

Do neighbourhoods matter? Neighbourhood disorder and long-term trends in serum cortisol levels

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Accepted 3 June 2010
 Published Online First 24 August 2010

ABSTRACT

Background Characteristics associated with low socioeconomic status neighbourhoods may put children at risk for unique chronic stressors that affect cortisol levels. This research sought to explore whether neighbourhood stressor exposure affected serum cortisol levels among children.

Methods A total of 148 African and European-American children with an average age of 8.28 years participated in a longitudinal study evaluating ethnic differences in body composition and disease risk. Five waves of data were included in analyses. Mixed modelling was used to explore neighbourhood stressors, which was a composite index of five items for zip code level poverty and physical disorder, and serum cortisol outcomes for the full sample, by race/ethnicity and gender.

Adjustments were made for individual level correlates age, pubertal status, gender and total fat mass.

Results Neighborhood disorder was predictive of lower serum cortisol levels among African-American children ($p < 0.05$), such that higher neighbourhood stressor exposure resulted in lower serum cortisol over time compared with individuals in socially ordered neighbourhoods. Neighbourhood disorder was marginally significant and predictive of higher serum cortisol among European-American children ($p < 0.10$). Transition to a higher pubertal status, nested in age was also predictive of lower serum cortisol levels ($p < 0.01$) among European-American children.

Conclusion Children who are exposed to negative socioenvironmental climates over time are more likely to have altered serum cortisol levels. This may be an adaptive mechanism to cope with stress; however, disrupted cortisol levels may have negative effects on general physical and mental health.

Neighbourhood environments may be sources of negative stressful stimuli that pose significant health risks for children. The work of William Julius Wilson suggests that health outcomes within specific geographical locations cannot be understood without taking into account the social and economic circumstances from which they emerge.^{1 2} Researchers posit that neighbourhood chronic stressors such as noise, violence and poverty may disrupt endocrine pathways such as the hypothalamic–pituitary–adrenal (HPA) axis and the sympathetic nervous systems.^{3–6} In turn, these disruptions may contribute to the development of metabolic disorders.⁷

While many studies have associated neighbourhood processes with psychosocial outcomes,^{8–12} few have assessed the direct effects of neighbourhood characteristics on the physiological

functioning of children.^{3 6 13} The studies that have been conducted primarily focus on neighbourhood socioeconomic status (SES), which leaves the effects of neighbourhood socioenvironmental conditions relatively unexplored. In addition, many of these studies have evaluated the relationships independently of body composition, although adiposity is known to affect cortisol secretion.^{14–16} The limited available data suggest that familial environment and individual and community level SES are highly correlated with cortisol.^{3 6 13 17 18} Disentangling the relationship between low community level SES and cortisol may show that neighbourhood characteristics such as physical and social disorder may be the chronic stressors that affect the HPA axis.^{3 6 13} Therefore, the aim of this research was to explore neighbourhood stressor exposure and the relationship to cortisol.

Cortisol is a physiologically induced mechanism whereby the HPA axis mediates the effects of stressful life events on biological functioning by means of increased cortisol output.^{18–22} Cortisol is activated when the HPA axis stimulates the production of corticotrophin-releasing hormone in response to a stressor. In turn, adrenocorticotrophic hormone is released, with the end result of cortisol secretion into the bloodstream.^{18–21 23} While cortisol is central in maintaining homeostasis during acute stress,¹⁵ exposure to chronic stressors can result in dysregulation of the HPA axis through either hyper or hypocortisolism^{9 22 24–26} and may increase the risks of hypertension, insulin resistance, neuronal damage, immune disorders, mental health disorders, and disrupt the ability to deliver cortisol to sites of inflammation within the body.^{9 22 24–28} Exposure to acute stressors may result in higher cortisol, which may have negative metabolic health outcomes, and conversely exposure to chronic stressors may result in lower cortisol secretion, which may increase inflammation and negative health risks. In order to evaluate the relationship between neighbourhood stressors and cortisol, it was hypothesised that neighbourhood chronic stressor exposure would result in lower serum cortisol.

METHODS

Study population and design

The study consisted of 178 participants who took part in a longitudinal study evaluating racial and ethnic differences in the relationship between body composition and disease risk. After accounting for attrition, a total of 148 children (African American 67 and European American 81) with an average age

of 8.30 years at baseline was included in the analytical cohort. Methods and research findings from this cohort have been published previously.^{29–31} After receiving institutional review board approval at the University of Alabama at Birmingham, participants were recruited by posting fliers at clinics and through participant referrals. A total of 39 children had data for one visit, 26 had data for two time points, 34 had data for three time points, 25 had data for four time points, and 24 had data for five time points.

