

9-23-2015

The Role of Residential Segregation in Explaining Racial Gaps in Childhood and Adolescent Obesity

Igor Ryabov

The University of Texas Rio Grande Valley, igor.ryabov@utrgv.edu

Follow this and additional works at: https://scholarworks.utrgv.edu/soc_fac



Part of the [Diseases Commons](#), and the [Sociology Commons](#)

Recommended Citation

Ryabov, I. (2018). The Role of Residential Segregation in Explaining Racial Gaps in Childhood and Adolescent Obesity. *Youth & Society*, 50(4), 485–505. <https://doi.org/10.1177/0044118X15607165>

This Article is brought to you for free and open access by the College of Liberal Arts at ScholarWorks @ UTRGV. It has been accepted for inclusion in Sociology Faculty Publications and Presentations by an authorized administrator of ScholarWorks @ UTRGV. For more information, please contact justin.white@utrgv.edu, william.flores01@utrgv.edu.

Residential Segregation and Racial Gaps in Childhood and Adolescent Obesity

Abstract

The present study used nationally-representative data from the U.S. Panel Study of Income Dynamics (PSID) merged with census-track data from the American Community Survey (ACS) to model race-ethnic disparities in overweight, obesity and obesity-related disease among children and adolescents as a function of neighborhood race-ethnic segregation, socioeconomic status, household size and structure, family history of obesity and other important predictors. Results indicate that African-American and Hispanic children and adolescents are more likely to suffer from obesity and obesity-related disease than their non-Hispanic white peers. We also found that race-ethnic segregation proxied by the Index of Dissimilarity has a strong and negative effect on the weight status and health outcomes mentioned above. Moreover, race-ethnic segregation appears to explain up to 20% of the difference between minority children and their non-Hispanic white peers in the prevalence rate of overweight, obesity and obesity-related disease.

Keywords: adolescent obesity, racial disparities in health, residential segregation.

INTRODUCTION

During the past three decades, the United States has witnessed an alarming increase in the prevalence of obesity, which has become a major public health concern (Ogden *et al.*, 2010; Ogden *et al.*, 2014). An estimated two-thirds of U.S. adults are overweight or obese, and this proportion is expected to continue to increase (Wang & Beydoun, 2007). A critical period for the development of obesity is childhood and adolescence (Bae *et al.*, 2014; Reilly *et al.*, 2005). In the United States, the share of adolescents aged 12–19 years who were obese increased from 5% in 1980 to nearly 21% in 2012. Likewise, the share of children aged 6–11 years who were obese increased from 7% to nearly 18% over the same period (Ogden *et al.*, 2014). Childhood obesity is not only a major antecedent of adult obesity, but also poses long-term consequences through the accumulation of various health risk factors over the life course (Biro & Wien, 2010; Blazer *et al.*, 2002; Reilly *et al.*, 2005). Childhood and adolescent obesity has been also found to co-occur with several physical and mental health problems (Dixon, 2010; Krebs *et al.*, 2007). Obese children and adolescents are at high risk for diabetes, hypertension, heart disease, and depression (Bae *et al.*, 2014; Gable & Lutz, 2000; Krebs *et al.*, 2007).

Sparked by a surging epidemic of obesity, a burgeoning literature on the topic has emerged in the United States. Specifically, a substantial body of research has focused on race-ethnic disparities in obesity and their determinants (e.g., Braveman *et al.*, 2010; Williams & Collins, 1995; Williams & Sternthal, 2010; Zhang & Wang, 2004). Despite the increased attention to social determinants of race-ethnic disparities in obesity in recent years, the impact of racial segregation on obesity among children and youth did not feature prominently in this literature. Prompted by a dearth of studies on this issue,

this research attempts to establish whether racial segregation as a neighborhood-level factor is an important risk factor for being overweight and obese among children and adolescents in the United States. Apart from racial segregation, which is analyzed herein as a neighborhood-level factor, this study seeks to assess the relative importance of family socio-economic status, family structure and size, family history of obesity and other socio-demographic determinants of obesity and chronic conditions associated with it. The results of this study will contribute to a better understanding of risk factors of adolescent adiposity and thus allow for better targeting of early interventions.

REVIEW OF RELEVANT RESEARCH

Race-Ethnic Gaps in Obesity

Although many factors contribute to the obesity epidemic, many researchers agree that obesity is a social disease, meaning that it mostly stems from larger social issues such as perpetual social inequalities based on race-ethnicity, socioeconomic status, gender, etc. (Williams & Collins, 1995; Wyatt *et al.*, 2003). Race is one of the major bases of division in American life and has been a critical factor in the social and economic structure of American society from its pre-colonial beginnings to the present (Bonilla-Silva, 2006; White & Borrell, 2011). Furthermore, in the United States race has traditionally been a more important determinant of individual life chances than socioeconomic status (Saperstein, 2012). Race is an antecedent of socio-economic status because institutionalized racism has blocked upward mobility for members of minority groups (Feagin, 2014). Given the history of racism in the United States and the ongoing reality of racial discrimination, it is not surprising that throughout U.S. history racial disparities in health have been pervasive (Subramanian *et al.*, 2005; Williams & Collins,

1995). These disparities are a product of the race privilege according to which whites have always been at the top, blacks at the bottom, and other groups in between. Even now, after years of policies that promoted equality of opportunity, by every measure non-Hispanic white Americans enjoy better health than their black co-nationals (Williams & Sternthal, 2010).

