

Contraception and Lactation Methods of Contraception

The benefits of breastfeeding for both the infant and the mother are undisputed. Lactation is an



effective contraceptive for the first 6 months postpartum only if women breastfeed exclusively and at regular intervals.

Each year, more than 100 million women worldwide make decisions about the use of a method of contraception after childbirth.^[1] These decisions include not only making a choice regarding a contraceptive method but also deciding the best time for initiation of the chosen method.^[2]

Hormonal Methods

There are some concerns about the use of hormonal contraceptive methods in lactating women. The American College of Obstetricians and Gynecologists' (ACOG) ^[7] and WHO^[8] recommendations for hormonal contraception in breastfeeding women are listed in table 1;

Natural Family Planning method- Natural family planning may be a contraceptive option for lactating women. Studies of cervical secretions in breastfeeding women indicate that mucus changes indicating fertility are reliable during lactation ^[3].

Intrauterine Devices

Non hormonal methods of contraception, such as or the copper intrauterine device (IUD), are the preferred choice for nursing mothers, because hormones in some contraceptive methods may interfere with lactation, and the transfer of hormones into milk poses a theoretical risk to the infant.^[2]

Tubal Ligation

If a tubal ligation is performed using general anesthesia, the mother should breastfeed just before the procedure to minimize the length of infant fast, with the following guidelines: 1) the mother should be alert; 2) short-acting benzodiazepines, muscle relaxants, inhalation agents, and local anesthetics are most likely safe, but meperidine or prolonged use of diazepam (Valium) should be avoided.^[4-6]

Special points of interest:

- Method of contraception must be safe for both mother and infant.
- 2-smoking may cause infertility for both males and females.

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ACOG	WHO
<ul style="list-style-type: none"> • Progestin-only oral contraceptives prescribed or dispensed at discharge from the hospital to be started 2-3 weeks postpartum (e.g., the first Sunday after the newborn is 2 weeks old). • Depot medroxyprogesterone acetate initiated at 6 weeks postpartum* • Hormonal implants inserted at 6 weeks postpartum* • The levonorgestrel intrauterine system can be inserted at 6 weeks postpartum. <p>Combined estrogen-progestin contraceptives, if prescribed, typically should not be started before 6 weeks postpartum, and only when lactation is well established and the infant's nutritional status is appropriate</p>	<ul style="list-style-type: none"> • Progestin-only methods of contraception (i.e., oral contraceptives, levonorgestrel-IUD, levonorgestrel implant, Depo-Provera injection) are not usually recommended before 6 weeks postpartum unless other more appropriate methods are not available or not acceptable. • Progestin-only methods can be used in any circumstances after 6 weeks postpartum. • Combined estrogen-progestin contraceptives (i.e., oral contraceptives, transdermal path, or vaginal ring) are not to be used before 6 weeks postpartum. • Combined estrogen-progestin contraceptives are not usually recommended between 6 weeks and 6 months postpartum unless other more appropriate methods are not available or not acceptable. • Combined estrogen-progestin contraceptives can be generally used after 6

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Endocrine Effects of Tobacco Smoking Gonadal and Reproductive Function

Males

Tobacco smoking in adulthood has a marginal impact on spermatogenesis.

Male fertility and spermatogenesis in adult men is surprisingly resistant to deleterious effects



of tobacco smoke. (Bonde & Storgaard, 2002). Other investigators found significantly lower sperm motility and percentage of morphologically normal spermatozoa, as well as altered sperm morphometric parameters and sperm function tests in smokers compared to nonsmokers (Sofikitis *et al.*, 1995). Furthermore, tobacco smoking might still be of importance if smoking during pregnancy has impact on the development of the foetal gonads.

Compared with nonsmokers and independent of relative weight and age, middle-aged male smokers have increased serum levels of DHEA, DHEAS, androstenedione, oestradiol, and SHBG, but normal total testosterone compared to nonsmokers (Attia *et al.*, 1989; Field *et al.*, 1994). Raised oestradiol may be among the mechanisms through which cigarette smoking impairs male reproduction. Of interest is the finding that smoking elevates oestradiol in men, while it lowers oestradiol in women. The finding of a lack of difference between smokers and nonsmokers in testosterone levels suggests that the steroidogenic function of the testis is not affected by smoking; on the other hand, smoking may affect the free fraction of testosterone (Attia *et al.*, 1989).

Females

Present evidence supports an adverse effect of smoking on ovarian function, which is prolonged and dose dependent (Shiverick & Salafia, 1999). Smoking may also alter fertility through effects on uterine–fallopian tube functions, which mediate gamete and conceptus transport (Shiverick & Salafia, 1999). Hughes and Brennan (1996) conducted a meta-analysis to assess the effects of female and male smoking on natural and assisted fertilization. In 13 studies of natural conception, all but one demonstrated a negative association between smoking and time to conception. Smoking one pack of cigarettes per day and starting to smoke before 18 years of age were further associated with an increased risk of infertility, providing evidence of dose- and age-related effects on natural fertility.



Maternal smoking during pregnancy is known to be associated with adverse pregnancy outcomes, including low birthweight, intrauterine growth retardation, premature delivery, spontaneous abortion, placental abruption, placenta praevia, perinatal mortality and ectopic pregnancy, especially in older mothers (Kistin *et al.*, 1996; Ahluwalia *et al.*,

metabolites of nicotine through the placenta (Shulman *et al.*, 1990). The increased miscarriage rate among mothers who smoke may be related to direct adverse effects of smoke components such as nicotine, cadmium and polyaromatic hydrocarbons on trophoblast invasion and proliferation (Shiverick & Salafia, 1999).

During early pregnancy, smoking is associated with significantly depressed levels of oestriol, oestradiol, human chorionic gonadotrophin and human placental lactogen, which may explain certain adverse effects of smoking; there appears to be a steady decline in these values with increasing cigarette consumption (Bernstein *et al.*, 1989; Shiverick & Salafia, 1999).



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