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Authors: Anne-Marie Saillenfait, Jean-Philippe Sabaté, Flavien Denis, Guillaume Antoine, Alain Robert, Alain-Claude Roudot, Dieynaba Ndiaye, Ethel Eljarrat



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Evaluation of the effects of α -cypermethrin on fetal rat testicular steroidogenesis

Anne-Marie Saillenfait ^{a*}, Jean-Philippe Sabaté ^a, Flavien Denis ^a, Guillaume Antoine ^a, Alain Robert ^a, Alain-Claude Roudot ^b, Dieynaba Ndiaye ^a, Ethel Eljarrat ^c

^a *Institut National de Recherche et de Sécurité, 1 rue du Morvan CS 60027, 54519 Vandoeuvre Cedex, France*

^b *Université de Bretagne Occidentale, UFR des Sciences et Techniques, 6 avenue Victor Le Gorgeau, CS 93837, 29238 Brest Cedex 3, France*

^c *Water and Soil Quality Research Group, Department of Environmental Chemistry, IDAEA-CSIC, Jordi Girona 18-26, 08034 Barcelona, Spain*

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

Highlights

- α -Cypermethrin had limited effects on the fetal rat testis up to maternal toxic doses.
- Testicular genes involved in steroid synthesis and transport were not disrupted.
- Ex vivo fetal testicular testosterone production was reduced at high doses.

ABSTRACT

Pregnant Sprague-Dawley rats were administered the insecticide α -cypermethrin at doses of 0.1, 1, 5, or 10 mg/kg/day, or di-isobutyl phthalate (DIBP) at 250 mg/kg/day, by gavage, from gestation day (GD) 13 to 19. Testicular testosterone production and the expression of several key genes related to cholesterol and androgen synthesis and transport were assessed in GD 19 male fetuses. Dams treated with 10 mg/kg/day of α -cypermethrin showed clinical signs of neurotoxicity and reduced body weight gain. α -Cypermethrin had no significant effect on post-implantation loss, fetal weight, incidence of male fetuses per litter, or anogenital distance of the male fetuses. In the fetal testes, mRNA expressions of *HMG-CoA synthase* and *reductase*, *SRBI*, *StAR*, *P450scc*, *3 β HSD*, *P450 17A1*, and *17 β HSD* were not affected by exposure to α -cypermethrin. Testosterone production by the fetal testis was significantly reduced at 5 and 10 mg/kg/day of α -cypermethrin, although to a much smaller extent than in DIBP-exposed fetuses.

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