Traditionally, racial segregation has been a key mechanism by which racial inequality has been created and reinforced (Williams & Sternthal, 2010). Some researchers (e.g., White & Borrell, 2011; Williams & Collins, 2001) argue that racial disparities in health reflect, in part, the legacy of racial segregation which limits access to quality care for minority groups. Moreover, earlier studies have noted that segregated communities are unable to meet their residents' dietary needs (Larson *et al.* 2009; Story *et al.*, 2008) because of a high concentration of fast food outlets (Morland & Evenson, 2009; Wang *et al.*, 2007) and a lack of community safety for outdoor physical activities (Kipke *et al.*, 2007; Smith *et al.*, 2011). Although there is limited literature on this topic (e.g., Larson *et al.*, 2009; Subramanian *et al.*, 2005; Williams & Collins, 2001), the relationship between racial segregation and childhood obesity has drawn little attention. To the best of our knowledge, there is only one study that explicitly tested the hypothesis whether and how that racial residential segregation contributes to obesity epidemic. Using Behavioral Risk Factor Surveillance System (BFRSS) data, Chang (2006) conducted multilevel analyses to compare weight status of non-Hispanic blacks and whites. Using racial isolation index as a neighborhood-level factor, she found that among non-Hispanic blacks, higher racial isolation is positively associated with both a higher body mass index (BMI), after adjusting for multiple covariates, including measures of

individual socioeconomic status. It is also worth mentioning a rather similar study by Wickrama *et al.*, (2006) which, using the same dataset (BFRSS), examined the influence of community poverty on adolescent weight status. The authors found that adolescents living in poor communities were more likely to be obese. Building on these studies, we also use multilevel regression whereby the socio-economic effects at the individual level are nested at the neighborhood level. This technique can give us a better understanding of the relationship between racial segregation as a neighborhood-level factor and obesity as an individual-level outcome. However, the present study differs from Chang's (2006) study in that our focus is not on adults, but on children and adolescents. Moreover, in contrast to Wickrama *et al.* (2006), we focus on residential segregation and not on community poverty, and we cover several obesity-related health outcomes of children as well as adolescents. Furthermore, unlike the two aforementioned studies, our study is longitudinal which will enable identifying causal factors which exert their influence over a prolonged period of time.

Other Risk Factors for Obesity

Although the favored model of risk for obesity is social, genetic factors have also been cited as those related to obesity in children and adolescents (Sørensen *et al.*, 1989; Strauss & Knight, 1999; Reilly *et al.*, 2005). For example, Davis *et al.* (2008) showed that parents' and grandparents' weight status also plays an important role among the causes of obesity. Another group of risk factors that has received prominence in the discussions of interventions to curb diabetes epidemic is related to socio-economic status (e.g., Bae *et al.*, 2014; Levine, 2011; Zhang & Wang, 2004). Structural factors, such as parental income and education, are known to determine level of access to sedentary and

fast-food lifestyles, with decreased physical activity opportunities and increased availability of high-fat energy-dense foods (Biro & Wien, 2010; Chang, 2006; Gordon-Larsen et al., 2003; Reilly *et al.*, 2005). More generally, it has been suggested that family socio-economic status is inversely related to prevalence of being overweight (Levine, 2011; Zhang & Wang, 2004). Apart from socio-economic status, family structure and size can affect the odds of overweight and obesity in children and adolescents (Chen & Escarce, 2010; Gable & Lutz, 2000). Generally, it has been documented that the presence or absence of a parent, as well as number of siblings may impact weight status of children (Chen & Escarce, 2010). Particularly, a strong relationship between single-parent status and excess weight in children has been suggested by Strauss & Knight (1999). Single parent households and large households have a tendency to favor the consumption of prepared food items, which tend to be high in fat and sodium (Gable & Lutz, 2000).

Hypotheses

On the basis of previous studies (Choi, 2008; Wexler & Pyle, 2012), we know that there exist race-ethnic disparities in overweight, obesity and obesity-related disease among children and adolescents in the U.S. The main objective of this study is to evaluate the relative roles of neighborhood race-ethnic segregation and individual-level factors (socioeconomic status, household size and structure, family history of obesity) as determinants of these disparities. Our main line of inquiry can be framed by two alternative hypotheses. The null hypothesis simply assumes that race-ethnic disparities in overweight, obesity and obesity-related disease among children and adolescents are fully explained by individual-level factors, such as socioeconomic status, household size and structure, family history of obesity. The alternative hypothesis is that the observable race-

ethnicity disparities in overweight, obesity and obesity-related chronic disease are not fully explained by the aforementioned socio-demographic characteristics and family history but attributable to persistent racial segregation of residential areas in the U.S.

METHODS

Data and Sample

The individual-level data were obtained from the Panel Study of Income Dynamics' (PSID's) Transition to Adulthood (TA) and Child Development Supplement (CDS) studies. The PSID is an ongoing longitudinal survey, begun in 1968, of a representative sample of U.S. individuals and their families. The initial PSID sample consisted of two independent samples: a cross-sectional, stratified national sample and a national sample of low-income families. Both samples that are combined into the PSID sample are probability samples. Thus, the PSID's initial households constitute a national probability sample of U.S. households as of 1967. It is worth mentioning that the original PSID sample contains too few Hispanics to draw reliable estimates about the U.S. Hispanic population. Moreover, the initial PSID sample is not representative of individuals who immigrated to the U.S. after 1968. To remedy these shortfalls, the PSID added 2,043 Hispanic households in 1990. Still, the PSID study undersampled post-1968 immigrants, particularly Asians. Due to a lack of sufficient funding, the Hispanic sample was dropped from the PSID study after 1995. As of now, the share of Hispanic and Asian households in the PSID study is disproportionately lower than their percentage of the U.S. population.

The TA study is part of PSID and a longitudinal follow-on to the Child Development Supplement (CDS). The CDS is an in-home interview survey of caregivers

(parents or guardians) about children and adolescents aged 5-18 years conducted in 1997, 2002/2003 or 2007. All TA participants had a completed family-level baseline interview in the CDS. This means that each TA sample member had information from the primary caregiver interview about the TA individual when they were a child. The TA study was initiated in 2005 when the oldest CDS respondents reached 18 to 20 years of age. The TA study has subsequently been conducted in 2007, 2009, and 2011. For this study, we use the most recent TA study conducted in 2011 (TA-11). Individuals were included in the TA-2011 sample if they completed a 2011 core PSID 2011 interview and CDS interview either in 1997, 2002/2003 or 2007. The response rate for the TA survey was 92%.

All of our analyses are conducted using the STATA 10.1 software. In order to account for unequal probability of selection and survey non-response, our analyses are based on weighted data. Further, cases with missing values one or more of the dependent variables were deleted, resulting in a total of 1,931 cases for the analysis. We also dropped 19 cases (approximately 1% of the sample) belonging to other race-ethnic groups rather than non-Hispanic whites, African-Americans and Hispanics. Hence the final sample comprised 1,910 respondents. Auxiliary analyses (not shown for parsimony) confirmed that this deletion did not result in any significant change in the means of the dependent variables. Missing values for all continuous individual-level variables were imputed by the Markov Chain Monte-Carlo technique (for more information on Monte-Carlo imputation see Rubin, 2004). This imputation technique yielded successive simulations of the distribution of missing values, assuming that the data are missing at random.

