FINAL COMPREHENSIVE REPORT MCHB RESEARCH PROGRAM

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I. Introduction

A. Nature of the Research Problem

Over the last three decades, the prevalence of obesity (defined as sex- and age-specific body mass index [BMI] at or above the 95th percentile of Centers for Disease Control [CDC] growth standards) at least doubled among children. Some subgroups are disproportionately affected by this epidemic with Hispanic and non-Hispanic Black children and those living in families with low socioeconomic status (SES) disproportionately affected.[1-3] Childhood obesity is associated with a host of physical and mental health problems—including type 2 diabetes, asthma, hypertension, sleep apnea, emotional and weight-related distress—and high health care costs[4-9]. Children who are obese are likely to be obese in adulthood,[10, 11] experience morbidity from cardiovascular disease, high blood pressure and stroke[8, 12] and incur higher health-related costs over the life course. Specific causes of the obesity epidemic and associated disparities remain elusive. Recent animal studies provide possible clues, finding that chronic stress among mice is associated with the release of neuropeptide Y, metabolic syndrome-like symptoms and growth of abdominal fat. The proposed research uses existing longitudinal, panel survey data to test hypotheses about relations between early childhood exposure to stress and the development of childhood obesity and the mediating role of stress in racial/ethnic and socioeconomic disparities.

B. Purpose, Scope, and Methods of the Investigation

Several specific aims were addressed. **AIM 1:** To test prospective associations between exposure to stress in early childhood (0 – 6 years) and body mass index (BMI) in later childhood (age 10 – 11 years); **AIM 2:** To investigate whether exposure to stress in early childhood mediates relations between race/ethnicity or family SES and BMI; and **AIM 3:** To test prospective associations between early childhood SES and later childhood BMI. In response to proposal reviewers' comments, the analysis was expanded to include a fourth aim: **AIM 4:** To test prospective associations between sleep duration in early childhood and the subsequent development of obesity. Data for the study were derived from two, large national surveys: 1) The Bureau of Labor Statistics (BLS) National Longitudinal Survey of Youth, Child Young Adult surveys (NLSY-CYA) linked to parent data in the NLSY79 cohort; and 2) The Panel Survey of Income Dynamics (PSID), a longitudinal survey directed by the National Science Foundation.

C. Nature of the Findings

AIM 1: Of the early childhood stress measures examined (age 0 – 6 years), none was consistently associated with the child's z-BMI score or with categorical measures of obesity or overweight measured at age 10 or 11 years. **AIM 2:** We found no evidence that the stress variables we examined mediated obesity disparities by SES or race/ethnicity. However, some but not all of the racial/ethnic disparities measured were accounted for by SES. **AIM 3:** Measures of early childhood income and poverty status were not associated with BMI at Age 10 or 11 when contemporaneous measures of SES were controlled. The effects of early childhood SES on later obesity were fully accounted for by maternal education. **AIM 4:** For children 5 – 9 years old in 2002, duration of night-time sleep measured five years prior (ages 0 - 4) was strongly associated with increased risk of subsequent obesity, controlling for contemporaneous sleep. For adolescents (aged 10-18), sleep duration at baseline was not associated with subsequent obesity; however, contemporaneous sleep was inversely associated with obesity. Day-time sleep had little effect on subsequent obesity at any age.

II. Review of the Literature

AIM 1 - Early Childhood Stress and Subsequent BMI: Several reviews suggest stress is a plausible contributor to obesity.[13-15]In animal studies, daily exposure to major chronic stressors led to increased subcutaneous and visceral fat mass (i.e. obesity) in mice fed a high fat diet and outcomes similar to a metabolic syndrome (i.e. decreased glucose tolerance; increased insulin, resistin, adiponectin and lipids).[16] The underlying biologic process was attributed to the release of neuropeptide Y (NPY) from sympathetic nerves in response to stress, with NPY and its receptors, in turn, up-regulated leading to metabolic syndrome-like symptoms and growth of abdominal fat.[16] We found no studies that explicitly tested stress-obesity relations in children.

