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CHANGES IN ANTHROPOMETRIC PARAMETERS IN VITAMIN D DEFICIENCY IN 7-11-YEAR-OLD CHILDREN.

Ergasheva Yulduz Sultonovna,

yulduzergasheva777@gmail.com

Bukhara State Medical Institute, assistant of the department of propaedeutics of children's diseases and children's neurology.

Abstract : The article discusses issues related to the negative impact of vitamin D deficiency on the body of low birth weight, prematurely born and full-term newborns. A review of the results of studies examining vitamin D availability is presented in pregnant women and their newborn children. There has been a wide prevalence of vitamin D deficiency in women during pregnancy, as well as in newborns. It has been shown that insufficient antenatal supply of vitamin D to the fetus can lead not only to congenital rickets and hypocalcemic neonatal convulsions during the newborn period, but also determines an increase in the incidence of necrotizing enterocolitis and bronchopulmonary dysplasia, as well as disorders of psychophysical development and an increased risk of developing lower respiratory tract infections (bronchiolitis, pneumonia) throughout first year of life.

Key words: vitamin D, vitamin D deficiency, calcidiol, low birth weight newborn, premature newborn, full-term newborn

Many regions worldwide are undergoing a rapid nutrition transition through which obesityrelated chronic conditions account for an increasing percentage of the disease burden. The rapid increase in the rates of obesity in school-age children is particularly concerning because childhood obesity is a risk factor for obesity and related risk factors for cardiometabolic disease (4) later in life. It is crucial to identify modifiable risk factors that are involved in the early development of adiposity to guide future prevention and treatment efforts. Vitamin D insufficiency is highly prevalent in the world; it is estimated that 1 billion people have 25-hydroxyvitamin D [25(OH)D] concentrations consistent with insufficiency (75 nmol/L). Even children who live in subtropical climates are at risk of vitamin D deficiency according to recent studies in Brazil and Costa Rica. Inadequate vitamin D status could be a risk factor for childhood obesity. Vitamin D affects lypolysis and adipogenesis in human adipocytes through its role in regulating intracellular calcium concentrations. Cross-sectional studies indicated that plasma 25(OH)D concentrations are inversely associated with body mass index (BMI; in kg/m2) and waist circumference in children. However, the interpretation of these associations is limited because vitamin D can be sequestered out of the blood and into the larger adipose tissue mass of obese subjects because of its hydrophobic properties. The cross-sectional nature of previous studies precludes the making of an inference regarding the directionality of the association between vitamin D and adiposity. We conducted a prospective study to evaluate the associations between vitamin D serostatus assessed in subjects at enrollment and changes in indicators of adiposity, including BMI, subscapular-totriceps skinfold-thickness ratio, and waist circumference, over 3 y of follow-up in a representative sample of low- and middle-income school-age children from Bogota, Colombia. In addition, we assessed the association between vitamin D serostatus and linear growth.

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It has been established that newborn children, both those born prematurely and full-term, belong to one of the most susceptible groups for the development of vitamin D deficiency. It was noted that the concentration of calcidiol (25(OH)D) in the umbilical cord blood of a newborn baby is no more than 50-80% of the level of 25(OH)D in the mother's blood, regardless of gestational age. It has been shown that vitamin D deficiency is observed in more than half of mothers and newborns. Failure vitamin D in pregnant women and newborns varies across countries depending on race, lifestyle, time of year, and vitamin D intake during pregnancy. These mixed models included random effects for the intercept and slope; we specified an unstructured variancecovariance matrix for these random effects (30). These methods do not require an even number of observations or that measurements be collected at exactly the same time in all subjects; thus, all measurements available for every child were included in the analyses. For the waist-circumference model, random effects for the slope were not included because measurements were only obtained in the second and third years of follow-up; the change in waist circumference represented the change between the 2 measurements taken during follow-up. Because there is some evidence that vitamin D may interact with estrogen, sex-specific estimates were obtained by analyzing boys and girls separately. Models in girls were adjusted for menarcheal status at baseline to control for the potential variability in sexual-maturation stage. Other variables that were related to overweight in this population or to 25 (OH)D concentrations in univariate analyses including maternal characteristics, socioeconomic-status indicators, and time spent playing outdoors were entered into the models. However, none of these variables were significantly related to the outcomes or changed the estimates of association between vitamin D status and anthropometric change. All models used empirical estimates of the variance. Tests for trend were estimated by introducing a continuous variable into the models that represented ordinal categories of vitamin D. Effect modification by sex was assessed with the use of the likelihood ratio test in each model. We examined the associations between vitamin D serostatus and changes in anthropometric indicators of total adiposity and fat distribution, as well as height, in a longitudinal study of school-age children from Bogota, Colombia. After baseline adiposity and other potential confounders were controlled for, a lower vitamin D serostatus was associated with greater increases in BMI and indexes of central adiposity.

