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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 effects in their children. The mechanisms of anatomical and behavioral teratogenesis may well differ, because it appears that the highest risk of anatomical defects is from first-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 associated with disturbed folate metabolism and increases plasma homocysteine levels. AEDs depressed synthesis of the neurotrophins 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 GABAergic inhibition, or block glutamate-mediated excitation, therefore major AEDs cause, sensitive neurons and apoptotic neuro-degeneration in the developing brain. Prostaglandin H synthase enzyme and lipoxygenase enzyme in the fetus are active, so they can convert AEDs to free radicals and cause anatomical defects. Findings suggest, the functional consequences of *in utero* AED exposure depend upon the type, dose and timing of treatment with AED that can induce long-lasting cognitive, behavioral and anatomical impairments and certain caution must be taken when prescribing these medications to pregnant or breastfeeding mothers.

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