AIM 2 - Stress as a Mediator of Racial/ethnic or SES Disparities in Obesity: In 2003 - 2004, 17% of children ages 2 – 19 years were obese; 34% were overweight nationwide according to established definitions.[2] White, non-Hispanic children fared slightly better than the national average, while 20% of non-Hispanic Black children were obese and 35% were overweight and 19% of Hispanic children were obese and 37% were overweight. In general, children 6 – 11 years old had the highest prevalence of obesity and overweight in all racial/ethnic groups. Black non-Hispanic and Hispanic (compared to White non-Hispanic) children and those in families with lower SES are expected to experience more intense exposure to chronic stress through several pathways including material deprivation or exposure to racism and discrimination.[17, 18] Adding measures of stress to analytic models of obesity could account for some of the variance attributed to race/ethnicity or SES.

AIM 3 - Early Childhood SES and Subsequent BMI: Two comprehensive reviews cover nearly 500 cross-sectional studies from 1933 – 2004 with contemporaneous measures of SES and obesity.[19, 20] Only one of the two available reviews included studies of children, with approximately one-third of the studies showing no relationship, one-third a direct relationship and one-third an inverse relationship, which was more evident in studies of girls than of boys.[20] Inconsistent findings could be due to differences in SES measures, demographics or variables controlled. However, emerging evidence from longitudinal studies suggests that SES-obesity relationships may reveal themselves over time, with low SES in early life an important predictor of overweight or obesity in adulthood, independent of the effects of adult SES.[21-27] Similar findings of inverse SES-obesity relations have been demonstrated in longitudinal studies with measures of early childhood SES and obesity in later childhood.[28-31] However, results are inconclusive when both SES and obesity in childhood were measured.[25]

AIM 4 - Early Childhood Sleep Duration and Subsequent BMI: Most studies of sleep duration and childhood obesity are cross-sectional and it is unknown whether the relationship is causal.[32-34] A U.S. sample followed from birth to age 3 years, generated two studies which showed sleep duration <12 hours in infancy was associated with increased odds of overweight[33, 34] and with higher BMI z-scores and skin-fold thickness measured at age 3 years.[34] A third study of 150 children found children's hours of sleep at ages 3 – 4 years associated with increased odds of overweight at age 9.5 years. Finally, a study of 900 children in the UK, found shortened sleep duration at age 3 years was associated with obesity at age 7 years.[35] Longitudinal studies are needed to examine the independent effects of day-time and night-time sleep on obesity as no prior studies made this distinction. Although precise physiological functions of sleep are not fully understood, day-time sleep and night-time sleep may have independent functions and thus distinct effects on subsequent obesity.[36-38]

III. Study Design and Methods

A. Study Design: The research involved secondary analysis of data collected from two, large nationally representative surveys. Each specific aim used a prospective cohort design with linked maternal-child data to test the proposed associations.

B. Population Studied: Data were derived from two sources: 1) The BLS **National Longitudinal Survey of Youth (NLSY)**, Child Young Adult surveys (NLSY-CYA) linked to parent data in the NLSY79 cohort. Originally designed to track labor market behaviors and experiences, the NLSY79 is a nationally representative panel survey that follows 12,686 young adults, ages 14 - 22 in 1979, every year until 1994 and biennially thereafter. Attrition from the NLSY79 has been low with retention rates close to 90%.[39] The initial cohort included substantial over-samples of African Americans and Hispanics providing sufficient observations for stratified analysis in these subgroups. As of 2006, the 6,283 women followed in the NLSY79 had attained ages 41 - 49 and were mothers of 11,466 children, representing more than 90% of the births expected in this cohort.[40] These children comprise the NLSY-CYA cohort and, since 1986, have been assessed every two years on a broad range of factors that influence health and social, emotional and cognitive development. NLSY-CYA data are obtained from the children and their mothers during in-person, in-home interviews conducted by experienced, trained field staff using computer assisted personal interviewing and valid survey instruments and assessments. **2) The Panel Survey of Income Dynamics (PSID)** is a longitudinal survey directed by the National Science Foundation. Since 1968, 4,800 families have been followed. In 1997 and 2002 a **Child Development Supplement (CDS)**, funded by the NICHD, was administered to the primary caregivers of 3,563 children ages 0 - 13.[41] The CDS included detailed demographic data, psychological and behavioral assessment of parents and children and time diaries.

