

# Maternal nutrition, pregnancy outcome and public health policy

Michael S. Kramer, MD

**H**ow important is maternal nutrition in determining the outcome of pregnancy? "Very important," most lay persons, prenatal care providers and public health policy-makers would reply. But a careful examination of the available evidence does not strongly support such an answer, at least not in industrialized countries such as Canada.

Before I review the evidence, it is essential to specify what is meant by both "maternal nutrition" and "pregnancy outcome." Maternal nutrition comprises anthropometric factors such as pre-pregnancy weight-for-height (i.e., body mass index [BMI]) and gestational weight gain (which partly reflects the balance between energy intake and energy expenditure, but also includes increases in body water), as well as intake of protein and micronutrients (vitamins and minerals). Of the pregnancy outcomes that might be affected by maternal nutrition, the one encountered most often in the research literature is low birth weight, that is, a birth weight less than 2500 g. Low birth weight is a concept developed and promulgated by epidemiologists and public health practitioners. As far as I can tell, its popularity can be attributed to 2 facts: infant mortality (particularly neonatal mortality) increases exponentially at birth weights below 2500 g,<sup>1</sup> and birth weight (and hence low birth weight) can be measured with excellent validity and precision. Thus, countries or regions where all newborn infants are weighed can be compared, regardless of the availability or validity of gestational age estimates and regardless of whether infant deaths are completely ascertained.

From a clinical, etiologic or prognostic perspective, however, low birth weight is *not* a very useful outcome. Birth weight is a function of 2 factors: duration of gestation and rate of fetal growth. Thus, the weight of newborns can be low either because they are born early (preterm birth) or because they are small for their gestational age or both.<sup>2</sup> I have almost never heard a pediatrician, obstetrician or family physician use the term "low birth weight" in characterizing an individual infant under his or her care. Instead, clinicians use specific terms such as "premature" (or "preterm" or "premie") and "growth-restricted" (or "growth-retarded" or "small-for-dates"). It is now clear that preterm birth and size that is small for gestational age differ in etiology both qualitatively (different etiologic determinants) and quantitatively (different relative risks for common determinants).<sup>2</sup> The 2 outcomes also exhibit vast differences in prognosis. Preterm infants are at increased risk for infant death; short- and long-term pulmonary, ophthalmologic and neurologic morbidity; and delayed psychomotor development.<sup>3-5</sup> Preterm birth is also responsible for high health care costs, particularly for neonatal intensive care, which is often required for many months for infants born extremely prematurely (at less than 32 weeks gestational age).<sup>6</sup> Term infants who are small for their gestational age are at much lower risk for death and short-term morbidity, although recent epidemiologic studies by Barker and colleagues<sup>7,8</sup> suggest that such infants may be at increased risk for type 2 diabetes mellitus, hypertension and coronary artery disease when they reach middle age many decades later. The use of low birth weight as a measure of pregnancy outcome therefore conflates 2 outcomes (shortened gestation and restricted fetal growth) with different causes and vastly different prognoses. In my view, the low-birth-weight concept has been a major hindrance to progress in perinatal epidemiology in general and to understanding the effects of maternal nutrition in particular.

Pre-pregnancy BMI and gestational weight gain both have strong, positive ef-



*Editorial*

*Éditorial*

**Dr. Kramer is with the Departments of Pediatrics and of Epidemiology and Biostatistics, McGill University Faculty of Medicine, Montreal, Que. He is a Distinguished Scientist of the Medical Research Council of Canada.**

