Modulation of Postnatal Neurogenesis by Perinatal Asphyxia: Effect of D1 and D2 Dopamine Receptor Agonists

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 Perinatal asphyxia (PA) is associated to delayed cell death, affecting neurocircuitries of basal ganglia and hippocampus, and long-term neuropsychiatric disabilities. Several compensatory mechanisms have been suggested to take place, including cell proliferation and neurogenesis. There is evidence that PA can increase postnatal neurogenesis in hippocampus and subventricular zone (SVZ), modulated by dopamine, by still unclear mechanisms. We have studied here the effect of selective dopamine receptor agonists on cell death, cell proliferation and neurogenesis in organotypic cultures from control and asphyxia-exposed rats. Hippocampus and SVZ sampled at 1?3 postnatal days were cultured for 20?21 days. At day in vitro (DIV) 19, cultures were treated either with SKF38393 (10 and 100 µM, a D1 agonist), quinpirole (10 µM, a D2 agonist) or sulpiride (10 ?M, a D2 antagonist) + quinpirole (10 ?M) and BrdU (10 ?M, a mitosis marker) for 24 h. At DI