Metabolic and behavioral predictors of weight gain in Hispanic children: the Viva la Familia Study[1–4]

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ABSTRACT

Background: Despite the high prevalence of overweight among Hispanic children in the United States, definitive predictors of weight gain have not been identified in this population.

Objective: The study objective was to test sociodemographic, metabolic, and behavioral predictors of 1-y weight gains in a large cohort of Hispanic children studied longitudinally.

Design: Subjects (n = 879) were siblings from 319 Hispanic families enrolled in the Viva la Familia Study. Families were required to have at least one overweight child aged 4–19 y. One-year changes in weight and body composition by dual-energy X-ray absorptiometry were measured. Data were from parental interviews, birth certificates, multiple-pass 24-h dietary recalls, 3-d accelerometry, 24-h respiration calorimetry, measurements of eating in the absence of hunger, and measurement of fasting blood biochemistry indexes by radioimmunoassay. Generalized estimating equations and principal component analysis were applied.

Results: Weight gain increased with age (P = 0.001), peaking at ≈10 y of age in girls and ≈11 y of age in boys. Mean (±SD) weight gain was significantly higher in overweight (7.5 ± 3.7 kg/y) than in nonoverweight (4.4 ± 2.4 kg/y) children and in boys than in girls. When adjusted for age, age squared, sex, and Tanner stage, the final model indicated a child's body mass index (BMI; kg/m^2) status, maternal BMI, energy expenditure (total energy expenditure, basal metabolic rate, and sleeping metabolic rate), and fasting blood biochemistry indexes (total triiodothyronine, insulin, leptin, and ghrelin) as independent, positive predictors of weight gain (P = 0.01–0.001).

Conclusion: Knowledge of the metabolic and behavioral predictors of weight gain in Hispanic children will inform prevention and treatment efforts to address this serious public health problem in the United States. *Am J Clin Nutr* 2007;85:1478–85.

KEY WORDS Food intake, eating behavior, energy expenditure, physical activity

INTRODUCTION

According to the National Health and Nutrition Examination Survey (NHANES), the prevalence of overweight is among the highest in Hispanic children and adolescents (1, 2). The increasing prevalence of childhood obesity in the United States is attributed to the interaction of genes and an environment that encourages sedentary lifestyle and excess food intake; however, metabolic and behavioral predictors of weight gain have not been identified in this population.

A systematic review evaluated risk factors for childhood obesity across several populations but not in Hispanic American children (3). Factors evaluated included parental fatness, social factors, birth weight, timing of sexual maturation, physical activity, dietary intake, and psychological state. Offspring of obese parents consistently were at increased risk of obesity. Higher birth weight and earlier sexual maturation were associated with greater subsequent fatness. A protective effect of activity in childhood on later obesity was seen in some studies. Early life risk factors identified in the comprehensive Avon Longitudinal Study included early adiposity rebound, television viewing, early catch-up growth, weight z score, high infant weight gain, and short sleep duration (4).

Metabolic predictors of weight gain have been studied primarily in adults. In Pima Indians, insulin sensitivity, low metabolic rate, low physical activity, low fat oxidation, low sympathetic nervous system activity, and low plasma leptin were significant predictors of weight gain (5). Given their familial resemblance, these predictors likely contribute to the inherited tendency toward obesity in Pimas, but possibly not in other adult populations (6) or children during growth and development.

Here, we evaluate both metabolic and behavioral predictors of weight gain in a large cohort of children in the Viva la Familia Study, which was designed to identify genetic and environmental factors that affect childhood obesity in the Hispanic population. We have established the heritability of body weight and fatness in the VIVA cohort (7). The specific aims of these analyses were to test putative sociodemographic, metabolic, and behavioral predictors of weight gain: *1)* familial characteristics, *2)* birth

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information, *3)* child acculturation, *4)* dietary intake, *5)* eating behavior, *6)* physical activity, *7)* energy expenditure (EE), and *8)* fasting blood biochemistries, controlling for sex, age, and sexual maturation.

SUBJECTS AND METHODS

Study design and subjects

Subjects were from 319 Hispanic families with a total of 1030 children who were participants in the Viva la Familia Study that was designed to identify genetic and environmental factors influencing childhood obesity (7). As part of the study design, 879 of the 1030 children returned after 1 y for repeated anthropometric and body composition measurements. To qualify for the study, Hispanic families were required to have at least one overweight child aged between 4 and 19 y; overweight was defined as body mass index (BMI; in kg/m^2) $\geq$ 95th percentile (8) and fat mass (FM) $>$ 85th percentile (9, 10). All children and their parents gave written informed consent or assent. The protocol was approved by the Institutional Review Board for Human Subject Research for Baylor College of Medicine and Affiliated Hospitals and the Southwest Foundation for Biomedical Research.

Methods

Interviews were conducted with the parents, in Spanish if necessary, to obtain health history and sociodemographic data. Texas birth certificates were obtained on a subset of children and used to extract birth weights and gestational ages. Acculturation was measured with the use of the Hazuda Acculturation Scale (11).