Eligibility requirements included being of African-American or European-American descent and being between 4 and 12 years of age at study entry. Participants were healthy, and were not taking any medications known to affect metabolism or body composition (eg, attention deficit hyperactivity, asthma medications and corticosteroids). Individuals who agreed to participate were provided with consent information. During the first visit, the study protocol was reviewed and both parents and children provided consent and assent respectively.

The sample consisted of male and female African-American and European-American children recruited from the Birmingham–Hoover MSA. Baseline data were acquired in 1994, and subjects returned annually for up to 9 years after initial evaluation. Due to increased attrition, available data for up to five time points were used. There were no significant differences in baseline data for the larger cohort and the analytical cohort. Geographical data obtained from the US Census Bureau (2000) were used to assess neighbourhood risk factors.

Measurements

This study included individual and group level data, obtained from clinical, survey and objective measurements. At each yearly time point, participants came to the university for two separate visits. During the first visit, body composition by dual energy x-ray absorptiometry (DXA) was recorded, anthropometric measurements were taken, surveys were conducted, and pubertal status was assessed by a physician according to the criteria of Marshall and Tanner.^{32–33} At the second visit, participants were admitted to the General Clinical Research Centre (GCRC) for an overnight evaluation during which intravenous glucose tolerance tests were administered and blood pressure measurements were obtained. Children were served a standard meal and snacks, which were consumed before 20:00 hours. After the overnight fast, blood samples were taken for hormone analyses at approximately 07:00 hours.

Dependent variable

Total serum cortisol was obtained from participants after an overnight fast at the GCRC. Unlike salivary samples, total serum cortisol levels are not sensitive to the diurnal rhythm and as such, all samples were obtained at approximately the same time for all individuals. Biochemical analyses were conducted in the Metabolism Core Laboratory of the Clinical Nutrition Research Centre and the GCRC at the university. Cortisol assays were performed using a coat-a-count radioimmunoassay method manufactured by Diagnostic Products Corporation, Los Angeles, California (now Siemens Medical Solutions Diagnostics, Cary, North Carolina, USA). This test has a sensitivity of 0.2 µg/dl and interassay and intra-assay coefficients of variation of 7.77% and 4.41%, respectively.

Independent variable

Objective indicators of neighbourhood disorder were compiled from the US Census. The only available participant identifiers

were zip codes. Zip code tabulation areas (ZCTA) were developed by the US Census (2000) to compute summary statistics at the zip code level. The ZCTA represents the zip code used by the majority of addresses within a given area and may vary slightly from US postal codes. Due to differences in development, it is possible that addresses within a zip code may be assigned to a ZCTA that does not correspond with the actual zip code.³⁴ At the time of the study period, the Birmingham–Hoover MSA had a population of 921 106 and ZCTA for the study participants covered 73% of the total population. The average population size was 17 749 (SD±8613) and an average geographical size of 78 799.27 km². For the study period, there was no neighbourhood mobility, which indicates that no children included in the analyses had moved during the five waves of data collection. As such, only one neighbourhood value was developed for each child. The neighbourhood index included percentages of unemployment, poverty, female-headed households with dependent children and vacant housing.^{1 2 35–37} While the first three items gauged neighbourhood SES, the percentage of vacant housing indicated physical disorder and within impoverished areas may indicate dilapidated or abandoned housing that could be sources of illegal activity.³⁸ The neighbourhood items were highly correlated with the percentage of female-headed households positively correlated with the percentage of vacant housing, poverty and unemployment ($r=0.769$, 0.901 and 0.793 , respectively). The percentage of vacant housing was strongly correlated with the percentage of poverty and unemployment ($r=0.896$ and 0.904 , respectively). Finally, the percentage of poverty was highly correlated with unemployment ($r=0.865$). For each individual item, Z scores were computed by subtracting the mean for the sample of ZCTA and then dividing by the SD. The Z scores for each measure were then summed to create a composite index. The Z score method has been established as a valid tool to assess neighbourhood characteristics.^{39 40} Higher scores indicated greater neighbourhood disorder relative to other children in the sample (expressed as SD units above the mean), whereas lower scores indicated that respondents encountered less disadvantage (expressed as SD units below the mean). The scale had a standardised Cronbach's α of 0.96.