With the exception of the outcome variables – adolescent overweight, obesity and health status – and independent variables monitoring residence and marriage statuses which come from TA-11, the CDS data were used in the present study as they provide the most complete information on the key variables of interest. The average age at time of interview for CDS was 14.2. The sample consists of 52% females. Approximately 50% of the sample is non-Hispanic white, 42% is African American and 8% Hispanic.

The PSID record each household's census tract and metropolitan area of residence. Using this information, we appended to each household's data record information describing the neighborhood characteristic of the census tract. As in most prior work in this area, we use census tracts as our approximation of neighborhoods. Tract-level data were obtained from the American Community Survey (ACS) summary file.

Measures

The dependent variables in our analyses are adolescent adiposity, proxied by weight status (overweight and obese), and the probability of being diagnosed with obesity-related chronic condition (asthma, diabetes or hypertension). Body mass index (BMI) was calculated as an indicator of adolescent adiposity using self-reported height and weight measures. Following CDC guidelines, overweight is defined as a BMI at or above the 85th percentile and lower than the 95th percentile for children of the same age and sex, and obesity is defined as a BMI at or above the 95th percentile for children of the same age and sex. CDC growth charts were used to define sex-specific cut-points for BMI (Kuczmarski *et al.*, 2000; Krebs *et al.*, 2007).

In order to avoid the observation of spurious associations between race-ethnicity (both as an individual- and neighborhood-level structural predictor) and obesity, this study controlled for different factors related to both. Hence, the individual-level characteristics used as control variables included parental income, educational attainment and employment status; household structure categorized as living with two parents (reference), single mother or other family arrangements; number of siblings; family history of obesity; age, sex (reference: male); residence status (living with parents or not) and marital status (reference: not married). For the exception of the latter two predictors (residence and marital status), all independent variables were obtained from the CDS questionnaire.

The Index of Dissimilarity (ID), perhaps the most commonly used measure of residential segregation in American social science since the 1950s, was used to estimate neighborhood residential segregation. Based always on a comparison of *two groups* (e.g., whites vs. blacks), this measure is very easy to interpret. The ID ranges from 0 to 1, and tells us the percentage of a given ethnic that would have to change its residential location in order to balance out the distribution of these groups across the geographic space. In this study, we used the ID's of specific minority groups (e.g. blacks, Hispanics) versus whites.

It is worth mentioning that neighborhood effects on weight status are small compared to individual effects in the study sample. An analysis of the intraclass correlations (not shown) reveals that 10.9 and 11.2% of the variance in childhood overweight and obesity, correspondingly, is accounted for by differences in the characteristics of the neighborhoods where PSID participants lived. It also means that,

owing to lack of statistical power, only a single predictor variable could be entered into the neighborhood-level models while controlling for demographic and family background variables. At the exploratory stage we considered a range of neighborhood-level predictors available from the most recent ACS tract-level summary file. Among the factors that we considered were: (1) the percentage of adults with less than a high school education; (2) the percentage of households with income below the poverty line; (3) the percentage of single-parent households; and (6) median household income. Still, the exploratory analyses (not shown) indicated that the most significant factor at the school level was the ID. Hence, our choice of a neighborhood-level predictor – the Index of Dissimilarity (ID) is not trivial.

Analytic Strategy

Logistic regression models were used to investigate race-ethnicity disparities in overweight, obesity and obesity-related chronic disease in relation to family socio-demographic, genetic, other contextual and neighborhood-level (residential segregation) correlates. Several sets of multivariate analyses are reported below. Parallel analyses are estimated for all three dependent variables, represented by the odds of being: (1) overweight, (2) obese, and (3) diagnosed with obesity-related chronic condition. Households were used as level-1 units and census tracts were used as level-2 units.

Because the health outcomes listed above were measured at two points of time – when participants were children (at CDS) and when they were adolescents (at TA) – separate regression models were designed for children and adolescent outcomes. Models 1-3 are identical for both CDS and TA outcomes. The base Model (Model 1) includes only race-ethnicity dummies. Dummies for African Americans and Hispanic race-

ethnicity (level-1 variables) were entered first so that the subsequent variables predicted any variance not accounted for by these variables. Indicators related to socio-economic status – parental income, educational attainment and employment status – are entered in Model 2. Model 3 adds other individual-level controls: family structure and size, family history of obesity, age and gender. Model 4 predicting health outcomes measured at CDS includes index of dissimilarity. This Model is identical to Model 5 for TA outcomes. Model 4 predicting health outcomes at the time when participants were adolescents (i.e., at the time of the TA survey) includes residence status (whether a participant lived with her/his parents or not) and marital status.

RESULTS

Descriptive Results

Descriptive statistics for the sample, by race-ethnicity, are provided in Table 1. The Table documents significant discrepancies between non-Hispanic white, African American and Hispanic children and adolescents in all three outcomes of interest – prevalence of overweight, obesity and obesity-related chronic disease. The sample estimates for the prevalence of overweight and obesity are generally in line with the national averages reported earlier (Ogden *et al.*, 2014). Approximately 11-12% of non-Hispanic white children (at CDS) were overweight. In contrast, around 17% of non-Hispanic black and 18-19% of Hispanic children were overweight. It is important to note that prevalence of overweight among non-Hispanic white children did not increase between two points of measurement – the CDS and TA. This was not the case, however, for African American and Hispanic children. Although African American prevalence of obesity was up only slightly at the time of TA, the percentage of overweight among

Hispanic children had risen 1%. A somewhat similar dynamic was observed with obesity. While the period between the CDS and TA showed increases of at least 1% in the prevalence of obesity for both African American and Hispanic children, the percentage of obese among non-Hispanic children (at CDS) and adolescents (at TA) was approximately the same. Furthermore, the disparity of 6-9% in the prevalence of obesity between non-Hispanic whites, on the one hand, and African American and Hispanics, on the other, was similar in magnitude to the difference in the prevalence of obesity-related chronic disease between these race-ethnic groups.