Body weight was measured to the nearest 0.1 kg with the use of a digital balance. Height was measured to the nearest 1 mm with the use of a stadiometer. BMI was calculated. Body composition was determined by dual-energy X-ray absorptiometry with the use of a Hologic Delphi-A whole-body scanner (Hologic Inc, Waltham, MA). Changes in weight, height, and body composition were standardized to 1 y. Tanner stages of sexual maturation, based on pubic hair and breast and penile development illustrated with drawings, were by self-report (12, 13).

A multiple-pass 24-h dietary recall was recorded on 2 random occasions 2–4 wk apart in person by a registered dietitian with the use of NUTRITION DATA SYSTEM FOR RESEARCH (NDSR) software (University of Minnesota, Minneapolis, MN) (14). The multiple-pass 24-h recall method uses 3 distinct passes to garner information about a subject's food intake during the preceding 24 h. The 24-h recalls were obtained without prior notice; children $\leq$7 y of age were assisted by their mothers. The average energy intake, energy density of the diet computed excluding noncaloric beverages and water, fiber, polyunsaturated-to-saturated fat ratio, cholesterol-to-saturated fatty acid index, and percent of energy derived from the macronutrients were tested for their effects on weight gain.

Actiwatch accelerometers (Mini Mitter Co, Bend, OR) were used to measure frequency, duration, and intensity of free-living physical activity for 3 consecutive days. These accelerometers have been calibrated and validated in children and adolescents against room respiration calorimetry (15). The percentage of awake time spent in sedentary, light, moderate, and vigorous activity was computed from thresholds established for children with the use of room respiration calorimetry, as described elsewhere (15). The time spent in nighttime sleep also was extracted from the data.

Total EE (TEE) was measured by room respiration calorimetry for 24 h with the use of a standardized protocol. Oxygen consumption ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$) were measured continuously in a 18- or 30-m^3 room calorimeter. The operation, calibration, and performance of the calorimeters (16) and the reproducibility in children (17) were described previously. TEE and respiratory quotient (RQ) were computed from $\dot{V}O_2$, $\dot{V}CO_2$, and nitrogen excretion according to Livesey and Elia (18). Children's dietary intake and physical activity while in the calorimeter were controlled; the children adhered to a set schedule that included meal times, cycling on a stationary bicycle for 15 min in the morning and afternoon, free time, and a set bedtime. After a 12-h fast, basal metabolic rate (BMR) was measured on awakening in the supine position without movement. Sleeping metabolic rate (SMR) was the average EE throughout nighttime sleep, confirmed by heart rate and activity monitoring. Diet provided 15% protein, 30% fat, and 55% carbohydrate as analyzed by the NDSR software. Three meals and one snack provided the estimated energy intake to achieve energy balance, based on a multiple of the subject's calculated BMR (19).

After the 24-h respiration calorimetry measurement, regulation of food intake was assessed by the eating in the absence of hunger (EAH) procedure, as described elsewhere (20–22). Briefly, the children were served a standard ad libitum dinner providing 50% of estimated energy requirements with instructions to eat as much as they wanted in 20 min. Next, after indicating an absence of hunger the children proceeded to individual observation rooms where they were offered 10 snack foods and told they could eat as much or as little as they desired in 10–15 min. The number, type, and amount of foods provided to assess EAH were consistent with previous research (20, 21). Ten foods, providing a total of 2495 kcal, were offered: popcorn (15 g; 43 kcal), potato chips (58 g; 331 kcal), pretzels (39g; 156 kcal), nuts (44 g; 251 kcal), fruit bars (88 g; 278 kcal), chocolate chip cookies (66 g; 318 kcal), fruit-chew candy (66 g; 267 kcal), chocolate bars (66g; 369 kcal), ice cream (154 g; 322 kcal), and frozen yogurt (112 g; 158 kcal). All food items were weighed before and after, and energy intakes during the EAH procedure were calculated with the use of manufacturers' data and NDSR software.

Commercial radioimmunoassay kits were used to measure fasting serum concentrations of insulin (CV: 7.1%) and leptin (CV: 4.6%) (Linco Research Inc, St Charles, MO), and ghrelin (CV: 7%) (Phoenix Pharmaceuticals Inc, Belmont, CA). Serum total thyroxine (CV: 8.1%) and total triiodothyronine (T_3; CV: 9.5%) were measured with the use of solid-phase immunoradiometric assays (Diagnostic Products Corp, Los Angeles, CA).

Statistical methods

ACCESS (version 9; Microsoft Corp, Seattle, WA) was used for database management, and STATA (version 9.1; STATA Corp, College Station, TX) was used for descriptive statistics, principal components analysis, generalized estimating equations (GEEs), and generalized least squares (GLS) random effects regression.

