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Childhood obesity and the associated roles of neighborhood and biologic stress

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ABSTRACT

Exposure to violence and obesity continues to be growing epidemics, particularly among children. Our objective was to increase our understanding of the association between neighborhood violence exposure and children's weight and how biologic stress may mediate this relation. A matched, community-recruited cross-sectional study of 90 children, ages 5–16 years, from 52 neighborhoods took place in the greater New Orleans, LA area between 2012 and 2013. Children were matched on their propensity for living in a high violence neighborhood and previous exposure to Hurricane Katrina. Primary neighborhood exposure included violent crime, operationalized as crime rates within specific radii of children's home. Rates of exposure within 500, 1000 and 2000 meter radii from the child's home were calculated. Primary outcomes were body mass index (BMI) and waist circumference, and the primary mediator was telomere length (TL), a marker of cellular aging. Significant variation in obesity and TL was observed at the neighborhood level and violent crime was significantly associated with weight status, with an increase of 1.24 units in BMI for each additional violent crime in the child's neighborhood and a significant mediated or indirect effect of TL in the crime-BMI relation (0.32, 95% bootstrapped CI = 0.05, 0.81; 32% total mediated effect). Findings strengthen existing evidence linking neighborhood violence to childhood health and identify biologic stress, indexed by TL, as one mechanistic pathway by which neighborhood violence may influence childhood obesity. Neighborhood violence may be an important target for interventions focused on reducing obesity and other stress related health outcomes in children.

1. Introduction

Since the 1970's in the U.S., childhood obesity rates have more than tripled (Ogden et al., 2016), and 20% of school-aged children were obese in 2014 (Fryar and Ogden, 2014). Both the number of children who become obese at younger ages and the prevalence of severe obesity among children continue to increase, with as much as 80% of obese children and adolescents remaining obese in adulthood (Ogden et al., 2016). In the U.S., racial/ethnic and socioeconomic disparities in childhood obesity are evident (Wang et al., 2011), and likely contribute to multiple adult health disparities. Multilevel, comprehensive obesity prevention strategies are needed to understand and address this serious public health concern.

Despite the growth in empirical research on environmental characteristics and their influence on children's diet, physical activity, and obesity (Gordon-Larsen et al., 2000; Sallis and Glanz, 2006; Chaparro et al., 2014), much remains to be learned as most work in childhood obesity has focused on the micro-environment (e.g., home, school influences) and few have examined neighborhood environments. Those that have, typically focused on the neighborhood food environment, looking at the impact of food availability on dietary behavior (An and Sturm, 2012; Elbel et al., 2015; Skidmore et al., 2010; Timperio et al., 2008; Van Hulst et al., 2012) and adiposity (Chaparro et al., 2014), or on the physical activity environment, such as the influence of the availability and condition of places to be physically active in the neighborhood (i.e., parks, recreation centers, playgrounds) on children's physical activity levels and adiposity (Ding et al., 2011; Galvez et al., 2010; Congdon, 2017; Papas et al., 2007).

Neighborhood violence has been linked to weight-related behaviors (i.e., physical activity (Janssen, 2014)), obesity (Miranda et al., 2012), and health disparities. Socioeconomically disadvantaged communities have higher violence exposure as well as greater rates of obesity,

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suggesting that the impacts of exposure may also be compounded. Unfortunately, before the age of 18, approximately two-thirds of children are exposed to multiple types of violence that potentially leave persistent biological "scars" across an individual's stress response systems (SRS) resulting in lasting physiologic and psychologic vulnerability (Finklehor et al., 2007). A greater understanding of the association of neighborhood violence exposure on children's health is critical as differential exposure to community violence may underlie persistent socioeconomic, racial and ethnic health disparities (Lee, 2002; Yen and Syme, 1999).