Total and regional body fat were assessed during each visit by DXA with the Lunar DPX-L scanner (GE-Lunar Radiation Corp, Madison, Wisconsin, USA). Participants were scanned in light clothing with arms at their sides. In children, DXA have shown high reliability and function as good indicators of body fatness with a correlation above 0.96.⁴¹

Additional covariates included self-reported age, gender and pubertal status. Pubertal status was assessed by the criteria of Marshall and Tanner^{32–33} by a physician during the annual physical examination. Pubertal staging defines physical measurements of reproductive maturity based on secondary sexual characteristics.^{32–33} There are five Tanner stages that have been demonstrated as reliable indicators of pubertal development. The staging based on the criteria of Marshall and Tanner is according to both breast and pubic hair development in girls (<http://www.fpnotebook.com/Endo/Exam/FmlTnrStg.htm>) and genitalia and pubic hair development in boys (<http://www.fpnotebook.com/Endo/Exam/MlTnrStg.htm>). The development is a continuous process in which an individual is assigned to one of the five categories. One value is assigned and represents the higher of the two values observed for breast/genitalia and pubic hair. Because very few children reached Tanner stage 5 ($n=5$), this stage was combined with Tanner stage 4.³⁰ Ethnicity was based on parent reports.

Statistical methods

The sample included 148 children (67 African American and 81 European American) and a total of 413 clinical observations. At baseline, descriptive statistics and t tests were performed to examine significant differences in the independent and dependent variables, with a significance criteria of $p < 0.05$. Variables were evaluated for normality and serum cortisol and total fat were log transformed.

The mixed modelling (SAS Proc Mixed, 2002) was used to examine longitudinal trends in serum cortisol for the full sample, by ethnic group and gender.⁴² This approach accounts for the high degree of within-subject correlations in cortisol values and includes both random and fixed effects. After accounting for intra-individual correlation, this model adjusts for between-subject variations in cortisol. Mixed modelling is flexible and can be fitted to handle the heterogeneity of variances across subjects. Because this method uses maximum likelihood estimation and the missing values were random, this method can handle missing values without discarding available data. Two separate models were evaluated and included individual level variables, gender, age, pubertal status and total fat mass to assess the independent contributions to cortisol. The second model included the individual level variables and adjustment for neighbourhood disorder. In all analyses, pubertal stage was nested in age due to the changes in age associated with the pubertal transition.³⁰

Post-hoc analyses examined whether the regression coefficients for neighbourhood disorder significantly differed by race/ethnicity and whether the pubertal stages were significantly different. For the post-hoc analysis of puberty, a series of dummy variables was created with stage 3 as the reference stage (chosen based on the relationship to maturation of the HPA axis)⁴³ to examine whether this stage exhibited greater effects on serum cortisol relative to other stages.

Due to heterogeneity within the majority of the ZCTA (ie, on average less than two children per ZCTA), no analyses accounted for neighbourhood clustering. Using neighbourhood level data as an independent level variable instead of as a hierarchical two-level model is valid for this type of research.³⁵ All statistical analyses were performed using SAS (version 9.1).

RESULTS

The average age at baseline was 8.28 years (± 1.68) (table 1). Approximately 57% of the sample was female. African-American children were younger ($p < 0.05$) and had slightly more total fat mass than European-American children. Neighbourhood composite index scores ranged between -1.94 and 5.68 SD units below and above the mean. Percentages for the individual items of the composite neighbourhood disadvantage score are also provided, African-American children were significantly more likely to live in disordered neighbourhoods ($p < 0.05$) characterised by higher unemployment, poverty, single female-headed households and vacant housing. Overall, there were no significant differences in baseline serum cortisol levels between European Americans and African Americans.

Results from the mixed model for the full sample indicate that over the study period pubertal stage emerged as a significant predictor of serum cortisol levels at all ages (table 2). Post-hoc analyses indicated no significant differences in the relationships between pubertal stages and serum cortisol. Fat mass was predictive of lower serum cortisol among children. The second model included the individual level covariates and neighbourhood disorder. Over the study period, neighbourhood

Table 1 Baseline descriptive statistics for total sample and ethnicity, mean (SD)

	Total sample (N=148)	European American (n=67)	African American (n=81)
Cortisol, $\mu\text{g/dl}$	11.42 (3.49)	11.47 (3.80)	11.35
Gender, % female	50.54	43.59	55.56
Age, years	8.30 (1.70)	8.72 (1.61)*	7.92 (1.67)*
Pubertal stage	1.11 (0.41)	1.07 (0.26)	1.15 (0.5)
Total fat, kg	10.28 (7.04)	9.29 (5.59)	11.46 (8.27)
Neighbourhood composite score†	0.046 (-1.94–5.68)	-0.81* (-1.94–1.53)	0.66* (-1.59–5.68)‡
% Unemployment	4.11 (2.89)	2.33 (0.87)	5.48 (3.21)
% Poverty	14.25 (9.48)	7.18 (4.60)	18.47 (9.15)
% Female-headed household‡	15.52 (7.83)	9.68 (4.40)	20.24 (6.74)
% Vacant housing	8.65 (3.12)	6.99 (1.76)	10.00 (3.35)

*Denote significant differences at $p < 0.05$.

