

Nonatherosclerotic vascular changes may also mediate the effect of smoking on genital function. The vasoconstrictive effects of nicotine in cigarette smoke may impair the complicated vascular processes involved in erection (Benowitz 1988). This may be due in part to disturbances of prostaglandin production in the vascular endothelium or to an enhancement of platelet aggregation noted by several investigators (Nadler, Velasco, Horton 1983; Alster et al. 1986; Taylor et al. 1987; Lassila et al. 1988; Jeremy et al. 1986; FitzGerald, Oates, Nowak 1988; Chapter 6).

Finally, hormonal effects of cigarette smoking could alter sexual responsiveness and spermatogenesis. Alterations in the secretion of luteinizing hormone releasing hormone (Moss, Riskind, Dudley 1979) or catecholamines (Patra, Sanyal, Biswas 1979; Klaiber and Broverman 1988) are two such possibilities, but disturbances in sex hormones, particularly low testosterone or high estradiol, have been suggested more often. In general, men who smoke cigarettes have similar or higher testosterone levels than nonsmokers; thus, it is difficult to associate low testosterone with sexual dysfunction among men who smoke (Briggs 1973; Shaarawy and Mahmoud 1982; Andersen, Semczuk, Tabor 1984; Handelsman et al. 1984; Deslypere and Vermeulen 1984; Vermeulen and Deslypere 1985; Vogt, Heller, Borelli 1986; Barrett-Connor and Khaw 1987; Dai et al. 1988; Lichtenstein et al. 1987; Meikle et al. 1987; Klaiber and Broverman 1988). The adrenal androgens (i.e., androstenedione, dehydroepiandrosterone, and dehydroepiandrosterone sulfate) are elevated in male smokers (Barrett-Connor, Khaw, Yen 1986; Barrett-Connor and Khaw 1987; Dai et al. 1988). Aromatization of these hormones may explain the elevated levels of estradiol among males who currently smoke (Entrican, Mackie, Douglas 1978; Lindholm et al. 1982; Klaiber, Broverman, Dalen 1984; Barrett-Connor and Khaw 1987; Lichtenstein et al. 1987; Dai et al. 1988; Klaiber and Broverman 1988). Elevations in circulating estrogens may interfere with spermatogenesis and sexual behavior (Klaiber and Broverman 1988); such an explanation remains speculative.

Several studies have suggested that the estradiol and testosterone levels of former smokers are comparable with those of never smokers (Deslypere and Vermeulen 1984; Vogt, Heller, Borelli 1986; Barrett-Connor and Khaw 1987; Lichtenstein et al. 1987). This observation implies that smoking cessation is likely to reverse any effect mediated by disturbances of these hormones. Alternatively, former smokers may have had a lower total dose. Androstenedione and dehydroepiandrosterone sulfate levels may be modestly higher in former smokers compared with those of never smokers (Barrett-Connor, Khaw, Yen 1986; Barrett-Connor and Khaw 1987; Lichtenstein et al. 1987). However, the relevance of these findings to sexual capabilities is unlikely to be significant. These hormones appear to have little intrinsic potency, and are important because of their capacity for conversion to more active hormones such as testosterone and estradiol (Baxter and Tyrrell 1987).

Sexual Activity and Performance

Surveys of the relationship between smoking and frequency of sexual episodes (intercourse or masturbation) have generally found smokers to be as sexually active as nonsmokers. In two studies of elderly men, sexual activity in smokers was comparable

with that of nonsmokers (Tsitouras, Martin, Harman 1982; Diokno, Brown, Herzog 1990); in a cross-sectional study of younger men, no differences were indicated (Vogt, Heller, Borelli 1986). Adolescent smokers are more sexually active than nonsmokers (Russell 1971; Malcolm and Shephard 1978). In contrast, Cendron and Vallery-Masson (1971), in studying 70 men older than age 45, found that those who reported smoking between ages 25 to 40 also reported being less sexually active at those ages than those who denied smoking. Overall, it appears that the relation between current cigarette smoking and the level of male sexual activity is not very strong. Among younger males, personality differences between smokers and nonsmokers may dominate any adverse physiologic effects (Russell 1971).

