

## Vitamin E Deficiency in Preterm Newborns

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### Abstract

Prematurity is due to diverse and unpredictable circumstances, affecting all places and social classes, with negative consequences for families and society in general, its incidence is variable and depends on population characteristics. Currently, prematurity is an important underlying cause or associated with perinatal, neonatal, and infant mortality, and the risk of morbidity and mortality is all the greater with lower gestational age and birth weight. In addition, the preterm newborn is more likely to have nutritional deficiencies, since they have metabolic peculiarities and a high nutritional demand, because they are in an accelerated phase of growth and development.

**Keywords:** Vitamin E; Preterm Newborns

Currently, prematurity is an important underlying cause or associated with perinatal, neonatal, and infant mortality, and the risk of morbidity and mortality is all the greater because lower gestational age and birth weight, due to the risks of adaptation to extrauterine life due to immature organs and vital systems [1]. In addition, the preterm newborn is more prone to nutritional deficiencies, since they have metabolic peculiarities and a high nutritional demand, because they are in an accelerated phase of growth and development. They can also present several diseases and complications in the postnatal period, which is associated with being premature, makes their nutrition a challenge and a great responsibility [2,3].

It is not yet clear in the literature the factors that make the premature newborn more susceptible to this deficiency. It is believed that this event may occur due to limited placental transfer, since the placenta acts as a barrier to the passage of vitamin E from the mother to the fetus, and this barrier does not occur at the entrance of vitamin E in the placenta, but rather, in the placental transport of this vitamin to the fetus [4,5]. Didenco [6] indicate that a high tocopherol intake during pregnancy does not interfere with the levels of this nutrient in children, suggesting that supplementation during gestation is not effective in preventing vitamin E deficiency in infants.

Other factors that may contribute to this vitamin deficiency in the newborn are: excessive fetal metabolism of vitamin E due to oxidative stress at birth; the limited absorption of the vitamin by the fetus because of the immaturity of its organism and reduced expression of the alpha-TTP protein; low levels of circulating lipids (triglycerides, phospholipids, total cholesterol); increased nutritional requirements in the preterm infant and alpha-tocopherol accumulation in the fetus occurred mainly in the third gestational trimester, period of higher fetal growth and increase in the reserve of adipose tissue [5-6]. Approximately 90% of vitamin E is localized in adipose tissue, however, as in premature infants these tissues are scarce, vitamin E reserves are lower [8].

Preterm infants with vitamin E deficiency have low levels of hemoglobin, morphological changes such as anisocytosis and fragmented erythrocytes, reticulocyte response, increased platelet count, and hyperbilirubinemia [9] and may develop infection, thrombocytosis, hemolytic anemia, retrolental fibroplasia, bronchopulmonary dysplasia, and spinocerebellar degeneration [10]. Vitamin E supplementation may be an additional benefit to premature newborns with anemia, reticulocytosis, and need for oxygen [11].

Vitamin E deficiency in adults is usually established as a result of genetic abnormalities in alpha-TTP and apolipoprotein B (apo-B), or as a result in fat malabsorption syndrome [12]. The initial symptom of vitamin E deficiency in humans is the reduction or absence of reflexes, being cerebellar ataxia, skeletal myopathy and pigmentary retinopathy among the first manifestations of deficiency. Patients with cystic fibrosis, chronic cholestatic and hepatobiliary disease may also develop symptoms of this deficiency [12,13].

Some studies have also reported the importance of vitamin E in the pathogenesis of peri-intraventricular hemorrhage (HPIV), since the brain is especially susceptible to the oxidative stress involved in the mechanisms of hypoxia-ischemia and reperfusion-hyperoxia. This vulnerability is a consequence of several factors such as the presence of nervous tissue with membranes rich in polyunsaturated fatty acids, an immature nervous system rich in iron, and an inadequate ability to block the oxidant chain due to the low activity of enzymes with antioxidant function, characteristics of very low birth weight preterm infants [8,10]. Thus, it is believed that vitamin E deficiency may worsen the prognosis of newborn cases with HPIV [10].

It is recommended exclusive breastfeeding until the sixth month of life as an ideal feeding for children [14]. The mechanism of transfer of vitamin E from the blood to the mammary gland is little known. It is believed that LDL plays an important role in this transfer, since one of the proposed mechanisms is LDL transfer, described through independent and dependent mechanisms of the LDL receptor [15,16]. In addition, the SR-BI scavenger receptor, which is expressed at high levels in the mammary gland of pregnant rats, suggested to be physiologically relevant for the entry of alpha-tocopherol, is involved in the transfer via HDL and LDL [16]. It is also proposed the participation of the CD36 protein, which has high affinity for HDL, LDL and VLDL, as well as lipoprotein lipase and VLDL receptor [5].

The supply of vitamin E to breast milk also seems to be selective, since the RRR- isomer is preferentially incorporated into milk. However, it is believed that maternal supplementation with alpha-tocopherol may increase the amount of this micronutrient in breast milk [17], important factor, since the concentration of vitamin E tends to decrease with the passage of time [18]. Thus, it is believed that in the mammary gland there may be the presence of mechanisms similar to what occurs in the liver, where the alpha form is the main one incorporated into VLDL, due to the presence of alpha-TTP [19].

Dimenstein [20] analyzed the association between alpha-tocopherol in serum and colostrum under fasted and postprandial conditions and concluded that the absence of correlation between these variables in both feeding states excludes the existence of passive transfer mechanisms during the passage of the vitamin and from the mammary gland to the milk. According to the authors, there are probably different transport mechanisms of this vitamin for the mammary gland, which are independent of the plasma concentration. Therefore, several protocols are being used for infant and infant supplementation to meet nutrient needs, including alpha-tocopherol.

## Conclusion

Particular attention is needed regarding vitamin E deficiency in preterm infants, in order to minimize the effects of vitamin E deficiency.

## Conflict of Interest

No conflict of interest or economic interest.

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