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Maternal exposure to novel atypical psychotropic drugs: Impact on fetal brain development, apoptosis, neurodegeneration and psychopathological sequelae in young offspring

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Clinical and non-clinical literature revealed that *in utero* exposure to classical psychotropic drugs may lead to abnormal brain development and related functional disorders in children, but reports on new generation psychotropic drugs are limited and inconclusive. Therefore, present study has been taken to reveal the impact of therapeutic psychotropic agents on fetal brain development and genesis of psychiatric disorders in young offspring. In this study, pregnant C-F rats were used and equivalent therapeutic doses of some atypical psychotropic drugs of different classes like antipsychotics (RIS, QUE, ARI and ASN), antiepileptics (PGB, ESL and GBP) and antidepressants (VEN) were administered during sensitive phase of fetal brain development. At GD 21, about half of the dams of drugs exposed and non-exposed dams were sacrificed and their fetal brains were assessed for architectural pattern of neurocortical layers, neuronal migrations and neuronal apoptosis (Bax, Bcl-2, Annexin-5 kit assay, EM & RT-PCR), and their lasting impact on neurodevelopment and genesis of psychiatric disorders in young offspring. This laboratory revealed that therapeutically relevant doses of atypical psychotropic drugs may induce default neural migration, altered neuroarchitectural pattern; enhanced apoptotic neurodegeneration in different neuronal layers of neocortex and hippocampus of fetal brain and overt expression of anxiety, depression and cognition (learning and memory) like psychiatric disorders in young offspring. The neurobiology and genesis of these psychiatric disorders is associated with confounding factors (intrinsic and extrinsic). This study concludes that prenatal exposure to atypical psychotropic agents may induce abnormal fetal brain development and neurobehavioral sequelae in young offspring, therefore precautions should be taken by the health care providers before prescribing these agents to pregnant population.

Recent Publications

1. Singh S K, Hidau M K, Gautam S, Gupta K, Singh K P, Singh S K and Singh S (2017) Glycol chitosan functionalized asenapine nanostructured lipid carriers for targeted brain delivery: pharmacokinetic and teratogenic assessment. International Journal of Biological Macromolecules, 108:1092-1100.
2. Singh K P and Singh M K (2017) *In utero* exposure to atypical antipsychotic drug, risperidone: effects on fetal neurotoxicity in hippocampal region and cognitive impairment in rat offspring. Progress in Neuro-Psychopharmacology and Biological Psychiatry 75:35-44.
3. Singh K P, Singh M K and Gautam S (2016) Effect of *in utero* exposure to the atypical anti-psychotic risperidone on histopathological features of the rat placenta. International Journal of Experimental Pathology 97(2):125-132.
4. Singh K P, Singh M K and Singh M (2016) Effects of prenatal exposure to antipsychotic risperidone on developmental neurotoxicity, apoptotic neurodegeneration and neurobehavioral sequelae in rat offspring. International Journal of Developmental Neuroscience 52:13-23.
5. Singh K P and Tripathi N (2015) Prenatal exposure to a novel antipsychotic quetiapine: Impact on neuro-architecture, apoptotic neurodegeneration in fetal hippocampus and cognitive impairment in young rat. International Journal of Developmental Neuroscience 42:59-67.

Biography

K P Singh is a Professor in the Department of Zoology, University of Allahabad, India. He started his research on "Developmental neurotoxicity of CNS acting drugs on different aspects of teratology in general and neuroteratology in particular like neuroanatomy, pharmacology, neurochemistry and neurobehavior in rodent (Rat/Mice) model". At present, his laboratory is involved to investigate the effect of novel psychotropic (antiepileptic, antipsychotic, antidepressant) drug exposure during pregnancy and lactation on fetal/neonatal birth defects, neuropathological alterations in different regions of fetal brain viz, cerebral cortex, caudate putamen, hippocampus and cerebellum etc., neurodevelopmental delay in offspring as well as long-lasting impact on neurobehavioral impairment in young-adult offspring.

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