



Contraception xx (2009) xxx–xxx

## Clinical Guidelines

# Contraceptive considerations in obese women

Release date 1 September 2009

SFP Guideline 20091

---

### Abstract

Contraceptive failure is the primary cause of unintended pregnancy in the United States. With obesity rates at epidemic proportions, any association between obesity and strategies that prevent undesired pregnancies constitutes a significant public health and economic concern. Unfortunately, the relationship between obesity and contraception has not been extensively studied. Evidence from several epidemiological studies suggests that obesity may increase failure of some hormonal contraceptives resulting in unplanned pregnancies. Obesity may make procedure-dependent contraceptive methods (i.e., sterilization and intrauterine devices) more technically challenging for the provider to perform. Hormonal contraceptives, on the whole, do not appear to adversely affect body weight and provide important noncontraceptive benefits (i.e., cancer protection). Some surgical interventions to treat bariatric issues may compromise the efficacy of orally dosed contraceptive methods. Overall, the Society of Family Planning strongly encourages the use of both hormonal and nonhormonal methods of contraception in obese women desiring pregnancy prevention with very few restrictions. Further studies are needed to determine the interrelationship between obesity and contraception. In addition, future contraceptive efficacy studies need to include women of differing BMIs to better reflect the population of women using these methods.

© 2009 Elsevier Inc. All rights reserved.

*Keywords:* Obesity; Body mass index (BMI); Body weight; Contraception; pregnancy prevention; Unplanned pregnancies; Pregnancy; Bariatric surgery; Contraceptive compliance; Sexual behavior

---

### Background

The rate of obesity worldwide is at epidemic proportions with 1 billion and 300 million adults meeting the criteria for overweight and obese, respectively [1]. Currently, the obesity rate in Europe and the United States is approximately 30% and rising [1,2]. The prevalence of unintended pregnancy rivals that of obesity. Forty-nine percent of all pregnancies per year in the United States are unintended (3.1 million), and roughly half a million of these are related to oral contraceptive failures [3]. Obesity is known to affect the health of both present and future generations with higher rates of both maternal and fetal morbidity and mortality, and increased rates of obesity and diabetes in offspring [4–8]. Thus, any association between obesity and the ability to prevent pregnancies constitutes a significant public health and economic concern. Unfortunately, most contraceptive research has excluded women above 130% of ideal body weight, making it difficult to counsel these women regarding their risk for contraceptive failure [9].

Use of a safe and effective contraceptive method in women with chronic medical conditions, like obesity, is

paramount since these women are at higher risk of pregnancy-related complications. In addition, many contraceptive methods may offer important noncontraceptive benefits for these women [10–13]. However, obese women are less likely to use contraception or to receive preventative health care services as compared to women with a normal BMI [14,15]. It is unclear whether this disparity is related to patient, provider or systems issues. In regard to contraceptive care, perhaps there is an assumption that fertility is impaired and contraception is unnecessary, other health issues supersede contraceptive counseling or there is a perception that contraception would be riskier than a pregnancy. However, it is essential that the risks of contraceptive use in obese women and the potential impact of obesity on contraceptive efficacy be compared to the health, financial and personal implications of an unplanned pregnancy. The use of contraception in obese women will always prevent more pregnancies than no contraception even in the event of impaired contraceptive effectiveness and is universally always less risky to these women than a pregnancy.

The inherent efficacy of hormonal contraception in obese users has not been well studied. The research is inconsistent

and demonstrates either no difference between BMI categories or an increase in contraceptive failures in the obese group [16–28]. There are also significant limitations to most of the studies, the majority of which are retrospective and underpowered with self-reported weight or BMI, self-reported oral contraceptive use/type, and many use databases where unintended pregnancies ending in abortion are significantly underreported or not included [29]. Overall, self-reported weight or BMI in women is fairly accurate in that height is overreported and weight is underreported; objective measurements of weights and heights would then only strengthen the findings of positive studies [30–34]. Several of the studies were based in Europe where contraceptive effectiveness has been demonstrated to be higher and thus findings would be biased towards a null result [35]. Many of these studies were performed at a time where the prevalence of obesity and morbid obesity was less than currently exists. Most importantly, no studies address the potential, if any, biological mechanism for failure of hormonal contraception.

Obesity is defined based on body mass index (BMI), which is an indirect measure of body fat. BMI has been shown to correlate well to direct assessments of body fat [i.e., dual-energy X-ray absorptiometry, underwater weighing and air displacement plethysmography (BOD POD)] [36,37]. BMI is calculated by dividing weight in kilograms by height in meters squared. Although BMI is not a perfect indicator of body fat, it is reliable, inexpensive and easy to perform in a clinical setting. BMI categories are defined by The Centers for Disease Control and Prevention and The World Health Organization as [33,38,39]:

- Underweight  $<18.5 \text{ kg/m}^2$
- Normal  $18.5\text{--}24.9 \text{ kg/m}^2$
- Overweight  $25\text{--}29.9 \text{ kg/m}^2$
- Obese  $30\text{--}39.9 \text{ kg/m}^2$  or Class I obesity  $30\text{--}34.9 \text{ kg/m}^2$  and Class II obesity  $35\text{--}39.9 \text{ kg/m}^2$
- Very obese  $\geq 40 \text{ kg/m}^2$  or otherwise referred to as severe, extreme, morbid or Class III obesity

This document will review the current evidence regarding the interrelationship between contraception and obesity.

### Clinical questions and recommendations

#### 1. Are obese women at increased risk for pregnancy as compared to their normal BMI counterparts?

Abnormalities in metabolism and extremes in body weight can adversely affect the reproductive system. Obesity is a known risk factor for reduced fertility because of menstrual abnormalities, anovulation, polycystic ovarian disease and insulin resistance [40,41]. However, the majority of women, both thin and obese, ovulate on a regular basis and are at risk for pregnancy [40].

