

RESEARCH PAPER:

## Effects of maternal lead acetate exposure on prenatal development of swiss albino mice

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### SUMMARY

In the present study the teratogenic effects of lead acetate on prenatal development of mice were investigated. Females aged 9-10 weeks weighing 25-30 g. and showing vaginal plug were selected and divided into two groups. These groups were (i) control (ii) lead exposed groups at different days of gestation- (a) from the 1<sup>st</sup> day of gestation (dg) (b) from the 8<sup>th</sup> dg and (c) from the 10<sup>th</sup> dg. In all, these groups lead acetate was administered orally in the dose of 8mg, 16mg, and 32mg/animal/day. At the end of 17<sup>th</sup> dg, their uteri were excised out for the examination. Result showed that implantation rate was approximately nil in the uteri of females which were exposed from 1<sup>st</sup> dg. Dual results were obtained when exposure was began from 8<sup>th</sup> dg. Most of the female uteri showed only implantation site while some female uteri contained developed fetus but in very few number. The administration of lead on 10<sup>th</sup> dg did not affect the ability to conceive, to carry a normal litter. The percentage of malformed fetuses, resorptions were unaffected/ less affected on this day. Results suggest that exposure of lead acetate from 10<sup>th</sup> dg and there after, fetal toxicity (resorption) sharply declined. In conclusion, it is inferred that gestational lead exposure has an adverse effect on development, with an effect that may be most pronounced during the first trimester.

### Key words :

Lead, Mice,  
Fetus, Prenatal,  
Gestation, Uteri,  
Implantation

Environmental lead toxicity is an old but persistent public health problem throughout the world and children are more susceptible to lead than adults (Ahamed and Siddiqui, 2007). Lead poisoning among pregnant women is a significant public health problem, as it effects development (Katharina Weizsaecker, 2003). The development of a child begins in utero and continues following birth, thus, both of these time frames must be examined as possible periods of lead intoxication. During development, the fetus is at the mercy of its mother. If the mother has high blood lead levels during pregnancy, the developing fetus will have the same. This is due to the lack of a transplacental barrier to lead (Goyer *et al.*, 1990). Lead freely crosses the placenta consequently, gestational lead poisoning is not only harmful to the woman but also to the developing fetus (Shannon *et al.*, 2003).

The presence of lead in placenta indicates that lead moves from mother's blood erythrocytes in the intervillous space released and received by the villous syncytiotrophoblast. This finding enriches relation between mother's erythrocytes, lead, calcium that is a lead carrier and syncytiotrophoblast (Foltinova *et al.*, 2007).

Lead can be incorporated into the bone structure of the mother as a result of previous lead exposure, up to thirty years before in some cases. Thus, whenever, net bone resorption occurs to increase blood calcium levels, lead may also be released into the circulation. During gestation, there are two such periods. The first is in the first trimester when maternal blood volume increases, thus increasing the need for calcium to hold a constant concentration. The second is the third trimester when fetal ossification begins, thus increasing the fetal requirement for calcium (Silbergeld *et al.*, 1991). Both cases can result in higher lead concentrations in the fetal blood. Maternal exposure to lead is more important during fetal development than during breast feeding (Dorea and Donangelo, 2006). Early gestational exposure of lead slightly delays the development of the embryo and inhibits its implantation (Jacquet, 1976). Implantation is an intricately timed event necessary in the process of viviparous birth that allows mammals to nourish and protect their young during early development (Kevin and Franceso, 2004). Implantation is the process that leads from blastocyst attachment to its

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