreased lean body mass, faulty insulin signaling mechanism, and increased protein kinase C activity [12]. All vitamins in the right proportions are essential for a healthy life, without which the body will not function properly [9]. However, several studies, including a systematic review and meta-analysis by Balbi et al. [13], show that vitamins of primary importance in the pathogenesis of DM are vitamin A, vitamin C, vitamin D, vitamin E, and B-vitamins. The major mechanisms by which deficiencies in these vitamins predispose to DM are summarized in Table 1.

**Table 1. Mechanistic links between vitamin deficiencies and diabetes mellitus.**

| Vitamin | Mechanism |
|---------|-----------|
| Vitamin A | Beta-cell death, causing decreased insulin synthesis and hyperglycemia[18] |
| Vitamin B6 | Affects T-cell composition, which may contribute to pancreatic islet autoimmunity in T1D[14,15]; It also impairs glucose and lipid metabolism[12] |
| Vitamin B12 | Promotes oxidative stress, autoimmunity, insulin resistance, β-cell dysfunction, systemic inflammation, obesity, and endothelial dysfunction [28–30–33] |
| Folate | Depletion of folate causes oxidative stress, abnormal glucose and lipid metabolism, insulin resistance, and endothelial disruption [37,40] |
| Vitamin C | Causes oxidative stress and endothelial dysfunction[59,61] |
| Vitamin D | Promotes beta cell autoimmunity, insulin resistance, and glucose metabolism disorders [65,66] |
| Vitamin E | Causes oxidative stress and beta-cell dysfunction, and promotes lipid peroxidation and glucose intolerance [56,63] |

**Vitamin A**

Vitamin A is a fat-soluble antioxidant that occurs naturally in several foods [16]. Vitamin A can be classified into two; preformed vitamin A (often called retinol) and pro-vitamin A (otherwise called beta-carotene) [17]. Retinol is found in fish, poultry, dairy products, and meat, while fruits and vegetables are the main sources of pro-vitamin A [17]. Vitamin A boosts antioxidant enzymes, supports immune functions, and regulates
spermatogenesis and embryonic development [18,19]. Vitamin A also plays a role in cell growth and differentiation through its gene regulatory activities, maintenance of epithelial cell integrity, and antimicrobial activities [18,20].

Studies show that vitamin A regulates metabolic pathways involved in the pathogenesis of DM. These findings have increased interest in the anti-diabetic properties of natural and synthetic vitamin A [19,20]. In a study, normal and transgenic mice fed no vitamin A expressed beta-cell death, insufficient insulin, and hyperglycemia, but these conditions improved upon restoration of vitamin A [18]. In another study, normal serum carotenoids were shown to reduce the risk of hyperglycemia and T2DM [21]. It was also shown in a rat study that vitamin A improves hyperglycemia and glucose-intolerance through regulation of intracellular signaling pathways and glycogen synthesis [22]. In a systematic review and meta-analysis comprising 11 studies, an inverse association was observed between total carotenoids and metabolic syndrome, including T2DM [23]. Overall, these findings suggest that vitamin A is necessary for beta-cell formation and glucose metabolism and its depletion may predispose to T2DM [18]. Similarly, vitamin A supplementation might ameliorate diabetic conditions [21]. To this end, diets rich in mixed carotenoids have been demonstrated to reduce the burden of metabolic syndrome [23]. Moreover, daily intake of vitamin A by some experimental rats has been demonstrated to improve pancreatic β-cell function and prevent or delay the progression from pre-diabetes to T2DM [24]. Beta-cells contain numerous cell surface receptors for vitamin A [24], which underscores the importance of vitamin A in insulin production and glucose metabolism.

Owing to the regulatory role of vitamin A on immune functions, particularly T-cell-mediated immunity, some scientists suggest that impaired vitamin A metabolism can cause autoimmune and T1DM [19,25]. In a study, vitamin A and its derivative (all-trans retinoic acid) significantly boosted the immune system and prevented islet inflammation and T1DM by inhibiting Teff cells and increasing Treg cells mass [26]. Similarly, diabetic mice treated with 4-IU vitamin A for 16 weeks showed a significantly reduced fat mass, lipid profile, and malonaldehyde as well as increased superoxide dismutase, glutathione peroxidase, and catalase compared to the control [27]. These suggest that vitamin A supplementation may protect tissues from free radical damage and thus may reduce or prevent DM complications [27]. Vitamin A may also contribute to the synthesis of insulin and glucagon [26]. The mechanistic links between vitamin A deficiency and DM are summarized in Figure 1.

