# Vitamin D moderates the interaction between 5-HTTLPR and childhood abuse in depressive disorders

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A complex interplay between genetic and environmental factors determines the individual risk of depressive disorders. Vitamin D has been shown to stimulate the expression of the tryptophan hydroxylase 2 (TPH2) gene, which is the rate-limiting enzyme for serotonin production in the brain. Therefore, we investigate the hypothesis that serum vitamin D levels moderate the interaction between the serotonin transporter promotor gene polymorphism (5-HTTLPR) and childhood abuse in depressive disorders. Two independent samples from the Study of Health in Pomerania (SHIP-LEGEND: n = 1 997; SHIP-TREND-0: n = 2 939) were used. Depressive disorders were assessed using questionnaires (BDI-II, PHQ-9) and interview procedures (DSM-IV). Besides serum vitamin D levels (25(OH)D), a functional polymorphism (rs4588) of the vitamin D-binding protein is used as a proxy for 25(OH)D. S-allele carriers with childhood abuse and low 25(OH)D levels have a higher mean BDI-II score (13.25) than those with a higher 25(OH)D level (9.56), which was not observed in abused LL-carriers. This significant three-way interaction was replicated in individuals with lifetime major depressive disorders when using the rs4588 instead of 25(OH)D (p = 0.0076 in the combined sample). We conclude that vitamin D relevantly moderates the interaction between childhood abuse and the serotonergic system, thereby impacting vulnerability to depressive disorders.
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