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POSTCOITAL ANTIFERTILITY EFFECT AND MECHANISM OF ACTION  
OF PIPERINE IN RATS

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OF PIPERINE IN RATS

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ABSTRACT

Antifertility effects and mechanism of piperine action were investigated in pregnant rats. Administration of piperine in a dose of 25 mg/kg, twice a day, intraperitoneally and 100 mg/kg, twice a day, intragastrically at the pre-implantation period from day 2 through 5 inhibited implantation by  $52.2 \pm 8.6\%$  and  $58.3 \pm 12.1\%$ , respectively. The antifertility effect of piperine was more pronounced when it was given at the post-implantation period from day 7 through 10. With the same dose as in the pre-implantation period, it produced resorption in  $90.6 \pm 5.1\%$  for intraperitoneal and  $73.8 \pm 9.6\%$  for intragastric routes, respectively. Circulating level of progesterone started to decline after receiving piperine and was shown to be significantly lower than the control on day 6 in the pregnant rats which received piperine from day 2 through 5 and on day 10 in pregnant rats which received the drug from day 7 through 10 of gestation. Piperine did not have anti-gonadotrophic activity as determined from the lack of effect on the cyclic changes of vaginal epithelium and alteration of the weights of accessory sex organs in immature male rats.

Histological examination of the pregnant uterus showed that piperine caused a degeneration of implanted embryos as well as surrounding decidual cells. The depression of decidual growth in pseudopregnancy was

also observed. Changes in weight of the uterus and some biochemical parameters in decidual cells were measured as indices of decidual growth. Piperine significantly depressed the uterine weight and content of protein, DNA and glycogen in the traumatized horn of pseudopregnancy. All the actions of piperine, including anti-implantation, abortifacient and depression of decidual growth could not be reversed by supplemental administration of progesterone with piperine. These results suggested that the antifertility effect of piperine in pregnant rats was primarily due to the impairment of uterine cell function. Luteolysis of corpora lutea might accelerate the process of impairment.