Violence exposure in children has been linked to altered biologic responses across a number of SRS, suggesting mechanistic roles in negative health effects (Moffitt, 2013). In both adults and youth, violence exposure has been associated with decreased telomere length and greater telomere loss (Drury et al., 2014; Shalev et al., 2013; Theall et al., 2017; Tyrka et al., 2010). These alterations may reflect initial physiologic adaptations to high risk environments, however persistent activation of these SRS, or decreased effective regulation, leads to a high biologic cost to long-term health, including obesity (Garcia-Calzon et al., 2014). Telomeres represent one novel biomarker that is: (a) responsive to environmental changes and stressors, (b) has diverse effects in different organ systems, and (c) may index a molecular pathway through which environments, both positive and negative, influence physiology and development. Telomeres are specialized nucleoprotein complexes located at the termini of chromosomes that prevent promiscuous chromosomal rearrangements and genomic instability as well as ensure complete chromosomal replication (Epel et al., 2004). Telomere length normally decreases with cellular replication but is also influenced by a myriad of other factors including inflammation, oxidative stress, environmental toxins, and ionizing radiation (Valdes et al., 2005; Bull and Fenech, 2008; Derradiji et al., 2008; Zglinicki, 2002). Once a critically short length is reached, cellular senescence or terminal differentiation can be triggered (Gilley et al., 2008). However, it is increasingly recognized that telomeres are dynamic complexes that likely epigenetically regulate regions throughout the genome influencing both cellular development and differentiation (Li and Lustig, 1996).

Shorter TL may be reflective of one molecular pathway linking neighborhood violence to obesity and other negative health trajectories. Links between obesity and shorter telomere length have been reported in cross-sectional studies in both adults and adolescents (Correia-Melo et al., 2014). Among adults, a meta-analysis reported that the majority of studies found an inverse relationship between leukocyte telomere length and obesity indices including BMI, waist circumference, hip circumference, body fat percentage, and excessive visceral adiposity (Lee et al., 2011; Müezzinler et al., 2014). Among children, a similar inverse association between telomere length and adiposity measures has been reported (Buske-Kirschbaum et al., 1997; Garcia-Calzon et al., 2014; Lamprokostopoulou et al., 2014) and may differ by both sex (Al-Attas et al., 2010) and race (Zhu et al., 2011).

The purpose of this study was to examine the relation between neighborhood violence and obesity-related outcomes in children, and to test the potential mediating role of telomere length, a putative marker of biologic stress. Examination of community factors such as neighborhood violence may lead to new arenas that can be targeted for wider public health impact. Furthermore, examination of potential mechanisms through which violence may lead to obesity-related outcomes further increases our understanding of this relation and provides additional targets for intervention.

2. Methods

2.1. Subjects

Children aged 5–16 years (mean age = 10 years) and their families were recruited using street outreach techniques from New Orleans, Louisiana, U.S. between January 2012 and July 2013. Just over half (54%) of participants were female. Participants were georeferenced to their census tract of residence and located in 51 census tracts (out of 177). Mothers provided data about multiple levels of their child(ren)'s social ecology using an interviewer-assisted computer survey (Nova Research, Bethesda, MD) administered face-to-face at the research site. Written informed consent was obtained from caregivers and assent was obtained for children over age eight.

Given the outcomes examined, potential structural confounding or selection into specific neighborhoods, and the importance of age, children were propensity score matched (Oakes and Kaufman, 2017) 3:1 (with replacement) on age within one year, exposure to Hurricane Katrina, and the propensity (with a caliber of 0.05 on the propensity score) of living in a high neighborhood violence environment. The final matched sample included 85 Black children.

2.2. Measures

Both primary and secondary data were included and a multilevel data system of children nested within their household and neighborhoods. Neighborhood was defined as geographic buffers (500, 1000 and 2000-meter radii) around the child's home as well as a U.S. census tract.

The ***primary outcomes*** of interest included body mass index (BMI) percentile and waist circumference (WC). For children, the Centers for Disease Control and Prevention sex-specific BMI-for-age growth charts were used to define overweight and obesity. Overweight was defined as at or above the 85th percentile but less than the 95th percentile of the sex-specific BMI-for-age chart, whereas children with BMI-for-age ≥ 95th percentile were classified as obese. Height was measured with a stadiometer and weight with a digital scale, calibrated each time prior to measurement. To determine WC, waist measurements were taken on a horizontal line at the high point of the iliac crest. The measurement was made at minimal respiration to the nearest 0.1 cm. All measures were taken twice, by trained research staff, and the average utilized as the final estimate.