Although cross-sectional studies reported inverse associations between vitamin D serostatus and BMI in children, it was not possible to conclude that vitamin D increased the risk of overweight because of reverse-causation bias given that vitamin D can be sequestered in adipose tissue. Only randomized trials or longitudinal studies that were adjusted for baseline adiposity could overcome the potential for reverse causation. Our results suggested that inadequate vitamin D status may prospectively lead to increased adiposity during childhood. It is unlikely that these findings reflect the catch-up growth of malnourished children because the exclusion of children who were thin at baseline did not alter results. In addition, the mean baseline BMI z score was above zero, and the BMI trends by age in this population were very close to the reference median of the World Health Organization; thus, greater BMI changes in vitamin D-deficient children likely represent unhealthy weight gains. There is limited and inconsistent evidence of the association between vitamin D and adiposity from prospective studies. In a calcium-intervention trial in 69 pubertal children, a higher baseline vitamin D status was significantly associated with less weight gain over 24 mo in univariate analyses. The Women's Health Initiative, which is a large trial that assigned women to receive either 1000 mg Ca plus 400 IU vitamin D/d or a placebo

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reported that women who received the regimen of calcium and vitamin D had a small significant lower gain in BMI and waist circumference over 7 y of follow-up; in that study, it was impossible to separate the effects of calcium and vitamin D. In contrast, a trial of overweight adults showed that supplementation with either 20,000 or 40,000 IU cholecalciferol/wk and 500 mg Ca/d did not lead to significantly greater weight loss than the calcium-only control group; however, none of the groups experienced significant weight changes over the 1-y study period. It is possible that vitamin D may limit weight gain but does not affect long-term weight loss in individuals who are already overweight.

The mechanisms by which vitamin D may influence adiposity are unknown, and possible explanations are still speculative. In vitro experiments suggested that vitamin D may prospectively influence the risk of obesity by modulating the catabolic and anabolic activity of adipocytes. Studies have shown that intracellular calcium concentrations modulate lipolytic activity in isolated human adipocytes , which raises the possibility that vitamin D could influence body weight and energy expenditure through calcium regulation. In vitro studies have also shown that vitamin D can inhibit the expression of a key adipogenesis regulator, peroxisome proliferator-activated receptor-gamma . In our longitudinal study, we showed that vitamin D status was negatively associated with changes in waist circumference and subscapular-to-triceps skinfold-thickness ratio after adjustment for baseline adiposity. These findings are particularly worrisome because central adiposity is strongly related to all components of metabolic syndrome in children, including hypertension and insulin resistance . Furthermore, children who accumulate central body fat may be at greater risk of central adiposity and its associated morbidities later in life.

Summarizing the data presented above, we can conclude that fetal development, health status a newborn, an infant and a child in subsequent periods of development are largely determined by the provision vitamin D. Considering the prevalence of vitamin D deficiency among pregnant women and newborns, we can talk about the advisability of its preventive use in these groups. However, taking Taking into account that the prevalence and severity of vitamin D deficiency in pregnant women and their newborns varies widely, it should be noted that the choice of dose of vitamin D for preventive reception must be adjusted taking into account factors causing its insufficiency. Wherein One of the most important indicators of the body's supply of vitamin D is the level of calcidiol in the blood. The criterion for sufficient supply of vitamin D in the body is the level of 25(OH)D in the blood within 30–50 ng/ml, when calcidiol concentration decreases below 20 ng/ml indicates vitamin D deficiency. Our study had several strengths. We collected blood samples from a large and representative sample of children in a setting where the increasing prevalence of child overweight is becoming a serious public-health problem. Our prospective design and use of repeated anthropometric measures enhanced our ability to explore the temporal relation between vitamin D and anthropometric measures and to account for potential reverse causation. One potential limitation of the study is that we assumed that baseline vitamin D serostatus was representative of the cumulative exposure during follow-up. Nevertheless, studies in which 25(OH)D has been measured repeatedly over long periods suggest that the within-subject correlation is high and that a single baseline measurement could be a valid indicator of long-term exposure. Outdoor physical activity could be a confounder of the association between vitamin D status and weight change. However, we did not find that adjustment for the time spent playing outdoors changed the estimates of association in our study; in addition, prospective studies suggested that the association between physical activity and BMI change in children is not strong

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. Another limitation is that we lacked detailed measurements of sexual maturation status (such as Tanner staging), which might be a potential confounder; however, adjustment for menarcheal status at baseline did not change the associations observed in girls.

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In conclusion, vitamin D serostatus was inversely associated with the development of adiposity in school-age children. Randomized intervention studies are needed to ascertain the effect of improving vitamin D status in children on the risk of obesity and other risk factors for chronic disease.

