Neurobehavioural Effects of Prenatal Exposure to Alcohol

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Abstract

Neurodevelopmental disabilities seem to be increasing in frequency and industrial chemicals (lead, methylmercury, polychlorinated biphenyls, arsenic, toluene, fluoride, chlorpyrifos, dichlorodiphenyltrichloroethane, tetrachloroethylene, polybrominateddiphenil ethers) that injure the developing brain are among the known causes for this rise in prevalence. These substances have been identified as developmental neurotoxicants. Grandjean and Landrigan postulated that even more neurotoxicants remain undiscovered and proposed a global prevention strategy. (1) However, we must consider that the intimate prenatal environment is maternal uterus. So, not only maternal environmental chemicals but also maternal prenatal exposure or consumption of substances of abuse (nicotine, alcohol and drugs of abuse) that must be considered as developmental toxicants. Prenatal exposure to alcohol is the most prevalent and unrecognized cause of neurobehavioural deleterious effects around the world. (2) The proposed global prevention strategies must include to avoid the consumption of these substances of abuse during pregnancy.

Rapid Communication

There is a growing interest in the scientific community to assess the impact of environmental pollutants on the process of fetal development [1]. However, the research has been focused on substances whose exposure is usually not preventable at small scale, so the capacity to affect change through primary prevention measures is limited and probably infeasible. Other highly neurotoxic substances such as alcohol, which is entirely preventable through abstinence during all the pregnancy and represents a high burden of disease leading to the most recurrent cause of acquired development disabilities in newborns, [2] remain in oblivion. In fact, with the information at our disposal so far, alcohol consumption should be considered as the most injurious and hazardous agent of a not well recognized pandemic of neurodevelopmental toxicity.

The deleterious effects caused by prenatal exposure to ethanol are related to variables as dose, time, duration and pattern of alcohol consumption. However, in many cases, the foetus is exposed to the teratogenic effects of ethanol during the critical period of organogenesis, before pregnancy is confirmed [3]. Newborns exposed to maternal alcohol during pregnancy can develop a spectrum of characteristic facial features, impaired neurodevelopment, cognitive and behavioural disabilities, and fetal growth restriction known as fetal alcohol spectrum disorder (FASD), with the most severe form, including specific morphological facial abnormalities, defined as fetal alcohol syndrome (FAS) [3].

Maternal consumption of alcohol during pregnancy, even in very small quantities, has been linked to a range of neurobehavioural adverse effects in offspring, including reduced IQ, impaired executive function and social judgment, delinquent behavior, seizures, other neurological signs, and sensory problems [4,5].

The true rate of prenatal alcohol consumption in different countries using reliable tools of estimation is unknown, but an estimated worldwide 30% (60% in certain countries) of pregnant women consume alcohol [2,6]. This prevalence is difficult to determine, since in many cases pregnant women refuse or under-report the alcohol consumption during pregnancy, most of them not considering drinking as being dangerous [7]. In this sense, objective biomarkers determined in biological matrices, as meconium or maternal hair, must be used in order to assess prenatal exposure [8,9].

In addition, it is of utmost importance taking into consideration that maternal uterus is the major window of developmental vulnerability and the whole fetal environment during prenatal development. Thus, its proper organogenesis is susceptible to be compromised by any subtle increase in the concentration of a substance capable to cross the placental barrier [2,5]. Moreover, there is a huge lack of awareness about alcohol consumption during pregnancy in general population, probably as a result of a deficient generation of complete abstinence-based recommendations by health professionals.

Although it is widely accepted that exposure to high doses of ethanol has longlasting detrimental effects on brain development, the case for moderate exposure remains controversial. It is not accurate to state that moderate or light drinking does not appear to be associated with adverse mental health consequences. Moreover, the amount of alcohol that reaches the foetus depends not only on timing and volume of consumed alcohol, but also on genetic or epigenetic factors and on placental passage [7,10]. There is no 100% assurance that there is no risk at all; so, it is mandatory to recommend that women not drink alcohol during pregnancy [2].

Lack of evidence is not the same as evidence of absence of risk and, in this case, no evidence of harm does not mean evidence of no harm; subsequently, no amount of alcohol during pregnancy can be considered safe based on research evidence [2].

The collective evidence from the animal studies suggests that moderate or low prenatal alcohol exposure can persistently alter multiple neurotransmitter and neuromodulatory systems throughout the brain, leading to significant neurobehavioral alterations in the offspring [11].

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Although brain damage caused by fetal exposure to ethanol cannot be reversed, early action is of extreme importance since appropriate follow-up programs for affected newborns may prevent the development of secondary disabilities and help lead to the best possible neurodevelopmental outcome [12]. To achieve this, we must emphasize the necessity of generating recommendations that provide clear and consistent information about the effects of alcohol consumption on the developing baby [2,12]. Additionally, it is important to ensure ongoing education for health professionals on the issue of prenatal alcohol consumption. The preventive message must be very clear: no amount of alcohol is safe during pregnancy.

References