The ***primary exposure*** of interest was *neighborhood violent crime rate*, calculated using New Orleans Police Department data from 2012. Substantiated crimes were mapped using the address where the crime occurred and then aggregated by type of crime to determine the number of homicide and assault (including rape) crimes within a 500, 1000 and 2000-meter radii of each child's residence. The rate per census tract and distance to nearest event from the child's home were also calculated and compared for consistency.

The ***primary mediator*** of interest was telomere length (TL), as represented by the triple to single gene (T/S) ratio and determined by monochromic multiplex quantitative polymerase chain reaction (MMqPCR) from extracted DNA taken from buccal swab samples, with two swabs per child. Measures were performed in triplicate and duplicated on a second plate in a different location. The average of the six replicates was used to calculate TL. Samples with high coefficients of variation (CVs) (10% intra-assay and 6% inter-assay) were removed (n = 2). Inter-plate CVs for the Ct (cycle threshold) values were below 1% for both the telomere and single-copy gene reactions, indicating consistent amplification between replicate plates. In addition to the samples, each 384-well plate contained a six-point standard curve from 0.625 ng to 20 ng of genomic DNA. The exponentiated T/S ratio (_dCt) for each sample was calculated by subtracting the average 36B4 Ct value from the average telomere Ct value and expressed as 2_(_dCt). sTL values were calculated as the ratio of telomere repeats to single-copy gene values (T/S ratio).

Key covariates, potential confounders or effect modifiers included socio-demographics: child sex, maternal age, maternal education, prenatal maternal smoking, smoke exposure in the home, as well as children's pubertal status as measured with the Tanner Scale (Marshall and Tanner, 1969; Marshall and Tanner, 1970), diet and physical activity. Questions regarding diet and asked to the caregiver

included whether the child consumed two or more servings of fruits or vegetables per day, on average. Physical activity was assessed by asking the caregiver how many hours, on a typical day, does the child spend engaging in moderate-intensity activities (activities that require moderate physical effort and cause small increases in breathing or heart rate, like brisk walking, bicycling or swimming) and then how many hours they spend in vigorous-intensity activities (activities that require hard physical effort and cause large increases in breathing or heart rate, like running or playing basketball). Those who spent one or more hours per day in moderate or vigorous activity were coded as having some type of physical activity, versus those who had less than 1 h per day of either activity.

2.3. Statistical analysis

Univariate, bivariate, and multivariable analyses were performed using SAS version 9.2, including PROC MIXED or GLIMMIX to take into account neighborhood clustering, multiple children per household, and conditioning on matched stratum. Thirty-eight percent of enrolled families had more than one child participate (range = 1–5). Mixed models were utilized for partitioning variance estimates at all levels, expressed as the intraclass correlation coefficient (ICC). When overweight/obesity status was examined as a dichotomous outcome (overweight or obese vs. normal or underweight), the pseudo-ICC was calculated following Snijders formula based on an underlying continuous variable with $V_{individual} = \Pi^2/3$ (Snijders and Bosker, 1999). Modeling was performed in the following steps: (1) examination of empty models to determine the extent of clustering of BMI and WC and TL values by neighborhood, (2) testing the unadjusted associations between the neighborhood violence environments and BMI and WC and the extent of neighborhood-level variance explained, (3) testing the adjusted association, after accounting for potential confounders, and (4) testing potential mediation. Any covariate that exhibited collinearity with other covariates (Variance Inflation Factor > 10) in the final model or did not change the measure of effect were not considered in the analyses. The proportion of variance in outcomes explained by neighborhood violence was examined by both traditional R-square (given that the exposures within radii of children's homes is an individual level measure) as well as with the proportion of variance explained at the neighborhood level, using the Proportional Change in Variance (PCV). All models compared violence rates at different radii around the child's home, as well as with tract-level exposures. Potential mediation of the violent crime and obesity outcome relations by TL were calculated, separately, utilizing the SAS PROCESS Macro, given the continuous nature of the mediator, and with bootstrap analyses (20,000 repetitions) to derive a 95% confidence interval (CI) for the indirect effect.

