

Glutamatergic neurotransmission system is impaired in an adult zebrafish FASD model

Suelen Baggio

Federal University of Rio Grande do Sul, Brazil



Abstract

Background: Fetal Alcohol Spectrum Disorder (FASD) is a syndrome related to ethanol (EtOH) exposure during development with neurological impairment. Glutamatergic neurotransmission is modulated by EtOH exposure, which affects synaptic plasticity and cognitive processes. Adult zebrafish is a known model to evaluate long-lasting impairments of milder forms of FASD, also used to investigate the glutamatergic system. Aim: Evaluate brain glutamatergic system of adult zebrafish exposed to ethanol during development. Methods: zebrafish larvae (24 h post-fertilization), were exposure to EtOH (0.0%, 0.1%, 0.25%, 0.5% and 1%) for 2 hours. After 4 months, the animals were euthanized and their role brain was used to access: glutamate transport uptake activity; glutamate binding in enriched membrane fraction; glutamine synthetase (GS) activity; Na+/K+ ATPase activity; and high-resolution respirometry. Results: Animals exposed to EtOH 0.5% and 1% presented a 50% reduction of brain glutamate uptake compared to control (p < 0,001). Ceftriaxone, a positive modulator of glutamate uptake, rescued 50% of this drop (p < 0,0001). Both groups presented reduced levels of glutamate binding compared to control (43% and 60%, respectively, p = 0,0041). Both groups presented 32% reduction in Na+/K+ ATPase activity compared to control (p = 0,0003). One-fifth of GS activity was reduced on EtOH 1% group compared to control (p = 0,0032). No alterations were observed in high-resolution respirometry. Conclusion: Embryonic alcohol exposure disrupts adult zebrafish glutamatergic neurotransmitter system



Biography

I am a Biologyst and PhD. student of Biochemistry in Brazil. My research involves brain glutamatergic system impairment on an adult zebrafish FASD model. My goal is to increase our understanding in the alterations of neurochemical functions in the glutamatergic system, in an addition to an attempt to modulate these impairments observed FASD with pharmacological modulation. I study this model since 2014, which resulted in two publications as first author: Embryonic alcohol exposure promotes long-term effects on cerebral glutamate transport of adult zebrafish (Neuroscience Letters, 2017), and Embryonic alcohol exposure leading to social avoidance and altered anxiety responses in adult zebrafish (Behavioral Brain Research, 2018).

Publication

• Baggio S, Mussulini BH, de Oliveira DL, Gerlai R, Rico EP (2018) Embryonic alcohol exposure leading to social avoidance and altered anxiety responses in adult zebrafish. Behavior Brain Research 352:62-69

• Baggio S, Mussulini BH, de Oliveira DL, Zenki KC, Santos da Silva E, Rico EP (2017) Embryonic alcohol exposure promotes long-term effects on cerebral glutamate transport of adult zebrafish. Neuroscience Letters 636:265-269

• Mahabir S, Chatterjee D, Gerlai R (2018) Short exposure to low concentrations of alcohol during embryonic development has only subtle and strain- dependent effect on the levels of five amino acid neurotransmitters in zebrafish. Neurotoxicology and Teratology 68:91-96.

• Fernandes Y, Gerlai R (2009) Long-term behavioral changes in response to early developmental exposure to ethanol in zebrafish. Alcoholism, clinical and experimental research 33(4):601-9

• Rothstein JD, Patel S, Regan MR, Haenggeli C, Huang YH, Bergles DE, Jin L, Dykes Hoberg M, Vidensky S, Chung DS, Toan SV, Bruijn LI, Su ZZ, Gupta P, Fisher PB (2005) Beta-lactam antibiotics offer neuroprotection by increasing glutamate transporter expression. Nature 6;433(7021):73-7

10th Global Summit on Neuroscience and Neuroimmunology | Paris | February 19-20, 2020

Citation: Suelen Baggio, Glutamatergic neurotransmission system is impaired in an adult zebrafish FASD model, Neuroimmunology 2020, Paris, February 19-20, 2020, PP. 27