Instructions for posters and abstracts at the VNPN-2
Virginia-Nordic Precision Neuroscience conference Oslo, Norway, 19-21 September 2018

Poster abstracts should be sent to jonsm@med.uio.no (Subject: VNPN Abstract) before 1st September as attached pdf in the PubMed 'Abstract' format (see sample below; text about 2000 characters with spaces).

Approved abstracts are to be published (with ISBN) at the web site.

Posters should be displayed throughout the meeting, size max 95cm x 135cm (WxH).

Underline the presenting author. Supply up to 5 KEYWORDS.

Template / sample format:

A ketogenic diet improves mitochondrial biogenesis and bioenergetics via the PGC1α-SIRT3-UCP2 axis

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A ketogenic diet (KD; high-fat, low-carbohydrate) can benefit refractory epilepsy, but underlying mechanisms are unknown. We used mice inducibly expressing a mutated form of the mitochondrial DNA repair enzyme UNG1 (mutUNG1) to cause progressive mitochondrial dysfunction selectively in forebrain neurons. We examined the levels of mRNAs and proteins crucial for mitochondrial biogenesis and dynamics. We show that hippocampal pyramidal neurons in mutUNG1 mice, as well as cultured rat hippocampal neurons and human fibroblasts with H_2O_2 induced oxidative stress, improve markers of mitochondrial biogenesis, dynamics and function when fed on a KD, and when exposed to the ketone body β-hydroxybutyrate, respectively, by upregulating PGC1α, SIRT3 and UCP2, and (in cultured cells) increasing the oxygen consumption rate (OCR) and the NAD^+/NADH ratio. The mitochondrial level of UCP2 was significantly higher in the perikarya and axon terminals of hippocampus CA1 pyramidal neurons in KD treated mutUNG1 mice compared with mutUNG1 mice fed a standard diet. The β-hydroxybutyrate receptor GPR109a (HCAR2), but not the structurally closely related lactate receptor GPR81 (HCAR1), was upregulated in mutUNG1 mice on a KD, suggesting a selective influence of KD on ketone body receptor mechanisms. We conclude that progressive mitochondrial dysfunction in mutUNG1 expressing mice causes oxidative stress, and that exposure of animals to KD, or of cells to ketone body in vitro, elicits compensatory mechanisms acting to augment mitochondrial mass and bioenergetics via the PGC1α-SIRT3-UCP2 axis (The compensatory processes are overwhelmed in the mutUNG1 mice by all the newly formed mitochondria being dysfunctional).

KEYWORDS: Bioenergetics; Biogenesis; Ketogenic diet; MutUNG1