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Prospects in the field of pharmacotherapies for Down syndrome

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Down syndrome (DS), a relatively high-incidence genetic condition due to triplication of chromosome 21, is characterized by brain hypotrophy and intellectual disability. There are currently no effective therapies for intellectual disability in DS. Since brain hypotrophy is due to impairment of neurogenesis and dendritogenesis starting from prenatal and early postnatal life stages, the perinatal period represents a crucial time window of opportunity for possible interventions aimed at restoring these processes and, possibly, intellectual disability. Based on these premises, the overall goal of our study was to establish whether it is possible to restore the trisomy-linked neurodevelopmental defects with early pharmacological intervention. To this purpose, we have exploited the Ts65Dn mouse, a model of DS that mimics the human condition. We have targeted pathways that are crucial for neurogenesis and are known to be deranged in the DS brain. We identified various drugs that can restore neurogenesis and obtained evidence that some of them elicit long-lasting effects on cognitive performance. In my presentation, I will compare and discuss the potential translational value of the tested drugs. In view of its relatively high incidence, DS represents a public health problem and source of concern for families and caregivers. Demonstration that early therapy may restore the defects of the DS brain in a DS model may prompt clinical trials in children with DS and open a breakthrough regarding intellectual disability in DS.

Recent Publications

1. Guidi S, Stagni F, Bianchi P, Ciani E, Giacomini A et al. (2014) Prenatal pharmacotherapy rescues brain development in a Down's syndrome mouse model. Brain. 137(Pt 2):380-401.
2. Stagni F, Raspanti A, Giacomini A, Guidi S, Emili M et al. (2017) Long-term effect of neonatal inhibition of APP gamma-secretase on hippocampal development in the Ts65Dn mouse model of Down syndrome. Neurobiol Dis.103:11-23.

Biography

Sandra Guidi graduated in Biology at the Bologna University, Bologna, Italy (2000). She carried out a Post-degree training at the Department of Human and General Physiology in Bologna (2000-2007). From 2008 she is Assistant Professor of Physiology, Medical School, University of Bologna and Lecturer of Neurophysiology. Her research work focuses on the mechanisms underlying impairment of brain development in a mouse model Down syndrome, with the goal to devise therapies potentially transferable to individuals with DS. She has authored over 30 publications that have been cited over 1200 times. Her publication H-index is 18.

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