We are grateful for the opportunity to respond to a number of discussion points (1) regarding our study on maternal folate intake during pregnancy and asthma in offspring (2). In our article, we did not claim that dietary exposure data for our study were collected prior to the introduction of voluntary folic acid fortification in Australia. We simply stated “prior to folic acid fortification” (2), since there was no mandated system in place at the time. Perhaps we should have stated “prior to effective folic acid fortification,” as we accept that it was possible, commencing in mid-1995, for manufacturers in Australia to nominate certain foods for a scheme of voluntary folic acid fortification. The scheme was explicitly designed to restore folate removed through processing in folate-rich foods, thereby reducing the risk of low folate intakes among women.

Implementation of the scheme nationally, however, was slow (3) and, to our knowledge, had no substantial impact on the foods both locally available on market shelves and consumed by members of our cohort during the period of dietary assessments. Our observations are supported by a review of the scheme which concluded that relatively few foods were fortified during 1995–1999, and by late 1998, voluntary fortification had had little effect on increasing daily folate intakes in the target population (from 213 μg to 235 μg) (3). Further, according to Abraham and Webb (3), the review’s authors, the rapid rise in the number of foods nominated by June 1999 comprised both foods that manufacturers intended to fortify and those which may have actually appeared upon grocers’ shelves. Abraham and Webb’s statement that the program did not meet its objectives (3) was an important argument for the recent mandatory fortification of foods in Australia.

Our dietary interviews were conducted between May 1998 and February 2000 (2). Based on the conclusions expressed in the above review and our specific, local observations at the time, we maintain that during this period it was reasonable to code foods according to nonfortified values. We understand that this approach may be viewed as conservative, in which case we might consider the potential impact of a 20-μg misclassification resulting from this decision. Such an effect would be modest, especially relative to the effect of a folate supplement of 400 μg or greater. Nevertheless, we would be pleased to reanalyze our data if accurate voluntary food fortification information became available for the specific dates of data collection.

The potential role of postnatal dietary folate intake by children is of considerable interest but was outside the scope of the present study and may form the basis for future work.

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REFERENCES


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