Postnatal betamethasone administration produces delayed changes in the immuno histochemical expression of brain-derived neurotrophic factor and the tyrosine kinase B receptor in the rat cerebellar cortex together with anxiety like behavior

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Postnatal synthetic glucocorticoids (GCs) are widely used in the prevention of chronic lung disease in premature infants and their pharmacologic use is associated with neurodevelopmental delay and various behavioral and brain changes. Using classic Golgi staining methods, we previously showed that the administration of GCs betamethasone (BET), in equivalent doses to those given in cases of human premature birth, generates long term alterations in Purkinje cell (PC) dendritic development in the cerebellar cortex. On the other hand, the brain-derived neurotrophic factor (BDNF) and the tyrosine kinase B receptor (TrkB) are involved in cerebellar PC dendritic development and maintenance and are located predominantly in the cerebellar molecular layer where PC dendritogenesis occurs. Therefore, we hypothesized that reductions in protracted lower PC dendritic arborization could be due, at least in part, to changes in dendritic expression of BDNF and TrkB. On the other hand, previous studies demonstrated that animals that experience stress, either via chronic maternal stress or exogenous GCs administration, exhibited a significant alteration in the neurotrophic factors regulation and behavioral changes associated with anxious behaviors. For these reasons, in the present study, we evaluated whether postnatal administration of betamethasone alters the immunohistochemical (IHC) expression of BDNF and TrkB in the cerebellar cortex along with anxiety behaviours in infants, adolescent and adult rats. Consistent with our previous studies, we observed that animals postnatal exposed to betamethasone showed long term alterations in the IHC expression of BDNF and TrkB in adolescent and adult rats. Additionally, these protracted molecular changes were accompanied by an increase of anxiety like behaviors in the elevated plus maze and marble burying test in adolescent and adult rats.

Recent Publications


Biography

Martina Valencia is a part of the research team at the Neuroscience Laboratory of the Pontifical Catholic University of Valparaíso, Chile and has her expertise in university teaching in subjects such as neurophysiology, research methodology and history of science. In parallel and more than seven years ago, she carried out research on the effect of prenatal stress on neurodevelopment. To achieve this goal, the work is based on a problem that occurs in the obstetric clinic: the use of antenatal and postnatal synthetic glucocorticoids in the prevention of chronic lung disease in premature infants and their adverse side effects at the level of the Central Nervous System, such as, neurodevelopmental delay and various behavioral and brain changes observed in the progeny. She has built this model after years of experience in research, evaluation, teaching and administration both in hospital and education institutions.

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