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Summary of the Presentations at the Conference on Preventing Childhood Obesity, December 8, 2003

Sally Ann Lederman, PhD*; Sharon R. Akabas, PhD‡; Barbara J. Moore, PhD§ with the collaboration of Margaret E. Bentley, PhD; Barbara Devaney, PhD; Matthew W. Gillman, MD; Michael S. Kramer, MD; Julie A. Mennella, PhD; Andrew Ness, PhD; and Jane Wardle, PhD

ABSTRACT. *Objective.* Because of the rising rates of childhood obesity, we set out to determine what is known about its causes and what could be done to prevent additional increases.

Methodology. A meeting was convened of experts in areas that bear on prevention of obesity development during intrauterine life, infancy, and very early childhood. They presented recent data and their interpretations of the stage of our current knowledge in related areas. They also proposed possible useful interventions and future directions for research.

Findings. The speakers' talks indicated that (1) breast-feeding as currently practiced seems to be significantly (albeit weakly) protective against obesity and should be encouraged as the preferred method of feeding infants for as long a duration as practical during the first year of life; (2) infant-feeding practices are changing in a way that may predispose to obesity (eg, soda and french fries are being fed to infants as young as 7 months of age), possibly altering taste preferences for foods and beverages that are energy dense and nutrient poor; (3) although little is known about parenting styles (eg, authoritative versus permissive), parenting style is likely to be a fruitful area of current research into childhood obesity etiology; and (4) the pattern of weight changes in the first few years of life may contribute to later risk of obesity.

Conclusions. Children's obesity will continue to be a growing problem unless we improve understanding of the key factors likely to be operative during intrauterine life, infancy, and very early childhood, identify those in whom intervention would have the greatest effect, design and evaluate preventive interventions, and promote those that are successful. *Pediatrics* 2004;114:1146-1173; *obesity, pediatrics, prevention, infant feeding, birth weight, activity, dietary choices, maternal weight, feeding styles.*

ABBREVIATIONS. IOM, Institute of Medicine; SES, socioeconomic status; BMI, body mass index; NCHS, National Center for Health Statistics; LGA, large for gestational age; SGA, small for gestational age; NHANES, National Health and Nutrition Examination Survey; WIC, Special Supplemental Nutrition Program for Women, Infants, and Children; OR, odds ratio; CI, confidence interval; FITS, Feeding Infants and Toddlers Study; IFSQ, Infant

Feeding Style Questionnaire; ALSPAC, Avon Longitudinal Study of Parents and Children.

In late 2001, Congress allocated monies to fund a study to be conducted by the Institute of Medicine (IOM), to develop an action plan to prevent obesity in children and youth. According to Senate Report 107-84, the study "should assess the primary factors responsible for the increasing prevalence of childhood obesity and identify the most promising methods for prevention."^{1(p92)} Accordingly, the IOM appointed a Committee on Prevention of Obesity in Children and Youth, which is charged with assessing the nature of obesity among children and youths in the United States and developing a prevention-oriented action plan to reduce its prevalence.

In February 2003, Dr B. J. Moore was appointed to the IOM committee, with 17 other individuals from throughout the United States. In August and September 2003, as a result of informal discussions among committee members, the idea was born of developing a conference to focus on very early critical periods of development that might predispose individuals to obesity. Dr Moore secured the necessary funding for the conference through her affiliation with Shape Up America!, a nonprofit 501(c)3 educational organization. The Gerber Product Company agreed to provide funding for the conference venue, food, audiovisual support, speaker travel, and honoraria. Although it was not an IOM event, this Shape Up America! conference was designed in consultation with the members of the IOM committee.

The focus of the conference was on the earliest critical periods considered important for the development of childhood obesity, namely, conception, intrauterine life, infancy, the postweaning period, and the preschool period. Certain members of the committee were particularly generous with their time and ideas in identifying topics and speakers. In that regard, we wish to acknowledge the contributions of Dr Robert Whitaker of Mathematica Policy Research, Dr Leann Birch of Pennsylvania State University, Dr Tom Robinson of Stanford University, Dr Shiriki Kumanyika of the University of Pennsylvania, Dr Dennis Bier of the Baylor College of Medicine, Dr Russell Pate of the University of South Carolina, and Dr Ross Brownson of St. Louis University.

Dr Sally Lederman was selected by Dr Moore be-

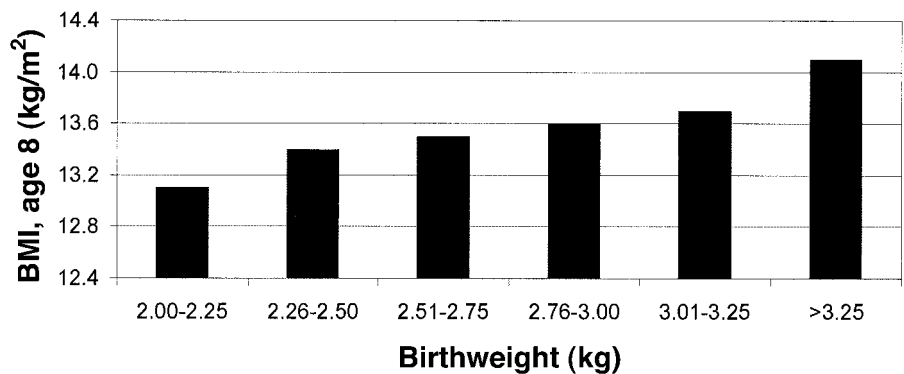
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Fig 1. Direct association of birth weight with child BMI among 8-year-old Indian children.⁶



cause of her expertise in the fields of nutrition, obesity, growth, pregnancy, and lactation. She directed the development of the conference summary and drafted the editors' overview, in collaboration with Dr Moore and Dr Sharon Akabas, who was selected as coauthor because of her background in nutrition education and her interest in the role of activity in preventing obesity among children.

The conference was held at the Marriott Metro Center Hotel in Washington, DC, on December 8, 2003. It included research-based presentations by 7 session speakers, Dr M. Gillman, Dr M. Kramer, Dr B. Devaney, Dr J. Mennella, Dr J. Wardle, Dr M. Bentley, and Dr A. Ness. In their invitations to speak at the conference, the speakers were asked to address, to the extent possible, a set of questions whose answers might guide future approaches to preventing childhood obesity and inform subsequent research efforts. Different speakers undertook to answer different selections of the questions posed, on the basis of their expertise and research findings, and focused on different broad areas, as indicated by the session titles in this summary. Several speakers included materials from the peer-reviewed literature and used that information to bridge gaps or suggest relationships that could be helpful in decision-making. New research findings, both their own and those of others, obtained through personal communications, were also included, which made for a stimulating, exciting, and challenging conference. The conference summary presented here is a detailed summary of the presentations, derived from a transcript of the proceedings. Each speaker reviewed the summary of his or her presentation for accuracy.

OBESITY ORIGINS IN FETAL DEVELOPMENT AND THE FIRST 6 MONTHS OF LIFE

Matthew Gillman, MD, Harvard Medical School, Boston, Massachusetts

During the course of life, exposures that determine obesity may be attributable to environmental, social, behavioral, or biological (including genetic) factors. The life course approach to chronic disease focuses on the fact that such exposures may occur at many stages of life, from preconception through fetal life, infancy, childhood, adolescence, and beyond. The exposures act in concert over time. At least 2 causal models can be considered, ie, the critical- or sensitive-period model, in which a specific exposure may

need to act at a particular time to have its effect, with little or no effect at other times, and the accumulation-of-risk model, which suggests that the effect of a given factor or exposure may increase with increasing duration of exposure.

Many factors, operating from preconception through childhood and adolescence, may affect obesity risk. Maternal prepregnancy body mass index (BMI) may determine pregnancy glucose and insulin levels in the mother and fetus, with high levels increasing newborn weight. Postnatally, the feeding of the newborn, infant, and child can determine the rate of growth and influence the timing and magnitude of the adiposity rebound seen in childhood, with subsequent dietary and activity patterns contributing to later BMI, adiposity, and a fat distribution characterized by central obesity. Such factors then contribute to morbid outcomes, including insulin resistance, cardiovascular disease, and type 2 diabetes mellitus.

The increasing importance of obesity development among children was illustrated with data on the changing prevalence of obesity among children 6 to 11 and 12 to 19 years of age.^{2,3} The data showed that, between 1976–1980 and 1999–2000, obesity rates more than doubled among children 6 to 11 years of age and more than tripled among those 12 to 19 years of age, although there had been little change in the prevalence between 1963 and 1976. Additional data from Kim et al⁴ were used to develop a linear model of weight changes among children 0 to 71 months of age, controlling for age, gender, race/ethnicity, Medicaid status, and clinic site. The data showed an 83% increase in overweight (>95th percentile) and a 27% increase in the risk of overweight (>85th to 95th percentile). Data reported by Mokdad et al⁵ showed changes in obesity prevalence according to state during the past decade. In 1991, only 5 states exhibited obesity prevalences of >15%; by 2001, all states exceeded this value and most demonstrated obesity prevalences of 20% to 24%, with Mississippi having even higher levels.

Dr Gillman then addressed the following question: Does obesity begin in the womb? To explore this question, >20 studies that considered birth weight and later BMI, taken from various sources, were reviewed. These studies were limited in being mostly from Europe, North America, and Australia, rarely having data on gestational age, socioeconomic status (SES), or parental height or weight, and mostly cov-

TABLE 1. Birth Weight Association With Adolescent Overweight Among >14 000 Boys and Girls, 9 to 14 Years of Age⁷

Model Covariates	OR per 1-kg Increase in Birth Weight	95% CI
Tanner stage	1.4	1.2, 1.6
+ Television, physical activity, and energy intake	1.5	1.3, 1.7
+ Multiple covariates*	1.4	1.2, 1.7
+ Mother's current BMI	1.3	1.1, 1.5

* Infant feeding, birth order, household income, and mother's dietary restraint, weight cycling, and smoking.

ering childhood, with only a few covering adulthood. The findings were of interest, however, because almost all showed a direct relationship between birth weight and later BMI and none showed an inverse relationship. The few with null findings were smaller studies that might have lacked the power needed.

Dr Gillman presented several examples of such data, examining the relationship of birth weight to later BMI or obesity development over a broad age range. Data reported by Bavdekar et al⁶ related birth weight, in 250-g groupings between 2 kg and >3.25 kg, to BMI at 8 years of age (Fig 1). Mean BMI increased linearly from ~13 to 14.1 across these birth-weight groups. Gillman et al,⁷ in a study of >14 000 children 9 to 14 years of age (the Growing Up Today Study), showed an increase in the odds of adolescent overweight of 30% to 50%, depending on the factors controlled, with a 1-kg increase in birth weight (3-5% increase in the odds ratio [OR] per 100-g increase in birth weight) (Table 1). Data reported by Sorenson et al,⁸ which adjusted for gestational age, birth length, and maternal factors, showed a similar linear increase in BMI among Danish conscripts at 18 to 26 years of age, as their birth weight, grouped in 500-g categories, increased from <2.5 kg to >4.5 kg (Fig 2). The largest increase in BMI was among those who exceeded 4.5 kg at birth. Mean BMI ranged from ~22.7 in the lowest-birth-weight group to ~24.8 in the highest-birth-weight group.

What mechanisms can be proposed for the observed relationship between birth weight and later BMI? First, potential confounders should be considered. For example, this BMI-birth weight relationship may be determined at least in part by differences in

lean mass, rather than fat mass, at birth, differences that may persist into adulthood. Some of the association may be determined by the prenatal and postnatal environments, which may determine differences in birth weight as well as subsequent BMI. In addition, genes that cause birth-weight differences may also determine BMI differences.

Independent of the potential contributions of such factors, direct effects may result from alterations in the fetal environment, including the transfer of fatty acids, leptin, and other hormones, fetal hyperinsulinemia, and the functioning of the fetal/placental unit. Supporting this view are data showing the growth patterns of children of diabetic mothers from birth to 8 years of age.⁹ These children had a higher weight for length at birth and at every year after age 1, relative to a National Center for Health Statistics (NCHS) reference. Data from a later report¹⁰ showed that this higher weight persisted through 14 to 17 years of age.

A within-family study indicated that family genetic factors and environment did not account for the relationship between birth weight and later weight among infants of diabetic mothers (Fig 3).¹¹ The authors studied Pima Indian siblings in cases in which only 1 sibling was exposed to maternal diabetes mellitus in utero. Individuals exposed in utero exhibited BMI values ~5 units higher than those of the unexposed siblings, measured either in late childhood or through age 24. A recent report,⁷ based on data from the Growing Up Today Study, examined obesity risk among ~14 000 children according to maternal diabetes status during pregnancy (Table 2). The results showed a weaker association between gestational diabetes and offspring obesity, perhaps because of less severe gestational diabetes or better diabetes treatment.

A model drawn from animal studies suggests that experimentally induced maternal gestational diabetes may result in fetal hyperinsulinemia, which in turn increases hypothalamic insulin levels and alters the fetal hypothalamus,^{12,13} increasing neuropeptide Y neurons.^{12,14} In later life, this perturbation results in hyperphagia, hyperinsulinemia, impaired glucose tolerance, and overweight.¹³ If applicable to human subjects, this model would suggest a potential for intergenerational nongenetic transmission of impaired glucose tolerance and overweight.

Because birth weight may be related to later obe-

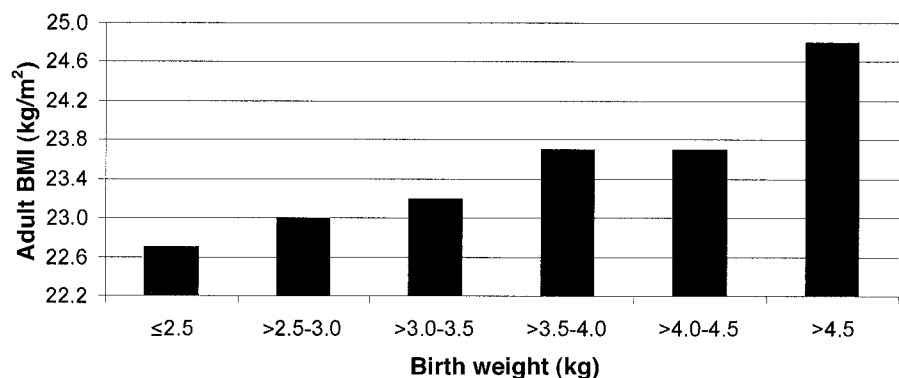


Fig 2. Direct association of birth weight with young adult BMI among 20- to 26-year-old Danish conscripts, with adjustment for gestational age, birth length, and maternal factors.⁸

Fig 3. Mean BMI of siblings from 19 Pima Indian families exposed to diabetes in utero, compared with siblings not exposed in utero (58 siblings).¹¹

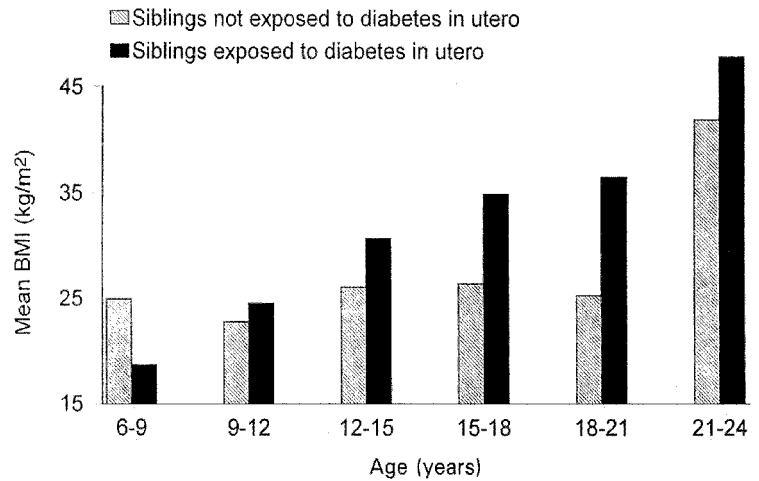


TABLE 2. Relationship of Maternal Gestational Diabetes to Obesity Among >14 000 Boys and Girls, 9 to 14 Years of Age⁷

Model Covariates	OR per 1-kg Increase in Birth Weight	95% CI
Tanner stage	1.4	1.1–2.0
+ Television, physical activity, energy intake, and other covariates*	1.4	1.0–2.0
+ Birth weight	1.3	0.9–1.9
+ Mother's current BMI	1.2	0.8–1.7

* Infant feeding, birth order, household income, and mother's dietary restraint, weight cycling, and smoking.

sity risk, it is relevant that the proportion of infants born large for gestational age (LGA; >90th percentile of weight for gestational age) in the United States has increased since 1990, particularly among whites (E. Oken, MD, verbal communication, 2003). Changes in the prevalence of factors that influence birth weight might have contributed to this increase. For example, the rate of diabetes mellitus during pregnancy increased from 2.2% to 3.2%, the rates of maternal smoking and adolescent pregnancy decreased, and the level of maternal education and first-trimester prenatal care increased. Changes in maternal BMI were not available, but increases in BMI could also increase birth weight. However, increases in birth weight resulting from these factors might be attenuated by other factors that tend to reduce birth weight, including decreased rates of postterm delivery because of more frequent labor induction, increased maternal age and rates of hypertension, increased numbers of births to unwed mothers, and increased numbers of preterm deliveries because of multiple births. (Dr Kramer later presented an analysis of data

from 1 hospital that estimated the magnitude of the effects, in a Canadian population, of several factors known to affect birth weight.)

