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Searching for new possibilities to target neuro-developmental disorders associated with cognitive disabilities

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It is well established that synapses undergo changes during the process of learning and the formation of memories, what is known as synaptic plasticity. This process, which depends on actin cytoskeleton remodeling, has been reported to be abnormal in several autistic disorders such as Fragile X syndrome (FXS). One of the proteins involved in these actin dynamics is Rac1, a protein of the Rho subfamily of GTP binding proteins. Thus far, the functional role of Rac1 in adult neuronal signaling is relatively unknown. Rac1 is highly expressed in the adult mouse hippocampus, a part of the brain crucial for learning and memory. *In vitro* and *in vivo* studies from our laboratory revealed that

1. activation of Rac1 is associated with NMDA receptor activation;
2. Rac1 function is associated with learning in the adult mice;
3. Rac1 is necessary for long-term plasticity in the hippocampus; and
4. regulation of Rac1 is altered in a mouse model of FXS.

Thus, it is hypothesized that the aberrant dendritic spine morphology and altered long-term plasticity observed in FXS is likely related to the observed up-regulation of Rac1. In an effort to define whether there is a critical role for Rac1 in FXS-associated learning deficiencies, Rac1 was pharmacologically targeted using novel agents that inhibit its activation and therefore, its function. Mice treated with Rac1 inhibitors and subjected to behavioural tasks revealed that these animals improved their cognitive capabilities. Moreover, this treatment was able to reverse the induction of audiogenic seizures, which is another strong phenotype of this mouse model and disease. These findings suggest that Rac1 hyperactivity may play an important role in FXS and autism-related molecular pathology contributing to cognitive problems and other alterations.

Biography

Maria Victoria Tejada-Simon is an Associate Professor of Pharmacology, Biology and Psychology, and member of the Biology of Behaviour Institute at the University of Houston in Houston, Texas. Current research in her laboratory examines the role of small GTP-binding proteins in cognition, with emphasis in mental retardation, schizophrenia, neurodegenerative as well as autistic disorders.

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