CMAJ 1998;159:663-5

‡ See pages 651 and 677 for other articles on antenatal health





fects on fetal growth,<sup>2,9,10</sup> but little if any impact on the duration of gestation.<sup>9-11</sup> Secular (temporal) increases in pre-pregnancy BMI and gestational weight gain (along with a reduction in maternal smoking) are probably responsible for the increase in mean birth weight and the modest decline in low-birth-weight rates observed over the last quarter century.<sup>12,13</sup> Because maternal anthropometry has a much smaller impact on the duration of gestation, however, the beneficial effects of these secular changes on preterm birth, and hence on infant mortality and morbidity, are highly questionable. Thus, despite these changes and the secular decrease in low birth weight in Canada, the number of preterm births in this country has not been reduced.<sup>14</sup> In addition, increases in maternal anthropometric factors carry risks as well as benefits: more is not always better. For example, pre-pregnancy obesity is strongly associated with late fetal death (stillbirth),<sup>9,15</sup> and excessive weight gain increases the risk of fetal macrosomia, cesarean section and maternal weight retention.<sup>9</sup>

Associations between maternal anthropometry and pregnancy outcome are based, by necessity, on observational (i.e., nonexperimental) research designs. What evidence is available from experimental and quasi-experimental studies of actual nutritional intake among pregnant women? The extreme reduction in energy intake imposed by the Germans on the Dutch during the so-called "hunger winter" of 1944/45 led to large reductions in birth weight among the babies of women affected during the third trimester of pregnancy, but no perceptible impact on the duration of gestation or other pregnancy outcomes.<sup>16</sup> Controlled clinical trials of balanced energy-protein supplementation during pregnancy have shown only modest effects on gestational weight gain and fetal growth and few, if any, benefits for other pregnancy outcomes.<sup>17</sup> A recent trial from the Gambia<sup>18</sup> indicated that the effect on fetal growth may be greater in extremely malnourished mothers, but maternal malnutrition to this extent probably does not exist in countries such as Canada.

Many nutritionists, prenatal care providers and public policy-makers would counter that it is the *quality* of the diet, not its *quantity*, that is most important. Thus, it is argued, pregnant women who eat calorically dense "junk food" may gain adequate (or even excessive) amounts of weight during pregnancy but are nevertheless at nutritional risk for adverse pregnancy outcomes. In women with adequate energy intake, however, protein is rarely if ever a limiting nutrient, and high-protein diets may even be detrimental.<sup>17,19</sup> Although the effectiveness of periconceptional folic acid supplementation in reducing neural tube defects is now well established,<sup>20</sup> the evidence from randomized trials of supplementation with nonenergy, nonprotein nutrients such as iron,<sup>21</sup> folic acid (after the

periconceptional period),<sup>22</sup> zinc,<sup>23</sup> calcium,<sup>24,25</sup> and  $\omega$ -3 and  $\omega$ -6 polyunsaturated fatty acids (fish oil)<sup>25,26</sup> have not demonstrated consistent effects on fetal growth, duration of gestation, perinatal mortality or infant morbidity. However, further research on micronutrients is warranted, particularly to identify genetic defects that might put women at risk for adverse effects due to minor micronutrient deficiencies.

Despite the evidence that has been reviewed here, the Canada Prenatal Nutrition Program (CPNP) was established by Health Canada in 1994 with the primary objectives of reducing the incidence of both low birth weight and preterm birth. Eighty-five million dollars was budgeted over a 4-year period. As a socially responsible citizen, I cannot argue with a federal program that provides food for the poor. At the very least, the CPNP provides a method of income redistribution and ensures that the money is spent on nutritious food. But why should such an initiative be undertaken by Health Canada? The evidence was sufficient at the time of the CPNP's inception to warrant extreme scepticism about the chances that this program would improve perinatal health.<sup>17,19</sup> Modelled in large measure on the Women, Infants, and Children (WIC) Program in the United States, the CPNP risks becoming embroiled in the same type of political controversy that has characterized its US counterpart since inception.<sup>27-29</sup> And as with the WIC Program, it will be impossible, despite Health Canada's best efforts, to satisfactorily evaluate the CPNP, because program beneficiaries were not randomly selected for participation and therefore differ from nonparticipants in many unmeasurable ways that confound the effect of the program itself, and because before-and-after trends are confounded by secular trends in pregnancy outcomes and their determinants.