The rate of sexual activity and use of contraception also affect the risk of pregnancy. Many assume that obese women

engage in less frequent sexual activity, making them at less risk of pregnancy. An analysis of the 2002 National Survey of Family Growth (NSFG) demonstrated no differences in sexual behaviors between BMI categories in sexually active reproductive-age women [42]. In regard to contraceptive use, an analysis of the Family Planning Module of the Behavioral Risk Factor Surveillance System (BRFSS; 7943 women) found that obese women were significantly less likely to use contraception as compared to normal BMI women [15]. It is unclear whether this disparity is due to patient, provider and/or systems issues. Of note, these obese women were also more likely to be older, Black, Hispanic, married, less educated and without health insurance.

Available information regarding female adolescent sexual activity based on weight differences is somewhat conflicting. A longitudinal study of 200 teens demonstrated that “thinner” girls dated more and thus had more opportunity for and higher frequency of petting and coital activity as compared to “heavier” girls [43]. A survey of 522 African-American female adolescents found that obese adolescents were more likely to have body image dissatisfaction and lower self-esteem issues. These factors were felt to lead to earlier coital debut ( $<14$  years old), fear of abandonment when trying to negotiate condom use, less confidence to refuse an unsafe sexual encounter and higher rates of unprotected intercourse [44].

Based on these findings, obese adult and adolescent women appear to be at a similar or higher risk of pregnancy as compared to normal BMI women.

#### 2. Does obesity affect oral contraceptive effectiveness?

Contraceptive effectiveness relies on medication compliance, sexual behavior (discussed above), fecundity and the inherent efficacy of the medication. Poor medication compliance has been blamed for the majority of oral contraceptive failures. Using an electronic device implanted in pill packages, researchers compared patient self-report and electronic data regarding compliance. Self-reported data significantly underestimated the number of pills missed [45]. However, obese women have never been demonstrated to be less compliant with medication than normal BMI women.

In general, the effect of obesity on drug pharmacokinetics is poorly understood. A study comparing the pharmacokinetics and hypothalamic-pituitary-ovarian activity of obese ( $\text{BMI} >30 \text{ mg/k}^2$ ) and normal ( $\text{BMI} <25 \text{ mg/k}^2$ ) BMI women using a 20-mcg ethinyl estradiol (EE) /100-mcg levonorgestrel oral contraceptive demonstrated that the obese group had a significantly longer levonorgestrel half-life ( $52.1 \pm 29.4$  vs.  $25.6 \pm 9.3$  h,  $p < .05$ ) which correlated with a lower maximum levonorgestrel serum level and a longer time to reach steady state (10 vs. 5 days) [46]. There were no significant differences in volume of drug distribution between the BMI groups. Consistent with these pharmacokinetic findings, more obese women demonstrated hormonal

changes associated with recruitment and maturation of a dominant follicle, and even ovulation, but the sample size was too small to achieve statistical significance. It is unknown whether these findings translate into an actual risk of pregnancy.

A few studies support an association between weight or BMI and combined oral contraceptive failure. Holt et al. [16] performed a retrospective cohort analysis of women from a single health maintenance organization (HMO) in Seattle, WA, who were involved in a dietary study. In the highest overall weight quartile ( $\geq 70$  kg) and the highest weight quartile of women using oral contraceptives with  $\leq 35$  mcg EE, the relative risk of contraceptive failure was significantly increased [RR 1.6 (CI 1.1–2.4) and RR 4.5 (CI 1.4–14.4), respectively]. The study was limited by no weight documentation immediately prior to the contraceptive failure, and self-reported contraceptive failure (no confirmation of pregnancy) and type of oral contraceptive used. In addition, the highest weight quartile was equivalent to the mean weight for the overall population and not reflective of an obese population.

Holt et al. [17] followed this report with a case-controlled study of women in the same HMO. Women with a BMI of 27.4–32.2 kg/m<sup>2</sup> were found to have increased odds of combined oral contraceptive failure [OR 1.58 (CI 1.11–2.24) and OR 1.72 (CI 1.04–2.82)]. Pregnancies were confirmed in this study, but weight in the reference month was self-reported, more cases than controls had been previously pregnant and subjects were excluded if they missed five or more pills in the reference month.

A case control study using the 1999 Pregnancy Risk Assessment Monitoring System (PRAMS) also found that both overweight and obese women using contraception (type not specified) had an increased chance of contraceptive failure [OR 1.73 (CI 1.2–2.36) and OR 1.75 (CI 1.21–2.52)] [19]. This study was limited by self-reported BMI and contraceptive type, and cases were only limited to unintended pregnancies leading to live births because abortions were not tracked. Although the type of contraception in this study was not specified, one could assume that the majority of the study group was using combined oral contraception since that is the most common form of hormonal contraception used in the United States.

A secondary analysis of a large prospective randomized trial ( $N=2812$  women) evaluated the relationship between contraceptive efficacy and weight/BMI. The primary study compared oral contraceptives containing 25 mcg EE/180/215/250 norgestimate vs. 20 mcg EE/1 mg norethindrone [26]. For the secondary analysis, body habitus indices were dichotomized to either  $<70$  or  $\geq 70$  kg or  $<25$  or  $\geq 25$  kg/m<sup>2</sup>. A slight but not statistically significant increase in the relative risk of pregnancy was found in the  $\geq 70$  kg and  $\geq 25$  kg/m<sup>2</sup> groups [RR 1.25 (CI 0.63–2.46) and 1.85 (CI 0.98–3.45), respectively]. This study excluded women with a BMI of  $>32$  kg/m<sup>2</sup> and thus does not provide us with information in a truly obese population.

