

Antioxidant vitamin and B vitamin supplements have been extensively studied for a presumed beneficial effect in the prevention of clinical cardiovascular events in large, high-quality, randomized controlled trials, with disappointing results. In view of the randomized evidence, the epidemiologic evidence discussed by Hathcock cannot be used as grounds for supporting the use of these vitamin supplements to prevent cardiovascular disease. Therefore, there is currently no sound evidence to support the use of antioxidant vitamin and B vitamin supplements to prevent clinical cardiovascular events or the progression of atherosclerosis.

None of the authors had a conflict of interest.

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human milk exerts antiinflammatory effects and still exerts significant protective action against infections in breastfed infants. In addition, human breast milk is a good source of polyunsaturated fatty acids (PUFAs), especially of  $\gamma$ -linolenic acid (GLA), arachidonic acid (AA), eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA) (7).

Breastfed infants have a significantly higher percentage of DHA and other PUFAs in muscle phospholipids than do nonbreastfed infants. Higher PUFA concentrations in the skeletal muscle membrane are associated with lower fasting plasma glucose concentrations (8), whereas low concentrations of DHA and other PUFAs can result in insulin resistance. We showed that prior oral administration of pure GLA, AA, EPA, and DHA (which are present in human breast milk) prevents alloxan-induced diabetes mellitus by protecting pancreatic  $\beta$  cells from the apoptotic actions of alloxan (9, 10) supports the conclusions made by Owen et al (1).

Early nutrition is an important environmental signal that can induce lifetime effects on metabolism, growth, and neurodevelopment and on major disease processes, such as diabetes mellitus (11). It is likely that breastfeeding ensures adequate nutrition and PUFAs that are essential for brain growth and development (7, 12). Recent studies indicate a significant role for brain insulin receptors in the control of insulin secretion and carbohydrate metabolism (12). It is likely that breastfeeding ensures an adequate supply of PUFAs, which, in turn, will lead not only to the growth and development of brain but also to adequate numbers of insulin receptors in the brain to maintain normal glucose metabolism (13). In view of this evidence, it will be interesting to study whether perinatal supplementation of PUFAs could prevent or postpone the development of diabetes mellitus in high-risk subjects (14, 15).

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### Breastfeeding prevents type 2 diabetes mellitus: but, how and why?

Dear Sir:

The conclusion of the recent systemic review of the published studies that breastfeeding in infancy is associated with a reduced risk of type 2 diabetes, with marginally lower insulin concentrations in later life, and with lower blood glucose and serum insulin concentrations in infancy (1) is interesting.

Human milk contains tumor necrosis factor  $\alpha$  (TNF- $\alpha$ ), interleukin (IL)-1, IL-6, transforming growth factor  $\beta$ 1 and  $\beta$ 2 (TGF- $\beta$ 1 and TGF- $\beta$ 2), chemokine growth-related oncogene protein  $\alpha$ , monocyte chemoattractant protein-1, IL-8, IL-1 receptor antagonist, soluble forms of the receptors for TNF- $\alpha$ , the antiinflammatory cytokine IL-10, and RANTES (regulated upon activation, normal T cell expressed, and secreted) (2–5). Lysozyme present in human milk suppresses chemotaxis and respiratory burst activity in human polymorphonuclear leukocytes (6). The presence of an ascorbate-like material, uric acid,  $\alpha$ -tocopherol, and  $\beta$ -carotene in human milk ensures that phagocyte-produced oxidant molecules cannot persist, and this contributes to the antiinflammatory effects of milk. Thus,



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### Poverty threshold as an indicator of the association between childhood overweight and socioeconomic status over time

Dear Sir:

I read with interest the recent Journal article by Wang and Zhang (1) on changes in the association between overweight and family income in children and adolescents using data from the National Health and Nutrition Examination Survey (NHANES) between 1971 and 2002. The authors are to be commended on their ambitious undertaking of examining secular trends in the relation between overweight and socioeconomic status (SES); such findings may have important insights for developing effective obesity prevention and management programs as well as public policies. Wang and Zhang point out that each of the commonly used variables of SES—including education, income, and occupational status—has its own strengths and limitations for studying the relation between SES and health outcomes. In their study, SES groups were determined by the poverty income ratio tertiles at each survey timepoint (NHANES I, 1971–1975; NHANES II, 1976–1980; NHANES III, 1988–1994; and NHANES 1999–2002), and then low-, middle-, and high-SES groups were compared on the basis of these tertiles. It is important to address some potential limitations to this approach.

The US Census Bureau's poverty threshold is an "absolute" threshold, fixed at a point in time (1963) and updated solely for price changes (2). As such, it does not reflect changes in standard of living, taxes, or other factors affecting SES over time. In addition, the poverty threshold is constant across the United States, and no allowances are made for dissimilar costs of living in different geographic areas. Thus, a child living in the rural Midwest with a family income of \$40 000 would be in the same SES category as a child living in a coastal metropolis on the same \$40 000 income. Furthermore, the poverty threshold does not account for family expenses, such as childcare costs for working and nonworking families; therefore, a working family spending 10% of their income on childcare would be in the same SES category as a nonworking family making the same income but without the extra expense. These concerns are important not only for determining the SES grouping of children in each individual NHANES cohort, but also for understanding the possible differing SES classifications across timepoints that result from temporal changes in the factors affecting the poverty threshold.

Wang and Zhang found in their analyses that, contrary to widely held perceptions, not all low-SES groups were at increased risk of overweight; they also found that, over the past 3 decades, there has been an overall weakening association between SES and overweight, especially in the later waves of NHANES (1988–1994 and 1999–2002). Given the above concerns regarding the definition of SES, their results should be interpreted cautiously. An alternative explanation for the findings could be that the poverty threshold was

a more accurate indicator of SES in the earlier cohorts, whereas, for the later groups, nationwide changes such as increases in the numbers of working or single-parent families, unequal geographic changes in the cost of living, or greater consumerism (or all 3 circumstances) make the poverty threshold a less reliable SES measure. These variables may also affect different races to greater or lesser degrees. Thus, the apparently changing associations over time among overweight, race, and SES could in fact reflect the comparison of unequal SES groups.

Despite these concerns over the authors' choice of SES indicator, their study shows the real and significant increase in child and adolescent overweight spanning the last 3 decades and highlights the urgent need for further research into the causes and potential prevention and management efforts required to fight the childhood obesity epidemic.

The author had no personal or financial conflict of interest with the study by Wang and Zhang or with those authors themselves.

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### Reply to M Bishop

Dear Sir:

We appreciate Dr Bishop's interest in and comments on our recent study (1), and we share her viewpoint that readers should be aware of the potential limitations of using poverty income ratio (PIR) as an indicator of socioeconomic status (SES), as well as of the limitations of other variables, such as education and occupation. As stated in our study, each of the commonly used variables of SES has its own strengths and limitations for studying the relation between SES and health outcomes. We agree that readers need to know how the PIR was constructed. It is the ratio of income to the family's appropriate poverty threshold set by the US Census Bureau in a given calendar year. The Census Bureau, working in accordance with the Statistical Policy Directive of the Office of Management and Budget, uses a set of money income thresholds that vary by family size and composition to determine who is poor. Thresholds are updated annually for inflation by using the Consumer Price Index. The official poverty definition counts pretax money income and excludes capital gains and noncash benefits such as public housing, Medicaid, and food stamps (2). PIR values for the National Health and Nutrition Examination Survey (NHANES) participants were computed by the National Center for Health Statistics by using family income data (3, 4). As pointed out by Bishop, a major limitation of the PIR is that the