**B-vitamins (Vitamin B6, Folate, and Vitamin B12)**

Vitamin B12, otherwise called cobalamin because it contains cobalt, is a water-soluble vitamin [28]. It is the most studied and structurally complex of the B-vitamins, and found in all cells where it is involved in DNA synthesis, optimal hemopoiesis, and neurological activities [28]. Vitamin B12 and some other B-vitamins lower homocysteine levels – an amino acid that predisposes to T2DM by promoting oxidative stress, insulin resistance, β-cell dysfunction, systemic inflammation, and endothelial dysfunction [29,30]. Depletion of vitamin B12 may lead to pernicious anemia, which is often associated with T2DM. The deficiency of vitamin B12 is also linked with autoimmune diseases, including T1DM [28]. Vitamin B12 is abundant in animal products such as meat, milk, eggs, poultry, eggs, and fish [31]. These show that individuals following vegan diets are more at risk of vitamin B12 deficiency [31]. Many studies have linked vitamin B12 deficiency with the pathogenesis of DM. In one study, vitamin B12 deficiencies in pregnant women were linked with obesity, a risk factor of both T1DM and T2DM [32]. In another study, an association was established between maternal vitamin B12 levels and risk of maternal obesity and gestational diabetes.
In a clinical trial, vitamin B12 therapy improved insulin resistance and endothelial function [34]. Additionally, low vitamin B12 levels were linked with an increased risk of adiposity [35]. Vitamin B12 modulates several cellular processes, particularly epigenetic changes that are necessary for gene expression. Some of these pathways may be involved in the fetal metabolic configuration that predisposes offspring to insulin resistance [32]. The mechanistic links between vitamin B12 deficiency and DM are summarized in Figure 2.

Folate, formerly known as folacin and sometimes as vitamin B9, is a water-soluble B-vitamin that is present naturally in several foods and also as an additive and dietary supplement [36]. Foods that contain folates include fruits, grains, poultry, eggs, vegetables, nuts, beans, peas, seafood, liver, dairy products, and meat.

**Figure 1.** Links between vitamin A deficiency and diabetes mellitus (CorelDraw 13).

**Figure 2.** Links between vitamin B12 deficiency and diabetes mellitus (CorelDraw 13).
Folate depletion in mice causes oxidative stress, abnormal glucose and lipid metabolism, and insulin resistance [37]. In a study of 1530 non-diabetic adults in the 2011–2012 US National Health and Nutrition Examination Survey, a relation was detected between serum folate levels and insulin resistance [38]. These findings collectively show that folate supplementation may lower homocysteine and lessen insulin resistance. Indeed, in a systematic review and meta-analysis, folate supplementation lowers insulin resistance and improves glucose metabolism [39]. Furthermore, a clinical trial showed that a high-dose of folate rapidly normalized endothelial dysfunction in children with T1DM [40]. The mechanistic links between folate deficiency and DM are summarized in Figure 3.

Vitamin B6, also called pyridoxine, is present in several foods, such as poultry, pork, fish, soya beans, peanuts, bananas, wheat germ, oats, and milk [41]. The vitamin assists the cells to utilize energy from food and store excess energy for later use [41]. Vitamin B6 is also involved in the formation of hemoglobin and its active form, known as Pyridoxal 5′-phosphate (PLP), helps catalyze 150 reactions that are involved in glucose and lipid metabolisms [42]. Vitamin B6 is important in cellular metabolism and acts as an antioxidant and blocks reactive oxygen species (ROS) and advanced glycation end-products (AGEs) [42]. Moreover, vitamins B6 and B12 promote nerve function and prevent diabetic complications like diabetic neuropathy [43]. Several studies have investigated the correlation between vitamin B6 and DM and its complications. In particular, insufficient PLP disrupts insulin production in rats, while PLP administrations lessen diabetic complications and maintain chromosome integrity and glucose homeostasis [44]. The most notable study that established a link between vitamin B6 deficiency and DM was demonstrated in Drosophila in which mutations in a gene involved in vitamin B6 metabolism known as the dPdxk gene caused hyperglycemia and DM [45,46]. In addition, studies show that reduced vitamin B6 levels may predispose people to pancreatic islet autoimmunity in T1D [47]. Qian et al. [48] demonstrated that reduced vitamin B6 may affect the T-cell composition and compromise the immune system and predispose it to autoimmune diseases. Overall, this shows that vitamin B6 may possess

![Figure 3. Links between folate deficiency and diabetes mellitus (CorelDraw 13).](image-url)
a protective or ameliorative effect on diabetic conditions. In a study, dietary vitamin B6 prevents endothelial disorders, insulin insensitivity, and hepatic lipid accumulation in mice treated with high fat-diets [49]. The mechanistic links between vitamin B6 deficiency and DM are summarized in Figure 4.