If, as the aforementioned studies suggest, current smokers (or ever smokers) are similar in sexual habits to never smokers, then no differences would be expected for former smokers. Vogt, Heller, and Borelli (1986) evaluated 239 healthy male volunteers aged 19 to 40 without genital abnormalities or diseases and taking no medications. The study results indicated that the 36 former smokers among them were comparable with both never smokers and current smokers in sexual activity (Vogt, Heller, Borelli 1986).

Impotence, the inability to maintain an erection sufficient for intercourse, has been more extensively investigated in relation to smoking. Among treated hypertensives aged 40 to 64, cigarette smokers were more likely to report impotence, although the differences were modest and not statistically significant (Bühler et al. 1988). A statistically significant association was reported among men undergoing radiation therapy for prostatic cancer (Goldstein et al. 1984). However, in both studies, potentially important covariates, such as alcohol intake and age, were not considered. Two other studies of men undergoing impotence evaluation indicated a high prevalence of smoking and suggested an association between smoking with impotence (Virag, Bouilly, Frydman 1985; Condra et al. 1986). Unfortunately, neither study included a sexually functional control group, and both studies based their conclusions on questionable comparisons of the smoking rate in their clinic patients with that of the general population. Vogt, Heller, and Borelli (1986) studied a group of young volunteers without selecting for impotence. These investigators found that smokers reported more difficulties with decreased libido and erection than nonsmokers (Vogt, Heller, Borelli 1986). This analysis did not consider former smokers separately.

An acute effect of smoking on sexual performance is suggested by a study of smokers monitored while viewing erotic films (Gilbert, Hagen, D'Agostino 1986). The successive smoking of 2 cigarettes high in nicotine content significantly impaired the rate of penile diameter change compared with that observed after smoking 1 cigarette or eating candy. However, the clinical relevance of these observations is unknown because frank impotence was not studied.

An important clinical measurement in the evaluation of impotence is the PBI, which indicates the systolic blood pressure in the penis divided by systolic blood pressure in the arm. A low value is considered to be evidence of compromise of the penile blood supply, a factor which may interfere with erection. Several studies of men undergoing evaluation of impotence reported an association between smoking and low PBI (Jacobs et al. 1983; Condra et al. 1986; Bornman and Du Plessis 1986; DePalma et al. 1987).

Among impotent diabetics, evidence of nocturnal erections was found less in smokers compared with nonsmokers, thus suggesting an increased risk of vascular compromise in smokers (Takahashi and Hirata 1988). However, other studies of impotent men have not reported differences between smokers and nonsmokers in vascular measurements (Wabrek et al. 1983; Virag, Bouilly, Frydman 1985; Kaiser et al. 1988). Most of these investigations did not consider covariates such as alcohol use, although one study suggested that smoking in isolation had little effect and that an association of smoking with an abnormal PBI may be due to the association of smoking with other arterial risk factors (Virag, Bouilly, Frydman 1985).

In many of the studies relating smoking and impotence, the investigators did not distinguish nonsmokers as ex-smokers or never smokers. However, two investigations considered former smokers separately (Table 15). Wabrek and associates (1983) studied 120 men who were referred to a hospital-based erectile dysfunction program. The percentage of former smokers was approximately the same among men with impaired, borderline, and normal PBI. Condra and colleagues (1986) reported on 178 patients also referred for impotence. Former smokers were not separated for analysis, but this study suggests that the PBI for ex-smokers is more normal than in current smokers (Condra et al. 1986). However, neither study considered important covariates, such as age and alcohol use (Wabrek et al. 1983; Condra et al. 1986).

Two recent investigations considered the effect of smoking cessation on impotence. Forsberg and colleagues (1979) noted that two smoking men who were impotent improved their functioning after smoking cessation at the same time that measures of penile blood flow improved. However, it is not clear how these two men were selected for this study, and control subjects were lacking. Elist, Jarman, and Edson (1984) reported on the treatment of 60 impotent men. Twenty nonsmokers were treated with the vasodilator isoxsuprine, and 40 smokers were either advised to stop smoking or advised to stop smoking and also given isoxsuprine. There was no mention of randomization, and there was no untreated control group. Similar proportions improved whether given isoxsuprine, convinced to stop smoking, or both (Elist, Jarman, Edson 1984).