[Table 1 is about here]

Race-ethnic disparities were observed not only in the outcome variables, but also in the independent variables. For example, parental income differed noticeably among the race-ethnic groups. Incomes for African Americans and Hispanics were approximately 23 and 12% lower than non-Hispanic white income. Average level of mother and father education was noticeably higher among non-Hispanic whites than among African Americans and Hispanics. Similarly, non-Hispanic white children were found to be at advantage over their African American and Hispanic peers with respect to parental employment status, living arrangements (the majority of African American children had not resided in two parent families), number of siblings (Hispanic children had more siblings than the other two race-ethnic groups) and family history of obesity.

Multivariate Results

Tables 2, 3 and 4 present our multivariate analyses which model race-ethnic disparities in the prevalence of overweight, obesity and obesity-related disease as a function of individual-level controls and race-ethnic residential segregation. Each table

reports the difference in odds ratios of being overweight, obese and having an obesity-related disease between non-Hispanic whites, on the one hand, and African Americans and Hispanics, on the other. For reasons of parsimony, the coefficients for the control measures are not presented. However, these variables generally conformed to expectations derived from the literature, as negative associations were found between the three dependent variables and parental income, educational attainment and employment status. Living in a single-mother household and having obese parents or grandparents were also found to statistically significant predictors of overweight, obesity and obesity-related disease among children and adolescents alike. Most importantly, we found that the index of dissimilarity is an important risk factor of overweight, obesity and obesity-related disease. The odds of being diagnosed with obesity and obesity-related disease among children and adolescents, regardless of their race-ethnicity, residing in more segregated neighborhoods were higher than among their peers from less segregated neighborhoods. Complete results are available upon request from the author.

Model 1 of Table 2 (Panel A) shows the odds of being overweight when the study participants were children at the time of CDS survey. The results indicate that, in absence of any controls, both non-Hispanic blacks and Hispanics are approximately 1.5 more likely to be overweight than non-Hispanic white children. The addition of variables monitoring family socio-economic status in Model 2 results in a noticeable decline of the odds ratios for both minority groups. The further expansion of the base Model through the inclusion of family structure, number of siblings, family history of obesity and other individual-level controls causes the discrepancy between non-Hispanic whites and minorities to decrease somewhat (see Model 3). The Index of Dissimilarity (ID) is

introduced in Model 4 of Panel A. Its effect eliminates the race-ethnic differences in the prevalence of overweight among children. In real terms, the odds ratios decline to 1.14 for African American and 1.11 for Hispanics.

[Table 2 is about here]

Comparing the results presented in Panels A (study population: children) and B (study population: adolescents) of Table 2, it is hard not to notice a high degree of similarity between the base Models of both Panels. The estimated race-ethnic disparities in the prevalence of overweight are approximately of the same magnitude among children as well as adolescents. That is, as compared to non-Hispanic white children and adolescents, their African American and Hispanic same-age peers are approximately 1.5 times more likely to be overweight (if none of the controls are accounted for). Put differently, race-ethnic disparities in overweight persist almost unchanged from childhood to adolescence. Quite similarly to the results of Panel A, the inclusion of variables monitoring family socio-economic status significantly alters the odds ratios for both African Americans and Hispanics. In fact, the largest drop in magnitude of the odds ratios is observed between Models 1 and 2 in both Panels of Table 2. This indicates that, as expected, parental income, education and employment status are important predictors of the prevalence of overweight in children and adolescents and, as such, these predictors exert a mediating effect on the relationship between race-ethnicity and weight status. The next two sets of independent variables entered in Models 3 and 4 of Panel B were also found to be important mediators of the race-ethnicity effect on the prevalence of overweight. Put differently, family structure, number of siblings, marital status and other predictors entered in Models 3 and 4 explained some of the variance in overweight

attributed to race-ethnicity. The odds ratios for African Americans and Hispanics declined steady from approximately 1.35 in Model 2 to 1.25 in Model 4. With the inclusion of ID in Model 5 of Panel B, the odds ratio dropped even further, thus rendering the race-based differences in overweight insignificant.

Turning to Table 3 which documents the race-ethnic discrepancies in obesity, we notice that in the base Models of both Panels A and B: (1) Hispanic-white disparity is larger than black-white disparity in obesity; and (2) the odds of being obese are higher among race-ethnic minorities than the odds of being overweight (if compared to non-Hispanic whites). The logistic regression results presented in Models 1 of Panels A and B of Table 3 show that, in the absence of any controls, African American children and adolescent are more than 1.6 and 1.7 times, respectively, are more likely to be obese than non-Hispanic whites. The corresponding numbers for Hispanic children and adolescents are 1.7 and 1.9 times.

Parallel to the results presented in Table 2 (outcome: the odds of overweight), parental income, education and employment status, the measures of socio-economic status, have a noticeable mediating effect on the relationship between race-ethnicity and weight status. As evidenced by the drop in the odds ratios for African Americans and Hispanics, family structure, number of siblings, family history of obesity, age and gender, too, explain some of the association between race-ethnicity and obesity. These mediating effects are similar in magnitude in both Panels A and B of Table 3, confirming that a significant share of the association between race-ethnicity and obesity among children and adolescents, is attributable to family socio-economic status, family structure and other individual-level risk factors. The introduction of the Index of Dissimilarity in

Model 4 of Panel A alters significantly the odds ratios for both African Americans and Hispanics, but this mediating effect of ID is more pronounced in the case of African Americans than in the case of Hispanics. As compared to Model 3 of Panel A, the odds ratio for African Americans in Model 4 that includes ID decreases 0.16 points, while that for Hispanics 0.12 points. A somewhat similar picture is observed in Panel B that shows the logistic regression models predicting the odds of adolescent obesity. Hence, it is reasonable to suggest that the portion of the race-based difference in overweight and obesity vis-à-vis non-Hispanic whites that ID explains is greater for African American than for Hispanics.