In addition to the effects of high birth weight on later BMI, the effects of low birth weight warrant review. Of particular concern is the effect of restricted fetal growth on central obesity. Central obesity, assessed as the waist-to-hip ratio or as skinfold measurements, is associated with insulin resistance and the metabolic syndrome, which is manifested as hypertension, hypertriglyceridemia, and glucose intolerance or frank type 2 diabetes, with an increased risk of cardiovascular disease. Several studies showed an inverse relationship of birth weight to central obesity, adjusted for attained BMI (Table 3),^{6,15–17} as well as to various measures of insulin resistance (Table 4).^{6,18,19} Two studies^{17,20} reported an inverse relationship between birth weight and measures of the insulin resistance syndrome, also with adjustment for attained BMI. Data from the Nurses' Health Study^{21–23} showed linear decreases in the risks of coronary heart disease, stroke, hypertension, and type 2 diabetes as birth weight increased from <5 to >10 lb, with adjustment for attained BMI (Fig 4). These various results, with adjustment for attained BMI, indicate that, at any given adult BMI, individuals with higher birth weights have lower risks of these outcomes, compared with those with lower birth weights.

We need larger studies of the effects of gestational diabetes mellitus on fetal growth; these studies should include less severe forms of glucose intolerance. We need to move beyond birth weight, which is only a momentary marker, a proxy for many determinants that operate preconceptionally and pre-

TABLE 3. Inverse Association of Birth Weight With Measures of Central Obesity

Variable	Estimate*	95% CI
Subscapular/triceps skinfold measurements, per kg birth weight ¹⁵	–6%	0, 12
Log subscapular/triceps skinfold measurements, per kg birth weight ⁶	–4.8	0.7, 8.7
Waist/hip ratio, per kg birth weight ¹⁶	–1.2%	0, 2.5
Waist/hip ratio, per kg birth weight ¹⁷	–0.8%	NA

* Adjusted for attained BMI. NA indicates not available.

TABLE 4. Association of Birth Weight With Measures of Insulin Resistance

Variable	Estimate, per kg birth weight*	95% CI
Insulin sensitivity ¹⁸	0.7 units	0.1, 1.2
Fasting glucose ¹⁹	-2.4%	0.1, 4.7
2-h glucose ¹⁹	-5.1%	0.7, 9.3
Fasting insulin ¹⁹	-9.7%	2.5, 16.5
2-h insulin ¹⁹	-14.0%	2.4, 24.2
Log homeostatic model assessment score (HOMA), insulin resistance ⁶	-9.7	-0.2, 18.6

* Adjusted for attained BMI.

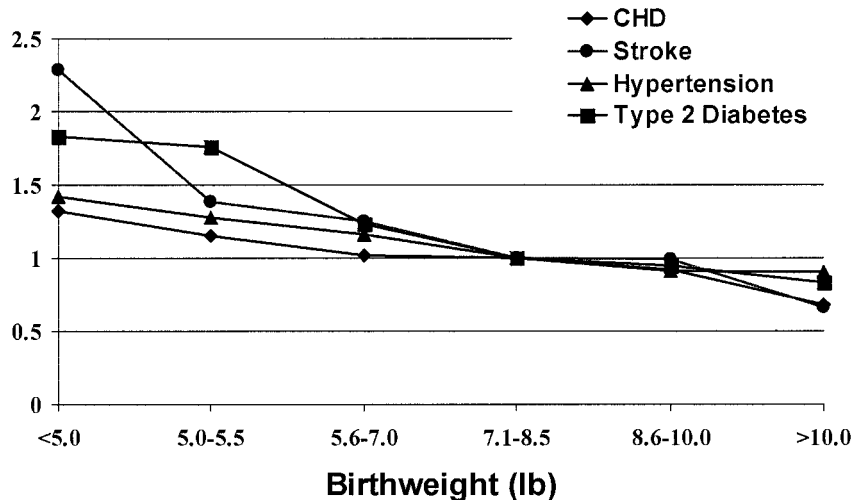


Fig 4. Inverse association of birth weight with cardiovascular disease outcomes in the Nurses' Health Study. Values were adjusted for attained BMI. CHD indicates coronary heart disease.²¹⁻²³

nately. For example, several reports have shown that maternal smoking can be related to an increase in obesity in the offspring, despite its relationship to reduced birth weight.²⁴⁻²⁶ The relative contributions of prenatal and postnatal influences to body mass, central obesity, and both fat and lean masses require elucidation. Data suggest that catch-up growth in infancy among individuals born small may be related to later obesity and other morbid outcomes. Interdisciplinary studies that examine the interplay of genes and environmental factors are needed.

What are the implications for pregnancy interventions of the findings on the effects of intrauterine life? Although there are valid epidemiologic links between birth weight and adult health, clear etiologic factors have not been identified or quantified. Because lower birth weight is related to worse cardiovascular outcomes, some workers have recommended increasing efforts to raise birth weights, even in the developed world. However, Dr Gillman cautioned that such efforts could be harmful because of their possible effects in increasing obesity, which could increase the risk of type 2 diabetes and other diseases.

In a study of insulin resistance at 8 years of age among children in India, birth weight and insulin resistance (on HOMA, the homeostatic model assessment scale) were divided into tertiles.⁶ The highest risk for insulin resistance was among individuals in the lowest birth-weight tertile who were in the highest weight tertile at age 8. In a study of adolescents from the Philippines, blood pressure increased with increased BMI tertile but decreased across birth-weight tertiles.²⁷ That is, with division of birth

weight and adolescent blood pressure into 3 groups, blood pressure was highest for individuals born at the lowest weights and attaining the highest weights. Within the group with highest adolescent BMI values, higher birth weight was protective. Similarly, among white and Mexican American adults, those who were in the highest birth-weight tertile and remained thin showed no insulin resistance syndrome, whereas those who were born small and were in the highest BMI tertile in adulthood demonstrated a 25% prevalence of insulin resistance syndrome, the highest rate.²⁸ It is clear that high adult BMI increases the risk at all birth weights, but the risk in all BMI tertiles is lowest for those with higher birth weight. In a cohort of 1256 Welsh men 45 to 59 years of age, the expected inverse relationship between birth weight and coronary disease was observed, with the lowest birth weight being related to more coronary disease than the highest birth weight.²⁹ High birth weight was protective; all of the risk seemed to be related to having a high BMI in adulthood, with birth weight in the lower 2 tertiles.

Studies in rats suggest that prenatal conditions may determine postnatal behaviors that influence body weight, suggesting a prenatal origin for the "couch potato" syndrome.³⁰ Rat mothers were either fed normally or restricted in energy during pregnancy. The pups were cross-fostered after birth, to either a normal-nutrition condition or overnutrition induced by a high-fat diet. This model provided adult rats with only prenatal undernutrition or undernutrition followed by postnatal overnutrition. Measurements showed increased energy intake and decreased physical activity (monitored as distance

traveled) in the postnatally overnourished groups, compared with the normally nourished comparison groups, whether they were well nourished or energy-restricted during pregnancy. Increased mass of the retroperitoneal fat pad in the postnatally overnourished rats indicated the development of a relative central adiposity; this was accompanied by a decrease in total muscle mass, with an increase in systolic BP and insulin resistance. Postnatal overnutrition increased BP, compared with a normal state, but the effect was greatest when combined with prenatal undernutrition. These findings suggest that fetuses in a growth-restricted environment are not prepared to live with a surfeit of energy postnatally. The combination of restricted fetal growth and excess energy postnatally yields the worst outcomes.

We have noted that the interaction of lower birth weight with higher adult BMI yields the highest risk for cardiovascular disease outcomes. This combination of conditions is characteristic of the developing world undergoing the epidemiologic nutritional transition. In South Korea between 1938 and the middle 1990s, for example, the rates of deaths resulting from circulatory system diseases and malignancies increased from ~1% to 28% and 22%, respectively, whereas the rate of deaths resulting from infections and parasitic diseases decreased from ~18.5% to ~2.4% and that of deaths resulting from respiratory system diseases decreased from 22% to 5%.³¹ In such settings, preventing the development of obesity is increasingly important.

The early postnatal level of nutrition appears able to influence later obesity. Therefore, it is logical to consider whether breastfeeding plays a role in preventing later obesity. Possible mechanisms include metabolic programming from the breast milk itself. Earlier studies suggested that breast milk and formula produce different insulin responses.^{32,33} Newer data suggest that there is a difference in leptin concentrations among breastfed versus bottle-fed infants.^{34,35} Another possibility is that breastfeeding leads to more internal control of energy intake by the child, whereas there might be more parental control over formula feeding. Whatever the relationship of feeding method to outcomes, there are problems in assuming that the observed associations are causal, because the social and cultural determinants of breastfeeding may also be related to later development of obesity.

Dr Gillman presented data he had collected from a

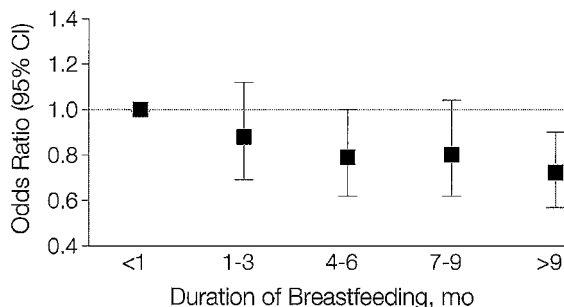


Fig 5. Risk of overweight in adolescence according to duration of breastfeeding in infancy.³⁶

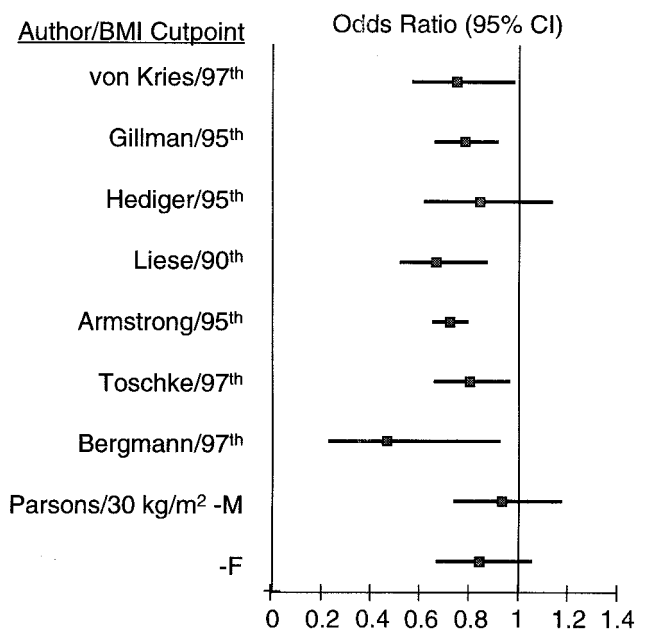


Fig 6. Breastfeeding, considered dichotomously (yes or no), and the OR for later obesity.

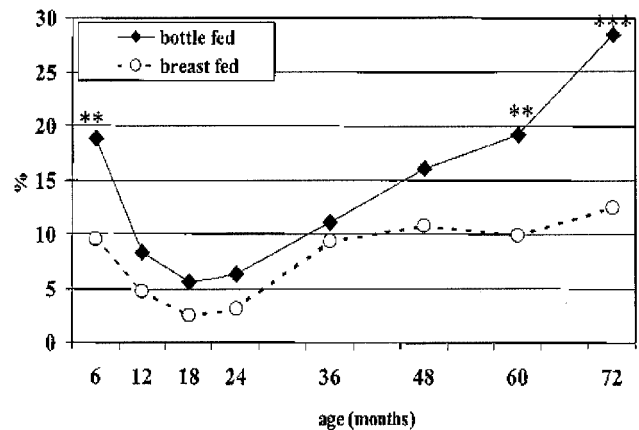


Fig 7. Prevalence of overweight (BMI in >90th percentile) among breastfed versus bottle-fed infants.⁴⁰ ** indicates $P = .01$; ***, $P = .001$.

brief literature review of reports since 1999 that dealt with breastfeeding and later overweight. All except 1 of the studies were cross-sectional and included populations of >2000 subjects; most were from the developed world. The collated results suggested a possible effect of breastfeeding duration, but results were mixed. The study by Gillman et al³⁶ showed that the risk of overweight in adolescence declined monotonically with increased duration of breastfeeding in infancy (Fig 5). In the combined set of studies, when breastfeeding was considered dichotomously (yes or no), there was a consistent mean reduction in the prevalence of later obesity (variously defined), although the finding was not always statistically significant (Fig 6).^{25,26,36-41} For example, Bergmann et al⁴⁰ examined children 9 times between birth and 6 years of age and compared the findings for subjects who were breastfed for >3 months or <3 months. In both BMI and triceps skinfold measurements, breastfed infants began diverging from bottle-fed infants at

3 to 4 years of age, with, for example, a smaller proportion above the 90th percentile of BMI (Fig 7).

Although these studies suggest protection, they do not take into account residual confounding by shared cultural determinants of both breastfeeding and obesity. To do that, it would be necessary to measure the confounders very carefully, which is difficult. Randomized trials of breastfeeding cannot be performed on an individual basis but might be accomplished somewhat with randomization of groups of individuals to breastfeeding promotion. Sibling-pair analyses also provide some control for these factors and may be possible with the data from the Growing Up Today Study.