**Vitamin C (Ascorbic Acid and Ascorbyl Palmitate)**

Vitamin C, otherwise called ascorbic acid or ascorbate, catalyzes several reactions, including collagen synthesis [50]. It is a water-soluble vitamin and an essential nutrient because it can only be obtained from diets (the body does not synthesize it) [51]. Like most mammals and some other animals, humans are unable to produce vitamin C due to mutations in the gene called L-gulono-γ-lactone oxidase (GLO), which encodes the enzyme that catalyzes the last step of vitamin C biosynthesis [52]. Vitamin C reduces free radical-mediated oxidation processes and thus can be referred to as an antioxidant [53]. It is not common to be chronically deficient in vitamin C because it is abundant in fresh fruits and vegetables [54]. However, certain traditional cooking practices, processed foods, and staple foods such as grains that contribute minute vitamin C to diets may reduce vitamin C levels [55]. Environmental factors, including geographic region, season, climate, as well as pollution (which increases oxidative stress) may also lower vitamin C levels [55]. Other factors that deplete serum vitamin C include physical inactivity, obesity, smoking, certain diseases, sex, age, race, and low socioeconomic status [55].

Some studies suggest that vitamin C deficiency predisposes to T1DM and T2DM [56,57]. In a cross-sectional study involving T2DM patients and controls, plasma vitamin C levels were significantly lower in the diabetics and pre-diabetics [57]. In another study, the prevalence of vitamin C deficiency among diabetics was 55.13% [58], and an inverse relationship was established between vitamin C and total cholesterol levels and fasting blood sugar [58]. These collectively show that vitamin C supplementation may reverse certain cases of DM. One major role of insulin in blood sugar regulation is its ability to induce blood vessel relaxation (vasodilation) to increase blood flow and glucose to all parts of the body [59]. This insulin function is regulated in part by nitric oxide, which is enhanced by

![Figure 4. Links between vitamin B6 deficiency and diabetes mellitus (CorelDraw 13).](image-url)
vitamin C [59]. Injections of vitamin C into the arteries of diabetic patients have been demonstrated to boost blood vessel response to nitric oxide in diabetic patients [59]. Thus, doses of vitamin C will boost insulin sensitivity and endothelium function in diabetics or individuals expressing vitamin C deficiency [59]. Vitamin C prevents sorbitol accumulation and glycosylation of proteins and thus reduces the microvascular complications of T1DM and T2DM, such as retinopathy, nephropathy, and diabetic foot [60]. In a study, administration of vitamin C alone or combined with vitamin E reduced blood glucose and increased superoxide dismutase and glutathione levels, resulting in reduced insulin resistance by lowering oxidative stress [61]. In a prospective study comprising 412 diabetic patients divided equally into two groups, the group supplemented with vitamin C showed a reduced level of glycated hemoglobin (HbA1c) and fasting blood glucose [62]. However, in a study conducted in Korea, there was no difference in dietary vitamin C intake among diabetics and controls [63]. The mechanistic links between vitamin C deficiency and DM are summarized in Figure 5.

**Figure 5.** Links between vitamin C deficiency and diabetes mellitus (CorelDraw 13).

**Vitamin D**

Vitamin D (also called ‘calciferol’) is a fat-soluble vitamin that occurs naturally in some foods and is also available as additives and dietary supplements [64]. Fatty fish, including salmon, tuna, mackerel, trout, and fish liver oils are among notable sources of vitamin D [64]. Furthermore, vitamin D is synthesized by the body when the skin absorbs ultraviolet rays from the sun and stimulates vitamin D synthesis [64]. However, vitamin D obtained from foods, ultraviolet rays, and food additives are biologically inactive and must pass through two hydroxylation processes in the body to become active [64]. The first hydroxylation occurs in the liver, where vitamin D is converted to 25-hydroxyvitamin D [25 (OH) D], also known as ‘calciol’. The second hydroxylation occurs primarily in the kidney and results in the metabolically active 1,25-dihydroxyvitamin D [125OH], also known as “calcitriol [64].