To normalize values for differences in body size and composition, the natural logarithms of energy intake, EE, or RQ were

regressed on fat-free mass (FFM) and fat mass (FM) (23). Residuals of the linear regression were then used in GEE population-averaged models and GLS random effects models to test the effect of energy intake, EE, or RQ on weight gain, with sex, age, age squared, and Tanner stage as covariates. The GEE population-averaged and GLS random effects models yielded nearly identical results.

Predictors of weight gain were examined with the use of GEE population-averaged panel data models. To account for correlated data within families, a family identification number was used as the cluster variable. Models were adjusted for sex (boys = 1, girls = 2), age, age squared, and Tanner stage. Preliminary graphical analysis indicated that weight gain increased nonlinearly with age; thus, a quadratic term was needed. Transformation to normality was performed as appropriate. The GEE model, with Gaussian family, identity link function, and exchangeable correlation structure, is asymptotically equivalent to a random intercept model and thus provides almost identical estimates of the variables. A random-effects linear regression model also was fitted to the data just to obtain R^2 values for the final model.

Principal components analysis was used to reduce the number of interrelated putative predictors of weight gain, with little loss of information in the original variables, by explaining the variance-covariance or correlation structure of a set of variables through a few uncorrelated linear combinations, called principal components (24). These uncorrelated components were used in place of the original predictor variables to overcome the problem of multicolinearity, to improve the model's predictive ability, and also to provide a better understanding of the factors that influence weight gain. We selected those principal components that accounted for a large proportion of the variance but also were correlated with the dependent variable, because low variance for a component by itself does not necessarily imply that it is an unimportant predictor.

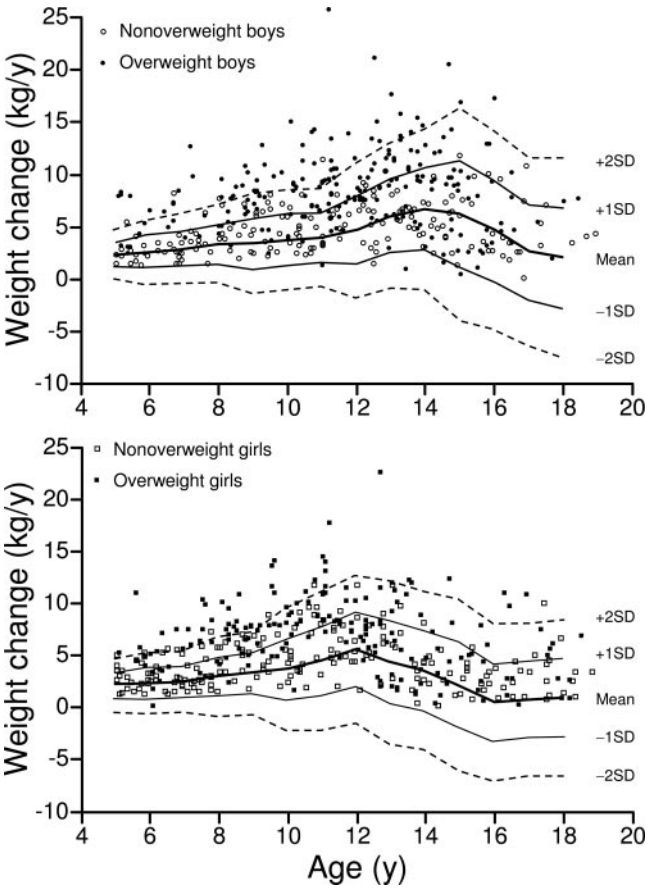

FIGURE 1. Weight gain over 1-y intervals in the nonoverweight and overweight boys and girls in the Viva la Familia Study, relative to the mean (±2 SDs) Fels Longitudinal Growth Reference (25).

RESULTS

In the 1-y follow-up interval, 91% of the children gained weight. Weight gain increased with age ($P = 0.001$), peaking at ≈10 y of age in girls and ≈11 y of age in boys, after which time it decreased (**Figure 1**). Mean weight gain was significantly higher in overweight than nonoverweight children and in boys more than girls, adjusted for age, age squared, and Tanner stage ($P = 0.001$) (**Table 1**). Weight gain was significantly correlated with FFM accretion ($r = 0.72$ and 0.84, $P = 0.001$) and FM accretion ($r = 0.76$ and 0.87, $P = 0.001$) in boys and girls, respectively. Relative to the Fels Longitudinal Growth Reference (25), the observed weight gain was >1 SD above the expected mean weight gain in 14% and 50% of the nonoverweight and overweight girls, respectively, and in 19% and 49% of the nonoverweight and overweight boys, respectively. The change in BMI *z* score was a poor indicator of weight gain in the overweight subjects because of a technicality in the development of the 2000 CDC growth charts which used a box-Cox transformation to account for skewness that had the unintended effect of mapping all high BMI values to essentially the same *z* score (26).