3. Results

Characteristics of the sample are presented in Table 1. More than one-third (32%) of children were overweight or obese. Children who were overweight or obese were significantly more likely to live in neighborhoods with a greater homicide rate (1.2 homicides within a 500-meter radius of the home versus 0.8, $t = 2.17$, $p = 0.03$) as well as a trend for greater assaults. The average TL was shorter among children who were overweight or obese (1.4 vs. 1.7, $t = 1.85$, $p = 0.08$). Neighborhood violent crime rate was significantly associated with shorter TL ($rho = -0.257$, $p = 0.006$).

3.1. Effect of neighborhood violence

In empty models, not considering any covariates, we observed substantial clustering of obesity-related outcomes with 23% of the variance in BMI, 22% in WC, and 15% in obesity explained at the census tract or neighborhood level based on the ICC. Table 2 shows multivariable results depicting the total effect of neighborhood violent crime rate on BMI (Model 1) and WC (Model 2), separately. Even after adjusting for potential confounders and predictors of BMI, the number of violent crimes within a 500-meter radius remained associated with BMI, with an increase in BMI by 1.241 for each additional reported crime (Table 2, Model 1, $\beta = 1.241$, SE = 0.489, $p = 0.016$). Considering only bivariate associations, violent crime exposure explained 5% of the variance (R-squared = 5%) in BMI and an additional 22% was explained by additional covariates. The amount of neighborhood-level variance explained by including violent crime in the empty model was 70% for BMI and 45% for obesity status (not shown). Results were nearly identical for crime exposure within a 1000-meter radius and approached significance within a 2000-meter radius. Controlling for the same covariates, the likelihood of being overweight or obese increased by 12% for each additional violent crime reported within a 500-meter radius of the child's home (adjusted OR = 1.12, 95% CI = 1.02–1.26; not shown).

Violent crime rate remained only marginally associated with WC after adjusting for several potential confounders, with violence within a 500-meter radius associated with an increase in WC of 0.723 for each additional crime (Table 2, Model 2, $\beta = 0.723$, SE = 0.493, $p = 0.151$). Considering only bivariate associations, violent crime exposure explained 2% of the variance (R-squared = 5%) in WC, with an additional 22% explained by additional covariates. The amount of neighborhood-level variance in WC explained by including violent crime in the empty model was 29%.

3.2. Indirect effect of neighborhood violence

Given the potential for biologic stress – measured by TL – to be a potential pathway from neighborhood violence exposure to obesity-related outcomes, mediation between neighborhood violent crime exposure and BMI and WC by TL were examined. As shown in Table 2, Models 3 and 4, significant indirect effects emerged for both BMI (0.318, 95% Bootstrapped CI = 0.048, 0.806) and WC (0.201, 95% Bootstrapped CI = 0.038, 0.520), with TL explaining 32% of the effect of neighborhood violent crime on BMI and 20% of the effect of neighborhood violent crime on WC.

4. Discussion

We demonstrate in this study the potential role that neighborhood violence may play in the development of obesity-related outcomes among children. Results indicate an association between neighborhood violent crime and obesity-related outcomes, including BMI, overweight/obesity status, and WC. We also observed substantial clustering at the neighborhood level for obesity-related outcomes and substantial changes in explained neighborhood level variance once violent crime in the neighborhood was considered.

