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Effect of lead exposure during gestation and lactation on developing ovary in Swiss mice

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ABSTRACT

Histopathological alterations in the ovaries were examined in the neonate from birth to 21 day of weaning on specific days *viz.*, 1, 7, 14 and 21 day of postnatal development. Lead acetate was administered orally at 530 mg/kg/bodyweight to pregnant Swiss mice from 10 day of gestation to 21 day of lactation. Studies conducted on females revealed mostly miscarriages, premature delivery and infant mortality. Lead suppresses the development of primordial follicles during fetal and neonatal life. Changed in the number of primordial follicles, primary and secondary follicles were also observed.

Key words : Lead acetate, Ovary, Organogenesis, Folliculogenesis

Many recent reports show that lead exposure continues to be a major public health problem worldwide (Dykeman *et al.*, 2002; Lidskey and Schneider, 2003; Huang and Schneider, 2004; Shalan *et al.*, 2005; Bellinger, 2006). The environmental contamination by lead generated from human activities has become an evident problem during the last decades (Kim *et al.*, 1996). Lead exposure is a leading environmental issue for children and women of child bearing age (Mushak, 1992). Lead can penetrate by inhalation, ingestion and skin absorption (El Feki *et al.*, 2000). Both in humans and experimental animals, lead readily crosses the placental -fetal barrier and its greater intestinal absorption in fetus results in toxic responses of exposed mothers, not only directly affecting the fertilized egg, it also includes all post fertilization toxicity relating to the developing offspring considered as developmental toxicology.

Lead is a strong teratogen which causes most of its congenital effect at the time of organogenesis during embryonic development. It creates specific defects during the period of organogenesis , shows a period of sensitivity corresponding to the development of the target structure. The ovarian follicle is the functional unit of the ovary. It contains the oocyte that may eventually ovulate, undergo fertilization and form an embryo. It also provides the steroid and protein hormones required for maintenance of the ovarian cycle, the secondary sex characteristics and preparation of the uterus for implantation (Findle *et al.*, 2009). The mammalian folliculogenesis is one of the most dynamic intricately regulated developmental processes in biology. In human, primordial follicle formation is initiated around 21 weeks of gestation (Bayne *et al.*, 2004; Pepe *et al.*, 2006) while in the mouse this process occurs shortly after birth. Histological examples of atretic primordial follicles in postnatal and adult ovaries are very limited (Perez *et al.*, 1999; Depalo *et al.*, 2003). The strain differences also account of major variations in the assessment of ovarian histology with regard to the types and number of follicles (Mayers *et al.*, 2004). Depletion of primordial follicles in the postnatal mouse ovary is well documented. In addition to recruitment into the growth phase, their diminishing number in part attributable to oocyte apoptosis (Peters, 1969) and also to follicular atresia (Pepling and Spradling, 2001; Johnson *et al.*, 2004).

The chelating agent removes the metals from the target tissues but they cannot be used during developing stages as this period is highly sensitive to these agents and chelators also have injurious side effect on development hence the treatment with antioxidants is more convenient. Female reproductive potential is limited in mammals with

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