In the 9% who lost weight, weight loss was significantly higher in the overweight than the nonoverweight children (−3.3 ± 4.0 kg/y compared with −1.7 ± 1.9 kg/d; $P = 0.009$), adjusted for sex, age, age squared, and Tanner stage. The children who lost weight were more often girls, older, more mature, taller, and heavier than the children who gained weight ($P = 0.001$). Interestingly, in boys who lost weight, FFM was accreted, whereas FM was mobilized, resulting in a decrease in the percentage of FM. In girls who lost weight, both FFM and FM were mobilized.

First, putative predictors of weight gain (in kg/y) were explored individually with the use of GEE. The mean values of the predictors for those children who gained weight are presented in **Table 2**, along with the β coefficients from GEE models, adjusted for sex, age, age squared, and Tanner stage.

Several demographic factors were explored, but it should be noted that the cohort was fairly homogenous in terms of socioeconomic status. No significant influence of family income or maternal or paternal education on weight gain was seen. Maternal and paternal BMI were positively associated with weight gain ($P = 0.001$ and 0.007).

Mean birth weight of the children ($n = 506$) was 3.50 ± 0.63 kg; birth weights were <2.5 kg in 6.1% and >4.0 kg in 20.2% of infants. Preterm births (<37 wk gestation) occurred in 10.8% of the deliveries. Gestational diabetes mellitus was present in 13.5% of pregnancies. Exclusive breastfeeding for a median duration of 5.1 mo was reported for 34.1% of the children. Adjusted for these variables, birth weight had a significant impact on weight gain ($P = 0.05$). Child's experience with English compared with Spanish language (scale A1), proficiency in English (scale A2), pattern of English compared with Spanish language

TABLE 1
Changes in anthropometry and body composition in the Hispanic children who gained weight in the 1-y follow-up interval[1]

	Boys		Girls	
	Nonoverweight (n = 182)	Overweight (n = 228)	Nonoverweight (n = 203)	Overweight (n = 185)
Weight (kg/y)[2]	4.7 ± 2.5	8.2 ± 3.8	4.1 ± 2.4	6.7 ± 3.5
Height (cm/y)[3]	5.4 ± 2.4	5.5 ± 2.2	4.6 ± 2.8	4.7 ± 2.6
BMI (units/y)[4]	0.9 ± 1.1	1.7 ± 1.5	1.1 ± 1.1	1.7 ± 1.6
Weight-for-age z score (SD/y)[4]	0.10 ± 0.29	0.06 ± 0.20	0.13 ± 0.31	0.03 ± 0.19
Height-for-age z score (SD/y)	−0.02 ± 0.28	−0.04 ± 0.30	−0.05 ± 0.32	−0.08 ± 0.26
BMI-for-age z score (SD/y)[4]	0.10 ± 0.36	−0.01 ± 0.18	0.14 ± 0.38	−0.001 ± 0.14
Fat-free mass (kg/y)[2]	3.5 ± 2.0	5.1 ± 2.3	2.2 ± 1.4	3.6 ± 2.0
Fat mass (kg/y)[5]	1.2 ± 1.8	3.1 ± 2.7	1.7 ± 1.5	3.2 ± 2.3
Percent fat mass (%/y)[5]	0.7 ± 3.5	0.3 ± 3.1	1.8 ± 3.0	1.0 ± 2.7

[1] All values are $\bar{x}$ ± SD.
[2] Adjusted for age, age squared, and Tanner stage with significant difference by sex and BMI status ($P = 0.001$) by generalized estimating equations.
[3] Adjusted for age, age squared, and Tanner stage with significant difference by sex ($P = 0.001$) by generalized estimating equations.
[4] Adjusted for age, age squared, and Tanner stage with significant difference by BMI status ($P = 0.001$) by generalized estimating equations.
[5] Adjusted for age, age squared, and Tanner stage with significant interaction between sex and BMI status ($P = 0.001$) by generalized estimating equations; splitting the data on sex, the effect of BMI status was significant ($P = 0.001$).

usage (scale A3), and child's interaction with members of mainstream society (scale S1) were not significantly associated with weight gain.

Energy intake normalized for FFM and FM, cholesterol-to-saturated fatty acid index, and the percent of energy from fat, monounsaturated fatty acids, and n−3 and n−6 polyunsaturated fatty acids were positively correlated with weight gain ($P < 0.05$). Energy density, fiber, polyunsaturated-to-saturated fat ratio, and the percent of energy from protein, saturated fatty acids, *trans* fatty acids, fructose, sucrose, and added sugars were not significant. The amount of energy consumed in the absence of hunger also was associated with weight gain ($P = 0.008$). The percentage of awake time in sedentary activity was positively associated with weight gain ($P = 0.04$), and the percentage of awake time in light activity was negatively associated ($P = 0.007$). Neither percentage of awake time in moderate and vigorous activity nor sleep duration was associated with weight gain.