Although there is mixed evidence on the role of community compared to other forms of violence on obesity (Midei and Matthews, 2011), these results are consistent with previous findings linking community violence to obesity and BMI in children, most often measured as neighborhood safety (An and Sturm, 2012). In a national, cross-sectional National Institute of Child Health and Human Development study (Lumeng et al., 2006), researchers found that children living in the lowest quartile of neighborhood safety rating had 4.43 times the odds of overweight at age 7 compared to the highest quartile of safety. In terms of overweight/obesity, only a handful of studies have investigated the association between exposure to objectively-measured crime and overweight/obesity among children or adolescents (Miranda et al., 2012; Gartstein et al., 2017). Among children across wide age ranges, these studies found that children exposed to higher levels of neighborhood violent crime had a greater likelihood of being overweight/obese and higher BMI.

Nonetheless, a recent meta-analysis suggested only a small increase in BMI and limited impact on childhood obesity related to living in an

Table 1
Characteristics of matched sample by overweight or obesity status.

	Total *N* = 90 n (%) or Mean (± s.d.)	Overweight or obese (≥85th percentile BMI) *N* = 29 n (%) or Mean (± s.d.)	Not overweight or obese (< 85th percentile BM) *N* = 61 n (%) or Mean (± s.d.)
Sex of child			
Male	41 (46)	11 (38)	30 (49)
Female	49 (54)	18 (62)	31 (51)
Age of child			
≤6 years old	7 (8)	1 (4)	6 (10)
7–9 years old	32 (36)	14 (48)	18 (29)
10+ years old	51 (56)	14 (48)	37 (61)
Caregiver's age*			
18–29	16 (18)	5 (17)	11 (18)
30–39	58 (64)	20 (70)	38 (64)
≥40	16 (18)	5 (17)	11 (18)
Mother's education†			
< High school	23 (26)	5 (17)	18 (30)
High school degree or GED	23 (26)	5 (17)	18 (30)
Some college or more	44 (48)	19 (66)	25 (40)
Low daily fruit and vegetable consumption (yes)†	17 (19)	8 (28)	9 (15)
Low physical activity (yes)†	48 (53)	19 (66)	29 (47)
Smoking during pregnancy (yes)*	13 (14)	1 (3)	12 (20)
Smoke exposure in the home (yes)	36 (40)	9 (31)	27 (44)
Mean telomere length (T/S ratio)*	1.5 (0.04)	1.4 (0.06)	1.7 (0.04)
Mean violent crime rate/500 m†	8.3 (8.2)	9.0 (1.2)	8.1 (1.2)

Note. Percentages and estimates based on non-missing data (< 5% were missing across all variables). Low daily fruit and vegetable consumption = consumed less than two or more servings of fruits or vegetables per day, on average. Low physical activity = spending less than 1 h per day in moderate or vigorous activity.
† p-Value < 0.05.
* p-Value < 0.10.

unsafe neighborhood (Müezzinler et al., 2014). This could be a result of differences in the measurement of neighborhood safety, or to the fact that violence may also operate in other ways through the home or community to increase risk—mechanisms that may be wiped out in traditional analyses examining community violence's impact while also including potential pathways or mediators (Oakes and Kaufman, 2017). Limited or null findings may also be a result of length of exposure and latency period. Obesity and even BMI changes may be too distal of outcomes to see the full impact of community violence on risk for obesity. Focusing on intermediate outcomes, such as TL or other biologic stress markers, that are potentially more proximal and that have been linked to excess adiposity, may reveal a more significant role of the neighborhood environment than is seen with adiposity-related outcomes.

Findings of this study suggest that differences in TL may mediate the relation between neighborhood violence and obesity in children. While violence in the community may lead to reduced physical activity among children due to safety concerns, its link to biologic stress represents an

Table 2
Neighborhood violence environment's association with BMI and WC (N = 90).