Vitamin D receptors are embedded in the immune cells (B-cells, T-cells, and antigen-presenting cells), and through chemical signals, vitamin D modulates the innate and adaptive immune responses [65]. This shows that vitamin
D enhances the immune cells, which is important in preventing autoimmune diseases like T1DM [65,66]. Thus, vitamin D influences the immune cells to commit fewer errors [65]. There are specific receptors in pancreatic beta cells that switch on only when they receive optimum vitamin D, suggesting a role for vitamin D in beta-cell function and insulin secretion [67]. Vitamin D attaches to these receptors and prevents beta-cell apoptosis, boosts insulin secretion, and decreases inflammation [67]. Vitamin D also improves metabolic functions and prevents metabolic disorders such as T2DM [68]. Decreased levels of vitamin D may cause insulin resistance, insulin insensitivity, and impaired insulin secretion through β-cell dysfunction [68]. Vitamin D is involved in the systemic circulation of insulin through the portal tract and binds to receptors on the cell surface, allowing insulin to enter the cell for further cellular activities. Low vitamin D levels may disrupt one or several of these steps, resulting in the abnormal response of the body to insulin [68]. Vitamin D reduces oxidative stress and inflammation and prevents genetic and epigenetic alterations associated with insulin resistance and DM [69].

Some studies in humans and animals have investigated the relationship between vitamin D levels in the body and the onset of DM. A study was conducted on animals in which vitamin D receptors were removed and the animals failed to produce normal insulin levels [70]. In a study that measured maternal vitamin D-binding protein throughout pregnancy and the risk of T1DM in offspring, lower concentration, particularly in the third trimester, tended to be associated with T1DM [71]. Moreover, in a non-randomized clinical trial involving 80 type 1 diabetics with 25-hydroxyvitamin D levels below 50 nmol/L, 4000 IU of vitamin D₃ supplementation for 12 weeks produced a positive effect [72]. A systematic review and meta-analysis also linked low vitamin D levels with T2DM and insulin resistance, which were improved following vitamin D supplementation [73]. Additionally, a randomized controlled trial found that vitamin D supplementation for six months improved insulin secretion and sensitivity in 96 pre-diabetics and type 2 diabetics [74]. These findings suggest that vitamin D may help delay or manage T2DM. However, some studies, including Bizzarri et al. [75] and Al Thani et al. [76], found no link between vitamin D supplementation and improved glycemic control. However, according to Gröber and Holick [77], diabetic subjects in studies reporting no effect of vitamin D supplementation are not deficient in the vitamin and thus may not benefit from its effects. The mechanistic links between vitamin D deficiency and DM are summarized in Figure 6.

**Vitamin E**

Vitamin E is a group of eight lipophilic molecules; four of which are tocopherols and the other four are tocotrienols [78]. γ-Tocopherol is the most abundant vitamin E in many plant seeds and the western diet, while α-tocopherol is the most abundant vitamin E in plasma, and is the most biologically active [79]. When the body is deficient in vitamin E, internal organs can be destroyed by free radicals [56]. Hence, vitamin E is an antioxidant, which prevents the generation of free radicals and reactive oxygen species from the oxidation of vitamin A and unsaturated fatty acids and thus may benefit diabetics [80]. The most compelling evidence for the effect of vitamin E in DM is on protection against lipid peroxidation [63]. Vitamin E improves oxygen supply to the blood, detoxifies toxins, and improves insulin function [56]. Raising the plasma levels of vitamin E may therefore reduce the chances of DM and as well improve glucose tolerance in individuals expressing DM [56]. In addition, the antioxidant activities of vitamin E may decrease the risk of diabetic complications [56].

The findings of many studies suggest that vitamin E supplementation can effectively normalize blood glucose levels. In a study that monitored
the effects of oral insulin and vitamin E supplementation on some type 1 and 2 diabetic patients, the patients who were on vitamin E supplementation showed better glycemic control and a slower progression of diabetic complications [81]. Tocotrienol-rich fraction supplementation reduces hyperglycemia-induced skeletal muscle damage through regulation of insulin signaling and oxidative stress in type 2 diabetic mice [82]. In a systematic review and meta-analysis of 33 clinical trials involving 2102 individuals, vitamin E supplementation (≥ 700 mg/day) significantly reduced C-reactive protein (CRP), cytokines, tumor necrosis factor-α (TNF-α), and insulin resistance [83]. The mechanistic links between vitamin E deficiency and DM are summarized in Figure 7.