[Table 3 is about here]

The next set of multivariate regression analyses shown in Table 4 predicts race-ethnic gaps in the prevalence of obesity-related chronic condition (asthma, diabetes or hypertension) among children (Panel A) and adolescents (Panel B). In the base Models of Panels A and B, the estimated odds ratio for both African Americans and Hispanics are strongly significant and range from 1.63 (Hispanic effect: children) to 1.96 (African American effect: adolescents). In essence, if no controls are included in the regression models, the gap between African American and non-Hispanic white adolescents in the prevalence of obesity-related chronic diseases is estimated to be almost twice as large. The corresponding difference between African American and non-Hispanic white children is smaller but still noteworthy – approximately 1.7 times. Although the Hispanic effect is lesser in magnitude, if compared to the African American one, it is still very significant. Hispanic children and adolescents are 1.6 and 1.7 times, respectively, more likely to be diagnosed with asthma, diabetes or hypertension than non-Hispanic white

children and adolescents. The introduction of socio-economic status measures in Model 2 of both Panels A and B significantly reduces the estimated race-ethnic gaps in chronic disease. Generally, as in previous analyses (see Tables 2 and 3), the effects for parental income, educational attainment and employment status are consistent across models. Likewise, the addition of other individual-level controls in Model 3 of Panels A and B further reduces the aforementioned race-ethnic disparities. Across models, family socio-economic status and other control measures entered in Models 2 and 3 reduce the race-ethnic gaps in chronic disease by approximately 15%. Residence status (living with parents or separately) and marital status at the time of TA entered in Model 4 of Panel B were also found to reduce the race-ethnic gaps in obesity-related disease. However, the effects for residence and marital statuses were uneven across race-ethnic groups. Adjusting for the aforementioned variables lowered the odds ratio for African Americans more (by 0.2 point) than that for Hispanics (by 0.04 point). The same is true about the effect of ID. The adjustment for the ID decreased the odds ratio for African American children (Panel A) and adolescents (Panel B) to 1.28 and 1.25 (or by about 0.2 point), correspondingly. The parallel values for Hispanics were 1.37 and 1.34 (or by about 0.1 point). Consequently, the effects for residence status, marital status and ID explain a larger share of the African-American-non-Hispanic white disparity in obesity-related chronic disease than the Hispanic-non-Hispanic white disparity.

[Table 4 is about here]

DISCUSSION

Prior research shows that there are sharp race-ethnic disparities in the prevalence of obesity and obesity-related disease. Not only the prevalence rate of obesity among

minorities is markedly higher than among non-Hispanic whites, but also racial disparities in obesity are known to grow over the time (Wang & Beydoun, 2007). The latter tendency has aroused significant interest in scientific communities and has stimulated numerous empirical investigations of the contributions of different risk factors to the creation of these disparities. By focusing on individual- as well as neighborhood-level determinants of race-ethnic disparities in childhood obesity, the present study was intended as a contribution to this stream of literature. The primary objectives of the present study were: (1) to investigate race-ethnic disparities between non-Hispanic whites, African Americans and Hispanic children and adolescents in the prevalence of overweight, obesity and obesity-related disease; and (2) to estimate the relative contribution of individual (socio-economic background, family structure, family history of obesity, etc.) and neighborhood (racial segregation) factors to these disparities.

In line with early studies (Chen & Escarce, 2010; Gable & Lutz, 2000; Ogden *et al.*, 2010; Ogden *et al.*, 2014), we found that African American and Hispanic children and adolescents are significantly more likely than their non-Hispanic white peers to be overweight, obese and suffering from obesity-related disease (diabetes, hypertension and asthma). These results are robust to inclusion of controls for family socio-economic status, family size and structure, family history of obesity, marital status, age, sex and other individual-level predictors and residential race-ethnic segregation (neighborhood-level predictor). In our sample derived from the PSID CDS and TA surveys, the effects of most predictors of the odds of overweight, obesity and obesity-related disease were found to be significant and in the directions predicted from previous research (e.g., Bae *et al.*, 2014; Chen & Escarce, 2010; Gordon-Larsen *et al.*, 2003). Among significant predictors

of overweight, obesity and obesity-related disease were parental income, education and employment status, family structure, family history of obesity, sex, marital status and the level of residential segregation proxied by the Index of Dissimilarity (ID). Importantly, our results (not shown for parsimony) established a positive association between the level of race-ethnic segregation and the odds of being diagnosed with overweight, obesity and obesity-related disease among children and adolescents, regardless of their race-ethnic group. Race-ethnic segregation explains approximately 5-20% of the difference in the odds of being overweight, obese or having developed an obesity-related illness. A similar effect in magnitude is reported for the variables that monitor family socio-economic status – parental income, education and employment status. Thus, consistent with other studies, one of the reasons that African American and Hispanic children and adolescents are more likely to be obese and in poorer health due to obesity than their non-Hispanic white peers is because they are less advantaged in terms of family human capital and other parental resources (Chen & Escarce, 2010; Gable & Lutz, 2000; Gordon-Larsen *et al.*, 2003).

While our results suggest that the race-ethnic differential in overweight and obesity would be significantly reduced if African Americans and Hispanics became less segregated, this conclusion must be drawn with caution because this study has several limitations. First, this research relied on a pan-ethnic category (e.g., Hispanic) that can obscure important differences by ethnicity and/or country of origin. This is due to the limitation of the PSID and, by implication, CDS and TA surveys which contain too few Hispanic households (8% versus 16% in the current U.S. population). Therefore, additional research using larger and more representative sample sizes is warranted to

examine the ways in which overweight and obesity prevalence differs among specific ethnic groups such as Cubans, Puerto-Ricans, Central Americans, etc. Finally, the present study uses only a single neighborhood-level measure – the ID. It is theoretically feasible that other characteristics of African-American and Hispanic neighborhoods, such as low availability of healthy food options, a high concentration of fast food outlets and a lack of community safety for outdoor physical activities, account for the observed neighborhood effect on adolescent adiposity. Future studies should explore the extent to which these proximal neighborhood-level predictors affect the prevalence of obesity among children and adolescents. Despite these limitations, the results of the present study is helpful in understanding what socio-demographic and neighborhood-level factors contribute to the children’s obesity epidemic. These findings also have important implications.

