4. Kwan ML, Weltzien E, Kushi LH, Castillo A, and colleagues (1) have concluded that
in a thoughtful mediation analysis, Walter and colleagues (1) also argue that “it is early exposure that influences both height and risk of cancers in adulthood” and that childhood insulin-like growth factor 1 (IGF-1) levels may be implicated. We had reached a similar conclusion, but we have also pointed to the intrauterine life as a critical period (2,3). Indeed, cord blood IGF-1 has been correlated with stem cell potential, much more so than steroid hormones (4), and both cord blood IGF-1 and stem cell potential are correlated with birth weight (5). Birth weight, in turn, is higher among boys than among girls and predicts both adult height (6) and overall (7) cancer risk.

It appears that what we have postulated (2) and what Walter and colleagues have elegantly documented (1) point to early life as a relevant period for carcinogenesis and represent a step toward our understanding of an important aspect of cancer etiology. Plausible mechanistic aspects of this web of causation have already been reported (4,5). However, as far as primary prevention is concerned, this particular aspect of cancer etiology remains, at present, all but intractable.

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References

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Erratum: “Effects of vascular-endothelial protein tyrosine phosphatase inhibition on breast cancer vasculature and metastatic progression,” by Shom Goel, et al. [J Natl Cancer Inst 2013; 105(16)]. Shom Goel, MD, is also affiliated with the Centenary Institute of Cancer Medicine and Cell Biology at the University of Sydney, Camperdown, Australia. The authors regret the error.

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