

2<sup>nd</sup> International Conference on

## Autism

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**Knock down of neuroligin-2 in cortical region leads to hyperactivity and autistic like behavior features**Deeba Noreen Baig<sup>1,2</sup>, Woo Yang Kim<sup>2</sup> and Katsuhiko Tabuchi<sup>3</sup><sup>1</sup>Forman Christian college, Pakistan<sup>2</sup>University of Nebraska Medical Center, USA<sup>3</sup>Shinshu University, Japan

Neuroligins (NL) and their genetic variants were found to be strongly implicated in the pathophysiology of autistic disorders. Neuroligin-2 (NL2) is a postsynaptic cell adhesion molecule, which is predominantly expressed at inhibitory synapses and required for synapse specification and stabilization. NL2 knockout mice lacking functional NL2 were shown to result in alterations of social behaviors as well as altered inhibitory synaptic transmission, hence modifying the excitation to inhibition balance. Here, we focused on the role of NL2 in the cerebral cortex in the regulation of social behaviors. To this purpose, we designed shRNA system based sh-NL2 construct and injected in lateral ventricles of embryonic brain of mice by in utero electroporation (IUE) to knock down the expression of NL2 at transcriptional level. The effects of NL2 gene silencing were explored by analyzing the expression of NL1 to check impairment in synaptic balance. Our results suggest the synchronization of IUE and shRNA silencing technology proved to be highly successful to determine the effect of local suppression of NL2 in defined compartment cortex of brain. Despite of their confined knockdown of NL2 in the cortex, *in vitro* studies indicated that it is strongly disturbed normal excitation to inhibition balance. The induced synaptic imbalance in cortex critically appeared in hyperactive stereotypies and impaired social interaction of mice, which are key features of autistic like behavior.

**Biography**

Deeba completed her masters in animal sciences, from Department of Zoology, University of Punjab and proceeded for PhD in the area of cell and molecular biology from School of biological sciences in the same university. She graduated in August 2008 continued as research associate in the same department. In 2010, Deeba Noreen Baig joined National institute of Physiological Sciences (NIPS), Okazaki, Japan, as post-doctoral fellow. In NIPS, she was working under direct supervision of Ryuichi Shigemoto, who is a renowned scientist in the field of freeze fracture replica plating and electron microscopy. She acquired immuno-histochemistry and confocal microscopy skills there. Keeping in view my interest in the areas of neurosciences and molecular biology, he directed Deeba to Tanaka Shinji, in University of Tokyo, to whom Deeba learned embryonic in vivo microinjection and electroporation system, which is break through now a days in the field of neuro-molecular biology. On returning to NIPS, Deeba Noreen Baig started working in my previous lab with Katsuhiko Tabuchi and established in vivo microinjection and electroporation system to generate autism candidate gene transgenic model mice. In 2012, she was awarded prestigious post-doctoral fellowship from Higher Education commission Pakistan and she joined University of Nebraska Medical Center as foreign post doctoral fellow. In UNMC she has been engaged in Woo-Yang Kim lab on autism project. In 2013, she appointed as Assistant Professor in Department of Biological Sciences, Forman Christian College University, Lahore. Here, based on my specialization and expertise she is pursuing research in continuation of autism project in Pakistani population.

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