Letters to the Editor

Breast Cancer Risk in Overweight Postmenopausal Women

To the Editors: We read with great interest the article by Spencer Feigelson et al. (1) showing that long-term weight gain and recent body mass index are strongly associated with postmenopausal breast cancer risk only among non–hormone replacement therapy (HRT) users in the Cancer Prevention Study II Nutrition Cohort. The effect modification by HRT on the association between obesity and postmenopausal breast cancer risk has been addressed by several prospective studies, including our study of Swedish women (2-6). However, Spencer Feigelson et al. in their summary of our report (6) did not mention the significant interaction between percentage body fat (derived from body composition by impedance analysis) and HRT use. Specifically, in our study, postmenopausal women who did not use HRT had a significantly increased breast cancer risk as a function of percentage body fat quintile (relative risk by quintile: 1.0, 1.94 [95% confidence interval (95% CI) 0.96-3.95], 2.57 [95% CI 1.32-4.99], 2.29 (95% CI 1.16-4.52), and 3.41 (95% CI 1.75-6.67); P for trend < 0.0001), whereas no risk was observed in women who used HRT [relative risk by quintile: 1.0, 0.95 (95% CI 0.50-1.80), 0.71 (95% CI 0.36-1.42), 0.69 (95% CI 0.32-1.50), and 1.00 (95% CI 0.42-2.36); P for trend = 0.450; ref. 6].

To our knowledge, this has been the first study to show that the effect of percentage body fat, a more direct measure of fatness, was modified by HRT use when investigating breast cancer risk. Moreover, the magnitude of excess risk was stronger for percentage body fat in comparison with the surrogate measure body mass index. Considering that weight gain during adult life mainly reflects the deposition of fat mass rather than lean body mass, the interaction between percentage body fat and HRT is particularly relevant to this area of investigation. These results support the etiologic hypothesis attributing increased risk of breast cancer in overweight postmenopausal women to the excess level of circulating estrogens derived from aromatization of androgen in adipose tissue together with low levels of sex hormone binding globulin.

References

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