Some preliminary results were presented from Project Viva, a cohort study exploring behavioral mechanisms of the breastfeeding effect and including >2000 deliveries. Three-year follow-up studies are now in progress. Interview data, biological samples, and dietary information were collected in this study. To examine the association of breastfeeding with behavioral factors that might affect the weight of the child, the effect of maternal restriction of feeding was examined, because this factor has been correlated with obesity among toddlers and preschool-aged children.⁴² The mother's level of agreement with the statement "I have to be careful not to feed my infant too much" was used as the measure of this factor. Data on prenatal concerns about the child eating too much or not enough or becoming overweight or underweight were also collected. This allowed a preliminary examination of breastfeeding duration and maternal feeding restriction at 12 months, controlling for infant gender.

For each additional 1 month of breastfeeding, there was an 11% decrease in the odds of the mother agreeing or strongly agreeing with the statement quoted above. Results were similar when demographic characteristics, mother's preexisting attitudes, and infant birth weight or 6-month weight for length were controlled, suggesting that, if breastfeeding protects against later obesity, then there might be a behavioral mechanism for the protection. Nevertheless, because the many other benefits of breastfeeding for the mother and child are well established, little harm would come from promoting breastfeeding to prevent obesity development.

Michael S. Kramer, MD, McGill University, Montreal, Quebec

Dr Kramer began by presenting data on the temporal trend in obesity, which demonstrated that, like the United States, Canada is experiencing an epidemic of obesity. Obesity prevalence has increased progressively since 1985, with most provinces exceeding 15% by 1998. The Maritime Provinces, the Northwest Territories, and the new territory in Canada, Nunavut, exhibited the highest prevalences of obesity ($\geq 20\%$) in the most recent data. There is a major problem with obesity in the aboriginal population. The 2 provinces with the lowest rates of obesity (10–14%) are British Columbia in the west and the province of Quebec in the east. It is probably not

an accident that these 2 provinces both have mountains and an outdoor lifestyle.

Data from the US National Health and Nutrition Examination Survey (NHANES) for children show increasing obesity not only among 6- to 17-year-old individuals but even in the toddler and preschool periods.³ Recent surveys show exponentially increasing rates of obesity among children in the United States, but there are no comparable data for Canada.

The relationship of birth weight to weight in childhood has been explored with data from the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC) in Tennessee.⁴³ The birth weights of WIC participants were obtained from birth certificates and linked to their weights and heights through age 5 in WIC. The z scores of weight for age, height for age, and weight for height were examined for 8 birth-weight groupings of 500 g, ranging from 1.0 to 1.5 kg through 4.5 to 5.0 kg. A z score of 0 indicates the NCHS reference mean weight-for-age z score. The 3.0- to 3.5-kg birth-weight grouping most closely approximated that value at 5 years of age. The lightest infants (1.0–1.5 kg) and the heaviest infants (4.5–5.0 kg) had the most extreme weight-for-age and height-for-age z scores initially but moved to less extreme values within ~12 months, attaining z scores of approximately -1 and +1, respectively (ie, ~1 SD from the mean). Differences in weight-for-height z scores in the most extreme birth-weight groups did not decrease with age.

A study based on data from NHANES III (also linked to birth certificates) examined weight-for-age z scores for children born LGA (>90th percentile) and for those born small for gestational age (SGA; <10th percentile).⁴⁴ There was some catch-up growth by the SGA infants and "catch down" by the LGA infants, reaching z scores of 0.5 below or above the mean, respectively, by the first year and remaining largely at those levels through 3 to 4 years of age.

Data from the Collaborative Perinatal Project, a large US study from the early 1960s, were recently used to examine the joint relationship of birth weight and early child weight gain (in the first 4 months) to overweight status at 7 years of age, considered as ≥ 95 th percentile of BMI with the use of current NCHS standards for age 7.⁴⁵ Birth weight and rate of weight gain during the first 4 months of life were both divided into quintiles. Within each early weight gain quintile, overweight status at 7 years of age tended to increase as birth weight increased, with the greatest increase in prevalence in the highest birth-weight quintile (3.61–5.56 kg). Within any birth-weight quintile, there was also an increased prevalence of overweight with increased rate of gain in the first 4 months, with a particularly large increase in the highest quintile of weight gain. Therefore, weight gain and birth weight had independent effects that persisted through at least age 7. (A table from this publication showed the OR for overweight status as 1.38 and 1.17 [2 models] for each 100-g increment in the rate of weight gain per month and 1.06 and 1.02 for each 100 g of birth weight.)

Data from a small study from Canada⁴⁶ considered this issue at later ages, to show the lasting effects of

low birth weight for gestational age on later fat content. SGA infants were matched, with respect to SES and other factors, with infants who were born between the 25th and 75th percentiles of weight for gestational age. In the subjects' teenage years, there was no significant difference in subscapular skinfold measurements, but significantly lower triceps skinfold measurements were recorded for those who were SGA, compared with those who were appropriate weight for gestational age. This finding may support relatively increased central adiposity among individuals born SGA, although the small sample size suggests the need for cautious interpretation. In a study of Swedish children monitored from birth through 18 years of age,⁴⁷ BMI at birth was correlated with BMI at 18 years of age (Spearman correlation coefficient: ~ 0.15). As might be expected, the correlation of current BMI with BMI at 18 years increased with increasing age.

A study of Finnish twin pairs examined the relationship of the intratwin difference in ponderal index at birth and the intratwin difference in BMI at 16 years of age.⁴⁸ For the 637 monozygotic twin pairs, there was a very weak, although highly statistically significant, correlation between the differences in ponderal index at birth and the differences in BMI at age 16 (Pearson r : ~ 0.05). The much stronger correlation among the same-sex dizygotic twins ($r = 0.15$) suggests that environmental factors played a much larger role than genetics in the degree to which these twins exhibited similar BMI values at age 16.

A Swedish study examined women's weight in early pregnancy and their own birth weights, with the use of a national birth register that included data on mothers' BMI values and could be linked to the mothers' earlier birth records.⁴⁹ Women who were born SGA (z scores of less than -2) had a slightly reduced (not statistically significant) chance of having a BMI of ≥ 25 when they became childbearers 20 to 30 years later, with an adjusted OR of 0.9 (95% confidence interval [CI]: 0.8-1.1). There was, however, a statistically significant increased risk (adjusted OR: 1.8; 95% CI: 1.5-2.1) of having a BMI of ≥ 25 during pregnancy among women who were born LGA (z scores of more than $+2$).

A study of Australian children related being SGA or LGA at birth to the risk of having a >94 th per-

centile BMI at 5 years of age, with the use of an internal standard based on this cohort.⁵⁰ Being SGA at birth reduced by one-half the risk of having a high BMI at age 5. Being LGA doubled the risk. Dr Kramer noted that these results were different from those presented by Dr Gillman. He proposed that there is no increased risk of obesity development at the low end of the birth-weight distribution; if attained BMI is not controlled, then low-birth-weight newborns are protected from obesity. He indicated that attained BMI should not be controlled, because BMI is on the causal path between fetal growth and later adiposity/obesity and "it actually artificially inflates the risk of obesity in SGA children."

The effect of high birth weight may become more important as birth-weight distributions move toward higher weights. Data for Canada and the United States are similar, but the increases in birth weight in the United States may be somewhat smaller. Canadian trends in low birth weight (<2500 g) and preterm births (births before 37 completed weeks) also differ from those in the United States. In the early 1980s, rates of both low-birth-weight and preterm births were declining very slightly in Canada (Fig 8).⁵¹ Since the middle 1980s, however, Canadian rates of preterm births have increased, as have rates in almost all developed countries and in many developing countries, primarily because of increases in obstetric interventions and in the numbers of multiple births. However, the low birth-weight rate, despite its being driven largely by preterm births, actually declined slightly. This finding suggests that the size of infants born at term is increasing, although more infants are being born early, with shorter gestations. Preterm births are more common, but birth weight is increasing, particularly for term births. The data showed that mean birth weight increased ~ 50 g from 1981 to 1997. Accounting for the decline in gestational age that occurred during the period (more preterm births and fewer postterm births) with the use of birth-weight z scores, the birth-weight increase was 0.2 z score, or ~ 100 g.

Compared with reference data for 1994-1996, with LGA and SGA being defined by the upper and lower 10th percentiles, respectively, in those years, the SGA rate in 1981 was almost 15%; the rate decreased by approximately one third during this time period, to

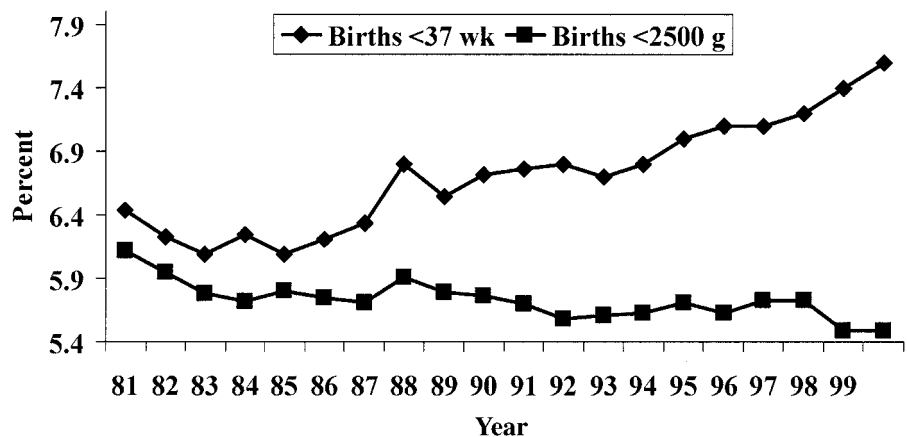


Fig 8. Trends in preterm birth and low birth weight in Canada from 1981 to 2000.⁵¹

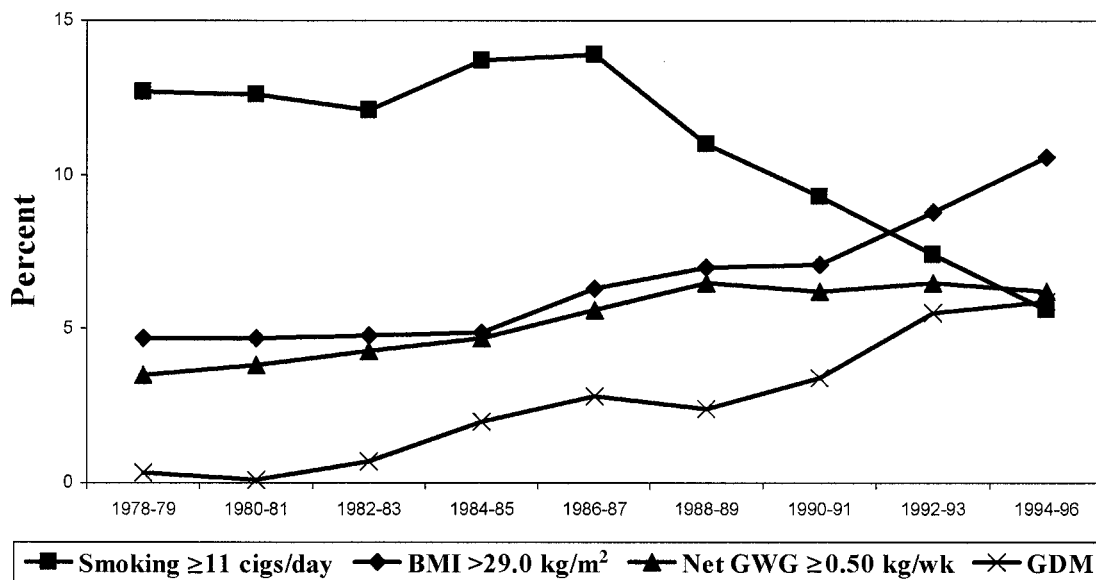


Fig 9. Trends in determinants at the Royal Victoria Hospital, 1978–1996. GWG indicates gestational weight gain; GDM, gestational diabetes mellitus.⁵²

~9.5% in 1997. Similarly, the LGA rate increased by ~25%, from ~8% to 10%, during this time period. Therefore, birth weight for gestational age has increased, as would be predicted from the contrasting trends in preterm births and low birth weight.

Like US birth certificates, Canadian birth certificates lack information on maternal BMI. Data from hospital records are more inclusive. Dr Kramer presented findings for an 18-year period at a maternity hospital at McGill University that enabled examination of factors that might have influenced birth-weight trends and fetal growth during the period.⁵² Differences in the hospital's absolute LGA and SGA rates, compared with the contemporaneous national figures presented above, likely reflect the relative SES advantage of the population the hospital serves. Nevertheless, the general decrease in SGA rates and increase in LGA rates during the time period were similar to those occurring throughout Canada. Changes in determinants that affect birth weight or birth weight for gestational age played a role. There was a 50% decrease in the proportion of mothers smoking more than one-half of a pack of cigarettes per day (Fig 9). Furthermore, the prevalence of maternal obesity (BMI of >29) doubled and the prevalence of net maternal pregnancy weight gain of at least 0.5 kg/week (exclusive of the weight of the infant) also almost doubled, increasing steadily during this time period. The prevalence of gestational diabetes mellitus increased dramatically to nearly

5%, partly because of increased screening but probably partly reflecting a real increase.

Starting with a base estimate adjusted for changes in postterm birth, ultrasonographic estimation of gestational age, and maternal height, the mean birth-weight-for-gestational age z score increased by 0.010 SD units per year (Table 5). Controlling successively for the increase in BMI, gestational weight gain, and gestational diabetes, the unexplained change in the birth-weight z score was reduced to 0.006 units per year. With control for decreased smoking, it was reduced to 0.004. With control for other factors, such as changes in maternal age distribution, marital status, and education, virtually all of the change in the mean birth-weight z score was explained.

Similar analyses showed that there was a decrease of ~2.2% per year in the risk of SGA (Table 5). Increased maternal prepregnancy BMI explained approximately one seventh of the change. Reductions in maternal smoking and the other standard factors that were controlled also explained most of the decrease in the SGA rate. The LGA rate increased ~2% per year. Approximately one quarter of that increase was explained by increases in BMI, but increases were also attributable to increases in the gestational diabetes rate and reductions in smoking. These analyses show that changes in maternal weight, weight gain, diabetes, smoking, age, and education largely explain the increases in birth-weight z scores and LGA rates and the decrease in SGA rates.

TABLE 5. Yearly Trends in Birth Weight for Gestational Age z Score and OR for SGA or LGA Birth Weight and Their Changes With Sequential Adjustment for Known Predictor Variables

	Base*	+ BMI	+ Gestational Weight Gain	+ Gestational Diabetes Mellitus	+ Smoking	+ Other Factors†
z score	0.010	0.008	0.007	0.006	0.004	0.001
SGA OR	0.978	0.981	0.983	0.984	0.989	0.999
LGA OR	1.020	1.015	1.014	1.010	1.006	1.002

* Adjusted for postterm birth, ultrasound-based gestational age, and maternal height.

† Factors included maternal age, marital status, and education.

In the above-cited Australian study,⁵⁰ maternal BMI of ≥ 95 th percentile (obesity) quadrupled the risk of obesity (BMI of > 95 th percentile) among 5-year-old subjects, even after controlling for size for gestational age at birth. Paternal BMI of ≥ 95 percentile doubled the risk. Parental obesity is an important factor, beyond the effect of birth weight. However, the larger effect of maternal (versus paternal) BMI suggests that obesity development in the offspring is not primarily a genetic effect. The relationship to maternal BMI may be partly genetic but probably reflects maternal and family lifestyles, especially regarding eating and physical activity.