In contrast, several studies have shown no association between weight or BMI and oral contraceptive failure. Retrospective analyses of the 1995 and the 2002 NSFG showed no evidence of decreased contraceptive efficacy or increased unintended pregnancies in different BMI categories after adjusting for confounders [20–22]. The NSFG is limited by self-reported BMI, oral contraceptive use, oral contraceptive type (does not differentiate between combined and progestin-only) and pregnancies. In addition, abortions are significantly underreported (only 43%) in the NSFG [29]. Using the 1999 BRFSS and the 2000 PRAMS for South Carolina, a case-cohort study was performed and found no decreased contraceptive efficacy in obese women after adjusting for confounders [23]. As previously mentioned, these databases are limited by self-reported height, weight, pregnancies and oral contraceptive use/type. In addition, only live births were tracked and very small numbers of overweight/obese women were included (overweight  $n=44$ , obese  $n=28$ ). A reanalysis of several recent combined oral contraceptive trials found no change in contraceptive efficacy for heavier women, but again these studies were limited by either small samples sizes — only 55 out of 1673 women studied were  $>198$  lb [24] — or a nontraditional dosing pattern (extended 91-day regimen) [25] which may have altered the risk of failure.

Finally, two large longitudinal studies in Europe found no effect of weight on oral contraceptive efficacy [27,28]. A study of British progestin-only pill users demonstrated no increase in contraceptive failures for obese women. This study had several significant limitations, including no report of the number of overweight/obese women, measurement of weight only at recruitment (1968–1974), study timing (the study occurred prior to the current obesity epidemic). The European Active Surveillance Study on Oral Contraceptives (EURAS-OC) monitored 59,510 combined oral contraceptive users from 2000 to 2005 with an impressively small lost to follow-up rate (2.4%) [28]. No association between BMI or weight and contraceptive failure was found with the exception of a small effect in pills with the progestin chlormadinone acetate (not currently available in the US market). The actual number of obese women in the study was not mentioned, but the mean BMI for the study population was in the normal range ( $<25$  kg/m<sup>2</sup>). As mentioned earlier, it is possible the results from these two studies are not generalizable to the US as European women tend to have a higher contraceptive effectiveness rate than the US women [35].

When all of the data is considered, overweight and obese oral contraceptive users (both combined and progestin-only) appear to be at a similar or slightly higher risk of pregnancy as compared to normal BMI women. However, even if this risk is higher, that actual increase (attributable risk) would be minimal. If the results in the studies by Holt et al. [17] are considered to be causal, then only two to four extra pregnancies per 100 women-years would be expected in

women with a BMI >27.3 as compared to women with a lower BMI.

### 3. Does obesity affect contraceptive effectiveness for nonoral contraceptive methods?

Compared to information on oral contraceptive methods, an even smaller number of obese women have been studied with other hormonal methods (contraceptive patch, contraceptive ring, implant, injection) [47–56]. An abstract reporting the pooled analysis of three multicentered cohort studies of the contraceptive patch reported a possible increase in contraceptive failure in women weighing  $\geq 90$  kg, but it did not report what the increase was or how many women weighing  $\geq 90$  kg were included in the analysis [48]. In regard to the contraceptive ring, an abstract reporting a reanalysis of the Phase III trials found no increase in failure for heavier women (weight 88–272 lb,  $n=6047$ ) [50]. The etonogestrel contraceptive implant has not been prospectively studied in women who weigh more than 130% of ideal body weight [55] and only a handful of users studied have weighed over 70 kg ( $n=134$ ); however, no pregnancies were reported in this group [56]. Accordingly, no definitive conclusions can be made regarding the impact of increased weight or BMI on their effectiveness.

Obesity may make procedure-dependent contraceptive methods more challenging for the provider to perform. A Cochrane review of interval laparoscopic tubal ligations found an increased complication rate in obese women [57]. Placement of an IUD may require longer instruments and an exam table with a higher weight capacity. Intramuscular injections may also require longer needles to ensure proper medication administration [58].

The number of overweight and obese women studied is too small to determine a difference in the inherent contraceptive effectiveness for nonoral hormonal methods. Effectiveness of intrauterine devices and sterilization should theoretically be unaffected by BMI but may be associated with an increase in technical difficulties and procedure-related complications.

### 4. What are the risks of contraceptive use in obese women?

The use of low-dose estrogen-containing contraceptives slightly increases the incidence of deep venous thrombosis (5–10 cases in nonusers vs. 15–30 cases in users per 10,000 women per year) [19,59]. At baseline, obesity doubles the risk of venous thromboembolism (VTE) as compared to a normal BMI [60]. As the absolute risk of VTE with combined contraceptives is quite small, the additional risk of obesity is still less than the VTE risk that pregnancy/postpartum poses in an obese woman [59–64].

The currently used EE dosages of combined contraceptives (15 to 35 mcg) have less VTE risk than older products ( $\geq 50$  mcg EE) [60]. However, no difference in venous thrombosis risk has been proven between very low (15–20 mcg EE) vs. low dose (30–35 mcg EE) products in

the general population [65]. Whether these very low dose products are safer in obese women is unknown.

Recent attention has been focused on the possible increased VTE risk with the contraceptive patch. The three published studies addressing this issue are conflicting and range from no increase in risk (OR 0.9, 95% CI 0.5–1.6) to a slight increase in risk (OR 2.4, 95% CI 1.1–5.5) [66–69]. Even when using the most conservative estimate of VTE risk (OR 2.4), the VTE risk is still lower in patch users than in pregnancy. There are no published studies focusing on VTE risk in obese contraceptive patch users.

