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Perspective

Association between Dialkyl Phosphate Urinary Metabolites and Chromosomal Abnormalities in Human Sperm

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DESCRIPTION

Recent research has increasingly focused on the impact of environmental endocrine disruptors on male reproductive health. Over 2 million couples each year face infertility and more than 2 million pregnancies are lost before the 20th week of gestation. A significant proportion of these losses, particularly those involving aneuploid conceptuses, are thought to result from pre-existing chromosomal abnormalities. Nearly 50% of all spontaneous abortions are linked to such abnormalities, many of which stem from errors in the father's sperm, particularly involving sex chromosomes (X and Y). These abnormalities arise when chromosomes fail to properly separate during cell division, a process known as non-disjunction. In germ cells, errors during meiosis I or II can lead to imbalances in chromosome numbers. Disomy, where an extra chromosome is present, is the most common form of aneuploidy observed in human sperm.

Children born with sex chromosomal abnormalities, such as those seen in Klinefelter and Turner syndromes, often face reproductive issues, behavioral challenges and intellectual disabilities compared to their siblings. Data from European birth defect registries suggest an increase in the prevalence of chromosomal abnormalities in infants between 1967 and 1988, with no corresponding rise in maternally-derived abnormalities, raising concerns about environmental factors affecting spermatogenesis. However, comparable data for the U.S. is lacking.

Pesticides, particularly Organophosphate (OP) insecticides, have raised concerns due to their potential as Endocrine-Disrupting Chemicals (EDCs). These chemicals can interfere with the endocrine system and cause adverse effects. Humans are shown to EDCs through various routes, including ingestion, skin absorption, inhalation and environmental contact (air, water, soil), with dietary intake being a major source. These chemicals were the first group of pesticides to be reviewed under the Food Quality Protection Act (FQPA) of 1996, and in 1999, the U.S. Environmental Protection Agency (EPA) identified a common mechanism of action for OPs, which involves binding to and phosphorylating the enzyme acetylcholinesterase in both the central and peripheral nervous systems. OP exposure has been linked to thyroid hormone disruption, decreased semen quality (including reduced sperm count, concentration and motility), abnormal sperm morphology, DNA fragmentation, and alterations in sperm chromatin structure. Despite these findings, the relationship between OPs and sperm abnormalities, particularly chromosomal issues like disomy, remains understudied.

Toxic environmental exposures may damage germ cell Deoxy Ribo Nucleic Acid (DNA) integrity, yet the precise causes of aneuploidy and the specific timing of exposures during the spermatogenic cycle remain poorly understood.

CONCLUSION

In conclusion, environmental endocrine disruptors, particularly organophosphate pesticides, may play a significant role in the increasing prevalence of chromosomal abnormalities in human sperm. The potential for these chemicals to interfere with spermatogenesis and DNA integrity raises concerns about their impact on male reproductive health. While studies have linked OP exposure to various sperm abnormalities, the precise mechanisms and timing of these effects remain unclear. Further research is essential to better understand how environmental toxins influence chromosomal disomy and to develop strategies to mitigate their impact on male fertility and reproductive outcomes. This study aimed to explore the association between environmental OP exposure and the frequency of disomy in adult men's sperm.

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