The strong and independent association of race-ethnic segregation with children’s obesity and obesity-related disease lends imperative to the development of innovative intervention strategies that target children residing in segregated neighborhoods. A few policy approaches can be proposed to improve the public health conditions of the segregated communities. One is community-based, participatory approach to neighborhood improvement through the cooperative efforts of community institutions and citizens. This approach relies on building partnerships with relevant public agencies, local businesses and community-based organizations that allow community leaders and agencies to devise appropriate strategies to improve their localities (Lovasi *et al.*, 2012). As the evidence shows, the community-based approach can be effective in increasing the availability of healthy foods and creating local recreational facilities and infrastructure in disadvantaged neighborhoods (Bodor *et al.*, 2010; Gordon-Larsen *et al.*, 2006). A second

approach is housing mobility, which provides tangible benefits to disadvantaged families who move to less segregated neighborhoods (Acevedo-Garcia *et al.*, 2004). The housing mobility policies are effectuated by local governments that act through a system of vouchers which subsidize rent in the private rental market. Finally, the third approach that we advocate is school desegregation which is many ways similar to housing mobility, albeit is not intended to move families out of segregated neighborhoods. Today school desegregation policies operate primarily through parents' choice, centered on magnet schools and a series of student transfers (Palardy, 2013). More generally, we believe that school environment is crucial for combatting the children's obesity epidemic as it allows to generate a variety of school-based interventions designed to improve childhood nutrition, physical activity, and other health-related behaviors (Ding *et al.*, 2011; Scott *et al.*, 2007). The school environment is an ideal location for health education, interventions against inactivity and maintaining healthy body weight.

Our findings suggest that some share of the association between race-ethnicity and children and adolescent weight status is a function of residential segregation. However, other environmental and genetic factors such as socioeconomic status, household size and structure, family history of obesity also contribute to the inter-racial variations in weigh status among U.S. children and adolescents. Based on our analysis we are confident that no simple solution will adequately remedy the weight status differentials observed in our study. Therefore, intervention strategies should be complex. Nevertheless, we strongly believe that widespread changes in the social structure including but not limited to the equalization of access to quality health care will have a positive effect on the observed race-ethnic disparities in childhood and adolescent

obesity. Above all, the results of this study suggest that substantial resources be committed to ensure all children, regardless of their race-ethnic and socio-economic background as well as of their place of residence, have high-quality developmental experiences. Equality so, all children should have access to healthy foods and do not encounter barriers to physical activities.

REFERENCES

Acevedo-Garcia, D., Osypuk, T. L., Werbel, R. E., Meara, E. R., Cutler, D. M., & Berkman, L. F. (2004). Does housing mobility policy improve health? *Housing Policy Debate, 15*(1), 49-98.

Biro, F. M., & Wien, M. (2010). Childhood obesity and adult morbidities. *The American Journal of Clinical Nutrition, 91*(5), 1499S-1505S.

Bae, D., Wickrama, K. A. S., & O'Neal, C. W. (2014). Social consequences of early socioeconomic adversity and youth BMI trajectories: Gender and race/ethnicity differences. *Journal of Adolescence, 37*(6), 883-892.

Blazer, D. G., Moody-Ayers, S., Craft-Morgan, J., & Burchett, B. (2002). Depression in diabetes and obesity: racial/ethnic/gender issues in older adults. *Journal of Psychosomatic Research, 53*(4), 913-916.

Bodor, J. N., Ulmer, V. M., Dunaway, L. F., Farley, T. A., & Rose, D. (2010). The rationale behind small food store interventions in low-income urban neighborhoods: insights from New Orleans. *The Journal of Nutrition, 140*(6), 1185-1188.

Bonilla-Silva, E. (2006). *Racism without Racists: Color-Blind Racism and the Persistence of Racial Inequality in the United States*. Rowman & Littlefield Publishers.

- Braveman, P. A., Cubbin, C., Egerter, S., Williams, D. R., & Pamuk, E. (2010). Socioeconomic disparities in health in the United States: what the patterns tell us. *American Journal of Public Health, 100*(S1), S186-S196.
- Chang, V. W. (2006). Racial residential segregation and weight status among U.S. adults. *Social Science & Medicine, 63*(5), 1289-1303.
- Chen, A. Y., & Escarce, J. J. (2010). Family Structure and Childhood Obesity, Early Childhood Longitudinal Study-Kindergarten Cohort. *Preventing Chronic Disease, 7*(3), A50.
- Davis, M. M., McGonagle, K., Schoeni, R. F., & Stafford, F. (2008). Grandparental and parental obesity influences on childhood overweight: implications for primary care practice. *The Journal of the American Board of Family Medicine, 21*(6), 549-554.
- Ding, D., Sallis, J. F., Kerr, J., Lee, S., & Rosenberg, D. E. (2011). Neighborhood environment and physical activity among youth: a review. *American Journal of Preventive Medicine, 41*(4), 442-455.
- Dixon, J. B. (2010). The effect of obesity on health outcomes. *Molecular and Cellular Endocrinology, 316*(2), 104-108.
- Drewnowski, A. (2004). Obesity and the food environment: dietary energy density and diet costs. *American Journal of Preventive Medicine, 27*(3), 154-162.
- Drewnowski, A., & Specter, S. E. (2004). Poverty and obesity: the role of energy density and energy costs. *The American Journal of Clinical Nutrition, 79*(1), 6-16.

Flodmark, C. E., Marcus, C., & Britton, M. (2006). Interventions to prevent obesity in children and adolescents: a systematic literature review. *International Journal of Obesity, 30*(4), 579-589.

Feagin, J. R. (2014). *Racist America: Roots, Current Realities, and Future Reparations*. Routledge.

Gable, S., & Lutz, S. (2000). Household, parent, and child contributions to childhood obesity. *Family Relations, 49*(3), 293-300.

Gortmaker, S. L., Swinburn, B. A., Levy, D., Carter, R., Mabry, P. L., Finegood, D. T., Huang, T., Marsh, T., & Moodie, M. L. (2011). Changing the future of obesity: science, policy, and action. *The Lancet, 378*(9793), 838-847.

Gordon-Larsen, P., Harris, K. M., Ward, D. S., & Popkin, B. M. (2003). Acculturation and overweight-related behaviors among Hispanic immigrants to the US: the National Longitudinal Study of Adolescent Health. *Social Science & Medicine, 57*(11), 2023-2034.

Gordon-Larsen, P., Nelson, M. C., Page, P., & Popkin, B. M. (2006). Inequality in the built environment underlies key health disparities in physical activity and obesity. *Pediatrics, 117*(2), 417-424.

Kahn, S. E., Hull, R. L., & Utzschneider, K. M. (2006). Mechanisms linking obesity to insulin resistance and type 2 diabetes. *Nature, 444*(7121), 840-846.

