Letters to the Editor

Epidemiology of Urinary Melatonin in Women and Its Relation to Other Hormones and Night Work

To the Editors: The article by Schernhammer et al. (1) presents interesting new data on a current topic in cancer research: does shift work, and the attendant nocturnal illumination, alter cancer risk by altering hormone levels? However, two points should be made about their article. The first is their citation of Jöchle (2) for the idea “...that light at night can alter a woman’s risk of breast cancer through the melatonin pathway.” The article by Jöchle is pioneering work, and the finding that light at night can alter breast cancer risk by altering hormone levels...” and then contributes new data on the effects on constant illumination on reproduction, mammary tumorigenesis, and molting in rats, mice, and laying hens. For mammary tumorigenesis, Jöchle (2) reports that C3H-A mice under constant light show accelerated tumor development, whereas C3H-HeJ mice under constant light showed delayed mammary tumor development and a longer life span. Jöchle notes that C3H-HeJ mice are rd (retinal degeneration), but it has now been shown that the C3H/HeJ mice react normally to nocturnal illumination in suppression of melatonin (3). Jöchle (2) makes no speculation on “light at night” and breast cancer in women, and the word “melatonin” does not appear in his article. The first speculation that light at night might increase risk of breast cancer in women was in 1987 (4), and the first published speculation that shift work would be associated with increased risk was in 1992 (5), although the idea was communicated to the Nurses’ Health Study in 1987 and subsequently incorporated into their 1988 questionnaire. The second point is that the findings of Schernhammer et al. (1) of lower melatonin and higher estradiol in long-term shift working nurses are consistent with an elevated breast cancer risk but inconsistent with an elevated risk of colon cancer in shift workers [as these authors have also reported (6)]. There are much less data on melatonin and colon cancer than for breast cancer, and existing evidence implicates higher estradiol in lower risk of colon cancer (7, 8).

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References

In Response: We appreciate the additional background information conveyed by Dr. Stevens. We have originally cited a 1978 article by Cohen et al. (1) to support the initial theories proposing that a diminished function of the pineal gland might promote the development of breast cancer in humans, but we were asked to change this by a reviewer. We also explicitly mention Dr. Steven’s seminal contribution to the melatonin hypothesis in the text. Dr. Stevens is correct in that higher estradiol levels as seen in long-term shift working nurses (2) would not be consistent with an elevated colorectal cancer risk (3); we have therefore put forward an alternate explanation, suggesting that the association may be mediated at least in part by the melatonin pathway itself (4).

Eva Schernhammer
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