Letter to Editor

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Genetic Polymorphisms of Vitamin D Receptor Gene and Depression

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ABSTRACT

Abbreviations: TPH2: Tryptophan Hydroxylase 2; SERT: Serotonin Reuptake Transporter; MAO-A: Monoamine Oxidase-A; SNP: Single Nucleotide Polymorphism; Bat: BsmI-ApaI-TaqI TAC; baT: BsmI-ApaI-TaqI CCT

Letter

Dear Editor,

It has been demonstrated that certain individuals are predisposed to depression due to a variety of genetic variations [1]. Decreased serum vitamin D levels have been investigated in relation to the manifestations of depressive symptoms. Research indicates that preserving optimal serum vitamin D levels may protect against the onset or severity of depressive symptoms [2]. Vitamin D's potential to influence the onset of mental disorders is emphasized by its multifaceted nature. It is important to note that the pathogenesis of depression is influenced by the expression of a nuclear hormone receptor known as the vitamin D receptor (VDR) and specific cytochrome P450 enzymes involved in converting vitamin D into its active form. This is particularly true for brain regions and cerebral cells. The biologically active form of vitamin D, 1, 25-dihydroxyvitamin D3, operates by binding to VDR. The tryptophan hydroxylase 2 (TPH2) gene is induced by this binding, which in turn regulates the expression of the serotonin reuptake transporter (SERT) and monoamine oxidase-A (MAO-A). This enzyme is implicated in serotonin catabolism [2,3]. Additionally, it has been proposed that vitamin D may have anti-inflammatory properties, which could indirectly alleviate symptoms of depression [1]. Vitamin D and VDR play a complex role in the mechanisms that underlie depression, as evidenced by their presence in cellular and brain compartments [1]. The functional activity of VDR can be influenced by a variety of genetic variations, such as the single nucleotide polymorphism (SNP) FokI (rs2228570), TaqI/BsmI, ApaI, and Cdx2 polymorphisms [3,4].

SNPs can influence theeffects of vitamin D on depressive symptoms by affecting inflammation, calcium homeostasis, and monoamine neurotransmission in the brain. The susceptibility to age-related changes in cognitive functioning and depressive symptoms is influenced by polymorphisms in the VDR gene, specifically BsmI (rs1544410) and TaqI (rs731236), but not of a prospective population-based study that included 563 participants in the Netherlands. In the same vein, a cross-sectional study conducted in Turkey with a sample of 86 individuals diagnosed with major depressive disorder did not disclose any discernible relationship between depression and the FokI polymorphism [3]. A recent study has shown that individuals who carry the BsmI-ApaI-TaqI CCT (baT) haplotype of the VDR

gene are less likely to experience depression than those who take the BsmI-ApaI-TaqI TAC (BAt) haplotype [4]. More specific variations, including Cdx-2, GATA, and 1b-G-886A, have also been identified within the promoter region of the VDR gene. These SNPs may influence the translation capacity or expression of the VDR gene. Previous research indicates that an alternative allele in GATA can increase the expression of VDR, resulting in increased levels of vitamin D in the brain. It is important to note that no significant correlation was discovered between the progression of depression symptoms and any SNPs in the VDR gene [5].

Declaration of Interest Statement

Conflict of Interest

None.

Declaration

None.

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