C. Sample Selection: AIM 1: The study sample included all children born to mothers followed in the NLSY79 cohort who were at least 10 or 11 years old by 2004 (n = 8,992). Because the NLSY-CYA data are collected biennially, children were assessed at age 10 or age 11. Children with missing height or weight were excluded from the analysis as their BMI (the dependent variable) cannot be computed (n =3,146). Too few with BMI classified as underweight[42] were available for separate analysis and were therefore excluded (n=365). The analysis sample included 5,481 children (2,766 girls; 2,715 boys) of whom 2,454 are White, non-Hispanic; 1,681 are non-Hispanic Black; 992 are Hispanic and 354 are other race/ethnicity. In one Aim 1 analysis, exposure to violent television content was considered as a plausible stressor in early childhood. To test this hypothesis, data from the PSID CDS were used as television content could not be ascertained from the NLSY. PSID CDS time diaries were completed by 2,569 families in 2002. Of these, 376 did not complete the 1997 time diaries, and an additional 92 were not assessed for BMI or were missing data for important covariates. Missing values were dealt with by case-wise deletion, which in observational data results in minimal bias. Underweight children (n = 24) were dropped from the sample leaving an analysis sample of 2,037. **AIM 2:** See Aim 1 (NLSY sample selection). **AIM 3:** See Aim 1 (NLSY sample selection). **AIM 4:** Of the 2,569 children in the PSID CDS-II, 990 were ages 0-59 months and 1,579 were 60 months or older in 1997. Children were excluded if they had implausibly low (<12 kg/m^2) or missing BMI in 2002, or missing Time Diary Data which provided the estimates of sleep duration. The total final sample size was 822 children ages 0 – 59 months and 1,108 children 60 months or older in 1997. We selected age 5 years as the sample cut-point to account for differences in sleep patterns between pre-school and school-age children because by age 5, the vast majority of children do not take naps.[38] Of the older children, 125 were missing BMI in 1997. Accordingly, the models that included baseline BMI were estimated with a sample size of 983. BMI in 1997 was not available for the younger children.

D. Instruments used: Outcomes: For all specific aims, age- and sex-specific body mass index z-scores (z-BMI) were developed using 2000 growth charts published by the Centers for Disease Control and Prevention[3] and child height and weight provided in the survey data. BMI z-scores were used rather than absolute BMI because child height and weight

increases as part of normal development and our sample combined boys and girls of different ages. In the NLSY-CYA, height and weight were both measured by the in-home interviewer using a portable scale and tape measure. Height was measured for more than 80% of the sample and weight for more than 75%; for the remaining children, height and weight were obtained by parent report.[43] In the 2002 wave of the PSID CDS both height and weight were measured. In 1997, the height of children 5 years and older was measured and their weight was recorded from parental report. To assess results in light of common definitions of obesity and overweight, BMI z-scores were also trichotomized based on established cut-points: normal weight (BMI < 85th percentile), overweight (BMI $\geq$ 85th and < 95th percentile) or obese (BMI $\geq$ 95th percentile).[1] We considered modifying the categorical variable for separate analysis of underweight children[42]; however, as too few observations in our data met these criteria, they were dropped from analysis.

Independent Variables: AIM 1: Stress was measured using items available in the survey data and plausibly associated with stress among children ages 0 - 6[44, 45]. We agreed with proposal reviewers that criteria for distinguishing between acute stress (i.e., circumscribed or discrete trauma) and chronic stress (i.e., ongoing and persistent frustrations and demands) in early childhood are arbitrary and not clear cut (e.g., death of a parent could result in acute stress as well as ongoing chronic stress as the living parent assumes the role of a single parent). Accordingly, we considered any plausible stressors in our analysis and did not attempt to distinguish between acute and chronic stress as originally proposed. Far fewer stress variables suggested in extant inventories[44, 45] were available and consistently measured in our cohort than originally anticipated. Using data collected for the NLSY, we measured: any change in marital status (separation, marriage, divorce), grandparent in the home, father (or mother) not present in the home, serious illness requiring hospitalization, injury requiring hospitalization, family lived in poverty, number of years of poverty. As multiple variables were measured as a proxy for a single underlying construct, we employed factor analysis to group the variables empirically. Using factor analysis with varimax rotation of stress measures adapted from prior work, we have identified 2 distinct uncorrelated stress constructs. These factors capture stress plausibly related to family structure (examples of variables with high factor loadings include change in marital status, father not present, grandparent living in household) and stress related to traumatic events (examples of variables with high factor loadings include illness and accident requiring medical attention or hospitalization). The NLSY-CYA includes the HOME-SF (a modification of the HOME inventory[46] as a unique age-specific observational measure of the quality of the cognitive stimulation and emotional support provided by a child's family. Our analysis considered scores on the Home-SF and on two subscales (cognitive and emotional) as measures of potential exposure to (versus buffering of) family-level stress. A measure of marital conflict was constructed as a plausible stress exposure for young children using a series of relationship satisfaction questions asked during interviews of mothers living with a spouse or opposite-sex partner (e.g. frequency the couple calmly discuss something, argue about money etc.). Analysis that considered marital conflict was restricted to children in families with both parents present. Using the PSID, we tested the associations of different content types of children's television viewing with subsequent body mass index (BMI) to assess the plausibility of different causal pathways in the television-obesity association. Time diary data from the 1997 and 2002 PSID asked parents to report their child's activities throughout the course of an entire day, for a randomly chosen weekday and weekend day September - May. Time diaries have excellent validity when compared to direct observation of activities.[47, 48] When the activity involved watching TV in any format, the parents were asked to report the format (i.e., TV or DVD or video) and the name of the show watched. These data were used to classify hours of TV viewing/day into 5 collectively exhaustive and mutually exclusive categories and to identify hours of exposure to violent (i.e. plausibly stressful) versus non-violent content.[49, 50] **AIM 2:** See