Animal data have not elucidated the relation between smoking and either sexual activity or impotence. Soulairac and Soulairac (1972) studied the sexual activity of male rats given either a 0.6 mg/kg or a 1.2 mg/kg dose of nicotine subcutaneously. The sexual activity of the rats after the nicotine administration was compared with that before treatment. Sexual activity was markedly increased with the 0.6 mg/kg dose, and at 1.2 mg/kg there was trembling and twitching and no sexual behavior for 2 to 3 hours. In contrast, exposure to smoke from 1 cigarette has been shown to interfere with the physiology of erection in male dogs (Juenemann et al. 1987).

In summary, the level of sexual activity does not appear to be affected by cigarette smoking. Cigarette smoking may be associated with impaired male sexual performance. Among impotent men, smokers are more likely to have an underlying vascular problem. These associations have been more commonly noted in groups already at high risk of impotence, such as hypertensives and diabetics. However, these associations have not been consistently observed, and the positive findings may be due to the association of smoking with other factors such as alcohol use. Moreover, because the

TABLE 15.—Sexual performance among male former smokers

| Reference | Study population | Findings | Comments |
|-----------------------------|-------------------|---|--------------------------------|
| Vogt, Heller, Obe (1984) | Volunteers | No differences in sexual activity between former, current, and never smokers | No consideration of covariates |
| Wabrek et al. (1983) | Impotent patients | Proportion of former smokers similar in men with abnormal, impaired, and normal PBI | No consideration of covariates |
| Condra et al. (1986) | Impotent patients | Indications that former smokers had more normal PBI than current smokers | No consideration of covariates |
| Forsberg et al. (1979) | Impotent patients | Two smokers improved sexual performance after smoking cessation | No controls |
| Elist, Jarman, Edson (1984) | Impotent patients | Smoking cessation improved sexual performance as well as vasodilator | No untreated and controls |

NOTE: PBI=penile brachial index.

studies of PBI are generated entirely in referral populations, it is unclear if these findings can be generalized. Because of limited and uncontrolled data, no conclusions can be drawn regarding sexual performance or PBI among former smokers.

Sperm Density and Quality

Measurements of sperm density, morphology, and motility are commonly used assessments of sperm quality (Rogers and Russell 1987). Over 20 studies have dealt with the relation of cigarette smoking to sperm density, motility, and morphology (Vicizian 1968a; Schirren and Gey 1969; Campbell and Harrison 1979; Vogel, Broverman, Klaiber 1979; Stekhun 1980; Nebe and Schirren 1980; Evans et al. 1981; Godfrey 1981; Rodriguez-Rigau, Smith, Steinberger 1982; Shaarawy and Mahmoud 1982; Buiatti et al. 1984; Andersen, Semczuk, Tabor 1984; Nordenson, Abramsson, Duchek 1984; Handelsman et al. 1984; Hoidas et al. 1985; Kulikauskas, Blaustein, Ablin 1985; Ablin 1986; Rantala and Koskimies 1987; Vogt, Heller, Borelli 1986; Klaiber et al. 1987; Dikshit, Buch, Mansuri 1987; Saaranen et al. 1987; Klaiber and Broverman 1988; Saaranen et al. 1989; Rui, Oldereid, Purvis 1989; Marshburn, Sloan, Hammond 1989; Oldereid et al. 1989). Table 16 summarizes the findings of those studies that reported mean values for smokers and nonsmokers. In most studies, men smoking cigarettes had lower sperm density, although many of these studies indicated differences that were not statistically significant. The smokers' average sperm density was at least 80 percent that of the nonsmokers. In several studies sperm morphology or motility was impaired

in smokers compared with nonsmokers, but this was a less consistent finding. Few studies have considered the spermatid chromosomal characteristics of smokers compared with nonsmokers. Nordenson, Abramsson, and Duchek (1984) found smokers to have more chromosome breaks than nonsmokers, but Oldereid and coworkers (1989) reported no differences in DNA condensation as assessed by flow cytometry.

