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Regulation of hypothalamic neuronal function in obesity

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Obesity develops with chronic consumption of palatable energy-dense diets, and also with increasing age. With persistent positive energy balance, the increase in body weight is accompanied by a steady rise in circulating leptin levels, indicating the progressive development of counter regulatory mechanisms to antagonize leptin's anorexigenic effects. Hypothalamic neurons co-expressing agouti-related peptide (AgRP) and neuropeptide Y are direct leptin targets. We have recently shown that AgRP neurons are the predominant cell type situated outside the blood-brain barrier in the mediobasal hypothalamus. AgRP neurons are able to sense slight changes in plasma metabolic signals, such as leptin, but they also more quickly develop cellular leptin resistance in contrast to proopiomelanocortin (POMC) and other hypothalamic neurons that remain leptin-sensitive. AgRP neurons also display age-dependent increase in innervation onto their target neurons, and this process is accelerated by chronic high-fat feeding. Our studies suggest that AgRP neurons are critical sensors for peripheral metabolic hormones and that they play a dynamic role in metabolic fine tuning in response to acute changes in nutritional status. Our studies also suggest that these neurons, with their unique anatomical relationship with the blood-brain barrier, could serve as important targets for therapeutic intervention for the treatment of metabolic disorders.

Biography

Allison W Xu completed her PhD at University of Texas - M.D. Anderson Cancer Center and conducted her Postdoctoral studies at Stanford University School of Medicine. Subsequently, she started her independent research program, and she is currently an Associate Professor in the Diabetes Center at University of California, San Francisco. Her laboratory focuses on understanding how hypothalamic neurons sense and respond to peripheral metabolic hormones and how these regulatory circuits regulate food intake, body weight, hepatic lipid and glucose metabolism in normal physiology and obesity.

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