Aim 1 above. **AIM 3:** Early childhood SES was measured as the total mean family income at age 5 or 6, adjusted to represent dollars in 2006 and log-transformed to correct skewness. Categorical family income variables were also tested with total mean family income at age 5 or 6 adjusted to represent dollars in 2006 and categorized as low (<25th percentile), moderate (25 – 75th percentile) and high (>75th percentile). **AIM 4:** PSID CDS family time diary data were used to calculate average duration of day-time and night-time sleep in hours in 1997 and in 2002. The sleep variables were tested as linear, quadratic and cubic terms to allow for potential non-linearity in the relationship with obesity. Cubic and quadratic terms were tested for each sleep variable to yield the best model fit.

Covariates: All statistical models were adjusted for child and parent attributes that may confound the proposed relationships. Unless noted, none of the covariates had more than 10% missing data. For each, missing data points were imputed with the sample mean or mode. An alternative approach of dropping observations from the sample if covariates were missing produced similar findings. **AIM 1:** Covariates included the child's race/ethnicity and sex, total net family income at age 5 or 6 and at age 10 or 11 (adjusted to represent dollars in 2006 and log-transformed to correct skewness), mother's education, mother's BMI, family structure (# siblings, birth order, single parent status, age of mother), the child's baseline BMI measurement at age 5 or 6, the child's year of birth, year of the NSLY wave, region and urbanicity of residence. Unless noted all time-varying covariates are measured at age 10 or 11. In the analysis of television content and obesity, covariates included gender, age, and race/ethnicity, average sleep duration, time engaged in vigorous physical activity (none, 1 – 30 minutes per day, > 30 minutes per day), maternal body mass index (self-reported in 1999), maternal education, and child's baseline BMI in 1997. Because BMI was not measured in children under 5, it was not possible to include baseline BMI in the regression for children under 7. **AIM 2:** See Covariates Aim 1 above. **AIM 3:** See Covariates Aim 1 above. **AIM 4:** Regression models controlled for child age in 1997 (linear and quadratic terms in months), gender, race/ethnicity, birth order, hours per day of entertainment television and non-commercial television (e.g., educational programs or DVD) watched in 1997 and 2002, family income in 1997 (dollars, log-transformed), maternal education in 1997 (years, log-transformed), mother's and father's BMI and the child's birth weight. The parents' BMI is a proxy for both the diet and physical activity patterns in the household, as well as genetic factors that might influence the child's BMI. For children over age 5, the child's baseline BMI z-score was controlled to account for the possibility that an association between sleep duration and subsequent obesity reflects an unmeasured tendency of obese children to sleep fewer hours. BMI was not available for children under 5 in 1997; therefore, it was not possible to include baseline BMI in the models for this age group.

