Teratogens



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Medications

ACE-Inhibitors: Kidney Damage
Ace-card-inhibited and injured-kidney
ACE inhibitors when used after the first trimester may cause decreased urine production in the fetus as a result of kidney injury. This can lead to several of the sequelae associated with poor fetal urine production, such as oligohydramnios, pulmonary hypoplasia, and intrauterine growth restriction.

Aminoglycosides: Ototoxicity
Amino-amigo-glider with toxic-green ears
Aminoglycoside use during pregnancy has been documented to be associated with ototoxicity in infants. This mechanism is thought to be mediated by damage to hair cells as well as mitochondrial DNA mutations that result in subsequent hearing loss in the child.

Lithium: Ebstein Anomaly
Lithium battery and egg-stein
When taken during pregnancy, lithium can cause Ebstein anomaly. Ebstein anomaly is a congenital heart malformation characterized by malformed and displaced tricuspid valves. This leads to tricuspid regurgitation and enlargement of the right heart. Ebstein anomaly may also be associated with other cardiac defects such as septal defects and patent foramen ovale.

Folate Antagonists: Neural Tube Defects
Ant-foliage and withered neuron-tube
Any medications that decrease folate or interfere in its metabolism can cause neural tube defects. These drugs include methotrexate, trimethoprim, and certain anti-epileptric drugs.

Substance Abuse

Cocaine: Low Birth Weight
Cocaine on down-arrow-scale
Cocaine is a cause of low birth weight. While the exact mechanism is not defined, a contributing factor may be the vasoconstriction and subsequent reduced blood flow to the fetus associated with cocaine use.

Cocaine: Placental Abruption
Cocaine and placenta-present-ruptured
Acute cocaine intoxication is known to be associated with elevated blood pressure leading to placental abruption. Placental abruption refers to complete or partial separation of the placenta from the uterine wall prior to delivery of the fetus and is a cause of significant maternal morbidity and fetal mortality.

Other

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Vitamin A Excess: Spontaneous Abortion

Up-arrow-viking-apple and spartan-aborting baby

Excess vitamin A has been linked to spontaneous abortion. Though the exact mechanism is unknown, it likely involves disruption of the normal cellular differentiation process that in the fetus is highly active and is largely dependent on appropriate concentrations of vitamin A for the process to take place correctly.

Vitamin A Excess: Cleft Palate

Viking-apple and cleft-plate

Vitamin A excess during pregnancy has been shown to be linked to higher rates of cleft lip and cleft palate. Vitamin A plays an important role in regulating embryonic development, therefore excess can cause dysfunction in this regulation process, leading to malformations such as cleft palate by affecting epithelial differentiation and apoptosis and preventing tissue development.

X-Rays: Intellectual Disability

X-ray of baby holding Tar Covered Book

Ionizing radiation has been associated with defects in development of the fetal central nervous system, leading to cases of intellectual disability. This is generally associated with exposure to doses over 10-20 rads with exposure between weeks 8 and 15 of gestation.

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