Kipke, M. D., Iverson, E., Moore, D., Booker, C., Ruelas, V., Peters, A. L., & Kaufman, F. (2007). Food and park environments: neighborhood-level risks for childhood obesity in east Los Angeles. *Journal of Adolescent Health, 40*(4), 325-333.

Kuczarski, R. J., Ogden, C. L., Grummer-Strawn, L. M., Flegal, K. M., Guo, S. S., Wei, R., Mei, Z., Curtin, L. R., Roche, A. F., & Johnson, C. L. (2000). CDC growth charts: United States. *Advance Data*, *314*(1), 1-27.

Krebs, N. F., Himes, J. H., Jacobson, D., Nicklas, T. A., Guilday, P., & Styne, D. (2007). Assessment of child and adolescent overweight and obesity. *Pediatrics*, *120* (Supplement 4), S193-S228.

Larson, N. I., Story, M. T., & Nelson, M. C. (2009). Neighborhood environments: disparities in access to healthy foods in the U.S. *American Journal of Preventive Medicine*, *36*, 74–81.

Levine, J.A. (2011). Poverty and obesity in the U.S. *Diabetes*, *60*: 2667–8.

Lovasi, G. S., Bader, M. D., Quinn, J., Neckerman, K., Weiss, C., & Rundle, A. (2012). Body mass index, safety hazards, and neighborhood attractiveness. *American Journal of Preventive Medicine*, *43*(4), 378-384.

Morland, K. B., & Evenson, K. R. (2009). Obesity prevalence and the local food environment. *Health & Place*, *15*, 491–495.

Ogden, C.L., Carroll, M. D., Kit, B. K., Flegal, K. M. (2014). Prevalence of childhood and adult obesity in the United States, 2011-2012. *Journal of the American Medical Association*, *311*(8), 806-814.

Ogden, C. L., Carroll, M. D., Curtin, L. R., Lamb, M. M., & Flegal, K. M. (2010). Prevalence of high body mass index in U.S. children and adolescents, 2007-2008. *Journal of the American Medical Association*, *303*(3), 242-249.

Palardy, G. J. (2013). High school socioeconomic segregation and student attainment. *American Educational Research Journal*, *50*(4), 714-754.

Reilly, J. J., Armstrong, J., Dorosty, A. R., Emmett, P. M., Ness, A., Rogers, I., Steer, C., & Sherriff, A. (2005). Early life risk factors for obesity in childhood: cohort study. *British Medical Journal*, *330*(7504), 1357.

Rubin, D. B. (2004). *Multiple Imputation for Nonresponse in Surveys*. New York: John Wiley and Sons.

Saperstein, A. (2012). Capturing complexity in the United States: which aspects of race matter and when? *Ethnic and Racial Studies*, *35*(8), 1484-1502.

Scott, M. M., Cohen, D. A., Evenson, K. R., Elder, J., Catellier, D., Ashwood, J. S., & Overton, A. (2007). Weekend schoolyard accessibility, physical activity, and obesity: the Trial of Activity in Adolescent Girls (TAAG) study. *Preventive Medicine*, *44*(5), 398-403.

Smith, K. R., Zick, C. D., Kowaleski-Jones, L., Brown, B. B., Fan, J. X., & Yamada, I. (2011). Effects of neighborhood walkability on healthy weight: assessing selection and causal influences. *Social science research*, *40*(5), 1445-1455.

Sørensen, T. I., Price, R. A., Stunkard, A. J., & Schulsinger, F. (1989). Genetics of obesity in adult adoptees and their biological siblings. *BMJ: British Medical Journal*, *298*(6666), 87

Story, M., Kaphingst, K.M., Robinson-O'Brien, R., & Glanz, K. (2008). Creating healthy food and eating environments: Policy and environmental approaches. *Annual Review of Public Health*, *29*, 253–272.

Strauss, R. S., & Knight, J. (1999). Influence of the home environment on the development of obesity in children. *Pediatrics*, *103*(6), e85-e85.

Subramanian, S. V., Acevedo-Garcia, D., & Osypuk, T. L. (2005). Racial residential segregation and geographic heterogeneity in black/white disparity in poor self-rated health in the U.S.: a multilevel statistical analysis. *Social Science & Medicine*, 60(8), 1667-1679.

Wang, Y., & Beydoun, M. A. (2007). The obesity epidemic in the United States—gender, age, socioeconomic, racial/ethnic, and geographic characteristics: a systematic review and meta-regression analysis. *Epidemiologic Reviews*, 29(1), 6-28.

Wang, M. C., Kim, S., Gonzales, A. A., MacLeod, K. E., & Winkleby, M. A. (2007). Socioeconomic and food-related physical characteristics of the neighborhood environment are associated with body mass index. *Journal of Epidemiology & Community Health*, 61, 491–498.

Whitaker, R. C., Wright, J. A., Pepe, M. S., Seidel, K. D., & Dietz, W. H. (1997). Predicting obesity in young adulthood from childhood and parental obesity. *New England Journal of Medicine*, 337(13), 869-873.

White, K., & Borrell, L. N. (2011). Racial/ethnic residential segregation: framing the context of health risk and health disparities. *Health & Place*, 17(2), 438-448.

Wickrama, K. T., Wickrama, K. A., & Bryant, C. M. (2006). Community influence on adolescent obesity: Race/ethnic differences. *Journal of Youth and Adolescence*, 35(4), 641-651.

Williams, D. R., & Collins, C. (1995). U.S. socioeconomic and racial differences in health: patterns and explanations. *Annual Review of Sociology*, 349-386.

Williams, D. R., & Collins, C. (2001). Racial residential segregation: a fundamental cause of racial disparities in health. *Public Health Reports*, 116(5), 404.

Williams, D. R., & Sternthal, M. (2010). Understanding racial-ethnic disparities in health sociological contributions. *Journal of Health and Social Behavior*, 51(1 suppl), S15-S27.

Wyatt, S. B., Williams, D. R., Calvin, R., Henderson, F. C., Walker, E. R., & Winters, K. (2003). Racism and cardiovascular disease in African Americans. *The American Journal of the Medical Sciences*, 325(6), 315-331.

Zhang, Q., & Wang, Y. (2004). Socioeconomic inequality of obesity in the United States: do gender, age, and ethnicity matter? *Social Science & Medicine*, 58(6), 1171-1180.

Table 1. Descriptive Statistics of the Sample.

