SFN-rich broccoli sprouts in subjects at a high-risk of developing psychosis may prevent the onset of psychosis at adulthood.

PM366

Three-dimensional analysis of morphological changes in dendritic spines and mitochondria in dentate gyrus granule cells in Schnurri-2 knockout mice, an animal model for schizophrenia

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Abstract

Accumulating evidence suggests that morphological changes of subcellular-scale structures such as dendritic spine and mitochondria may be involved in the pathogenesis/pathophysiology of schizophrenia. Previously, we have proposed mice lacking Schnurri-2 (Shn2), an MHC enhancer binding protein, as a schizophrenia model with mild chronic inflammation. In the mutants, there are decreases in the expression level of PSD95, synaptic marker, and increases in C1q family genes (C1qa, C1qc and C1ql2), which are considered to mediate synapse elimination during the postnatal development. In the present study, we analyzed three-dimensional morphological changes in dendritic spines and mitochondria in dentate gyrus granule cells in Shn2 KO mice by serial block-face scanning electron microscopy. The mutants showed about 13% increase in spine length, and about 25% increase in spine neck length. There were no significant differences between Shn2 KO and wild-type mice in their spine density, volume of spine, spine head length, or spine head diameter. The mutants exhibited reduced complexity in mitochondrial morphology, suggesting that a balance between mitochondrial fusion and fission is compromised in Shn2 KO mice. These morphological changes in spines and mitochondria may be associated with functional impairments of those subcellular-scale structures, and represent potential endophenotype of schizophrenia.

PM367

Effect of cannabidiol on cognition in a maternal immune activation (poly IC) model of schizophrenia

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Abstract

Background: Maternal immune activation (MIA) during pregnancy is a risk factor for schizophrenia and may be linked to cognitive deficits of the disorder [1, 2]. Current antipsychotics show minimal improvement of cognitive deficits [3] and have adverse side-effects [4]. Cannabidiol (CBD), a constituent of cannabis, possesses anti-inflammatory, antipsychotic and neuroprotective properties [5]; however, whether CBD can improve cognition in schizophrenia is unknown. The aim of this study was to examine effects of CBD on cognition in a MIA model of schizophrenia.

Methods: Pregnant dams were administered either poly IC (POLY; 4mg/kg) or saline (CONT). From postnatal day 56, offspring were administered either vehicle (CONT+VEH, POLY+VEH) or CBD (10 mg/kg; CONT+CBD, POLY+CBD) twice daily for 3 weeks (n=12 males, 12 females/group). Novel object recognition (NOR) and T-maze tests were performed to assess recognition and working memory.

Results: In the NOR test, POLY+VEH males spent significantly less time exploring the novel object compared to CONT+VEH counterparts ($p<0.01$), and in the T-maze test, POLY+VEH males exhibited a lower number of correct entries ($p<0.01$ vs. CONT+VEH). Performance of POLY+CBD males was significantly better than POLY+VEH in both tests ($p<0.05$) and did not differ from CONT+VEH ($p>0.05$). No differences were observed between CONT+VEH and CONT+CBD males ($p>0.05$). There was no effect of poly IC or CBD treatment on female offspring performance in either test ($p>0.05$).

Conclusions: Interestingly, CBD treatment restored working and recognition memory impairments in the poly IC model of schizophrenia, but had no effect in control rats. These effects were observed in male offspring, whereas females showed no cognitive impairment. The mechanisms underlying the apparent protection in females and sensitivity in males warrant further investigation. This is the first study to examine chronic CBD treatment effects in a MIA model, and suggests that CBD may have sex-specific therapeutic potential for schizophrenia-related cognitive impairments.

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PM368

Ketogenic diet normalises schizophrenia-like behaviours in acute and chronic NMDA-receptor hypofunction models

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Abstract

Objective: Impaired glucose and energy metabolism may play a role in the pathobiology of schizophrenia. Abnormal brain energy metabolism has been found in animal models of