Other studies have also explored the relationship of parental weight status to the weight status of the child. An analysis based on NHANES III data³⁷ classified maternal BMI as underweight, normal, overweight, or obese and compared the children's weight status at 3 to 5 years of age, grouped as BMI of < 85 th percentile, 85th to 94th percentile (overweight), or ≥ 95 th percentile (obese). The proportions of children in the 2 highest BMI categories increased monotonically as maternal weight status increased. With logistic regression analysis of these data, controlling for birth weight, race/ethnicity, gender of the child, breastfeeding, and timing of the introduction of solid foods, overweight mothers had a 54% increased risk of their children being overweight and a tripled risk of the children being obese (Table 6). Obese mothers had a tripled risk of their children being overweight and a more than quadrupled risk of their children being obese. These effects of maternal BMI may be quite long-lasting. Among African American adults, the sum of the triceps and subscapular skinfold measurements has been shown to increase with an increase in the quintile of their mothers' prepregnancy BMI.⁵³

The relationship between breastfeeding and obesity was also discussed by Dr Kramer. There are several very difficult methodologic challenges in studying this relationship, including adequately defining breastfeeding (ie, specifying whether breastfeeding was exclusive and determining the duration of exclusive or partial breastfeeding). Accuracy is affected by whether the data on infant feeding are obtained prospectively or retrospectively. Without good information on exclusive breastfeeding and the duration of breastfeeding, it is difficult to identify a dose-response relationship, which is important because the effect is probably not an all-or-none phenomenon. Also, if the breastfeeding effect is small,

then it cannot be detected in a small study. In all breastfeeding studies, the issue of residual confounding is a particular problem, because breastfeeders differ in many ways from bottle feeders.

One confounder is maternal BMI. It has been shown that high maternal BMI is associated with reduced breastfeeding initiation and duration.⁵⁴ Fat mothers are less likely to breastfeed and, if they try, they are less likely to be successful at it. Therefore, the higher weight status of bottle-fed infants could be attributable to selection of children of higher-weight mothers, who were both less likely to have breastfed and more likely to have higher-weight children. In addition, mothers who breastfed for at least 12 months reported lower levels of control over child feeding at 18 months.⁵⁵ Highly controlling feeding practices may interfere with the child's ability to self-regulate energy intake, an effect that may be long-lasting. Breastfeeding also may be associated with a healthier lifestyle, including increased physical activity, and with other factors that are hard to measure or often are not measured but may be responsible for part of the effect that has been attributed to breastfeeding.

Dr Kramer concluded that there is probably a protective effect of breastfeeding on child obesity but it must be a fairly small effect. The obesity epidemic developed in the United States during a 30-year period when breastfeeding initiation in hospitals increased and breastfeeding, even at 6 months, also increased.⁵⁶ These increases did not prevent obesity development. However, others noted that the increase in obesity might have been even worse without the increase in breastfeeding. Furthermore, the increase in obesity might not have occurred equally, or even at all, among infants who were breastfed for a significant period, because few infants are exclusively breastfed and most are not breastfed long.

Neither birth-weight changes nor breastfeeding explains the obesity epidemic. What other factors might explain it? At this point, Dr Kramer acknowledged material provided by 2 colleagues, Dr Alison Stephen, Director of Research at the Heart and Stroke Foundation of Canada, and Dr Diane Finegood, Scientific Director of the Institute of Nutrition, Metabolism, and Diabetes of the Canadian Institutes of Health Research. Body fat is the net result of what goes in and what comes out. What goes in has to do with portion sizes, meal and snack frequencies, and the energy density of food. What comes out is basal

TABLE 6. Odds of Overweight Among Children 3 to 5 Years of Age According to Maternal BMI Group in NHANES III³⁷

Mother's Weight Group	Child's Weight Status			
	BMI in 85th to 94th Percentile		BMI in ≥ 95 th Percentile	
	AOR*	95% CI	AOR*	95% CI
Normal weight (BMI of < 25.0)	1.00		1.00	
Overweight (BMI of 25.0–29.9)	1.54	0.93–2.57	2.95	1.35, 6.42
Obese (BMI of ≥ 30.0)	2.97	1.88–4.69	4.34	2.50, 7.54

* AOR indicates adjusted OR, adjusted for birth-weight status, race/ethnicity, gender, child's age, duration of full breastfeeding, and timing of introduction of solids.

energy expenditure, obligatory energy expenditure, adaptive thermogenesis, and physical activity.

Just 40 to 125 kJ extra every day leads to an increase in weight of 1 to 3 lb per year. The tools for measuring energy intake in the field, such as food frequency and 24-hour recall, cannot measure energy intake or energy expenditure that precisely. Consider the 8-year weight changes observed among adults between 20 and 40 years of age in 2 NHANES studies. The modal increase in weight was ~10 lb, equivalent to an excess of ~65 kJ per day. An excess of 210 kJ per day for 8 years would result in a gain of 40 lb, the amount gained by ~10% of the subjects studied.⁵⁷ Even 210 kJ per day is too small a difference to measure reliably with current methods. Typical food items have much greater energy levels (Big Mac: ~2475 kJ; large order of fries: 2270 kJ; grand latte: 925 kJ; bagel: ≥670 kJ). On the energy expenditure side, cycling for 15 minutes requires only 315 to 630 kJ (depending on speed and resistance), using a stair climber for 15 minutes requires 650 kJ, and walking 2000 steps on a level requires ~420 kJ.

Dr Kramer presented data assembled from several countries between 1900 and 2000.⁵⁸⁻⁶⁰ The data suggested that there was an increase in the percentage of energy as fat in the diet before World War II in the United States and probably in the United Kingdom, whereas there was a decrease in the 1970s and 1980s. The available data for Canada indicated a similar pattern of declining percentages of dietary fat in the past few decades. Carbohydrate intake has also decreased in the United Kingdom since values have been measured, in terms of grams per day or percentage of energy.

Dr Kramer cited an array of sources of data for the United Kingdom, including *Nutrition Abstracts Review* (1933 to present), the *British Journal of Nutrition* (1947 to present), the *Journal of Human Nutrition* and the *European Journal of Clinical Nutrition* (1947 to present), the *American Journal of Clinical Nutrition*, United Kingdom government publications, Medical Research Council special reports, and various medical journals. The data presented indicated that reported energy intake declined in all 3 countries, whether measured between 1972 and 1990 (Quebec and Saskatchewan) or between 1940 and 2000 (United States and United Kingdom). Use of low-energy products among US adults has skyrocketed, particularly since the middle 1980s, and there has been a concurrent increase in overweight.⁶¹ Combined results of 2 United Kingdom national surveys indicated a decrease in energy intake among children 3 to 4.5 years of age between 1950 and the early 1990s.⁶² Data from NHANES for US children from the 1970s to the 1990s also showed slight decreases in energy intakes among 2- to 5-year-old and 6- to 11-year-old children, with a possible slight increase in intake among teenagers in the most recent survey (1988-1994).⁶³ The same NHANES reports indicated that the percentage of energy intake as fat has declined for all 3 age groups.⁶³

Cross-sectional data from Nova Scotia show a decline in physical activity among both boys and girls in grades 3 through 11.⁶⁴ Data on television watching

among teenagers 12 to 17 years of age in the United States, taken from 2 Centers for Disease Control and Prevention surveys from the late 1960s and 1990, are consistent. Much time was spent watching television even in the 1970s, but the amount has increased in ~20 years. Now, one third of teenagers say they watch ≥5 hours of television per day, which surely affects energy expenditure.

In conclusion, high birth weight and infant weight gain are both associated with later obesity. There has been a temporal trend toward increasing birth weight, which is attributable mostly to increases in maternal BMI, gestational weight gain, and gestational diabetes rates and a reduction in maternal smoking. Parental, and particularly maternal, BMI is associated with child obesity and subsequent adult obesity, beyond its effect on birth weight. The evidence is equivocal but perhaps suggests a protective effect of breastfeeding, although this is unlikely to be of major public health importance, in terms of its effect on adult obesity. Dr Kramer considered the evidence weak for higher energy intake being the primary factor responsible for the obesity epidemic but considered the evidence strong for decreased physical activity playing a key role.

WHAT ARE INFANTS AND TODDLERS EATING?

Barbara Devaney, PhD, Mathematica Policy Research, Princeton, New Jersey

Dr Devaney's presentation, prepared in collaboration with Ronette Briefel, was based on findings from the Feeding Infants and Toddlers Study (FITS), some of which have been published.⁶⁵⁻⁶⁷ This study was performed with colleagues from Gerber and Mathematica, and recent results were compiled for the conference. The overall objective of the FITS was to update knowledge on the food and nutrient intakes of US infants and toddlers 4 to 24 months of age. When the FITS was conceived, concern about the prevalence of overweight and obesity in this country was increasing. There was also new information on nutrient requirements being used by the IOM committees on reference dietary intakes.

The FITS consisted of a survey of the parents and caregivers of a sample of children 4 to 24 months of age. A commercial frame, a listing judged to have the greatest coverage of all infants and toddlers, was used to draw the sample. A household survey was used to establish eligibility and recruit participants for the study. Participants answered questions on socioeconomic and demographic characteristics and were sent a food guide, a study brochure, and an incentive check. Approximately 7 to 10 days later, a 24-hour dietary recall was administered via telephone. The University of Minnesota nutrition data system was used for data collection. For statistical purposes, a second 24-hour dietary recall was administered to a random subsample of the population.

The 3022 participating children were grouped according to age, ie, 4 to 6 months, 7 to 8 months, 9 to 11 months, 12 to 14 months, 15 to 18 months, and 19 to 24 months. The ages of 4 to 6 months and 9 to 11 months were intentionally oversampled, because of

TABLE 7. Reported Energy Intakes of Infants and Toddlers, Compared With Estimated Energy Requirements (EER)⁶⁸

Child's Age	Mean EER, kJ	Mean Dietary Intake, kJ	Difference, %
4–6 mo	2632	2887	10
7–11 mo	3092	3816	23
1–2 y	3975	5226	31

the transitions in infant feeding that occur at these ages. There were 308 to 862 children in each group. The sample was reasonably representative of all US infants and toddlers, with 20% nonwhite, 12% Hispanic, 48% first-born, and 55% with working mothers (all close to national rates). Only 27%, however, participated in WIC, with lower rates for the 1- to 2-year-olds than for infants (below national rates); 80% of the sample had household incomes between \$25 000 and \$100 000, with 11% having lower incomes and 9% having higher incomes. This represents slightly smaller proportions at the low-income and high-income levels and a slightly greater proportion at the middle-income level, compared with national data.

The first question considered was: Are infants and toddlers overeating? The energy intakes reported in the 24-hour dietary recalls for these infants and toddlers exceeded the Estimated Energy Requirement, the new dietary reference standard for food energy that was released ~1 year ago by the Macronutrient Panel.⁶⁸ On average, individuals should be consuming the amount of food energy that they need in a day. In a population group, the mean intake should equal the mean energy requirement. In this population, the mean intake exceeded the mean requirement by 10% for infants 4 to 6 months of age and by 31% for children 1 to 2 years of age (Table 7). Although these intakes may represent overeating, it is also possible that the mean Estimated Energy Requirement is underestimated or that intake is overreported; all of these may contribute to the difference.

The Estimated Energy Requirement is based on an infant's or toddler's age (in months), gender, and weight. The parents reported the children's weight. If parents tended to underestimate their children's weight, as could happen if the reported weight was measured several weeks or 1 month previously, when the child was last weighed by the doctor, then

the energy requirement would be underestimated. However, the distribution of weight for age in this population did correspond approximately to the NCHS growth charts. Regarding possible overreporting of intake, parents were instructed to report not what was served but what the child consumed. If overreporting did occur, then this represents one of the few age subgroups in which that happens. Most groups underreport intake. If parents are overreporting, then this suggests that it is socially desirable to have the child eat more. This view might have implications regarding whether parents are actually overfeeding their children.

The second question addressed was: What are infants and toddlers eating? The data showed that 29% of infants have solid foods introduced before 4 months of age, whereas introduction at 4 to 6 months is usually considered to be developmentally appropriate. Only 6 percent of the infants reach the age of 6 months before the introduction of solid foods.

The percentages of total daily energy intake attributable to 7 broad food groups were presented for each of the 6 age groupings (Fig 10). For very young infants (4–6 months and 7–8 months of age), >80% of daily energy intake is obtained from milk, either breast milk or formula. The remainder is obtained mostly from infant cereal or baby foods. A transition starts at ~9 to 11 months of age and continues through the second year, as the child moves from infant feeding to baby foods and to adult foods, including table foods, cow's milk, and other beverages. By age 2, the bulk of the food energy is obtained from table foods, including 100% juices. Other beverages, which tend to be sweetened beverages, colas and ades, account for ~10% of daily energy by age 2. Cow's milk continues to play a role up to age 2.

This transition, from 9 to 11 months through the second year, is of particular interest. By age 2, approximately one third of the toddlers did not consume fruit on the day of the recall. The fruit category does not include 100% juices; it represents discrete servings of fruit, either fresh or canned. It should also be noted that ~20% of subjects did not consume a vegetable on the day of the recall. There are 2 interesting results related to vegetables. First, by 15 to 18 months of age, French fries or fried potatoes are the vegetables most commonly reported as being con-

Fig 10. Sources of food energy for infants from 4 to 24 months of age.

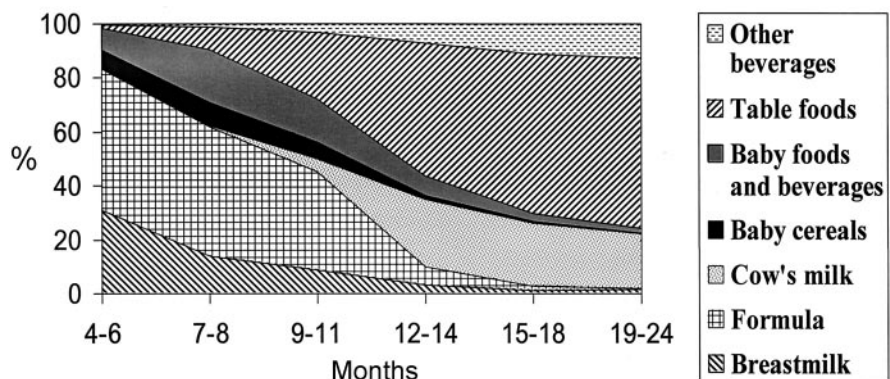
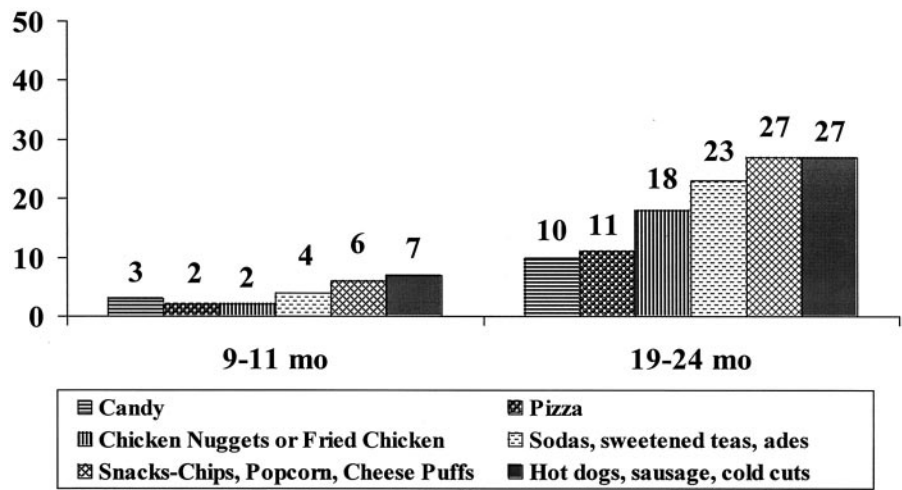


Fig 11. Percentages of infants consuming high-energy foods at least once on the recall day.



sumed. Second, 29% of the infants are consuming deep yellow vegetables at 9 to 11 months of age, when they are still consuming mostly infant foods. At 7 to 8 months, that percentage is even higher. As the children make the transition to table foods, however, the proportion consuming deep yellow vegetables decreases dramatically, to 12% at 15 to 18 months and to 8% at 19 to 24 months.

