The side effects of developmental treatment with maternal antiepileptic drugs

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Most women with epilepsy require one or more antiepileptic drugs (AEDs) throughout their entire pregnancy to control seizures. Long-term use of AEDs in epileptic mothers can elicit lasting side e ects in their children. e mechanisms of anatomical and behavioral teratogenesis may well di er, because it appears that the highest risk of anatomical defects i from rst-trimester, whereas the highest risk of behavioral defects appears to be from exposure during the third trimester. Proposed mechanisms underlying teratogenicity of AEDs include impaired folate, ischemia-hypoxia, neuronal suppression, reactive intermediates and AED-induced neuronal apoptosis. Disturbed folate metabolism during administration of AEDs, reduces neurogenesis, increases apoptosis. AEDs inhibit S-adenosyl methionine and dihydro folic acid reductase, so associat with disturbed folate metabolism and increases plasma homocysteine levels. AEDs depressed synthesis of the neurotrophin BDNF and NT-3 and reduced levels of the active phosphorylated forms of c-RAF, ERK1/2 and AKT. Suppression of synaptic neurotransmission is the common denominator in the action of AEDs, via block voltage-gated sodium channels enhance

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