TEE adjusted for FFM and FM was positively related to weight gain, controlling for sex, age, age squared, and Tanner stage ($P = 0.001$). BMR and SMR, adjusted for FFM and FM, also were positively associated with weight gain ($P = 0.001$). RQs adjusted for energy balance and body composition were not correlated with weight gain. Independent of age, age squared, sex, and Tanner stage, weight gain was positively associated with fasting circulating concentrations of total T_3, insulin, leptin, and ghrelin ($P = 0.001$).

To address potential confounding between BMI status and the putative predictors of weight gain, the GEE analyses were repeated and adjusted for BMI status, age, age squared, sex, and Tanner stage (Table 2, columns 5 and 6). Individual effects of maternal BMI; energy from dietary fats; rates of EE, and fasting concentrations of total T_3, insulin, leptin, and ghrelin persisted ($P = 0.05–0.001$). Significant effects of paternal BMI, birth weight, EAH, and sedentary and light physical activity were not apparent, independent of BMI status.

The principal components and their corresponding coefficients for dietary intake, physical activity, EE, and fasting blood biochemistries used in the final GEE models to predict weight gain are shown in **Table 3**. The final GEE model for weight gain is presented in **Table 4**. Adjusted for age, age squared, sex, and Tanner stage, the final GEE model retained child's BMI status, maternal BMI, principal component 1 for EE (TEE, BMR, SMR), and principal component 1 for fasting blood biochemistries (total T_3, insulin, leptin, and ghrelin) as independent, positive predictors of weight gain ($P = 0.01–0.001$). The intraclass coefficient for family cluster was 0.26. The final GEE model explained 30% of the overall variance in weight gain.

DISCUSSION

In this prospective study Viva la Familia, we have documented the natural progression of childhood obesity in a large cohort of Hispanic families selected on the basis of child overweight. Weight gain tended to peak at ≈10–12 y of age and was higher in boys than in girls, consistent with NHANES of a higher prevalence of overweight in Mexican American boys than girls (1, 2). Rates of weight gain, up to 26 kg/y, were most alarming in the overweight children with 50% gaining >1 SD above reference weight velocities (25).

It is widely acknowledged that genetics has a substantial influence on body weight regulation (27). Consistent with significant heritabilities for weight, BMI, FFM, and FM in the VIVA cohort (7), we found that maternal BMI was a significant predictor of weight gain, confirming the genetic or shared familial influence on the development of childhood obesity. Birth weight, a trait influenced by genetics and intrauterine environment, was positively associated with weight gain, but this seemed to be mediated by the child's BMI status, in agreement with reported positive associations between birth weight and later BMI (28).

We did not observe an influence of early feeding practices on weight gain. About one third of the children were exclusively breastfed and another third were partially breastfed; we did not see an effect of any exposure to breastfeeding on weight gain. Although breastfeeding appears to be associated with a reduced risk of overweight, it is unclear whether this is due to confounding of socioeconomic, maternal, or other unmeasured factors (29, 30).

TABLE 2
Predictors of weight gain (kg/y) in Hispanic children (n = 798) evaluated individually in generalized estimating equations[1]

