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Modulation of microglia-mediated neuroinflammation as a novel aspect of insulin activity in the central nervous system

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Recently, type 2 diabetes (T2D) has been identified as a risk factor for Alzheimer's disease (AD). Peripheral inflammation and insulin resistance characterize T2D, while AD brains display chronic neuroinflammation, and central nervous system (CNS) insulin deficiency. The role of insulin in neuronal survival and plasticity has been described extensively; additionally, this hormone is known to regulate peripheral inflammation. We hypothesized that inflammation link T2D with AD. Using an *in vitro* human cell culture system we investigated the effects of insulin on neuroinflammation. We demonstrate that human primary microglia express insulin receptors and insulin receptor substrates. We further show that insulin upregulates the secretion of interleukin (IL)-8, and suppresses the secretion of monocyte chemoattractant protein (MCP)-1 by stimulated human microglia. Additionally, we show that insulin reduces the toxicity of stimulated human THP-1 monocytic cells (used as a model of microglia) towards human SH-SY5Y neuronal cells. These data demonstrate that insulin can affect the viability of neurons not only directly, but also indirectly by modulating glial cell secretions, which in turn impacts the survival of surrounding neuronal cells. This novel insulin-microglial interaction may provide new targets for drug development and therapies aimed at alleviating the symptoms and progression of AD.

Biography

Lindsay J Spielman completed her BSc with distinction from Vancouver Island University and is currently completing her PhD at the University of British Columbia Okanagan. She has published 4 papers in the field of neuroscience and an additional 4 publications in the field of education.

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