In my view, it would have been much better to emulate the US in a different way. Instead of spending such a large sum of money on a program with so little chance of success, would it not have been preferable to admit what we don't know and spend the money on trying to fill the knowledge gap, that is, on research into the causes of preterm birth? In 1990/91, the US spent almost Can\$40 per capita on its National Institutes of Health (NIH), whereas this country spent less than Can\$9 per capita on the Medical Research Council of Canada (MRC). By 1997/98, the NIH:MRC ratio had nearly doubled, to \$66:\$8 per capita. Moreover, the investigator-initiated research budget of Canada's National Health Research and Development Program has been virtually decimated. The MRC has been given the mandate to conduct population health research but not the budget it would need to do so.

I believe that the faulty logic underlying the CPNP reflects the troubling divergence within Canada between



public health practice and epidemiologic research. Even as efforts succeed to make clinical practice more evidence-based, those setting health care policy have adopted a health promotion ethos that can extend well beyond the limits of, or even ignore, the available evidence. Tension between “evangelists” and “snails”<sup>30</sup> can be a good thing: policy-makers can coax academics out of their ivory towers to address real-world problems and solutions, while academics can apply the brake to the unbridled enthusiasm and inadequately tested interventions of program developers. But if this tension is to be productive and mutually beneficial, the evangelists and the snails must work together. In this regard, Health Canada’s separation of health protection (the “thinkers”) from health promotion (the “doers”) runs the dual risks of thinking without doing and doing without thinking.

As a country, we owe it to future generations to invest far more heavily in research on the causes and prevention of adverse pregnancy outcomes. So as not to appear too self-serving, this epidemiologist believes that improved understanding of the biologic mechanisms underlying the onset of premature uterine contractions and preterm, prelabour rupture of membranes (i.e., fundamental, basic research) would provide a much greater return on Health Canada’s investment than its superficially laudable but fundamentally flawed policy of providing milk, eggs and orange juice to poor pregnant women. Resources are not unlimited, and although it is important to apply what we know, it is equally important not to pretend to know more than we do. The unfortunate legacy of the CPNP will be not only wasted money, but wasted opportunity and disillusionment over hopes that remain unfulfilled.

