Perspective

Toxicology in reproduction

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Human reproductive disruption caused by xenobiotics, such as medications, occupational, and environmental exposures, has become an increasing concern over the last decade. For example, the cover story of a recent issue of US News and World Report (October 5, 1987) focused on infertility in the United States and indicated that some of the disease was caused by environmental contamination. At the moment, reproductive risk [1] is assessed by multiplying the lowest dose that has no effect on reproduction by a protection factor.

Statistical modeling of the "dose response curve" is being studied to establish an appropriate degree of excess population risk for the reproductive effect. The use of these methods to determine reproductive risk carries a lot of uncertainty, until a better formulation for characterizing the site and mechanism of action of reproductive toxicants across organisms is obtained, reproductive risk assessment using these techniques will be fraught with uncertainty.

Gametogenesis is the first step in the reproduction process, which continues through gamete interaction, implantation, embryonic development, growth, parturition, and sexual maturation. Reproductive processes, unfortunately, take place in a polluted area. To identify xenobiotics that are human reproducible, the process of hazard identification and characterization is necessary. While certain xenobiotics are considered to be harmful to the reproductive system, the vast majority have not been thoroughly tested for reproductive effects. Absorption, distribution, metabolism (toxification, and/or detoxification), excretion, and repair are all part of the biological pathways underlying reproductive toxicity. Over the next decade, reproductive toxicologists will face a difficult and important challenge in combining toxicokinetics and toxicodynamics with reproductive physiology through organisms.

Reproductive toxicology [2] varies by species and is affected by hormonal regulation systems, anatomy, pharmacokinetics, and metabolism. Because of variations in reproductive and/or toxicological processes, a reproductive toxicant in one species may not be toxic in another (including humans).

This complicates the assumptions that must be made in order to extrapolate reproductive effects from the data. The developmental toxicity of thalidomide is a good example; rats and mice are unaffected, but rabbits, humans, and non-human primates are affected.

Another important consideration in reproductive toxicology is gender disparities. Because of the variations in anatomy and biological control mechanisms for reproduction between men and women, this is a cause for concern. Xenobiotics [3] can be checked for toxicity to male reproductive processes more easily than female reproductive processes because access to gametes and gonads is so simple. Another significant feature of reproductive toxicity is the window of exposure to toxicity. This has been shown in studies of developmental toxicants and toxins that affect spermatogenesis. Toxins that affect the production of ovarian follicles.

Several studies have shown that substances like galactose or azathioprine are toxic to the developing ovary during pregnancy but have no effect afterward. The impact of age on gonadal sensitivity to chemotherapeutic agents or other xenobiotics have been studied in a limited number of experimental and clinical studies. Given the importance of reproduction for the survival of any species, it is critical to gain a better understanding of the mechanisms of action, sites of action, and species sensitivity to reproductive toxicants

REFERENCES

1. Kulier R, Campana A. Reproductive health research challenges. Reprod health. 2004;1(1):2.

2. Mattison DR, Thomfo PJ. The Mechanisms of Action of Reproductive Toxicants. Toxicol Pathol. 1989;17:364-376.

3. Gagne F. A Research Journal Dedicated to the Effects of Xenobiotics in OrganismsJ Xenobiot. 2020,10:11–13.

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