E. Statistical techniques employed: All analysis was conducted with Stata, Version 10.1 statistical software (College Station, TX). Descriptive statistics were computed. As needed, continuous variables were transformed to correct skewness. In all models, standard errors were corrected for family-level clustering using the Huber-White estimate of variance.[51] Sampling weights were used to account for the complex sampling designs and to allow inferences valid for the population. Because of the salience of potential effect modification by sex, partial f-tests were used to formally test the assumption that sex- or race-specific specific analyses are indicated. If significant, analyses were stratified and results presented accordingly. **AIM 1:** Continuous measures of z-BMI were modeled with linear regression. Categorical measures (normal weight = reference, overweight, obese) were modeled using multinomial regression. In the analysis of television content separate multivariate linear regressions were performed for children under age 7, and those 7 and over. **AIM 2:** To test for significant mediation of relations between race/ethnicity (or SES) and BMI by stress, regressions were develop to test conditions:[52, 53] **Condition 1:** SES (or race/ethnicity) is significantly associated with z-BMI in a regression

that does not include stress as a covariate. **Condition 2:** Stress is significantly associated with BMI. **Condition 3:** Stress is significantly associated with SES (or race/ethnicity). Maternal education and family income were considered separately, with each measured as: i.e. low <25th percentile of the study sample, moderate 25th – 50th percentile; high >50th percentile. **AIM 3:** See Aim 1. The estimated coefficients for early childhood SES were examined for direction, magnitude and significance. **AIM 4:** Logistic regression was used to model associations between 1997 sleep and 2002 weight classification, controlling for the covariates listed above. Analysis was developed separately for children who were ages 5 – 9 years in 2002 (0 – 59 months in 1997) and those who were ages 10 – 18 years in 2002 (60 – 154 months in 1997). Two models were estimated in the older cohort, one including baseline BMI and one without this variable. Results from the two models were compared to assess the contribution of baseline BMI to the estimated association between sleep duration and subsequent BMI. This comparison allowed us to gauge whether missing baseline BMI in the younger cohort is likely to affect the results. Results of the logistic regression were used to compute the predicted risk of obesity conditional on hours of sleep. The 95% confidence intervals for the relative risk estimates were bootstrapped with 1,000 repetitions. The estimates and 95% confidence intervals were graphed to illustrate the relative risk associated with obesity by hours of sleep. Results from this approach were similar to those derived from ordered logistic regression models.

IV. Detailed Findings

AIM 1: In the NLSY analysis of early childhood stress measures at age 0 – 6 years, there was no consistent pattern of a significant association with the child's z-BMI score at age 10 or 11 or with categorical measures of obesity or overweight measured at age 10 or 11 years. This finding held when the stress variables were modeled together, separately, as simple sums, or as factors. F-tests of effect modification by sex (but not by race/ethnicity) were significant; therefore, the analysis was developed separately by sex. Some of the stress variables were significant in some models. In girls, residence in a single-parent household in early childhood was associated with lower z-BMI at age 10 or 11 (β =-0.14, 95% CI: -0.27, -0.01) and living with a grandparent in the household was associated with higher BMI (B = 0.24; 95% CI: 0.04, 0.44) when the other available stress measures were controlled. Also significantly associated with z-BMI at age 10 or 11 were maternal overweight (β =0.19; 95% CI: 0.06, 0.32), maternal obesity (β = 0.47; 95% CI: 0.32, 0.61), maternal age at birth measured continuously (β = 0.03; 95% CI: 0.01, 0.04). Consistent with the effect of grandparents in the household, the factor measuring family structure was associated with higher BMI in girls (β =0.07; 95% CI: 0.01, 0.13). In boys, none of the stress variables were significant; however, maternal overweight (β =0.30; 95% CI: 0.17, 043), maternal obesity (β =0.52; 95% CI: 0.37, 0.66) and z-BMI at age 5 or 6 (β =0.28; 95% CI: 0.25, 0.32) were all strongly associated.

In the PSID analysis, violent television content was not independently associated with subsequent obesity in either age group in any of the models tested. Among children aged 0-6 in 1997, each hour/day of commercial viewing in 1997 is significantly associated with a 0.11-unit increase BMI z-scores in 2002 controlling for of the socio-demographic covariates, including mother's BMI. No other category of TV viewing was significant. Among children 7 and over, commercial viewing in 2002 was associated with 2002 BMI. All results were robust to the inclusion of exercise and eating while watching TV as possible mediators.