During this period, the proportion of children consuming high-energy foods increases with age (Fig 11). Intakes of some common adult foods (foods that are part of the diets of older children and adults) are revealing. Candy, pizza, chicken nuggets or fried chicken, sodas, sweetened teas and ades, salty snacks (including chips, popcorns, and cheese puffs), hot dogs, sausages, and cold cuts are included in Fig 11. By 19 to 24 months of age, 1 in 10 toddlers consumed candy on the day of the recall, 23% consumed sodas or other sweetened beverages, 27% consumed salty snacks such as chips, popcorn, or cheese puffs, and 27% consumed hot dogs, sausages, or cold cuts.

The final question addressed in the same study was: What do the diets of infants and toddlers in WIC look like? Table 8 shows data similar to that presented earlier for the general FITS population. It compares reported energy intakes with the Estimated Energy Requirements⁶⁸ for infants and toddlers in WIC. The mean energy intakes exceeded the mean requirement by 32% for older infants and by up to 40% for toddlers. These results should not be interpreted as estimates of the effects of WIC. Many of the confounders discussed previously apply here. Also, differences in energy intakes of WIC and non-WIC infants and toddlers may be attributable to differences in their demographic and socioeconomic characteristics. These are important findings, however, because WIC is a major public health vehicle for addressing the issue of overweight and obesity

TABLE 8. Reported Energy Intakes of Infants and Toddlers in WIC, Compared With Estimated Energy Requirements (EER)⁶⁸

WIC Child's Age, mo	Mean EER, kJ	Mean Dietary Intake, kJ	Difference, %
7-11	3142	4134	32
12-24	3966	5552	40

among its target population and among all US infants and toddlers. The results reported need to be interpreted in that light. New knowledge could facilitate evaluation of the WIC food package, as well as indicating the nutrition education that could be provided.

Foods that are consumed by WIC infants and non-WIC infants 7 to 11 months of age are shown in Fig 12. WIC infants are significantly more likely to be consuming 100% juices, compared with non-WIC infants. They are less likely to be consuming fruit (not including 100% juices). They are also more likely to be consuming desserts, sweets, and fruit drinks. There is no difference in vegetable consumption between WIC and non-WIC infants. The findings for toddlers are similar (Fig 13). WIC toddlers are more likely to be consuming 100% juice, less likely to be consuming fruit, and more likely to be consuming fruit drinks. In this case, though, the non-WIC toddlers are consuming desserts, sweets, and candy at about the same rate as WIC toddlers.

Dr Devaney summarized her conclusions as follows. Reported energy intakes of infants and toddlers are exceeding estimated requirements. The transition in infant feeding from true infant foods to more adult foods occurs during a long period but begins mostly at 9 to 11 months. As infants start to make the transition, the adult diet has a significant influence on what children eat. Fruit and vegetable consumption was fairly low on the day of the recall. With the importance of table foods in the diets of children through the second year, changing what toddlers are eating may require changing what adults and older siblings are eating. Finally, WIC faces significant challenges in serving its target population, given the excess of energy intakes over requirements.

TRANSITIONING TO SOLID FOODS AND THE FAMILY TABLE: EFFECTS OF PARENTING STYLES, FEEDING PRACTICES, AND CULTURE

Julie Mennella, PhD, Monell Chemical Senses Center, Philadelphia, Pennsylvania

Poor nutrition is a leading lifestyle factor related to the development of several noncommunicable dis-

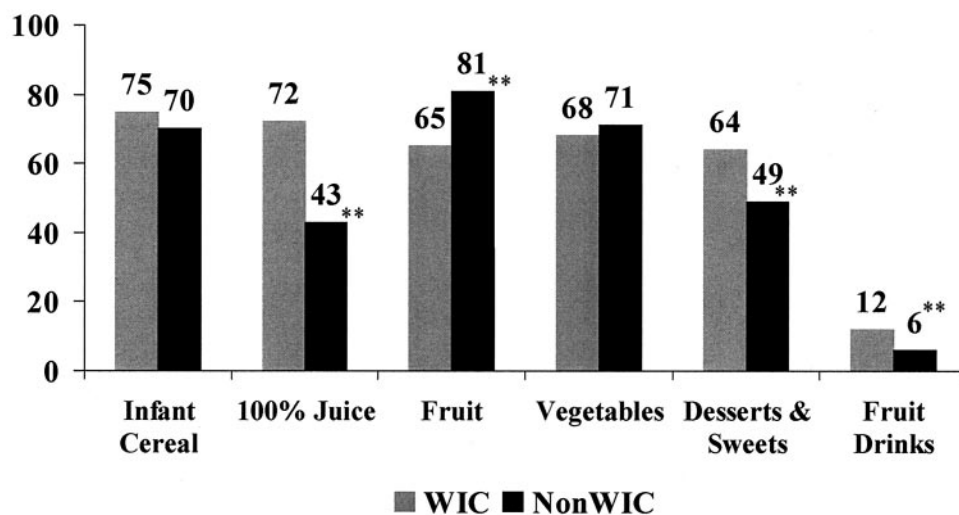


Fig 12. WIC status and percentages of children consuming different foods at least once on the recall day among infants (7-11 months of age). ** $P < .01$, non-WIC significantly different from WIC.

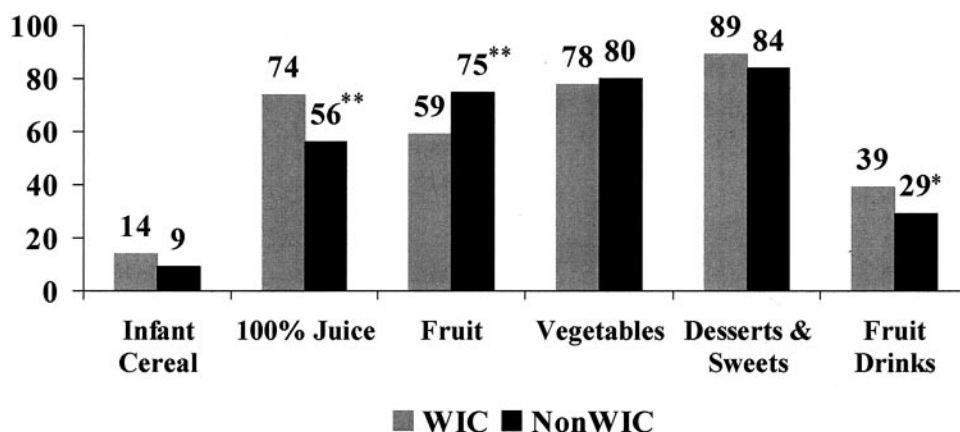


Fig 13. WIC status and percentages of children consuming different foods at least once on the recall day among toddlers (12-24 months of age). * $P < .05$, ** $P < .01$, non-WIC significantly different from WIC.

eases, including obesity. Worldwide trends indicate reduced intake of fruits and vegetables and overconsumption of sugar, salt, and fats^{69,70} (see discussion by Wardle et al⁷¹). Because eating habits established early in life continue into childhood and adulthood, recent campaigns have targeted children.^{72,73}

In general, interventions targeted at children have failed.⁷¹ The reasons for the failure are unclear, but one primary reason might be that infants and young children eat what they like. In other words, their preferences are guided by their senses and not cognitive decisions. Taste and smell are our oldest senses; they are critical for the acceptance or rejection of food. These senses are well developed in utero but continue to change during development, such that children live in their own sensory worlds. During infancy and childhood, children learn what to like, how to eat, and when to eat. There is mounting evidence of nutritional and flavor programming early in life, but one of the gaps in our knowledge remains one of the most fundamental mysteries of human behavior, ie, why do we like the things we do?

To answer this fundamental question, Dr Mennella explored a series of questions, as follows. 1) What are

the sensory capabilities and preferences of infants and children? 2) Does early exposure to salt and sweets shape preferences? 3) How do infants learn about flavors and foods? 4) Do practices early in life set the stage for lifelong food preferences? What practices facilitate later acceptance of fruits and vegetables?

Findings from developmental biology studies show that the chemical senses (taste and smell) develop in utero after tactile and vestibular capabilities but before the auditory sense. The flavor of a food includes, among other chemosensory stimuli, the oral sensation of taste (sweet, sour, salt, bitter, and umami) and the retronasal sensation of smell. The intimate connection was captured by Brillat-Savarin, who noted that "... smell and taste are in fact but a single sense, whose laboratory is the mouth and whose chimney is the nose. ..."⁷⁴ The sensory world of infants and children is different from that of adults, because their ability to detect and prefer certain tastes appears to develop after birth and is heightened during development. Within hours after birth, infants exhibit a strong preference for sweet tastes⁷⁵⁻⁷⁸ and can detect several different sugars, including lactose, glucose, and sucrose.⁷⁵ In general,

sweet sensation serves as a guide to foods that are rich in carbohydrates, and the heightened preference during childhood may serve the need for energy. Sweet tastes also produce a morphine-like analgesia among infants and children, and stimulation of the taste buds by the sweet taste is necessary to produce the analgesia.⁷⁹⁻⁸²

There is strong evidence that infants are born with a preference for sweetness, and sweetness is the predominant taste quality of breast milk. In addition to other sensory features of the mother that are preferred by the infant, such as her voice and her odor, these cues are powerful reinforcers for early learning.^{83,84} The heightened preference for sweet taste during early development is universal and is evident in children around the world, eg, in Brazil, France, Iraq, Israel, Mexico, the Netherlands, and North America.⁸⁵ Experience can modify this preference but, in general, sweet preference decreases to adult levels during late adolescence.^{86,87}

Bitter taste sensitivity is another critical early development among newborns. Such sensitivity protects the individual from poisoning, because most toxic plants and toxins contain bitter-tasting substances. Newborns grimace, arch their lips, and protrude their tongue in response to a bitter taste.⁷⁸

Unlike the other taste sensitivities, the ability to detect salt does not develop until 4 to 6 months of age. Like sweet preference, salt preference remains heightened during later infancy and into childhood; the degree of preference is related to experience with salty foods. Salt imparts a salty flavor to food and also enhances other flavors in food.⁸⁸ The most preferred level for the saltiness of food can be influenced by sodium status and dietary exposure. The sodium ion is a powerful modifier of off-tastes or bad tastes and thus enhances food palatability.^{88,89} Its ability to modify bitterness is enhanced during childhood, because of children's heightened preference for salt. In addition to age-related preferences for sweetness and sodium among children, compared with adults, there are ethnic differences.

How do infants learn about flavors and foods? Dr Mennella speculated that nutrition, a key environmental influence, might act on the genome during a sensitive period in development. This influence might have long-term effects on a wide variety of metabolic, developmental, and pathologic processes in later life. The flavors associated with the foods eaten by the mother and her infant are also being programmed. Dietary experience during the first years of life is critical for the development of several aspects of food and flavor preferences^{72,73,90,91} (see ref 92 for review).

There is evidence that a variety of flavors are transmitted from the mother's diet to amniotic fluid and mother's milk. These transmitted flavors include garlic, carrot, mint, vanilla, bleu cheese, alcohol, and tobacco.⁹³⁻⁹⁷ One-day-old infants of mothers who consumed anise-flavored beverages or foods containing garlic during pregnancy displayed less-negative facial expressions when exposed to the odors of anise and garlic, respectively. Prenatal taste experiences dictate later preferences.^{98,99} Infants can detect a diversity of flavors in amniotic fluid and mother's milk.¹⁰⁰ They accept new foods, such as cereals, more readily if they are prepared with their mother's milk.⁹⁷ The flavor profile of human milk reflects the mother's diet and the culture in which the infant is born and is similar to the flavor profile experienced in utero. The breastfed infant's sensory experience is quite varied. In contrast to this varied sensory experience, the flavor profile of formula does not vary, nor does it vary according to culture.

This difference in breast milk versus formula led Dr Mennella to explore whether the mother's diet during pregnancy and lactation influenced the infant's acceptance of vegetables (Table 9).⁹¹ The results demonstrated that infants who had exposure to the flavor of carrots in either amniotic fluid or mother's milk consumed significantly more carrot-flavored cereal and were perceived by their mothers as enjoying the carrot-flavored cereal more than plain cereal. In contrast, infants whose mothers drank water during pregnancy and lactation exhibited no such preference. These findings are the first experimental demonstration that prenatal and early postnatal exposure to a flavor enhances the acceptance and enjoyment of that flavor during weaning. These very early flavor experiences may provide the foundation for cultural and ethnic differences in cuisine.

Support for the idea that maternal exposure can enhance preferences for vegetables at weaning is demonstrated by the observation that, the more varied the mother's diet is during pregnancy and lactation, the more likely it is that the infant will accept new flavors.¹⁰⁰⁻¹⁰² Breastfed infants were more accepting of green beans, compared with formula-fed infants.¹⁰³ It seems that the minimal number of days of exposure needed is 9 days. Nine days of exposure to a variety of vegetables was associated with increased acceptance of a target vegetable and a novel food (eg, chicken).¹⁰⁴

A common concern among mothers involves the timing and order of introduction of solid foods to the child. Parents are often advised first to complement milk feedings with a grain product, such as pre-cooked cereal, and then to introduce gradually other

TABLE 9. Experimental Design for Study of Effects of Prior Exposure on Food Preferences

	Period Beverage Was Consumed		Preference Test at Weaning
	Pregnancy	Lactation	
Group CW	Carrot	Water	Carrot-flavored versus plain cereal
Group WC	Water	Carrot	Carrot-flavored versus plain cereal
Group WW	Water	Water	Carrot-flavored versus plain cereal

solid foods, such as pureed fruits, vegetables, and meats. Animal model studies suggest that the long-term effects of variety are more pronounced when variety is experienced during infancy.¹⁰⁵ In many countries, there exists a common folklore that fruits should not be introduced before vegetables, because children's inherent preference for sweets might interfere with their later acceptance of vegetables. In cultures in which fruits are introduced early, however, such introduction does not seem to interfere with vegetable consumption during infancy. The results of these studies on maternal diet and later preferences for vegetables and on early exposure to vegetables and fruits suggest that mothers should be encouraged to consume a wide variety of vegetables during pregnancy and lactation and that the order of introduction of food groups should be reexamined.

Dr Mennella speculated that, because flavor variety is often related to greater variety in the nutrient content of foods, a preference for varied flavors should increase the range of nutrients consumed and thus increase the likelihood of a well-balanced diet being obtained. In other words, the response to variety may be an important adaptive mechanism in the regulation of food intake. Whether similar effects would be observed for older infants (>6.5 months) is not yet known. It is also not known how long such effects persist in the absence of continued exposure to variety. Limited long-term effects that have been observed indicate that variety in fruit and vegetable intake among school-aged children is predicted by breastfeeding duration, food-related experiences during early life, or mothers' preferences. Data also indicate that food preferences at 2 to 4 years of age predict preferences at 8 years.^{72,73}

Despite the apparent importance of exposure to a variety of flavors in utero, in mother's milk, and during early infancy, Dr Mennella cautioned that much of children's fruit and vegetable intake cannot be explained by their mother's diet and early experience. Some of these individual differences are likely attributable to the substantial degree of sequence diversity that exists in bitter taste receptor genes. There is a lack of information regarding genetic, experiential, and developmental effects on bitter taste and on the acceptance of bitter-tasting foods such as vegetables. More specifically, how variations in human taste receptor genes influence individual differences in perception and preference is not known. It is also not known how genes and environmental factors interact to influence food preferences. Do practices early in life set the stage for lifelong food preferences? What practices facilitate later acceptance of fruits and vegetables?