Obese women are also at risk for comorbidities such as hypertension, hypercholesterolemia and diabetes. Increasing age is also an independent risk factor for VTE and may also be considered a comorbidity [60]. There is no safety information regarding the use of hormonal and nonhormonal contraception in obese women with comorbidities.

No safety information exists regarding the use of contraception in women with a BMI  $\geq 40$  mg/kg<sup>2</sup>.

### 5. What are the benefits of contraceptive use in obese women?

Pregnancy prevention is usually the primary contraceptive benefit for women of any weight desiring to avoid a pregnancy. An unplanned pregnancy in an obese woman carries greater risk than a normal-weight woman as obesity adversely affects the health of both present and future generations with higher rates of both maternal and fetal morbidity and mortality, and increased rates of obesity and diabetes in the offspring [4–8].

Obesity is associated with endometrial hyperplasia and cancer [70]. Although not directly studied in obese women, both hormonal and nonhormonal contraception (copper IUD) have been shown to decrease the risk of endometrial hyperplasia and cancer [13,71–74].

### 6. Does contraception adversely affect body weight?

Weight regulation is a major health and personal concern for many women. It is not surprising that women often blame contraception for their weight gain, as this may be the only medication they use consistently throughout their lifetime. However, adults tend to gain weight over time regardless of contraceptive use (hormonal or nonhormonal); most likely due to a combination of genetics, environment and lifestyle factors [2,75]. Women's perceptions of weight gain have been shown to be incongruent with their actual weight [76]. Additionally, women rarely blame nonuse of contraception as a reason for weight gain when pregnancy is clearly associated with weight gain and many women have difficulty returning to their prepregnancy weight [77].

Unfortunately, discontinuation of hormonal contraception due to perceived side effects plays a major role in the rates of unplanned pregnancy in the United States. Many women will discontinue their birth control because of these perceived side effects even though they are still at

risk for an undesired pregnancy. Studies of oral contraceptive users have found that a perceived weight gain is one of the leading reasons for discontinuation in US women [78,79].

Of note, the majority of studies researching hormonal contraception and its potential impact on weight have used a population of women that are no more than 130% of ideal body weight. The effect of hormonal contraception on weight may vary depending on the initial baseline weight, but this has not been thoroughly studied. In regard to specific contraceptive methods and weight gain:

- Nonhormonal contraception (i.e., copper intrauterine device, barrier methods) has not been associated with a change in body weight [75].
- Combined hormonal contraception (pill, patch, ring) has not been associated with a change in body weight [76,80].
- The levonorgestrel-releasing intrauterine device, in long-term users, has been associated with a small increase in weight that is equivalent to the weight gain associated with increasing age [77,81].
- The etonogestrel implant has not been well studied in regard to weight gain but appears to have little or no impact on weight [82,83].
- Depot medroxyprogesterone acetate (Depo Provera®) and weight change are more controversial. Studies have been conflicting with some showing no change in weight and others finding an increase (particularly in already obese teens) [84–87].

#### 7. Does bariatric surgery affect contraceptive efficacy?

It makes intuitive sense that any bariatric surgery which impairs gastrointestinal absorption may impair *oral* contraception. These surgeries include jejunioleal bypass, biliopancreatic diversion with/without duodenal switch, and Roux-en-Y bypass (gastric bypass). Currently, there are no published studies comparing oral contraceptive effectiveness before and after bariatric surgery, but one small study reported two out of nine patients with oral contraceptive failures after biliopancreatic diversion [88] and another found lower drug levels in morbidly obese women after jejunioleal bypass ( $n=7$ ) as compared to normal-weight controls [89]. Other forms of nonoral contraception, both hormonal and nonhormonal, should remain unaffected by bariatric surgery but have not been studied.

#### 8. What changes in contraceptive prescribing habits should be made for the obese patient?

In general, the level of evidence regarding the efficacy of some hormonal contraceptives in overweight and obese women is limited and inconsistent (i.e., Level B). Currently, there is not enough information to change clinical prescribing practices, other than helping our patients choose a contraceptive method with the overall highest inherent efficacy (i.e., IUDs, implants, sterilization). However,

hormonal contraception, both combined and progestin only, can be safely and effectively used in healthy obese women. All women using combined hormonal contraception should be counseled regarding their increased VTE risk and obese women are no exception.

### Conclusions and recommendations

The following recommendation is based on good and consistent scientific evidence (Level A):

- Use of contraception prevents more pregnancies in women regardless of BMI than nonuse of contraception.

The following recommendation is based on limited or inconsistent scientific evidence (Level B):

- Obese women, both adults and adolescents, appear to be at increased risk for pregnancy as compared to their normal BMI counterparts due to higher rates of contraceptive nonuse.
- Effectiveness of *oral* contraception (combined and/or progestin only) may be impaired in overweight and obese women.
- Healthy obese women using combined hormonal contraception (pill, patch, ring) moderately increase their risk of VTE as compared to nonobese combined hormonal contraceptive users, but this is not a contraindication to use as it is still less than the risk of VTE associated with pregnancy.
- Overall, hormonal contraception appears to have little effect on baseline body weight when studied in a nonobese female population.
- Effectiveness of oral contraception may be impaired in women undergoing bariatric surgery that causes gastrointestinal malabsorption (jejunioleal bypass, biliopancreatic diversion with/without duodenal switch, and Roux-en-Y bypass) and thus should be avoided.