**Recommended daily dietary intake of vitamins**

The findings of several studies showed no consensus on the total daily dosage of the studied vitamins and treatment duration for diabetics. However, Table 2 shows the recommended daily dietary requirements of the studied vitamins for overall wellbeing, including prevention and management of DM. It is worth noting that people who have vitamin deficiencies may require higher doses than is recommended.

**Conclusion**

The study established that certain vitamins, including vitamins A, C, D, E, and B-vitamins are necessary for glycemic control. These vitamins enhance the immune cells and prevent free radical generation and thus protect and enhance the insulin-producing activities of the pancreatic beta cells. In particular, vitamins C and D promote the entry of insulin into the cells and the circulation of glucose around the body. Thus, deficiencies in these vitamins may affect insulin secretion, and glucose circulation and metabolism, resulting in DM. Dietary supplementation or therapeutic intervention with these vitamins may produce positive effects in diabetics, particularly those having a vitamin deficiency etiology.

**Disclosure statement**

No potential conflict of interest was reported by the author(s).
Figure 7. Links between vitamin E deficiency and diabetes mellitus (CorelDraw 13).

Table 2. Recommended daily dietary intake of vitamins.

| Vitamin | Recommended Dietary Intake (RDI) |
|---------|---------------------------------|
| Vitamin A | Birth to 6 months (400 mcg RAE), infants 7–12 months (500 mcg RAE), children 1–3 years (300 mcg RAE), children 4–8 years (400 mcg RAE), children 9–13 years (600 mcg RAE), teen boys 14–18 years (900 mcg RAE), teen girls 14–18 years (700 mcg RAE), adult men (900 mcg RAE), adult women (700 mcg RAE), pregnant teens (750 mcg RAE), pregnant women (770 mcg RAE), breastfeeding teens (1,200 mcg RAE), and breastfeeding women (1,300 mcg RAE) |
| Vitamin B6 | Infants 0–6 months (0.1 mg), infants 7–12 months (0.3 mg), children 1–3 years (0.5 mg), children 4–8 years (0.6 mg), children 9–13 years (1 mg), males 14–50 years (1.3 mg), males over 50 years (1.7 mg), females 14–18 years (1.2 mg), females 19–50 years (1.3 mg), females over 50 years (1.5 mg), pregnant women (1.9 mg), and breastfeeding women (2 mg) |
| Folate | Birth to 6 months (65 mcg DFE), 7–12 months (80 mcg DFE), 1–3 years (150 mcg DFE), 4–8 years (200 mcg DFE), 9–13 years (300 mcg DFE), 14–18 years (400 mcg DFE), 19+ years (600 mcg DFE), breastfeeding 14–18 and 19 + years (500 mcg DFE) |
| Vitamin B12 | 0–6 months (0.4 mcg), 7–12 months (0.5 mcg), 1–3 years (0.9 mcg), 4–8 years (1.2 mcg), 9–13 years (1.8 mcg), 14+ years (2.4 mcg), pregnant 14+ years (2.6 mcg), and breastfeeding 14+ years (2.8 mcg) |
| Vitamin C | 0–6 months (40 mcg), 7–12 months (50 mcg), 1–3 years (15 mg), 4–8 years (25 mg), 9–13 years (45 mg), 14–18 years (75 mg for males, 65 mg for females, 80 mg for pregnant women, 115 mg for breastfeeding women), 19+ years (90 mg for males, 75 mg for females, 85 mg for pregnant women, 120 mg for breastfeeding women) |
| Vitamin D | Birth to 12 months (400 IU), children 1–13 years (600 IU), teens 14–18 years (600 IU), adults 19–70 years (600 IU), adults 71 years and older (800 IU), and pregnant and breastfeeding teens and women (600 IU) |
| Vitamin E | Birth to 6 months (4 mg), infants 7–12 months (5 mg), children 1–3 years (6 mg), children 4–8 years (7 mg), children 9–13 years (11 mg), teens 14–18 years (15 mg), adults (15 mg), pregnant teens and women (15 mg), breastfeeding teens and women (19 mg) |

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