Predictors	Value	β coefficient ± SD[2]	P[2]	β coefficient ± SD[3]	P[3]
Group 1 familial characteristics					
Income ($/y)	29 616 ± 15 709[4]	$1.2 \times 10^{-5} \pm 8.9 \times 10^{-6}$	0.17	$-8.2\times10^{-6} \pm 7.9\times10^{-6}$	0.3
Paternal education (y)	8.8 ± 4.4	−0.04 ± 0.03	0.19	−0.005 ± 0.03	0.86
Maternal education (y)	9.6 ± 4.1	−0.03 ± 0.03	0.35	−0.02 ± 0.03	0.46
Paternal BMI (kg/m^2)	30.7 ± 4.7	0.07 ± 0.03	0.007	0.03 ± 0.02	0.24
Maternal BMI (kg/m^2)	33.8 ± 8.2	0.08 ± 0.02	0.001	0.06 ± 0.01	0.001
No. of siblings	3.6 ± 1.2	0.03 ± 0.12	0.78	0.20 ± 0.10	0.05
Group 2 birth information					
Gestational age (wk)	39.4 ± 2.3	0.07 ± 0.06	0.25	0.04 ± 0.05	0.35
Gestational diabetes mellitus (%)	13.5	−0.15 ± 0.32	0.64	−0.004 ± 0.30	0.49
Exclusive breastfeeding (%)	34.1	−0.02 ± 0.26	0.92	−0.05 ± 0.23	0.82
Birth weight (kg)[5]	3.50 ± 0.63	0.52 ± 0.26	0.05	0.26 ± 0.24	0.26
Group 3 child acculturation					
Scale A1	1.8 ± 1.1	0.13 ± 0.11	0.23	0.10 ± 0.10	0.3
Scale A2	3.0 ± 0.9	−0.01 ± 0.17	0.97	−0.03 ± 0.15	0.82
Scale A3	3.0 ± 0.8	0.10 ± 0.16	0.54	0.08 ± 0.15	0.58
Scale S1	2.1 ± 0.8	−0.13 ± 0.14	0.36	−0.06 ± 0.13	0.66
Group 4 dietary intake					
lnEnergy (kcal/d)[6]	7.54 ± 0.31	0.82 ± 0.05	0.05	0.63 ± 0.38	0.1
Energy density (kcal/g)	1.32 ± 0.31	0.24 ± 0.39	0.53	0.23 ± 0.35	0.5
Fiber (g/d)	13.8 ± 6.5	0.03 ± 0.02	0.08	0.03 ± 0.02	0.04
P:S	0.56 ± 0.27	0.71 ± 0.45	0.11	0.54 ± 0.41	0.18
C:SFA index	40.6 ± 16.4	0.021 ± 0.007	0.006	0.015 ± 0.007	0.02
Energy from protein (%)	14.2 ± 3.0	0.022 ± 0.039	0.58	0.005 ± 0.04	0.89
Energy from carbohydrates (%)	53.1 ± 7.2	−0.044 ± 0.016	0.007	−0.031 ± 0.015	0.04
Energy from fat (%)	34.0 ± 6.0	0.055 ± 0.020	0.006	0.044 ± 0.018	0.014
Energy from SFAs (%)	12.4 ± 2.7	0.032 ± 0.043	0.45	0.038 ± 0.039	0.34
Energy from MUFAs (%)	12.5 ± 2.6	0.11 ± 0.04	0.02	0.08 ± 0.04	0.05
Energy from n−3 PUFAs (%)	0.56 ± 0.23	138 ± 52	0.008	119 ± 47	0.01
Energy from n−6 PUFAs (%)	5.6 ± 2.2	14.1 ± 5.4	0.009	13.2 ± 4.9	0.008
Energy from *trans* FAs (%)	2.2 ± 1.0	14.6 ± 11.7	0.21	9.8 ± 0.6	0.36
Energy from fructose (%)	6.6 ± 3.1	−2.66 ± 3.86	0.49	−3.54 ± 3.51	0.31
Energy from sucrose (%)	11.1 ± 4.8	−1.29 ± 2.43	0.60	−1.53 ± 2.21	0.49
Energy from added sugars (%)	19.7 ± 9.8	−0.54 ± 1.23	0.66	−1.52 ± 1.12	0.18
Group 5 eating behavior					
Eating in absence of hunger (kcal)	389 ± 224	0.002 ± 0.001	0.008	0.001 ± 0.001	0.17
Group 6 physical activity levels					
Total (counts/d)	$23.0 \pm 8.8 \times 10^4$	−0.001 ± 0.002	0.72	0.001 ± 0.002	0.53
Sleep time (min/d)	532 ± 64	−0.001 ± 0.002	0.60	−0.0003 ± 0.002	0.85
Awake sedentary (%)	36 ± 13	2.30 ± 1.11	0.04	−0.33 ± 1.03	0.74
Awake light (%)	54 ± 10	−3.64 ± 1.35	0.007	−0.20 ± 1.25	0.87
Awake moderate (%)	10 ± 6	0.74 ± 2.36	0.75	2.03 ± 2.16	0.34
Awake vigorous (%)	0.3 ± 0.5	−4.78 ± 22.6	0.83	2.92 ± 20.4	0.89
Group 7 energy expenditure					
lnTEE (kcal/d)[6]	7.60 ± 0.24	11.18 ± 1.77	0.001	8.04 ± 1.69	0.001
lnBMR (kcal/d)[6]	7.28 ± 0.23	9.41 ± 1.70	0.001	7.64 ± 1.59	0.001
lnSMR (kcal/d)[6]	7.20 ± 0.24	12.44 ± 1.88	0.001	9.34 ± 1.79	0.001
TEE RQ[7]	0.87 ± 0.03	−0.39 ± 4.93	0.94	1.16 ± 5.05	0.82
BMR RQ[7]	0.84 ± 0.05	−1.91 ± 2.88	0.51	−1.27 ± 2.77	0.65
SMR RQ[7]	0.84 ± 0.03	−1.21 ± 4.51	0.79	−1.04 ± 4.38	0.81
Group 8 fasting blood biochemistry indexes					
lnTotal T_3 (ng/dL)	5.01 ± 0.16	4.30 ± 0.85	0.001	3.23 ± 0.79	0.001
lnTotal T_4 (mg/dL)	8.15 ± 1.42	0.04 ± 0.09	0.62	−0.01 ± 0.08	0.89
lnInsulin (mIU/mL)	2.81 ± 0.78	1.74 ± 0.16	0.001	0.88 ± 0.20	0.001
lnLeptin (ng/mL)	2.46 ± 0.96	1.69 ± 0.12	0.001	1.18 ± 0.18	0.001
lnGhrelin (pg/100 mL)	3.57 ± 0.40	−2.22 ± 0.36	0.001	−0.72 ± 0.36	0.05