The following recommendation is based primarily on consensus or expert opinion (Level C):

- No safety information exists regarding the use of any type of contraceptive method in women with a BMI  $\geq 40$  mg/kg<sup>2</sup>, but this is not an absolute contraindication to use.
- Hormonal contraception and the copper IUD prevent endometrial hyperplasia and cancer in obese women.

### Important questions to be answered

Although contraception has been scrutinized since its inception, there are many unanswered questions in regard to obesity and contraception. Previous contraceptive studies have deliberately excluded obese women. This practice is no longer conscionable, as obese individuals make up a

significant portion of our population. Future contraceptive studies, in particular efficacy studies, must be a better reflection of our current population.

## References

- [1] World Health Report 2002. Overweight, obesity, and high body mass, p 60. Accessed and downloaded 1.27.09. [http://www.who.int/whr/2002/en/whr02\\_en.pdf](http://www.who.int/whr/2002/en/whr02_en.pdf). [Evidence grade: III].
- [2] Hedley AA, Ogden CL, Johnson CL, Carroll MD, Curtin LR, Flegal KM. Overweight and obesity among US children, adolescents, and adults, 1999–2002. *JAMA* 2004;291:2847–50 [Evidence grade: II-3].
- [3] Finer LB, Henshaw SK. Disparities in rates of unintended pregnancy in the United States, 1994 and 2001. *Perspect Sex Reprod Health* 2006;38:90–6 [Evidence grade: II-3].
- [4] Rode L, Nilas L, Wojdemann K, Tabor A. Obesity-related complications in Danish single cephalic term pregnancies. *Obstet Gynecol* 2005;105:537–42 [Evidence grade: II-2].
- [5] Nohr E, Bech B, Davies M, Frydenberg M, Henriksen T, Olsen J. Prepregnancy obesity and fetal death: a study within the Danish national birth cohort. *Obstet Gynecol* 2005;106:250–9 [Evidence grade: II-2].
- [6] Catalano PM, Ehrenberg HM. The short- and long-term implications of maternal obesity on the mother and her offspring. *BJOG* 2006;113:1126–33 [Evidence grade: II-3].
- [7] Boney CM, Verma A, Tucker R, Vohr BR. Metabolic syndrome in childhood: association with birth weight, maternal obesity, and gestational diabetes mellitus. *Pediatrics* 2005;115:290–6 [Evidence grade: II-2].
- [8] Whitaker RC. Predicting preschooler obesity at birth: the role of maternal obesity in early pregnancy. *Pediatrics* 2004;114:e29–36 [Evidence grade: II-2].
- [9] Grimes DA, Shields WC. Family planning for obese women: challenges and opportunities. *Contraception* 2005;72:1 [Evidence grade: III].
- [10] World Health Organization. Endometrial cancer and combined oral contraceptives: the WHO Collaborative Study of Neoplasia and Steroid Contraceptives. *Int J Epidemiol* 1988;17:263–9 [Evidence grade: II-3].
- [11] World Health Organization Collaborative Study of Neoplasia and Steroid Contraceptives. Depot-medroxyprogesterone acetate (DMPA) and risk of endometrial cancer. *Int J Cancer* 1991;49:186–90 [Evidence grade: II-3].
- [12] Guleria K, Agarwal N, Mishra K, Gulati R, Mehendiratta A. Evaluation of endometrial steroid receptors and cell mitotic activity in women using copper intrauterine device: Can Cu-T prevent endometrial cancer? *J Obstet Gynaecol Res* 2004;30:181–7 [Evidence grade: III].
- [13] Varma R, Sinha D, Gupta JK. Non-contraceptive uses of levonorgestrel-releasing hormone system (LNG-IUS)—A systematic enquiry and overview. *Eur J Obstet Gynecol Reprod Biol* 2005;125:9–28 [Evidence grade: III].
- [14] Cohen SS, Palmieri RT, Nyante SJ, Koralek DO, Kim S, Bradshaw P, et al. Obesity and screening for breast, cervical, and colorectal cancer in women: a review. *Cancer* 2008;112:1892–904 [Evidence grade: III].
- [15] Chuang CH, Chase GA, Bensyl DM, Weisman CS. Contraceptive use by diabetic and obese women. *Women's Health Issues* 2005;15:167 [Evidence grade: II-3].
- [16] Holt V, Cushing-Haugen K, Daline J. Body weight and risk of oral contraceptive failure. *Obstet Gynecol* 2002;99:820–7 [Evidence grade: II-2].
- [17] Holt V, Scholes D, Wicklund K, Cushing-Haugen K, Daling J. Body mass index, weight, and oral contraceptive failure risk. *Obstet Gynecol* 2005;105:46–52 [Evidence grade: II-2].