Although differences in mean values of any of these measurements suggest an effect of smoking, the most relevant parameter may be the percentage of smokers and nonsmokers who exhibit deficiencies in sperm density, morphology, or motility. Several researchers have investigated the relative risk of azoospermia (no sperm in the ejaculate) or oligospermia (reduced number of sperm) in smokers versus nonsmokers or never smokers (Table 17). Although the range of relative risks is wide, there is a clear pattern of increased risk among smokers. However, the clinical significance of oligospermia is uncertain. Most studies have used one ejaculate per man, although the within-man coefficient of variation can be as much as 60 percent (Schenker et al. 1988).

The available information suggests that current smoking is related to low sperm density. However, these data are limited. Many studies investigated men visiting infertility clinics, limiting generalization. Moreover, if male smokers with poor sperm quality are most likely to attend these clinics, selection biases may distort the results. Also, many of these studies were relatively informal. Few of the studies accounted for potentially confounding factors such as alcohol use and age. Less than half of the studies documented that a period of sexual abstinence was required for subjects before giving the sperm sample, and few of the studies analyzed multiple semen specimens as some authorities recommend (Zaneveld and Jeyendran 1988). Most studies have a small number of subjects, and their statistical power is limited for this reason. In some of the studies, it is not clear whether former smokers were included in the smoker or nonsmoker group.

A few studies investigated ex-smokers (Table 18). One was a case-control study of male infertility in Italy (Buiatti et al. 1984). The cases were azoospermic or oligospermic men being treated for infertility at the University of Florence. Controls were University outpatients who had normal sperm counts. There were no significant differences between smoking categories in the percentage of men with low sperm counts. Vogt, Heller, and Borelli (1986) evaluated 239 male volunteers. Among former smokers (those who had smoked for at least 1 year and those who had stopped smoking for at least 1 year), percent normal spermatozoa, percent young forms, percent old forms, and percent degenerate forms were comparable with those of never smokers. Stekhun (1980) reported that 42 percent of former smokers had oligospermia compared with 18 percent of never smokers. Schirren and Gey (1969) reported that three men with low sperm density and motility showed substantial increases in these parameters 3 to 6 months after smoking cessation. However, there were no controls defined in this analysis. Because of the limitations of the four studies, no conclusions are possible regarding the effects of smoking cessation on sperm quality in humans.

Animal studies have not been particularly informative. In some studies, rodents that were heavily exposed to nicotine or cigarette smoke demonstrated testicular atrophy, but this has not been a general finding (Larson, Haag, Silvette 1961; Larson and Silvette 1968; Dontenwill et al. 1973b; Essenberg, Fagan, Malerstein 1951; Thienes 1960;

TABLE 16.—Sperm quality among smokers and nonsmokers

| Reference | Study population (number of nonsmokers/number of smokers) | Ratio of measure among smokers to that among nonsmokers | | | Comments |
|--|--|--|-------------------|-------------------|--|
| | | Sperm density | % Normal sperm | % Motile sperm | |
| Viczian (1968a) | Obstetrics clinic (smokers only) (50/120) | 0.82 | 0.90 | 0.77 | No decrease in sperm density with increasing amounts smoked; controls were fertile men |
| Vogel, Broverman, Klaiber (1979) | Unstated (39/17) | 0.60 ^d | NS | 0.87 ^d | |
| Nebe and Schirren (1980) | Andrology clinic (455/451) | 1.01 | — | — | |
| Evans et al. (1981) | Subfertility clinic (43/43) | — | 0.92 ^d | — | Smokers and nonsmokers matched on sperm density |
| Godfrey (1981) | Infertility clinic (74/75) | — | 0.94 | — | Oligospermic ^b men omitted ($<1 \times 10^6$ /mL) |
| Spira et al. (1981) | Vasectomy candidates (173/122) | 0.75 ^d | 0.94 | 0.93 ^d | |
| | Infertility clinic (228/292) | 0.86 | 0.91 ^d | 0.97 | |
| Rodriguez-Rigau, Smith, Steinberger (1982) | Infertility clinic (101/58) | 0.95 | 1.00 | 1.00 | |
| Shaarawy and Mahmoud (1982) | Volunteers (20/25) | 0.93 | 0.69 ^d | 0.67 ^d | All subjects were fertile |
| Andersen, Semczuk, Tabor (1984) | Infertility clinic (86/137) | 0.99 | 1.07 | 1.08 | 10 azoospermic ^c smokers omitted from analysis |
| Handelsman et al. (1984) | Semen donors (71/23) | 0.67 | 0.98 | 0.93 ^d | |
| Kulikauskas, Blaustein, Ablin (1985) | Fertility clinic (135/103) | 0.43 ^d | 1.00 | 0.78 ^d | |
| Rantala and Koskimies (1987) | Infertility clinic (50/60) | 0.90 | 0.98 | 0.95 | Oligospermic ^b men omitted ($<1 \times$ 10^6 /mL) |
| Vogt, Heller, Borelli (1986) | Volunteers (52/150) | 0.81 ^d | 1.01 | 0.99 | |

