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Polycyclic Aromatic Hydrocarbons and Birth Outcomes - A Systematic Review

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Abstract—*Polycyclic Aromatic Hydrocarbons (PAHs) are of special interest due to their ubiquitous presence in the environment and carcinogenic property, also samples from population throughout the world have been found to contain detectable concentrations of these compounds. In the light of many significant studies PAHs have long been recognized for their potential to a) Cause health effects including carcinogenic and mutagenic effects, b) stored in human tissues like blood, placenta, etc., c) cross placental barriers, d) alter trophoblast proliferation in placenta, in addition to disturbing its endocrine functions, e) increase the risk of adverse birth outcomes. Considering their toxic potential, it is essential to understand the health risks from PAHs in different sources with proximity to humans for effective environmental management. In conclusion, the association between adverse birth outcomes and exposure to air pollutants like PAHs has recently become a major concern. This review summarizes the available information of Polycyclic aromatic hydrocarbons, its properties, fates, major sources of human exposure, and their effect on delivery outcomes. Mainly the effect of PAHs exposure on birth outcomes like preterm birth and low birth weight are stressed.*

Keywords*: Polycyclic Aromatic Hydrocarbons, human exposure, delivery outcomes, preterm births.*

1. INTRODUCTION

There is convincing evidences showing that exposures to one of the environmental pollutants, Polycyclic Aromatic Hydrocarbon (PAHs), either as indoor pollutant or as outdoor pollutant can show adverse birth outcomes. PAHs are released into the air as a result of incomplete combustion of organic materials such as fossil fuels, automobile emissions (diesel or gasoline), cooking (fuel burning) and waste refuse incineration , cigarettes and tobacco smoking, ingesting smoked/grilled foods. In the light of above sources PAHs shows toxic effect to human through varies routes like breathing polluted air , eating contaminated food, coming in contact with air, water or soil , near hazardous waste sites, etc.(Newsletter 2003)

Certain PAH compounds are known to be toxic, mutagenic and/or carcinogenic to mammals [1]**.** It has been proved that PAHs can cause carcinogenic and mutagenic effects and are potent immune-suppressants [2]. They are reported to show their adverse effects by altering the trophoblast proliferation in placenta, in addition to disturbing its endocrine functions, which may be able to increase the risk of preterm delivery in pregnant women. The developing fetus and neonates are particularly at risk because they are known to be more susceptible than adults to the toxicological consequences of indoor environmental pollution, including polycyclic aromatic hydrocarbons [3,4,]

2. SOURCES

Starting from Indoor PAHs exposure, the combustion of household fuels is the major source of PAHs. Emission of PAHs vary with the use of biomass fuel like cow dung, firewood, coal, LPG, kerosene, etc. WHO (2016) guidelines states that around 3 billion people use burning biomass(wood, animal dung and crop waste) and coal for cooking and heat their homes using open fires and due to their incomplete combustion PAHs are released along with other pollutants contributing in high levels of indoor air pollution. Thus the use of traditional cook stoves commonly known as chulla using wood and other biomass fuel obviously effect not only the women who do the cooking but also young children and infants who spend most of the time with their mothers. Many studies have shown the association of various diseases (pneumonia, tuberculosis, lung cancer, preterm and low birth weight) with household air pollution due to the use of biomass fuels [5]. The Regional Wood Energy Development Programme (1997) have illustrated that the hazards of kitchens continue to affect many millions of people largely in the form of polluted smoke from improper cooking devices used in rural poorly ventilated areas. C.V. Raiyani et al 1993 have observed the findings from the five groups of houses each using either cattle dung, wood , coal, kerosene or liquid petroleum gas(control) as cooking fuel and revealed that air quality of biomass using houses was worst [6]. According to WHO guideline 2010 the concentrations of PAHs using different types of cooking fuel, are in the order LPG < kerosene < coal < wood < dung cake/wood mixture < dung cake .Women who uses indoor firewood combustion are at

high risk of PAHs exposures than women who use outdoor firewood combustion [7].

World Health Organization (WHO) estimates that pollution levels in rural Indian kitchens are 30 times higher than recommended levels and six times higher than air pollution levels found in the national capital. Rural women using more of the traditional biomass fuel cook stove are more at risk to indoor air pollution and exposure to PAHs . In Agra rural rates of low birth weight (17.6%), prematurity (16%), birth asphyxia (13.5%), hypothermia (12.6%) and pneumonia (12.6%) emerged as chief medical causes of neonatal deaths [8].

Secondly the outdoor pollution through PAHs mainly includes the industrial sources and automobile exhausts. Human exposures to PAHs can be at high risk in urban areas due to more dense population and having drastic rate of industrial (aluminum production, iron and steel foundries, coke production, etc) spread, accompanied by increase in the number of automobiles following massive rate of motor vehicle emissions (especially diesel vehicles) making a considerable contribution to PAHs concentration in air due to burning and incomplete combustion of diesel or gasoline. Rajput et al 2009 has shown PAHs concentration of 269 ng m^{-3} in the urban area of Agra regions[9].

3. OTHER SOURCES :

Gullaya Wattayakorn (2003) have shown the pollution loads especially of PAH in contaminated Chao Phraya river , due to having some industrial sites and urban areas nearby [10]. Bhupander Kumar et. al. 2014 studied the PAHs concentrations in street soil of residential areas of Ghaziabad, India, a part of National Capital Region (NCR) which is a developing industrial city, and found the concentrations of ∑PAHs ranged 36 – 898 µg kg-1[11].