- [18] Norris P, Kamat A, Estes C, Medina C, Pietro P, Whitted W. Oral abstract presentation: contraceptive failure in overweight patients taking combination oral contraceptive pills. Association of Reproductive Health Professionals; 2003 [Evidence grade: II-2].
- [19] Brunner Huber LR, Hogue CJ. The association between body weight, unintended pregnancy resulting in a livebirth, and contraception at the time of conception. *Matern Child Health J* 2005;9:413–20 [Evidence grade: II-3].
- [20] Brunner LR, Hogue CJ. The role of body weight in oral contraceptive failure: results from the 1995 National Survey of Family Growth. *Ann Epidemiol* 2005;15:492–9 [Evidence grade: II-3].
- [21] Brunner Huber LR, Toth JL. Obesity and oral contraceptive failure: findings from the 2002 National Survey of Family Growth. *Am J Epidemiol* 2007;166:1306–11 [Evidence grade: II-3].
- [22] Kaneshiro B, Edelman A, Carlson N, Nichols M, Jensen JT. The relationship between body mass index and unintended pregnancy results from the 2002 National Survey of Family Growth. *Contraception* 2008;77:234–8 [Evidence grade: II-3].
- [23] Brunner Huber LR, Hogue CJ, Stein AD, Drews C, Zieman M. Body mass index and risk for oral contraceptive failure: a case-cohort study in South Carolina. *Ann Epidemiol* 2006;16:637–43 [Evidence grade: II-2].
- [24] Zhang H, LaGuardia K, Creanga D. Higher body weight and body mass index are not associated with reduced efficacy in Ortho Tri-Cyclen Lo Users (Abstract). *Obstet Gynecol* 2006;50S:107 [Evidence grade: II-3].
- [25] Westhoff C, Reape K, Hait H. Subject weight and oral contraceptive efficacy in recent clinical trials (Abstract). *Contraception* 2008;78:167 [Evidence grade: II-3].
- [26] Burkman R, Fisher A, Wan G, Barnowski C, LaGuardia K. Association between efficacy and body weight or body mass index for two low-dose oral contraceptives. *Contraception* 2009;79:424–7 [Evidence grade: II].
- [27] Vessey M, Painter R. Oral contraceptive failures and body weight: findings in a large cohort study. *J Fam Plann Reprod Health Care* 2001;27:90–1 [Evidence grade: II-2].
- [28] Dinger J, Cronin M, Mohner S, Schellschmidt I, Minh T, Westhoff C. Oral contraceptive effectiveness according to body mass index, weight, age, and other factors. *AJOG* 2009;201 in press. [Evidence grade: II-2].
- [29] National Survey of Family Growth Cycle 6:2002 (Public use file). [http://www.cdc.gov/nchs/data/nsfg/UserGuide\\_2002NSFG.pdf](http://www.cdc.gov/nchs/data/nsfg/UserGuide_2002NSFG.pdf). Accessed October 27, 2008. [Evidence grade: II-3].
- [30] Stewart AW, Jackson RT, Ford MA, Beaglehole R. Underestimation of relative weight by use of self reported height and weight. *Am J Epidemiol* 1987;125:122–6 [Evidence grade: III].
- [31] Brunner Huber LR. Validity of self-reported height and weight in women of reproductive age. *Matern Child Health J* 2007;11:137–44 [Evidence grade: III].
- [32] Engstrom JL, Paterson SA, Doherty A, Trabulsi M, Speer KL. Accuracy of self-reported height and weight in women: an integrative review of the literature. *J Midwifery Women's Health* 2003;43:338–45 [Evidence grade: III].
- [33] Perry GS, Byers TE, Mokdad AH, Serdula MK, Williamson DF. The validity of self-reports of past body weights in US adults. *Epidemiology* 1995;6:61–6 [Evidence grade: II-3].
- [34] Troy LM, Hunter DJ, Manson JE, Colditz GA, Stampfer MJ, Willett WC. The validity of recalled weight among younger women. *Int J Obes Relat Metab Discord* 1995;19:570–2 [Evidence grade: II-3].
- [35] Moreau C, Trussell J, Rodriguez G, Bajos N, Bouyer J. Contraceptive failure rates in France: results from a population-based survey. *Hum Reprod* 2007;22:2422–7 [Evidence grade: II-3].
- [36] Mei Z, Grummer-Strawn LM, Pietrobelli A, Goulding A, Goran MI, Dietz WH. Validity of body mass index compared with other body-composition screening indexes for the assessment of body fatness in children and adolescents. *AJCN* 2002;7:597–985 [Evidence grade: III].
- [37] Garrow JS, Webster J. Quetelet's index (W/H<sup>2</sup>) as a measure of fatness. *Int J Obes* 1985;9:147–53 [Evidence grade: III].