	Beta coefficient (β)	Standard Error (SE)	p-Value
Model 1. Effect of neighborhood violent crime on BMI			
Intercept	8.709	3.080	0.007
Number of violent crimes within 500 m	1.241	0.489	0.016
Child sex (female vs. male)	−0.125	1.113	0.911
Child age (ordinal)	2.268	0.858	0.012
Maternal education (ordinal)	1.467	0.674	0.036
Lower daily fruit and vegetable consumption (yes)	0.191	1.156	0.869
Lower daily physical activity (yes)	2.787	1.394	0.053
R-square	27%		
c (PCV)[a]	70%		
Model 2. Effect of neighborhood violent crime on waist circumference			
Intercept	35.292	9.503	0.001
Number of violent crimes within 500 m	0.723	0.493	0.151
Child sex (female vs. male)	1.510	3.138	0.634
Child age (ordinal)	9.882	2.417	0.0002
Maternal education (ordinal)	0.915	2.317	0.695
Lower daily fruit and vegetable consumption (yes)	0.256	3.467	0.942
Lower daily physical activity (yes)	5.510	4.271	0.206
R-square	15%		
Proportional Change in Variance (PCV)[a]	29%		
Model 3. Indirect effect of telomere length on BMI			
Indirect Effect = 0.318 (95% Bootstrapped CI = 0.048, 0.806) = the proportion of the effect of neighborhood violent crime on BMI that acts through telomere length			
Model 4. Indirect effect of telomere length on waist circumference			
Indirect Effect = 0.201 (95% Bootstrapped CI = 0.038, 0.520) = the proportion of the effect of neighborhood violent crime on WC that acts through telomere length			

[a] $PCV_{neighborhood(n)} = [(V_{n-1} - V_{n-2}) / V_{n-1}]$, where V_{n-1} = neighborhood variance in the empty model and V_{n-2} is neighborhood variance in the model including violent crime rate.

additional, likely additive or synergistic, mechanism through which such environments increase health risks. This is critical for community-based intervention and prevention efforts as it suggests that neighborhood stress exerts a more direct influence on obesity development through biological pathways, even after accounting for diet and physical activity. Such factors are also linked to neighborhood violence as noted, and increased violence in the neighborhood may be a potential marker for more deprived neighborhoods whereby healthy food availability and access to safe places to play are also limited. In models without diet and physical activity, the association of neighborhood violence on TL was even stronger; however, the fact that the association persists even when controlling for these factors highlights the fact that multiple pathways contribute to obesity risk and that violence not only influences health related behaviors it is also influencing children's biology. For example, Bacha et al. (2010) found that neighborhood safety was associated with a higher weight status in 5th grade for girls, but this association was not explained by less time spent outdoors or more television viewing, similar to these results where neither diet nor physical activity fully accounted for the effect of violence on child weight. Dietary and physical activity behavioral factors considered could also be mediators in the relation between neighborhood violence and obesity-related outcomes. When considered together both the current findings and the previous research suggest that behavioral and biological factors contribute, potentially pointing to a stress inducing hypothesis that needs to be examined in future research.

Despite important findings, there are key limitations to the current study including the cross-sectional nature, small sample, and applicability to other demographic groups. Given the cross-sectional nature of the study, causality cannot be determined. We had no information on relevant transgenerational, pre-conception, prenatal or early life exposures that may shape the stress response system, telomere length or risk for obesity. The role of biologic stress markers such as TL and its relation to neighborhood exposure as well as other factors associated with obesity are complex and difficult to test in a small cross-sectional study. We also had no information on additional environments, such as the school or other neighborhood exposures that likely play a role in obesity. While our neighborhood definition for the multilevel models are based on administrative boundaries (census tract), and may not truly represent a child's neighborhood, our primary exposures were within radii from the child's home. The self-report measures of children's diet and physical activity are also limited and likely do not capture the full impact of diet and physical activity on obesity-related outcomes. Nonetheless, there are a number of strengths, including utilization of objective measures of neighborhood safety; examination of additional adiposity outcomes; and this being one of the few studies to examine biological mediators of the link between neighborhood factors and adiposity outcomes.

With persistent violence exposure in the U.S. and elsewhere, it is pertinent that we gain a deeper understanding of the role violence plays in health outcomes across the life course. Even though children may not be fully aware of their exposure to neighborhood adversity, findings suggest a powerful molecular association of this adversity on their health. This complex relation points to the larger, structural forces that shape child health and development and direct attention to neighborhoods as critical arenas that can be effectively targeted for even greater public health impact on a range of child physical and mental health outcomes.

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