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VITAMIN D DEFICIENCY INCREASES SCHIZOPHRENIA

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Abstract:
A recent case-control study based on neonatal blood samples identified a significant association between neonatal vitamin D status and risk of schizophrenia. Inadequate vitamin D intake during pregnancy can lead to abnormal bone growth, fractures or rickets in newborns, low maternal vitamin D may impact adversely on the developing fetal brain, leaving the affected offspring at increased risk of adult-onset schizophrenia. This hypothesis may explain diverse epidemiological findings including season of birth, the increased risk in dark-skinned migrants to cold climates and the increased rate of schizophrenia births in urban versus rural setting.

Key Words: *Vitamin D deficiency, Schizophrenia, cofactors of Vitamin-D, epidemiological findings, migrants.*

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INTRODUCTION:

VITAMIN D:

Vitamin D is a fat-soluble vitamin that is naturally present in very few foods, added to others, and available as a dietary supplement. It is also produced endogenously when ultraviolet rays from sunlight strike the skin and trigger vitamin D synthesis. Vitamin D obtained from sun exposure, food, and supplements is biologically inert. Vitamin D promotes calcium absorption in the gut and maintains adequate serum calcium and phosphate concentrations to enable normal mineralization of bone. Without sufficient vitamin D, bones can become thin, brittle, or misshapen. Vitamin D sufficiency prevents rickets in children and osteomalacia in adults.

SCHIZOPHRENIA:

Schizophrenia is a serious disorder which affects how a person thinks, feels and acts. Someone with schizophrenia may have difficulty distinguishing between what is real and what is imaginary; may be unresponsive or withdrawn; and may have difficulty expressing normal emotions in social situations. The cause of schizophrenia is still unclear. Some theories about the cause of this disease include: genetics (heredity), biology (the imbalance in the brain's chemistry); and/or possible viral infections and immune disorders.

VITAMIN D AND SCHIZOPHRENIA:

The link between vitamin D deficiency and the development of schizophrenia has been researched among patients of all ages around the globe. One meta-analysis reviewed 19 studies published between 1988 and 2013 and found a strong association between vitamin D deficiency and schizophrenia. Of the 2,804 participants from these studies, over 65% of the participants with schizophrenia were vitamin D deficient. Vitamin D deficient participants were 2.16 times more likely to have schizophrenia than vitamin D sufficient participants [1-4].

EPIDEMOLOGICAL SURVEY:

Vitamin D concentrations were measured in 50 schizophrenia patients in Israel aged 19-65 Lower mean vitamin D concentrations were detected among patients with schizophrenia (15 ng/ml) compared to controls (20 ng/ml) after adjusting for the impact of sun exposure and supplements [5,6,7]. Likewise, 92% of 102 adult psychiatric inpatients in New Zealand also had suboptimal vitamin D levels and were more than twice as likely as Europeans to have severely deficient levels below <10 ng/ml [8,9].

In a prospective birth cohort of 3,182 children in England, researchers measured vitamin D levels at age 9.8 years and assessed psychotic experiences at age 12.8 years. Vitamin D concentrations during childhood were associated with psychotic experiences during early adolescence. If psychotic experiences are related to the development of schizophrenia, this supports a possible protective association of higher vitamin D concentrations with schizophrenia [10,11,12].

Vitamin D deficiency is associated with more severe symptoms. Cross sectional analyses were carried out on mentally ill adolescents aged 12-18 who required either inpatient or partial hospitalization. Of the 104 patients evaluated, 72% had insufficient vitamin D levels. Vitamin D status was related to mental illness severity. Those with vitamin D deficiency were 3.5 times more likely to have hallucinations, paranoia, or delusions [13]. A second study supports this finding. Vitamin D was analyzed from 20 patients with first-episode schizophrenia. Greater severity of negative symptoms (blunted affect, emotional withdrawal, poor rapport, passive-apathetic social withdrawal, abstract thinking, and stereotyped thinking) was strongly correlated with lower vitamin D status. Lower vitamin D levels were also associated with more severe overall cognitive deficits [14].