- [38] World Health Organization. BMI categories. <http://www.euro.who.int/nutrition>. Accessed and downloaded October 7, 2008. [Evidence grade: III].
- [39] The Centers for Disease Control and Prevention. BMI categories. <http://www.cdc.gov/nccdphp/dnpa/healthyweight>. Accessed and downloaded October 7, 2008. [Evidence grade: III].
- [40] Speroff L, Glass RH, Kase NG. Clinical gynecologic endocrinology and infertility. 6th ed. Baltimore (Md): Lippincott Williams & Wilkins; 1999 [Evidence grade: III].
- [41] Farrow A, Hull M, Northstone K, Taylor H, Ford WC, Golding J. Prolonged use of oral contraception before a planned pregnancy is associated with a decreased risk of delayed conception. *Hum Reprod* 2002;17:2754-61 [Evidence grade: II-3].
- [42] Kaneshiro B, Jensen JT, Carlson NE, Harvey SM, Nichols MD, Edelman AB. Body mass index and sexual behavior. *Obstet Gynecol* 2008;112:586-92 [Evidence grade: II-3].
- [43] Halpern C, Udry JR. Effects of body fat on weight concerns, dating, sexual activity: a longitudinal analysis of black and white adolescent girls. *Developmental Psychology* 1999;721-36 [Evidence grade: II-2].
- [44] Wingood GM, Diclemente RJ, Harrington K, Davies SL. Body image and African American females' sexual Health. *J Wom Health Gen Base Med* 2002;11:433-9 [Evidence grade: II-2].
- [45] Potter L, Oakley D. Measuring compliance among oral contraceptive users. *Fam Plann Perspect* 1996;28:154-8 [Evidence grade: II-2].
- [46] Edelman A, Carlson N, Cherala G, Munar M, Stouffer R, Cameron J, et al. Impact of obesity on oral contraceptive pharmacokinetics and hypothalamic-pituitary-ovarian activity. *Contraception* 2009 in press. [Evidence grade: II-1].
- [47] Audet M, Moreau M, Koltun W, Waldbaum A, Shangold G, Fisher A, et al. Evaluation of contraceptive efficacy and cycle control of a transdermal contraceptive patch vs an oral contraceptive. *JAMA* 2001; 285:2347-54 [Evidence grade: 1].
- [48] Ziemann M, Guillebaud J, Weisberg E, Shangold G, Fisher A, Creasy G. Contraceptive efficacy and cycle control with the Ortho Evra/Evra transdermal system: the analysis of pooled data. *Fertil Steril* 2002;77: S13-18 [Evidence grade: II-3].
- [49] Dieben T, Roumen F, Apter D. Efficacy, cycle control, and user acceptability of a novel combined contraceptive vaginal ring. *Obstet Gynecol* 2002;100:585-93 [Evidence grade: II-2].
- [50] Westhoff C. Higher body weight does not affect Nuvaring's efficacy. *Obstet Gynecol* 2005;56S:105.
- [51] Roumen FJME, Apter D, Mulders TMT, Dieben TOM. Efficacy, tolerability and acceptability of a novel contraceptive vaginal ring releasing etonogestrel and ethinyl oestradiol. *Hum Reprod* 2001;16: 469-75 [Evidence grade: II-2].
- [52] Croxatto H, Mäkäräinen L. The pharmacodynamics and efficacy of Implanon®: an overview of the data. *Contraception* 1998;58:91-7 [Evidence grade: II-2].
- [53] Huber J. Pharmacokinetics of Implanon®: An integrated analysis. *Contraception* 1998;58:85S-90S [Evidence grade: II-2].
- [54] Jain J, Jakimiuk J, Bode F, Ross D, Kaunitz A. Contraceptive efficacy and safety of DMPA-SC. *Contraception* 2004;70:269-75 [Evidence grade: II-2].
- [55] Personal communication with Schering-Plough; 2009. Kenilworth, NJ. March 25. [Evidence grade: III].
- [56] Graesslin O, Korver T. The contraceptive efficacy of Implanon: a review of clinical trials and marketing experience. *Euro J Contra Reprod Health Care* 2008;13:4-12 [Evidence grade: II-3].
- [57] Jamieson D, Hillis S, Duerr A, Marchbanks P, Costello C, Peterson H. Complications of interval laparoscopic tubal sterilization: findings from the United States Collaborative Review of Sterilization. *Obstet Gynecol* 2000;96:997-1002 [Evidence grade: II-3].
- [58] Nisbet A. Intramuscular gluteal injections in the increasingly obese population: retrospective study. *BMJ* 2006;332:637-8 [Evidence grade: III].
- [59] Heinemann LAJ, Dinger JC. Range of published estimates of venous thromboembolism incidence in young women. *Contraception* 2007;75: 328-36 [Evidence grade: II-2].
- [60] Abdollahi M, Cushman M, Rosendaal F. Obesity: risk of venous thrombosis and the interaction with coagulation factor levels and oral contraceptive use. *Thrombosis & Haemostasis* 2003;89:493-8 [Evidence grade: II-2].
- [61] Nightingale A, Lawrenson R, Simpson E, Williams T, Farmer K. The effects of age, body mass index, smoking, and general health on the risk of venous thromboembolism in users of combined oral contraceptives. *Eur J Contracept Reprod Health Care* 2000;5:265-74 [Evidence grade: II-2].
- [62] World Health Organization. Venous thromboembolic disease and combined oral contraceptives: results of international multi-centre case control study. *Lancet* 1995;346:1575-82 [Evidence grade: II-1].
- [63] Trussell J, Guthrie K, Schwarz E. Much ado about little: obesity, combined hormonal contraceptive use and venous thrombosis. *Contraception* 2008;77:143-6 [Evidence grade: III].
- [64] Larsen TB, Sorensen HT, Gislum M, Johnsen SP. Maternal smoking, obesity, and the risk of thromboembolism during pregnancy and the puerperium: a population-based nested control study. *Thromb Res* 2007;120:505-9 [Evidence grade: II-2].
- [65] World Health Organization. Cardiovascular disease and steroid hormone contraception. Report of a WHO Scientific Group. *World Health Organ Tech Rep Ser* 1998;877:i-89.
- [66] Cole JA, Norman H, Doherty M, Walker A. Venous thromboembolism, myocardial infarction, and stroke among transdermal contraceptive system users. *Obstetrics & Gynecology* 2007;109:339-46 [Evidence grade: II-2].
- [67] Jick SS, Kaye JA, Russmann S, Jick H. Risk of nonfatal venous thromboembolism in women using a contraceptive transdermal patch and oral contraceptives containing norgestimate and 35 µg of ethinyl estradiol. *Contraception* 2006;73:223-8 [Evidence grade: II-2].
- [68] Jick S, Kaye JA, Li L, Jick H. Further results on the risk of nonfatal venous thromboembolism in users of the contraceptive transdermal patch compared to users of oral contraceptives containing norgestimate and 35 µg of ethinyl estradiol. *Contraception* 2007;76:4-7 [Evidence grade: II-2].
- [69] Boston Collaborative Drug Surveillance Program. Postmarketing study of ORTHO EVRA and levonorgestrel oral contraceptives containing hormonal contraceptives with 30 µg of EE in relation to non-fatal venous thromboembolism, ischemic stroke, and myocardial infarction. Accessed March 27, 2008. <http://www.clinicaltrials.gov/ct2/show/NCT00511784>. [Evidence grade: III].
- [70] Brinton L, Berman M, Mortel R, Twigg L, Barrett R, Wilbanks GD, et al. Reproductive, menstrual, and medical risk factors for endometrial cancer: results from a case-control study. *AJOG* 1992;167:1317-25 [Evidence grade: II-2].
- [71] World Health Organization. Endometrial cancer and combined oral contraceptives: the WHO Collaborative Study of Neoplasia and Steroid Contraceptives. *Int J Epidemiol* 1988;17:263-9 [Evidence grade: II-2].
- [72] Jick SS, Walker AM, Jick H. Oral contraceptives and endometrial cancer. *Obstet Gynecol* 1993;82:931-5 [Evidence grade: II-2].
- [73] World Health Organization Collaborative Study of Neoplasia and Steroid Contraceptives. Depot-medroxyprogesterone acetate (DMPA) and risk of endometrial cancer. *Int J Cancer* 1991;49:186-90 [Evidence grade: II-2].
- [74] Curtis KM, Marchbanks PA, Peterson HB. Neoplasia with use of intrauterine devices. *Contraception* 2007;75:S60-9 [Evidence grade: III].
- [75] Hassan DF, Petta CA, Aldrighi JM, Bahamondes L, Perrotti M. Weight variation in a cohort of women using copper IUD for contraception. *Contraception* 2003;68:27-30 [Evidence grade: II-2].
- [76] O'Connell KJ, Osborne LM, Westhoff C. Measured and reported weight change for women using a vaginal contraceptive ring vs. a low-