[1] FFM, fat-free mass; FM, fat mass; P:S, polyunsaturated-to-saturated fat ratio; C:SFA index, cholesterol-to-saturated fatty acid index; SFAs, saturated fatty acids; MUFAs, monounsaturated fatty acids; PUFAs, polyunsaturated fatty acids; TEE, total energy expenditure; BMR, basal metabolic rate; SMR, sleeping metabolic rate; RQ, respiratory quotient; T_3, triiodothyronine; T_4, thyroxine.
[2] Adjusted for child characteristics of sex, age, age squared, and Tanner stage by generalized estimating equations.
[3] Adjusted for child characteristics of sex, age, age squared, and Tanner stage and BMI status by generalized estimating equations.
[4] $\bar{x}$ ± SD (all such values).
[5] Adjusted for gestational age, gestational diabetes mellitus, and exclusive breastfeeding.
[6] Adjusted for lnFFM and lnFM.
[7] Adjusted for lnFFM, lnFM, and energy balance.

TABLE 3
Principal components for dietary intake, physical activity levels, energy expenditure, and fasting blood biochemistry indexes measured in the Hispanic children ($n = 798$)[1]

	Eigenvectors[2]		
Predictors	Component 1	Component 2	Component 3
Dietary intake			
lnEnergy (kcal/d)[3]	0.216	0.621	0.422
Cholesterol-to-SFA index	0.372	0.526	0.141
Energy from carbohydrate (%)	−0.477	0.122	0.288
Energy from fat (%)	0.511	−0.103	−0.218
Energy from MUFAs (%)	0.473	−0.088	−0.293
Energy from n−3 PUFAs (%)	0.210	−0.374	0.576
Energy from n−6 PUFAs (%)	0.243	−0.405	0.504
Eigenvalue (proportion)[4]	3.38 (0.48)	1.42 (0.20)	1.21 (0.17)
Physical activity levels			
Awake sedentary (%)	−0.71		
Awake light (%)	0.71		
Eigenvalue (proportion)[4]	1.90 (0.95)		
Energy expenditure			
lnTEE (kcal/d)	0.56		
lnBMR (kcal/d)	0.58		
lnSMR (kcal/d)	0.59		
Eigenvalue (proportion)[4]	2.49 (0.83)		
Fasting blood biochemistry indexes			
lnTotal T_3 (ng/dL)	0.003	0.96	
lnInsulin (mIU/mL)	0.61	0.06	
lnLeptin (ng/mL)	0.58	0.13	
lnGhrelin (pg/100 mL)	−0.54	0.22	
Eigenvalue (proportion)[4]	2.24 (0.56)	1.04 (0.26)	

[1] FFM, fat-free mass; FM, fat mass; SFA, saturated fatty acid; MUFAs, monounsaturated fatty acids; PUFAs, polyunsaturated fatty acids; TEE, total energy expenditure; BMR, basal metabolic rate; SMR, sleeping metabolic rate; T_3, triiodothyronine.
[2] Derived from principal component analysis; the eigenvectors for individual predictors are presented.
[3] Adjusted for lnFFM and lnFM.
[4] Eigenvalues and the proportion of variance explained by the component are presented.

Sociodemographic factors may influence the prevalence of obesity in the United States with decreased access to recreational activities and healthy diets among low-income populations. In NHANES III, the overweight prevalence among Mexican American children, however, was not related to family income (31), as we found. Acculturation has been associated with the striking increase in obesity between first- and second-generation Hispanic adolescents (32). In the National Longitudinal Study of Adolescent Health, foreign-born Hispanic adolescents were more active and had healthier diets than their US-born counterparts. In the VIVA cohort, 82% of the children were born in the United States of foreign-born parents; acculturation did not have an impact on weight gain in these second-generation Hispanic children.

TABLE 4
Final generalized estimating equation (GEE) model for the prediction of weight gain in the Hispanic children ($n = 798$)[1]

Independent variables	β coefficient (SE)	P
Age	0.80 (0.35)	0.02
Age squared	−0.03 (0.01)	0.03
Sex	−0.57 (0.33)	0.09
Tanner stage	−0.48 (0.22)	0.03
BMI status	1.51 (0.40)	0.001
Maternal BMI	0.06 (0.02)	0.002
PC1–energy expenditure	0.52 (0.11)	0.001
PC1–fasting blood biochemistry indexes	0.41 (0.17)	0.01
Constant	0.79 (2.32)	0.73

[1] Principal component 1 (PC1)–fasting blood biochemistry indexes represents serum total triiodothyronine, insulin, leptin, and ghrelin. PC1–energy expenditure represents total energy expenditure, basal metabolic rate, and sleeping metabolic rate. The dependent variable was weight gain (kg/y). r^2 overall was 0.30.