TABLE 16.—Continued

| Reference | Study population (number of nonsmokers/number of smokers) | Ratio of measure among smokers to that among nonsmokers | | | Comments |
|-------------------------------------|--|--|-------------------|-------------------|---|
| | | Sperm density | % Normal sperm | % Motile sperm | |
| Saaranen et al. (1987) | Infertility clinic (110/54) | 0.81 | 1.00 | 0.97 | Azoospermic ^c men omitted |
| Klaiber et al. (1987) | Paid volunteers (90/60) | 0.77 ^d | 0.98 | 0.89 ^d | |
| | Males from infertile couples (43/51) | 0.52 ^d | 0.94 | 0.80 ^d | |
| Dikshit, Buch, Mansuri (1987) | Infertility clinic (288/219) | 0.96 | 0.99 | 1.01 | |
| Klaiber and Broverman (1988) | Volunteers (21/22) | 0.93 | 1.02 | 0.97 | |
| Saaranen et al. (1989) | Semen donors and fertile men (32/28) | 0.83 | 0.95 | 1.01 | |
| Marshburn, Sloan, Hammond (1989) | Infertility clinic (294/152) | 0.92 | 0.99 | 0.94 | |
| Rui, Oldereid, Purvis (1989) | Infertility clinic (203/147) | 1.17 | 1.05 | 0.96 | Azoospermic ^c men omitted |
| Effendy and Krause (1987) | Infertility clinic (61/31) | 1.13 | 1.06 | 1.12 | |

^dStatistically significant difference ($p < 0.05$) between smokers and nonsmokers.

^bOligospermia is a low sperm count.

^cAzoospermia is the absence of sperm.

Thompson et al. 1973; Patra, Sanyal, Biswas 1979; Biswas and Patra 1981). Some studies have noted a disturbance of spermatogenesis, a decrease in the interstitium, or a destruction of the seminiferous epithelium (Larson, Haag, Silvette 1961; Larson and Silvette 1968; Essenberg, Fagan, Malerstein 1951; Viczian 1968b; Wyrobeck and Bruce 1975; Biswas and Patra 1981; Alwachi et al. 1986; El-Sayad et al. 1987). The results may depend on the duration and dose of exposure, as well as on the ages at which exposure takes place. Moreover, the relevance to humans of the large doses given to the animals is uncertain. None of these investigations considered spermatogenesis after exposure ended; thus, few conclusions may be drawn regarding the effect of cessation of exposure even within the limitations of the animal studies.

TABLE 17.—Estimated relative risk of azoospermia or oligospermia among smokers versus nonsmokers or never smokers

