Example of research paper on long term effects of teratogenic abuse on child deve...

Health & Medicine, Addiction

Teratogens are genotoxins that can potentially disrupt the genetic makeup of developing cells, resulting in aberrations that can manifest disabling physical and mental abnormalities in a fetus. Teratogens can cross the placenta and reach the fetus, which quashes the popular belief that a fetus is shielded from external harmful influences. This indicates that the placenta is incapable of blocking teratogenic metabolites from reaching the fetus. Teratogenesis can be defined as birth defects or congenital malformations occurring in utero due to exposure to external factors. Such factors could be environmental, chemical or biological in nature. Factors such as the time of exposure, duration of exposure, dosage, number of exposures and genetic susceptibility of the individual to a potential teratogen, determine the likelihood of a teratogenic effect (Maret, 2009).

In the USA, infant mortality has been associated with teratogen exposure in 20% of the cases. Teratogen associated-congenital malformations account for 3 to 5% of the births in the US. It appears that teratogenic abuse or accidental exposure to teratogens at adverse level dosage disrupts specific enzyme functioning, blood vessel development, folate production, endocrine functions, neural crest development and causes oxidative stress (Sen, Shishoo & Lahiri, 2011).

Prenatal substance abuse of opiates, narcotics, nicotine and other drugs by pregnant women has been linked to both mental and physical abnormalities in the fetus. Behavioral teratogeny is one of the consistent consequences observed in infants who were prenatally exposed to substance abuse drugs and alcohol. Long term effects are seen in growth, achievement, behavior, cognitive functions and language learning (Behnke & Smith, 2013). The

rationale behind this assumption is that the teratogens could damage the central nervous system (CNS) in utero, which in turn could produce behavioral teratogenesis instead of physical malformation (Minnes, Lang & Singer, 2011). Pregnant women with a history of substance abuse would typically abuse a combination of multiple illicit drugs; such an abuse could lead to cumulative teratogenic effect on the fetus.

This paper aims at analyzing the long term effects of teratogen abuse on the psychological development of a child. The paper also explores the possibility of the contribution of paternal teratogenic abuse to the child's psychological development.

Types of teratogens and their long-term effects on a child's development

Legal and illicit drugs make up the largest category of teratogens that are prone to abuse. The US Food and Drug Administration (FDA) has divided the prescription and non-prescription drugs into five categories, namely, A, B, C, D and X, based on their risk to the fetus. Categories A and B drugs are those that have demonstrated no risk to the human fetus. Categories C and D drugs are those that might result in teratogenic effects. Category X drugs are those that have been proven to cause fetal abnormalities at all dosage levels. Some of the substance abuse drugs fall under this category. Potential behavioral teratogenic effects of such drugs have been discussed below.

Substance abuse drugs

Amphetamines. Amphetamines are a popular first-line stimulants used for managing ADHD. Methamphetamine is an illegal form of the drug that has a

high abuse potential. Prenatal abuse of both of these drugs has been associated with sudden infantile death syndrome (SIDS), 3. 5 times increased likelihood of a low birth weight, size and presentation of withdrawal symptoms (Minnes, Lang & Singer, 2011). The long-term effects might include breathing trouble, diminished learning capability and problems associated with visions and hearing (Maret, 2009).

Cocaine. Cocaine is a narcotic drug that boomed in abuse during the late 1980s and early 1990s. During that time, nearly 2 million infants were prenatally exposed to this drug. Infants who were born to cocaine abusing mothers were found to have a low head circumference, low birth weights and length. Long term effects of low non-verbal reasoning scores, poor muscular control, low Bayley Scales of Infant Development (BSID) scores, delay in cognitive development, attention deficit, aggressive behavior, inability to self-regulate, low visual processing and impaired visual memory have been attributed to prenatal cocaine exposure (Maret, 2009). In a study 12% of the exposed infants presented ADHD as toddlers and 17% of the exposed infants exhibited oppositional defiant disorder. Brain imaging studies have recorded a retarded brain development in cocaine-exposed infants during the toddler years and adolescence (Minnes, Lang & Singer, 2011).

Opiates. Opiate (usually heroin) addiction and prenatal abuse are thought to have a greater impact when compared to cocaine. However, research has been inconclusive in this regard. Nevertheless, research has proven that opiate exposure prenatally could cause ADHD, delay in cognitive development, aggressive behavior and neonatal abstinence syndrome (NAS). Methadone is used for addiction management in pregnant women; however,

this therapy has not been approved by FDA due to lack of evidence regarding the impact and efficacy of methadone on fetuses (Minnes, Lang & Singer, 2011).