	White (N=955)	Black (N=782)	Hispanic (N=153)	All (N=1,910)
<i>Dependent Variables</i>				
Overweight at CDS	11.6%	16.7%	18.1%	14.7%
Overweight at TA	11.4%	17.0%	19.3%	14.8%
Obese at CDS	14.9%	21.7%	22.3%	18.4%
Obese at TA	15.0%	23.6%	24.2%	19.4%
Chronic Disease at CDS ^A	11.4%	17.5%	17.2%	14.4%
Chronic Disease at TA ^A	12.5%	20.2%	19.7%	16.3%
<i>Independent Variables</i>				
Parental Income, Thousands of Dollars (at CDS)	52.1	40.2	46.1	46.6
Mother's Education, years (at CDC)	14.3	12.3	10.9	13.2
Father's Education, years (at CDC)	13.6	12.3	11.6	12.9
At Least One Parent Employed (at CDS)	89.3%	76.5%	87.3%	84.0%
Two-Parent Household (at CDS)	62.4%	40.2%	52.6%	52.3%
Single-Mother Household (at CDC)	19.1%	41.6%	27.9%	28.8%
Number of Siblings (at CDS)	1.3	1.4	1.7	1.4
Obese Parent or Grandparent (at CDS)	20.7%	26.7%	22.4%	23.0%
Age, years (at CDS)	14.2	14.3	14.2	14.2
Gender (male)	47.9%	48.4%	48.1%	48.2%
Resided with One or Both Parents (at TA)	59.4%	68.0%	60.2%	62.9%
Married or Cohabited (at TA)	20.3%	14.4%	20.3%	18.0%
<i>Residential Segregation (Level-2)</i>				
Dissimilarity Index		0.65	0.41	0.52

Note: A – Includes asthma, diabetes and hypertension.

Table 2. Predicted Odds Ratios of Being Overweight ($\geq 85\%$ BMI $\leq 95\%$) with and without Controls; Non-Hispanic White – Reference Category; 95% Confidence Intervals are in Parenthesis

Model	Regression Controls Included	African American Effect	Hispanic Effect
Panel A: Overweight as a Child (at CDS)			
1	Race-Ethnicity Dummies, No Controls	1.47 (1.32-1.71)	1.49 (1.22-1.69)
2	Parental Income, Education and Employment Status	1.24 (1.02-1.48)	1.31 (1.16-1.48)
3	Family Structure, Number of Siblings, Family History of Obesity, Age and Gender	1.21 (1.00-1.44)	1.29 (1.06-1.45)
4	Dissimilarity Index	1.11 (0.89-1.25)	1.14 (0.92-0.39)
Panel B: Overweight as an Adolescent/Young Adult (at TA)			
1	Race-Ethnicity Dummies, No Controls	1.49 (1.25-1.76)	1.54 (1.26-1.84)
2	Parental Income, Education and Employment Status	1.35 (1.15-1.58)	1.37 (1.13-1.63)
3	Family Structure, Number of Siblings, Family History of Obesity, Age and Gender	1.30 (1.10-1.53)	1.26 (1.04-1.48)
4	Residence Status and Marital Status	1.25 (1.08-1.44)	1.24 (0.95-1.44)
5	Dissimilarity Index	1.06 (0.87-1.28)	1.18 (0.99-1.51)

Table 3. Predicted Odds Ratios of Being Obese ($\geq 95\%$ of BMI) with and without Controls; Non-Hispanic White – Reference Category; 95% Confidence Intervals are in Parenthesis

Model	Regression Controls Included	African American Effect	Hispanic Effect
Panel A: Overweight as a Child (at CDS)			
1	Race-Ethnicity Dummies, No Controls	1.63 (1.35-1.94)	1.71 (1.35-1.99)
2	Parental Income, Education and Employment Status	1.53 (1.28-1.81)	1.56 (1.32-1.83)
3	Family Structure, Number of Siblings, Family History of Obesity, Age and Gender	1.41 (1.18-1.66)	1.49 (1.29-1.71)
4	Dissimilarity Index	1.25 (0.99-1.49)	1.37 (1.12-1.56)
Panel B: Overweight as an Adolescent/Young Adult (at TA)			
1	Race-Ethnicity Dummies, No Controls	1.73 (1.35-2.04)	1.86 (1.86-2.13)
2	Parental Income, Education and Employment Status	1.62 (1.35-1.93)	1.73 (1.71-2.06)
3	Family Structure, Number of Siblings, Family History of Obesity, Age and Gender	1.49 (1.22-1.70)	1.55 (1.49-1.83)
4	Residence Status and Marital Status	1.45 (1.18-1.69)	1.45 (1.20-1.68)
5	Dissimilarity Index	1.28 (1.03-1.50)	1.34 (1.09-1.51)

Table 4. Predicted Odds Ratios of Being Diagnosed with a Chronic Condition (Asthma, Diabetes or Hypertension) with and without Controls; Non-Hispanic White – Reference Category; 95% Confidence Intervals are in Parenthesis

Model	Regression Controls Included	African American Effect	Hispanic Effect
Panel A: Diagnosed Chronic Disease as a Child (at CDS)			
1	Race-Ethnicity Dummies, No Controls	1.71 (1.35-1.99)	1.63 (1.35-1.94)
2	Parental Income, Education and Employment Status	1.56 (1.32-1.83)	1.53 (1.28-1.81)
3	Family Structure, Number of Siblings, Family History of Obesity, Age and Gender	1.49 (1.29-1.71)	1.41 (1.18-1.66)
4	Dissimilarity Index	1.28 (1.03-1.50)	1.37 (1.12-1.56)
Panel B: Diagnosed with Chronic Disease as an Adolescent/Young Adult (at TA)			
1	Race-Ethnicity Dummies, No Controls	1.96 (1.76-2.23)	1.73 (1.35-2.04)
2	Parental Income, Education and Employment Status	1.83 (1.61-2.16)	1.62 (1.35-1.93)
3	Family Structure, Number of Siblings, Family History of Obesity, Age and Gender	1.65 (1.39-1.93)	1.49 (1.22-1.70)
4	Residence Status and Marital Status	1.45 (1.20-1.68)	1.45 (1.18-1.69)
5	Dissimilarity Index	1.25 (0.99-1.49)	1.34 (1.09-1.51)