Complementing this work on breastfed infants is a research program currently underway at Monell Chemical Senses Center, investigating the short- and long-term effects of formula feeding. Different brands of formula differ in their flavor profiles. Formulas fed to infants who are protein-intolerant and colicky contain proteins in a "predigested" form, usually a hydrolysate of casein or whey. These formulas have a distinctive, unpleasant odor and after-taste, as well as bitter and sour tastes, which are

attributable in part to processing and in part to the bitterness of certain amino acids. Infants who consumed hydrolysate formulas continued to accept these unpalatable hydrolysates at 7.5 months of age.¹⁰⁶ Even when tested several years after their last exposure to hydrolysates, children had heightened preferences for sour tastes, as well as for the flavor and aroma of these formulas.¹⁰⁷ The mothers of these children were also significantly more likely to report that one of their children's favorite vegetables was broccoli, compared with mothers of children fed milk-based formulas.

Dr Mennella surmised that the response to these formulas is adaptive and may reflect a fundamental aspect of early flavor learning. It is important for the infant to accept and be particularly attracted to the flavors consumed by the mother. All else being equal, these are the flavors that are associated with nutritious foods, or at least foods to which the mother has access, and thus the foods to which the infant has the earliest exposure.

The ability of our senses to be trained, our excellent long-term memory for flavors, and the reward systems that encourage us to seek out pleasurable sensations play roles in the strong emotional component of food and flavor preferences and habits. Understanding both the causes of and likely solutions to poor food habits and choices requires attention to the complex interactions of our genes, experiences, and lifestyles.

Jane Wardle, PhD, University College, London, England

Dr Wardle presented an overview of what is known about the relationships between parental feeding styles and children's eating and weight, drawing on studies that used a variety of methods to examine the relationships. Feeding style includes family eating patterns (shared or separate meal times and foods), parental control over children's eating (pressure, restriction, or monitoring), emotional feeding (feeding as a response to the child's distress), and instrumental feeding (giving food as a reward). Dr Wardle's review focused particularly on the issue of control. She used the Parental Control Scale described by Johnson and Birch¹⁰⁸ as an example of how control can be characterized and measured. The scale (Table 10) has been adapted in a variety of ways and was used in several of the studies reviewed by Dr Wardle.

Ecologic studies compare findings across times, between countries, or between population sub-

TABLE 10. Parental Control Scale¹⁰⁸

When my child does not finish dinner, s/he should not get dessert.
My child should always eat all of the food on his/her plate.
Generally, my child should only be allowed to eat at set meal times.
My child often has to be strongly encouraged to eat things that are good for him/her.
My child should be told off for playing or fiddling with food.
I have to be especially careful to make sure my child eats enough.

groups, to determine whether patterns of exposure (in this case, parental control over feeding) and outcomes (in this case, children's weights) suggest an association. This is a comparatively weak method for establishing causal processes; nevertheless, it can yield insights into processes that are hard to investigate in any other way. For example, the types and variety of foods eaten have changed dramatically in this century, and examination of the weight changes that have been correlates of these trends could yield useful hypotheses. Dr Wardle remarked that a major trend in recent years has been the emergence of a large market of foods for children, which are increasingly marketed directly to children.¹⁰⁹ This might have greatly increased children's influence over the dietary patterns in the home. It is well known that, in general, children prefer less healthful foods than adults; if parents elect to comply with children's preferences, then this could reduce the quality of the diet of the entire family.^{110,111}

Cross-cultural comparisons, such as between France and the United States,^{112,113} also provide insights. The analysis by Stearns¹¹³ shows that child-feeding manuals in France and the United States differ, with those in France emphasizing the avoidance of overweight and those in the United States emphasizing the avoidance of underweight. This distinction is supported by observations of French parental feeding styles, which exclude between-meal snacks except "gouter" when children come in from school. Even the gouter is now thought to be a bit excessive, given that French children's weights are increasing. In contrast, between-meal snacks are commonplace in the United States. The French view is that parents are helping children learn to eat the kinds of foods that adults eat. In contrast, the predominant ethic in the United States is that food choices for children should be largely child-centered; what the child likes determines what is served.

Additional observations that support these differ-

ent cultural approaches can be seen in how pediatric feeding manuals suggest handling fussy eaters. In France, pediatric advice is never to allow substitutions for the child. Children who refuse to eat can easily go without food for up to 1 day without a problem. Most children who refused food then eat. In contrast, in the United States, it is recommended that fussiness be respected, to ensure that the child eats enough. Because most studies of children's weights use national rather than international criteria for obesity, and these criteria vary considerably, there are few comparative studies. Values for the United States and France that were determined with the same criteria (from the International Obesity Task Force¹¹⁴) showed that the prevalence of obesity among French children is lower than that among US children 6 to 9 years of age (Fig 14).^{115,116}

The final ecologic comparisons examine sociocultural differences, specifically differences between groups from different SES backgrounds. Many studies have indicated that children from higher SES backgrounds are less likely to be overweight and generally have more healthful diets, which raises the question of possible causal associations. To illustrate SES differences, Dr Wardle presented data from a study she and colleagues have been conducting in 36 schools in London, where the highest rates of overweight and obesity were seen among children from the lowest SES backgrounds. Lower SES was also associated with greater consumption of high-fat foods.¹¹⁷ One of the few studies that compared parental feeding styles according to SES was a study by Hupkens et al.¹¹⁸ Those authors examined the use of "food rules" in Germany, Belgium, and the Netherlands, using a large, population-based sample in each country. Questionnaires were delivered to the homes, and the investigators returned to the homes to collect the completed questionnaires. In general, there were more prescriptive food rules about sweets (candy), soft drinks, chips (fries), and white bread among the higher SES families, and parents in higher SES groups tended to be more restrictive, allowing fewer snacks between meals. Parents in higher SES groups also showed less concern with pleasing the children and were more health conscious than those in lower SES groups. The same trends were seen for all food types and in all 3 countries; therefore, although individually (food by food) some of the effects did not reach conventional levels of statistical significance ($P < .05$), the totality of the pattern was consistent with greater parental control among the higher SES families.

Despite the limitations of ecologic studies, there do seem to be some consistencies in the data. Parental control is probably lower now than in the past, partly as a consequence of increased marketing of foods directly to children. Family feeding styles in United States-type cultures tend to be more child-centered, and thus show lower levels of parental control, than do those in some European cultures. Lower SES groups tend to exhibit lower levels of parental control than higher SES groups. In all cases, these variations correspond approximately to the variations in the prevalence of obesity.

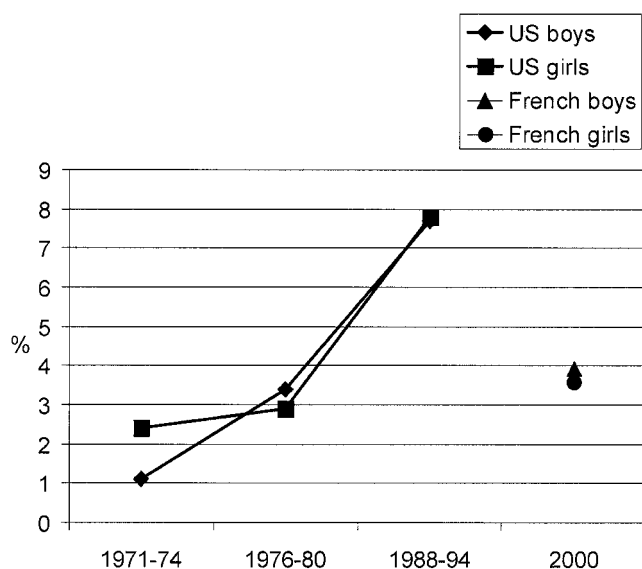


Fig 14. Prevalence of obesity, based on International Obesity Task Force criteria, among 6- to 9-year-old children in the United States and France.^{115,116}

A case-control study involves a comparison between individuals with the condition (obesity) and those without. A limited number of case-control studies have compared parental feeding styles for families of obese versus nonobese children. Observations of feeding styles (prompts or discouragements) among mothers with either fatter or thinner children (between-family comparisons) have yielded inconsistent results.^{119–121}

Within-family studies allow better control of extraneous factors, but comparisons of feeding styles within families for obese versus nonobese children also reveal no consistent patterns of feeding. Larger portions were given to the obese sibling in 1 study,¹²² but a more recent study found no significant differences.¹²³ These studies involved very few families (4 and 18 respectively), and it is possible that they lacked power to demonstrate an effect. A more general problem with this design is that, because the obese children already have a visible problem, it is possible that any difference (or lack of difference) is a consequence of the obesity, rather than a cause of it. In the study by Saelens et al,¹²³ it is possible that parents had previously fed the obese child larger portions but stopped because of the visible weight gain.

To avoid the limitations of case-control and ecologic studies, Dr Wardle and colleagues¹²⁴ enrolled children from families with high and low obesity risks (defined on the basis of parental weights) at a very early age, to compare feeding styles for children with higher or lower likelihoods of becoming obese. The authors proposed that this design made it possible to study feeding styles that might be related to obesity without the complication of existing obesity, by studying children before their obesity had begun to be expressed. The sample was drawn from families with same-sex twins enrolled in the Twins Early Development Study.¹²⁴ Children with

obese parents were compared with geographically matched and SES-matched children with normal-weight parents.

Sample characteristics are shown in Table 11. In the lean families ($n = 114$), the mother's BMI (self-reported or measured) and the father's BMI were relatively low (BMI: ~ 22 – 23). In the obese families ($n = 100$), both measured and reported average BMI values for the mother were ~ 36 . The fathers' mean reported BMI (no father's BMI was measured) was slightly below 30. In other respects, including occupation, age, and education level, the groups were the same.

Children were examined at ~ 4 years of age; by that time, there was already a slight divergence between twins with obese versus lean parents. The children from the obese families were slightly heavier, with a higher BMI than the children from the normal-weight families, but none of the children was strikingly obese. Parental feeding styles were measured with an instrument that had been developed for the study, the Parent Feeding Style Questionnaire,¹²⁴ which included measures of control, prompting, emotional feeding, and instrumental feeding. Obese mothers showed significantly less control over child feeding than did normal-weight mothers (Table 12). There were no other consistent differences between the groups.

In studies conducted by Johnson and Birch,¹⁰⁸ greater maternal control was consistently associated with poorer energy compensation. In a more recent study, Birch et al¹²⁵ showed that greater maternal restriction, which was measured when the child was 5 years of age, was associated with more eating in the absence of hunger at later ages. In terms of eating specific foods, pressure to eat was negatively associated with fruit and vegetable consumption among 5-year-old children, ie, the more the parents pres-

TABLE 11. Demographic Characteristics of Families and Their Children Selected From the Twins Early Development Study¹²⁴

	Lean Families* ($n = 114$)	Obese Families* ($n = 100$)	Significance of Difference (P Value)
Mother's reported BMI	22.3 \pm 1.7†	33.7 \pm 3.7	.001
Mother's measured BMI	23.3 \pm 2.3	36.0 \pm 4.6	.001
Father's reported BMI	22.9 \pm 1.3	29.2 \pm 3.2	.001
Mother's age, y	34.9 \pm 3.9	34.8 \pm 4.7	NS
Father's age, y	38.3 \pm 5.0	38.6 \pm 5.3	NS
Father's occupation, %			
Manual	46	46	NS
Nonmanual	54	54	NS
Mother's education, %			
O-level or less	65	71	
A-level or more	35	29	
Children's age, y	4.4 \pm 0.3	4.4 \pm 0.4	NS
Girls, %	49	55	NS
Dizygotic, %	50	54	NS
Child's weight, kg	17.5 \pm 2.5	18.3 \pm 2.8	.003
Child's height, cm	104.7 \pm 4.8	104.9 \pm 5.3	NS
Child's BMI	15.9 \pm 1.5	16.6 \pm 1.7	.001
Body fat, %	19.5 \pm 4.5	20.1 \pm 4.9	NS

NS indicates not significant.

* Lean families are families of twins with parents who are not obese; obese families are families of twins with parents who are obese.

† Mean \pm SD.

TABLE 12. Parental Feeding Style Scores for Obese and Nonobese Parents of Same-Sex Twins¹²⁴

	First-Born Twin		Second-Born Twin	
	Normal-Weight Families (<i>n</i> = 114)	Obese Families (<i>n</i> = 100)	Normal-Weight Families (<i>n</i> = 114)	Obese Families (<i>n</i> = 100)
Control over eating	4.05 ± 0.5*	3.87 ± 0.5†	4.04 ± 0.5	3.86 ± 0.5†
Prompting/encouragement	3.88 ± 0.4	3.95 ± 0.6	3.87 ± 0.4	3.95 ± 0.6
Instrumental eating	2.40 ± 0.7	2.21 ± 0.6	2.37 ± 0.7	2.25 ± 0.7
Emotional eating	1.87 ± 0.6	1.83 ± 0.6	1.89 ± 0.6	1.86 ± 0.6

* Mean ± SD.

† *P* < .001, for comparison with normal-weight families.**TABLE 13.** Correlations Between Parental Control and Fruit and Vegetable Intake of Girls and Boys

	Correlation, <i>r</i>		
	Parental Control and Fruit Intake	Parental Control and Vegetable Intake	Parental Control and Fruit and Vegetable Intake
Girls	-0.20*	-0.12*	-0.17*
Boys	-0.23*	-0.15*	-0.17*

* *P* < .001 for relationship of parental control to child's intake.

sured their children to eat, the fewer fruits and vegetables the children ate.¹²⁶

To confirm these findings in a different culture and among boys as well as girls, Dr Wardle and her colleagues studied 564 parents of 2- to 6-year-old children in London. The study also aimed to elucidate the likely mechanisms involved, ie, does control cause poor eating or does poor eating induce maternal attempts at control?

The results of the study, which are shown in Table 13, replicated Dr Birch's earlier findings. Greater parental pressure was associated with lower fruit and vegetable consumption among both girls and boys. Dr Wardle's group then added a third variable to the analysis, ie, neophobia (reluctance to eat novel foods), to attempt to ascertain the direction of causation. The investigators had previously shown that children who rate high in the neophobic characteristic eat fewer fruits and vegetables.¹²⁷ Their new results indicated that parents of these neophobic children tended to have higher control scores and, when neophobia was included in the analytic model, there was no longer a strong relationship between parental control and children's fruit and vegetable intake. Partial correlations controlling for neophobia and parental intake reduced the correlations between parental control and fruit and vegetable intake to non-significance (from *r* = -0.17 to *r* = -0.02). Given that neophobia is widely viewed as a trait characteristic, these findings are consistent with the idea that parents with neophobic children use more control in an attempt to get their fussy children to eat healthful foods, although in a cross-sectional study the possibility remains that control induces neophobia.

Three community-based studies (2 large and 1 smaller) have examined associations between parental control, measured in various ways, and aspects of the child's weight. Baughcum et al¹²⁸ found no association between any aspect of parental feeding and the child's BMI in a sample of 634 preschool-aged children. In another large study (~800 children, 7-9 years of age), higher levels of parental control were

associated with lower weight among girls but there was no association among boys.¹²⁹ The third study examined 74 children, 7 to 14 years of age.¹³⁰ Greater parental pressure was associated with lower fat mass in the children, but there was no association with restriction (restriction denotes limiting the amounts or types of foods and pressure denotes trying to persuade the child to eat certain types or amounts of foods, usually healthy foods, with both usually being motivated by concerns about health or weight).

