Neuroscience & Non-Communicable Diseases WEBINAR – All welcome

Friday 18 September, 3pm

"Feeding regulation by NPY system under stress and obese conditions"

Speaker: Prof Herbert Herzog, Garvan Institute of Medical Research; UNSW

Prof Herbert Herzog is a NHMRC Senior Principal Research Fellow. He received Bachelor of Science in 1986 and PhD in 1989 (University of Innsbruck, Austria), and Doctor of Science in 1996 (Free University of Berlin). Herbert undertook postdoctoral research at Garvan Institute in 1991, where he established his research group and have had a great success since. He was a Program Director of the Neuroscience Research Program at Garvan (2005-2015). Herbert is an international authority in obesity research, particularly known for his ground-breaking work on neuropeptides. He has published close to 300 papers.

Synopsis:

In addition, NPY in the arcuate nucleus (Arc) is known to be strongly upregulated under negative energy balance to increase feeding and reduce energy expenditure. Paradoxically however, reduced NPY levels as seen in response to a positive energy balance still seem to be able to drive feeding. We have now discovered that at least two distinct Arc NPY populations exist, AgRP-positive and AgRP-negative NPY neurons, which as confirmed by RNAseq, respond differently to fasting. Employing DREADD technology further revealed that AgRP-positive NPY neurons are critical for initiating food-seeking behaviour, inducing physical activity and protecting fat stores, while AgRP-negative NPY neurons specifically drive feeding in response to fasting and also under obese conditions, whilst not influencing energy expenditure. Importantly, mRNA expression of the high affinity Y2 receptor is increased in the Arc of HFD fed as well as genetically obese db/db mice suggesting that feeding under positive energy balance conditions is predominantly controlled by Y2 receptors compared to the control of feeding under normal energy balance conditions by the lower affinity Y1/Y5 receptors. Especially, NPY originating from the AgRP-negative NPY neurons is critical in activating Y2 receptors located on POMC neurons under positive energy balance conditions to drive food intake higher and accelerating the development of an obese phenotype.

All welcome!

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