the father's ethnic background, the degree of First Nations genes or potential admixture with white genotypes. Such admixture has occurred in the past, so caution is needed in interpreting ethnicity (unless a detailed family history for at least 2 generations is obtained). Although that was not done for the BC study,[2] only subjects known to be registered under the federal *Indian Act* (1959) and known to be registered with an Indian Band (as they were then known) were considered as First Nations. Although there have been a few instances of a white person marrying a First Nations person and thus becoming registered, the number is minuscule; we are therefore confident that in our sample both parents and probably all 4 grandparents of the babies were of First Nations background. Ethnicity is extremely important in many genetic and congenital anomaly disorders, but unfortunately it has been deemed politically incorrect to obtain this information routinely on vital statistics documents. This loss of data affects not only those who are attempting to do etiologic research but also those who might benefit from such research.

R. Brian Lowry
Alberta Congenital Anomalies Surveillance System
Department of Medical Genetics
Alberta Children's Hospital
Calgary, Alta.

References

1. Ray JG, Vermeulen MJ, Meier C, Cole DEC, Wyatt PR. Maternal ethnicity and risk of neural tube defects: a population-based study. *CMAJ* 2004;171(4):343-5.
2. Lowry RB, Thunem NY, Silver M. Congenital anomalies in American Indians of British Columbia. *Genet Epidemiol* 1986;3:455-67.

DOI:10.1503/cmaj.1041670

[One of the authors responds:]

We appreciate Chris Delaney's points about our study of ethnicity and neural tube defects.[1] First, we did not discuss the lower risk of neural tube defects in the group of 10 009 women categorized as "other" because of the nondescript nature of this category. Second, nondifferential misclassification of exposure (in our case, ethnicity) might be expected to bias the results toward the null. Thus, the observed effect size of the associated risk of neural tube defects among women of First Nations descent was probably an underestimation, not the false-positive result that Delaney contends. Third, we have yet to see someone perform an adjustment for multiple comparisons in a single logistic regression analysis conducted on five levels (in our case, ethnicity). By analogy, if we had examined weight as the exposure, divided into quintiles, with the risk of neural tube defects as the outcome, we would not have adjusted for multiple comparisons as Delaney suggests. The reference that Delaney cites does not support this idea either.[2] Given that neural tube defects are becoming so rare in Canada[3] and that data on maternal ethnicity is not typically recorded in large databases, we are unsure if there will be another opportunity in the near future to address the question of ethnicity and risk of neural tube defects with greater statistical power or accuracy.

Fu-Lin Wang and colleagues correctly suggest that some Ontario First Nations women may not undergo maternal serum screening and are thus underrepresented in our study. They are incorrect, however, in stating that "[f]ailure to include all pregnant First Nations women ... in the denominator for a risk calculation ... could lead to overestimation of the risk for neural tube defects." Rather, our risk estimate was calculated as all women within a given ethnic group whose children had neural tube defects and who underwent maternal serum screening (the numerator) divided by all women within the same ethnic group who underwent maternal serum screening (the denominator), which provides a valid prevalence rate ratio for those women. Because Wang and colleagues' Alberta live-birth data on neural tube defects do not capture the 50% or more of affected pregnancies that end in termination, as they admit, they are much more likely to miss a large number of First Nations women who may undergo termination in the presence of a fetal neural tube defect. For now, our "premature" conclusions are based on the some of the best available data in Canada.

We agree with Vinita Dubey that ethnicity may simply be a confounder of neural tube defects, related to poor folic acid intake. Not only might estimating periconceptional use of folic acid tablets within a maternal serum screening program improve future research, but it could also help to focus on which women are not receiving supplements.[4]

Heather Dean and coauthors are right: all women of reproductive age with type 1 or type 2 diabetes mellitus should be taking a daily folic acid supplement if a future pregnancy — planned or unplanned — is possible. Observational data strongly support both this notion and the value of multidisciplinary preconception care among women with diabetes mellitus,[5] no matter where they live in Canada.

Joel G. Ray
St. Michael's Hospital
University of Toronto
Toronto, Ont.

References

1. Ray JG, Vermeulen MJ, Meier C, Cole DEC, Wyatt PR. Maternal ethnicity and risk of neural tube defects: a population-based study. *CMAJ* 2004;171(4):343-5.
2. Aickin M, Gensler H. Adjusting for multiple testing when reporting research results: the Bonferroni vs Holm methods. *Am J Public Health* 1996;86:726-8.
3. Ray JG. Folic acid food fortification in Canada. *Nutr Rev* 2004;62(6 Pt 2):S35-9.
4. Ray JG, Singh G, Burrows RF. Evidence for suboptimal use of periconceptional folic acid supplements globally. *Br J Obstet Gynaecol* 2004; 111:399-408.
5. Ray JG, O'Brien TE, Chan WS. Preconception care and the risk of congenital anomalies in the offspring of women with diabetes mellitus: a meta-analysis. *Q J Med* 2001;94:435-44.

DOI:10.1503/cmaj.1041541

Does the C in CME stand for "Continuing" or "Commercial"?

The commentaries on commercial sponsorship of continuing medical education (CME) by David Davis[1] and Bernard Marlow[2] contain good recom-