## References

- Hogue CJR, Buehler JW, Strauss LT, Smith JC. Overview of the National Infant Mortality Surveillance (NIMS) project — design, methods, results. *Public Health Rep* 1987;102:126-38.
- Kramer MS. Determinants of low birth weight: methodological assessment and meta-analysis. *Bull World Health Organ* 1987;65:663-737.
- McCormick MC. The contribution of low birth weight to infant mortality and childhood morbidity. *N Engl J Med* 1985;312:82-90.
- Committee to Study the Prevention of Low Birthweight, Division of Health Promotion and Disease Prevention, Institute of Medicine. *Preventing low birthweight*. Washington (DC): National Academy Press; 1985.
- Dollfus C, Paletta M, Siegel E, Cross AW. Infant mortality: a practical approach to the analysis of the leading causes of death and risk factors. *Pediatrics* 1990;86:176-83.
- Morrison JC. Preterm birth: a puzzle worth solving. *Obstet Gynecol* 1990;76(Suppl):5S-12S.
- Barker DJP. *Fetal and infant origins of adult disease*. London: BMJ Publishing Group; 1992.
- Barker DJP. Fetal origins of coronary heart disease. *BMJ* 1995;311:171-4.
- Subcommittee on Nutritional Status and Weight Gain During Pregnancy, Food and Nutrition Board, US Institute of Medicine/National Academy of Sciences. *Nutrition during pregnancy*. Washington (DC): National Academy Press; 1990.
- Maternal anthropometry and pregnancy outcomes: a WHO collaborative study. *Bull World Health Organ* 1995;73(Suppl):1-98.
- Carmichael SL, Abrams B. A critical review of the relationship between gestational weight gain and preterm delivery. *Obstet Gynecol* 1997;89:865-73.
- Kramer MS. Birth weight and infant mortality: perceptions and pitfalls. *Paediatr Perinat Epidemiol* 1990;4:381-90.
- Kramer MS. Preventing preterm birth: Are we making any progress? *Yale J Biol Med* 1997;70:227-32.
- Joseph KS, Kramer MS. Recent trends in infant mortality rates and proportions of low-birth-weight live births in Canada. *CMAJ* 1997;157(5):535-41.
- Cnattingius S, Bergström R, Lipworth L, Kramer MS. Prepregnancy weight and the risk of adverse pregnancy outcomes. *N Engl J Med* 1998;338:147-52.
- Stein Z, Susser M, Saenger G, Marolla F. *Famine and human development: the Dutch hunger winter of 1944-45*. New York: Oxford University Press; 1975.
- Kramer MS. The effects of energy and protein intake on pregnancy outcome: an overview of the research evidence from controlled clinical trials. *Am J Clin Nutr* 1993;58:627-35.
- Ceesay SM, Prentice AM, Cole TJ, Foord F, Weaver LT, Poskitt EME, et al. Effects on birth weight and perinatal mortality of maternal dietary supplements in rural Gambia: 5 year randomised controlled trial. *BMJ* 1997;315:786-90.
- Rush D. Effects of changes in protein and calorie intake during pregnancy on the growth of the human fetus. In: Chalmers I, Enkin M, Keirse MJNC, editors. *Effective care in pregnancy and childbirth*. New York: Oxford University Press; 1989. p. 255-80.
- MRC Vitamin Study Research Group. Prevention of neural tube defects: results of the Medical Research Council Vitamin Study. *Lancet* 1991;338:131-7.
- Mahomed K. Routine iron supplementation during pregnancy [Cochrane review]. In: Neilson JP, Crowther CA, Hodnett ED, Hofmeyr GJ, Keirse MJNC, editors. *Pregnancy and childbirth module of the Cochrane Database of Systematic Reviews*. In: The Cochrane Library, Issue 2, 1998. Oxford: Update Software.
- Mahomed K. Routine folate supplementation in pregnancy [Cochrane review]. In: Neilson JP, Crowther CA, Hodnett ED, Hofmeyr GJ, editors. *Pregnancy and childbirth module of the Cochrane Database of Systematic Reviews*. In: The Cochrane Library, Issue 2, 1998. Oxford: Update Software.
- Mahomed K. Zinc supplementation in pregnancy [Cochrane review]. In: Neilson JP, Crowther CA, Hodnett ED, Hofmeyr GJ, editors. *Pregnancy and childbirth module of the Cochrane Database of Systematic Reviews*. In: The Cochrane Library, Issue 2, 1998. Oxford: Update Software.
- Levine RJ, Hauth JC, Curet LB, Sibai BM, Catalano PM, Morris CD, et al. Trial of calcium to prevent preeclampsia. *N Engl J Med* 1997;337:69-76.
- Kesmodel U, Olsen SF, Salvig JD. Marine n-3 fatty acid and calcium intake in relation to pregnancy induced hypertension, intrauterine growth retardation, and preterm delivery. *Acta Obstet Gynecol Scand* 1997;76:38-44.
- Olsen SF, Hansen HS, Secher NJ, Jensen B, Sandström B. Gestation length and birth weight in relation to intake of marine n-3 fatty acids. *Br J Nutr* 1995;73:397-404.
- Rush D, Leighton J, Sloan N, Alvir J, Garbowshi G. Review of past studies of WIC. *Am J Clin Nutr* 1988;48:394-411.
- Alexander G, Weiss J, Hulsev T, Papiernik E. Preterm birth prevention: an evaluation of programs in the United States. *Birth* 1991;18:160-9.
- Brown JL, Gershoff SN, Cook JT. The politics of hunger: when science and ideology clash. *Int J Health Serv* 1992;22:221-37.
- Sackett DL, Holland WW. Controversy in the detection of disease. *Lancet* 1975;2:170-2.

**Correspondence to:** Dr. Michael S. Kramer, 1020 Pine Ave. W, Montreal QC H3A 1A2