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The pyrethroid insecticides permethrin and esfenvalerate do not disrupt testicular steroidogenesis in the rat fetus

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* Correspondence to: AM Saillenfait, Institut National de Recherche et de Sécurité, Rue du Morvan, CS, 60027, 54519 Vandœuvre Cedex, France. E-mail address: anne-marie.saillenfait@inrs.fr Highlights

- The insecticides permethrin or esfenvalerate were administered to pregnant rats.
- They had no effects on the fetal rat testis up to maternal toxic doses.
- mRNA levels of testicular testosterone biosynthetic enzymes were not impaired.
- Ex vivo fetal testicular testosterone production was not affected.
- Levels of their metabolite, 3-PBA, in the amniotic fluid increased with the dose.

ABSTRACT

The present study investigated the effects of maternal exposure to the widely used pyrethroid insecticides, permethrin and esfenvalerate, on fetal testicular steroidogenesis. Pregnant Sprague-Dawley rats were administered permethrin at doses of 1, 10, 50, or 100 mg/kg/day, or esfenvalerate at 0.1, 1, 7.5 or 15 mg/kg/day, by gavage, from gestation day (GD) 13 to 19. Testicular testosterone production and the expression of several key genes necessary for cholesterol and androgen synthesis and transport were assessed in GD 19 male fetuses. Dams treated with 100 mg/kg/day of permethrin or 15 mg/kg/day of esfenvalerate showed clinical signs of neurotoxicity. The highest dose of esfenvalerate also resulted in reduced maternal body weight gain throughout the treatment period. In the fetal testes, mRNA expressions of *HMG-CoA synthase* and *reductase*, *SR-B1*, *StAR*, *P450scc*, *3 β HSD*, *P450 17A1*, and *17 β HSD* were not affected by exposure to either pyrethroid. No significant change was observed in *ex vivo* testosterone production. In conclusion, *in utero* exposure to permethrin or esfenvalerate has no effect on the testosterone biosynthesis pathway in the fetal rat testis up to maternal toxic doses.

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