1,25(OH)2D3 dependent overt hyperactivity phenotype in klotho-hypomorphic mice

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Klotho, a protein mainly expressed in kidney and cerebral choroid plexus, is a powerful regulator of 1,25(OH)2D3 formation. Klotho-deficient mice (kl/kl) suffer from excessive plasma 1,25(OH)2D3-, Ca(2+)- and phosphate-concentrations, leading to severe soft tissue calcification and accelerated aging. NH4Cl treatment prevents tissue calcification and premature ageing without affecting 1,25(OH)2D3-formation. The present study explored the impact of excessive 1,25(OH)2D3 formation in NH4Cl-treated kl/kl-mice on behavior. To this end kl/kl-mice and wild-type mice were treated with NH4Cl and either control diet or vitamin D deficient diet (LVD). As a result, plasma 1,25(OH)2D3-, Ca(2+)- and phosphate-concentrations were significantly higher in untreated and in NH4Cl-treated kl/kl-mice than in wild-type mice, a difference abrogated by LVD. In each, open field, dark-light box, and O-maze NH4Cl-treated kl/kl-mice showed significantly higher exploratory behavior than untreated wild-type mice, a difference abrogated by LVD. The time of floating in the forced swimming test was significantly shorter in NH4Cl treated kl/kl-mice compared to untreated wild-type mice and to kl/kl-mice on LVD. In wild-type animals, NH4Cl treatment did not significantly alter 1,25(OH)2D3, calcium and phosphate concentrations or exploratory behavior. In conclusion, the excessive 1,25(OH)2D3 formation in klotho-hypomorphic mice has a profound effect on murine behavior.
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