Marijuana. The researches on the teratogenic effects of marijuana have been inconclusive. Cognitive impairment has not been clearly established as a consequence of prenatal marijuana due to fewer studies (Maret, 2009); however, there have been some consistent pattern of behavioral teratogenesis in infants, toddlers and adolescents whose mothers smoked marijuana more than five times a week during pregnancy. Though IQ does not seem to be affected in prenatal-marijuana exposed infants, their ability to solve problems, higher level thinking, planning and memory are below average when compared to control groups (Behnke & Smith, 2013).

Tobacco. Nicotine is one of the active substances in a cigarette that has abuse potential right after a single use. A cigarette also contains potential carcinogens that are harmful to the smoker in the long run. It is an established fact that tobacco chewing and smoking directly correspond to low birth weight, smaller head circumference, premature delivery, still birth, SIDS, low IQ, attention deficit, higher muscle tension, diminished capacity to self-regulate, etc. (Behnke & Smith, 2013). Tobacco is thought to disrupt the enzyme monoamine oxidase (MAO) that is imperative for fore brain development in utero, which in turn is thought to be responsible for disruptive behavior in adolescence, depression and anxiety. Imaging studies have shown that prenatal exposure to tobacco results in reduced cortical grey matter. Tobacco exposure during infancy is also thought to hasten puberty in males (Minnes, Lang & Singer, 2011).

Alcohol

Next to tobacco, prenatal alcohol exposure is one of the most extensively studied subjects. Unlike substance abuse drugs, even moderate recreational alcohol drinking during the early weeks of pregnancy has been proven to affect the fetus. Alcohol abuse during early pregnancy is associated with miscarriage, while continued abuse could result in fetal alcohol effect (moderate drinking), fetal alcohol syndrome (severe drinking), impaired executive functions, impaired learning, underachievement, impaired cognition and disruptive-aggressive behavior (Behnke & Smith, 2013).

Paternal contribution during preconception exposure

A recent study has established that paternal preconception exposure to substance abuse drugs and alcohol also contributes to behavioral teratogenesis and genetic vulnerability. In the study, it was observed that children born to non-alcoholic mothers and chronic alcoholic fathers exhibited fetal alcohol syndrome, underachievement in studies and attention deficit. Paternal smoking has found to affect the quality of the sperm, and therefore, indirectly contribute to genetic vulnerability. Paternal marijuana abuse has not been documented or studied well. Studies conducted in the late 1990s suggested that paternal marijuana smoking could lead to congenital heart defect. Cocaine abuse in males results in accumulation of cocaine in the testes. Thus, it is possible that a cocaine-bound sperm could fertilize a healthy ovum, thereby introducing the cocaine into the ovum. This possibility suggests that cocaine addiction could be heritable via paternal contribution (Vassoler, Byrnes & Pierce, 2014).

Identification of prenatal exposure

Prenatal drug exposure could be identified via self-reporting by the parent or could be deduced from body fluids sampling. Self-reporting is cheaper and more reliable as the parent would be forthcoming with the necessary information to prevent extensive damage. However, a major drawback in this method would be the accuracy of the information regarding dosage and duration. Detection using biological samples is expensive due lack of specific substance abuse drug tests. Immunoassay is a non-specific test that could be modified for low-exposure or high-exposure detection; gas chromatography-mass spectrometry is required to confirm the results (Behnke & Smith, 2013).

Conclusion

A fetus is susceptible to teratogens such as chemicals, environmental factors, therapeutic drugs and infectious diseases. The teratogenic effect could be physiological, phenotypic or behavioral. Exposure to substance abuse drugs has found to contribute to behavioral teratogeny. Imaging studies of the brains of such prenatally exposed children have now confirmed that the psychological impairments are a direct consequence of the exposure due to maternal and, to some extent, paternal teratogen abuse. Prenatal exposure to alcohol, marijuana, cocaine, opiates, amphetamine and tobacco result in similar behavioral teratogeny, such as underachievement, ADHD, lack of self-regulation, aggressive behavior, tendency for substance abuse during adolescence, etc., which indicate that

teratogenic abuse during pregnancy, preconception and prenatal exposure result in long term detrimental psychological effects in a child.

References

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