

## Publication

### 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 promoter 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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