Dr Wardle and associates are using their twin study data to examine associations between parental control and feeding. In this analysis, they are using a much larger sample of the twins. Parental control is measured with the version of the Parental Control Scale described above, which is completed for each twin at age 2. The investigators are examining differences in parental control between pairs who are discordant with respect to weight, differences across the whole sample for heavier versus lighter children, and associations between parental control and subsequent weight gain among these children. Unpublished data show that the correlation between parental control and children's weight across the whole sample was slightly negative (-0.09), ie, there were slightly higher levels of control for the thinner children. The small correlation coefficient is significant, because of the large sample size, but explains little of the variance.

When children above the 90th percentile or below the 10th percentile for weight were compared with the normal-weight group, the same pattern emerged. Levels of parental control were slightly lower for parents of overweight versus underweight children, but it was a very small effect. The scale was then examined item by item, and analysis showed that the effect was produced by 2 items on the scale, ie, "I have to encourage my child to eat" and "I have to be careful that my child eats enough." This suggests that the observed difference according to weight occurs because the parents of the underweight children are trying to "feed them up a little bit." Data from

discordant twin pairs show the same pattern; there are slightly lower levels of control for the heavier twin of each pair. Again, the effects appear to be attributable entirely to efforts to increase intake for the lower-weight twin, rather than constrain intake for the heavier one.

Across all of the studies described, the associations between parental control and child weights are inconsistent but, when the results are considered together, a general sense emerges that greater control is probably associated with slightly lower weight and lower weight gain. However, uncertainties regarding the direction of the effect remain.

Intervention studies have the advantage of being able to show cause and effect. They would also allow us to examine the impact of larger differences in parental feeding styles than are usually observed in naturalistic, community-based settings. Although there have been no such intervention studies relating parental feeding styles to children's weight, Dr Wardle described a study whose design could be adapted to intervention studies. The study was designed to show that intervention could affect acceptance of novel or disliked foods by children. The goal of the intervention was to teach parents how to increase vegetable and fruit consumption, using a method that had been developed and validated in a laboratory setting by Dr Birch and tested in a community setting by Dr Wardle and her colleagues.¹³¹

In a sample of 156 parents, a moderately disliked vegetable was used as the target food.⁷¹ The sample was randomized into 3 groups of families; the first group learned the exposure-based feeding approach, the second received a health information leaflet, and the third was a control group that underwent only the pretesting. The exposure-based feeding group, compared with the control and information groups, almost doubled the children's liking for and consumption of the target vegetable each family had chosen. This study suggests that teaching parents how to use a particular feeding strategy can produce a positive change in eating behavior. Dr Wardle remarked that the parents were thrilled at being taught the exposure techniques and said it was the first time anyone had shown them any way in which they could get their children to eat vegetables. Dr Wardle's group is extending the study to determine whether the approach succeeds in modifying vegetable intake in everyday life, compared with these tests in the laboratory.

Dr Wardle briefly reviewed family-based, behavioral therapy programs that have demonstrated short-term beneficial effects on weight loss and, over the much longer-term, beneficial effects of parent- and child-based treatment.¹³² This treatment approach involves the entire family's commitment to modifying weight-related behaviors. It includes modifying the home environment and decreasing cues for unhealthful eating and sedentary behavior. It uses behavioral therapy techniques to promote behavioral changes. Given the evidence of successful, long-term, weight change, these study findings might be taken as support for a role for the way parents feed their children. If increases in parental

control during the treatment period were noted, then this would provide evidence for a beneficial impact of control on children's weight in a clinical setting.

In collaboration with the Institute of Child Health in London, Dr Wardle and colleagues worked with 4 groups of children and their families ($n = 36$) and completed a program based on Dr Epstein's approach (J. Wardle, unpublished data). Children in the first 3 groups achieved ~10% reduction in percent overweight, which is comparable to the loss of overweight in the original study by Epstein et al.¹³² As part of this study, parents in the trial completed Dr Wardle's Parent Feeding Style Questionnaire before and after the 12-week program. Parents in the trial had measured levels of control and prompting that were lower than the population levels, but instrumental and emotional feeding levels were similar to the population levels. During the course of the treatment program, parents showed a significant increase in overall control over child feeding, bringing them toward normal levels. They showed an increase in prompting (typically, prompting to eat healthful foods) and decreases in instrumental and emotional feeding.

In summary, the pattern of results from ecologic studies is consistent with higher levels of parental control at times or in settings in which children tend to have lower weights. Community study results are mixed but suggest that higher levels of control may be associated with slightly lower levels of fatness. Intervention studies are needed to determine the direction of causality, but it was noted that parental control increased among parents who were following an effective, family-based, behavioral treatment program. These varied results suggest that greater control might be beneficial for weight control, but any conclusions must be tentative, given the inconsistent pattern of results and the difficulty of discerning causal processes.

This uncertainty exists at a time when parents desperately want to know about feeding styles that minimize, rather than promote, overweight. Dr Wardle recommended continued integration of results of broader studies that associate a measure of control with weight with the results of more detailed studies of children's ability to regulate food intake. She emphasized the importance of conducting intervention studies and cautioned against drawing conclusions on the basis of cross-sectional or prospective studies. Dr Wardle also stressed the importance of recognizing that parental control has been measured in a variety of ways and is certainly multidimensional. Different aspects of control might have very different associations with various outcomes of interest. The concept of control should be investigated in much more detail, because it is likely that both the types and amount of control play important roles in children's weight trajectories.

Margaret E. Bentley, PhD, University of North Carolina, Chapel Hill, North Carolina

Dr Bentley presented preliminary results from the analyses she, Dr Michelle Mendez, Dr Judith Borja, and other colleagues at the University of North Caro-

lina are conducting with data from the ongoing Infant Feeding and Care Project. This research project examines risk factors for the development of obesity in the first 2 years of life among low-income African Americans in North Carolina. The broad aim of the project is to investigate the role of caregiver feeding patterns and other factors, including environmental factors and physical activity, on the risk of obesity during infancy among African American families who are at high risk of obesity. More specifically, the objectives are 1) to develop an infant feeding questionnaire that would allow measurement of some of the practices and feeding styles, 2) to compare these practices and styles with child factors such as growth, 3) to assess whether styles that promote overfeeding and possibly obesity risk are highly prevalent, and 4) to identify the maternal and child characteristics associated with obesogenic feeding styles.

Although there is literature on feeding styles, patterns, practices, and behaviors among children >3 years of age, such data are scant for the first 2 years of life. Dr Bentley noted that much more is known from international studies that focus on specific feeding behaviors and their relationships to underweight and growth faltering than is known about what happens when children are eating too much and gaining too much weight.

During the first phase of the study, in-depth interviews were conducted with 20 African American mothers participating in WIC. Ethnographic data from this first phase, with additional information from the literature, led to the development of the Infant Feeding Style Questionnaire (IFSQ). The IFSQ, which is designed to characterize feeding styles among low-income African American mothers, includes items that assess 4 constructs involving both beliefs and behaviors. On the basis of the work by Birch et al¹³³ (among older children), the constructs included: 1) *laissez-faire*, in which the parent has no limits regarding food (either quality or quantity) and little or no interaction with the child during feeding; 2) *pressuring/controlling*, in which a parent feeds a child to soothe the child or because of concern that the child is undereating; 3) *restrictive/controlling*, in which the parent limits a child to healthful foods and limits the quantities of food (with this style of feeding, there may be a disruption of self-regulation of energy); and 4) *responsive*, in which parents are attentive to the child's cues, set appropriate limits, and encourage exploration in positive feeding environments. The fourth style is assumed to be the most desirable one.

The IFSQ was pretested with 154 African American mothers with children ≤ 2 years of age. If a mother had ≥ 1 eligible child, then the referent child was selected on the basis of an age group sampling quota. The data presented were cross-sectional; longitudinal data from the study will eventually be available for the elucidation of causal relationships. Ongoing confirmatory factor analysis is assessing how well the items and the measures work. The analysis of the IFSQ data aims to answer the following questions. 1) Do prevailing feeding styles among

African Americans promote overfeeding or the risk of obesity? 2) Are pressuring or *laissez-faire* feeding styles highly prevalent? 3) Do caregivers' beliefs of ethnotheories support the early introduction of solids or cereals? 4) Are maternal and child characteristics associated with obesogenic feeding styles, if they exist? 5) What is the relationship of these factors to the mother's BMI and her perception of her child as thin or fat?

Most of the women were WIC participants, between 18 and 36 years of age (mean: 25.3 years), and had a mean parity of 2.2. The mean age of the children was 9 months, and ~49% were female. Approximately 22% of the women were overweight and ~46% were obese. The prevalences of overweight and obesity are consistent with the high rates seen nationally for this ethnic group. Of great concern is that the finding that only 14% of the children were reported to be exclusively breastfed for >3 months; more than one-half of the mothers put cereal in the bottle. By 5 months of age, 42% of the children had been fed solid foods and 27% had been given juice.

In addition to assessment of behaviors and beliefs, ethnotheories were also explored. An ethnotheory is an element of a belief system used by groups of people to rationalize specific behaviors or behavior patterns. For example, in this local population of African American mothers, the term "greedy" infant is commonly used.¹³⁴ It is defined in local terms as an infant who has a huge appetite, is always reaching for food, and cannot get filled up; often such infants are big. Interestingly, the research team observed that grandmothers tend to use this term more often and to attribute positive meaning to it, whereas younger mothers do not want their infants to be greedy.

There was substantial concern about overfeeding and promotion of breastfeeding among the mothers. Most of the mothers disagreed with the idea that infants fed solids too soon will become fat. Nearly one-half disagreed that feeding an infant too much will result in a fat child, that a greedy infant may eat too much and become fat, or that a breastfed infant will be healthier than a formula-fed infant. Most disagreed that a breastfed infant is less likely to become fat than is a bottle-fed infant. Taken collectively, these data suggest that most mothers do not think that early feeding of solids or overfeeding during early infancy leads to fatness in childhood and there is little belief that breastfeeding is better for the infant's health than formula feeding.

Dr Bentley also quoted individual mothers participating in the ethnographic study, to illustrate many of the points made by the data. Consistent with the finding that cereal use began early and was rationalized to be an appropriate practice, one woman said, "I started introducing cereal at maybe 2, 2½ months because the doctor said my milk was too thin." In regard to feeding styles, the quotes reflect more variable attitudes and behaviors. "I don't really believe in feeding children fast food," one woman said, whereas another said, "Well, they will have a lot of on-the-go meals, McDonald's or Bojangle's. She will fall into that when she starts eating solid foods."

Regarding the concept of greedy infants, a range of sentiments is reflected in 2 different quotes. One woman said, "They will be overweight from overfeeding and just plain-out greedy. I hate to see that. That is why I get mad at my momma for giving her so much stuff." Another woman said, "She is a little chunky, in the 95th percentile, so she is one of those that is overweight. But I know it is in part due to us giving her regular table foods. Everybody is not meant to be small."

In addition to evaluation of these beliefs, perceived child size was compared with actual size. The results indicate that mothers often think that their infants are thin when they are not or that they are not fat when they are at the high end of the weight distribution. Dr Bentley proposed that this mismatch between perception and reality is important, because maternal perception could help determine how mothers feed their infants.

To explore how these perceptions are related to feeding styles, the mothers' behaviors were assessed. The data indicate a high prevalence of pressuring behaviors. More than one-half of the mothers reported trying to get their infants to finish their milk more than one-half of the time. More than two-thirds of the mothers said that they often (more than one-half of the time) tried to get their children to finish their food and that they praised the children after each bite. Pressuring behavior was examined with respect to the perceived thinness variable. Perceived thinness was positively associated with pressuring; infants who were judged to be thin by their mothers were more likely to be encouraged to finish their food even if full or to be coaxed to eat when not hungry.

Analysis of the laissez-faire feeding style behaviors indicated that ~20% of the mothers propped the infant's bottle more than one-half of the time and nearly one-half of the mothers reported that they watched television while feeding. The majority of mothers of infants ≥ 6 months of age reported that they seldom made sure that their children did not eat junk food or sugary foods. Laissez-faire responses were associated with perceived child fatness. Mothers who perceived their children to be fat were more likely to report watching television while feeding or to report that their children watched television while feeding. These mothers were also less likely to make sure that their children did not eat junk food or sugary food.

In terms of restrictive feeding behavior, most of the mothers said that they carefully controlled how much their children ate more than one-half of the time, and some were concerned that their children would eat too much if someone else fed them. The majority of mothers of older infants seldom allowed their children to eat fast food, junk food, or sugary foods. They also did not offer sweets if the children finished their food. Perceived fatness was associated with a heightened concern about others feeding the infant. Mothers who perceived their infants as fat were approximately twice as likely to worry that others might feed the infant too much or to worry about what types of food would be given by others.

The mother's own weight was also associated with restrictive feeding. Obese mothers were more likely to let their children eat fast food or to offer sweets if the infants finished their food.

The responsive feeding style is considered optimal and involves monitoring rather than controlling behaviors. The great majority of the mothers of infants ≥ 6 months of age reported that they kept track of how much junk food, high-fat foods, and sugary foods their children ate, although there was much variation in whether these mothers also made sure that their children did not eat these foods. Most mothers reported being attentive to their children while they were eating. Interestingly, infants who were underweight, on the basis of reported weight and length, had mothers who reported less responsiveness in terms of attention during feeding.

Dr Bentley emphasized the significance of the high proportion of mothers in this population who put cereal in the bottle. She and her colleagues began documenting this phenomenon in a Baltimore, Maryland, community >10 years ago. At that time, it was not considered an alarming behavior; however, several subsequent studies demonstrated that early feeding of solids increases the risk of developing early indicators of type 1 diabetes (islet autoimmunity) or its associated antibodies.^{135,136}

Dr Bentley stressed that it is not yet known what amount of cereal or what period of cereal provision creates the highest risk but noted that introduction during the early months may be a problem (Ziegler et al¹³⁵ found months 0-3 and Norris et al¹³⁶ found months 0-3 and ≥ 7). Earlier work with WIC mothers, performed by Dr Bentley's colleagues in Baltimore, showed substantial rates of provision of cereal in the bottle by 7 to 10 days (16%), 2 months (44%), and 4 months (60%).¹³⁷ The early introduction of cereal in the bottle represents a major public health challenge, because any intervention is likely to conflict with this strong cultural norm. Despite this challenge, there have been successful interventions that delayed the age of introduction of solid foods in these communities.¹³⁸

In addition to considering early introduction of solid foods as a risk factor for obesity and perhaps type 1 diabetes, Dr Bentley reported on an ongoing study with Dr Maureen Black to identify risk factors for early rapid weight gain (at 0-6 months of age). Preliminary data suggest that characteristics of the infant, of the mother, and of the mother's response to the infant's irritability increase the risk of rapid weight gain during this period. Important predictors among the infants included lower birth weight (all infants were >2500 g), male gender (there were no gender differences in weight at birth), whether the child was perceived to be fussy or temperamentally difficult, and whether junk food was permitted. The mothers of rapid gainers tended to have more education, fewer negative life events, fewer symptoms of depression, and high parenting satisfaction.¹³⁹

The investigators also examined the interaction between maternal responsiveness and infant fussiness. With validated scales of mothers' responsiveness, it was shown that, among the irritable infants,

those with responsive mothers experienced very rapid weight gain; among those with mothers characterized as easygoing, however, there was only a weak relationship between maternal responsiveness and rapid weight gain. This suggests that there is an interaction between maternal perceptions, infant characteristics, and growth.

