Letters to the Editor

Correspondence re: Yasui et al., Breast Cancer Risk and “Delayed” Primary Epstein-Barr Virus Infection. 10: 9–16, 2001

Letter

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Although breast cancer is the most common malignancy of United States women (1), approximately half of all breast cancer patients are estimated to have no known risk factors (2, 3). Thus, researchers need to consider other potential causal pathways. Citing speculation that delayed viral infection might be involved with breast cancer (4) and invoking a possible common etiology for breast cancer and HD,1 Yasui et al. (5) proposed that late primary infection with EBV is a risk factor for breast cancer, as it is for HD in young adults in some populations (5, 6). Testing common incidence patterns in the two malignancies, these authors correlated age-adjusted incidence rates of breast cancer in women with age-specific incidence rates of HD in women ages 15–34 years across 135 populations in international registry data and across 27 population groups in United States registry data. They interpreted the resulting strong correlations (R = 0.74 and 0.88) as support for their hypothesis under the stated assumption that, in populations with low incidence rates of the two diseases, the rate levels were a consequence of early primary EBV infection. However, in the analysis of international data, 20 of the 34 populations with low HD rates were Asian. Young adult Asians have been shown to have low HD rates not only in Asia but also in the United States, despite their socioeconomic conditions being more likely to favor delayed primary EBV infection (7, 8). Thus, the assumption of Yasui et al. (5) is questionable, and the correlation in international rates may have an explanation other than the proposed hypothesis.

In data from a population-based case-control study of women ages 50–64 years, these authors detected a monotonic increase in breast cancer risk with self-reported age at IM, a clinical manifestation of primary EBV infection occurring in up to 50% of persons infected in late childhood or young adulthood (9, 10). Only 4.7% of the 489 controls reported a history of IM; in a population-based case-control study conducted in a similar period (1990–1995) in the Greater Bay Area, 1 of the 71 control women over age 45 years (1.4%) reported having had IM, in contrast with 45 of 254 (18%) control women under age 45 years (11, 12). This apparent extremely low prevalence of IM in older women further confuses the interpretation of the international correlations because they were based on breast cancer rates including women of all ages; breast cancer statistics show that the majority of these women would have been over age 50 years at diagnosis (1) and thus unlikely to have had delayed EBV infection, even if as many as 5% reported IM and this group constituted as few as 25% of all persons with delayed infection, as some data indicate (10).

The hypothesis of Yasui et al. (5) regarding delayed primary infection with EBV as a risk factor for breast cancer is intriguing, particularly in light of the biological mechanism they outline. However, their interpretation of some of the data presented in support fails to consider certain limitations. Moreover, in a population-based prospective study of cancer outcomes in 35,562 persons with serologically confirmed IM that found the anticipated excess of young adult HD, no excess of female breast cancer was detected [189 cases observed, 177.3 expected; standardized incidence ratio, 1.01 (0.92–1.23)], although these data were not stratified by age at breast cancer diagnosis (13). Thus, a stronger preliminary test of the Yasui hypothesis might be undertaken in younger breast cancer patients, a group more likely to have had delayed EBV infection than women studied to date.

References


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1 The abbreviations used are: HD, Hodgkin’s disease; EBV, Epstein-Barr virus; IM, infectious mononucleosis.
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