SAN2021
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CELLULAR AND MOLECULAR NEUROBIOLOGY

The ketone body β-hydroxybutyrate (βHB) rescues behavioral defects in DAF-18/PTEN mutants of C. elegans

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Mutations in the phosphatase and tensin homolog (PTEN) gene, a negative regulator of the Akt/PKB pathway, are associated with neurodevelopmental disorders (NDDs). In recent years, ketogenic diets (KGDs) have been shown to have beneficial behavioral effects in animal models of NDDs. Ketogenic diets trigger a metabolic shift by forcing the production of ketone bodies (KBs) to generate ATP. The mechanisms underlying the beneficial effects of KGDs on NDDs are unknown. Here we used daf-18/PTEN mutants of C. elegans to gain molecular and cellular insights into the effects of KGDs on neurodevelopment. We find that these mutants are defective in exerting a complex behavior such as the escape response. These behavioral defects improve in animals cultured in the presence of KB β-hydroxybutyrate (βHB). Surprisingly, exposure to βHB at early stages is sufficient to achieve this improvement throughout adulthood, suggesting that βHB is necessary at a critical stage of development. We have also found that the effect of βHB is abolished in daf-16/FOXO mutants, revealing a key role for this transcription factor. Finally, we observed morphological defects in GABAergic motor neurons in daf-18 mutants. We are exploring whether exposure to βHB can amend these abnormalities. Given the high level of conservation of the pathways involved (PTEN/AKT/FOXO) across the animal kingdom, this work could contribute to better understand NDDs and establishing potential therapeutic options in mammals.