- dose oral contraceptive. *Contraception* 2005;72:323–7 [Evidence grade: I].
- [77] Linne Y, Dye L, Barkeling B, Rossner S. Weight development over time in parous women—the SPAWN study—15 years follow-up. *Int J Obesity* 2003;27:1516–22 [Evidence grade: II-2].
- [78] Picardo CM, Nichols M, Edelman A, Jensen J. Women's knowledge and sources of information on the risks and benefits of oral contraception. *J Am Med Women's Assoc* 2003;58:112–6 [Evidence grade: III].
- [79] Rosenberg M. Weight change with oral contraceptive use and during the menstrual cycle: results of daily measurements. *Contraception* 1998;58:345–9 [Evidence grade: II-1].
- [80] Gallo M, Grimes DA, Schulz K, Helmerhorst F. Combination estrogen-progestin contraceptives and body weight: systematic review of randomized controlled trials. *Obstet Gynecol* 2004;103:359–73 [Evidence grade: I].
- [81] Ronnerdag M, Odland V. Health effects of long-term use of the intrauterine levonorgestrel-releasing system: a follow-up study over 12 years of continuous use. *Acta Obstet Gynecol Scand* 1999;78:716–21 [Evidence grade: II-2].
- [82] The Implanon US Study Group. Safety and efficacy of Implanon, a single-rod implantable contraceptive containing etonogestrel. *Contraception* 2005;71:319–26 [Evidence grade: II-2].
- [83] Kiriwat O, Patanayindee A, Koetsawang S, Korver T, Coelingh Bennink H. A 4-year pilot study on the efficacy and safety of Implanon, a single-rod hormonal contraceptive implant, in healthy women in Thailand. *Eur J Contracept Reprod Health Care* 1998;3:85–91 [Evidence grade: II-2].
- [84] Bahamondes L, Del Castillo S, Tabares G, Arce XE, Perrotti M, Petta C. Comparison of weight increase in users of depot medroxyprogesterone acetate and copper IUD up to 5 years. *Contraception* 2001;64:223–5 [Evidence grade: II-2].
- [85] Mangan SA, Larsen PG, Hudson S. Overweight teens at increased risk for weight gain while using depot medroxyprogesterone acetate. *J Pediatr Adolesc Gynecol* 2002;15:79–82 [Evidence grade: III].
- [86] Bonny AE, Ziegler J, Harvey R, Debanne SM, Secic M, Cromer BA. Weight gain in obese and nonobese adolescent girls initiating depot medroxyprogesterone, oral contraceptive pills, or no hormonal contraceptive method. *Arch Pediatr Adolesc Med* 2006;160:40–5 [Evidence grade: II-2].
- [87] Taneepanichskul S, Reinprayoon D, Khaosaad P. Comparative study of weight change between long-term DMPA and IUD acceptors. *Contraception* 1998;58:149–51 [Evidence grade: II-2].
- [88] Gerrits EG, Ceulemans R, van Hee R, Hendrickx L, Totté E. Contraceptive treatment after biliopancreatic diversion needs consensus. *Obes Surg* 2003;13:378–82 [Evidence grade: III].
- [89] Victor A, Odland V, Kral JG. Oral contraceptive absorption and sex hormone binding globulins in obese women: effects of jejunoileal bypass. *Gastroenterol Clin North Am* 1987;16:483–91 [Evidence grade: III].

## Sources

Cochrane Central Register of Controlled Trials (CENTRAL), MEDLINE and PUBMED were searched for publications regarding BMI/weight and contraception since 1970. In addition, reference lists of these identified trials or review articles were searched for any additional trials that may have been missed.

## Authorship

These guidelines were prepared by Alison Edelman, MD, MPH, and were reviewed and approved by the Board of the Society of Family Planning.

## Conflict of interest statement

Alison Edelman, MD, MPH., reports that she serves as a faculty trainer for the Implanon™ Clinical Training Program

(Schering-Plough, Kenilworth, NJ, USA). The Society of Family Planning receives no direct support from pharmaceutical companies or other industries.

## Intended audience

This Society of Family Planning guideline was developed for its members and other clinicians who counsel, provide and/or manage family planning services. This guideline may be of interest to other professional groups that set practice standards for family planning services. The purpose of this document is to review the medical literature regarding obesity and contraception. This evidence-based review should help to guide clinicians providing this care, but it is not intended to dictate clinical care.