Lastly human dietary habits can also be another cause of PAHs exposure, which includes the thermal treatment of food, especially grilling or barbecuing. Many studies have shown the exposures to PAHs from diet [12,13]

Thus the sources of PAHs are ample and we humans are at high risk to its exposures that cannot be ingnored. Polycyclic aromatic hydrocarbons and its consequences constitute a major health problem.

4. BIRTH OUTCOMES

Prenatal exposure to PAHs has been associated with adverse birth outcomes like preterm deliveries and reduction in birth weight. Preterm defines as neonates born at less than 37 weeks gestational age. According to World Health Organization (WHO) every year, an estimated 15 million babies are born preterm and over 1 million babies die annually from preterm birth complications. Thus Preterm delivery (PTD) remains the leading cause of prenatal mortality and occurs in approximately 4–10% of pregnancies [14,15]. Known cause coupled with preterm birth and low birth weight are therefore serious threats having risk factors due to socioeconomic status, ethnicity, smoking, drug abuse, and environmental contaminants [16]. In the overall pregnancy period women are exposed to wide variety of chemicals including PAHs through their life style which include tobacco smoking, drug abuse and also due to outdoor and indoor (poor ventilated) environmental pollution. [17,18]. The association between preterm delivery (PTD) and exposure to air pollutants has recently become a major concern. Previous studies have already reported that environmental levels of PAHs in India are much higher than the standard limits prescribed by government regulatory agencies [19] which increases the chances of getting exposed to high PAHs through environment. As there is no barrier to prevent inward movement of these chemicals and can be easily transported from mother to fetus via placenta by simple diffusion.The human placenta, a fetomaternal organ, consist of both fetal (amnion and chorion) and maternal (decidua) tissues [20] and as previously stated, many studies have reported the ability of PAH to cross the placenta by variety of mechanisms and even have evidence showing that PAHs are present in human placenta and their concentrations in cord blood are often higher than in maternal blood [21]. Placental studies can be done to observe the effects of exposure as it provides a large sample for analysis and is the most accessible and readily available component of the triad mother-infant-placenta. An Indian study [22] reported placental PAHs levels (viz. chrysene, benzo(a)pyrene and benz(a,-h)anthracene concentrations) of healthy non-smoking pregnant women from the general population.

Five PAH compound were detected by Iman Al-Saleh et al (2013), benzo(*a*)anthracene (B*a*A), chrysene (C*h*), benzo(*b*)fluoranthene (B*b*F), benzo(*a*)pyrene (B*a*P), and dibenzo(*a*,*h*)anthracene (DB*a*hA) in placentas and maternal and umbilical cord blood and assessed their influence on various anthropometric measures of birth outcomes suggesting an inverse relation between placental weight, cord length and Benzo(a)pyrene(24). Many studies have concluded that the risk of delivering a growth-retarded infant increases with the level of PAHs in early gestation [23]. Jan Dejmek (2000) experimented that , carcinogenic PAHs may directly modulate the proliferation of the trophoblast due to their reaction with receptors for placental growth factors [24]. In this way the feto-placental exchange of oxygen and nutrients may be reduced and fetal growth can be impaired. Huel G et al (1993) examined the possible impact of PAH exposure on the normal course of the pregnancy using induction of aryl hydrocarbon hydroxylase (AHH) enzyme as an indicator of PAHs exposure which can cause preterm delivery in the normal course of pregnancy [25]. A case control study in city of Lucknow has been performed by Singh et al 2008 and approached with significantly higher level of fluoranthene and benzo(*b*)fluoranthene in pre-term deliveries [26]. Different

concentrations of PAHs in varied biological samples worldwide is shown in table below.

Table 1: Concentration of PAHs in different biological matrixes globally

City/Country	No. of Sample	Matrix	PAHs	References
Texas USA	35	Maternal Blood	7.4 ng/ml	Ken Sexton et al, 2011
Texas USA	35	Cord Blood	14.1 ng/ml	Ken Sexton et al, 2011
Lucknow India	60	Placenta	1590.79 ppb	Singh et al., 2008
Riyadh Saudi Arabia	1543	Placenta	32.016 ppb	Iman., 2013
Beijing China	3254	Placenta	8.53 ppb	Yanxin et al., 2011
Chennai India	24	Placenta	240 ppb	Madhavan et al., 1995

Thus the above article gives a brief review of the status of PAHs in the environment and in the biological samples and also the results of some biomonitoring studies showing some significant association between polycyclic aromatic hydrocarbons and adverse birth outcomes. Focussing on the women health it reviles that the use of the biomass fuels-implies serious health hazards .

5. RECOMMENDATIONS:

In the light of above information, it can be recommended that focus should be made to improve the methods for identification of women at risk of pretem birth. The regulatory agencies including few nongovernmental organizations, companies, development and public health agencies should be promoted to adopt the measures to replace these traditional stoves by focusing on stove replacement programmes. It would definitely be beneficial to some extent if more of the assessment survey, training and workshops can be conducted regarding household fuel, stove, exposure to pollutants (PAHs) and threatened risk to pregnancy especially in the rural areas. A quality standard should be defined to the full strength of the providers who treat women giving preterm births & infants born preterm.

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