Editorial

Environmental Exposures and Reproductive Health

Chan Tian[#]

In recent years, the impacts of climate change and air pollution on human reproductive health have emerged as an important issue in global public health. Broadly, reproductive health is not confined to any single stage between conception and pregnancy; it encompasses a continuum that includes gametogenesis, embryonic development, maintenance of placental function, fetal growth, and birth. Adverse environmental exposures may affect any stage of early life. Accordingly, central questions in this field have shifted from whether an exposure is harmful to a more refined understanding of when it acts, during which window, in which susceptible population, and with what consequences. Recent studies published in Biomedical and Environmental Sciences addressed, respectively, the effects of $PM_{2.5}$ on oocyte development during assisted reproduction and the association between prenatal exposure to high ambient temperatures and heatwaves and the risk of neonatal necrotizing enterocolitis (NEC). These studies extend environmental reproductive health research toward earlier and more finely resolved stages of the life course. In addition, profiling neonatal hair metabolites has provided evidence linking early metabolic signatures and developed prediction models to adverse neurobehavioral outcomes in childhood.

Identifying Critical Exposure Windows

Oocyte development involves multiple stages, including follicular recruitment, growth, and maturation. The impact of environmental pollutants on oocyte quality may therefore not be restricted to the final stage of maturation, but may accumulate across earlier phases of follicular development. To more precisely identify the critical periods during which $PM_{2.5}$ exposure may affect oocyte development, the investigators defined exposure windows of 0–3 months, 4–12 months, and 0–12 months before oocyte retrieval. The study showed that higher $PM_{2.5}$ and component exposures were associated with poorer oocyte-related outcomes, with stronger effects observed for distal exposure (4–12 months). It further identified one month and 6–11 months before oocyte retrieval as sensitive exposure windows requiring particular attention[1]. These findings refine our understanding of how air pollutants affect reproductive outcomes and suggest that environmental risk management in assisted reproduction should begin well before the treatment cycle.

In the study on prenatal high ambient temperature and heatwave exposure and NEC risk, the investigators likewise highlighted the importance of exposure timing. Prenatal exposure to high ambient temperature and heatwaves was associated with an increased risk of NEC, with the first and second trimesters appearing to be more sensitive windows[2]. This finding shifts NEC risk assessment further upstream, from traditional postnatal factors such as feeding, infection, hypoxia, and the gut microbiota to the intrauterine environment.

Uncovering High-risk Components

$PM_{2.5}$ is a complex mixture composed of multiple constituents, including sulfate, nitrate, ammonium, organic matter, and black carbon. Recent research has gradually shifted from evaluating the overall effects of $PM_{2.5}$ mass to disentangling the effects of specific components. In 51,122 assisted reproductive technology (ART) cycles, the investigators assessed associations between $PM_{2.5}$ mass and its major components and total oocyte yield, mature oocyte yield, and the number of normally fertilized oocytes, and identified SO_4^{2-} and NH_4^+ as potential key contributors to impaired oocyte development[1]. Although dominant components may vary across regions and pollution sources, these findings indicate that reproductive risk assessment should move beyond $PM_{2.5}$ mass alone and consider the adverse effects of specific components, supporting more targeted prevention and control strategies.

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Center for Reproductive Medicine, Peking University Third Hospital, Beijing 100191, China

Recognizing Susceptible Populations

Previous studies have suggested that heat exposure is associated with adverse perinatal outcomes, including preterm birth, low birth weight, fetal growth restriction, placental abruption, and premature rupture of membranes[3]. The study in this issue further strengthens this evidence base[2]. It showed that prenatal exposure to high ambient temperature and heatwaves was associated with an increased risk of NEC, and that preterm birth partially mediated the association between high ambient temperature exposure across the entire pregnancy and NEC. In the context of global warming and the increasing frequency of extreme weather events, these findings have important public health implications. For pregnant women in high-temperature areas, heat protection should be more fully integrated into perinatal care and strategies to promote maternal and infant health.

Predicting Adverse Outcomes

Early life, especially the intrauterine period, represents a critical window of developmental plasticity. Changes in the environment, including exposures and nutrition, may have long-term consequences for health after birth and even into adulthood by altering metabolism and epigenetic regulation. Thus, detecting biomarkers earlier and predicting adverse outcome risks are important components of reproductive health promotion. The study in this issue offers useful methodological insights in this regard[4]. Using neonatal hair, an accessible biological sample that reflects cumulative exposure and metabolic status over time, the investigators conducted neonatal hair metabolomic signatures of sFGR neonates in dichorionic-diamniotic (DCDA) twins and found downregulation of cysteine and methionine metabolism, etc., which was associated with lower problem-solving and personal-social abilities later in childhood. Neurodevelopmental abnormalities are difficult to identify in the prenatal and early stages after birth. The study provided a possible tool to predict these disorders.

#Correspondence should be addressed to Chan Tian, E-mail: tianchan@bjmu.edu.cn

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