

# Abstract

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## Role of vitamin A in determining nephron mass and possible relationship to hypertension.

Bhat PV, Manolescu DC.

Laboratory of Nutrition and Cancer, Centre Hospitalier de l'Université de Montréal, CHUM-Hotel Dieu, Université de Montreal, H2W 1T8 Montreal, Canada.

**BACKGROUND:** Vitamin A (retinol) and its analogs (retinoids) are important regulators of cell proliferation, differentiation, immune function, and apoptosis. The kidneys are target organs for vitamin A action. Retinoic acid (RA), a vitamin A metabolite, is involved in embryonic kidney patterning through the control of receptor tyrosine kinase expression, which modulates ureteric bud branching morphogenesis.

**FINDINGS:** Vitamin A status of the mother profoundly affects kidney organogenesis of the newborn. In rodents, mild vitamin A deficiency results in a 20% reduction of nephron number. In adult humans, nephron number varies between 0.3 and 1.3 million per kidney, which is accepted as normal. However, recent studies indicate that humans at the low end of nephron number are predisposed to primary hypertension. Because RA regulates nephron mass, its optimal availability during nephrogenesis is critical. RA levels in the embryo are affected by several factors, such as maternal vitamin A nutrition and disturbances in retinol metabolism.

**CONCLUSIONS:** Maternal vitamin A deficiency during pregnancy is widespread in developing countries and segments of these populations may be exposed to low vitamin A during fetal life when nephron number is determined. Infants are likely to be born with suboptimal nephrons and may develop primary hypertension later in life. Although maternal vitamin A deficiency is not common in developed countries, congenital nephron number nevertheless varies widely, indicating low fetal RA levels due to common variants of the enzymes that convert retinol to RA. These infants might require heightened surveillance for hypertension later in life.

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