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213-OR: Obesity-Induced Astrogliosis Is Regulated by the Diabesity Factor HMG20A

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Previous

Article Info & Metrics

Abstract

It is well established that systemic glucose homeostasis is imparted by an intricate cross-talk among several organs including the central nervous system (CNS) and pancreatic islets. In analogy to islet beta cells, hypothalamic astrocytes are glucose sensors that facilitate the CNS response to changing metabolic environment. Therefore, astrocytes and beta cells may share a common genetic signature implicated in coupling glucose metabolism to cellular output. We recently demonstrated that the diabesity factor HMG20A is essential for beta-cell functional maturity and adaptation to physiological stress (obesity, type 2 diabetes and pregnancy). As this chromatin remodelling factor also dictates CNS development, we sought to determine whether HMG20A is expressed in astrocytes and whether it potentiates astrocyte function in response to

environmental cues. We found that HMG20A is expressed in hypothalamic astrocytes and is upregulated in diet-induced obesity and glucose-intolerant mice, correlating with increased transcript levels of GFAP and IL-1β, indicative of reactive astrocytes (astrogliosis). High glucose, but not lipids, transiently decreased HMG20A expression in isolated mouse primary astrocytes. HMG20A silencing in astrocytes resulted in repression of pro-inflammatory as well as lipid and glucose metabolism genes. These pathways are up-regulated in activated astrocytes (astrogliosis) to resolve stress conditions. As such astrocyte depleted of HMG20A were more susceptible to cell death. Furthermore, motor neuron viability in astrocyte conditioned media was significantly hindered after HMG20A depletion in astrocytes. We posit that HMG20A is involved in the astrocyte polarization state. Under physiological pressure such as obesity, HMG20A expression may be transiently decreased by high glucose but long-term it is increased to induce astrogliosis in an attempt to preserve the neuronal network and glucose homeostasis.

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Previous

Back to top

In this Issue

June 2020, 69(Supplement 1)
Table of Contents
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