How 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,¹ 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.² 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-re 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).² 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.³-⁴ 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).⁶ 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 colleagues⁷,⁸ 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-
flects on fetal growth, but little if any impact on the duration of gestation. 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. 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. 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) and excessive weight gain increases the risk of fetal macrosomia, cesarean section and maternal weight retention. 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. 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. A recent trial from the Gambia 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. Although the effectiveness of periconceptional folic acid supplementation in reducing neural tube defects is now well established, the evidence from randomized trials of supplementation with nonenergy, nonprotein nutrients such as iron, folic acid (after the periconceptional period), zinc, calcium and ω-3 and ω-6 polyunsaturated fatty acids (fish oil) 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. 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.

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 its National Institutes of Health (NIH). 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” 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


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