AIM 2: In unadjusted models, both Black/non-Hispanic children and Hispanic children had higher BMI z-scores than White non-Hispanic children (Black/NH boys: β =0.18; 95% CI: 0.05, 0.30; Black/NH girls β = 0.36; 95% CI 0.23, 0.48; Hispanic boys: β = 0.27; 95% CI: 0.13,

0.41; Hispanic girls β = 0.16; 95% CI: 0.003, 0.31). Those with other race/ethnicity had BMI z-scores no different than Whites. None of the SES variables (income, maternal education) were significantly associated with obesity at age 10 or 11 in boys. In girls, only high maternal education (16 years or more) was associated with lower z-BMI (β = -0.17; 95% CI: -0.31, -.02) as was being in the highest income category (β = -0.12; 95% CI: -0.24, -0.01). **Condition 1:** By adding the other (non-stress) covariates to the model, the disparity is eliminated for Black non-Hispanic boys (β =0.09; 95% CI: -0.04, 0.21) and for Hispanic girls (β = 0.02; 95% CI: -0.12, 0.16); however, the disparity persists in Hispanic boys (0.23; 95% CI: 0.09, 0.37) and Black/non-Hispanic girls (β = 0.16; 95% CI: 0.03, 0.28). Thus, condition 1 for mediation is met in some but not all sub-groups. The SES disparities were eliminated by the addition of the non-stress covariates to the model. **Condition 2**: Adding the stress variables to the model, separately and together (measured as individual variables or as factors) did little to change the adjusted estimates reported for Condition 1. Furthermore, none of the stress variables were significant in these regressions. **Condition3:** The stress variables were significantly associated with some but not all of the SES variables. The direction of the associations was not consistent. Some of the stress variables were more prevalent in high SES groups and some in low SES groups.

AIM 3: In models controlling for contemporaneous family income and other covariates, family income in early childhood (measured at age 5 or 6) was not significantly associated with z-BMI at age 10 or 11. This finding persisted when the models were repeated with measures of maternal education at age 5 or 6 as the measure of early childhood SES.

AIM 4: Duration of night-time sleep in early childhood was associated with subsequent obesity measured at ages 5 – 9 years. These findings persisted when controlling for contemporaneous sleep and other important confounding variables including parents' body mass index, family socioeconomic status (parental education, income, single parent) and hours of television viewed. Relationships between duration of sleep and obesity were significant and non-linear. The final model for the younger cohort included linear and quadratic terms in all sleep variables and cubic terms in 1997 day-time and night-time sleep. The joint effect of the three 1997 night-time sleep variables was significant within the range of the data. The optimal amount of night-time sleep was a little more than 10 hours, with an extra hour for children having less than this amount associated with reduced risk of obesity. Although the 1997 day-time sleep variables were individually significant, the direction of the association differed for the linear and cubic terms versus the quadratic term and the net effect of sleep was not significant. Night-time sleep in 2002 was not associated with obesity in this cohort. In the younger cohort, 1997 night-time sleep, 2002 night-time sleep and 1997day-time sleep were not significant when the models were re-estimated for obesity and overweight relative to normal weight. In adolescents aged 10 – 18 years in 2002, contemporaneous sleep was strongly associated with obesity while sleep five years prior had no significant effect. The final model in the older cohort included linear and quadratic terms in sleep. Contemporaneous sleep was strongly associated with obesity in 2002 while 1997 night-time sleep and 1997 day-time sleep were not significant. These findings were robust in models estimated with and without the inclusion of baseline BMI. As expected, parents' BMI was highly significant in the model without the adolescents' baseline BMI. In the older cohort, 1997 night sleep was significantly associated with overweight/obesity; whereas, napping and 2002 night sleep were not significant.

V. Discussion and Interpretation of Findings

A. Conclusions: In light of important study limitations described below, future research is required to confirm or refute the following conclusions: 1) Stress in early childhood (age 0 – 6)