SEASON OF BIRTH:

The risk of schizophrenia and vitamin D status vary with season of birth, The UV rays required to make vitamin D are reduced in the months most associated with an increase in the birth of individuals who later develop schizophrenia. One review including a total of 437,710 individuals with schizophrenia found that most individuals were born in January and February. These newborns were thus exposed to lower levels of UV rays in their prenatal and perinatal periods.

An increased rate of schizophrenia is also seen at higher latitudes, especially among immigrants. This may again be related to UV availability and subsequent vitamin D status. At higher latitudes, a dark skinned individual will also have a more pronounced reduction in vitamin D than a lighter skinned individual. The lighter skinned individual will have less melanin which allows the skin to absorb UV rays more effectively. It is estimated that individuals with darker skin at higher latitudes are more likely to develop schizophrenia than the general population [15].

THE URBAN-RURAL CONTRAST:

Some studies defined urban city as the number of people relative to area surface; others, as number of addresses relative to area surface; and yet others, as large city versus small city based on absolute population counts. Some examined urban residence, and most examined urban birth or urban upbringing, although the importance of this distinction is limited, given the fact that urban birth and urban residence are strongly associated. The meta-analysis of these studies indicates that the rate of schizophrenia in (variably defined) urban areas is around double the rate of that in (variably defined) rural areas. The studies address a range of possible confounders such as age, sex, ethnicity, drug use, social class, family history, season of birth, and many others, none of which could explain the apparent association between urban city and psychotic disorders. Also, results were apparent both for studies that depended on hospital admission in defining schizophrenia and for studies that defined the outcome regardless of any contact with services. Therefore, the oldest proposed explanation for the urban–rural difference—reduced distance T a psychiatric hospital in urban areas can also be discarded.

SELECTIVE MIGRATION:

Odegaard argued that the high rates of schizophrenia he observed in Norwegian migrants to the United States could be explained by selective migration, i.e., the greater tendency for individuals with an existing (genetic) predisposition or vulnerability for schizophrenia to migrate. This conclusion was based on Odegaard's observation that many of the migrants who developed schizophrenia had histories of poor social adaptation in Norway. However, when applied to other populations in which high rates have been reported, this explanation seems unlikely. Selten and colleagues conducted an intriguing thought experiment to test this in relation to Surinamese migrants to the Netherlands. They imagined that the entire population of Surinam had migrated to the Netherlands and, using this to inflate denominator data and assuming none of these contributed any further cases of schizophrenia, recalculated incidence rates from an earlier study. Having done this, they found that the risk for Surinamese migrants was still significantly higher than for Dutch individuals (RR 1.46, 95% CI 1.35–1.57). What is more, intuitively it seems reasonable to expect that the cognitive deficits and negative symptoms that are often evident prior to the onset of schizophrenia (and which are assumed to reflect underlying genetic and neurodevelopmental risk) will in fact reduce the likelihood of successful migration. In the only study, we are aware of that has attempted to investigate the impact of psychosis proneness on likelihood of migration, Lundberg et al evaluated potential future migrants in Kampala (Uganda) and found no differences on measures of psychosis-like experiences and mania between those actively planning to migrate and a comparison group with no intention to migrate.

CONCLUSION:

There are supplements in the market that contain all the co-factors vitamin D needs to work properly (including magnesium): zinc (the base of the fingers of the Vitamin D Receptor each contains a zinc molecule), Vitamin K2 (Vitamin K helps direct Vitamin D to calcify the proper organs), boron (boron is involved in the rapid, non-genomic action of Vitamin D on the cell wall), which helps activated Vitamin D stay around longer at the receptor site, and a tiny amount of Vitamin A. The wisest thing to do is to eat green leafy vegetables and a handful of seeds every day as that combination contains the co-factors of Vitamin D.

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