Ferdinando Rossi Lectures on Neuroscience

Neuroscience Institute Cavalieri Ottolenghi

UNIVERSITÀ DEGLI STUDI DI TORINO

DIPARTIMENTO DI NEUROSCIENZE RITA LEVI MONTALCINI

NICO
Neuroscience Institute Cavalieri Ottolenghi

FONDAZIONE CAVALIERI OTTOLENGHI

NIT
ISTITUTO NAZIONALE DI NEUROSCIENZE

SSST
SCUOLA DI STUDI SUPERIORI FERDINANDO ROSSI
UNIVERSITÀ DEGLI STUDI DI TORINO

Giovedì 24 gennaio - ore 14:00

Università di Torino
Aula Magna G. Levi, Istituto di Anatomia
Corso Massimo D'Azeglio 52 - Torino
Ingresso libero fino a esaurimento dei posti disponibili

A cinque anni dalla sua scomparsa, un ricordo del neuroscienziato, docente e fondatore della SSST.

Alessandro Vercelli, Direttore scientifico del Neuroscience Institute Cavalieri Ottolenghi - Università di Torino

Alessandro Mauro, Direttore del Dipartimento di Neuroscienze Rita Levi-Montalcini dell'Università di Torino

Michele Graziadei, Vice presidente della Scuola di Studi Superiori Ferdinando Rossi dell'Università di Torino

GABAergic synapses as possible targets of Autism Spectrum Disorders

Enrico Cherubini
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Early in postnatal life, γ-aminobutyric acid (GABA), the primary inhibitory transmitter in adulthood, excites targeted neurons by an outwardly directed flux ofchloride which results from the unbalance between thecation–chloride cotransporters NKCC1 and KCC2, involved in chloride uptake and extrusion, respectively. The depolarizing action of GABA leads to intracellular calcium rise through voltage-dependent calcium channels and/or N-methyl-D-aspartate receptors. GABA-mediated calcium signals regulate a variety of developmental processes from cell proliferation migration, differentiation, synapse maturation, and neuronal wiring. Therefore, it is not surprising that some forms of neuro-developmental disorders such as autism spectrum disorders (ASDs) are associated with alterations of GABAergic signaling and impairment of the excitatory/inhibitory balance within selective neuronal circuits.

ASDs comprise a heterogeneous group of neuro-developmental disorders, mainly of genetic origin, characterized by impaired social interactions, communications deficits, stereotyped and repetitive behaviors. In rare cases, monogenic heritable forms, associated with single mutations in genes involved in synaptic function have been identified. One of these, the R451C mutation of the gene encoding for Neuroligin3 (NLG3), has been found in patients with familial forms of ASDs. NLGs are postsynaptic adhesion molecules that interacting with their presynaptic partners neurexins ensure trans-synaptic signaling and synapse's stability. Animals carrying this mutation ($NL3^{R451C}$ knock-in mice) exhibit impaired social behavior, reminiscent of those observed in ASD patients. In addition these mice present major alterations in GABAergic signaling and synaptic plasticity processes.