is not significantly associated with z-BMI at age 10 or 11. While successful stress reduction or stress management interventions are available for children undergoing acute (e.g. medical and dental procedures) and chronic stress (e.g. chronic health conditions, parents' divorce),[54-58] such intervention may be of limited success in obesity prevention. A the same time, the effects of stress on obesity are not likely to be unidirectional—we found some stress variables were associated with higher and some with lower z-BMI at age 10 or 11 **(AIM 1).**. 2) Our analysis of television content in the PSID confirms the findings from the NLSY study, given that violent television content (a plausible stressor) was not associated with the development of obesity. This analysis also challenges the frequent contention that television viewing contributes to obesity because it is a sedentary activity. It was not television per se, but television advertising that was associated with obesity. This result was robust in the presence of controls for potential confounders and to the inclusion of possible mediators, including the child's baseline BMI and the frequency of eating in front of the television. Consistent with expectations based on children's different cognitive ability to understand advertising before and after age 7, our analysis finds a slightly stronger association of commercial content with obesity before age 7 than afterward **(AIM 1).** 3) Exposure to stress is not a significant mediator of racial/ethnic or SES disparities in obesity **(AIM 2).** 4) Early childhood family income (age 5 or 6) is not associated with subsequent obesity when current family income is controlled. **(AIM 3)** 5) Sleep duration is a modifiable risk factor with important implications for obesity prevention and treatment. Our findings suggest that there is a critical window prior to age five years when night-time sleep may be important for later obesity status. In our models, the estimated optimal amount of night-time sleep at age 0 – 4 associated with subsequent obesity was approximately 9.5 hours, with additional hours associated with a reduction in the risk of obesity **(AIM 4).** 6) Our findings also indicate that day-time sleep had little effect on subsequent obesity at any age. This result suggests that napping is not a substitute for night-time sleep. There is some evidence that night-time sleep and naps serve different physiological functions. Naps may reduce day-time psychosocial stress, increase attention span and increase alertness for learning, while nocturnal sleep involves complex biological, psychosocial and restorative functions.[36-38] Problem napping and disruptive behaviors are associated with higher cortisol levels and shorter nap duration[36] **(AIM 4).**

B. Explanation of study limitations: Study results must be interpreted in light of several limitations: 1) All analysis used observational data and can only test associations, not causality. Instrumental variables analysis could lead to insights about causal relationships, but we know of no valid instruments available for our questions. 2) Our measures of stress were not significantly associated with childhood obesity. We interpret these findings cautiously in light of the adult-centric nature of measures of stress for children which may not capture the exposure in question. We examined only one time period for the exposures (age 0 – 6) and the outcome (age 10 or 11). The associations we tested may be significant in other time periods or with more robust measures of stress. The effects of early childhood exposure to adversity may manifest over longer time periods. For instance, others have shown significant associations between several types of abuse (physical, verbal, witnessed) prior to age 16 years and obesity at age 45.[59] 3) PSID time diary data (used for television viewing and sleep) measure only two days per child per wave, thus there is considerable room for measurement error. 4) Although the sample sizes were relatively large, they were not large enough to drill down on some gender-racial/ethnic- or socio-economic subgroups for some analysis or to examine children with BMI classified as underweight. 5) BMI at baseline was not measured for the younger children in the PSID. We controlled for parents' BMI, however, which is positively associated with child BMI [60] and is expected to capture much of the variance associated with the tendency of children to be overweight at such a young age. 6) Physical activity and diet are potential confounders of the

associations of stress, television or sleep with obesity. These variables are not available in the NLSY and few such variables are available in the PSID. Those in the PSID are of insufficient quality; however our results using the PSID were robust to the inclusion of diet and physical activity variables available. We are aware of no datasets that include measured height and weight plus measures of stress, diet and physical activity. We combined two datasets for this study to test stress-obesity relations with the best available data.

C. Comparison with findings of other studies:

AIM 1: We found no consistent association between available measures of stress in early childhood and the development of obesity in young children. There is little extant work with which to compare these findings. A small group of studies examined relations between obesity and family factors that plausibly reflect exposure to stress[61-65] with mixed findings and no definitive factor or pattern of family dynamics consistently associated. Our findings add to this literature. Results of our analysis of television content are consistent with prior research.[66, 67] Most convincingly, two randomized trials to reduce television viewing found statistically significant effects on calorie intake and obesity, but not on physical activity.[68, 69] The results of these trials, together with the evidence from our very different approach make a strong case that television viewing does not affect obesity through pathways involving stress or reduced physical activity. **AIM 2:** We are aware of no prior studies that have considered stress as mediator of disparities in obesity by SES or race/ethnicity. **AIM 3:** Consistent with some but not all prior studies[20, 25], we found that childhood SES was not associated with childhood obesity at age 10 or 11. Cross-sectional studies of children, recently reviewed[20], have mixed findings but do not offer the best point of comparison for our results. Results of longitudinal studies have demonstrated inverse SES-obesity with measures of early childhood SES and obesity in later childhood.[28-31] However, results are inconclusive when both SES and obesity in childhood were measured[25] as we did in our study. Others have shown associations between childhood weight and socioeconomic status on adult BMI measured later than age 20.[26, 70] Together, with our findings this suggests that the effects of early childhood SES on subsequent weight status may manifest later in life than age 10 or 11. In this case, early obesity prevention efforts among children from low income families could prove fruitful. **AIM 4:** Our finding that sleep duration in early childhood was associated with subsequent obesity is consistent with other work in cohorts of different ages. A U.S. sample followed from birth to age 3 years, generated two studies which showed sleep duration of less than 12 hours in infancy was associated with increased odds of overweight[33, 34] and with higher BMI z-scores and skin-fold thickness all measured at age 3 years.[34] A third study of 150 children found that hours of sleep at ages 3 – 4 years was associated with increased odds of overweight at age 9.5 years. Finally, a study of 900 children in the UK, found shortened sleep duration at age 3 years was associated with obesity at age 7 years.[35] to our knowledge ours was the first study to consider independent effects of day-time and night-time sleep on obesity. Although the precise physiological functions of sleep are not fully understood, day-time sleep and night-time sleep may have independent functions and thus distinct effects on subsequent obesity.[36-38] Interestingly, in our analysis of sleep duration, several variables associated with obesity in other work—including race/ethnicity, socioeconomic status[1]—were not significant in our models. Significant racial/ethnic disparities have been reported in nocturnal sleep duration.[71] Consistent with our results, sleep duration may be a plausible contributor to racial/ethnic disparities in obesity documented elsewhere.

