Re: Semen of Smokers and Cervical Cancer Risk

Tokudome (1) suggested that key constituents of tobacco smoke inhaled by women are conveyed internally to the cervical mucus. He also said that another route for carcinogenic agents in tobacco smoke to be introduced into the cervix mucus is through the direct application of ‘‘tobacco-related mutagens/carcinogens in a smoking sex partner’s semen/semen fluid.’’

Two early reports (2,3) about cervical cancer that dealt with male partners’ smoking habits brought up the possibility that important agents in tobacco smoke adhering to the male’s fingers could be introduced into the vaginal mucus by ‘‘men using their fingers regularly within the vagina and on the clitoris during coital caressing’’ (3). The fingers of a male smoker might well be a very heavily coated part of his anatomy as regards the tar of tobacco smoke.

Thus, these three routes of exposure for the cervix to mutagenic and carcinogenic agents derived from tobacco smoke—inhaling, including that by passive smokers (4); contaminated semen/semen fluid; and digital stimulation of the vagina by fingers coated with tobacco tars—might well add up to a significant burden and might provide a trigger for a rise in risk for cervical cancer in women whose long-time male sexual partners are smokers.

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References


Note

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Re: Hormone Levels During Dietary Changes in Premenopausal African-American Women

Woods et al. (1) report significant differences in steroid hormone metabolism between African-American and Caucasian women, and they relate these variations to differences in 5-year survival.

The study is based on the hormonal effect of estrogens on mitotic activity and the hypothesis that dietary manipulation reduces plasma estrogen levels, thereby reducing mammary parenchymal mitotic activity and the risk of breast cancer. Controversy exists, however, on whether estrogen therapy, except perhaps after prolonged use, alters the risk of breast cancer (2).

Although Woods et al. (1) have consistently reported that low-fat, high-fiber diets decrease plasma estradiol concentration, two prospective studies—the Dutch study (n = 62,573) (3) and the Nurses’ Health Study (n = 89,494) (4)—revealed no association between fat or fiber intake and the risk of breast cancer. A meta-analysis of hormone replacement (5) revealed no increased risk of breast cancer.

In regard to the finding that estrone sulfate and androstenedione were associated with the waist/hip (W/H) ratio, den Tonkelaar et al. (6) in a study of premenopausal Dutch women (n = 5923) found no association between increasing W/H ratio and increased occurrence of breast cancer. Furthermore, Evans et al. (7) reported no association between estradiol concentration and W/H ratio in premenopausal women (n = 80) (92%-251% of ideal body weight; W/H ratio = 0.64-1.02).

Clearly, changes in mitotic activity during the menstrual cycle, resulting from estrogen excess or deficiency, will have an impact on the mammographic patterns (8); atypical lobules and dysplasia (DY, P2, etc.) being associated with an increased risk of breast cancer (9).

Early first birth and increasing parity decrease mammary dysplasia and atypical lobules (10), whereas physical activity may influence parenchymal patterns in premenopausal women (11).

A number of growth factors, however, are involved in the development of breast cancer. In postmenopausal Japanese (12) and Dutch (13) women, lean-ness was reported to be associated with a higher incidence of estrogen receptor (ER)-negative tumors, and ER-negative tumors were found to have a higher percentage of epidermal growth factor (EGF)/EGF-binding proteins, to grow faster, and to be associated with an earlier recurrence after surgery (14).

The insulin-like growth factor-1 (IGF1) system, which is modified in breast cancer patients, can alter its mitotic activity potential by proteolysis (15). Insulin-like growth factor-binding protein-3 (IGFBP3) inhibits estrogen-stimulated breast cancer cell proliferation (16), whereas IGFBP1 in vitro inhibits proliferation of breast cell lines (17).

Since environmental factors concomitantly modulate estrogen metabolism and the IGF1 system, it is essential to determine 1) whether changes in mitotic activity are affected by estrogens sensitizing the IGF1 system, 2) whether the IGF1 system or other growth factors modify mitotic activity and concomitantly enhance or inhibit the effect of estrogen, and 3) whether modification of estrogen metabolism by environmental factors reverses adverse mammographic patterns and, therefore, is ‘‘seen’’ by mammary parenchymal cells.

In view of the increase in in situ breast cancer in women under 45 years of age (18) and the finding that 50%-60% of patients with breast cancer develop metastatic disease for which no cure is available (19), it is time to ask what are the differences environmentally and/or metabolically in neonates and/or during adolescence that put African-American versus Caucasian women at higher risk for this disease.

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References


Note

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