Obesity can only arise from an imbalance of energy intake and EE, yet most studies have failed to detect significant effects of either on subsequent fatness (3). Our quantitative methods and large sample size have allowed us to identify significant positive associations among dietary intake, eating behavior, sedentary level of physical activity, and child's BMI status. Energy intake, EAH, and sedentary activity were higher in overweight subjects, who also experienced greater weight gains. Of these behavioral factors, only dietary intake from fats was positively correlated with weight gain, independent of BMI status. The effects of eating behavior and sedentary activity and BMI status on weight gain are confounded and cannot be distinguished from each other in our study.

In a systematic review of childhood predictors of obesity, 7 studies were identified that measured dietary intake and later childhood fatness (3). Findings, for the most part, were nonsignificant and inconsistent. One study showed a positive relation

between energy intake between 4 and 6 y of age and fatness at 8 y of age (33), whereas others reported negative relations at 9 y of age (34) and at 15 y of age in girls only (35). One study found a positive relation between percent energy from fat at 6 y of age (36), and another reported a positive association between percent energy from protein and fatness at 8 y of age (33).

Evidence of the effect of physical activity on later fatness in childhood is also inconsistent, in part because of limitations of methods, sample sizes, and confounding variables (3). Of the 9 studies identified, 5 showed a significant effect of physical activity on fatness in childhood. Longer television viewing was associated with greater fatness (37). Higher leisure activity was positively and aerobic activity was negatively associated with changes in BMI (36). Total activity was inversely related to increases in percentage of body fat (38) and skinfold thicknesses (34). Others failed to detect an effect (39).

The metabolic predictors of weight gain in these Hispanic children appear opposite to those in adulthood. The putative role of EE in the development of obesity was studied extensively in adult Pima Indians. Prospective studies were conducted to determine whether lower rates of EE measured in a respiratory chamber contribute to the pathogenesis of obesity in adult Pima Indians (40). Adjusted for FFM, FM, age, and sex, resting metabolic rate ($r = -0.19$, $P = 0.04$) and 24-h TEE ($r = -0.39$; $P = 0.001$) were negatively correlated with the 2–4 y weight change. The 24-h RQ was positively correlated with changes in weight and fat mass ($r = 0.27$, $r = 0.19$, $P < 0.05$) (41). Low spontaneous physical activity in the respiratory chamber was associated with higher weight gain (42). Low metabolic rate predisposing to weight gain has only been shown in the Pima adult population. Two other adult studies failed to find an association between metabolic rate and weight gain (43), and a third study reported a positive association in lean Nigerians (6). Our calorimetry results are contradictory to the Pima findings, but they are consistent with 2 pediatric studies (44). In the VIVA study, TEE, BMR, and SMR adjusted for body composition were positively correlated with weight gain. Similarly, positive relations were found among TEE and changes in fat mass (44) and percentage of FM in 8-y-old children (45).

Hormonal predictors of weight gain in Pima adults included low activity of the sympathetic nervous system, low plasma leptin, and insulin sensitivity (5). We found fasting serum insulin was positively associated with weight gain, as seen in Pima children. Odeleye et al (46) found that fasting hyperinsulinemia was a predictor of increased weight gain in Pima children, after a mean follow-up period of 9.3 y. Consistent with our findings, fasting insulin in Pima children was positively correlated with weight gain (5.3 kg/y) ($r = 0.27$, $P = 0.0001$). Insulin is known to have potent lipogenic effects and clearly plays an important anabolic role in the pathogenesis of obesity. In addition to fasting insulin, we also found positive associations among total T_3, leptin, and weight gain and a negative association between ghrelin and weight gain in the VIVA children. Leptin resistance and altered metabolism of ghrelin, an orexigenic gastrointestinal peptide, may predispose children to excessive weight gain (47). In contrast to adults, metabolic predictors of weight gain in growing children may reflect increased activity of the sympathetic nervous system, substrate cycling, ion pumping, protein synthesis or turnover, or fat synthesis associated with growth and development.

Adjusted for age, age squared, sex, and Tanner stage, the final GEE model indicated child's BMI status, maternal BMI, EE (TEE, BMR, SMR), and fasting blood biochemistries (total T_3, insulin, leptin, and ghrelin) as independent, positive predictors of weight gain ($P = 0.01–0.001$), accounting for 30% of its variance. Although the intrinsic metabolic factors may seem to be stronger predictors, it does not imply the behavioral predictors are unimportant. Identification of the critical factors in the development of childhood obesity provides a rational basis for the design of effective interventions for the prevention and treatment of excess weight gain in this population. Central to any intervention for the prevention and treatment of excessive weight gain should be strategies aimed at shifting awake time from sedentary into light activities, decreasing total energy intake especially from dietary fat, and minimizing hyperphagic eating behaviors.

In conclusion, we have documented the progression of obesity in a large cohort of Hispanic children and identified independent predictors of weight gain, including child's BMI status, maternal BMI, rates of EE, and fasting blood biochemistries (total T_3, insulin, leptin, and ghrelin). Knowledge of the metabolic and behavioral predictors of weight gain will inform efforts to address this serious public health problem in Hispanic children in the United States.

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