More longitudinal data are needed to determine the effect of each of the feeding styles and parental perceptions on the development of obesity. Feeding behaviors in the first 2 years of life may either promote or protect against obesity, although more data are needed to establish risk and causality. Dr Bentley stressed the importance of providing health professionals with simple effective tools to address caregivers' concerns about child size, growth, temperament, and feeding and to counsel them regarding responsive feeding behaviors. Dr Bentley concluded with an emphasis on the importance of breastfeeding and the need for continued promotion of exclusive breastfeeding, as well as the appropriate timing of and type of complementary foods.

LONGITUDINAL PERSPECTIVE ON PREVENTION: THE AVON LONGITUDINAL STUDY OF PARENTS AND CHILDREN

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Dr Ness presented results from the Avon Longitudinal Study of Parents and Children (ALSPAC). The study began as the Avon Longitudinal Study of Pregnancy and Childbirth and is led by Professor Jean Golding. A number of people worked on the analyses presented, including John Reilly (who led these analyses), Julie Armstrong, Andrea Sherriff, Ahmad Dorosty, Pauline Emmett, Imogen Rogers, and Colin Steer.

The cohort was established in Bristol in the early 1990s; >14 000 pregnant women with an expected date of delivery between April 1991 and December 1992 were enrolled. Some mothers had 2 pregnancies within the eligibility period, and some mothers had >1 child with a pregnancy. There were also some losses attributable to stillbirths and postnatal deaths.

Mothers were sent questionnaires throughout pregnancy and have been sent questionnaires regularly since the birth of their children. Many of the data presented were based on the 38-month questionnaire, for which there were ~12 500 responses. Approximately 8000 children underwent a health check at ≥ 7 years of age. A subset of ~10% of the cohort (called the Children in Focus group) was examined in the clinic every 6 months from birth through 5 years of age. Some of the data presented were for this subsample.

Annual health checks began with the clinic visit at 7.5 years of age. Total-body dual-energy x-ray absorptiometry scans were performed at the 9.5-year check and are being repeated at the 11.5- and 13.5-year checks. Actigraph uniaxial movement sensors are being used at the 11.5-year check to collect objective data on activity during a 7-day period. These sensors were given to the children at the clinic, and

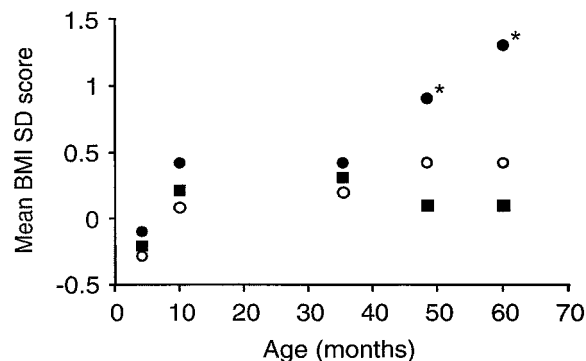


Fig 15. Adiposity rebound in ALSPAC¹⁴¹; mean BMI SD score (z score) in children characterized by very early (filled circles), early (open circles), and late (filled squares) adiposity rebound. * $P < .001$ for differences between very early adiposity rebound and the other 2 groups.

~90% of them are being returned. Therefore, detailed data on physical activity and body composition will be available in the future.

Previous work in ALSPAC compared the weight distributions of participant children with the 85th and 95th percentile weights in 1990 for the United Kingdom¹⁴⁰ On the basis of this standard, 15% of the children should have been overweight (>85th percentile) and 5% obese (>95th percentile). In the early 1990s, however, the sample showed 15.8%, 20.3%, and 18.7% overweight at ~2, 4, and 5 years of age, respectively, with obesity rates of 6.0%, 7.6%, and 7.2% for the same ages. In an analysis based on 7758 of the children measured at 7.5 to 8 years of age, 8.6% had a BMI of >95th percentile of the 1990 United Kingdom reference. When obesity is defined in this way, with extreme BMI cutoff points, the majority of children classified as obese are indeed obese; however, some of the children labeled as normal are also obese.

Some data suggest that later weight may be affected by the timing of the early adiposity rebound (the age in the first years of life when BMI begins to increase from its nadir). The children had fairly similar BMI values initially, but those with very early increases and those with later increases exhibited different BMI trajectories (Fig 15),¹⁴¹ suggesting that changes in growth in the first years may be predictive of subsequent obesity. Study children who were growing rapidly during the first 2 years were heavier, had higher BMI values, and had greater fat mass (estimated from skinfold thickness measure-

TABLE 14. Maternal Education and Child's Odds of Obesity at 7.5 Years of Age in ALSPAC

Maternal Education	OR	95% CI
CSE or less*	2.57	1.83–3.61
Vocational	2.65	1.84–3.83
O-level*	1.70	1.24–2.31
A-level*	1.74	1.26–2.39
Degree	1.00	—

* CSE indicates Certificate of Secondary Education. O-level examinations are taken at ~16 years of age; A-level examinations are taken at ~18 years of age.

TABLE 15. Relationship of Perinatal Factors to Risk of Obesity at ≥ 7 Years of Age in the ALSPAC

Risk Factor	Crude OR (95% CI)	Adjusted OR* (95% CI)
Birth weight (per kg)	1.05 (1.03–1.07)	1.05 (1.03–1.07)
Gender (female versus male)	0.87 (0.74–1.02)	0.99 (0.81–1.21)
Parity (>2 vs 0)	0.95 (0.75–1.20)	†
Maternal smoking (20 per day versus none)	1.96 (1.20–8.22)	1.80 (1.01–3.39)
Season of birth (winter versus spring)	0.98 (0.71–1.22)	†
Gestational age (≥ 42 vs < 37 wk)	1.75 (0.68–4.50)	†
Twin versus singleton pregnancy	0.88 (0.53–1.43)	†

* OR adjusted for other variables in final model, including gender, maternal education, birth weight, maternal smoking, breastfeeding, introduction of solids, parental BMI, number of siblings, hours watching television, hours asleep at night, energy intake, and the 4 food factors (junk, healthy, traditional, and fussy/snack).

† Did not enter the final model.

ments) at 5 years of age.¹⁴² For those who were growing slowly, the converse was the case.

Dr Ness presented some preliminary analyses examining associations between obesity at ≥ 7 years of age (BMI in >95 th percentile of the 1990 United Kingdom reference data) and various risk factors that had been postulated previously to increase obesity risk. Maternal education showed a clear inverse association with child obesity, with a nearly three-fold greater risk in the least educated group (Table 14). This relationship creates a problem within an observational data set, because such social gradients are associated with a series of behaviors likely to influence weight status. These behaviors include foods eaten and activities performed, and these behaviors are likely to cluster together. Although the models can be adjusted for these factors, that adjustment is probably imperfect and there is always the danger that the findings represent social patterning rather than causal associations. Cross-country comparisons and studies of cohorts that have different confounding structures can be helpful. If behaviors are identified that have different social distributions in different cohorts but for which the observed associations are of similar size, then the findings would be more convincing than the results from 1 study.

Table 15 shows the set of perinatal factors explored in relation to later obesity risk. Only the associations with birth weight and maternal smoking were significant in adjusted or unadjusted models. Each 100 g of birth weight was associated with a 5% increase in the odds of obesity. The changes in birth weight that have been seen with time are approximately that size, and the changes that can be achieved with interventions such as smoking cessation and dietary experiments are of that order. Although the associa-

tion was essentially unaltered with adjustment for a number of factors, including gestational age, the observed increase in risk was small. There was no difference in obesity risk between boys and girls, no effect of parity or season of birth, and no effect of gestational age or whether it is a single or twin pregnancy. There was an increased risk of obesity with maternal smoking, which was not attenuated with adjustment.

Table 16 shows the unadjusted OR and adjusted OR for a range of growth variables examined for a 10% subsample with more detailed measures. These measures of growth were all correlated; therefore, they were examined in separate models, both unadjusted and adjusted for birth weight, maternal smoking, parental obesity, hours of sleep, maternal education, energy intake, and infant's gender. The pattern of growth in childhood was examined with SD scores relative to the United Kingdom 1990 BMI reference data, which classified each child's BMI with respect to the mean BMI. The scores were divided into quartiles, and the highest quartile was compared with the other quartiles at 8 and 18 months. High infant weight in childhood was associated with later obesity, with the most marked increase in risk being associated with early adiposity rebound.

The associations of breastfeeding and introduction of solids with obesity risk were examined. Breastfeeding behavior was divided into 3 categories, ie, children who were exclusively breastfed at 2 months, children who had been breastfed but who were not exclusively breastfed at 2 months, and children who had never been breastfed. Breastfeeding appeared to be protective against obesity in unadjusted models, but these associations did not remain after adjust-

TABLE 16. Growth and Obesity at 7.5 Years of Age in ALSPAC Among a 10% Subsample With Detailed Measures

Risk Factor	Crude OR	Adjusted OR*
Weight SD score at 8 mo (quartile 4 versus the rest)	3.03 (1.89–4.85)	3.13 (1.43–6.85)
Weight SD score at 18 mo (quartile 4 versus the rest)	3.71 (2.29–6.00)	2.65 (1.25–5.59)
Rapid growth versus normal growth	2.21 (1.30–3.80)	2.60 (1.09–6.16)
Adiposity rebound (very early versus later)	12.0 (6.01–24.0)	15.0 (5.32–42.3)
Weight gain in first 12 mo (per 100 g)	1.07 (1.05–1.10)	1.06 (1.02–1.10)

* OR adjusted for gender, maternal education, birth weight, maternal smoking, breastfeeding, introduction of solids, parental BMI, number of siblings, hours asleep at night, energy intake, and the 4 food factors (junk, healthy, traditional, and fussy/snack).

TABLE 17. Effects of Infant Feeding on Odds of Obesity at 7.5 Years of Age in ALSPAC Among a 10% Subsample With Detailed Measures

Risk Factor	Crude OR	Adjusted OR*
Breastfeeding (exclusive for ≥ 2 mo versus never)	0.64 (0.50–0.82)	1.22 (0.87–1.71)
Breastfeeding (<2 mo versus never)	0.81 (0.64–1.01)	1.08 (0.80–1.45)
Timing of introduction of solids (2–3 vs 4–6 mo)	1.78 (1.33–2.39)	1.48 (1.01–2.16)
Timing of introduction of solids (3–4 vs 4–6 mo)	1.22 (0.99–1.51)	1.08 (0.83–1.39)

* OR adjusted for gender, maternal education, birth weight, maternal smoking, breastfeeding, introduction of solids, parental BMI, number of siblings, hours watching television, hours asleep at night, energy intake, and the 4 food factors (junk, healthy, traditional, and fussy/snack).

TABLE 18. Child Activities at 38 Months of Age and Odds of Obesity at 7.5 Years of Age in ALSPAC Among a 10% Subsample With Detailed Measures

Risk Factor	Crude OR (95% CI)	Adjusted OR* (95% CI)
Hours of television viewing (>8 vs ≤ 4 h/wk)	2.10 (1.60–2.77)	1.55 (1.13–2.12)
Time in car on weekdays (≥ 1 vs <1 h/d)	1.37 (0.90–2.07)	†
Time in car on weekends (≥ 1 vs <1 h/d)	0.64 (0.41–0.99)	†
Hours of sleep (<10.5 vs >12 h)	1.57 (1.23–1.99)	1.45 (1.10–1.89)

* OR adjusted for gender, maternal education, birth weight, maternal smoking, breastfeeding, introduction of solids, parental BMI, number of siblings, hours watching television, hours asleep at night, energy intake, and the 4 food factors (junk, healthy, traditional, and fussy/snack).

† Did not enter the final model.

TABLE 19. Dietary Patterns at 38 Months of Age and Odds of Obesity at 7.5 Years of Age in ALSPAC Among a 10% Subsample With Detailed Measures

Dietary Pattern	Crude OR (95% CI)	Adjusted OR* (95% CI)
Junk food (quartile 4 vs 1)	1.73 (1.32–2.27)	1.24 (0.87–1.76)
Healthy food (quartile 4 vs 1)	0.70 (0.54–0.91)	0.87 (0.61–1.22)
Traditional food (quartile 4 vs 1)	1.30 (1.01–1.66)	1.26 (0.92–1.72)
Fussy/snack food (quartile 4 vs 1)	0.64 (0.50–0.83)	0.79 (0.55–1.14)

* OR adjusted for gender, maternal education, birth weight, maternal smoking, breastfeeding, introduction of solids, parental BMI, number of siblings, hours watching television, hours asleep at night, energy intake, and the 4 food factors (junk, healthy, traditional, and fussy/snack).

ment (Table 17). Some models examined the effect of the timing of the introduction of solids. Introduction at 2 or 3 months of age was compared with that at 4 to 6 months, and introduction at 3 to 4 months was compared with that at 4 to 6 months. There was a suggestion in the unadjusted odds that the later introduction of solids reduced the risk of obesity, but this was not the case after adjustment (Table 17).

Some family characteristics have been explored. These included parental BMI, number of siblings at 18 months, ethnicity of child (white versus non-white), and maternal age. For children whose mother's BMI was >30 , there was a 4.5-fold increase in obesity risk, which was reduced to 4.2-fold after adjustment. For children whose father's BMI was >30 , there was a nearly 3-fold increase in obesity risk, which was reduced to 2.7-fold after adjustment. If both parents had BMIs of >30 , then the child's risk was increased 11.7-fold. Children with no siblings had an increased risk of obesity, with an OR that increased to 2.2 and became statistically significant with adjustment. None of the other family factors was a significant predictor of obesity risk.

To explore the effects of activity, several characteristics of children's lifestyles were considered. The characteristics included time per week spent watching television, time spent in the car on weekdays and

on weekend days, and sleeping time overnight, all measured at 38 months. Table 18 shows the results. Watching television >8 hours/week increased obesity risk, compared with <4 hours/week. There was a suggestion of a difference between weekday and weekend travel in the unadjusted model, but this was not evident in the final model. If less time was spent asleep, then obesity risk was increased, even after adjustment. This parameter refers to time spent sleeping overnight; it may be that more-active children sleep better or that children who are not sleeping are sitting and eating in front of the television.

Childhood dietary patterns were also examined. Dietary data were obtained from a food frequency questionnaire completed at 38 months. Principal-components analysis was used to identify 4 factors. One factor was termed "junk," with high factor loadings for carbonated drinks, candy, chocolates, fries, fried foods, and sausages. The second was called "healthy," with high factor loadings for legumes, vegetarian foods, rice, pasta, salad, fruit juice, and fruit. The third was called "traditional," with high factor loadings for meat and vegetables as meals, meat, poultry, root vegetables, potatoes (not fries), and green vegetables. The fourth factor was termed "fussy/snack," with high factor loadings for puddings, cakes/buns, cookies, ades, chips, cheese, and

fruit. Values for each factor were divided into quartiles, and the effects on obesity risk, determined with comparisons of the highest and lowest quartiles, were examined as crude or adjusted ORs (Table 19). Although the junk factor was associated with increased risk of obesity in unadjusted analyses, this association was attenuated and no longer significant after adjustment.

Future work will focus on the dual-energy x-ray absorptiometry scans, which will allow examination of the effects of these various factors on total fat and lean masses and on regional fat masses. In addition, the physical activity measurements and diet diary data being collected and coded will be related to the changes in fat mass through puberty.

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Summary of the Presentations at the Conference on Preventing Childhood Obesity, December 8, 2003

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