D. Possible application of findings to actual MCH health care delivery situations:

Findings from our studies of the effect of sleep and television on obesity have direct implications for MCH healthcare delivery settings. Parents should be instructed that sleep duration—especially in early life—is a modifiable risk factor with potentially important implications for

obesity prevention and treatment. Intervention to promote sleep in early life could have lasting effects on obesity. In counseling parents, it is important to note that sleep duration of longer than 9.5 hours may be required for other benefits.[38] Parents should also understand that napping is not likely a substitute for sufficient night-time sleep. While television viewing is a sedentary activity, we find it is not for that reason that it is associated with obesity in children. The relationship between television viewing and obesity among children is limited to commercial television viewing, and probably operates through the effect of advertising obesigenic foods on TV. The current emphasis on reducing sedentary activities—particularly television—may be misplaced. It may be more effective to focus on promoting physical activity directly, than to try to limit television viewing. At the same time, steering children away from commercial television may have a meaningful effect in reducing childhood obesity. How parents talk to their children about advertising could be an important mediator of advertising's influence on children's choices.

E. Policy implications: It may be appropriate to limit the advertising of obesigenic foods on television targeted to children. Given the huge sums spent by advertisers to fund commercial children's programming, such a policy change will be politically difficult, but given the enormous costs to society of obesity, it may be worth pursuing. The practice implication is that primary care providers and others who advise parents may find it easier—and just as effective for obesity outcomes—to steer parents away from commercial programming as away from television altogether. The existence of many high-quality, enjoyable, and educational programs available on DVD for all ages should make it relatively easy for health-educators and care-providers to nudge children's viewing toward less obesigenic television content.

F. Suggestions for further research: Taken together, our findings suggest several areas for future research. 1) Study the nature of stress in early childhood by race/ethnicity and SES. In our data, the direction of the associations was not consistent. Some of the stress variables were more prevalent in high SES groups and some in low SES groups. 2) Test longitudinal stress-obesity relations with other (ideally direct) measures of stress, and over longer time horizons. 3) Analyze the longer-term and real-world effects of advertising on obesity. Food advertisers have extensive presence on the internet, broadcast television, movies, video games, and web and cell-phone advertising. 4) Study prospective sleep-obesity relations in other cohorts and over longer time horizons. 5) Assess whether obese adolescents sleep fewer hours due, for instance, to greater predisposition to sleep apnea or whether the finding that contemporaneous sleep is inversely associated with obesity among adolescents reflects a true protective effect of sleep on obesity.

VI. List of products

The research resulted in three scientific papers for publication in peer-reviewed journals. Of these, one is close to submission, one is currently under review and one is forthcoming in the American Journal of Public Health.

1. Bell JF, Zimmerman FJ. Exposure to Stress in Early Childhood SES and Body Mass Index at Age 10 or 11 (in progress)
2. Bell JF, Zimmerman FJ. Sleep Duration in Early Life is Associated with Subsequent Obesity (Under review)
3. Zimmerman FJ, Bell JF. Only Commercial Television Viewing is Associated with Obesity in Children, forthcoming *American Journal of Public Health.*

Findings from Paper 2 will be presented at the upcoming MCH Epidemiology Conference in Tampa, FL, December 9 – 11.

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