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The zebrafish: as a model to decipher the molecular and cellular deficits underlying Autism Spectrum Disorders (ASD) and identify therapeutic targets

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Background: Autism spectrum disorders (ASD) are neurodevelopment disorders characterized by typical triad of symptoms: impaired social interactions, communication deficits, and stereotypical behaviors. Incidence rates vary widely around the world, from 1.85% of children in the US to 0.38% in the UK, and twin studies supported a high heritability ranging between 60 and 80%. Recent WES/WGS indicated that genetic factors are causative in at least 50% of the cases, involving ~ 1000 genes. However, besides purely genetic forms, other causative factors linked to disturbances of the fetus environment in utero have also been identified. First, epidemiological data showed that maternal infection during gestation and subsequent immune activation increase the risk of autism in the child. Also, sodium valproate (VPA), an antiepileptic drug widely used since the 1980s, has been more recently identified as a teratogenic agent that increases a pregnant woman's risk of having children with birth defects and ASD. Here, our objective was to study the neurotoxicity of VPA in zebrafish larvae and the behavioral consequences of early stage exposure to VPA. Methods: By taking advantage of the behavior of this small fish that lives in large schools (shoaling), we designed an experimental setup allowing to rapidly evaluate and quantify the social behavior (visual attirance toward conspecifics) of a dozen of larvae simultaneously, using individuals exposed to VPA (10 and 20 µM) from 4 to 24 hours post-fertilization and then grown for at least 24 days. Results: Individuals exposed early to VPA display social deficit as indicated by their lack of attraction towards conspecifics (Fig. 1).

Conclusion: Our zebrafish model reproduces a core symptom of ASD, social interaction deficit, supporting a strong, albeit unexpected, evolutionarily conservation of the neurobehavioral consequences of early VPA exposure and, thus, of the neuronal deficits underlying ASD

Biography

Constantin Yanicostas is a CNRS researcher with a long experience in molecular biology and genetics, and, more recently, neurosciences, and he is the author/co-author of some thirty articles in peer-reviewed journals. For the past ten years, he has been studying neuron-microglia interactions in physiological and disease situations, using the zebrafish as a model. He has also a solid knowledge of zebrafish models of human neurological diseases; tauopathies, spastic paraplegia, spinocerebellar ataxia, and neurodevelopmental disorders, such as epileptic encephalopathies and autism spectrum disorders. More recently, he has also focused on the environmental toxicity of pesticides and other toxins, and their effects on the nervous system, particularly the developing brain.

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