| Reference | Study population (number of nonsmokers/ number of smokers) | Contrast | Estimated relative risk in smokers | Comments |
|--|---|--|--|-----------------------------|
| Schirren and Gey (1969) | Andrology clinic (580/1377) | Azoospermia; smokers vs. nonsmokers | 1.2 | |
| | | Oligospermia; smokers vs. nonsmokers | 1.2 | Oligospermia not defined |
| Campbell and Harrison (1979) | Fertility clinic (119/134) | Oligospermia ($<40 \times 10^6/\text{mL}$); smokers vs. nonsmokers | 1.6 ^d | Azoospermic men omitted |
| Stekhun (1980) | Not stated (33/105) | Oligospermia; current smokers vs. never smokers | 3.2 ^d | Oligospermia not defined |
| Rodriguez-Rigau, Smith, Steinberger (1982) | Fertility clinic (101/58) | Oligospermia; ($<20 \times 10^6/\text{mL}$); current smokers vs. nonsmokers | 0.9 | |
| Buiatti et al. (1984) | Fertility clinic (80/135) | Oligospermia ($<20 \times 10^6/\text{mL}$); smokers vs. nonsmokers | 1.0 | |
| Andersen, Semczuk, Tabor (1984) | Fertility clinic (86/147) | Azoospermia; current smokers vs. nonsmokers | ∞^d | |
| Ablin (1986) | Not stated (135/238) | Oligospermia ($<40 \times 10^6/\text{mL}$); smokers vs. nonsmokers | 2.9 ^d | |
| Vogt, Heller, Borelli (1986) | Volunteers (52/150) | Oligospermia ($<1 \times$ $10^6/\text{mL}$); current smokers vs. never smokers | ∞ | |
| | | Azoospermia; current smokers vs. never smokers | ∞ | |
| Klaiber et al. (1987) | Volunteers with varicocele (11/9) | Oligospermia ($<20 \times$ $10^6/\text{mL}$); current smokers vs. never smokers | ∞ | |
| | Volunteers without varicocele (79/61) | Oligospermia ($<20 \times$ $10^6/\text{mL}$); current smokers vs. never smokers | 1.3 | |
| | Fertility clinic with varicocele (8/21) | Oligospermia ($<20 \times$ $10^6/\text{mL}$); current smokers vs. never smokers | 7.7 | |

TABLE 17.—Continued

| Reference | Study population (number of nonsmokers/ number of smokers) | Contrast | Estimated relative risk in smokers | Comments |
|---|---|--|--|----------|
| Klaiber et al. (1987) (continued) | Fertility clinic without varicocele (35/30) | Oligospermia (<20 × 10 ⁹ /mL); current smokers vs. never smokers | 1.5 | |
| Dikshit, Buch, Mansuri (1987) | Fertility clinic (219/288) | Oligospermia (<20 × 10 ⁹ /mL); current smokers vs. never smokers Azoospermia; current smokers vs. never smokers | 1.2 1.1 | |

NOTE: Azoospermia is the absence of sperm; oligospermia is a low sperm count.

*Estimated relative risk statistically significantly (p<0.05) different from 1.0.

TABLE 18.—Sperm quality among former smokers

| Reference | Study population | Findings | Comments |
|---------------------------------|---------------------------------------|--|-----------------------------------|
| Schirren and Gey (1969) | Andrology patients | Smoking cessation improved sperm density and motility in 3 smokers | No control |
| Stekhun (1980) | Not stated | Former smokers had RR of 2.3 for oligospermia | Oligospermia not defined |
| Buiatti et al. (1984) | Male partners of infertile couples | No difference between current, former, and never smokers in prevalence of azoo-/oligospermia | |
| Vogt, Heller, Borelli (1986) | Healthy volunteers | No difference between current, former, and never smokers in sperm morphology | No consideration of covariates |

NOTE: RR=relative risk.

CONCLUSIONS

1. Women who stop smoking before becoming pregnant have infants of the same birthweight as those born to never smokers.
2. Pregnant smokers who stop smoking at any time up to the 30th week of gestation have infants with higher birthweight than do women who smoke throughout pregnancy. Quitting in the first 3 to 4 months of pregnancy and abstaining throughout the remainder of pregnancy protect the fetus from the adverse effects of smoking on birthweight.
3. Evidence from two intervention trials suggests that reducing daily cigarette consumption without quitting has little or no benefit for birthweight.
4. Recent estimates of the prevalence of smoking during pregnancy, combined with an estimate of the relative risk of low birthweight outcome in smokers, suggest that 17 to 26 percent of low birthweight births could be prevented by eliminating smoking during pregnancy; in groups with a high prevalence of smoking (e.g., women with less than a high school education), 29 to 42 percent of low birthweight births might be prevented by elimination of cigarette smoking during pregnancy.
5. Approximately 30 percent of women who are cigarette smokers quit after recognition of pregnancy, with greater proportions quitting among married women and especially among women with higher levels of educational attainment.
6. Smoking causes women to have natural menopause 1 to 2 years early. Former smokers have an age at natural menopause similar to that of never smokers.

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CHAPTER 9
SMOKING, SMOKING CESSATION, AND
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