†Neighbourhood composite score represents Z-score index, sample means and range. Higher scores indicate greater neighbourhood disorder.

‡Female-headed households with dependent children under age 18 years.

disadvantage predicted lower serum cortisol levels such that children who lived in disordered neighbourhoods had lower serum cortisol levels than children in more socially ordered areas. In this model, the most significant predictor of serum cortisol levels was pubertal stage 2, in which, at this stage of puberty, reduced serum cortisol levels were evident. With the inclusion of neighbourhood effects in the model, pubertal stages 2 and 3 became significant and both negatively affected serum cortisol levels. Post-hoc analyses indicated that children in pubertal stage 2 had significantly lower serum cortisol levels relative to children in pubertal stage 3 ($p < 0.01$).

Table 3 reports longitudinal results for African-American children. In the first model, none of the independent variables were significant predictors of total serum cortisol. When the neighbourhood disadvantage measure was included, pubertal stage 3, nested in age, was significantly predictive of lower serum cortisol levels ($p < 0.05$). The composite neighbourhood disorder index was also predictive of lower serum cortisol levels ($p < 0.05$), such that over time, neighbourhood stressors affected cortisol and resulted in lower serum cortisol levels. Post-hoc analyses did not indicate any significant differences across pubertal stages.

Table 2 Mixed model for longitudinal trends in serum cortisol levels among a bi-ethnic cohort of children

	Model 1			Model 2		
	B	SE	β	B	SE	β
Age (Tanner 1)	-0.014	0.014	-1.00	-0.025	0.016	-1.56
Age (Tanner 2)	-0.020*	0.012	-1.67	-0.028**	0.014	-2.00
Age (Tanner 3)	-0.014	0.012	-1.16	-0.023*	0.013	-1.76
Age (Tanner 4 and 5)	-0.011	0.011	-1.00	-0.016	0.012	-1.33
Total fat mass	-0.048	0.030	-1.60	-0.034	0.033	-1.03
Gender, female	-0.014	0.045	-0.31	0.004	0.047	0.085
Neighbourhood index				-0.018*	0.011	-1.64
Log likelihood	396.3			350.5		
χ^2	34.36			31.33		
p Value	0.001			0.001		

Age (Tanner) refers to Tanner stage nested in age.

Neighbourhood index consisted of z-score tabulations of percentage of unemployment, poverty, single female-headed households with dependent children and percentage of vacant housing.

Significance levels were set at * $p < 0.05$; ** $p < 0.01$.

B, unstandardised coefficient; β , standardised coefficient.

Table 3 Mixed model for longitudinal trends in serum cortisol levels among African-American children

	Model 1***			Model 2***		
	B	SE	β	B	SE	β
Age (Tanner 1)	-0.015	0.022	-0.68	-0.022	0.022	-1.00
Age (Tanner 2)	-0.019	0.019	-1.00	-0.024	0.019	-2.10
Age (Tanner 3)	-0.024	0.017	-1.41	-0.031*	0.018	-1.72
Age (Tanner 4 and 5)	-0.017	0.016	-1.06	-0.023	0.016	-1.44
Total fat mass	-0.048	0.045	-1.06	-0.027	0.046	-0.586
Gender, female	-0.007	0.068	-0.10	.001	0.067	0.014
Neighbourhood index				-0.022*	0.012	-1.83
Log likelihood	191.7			195.5		
χ^2	27.98			26.75		
p Value	0.001			0.001		

Age (Tanner) refers to Tanner stage nested in age.

Neighbourhood index consisted of z-score tabulations of percentage of unemployment, poverty, single female-headed households with dependent children and percentage of vacant housing.

Significance levels were set at * $p < 0.05$; *** $p < 0.001$.

Full model was significant at $p < 0.001$.

B, unstandardised coefficient; β , standardised coefficient.

Table 4 provides results for the European-American children. As children progressed through the pubertal transition, significant effects on serum cortisol emerged. Pubertal stages 1, 2 and 3 nested in age, significantly predicted serum cortisol such that, at each pubertal stage, children had lower serum cortisol levels. Children entering pubertal stage 2 had significantly lower serum cortisol levels relative to pubertal stages 3 and 4 ($p < 0.05$). The neighbourhood composite index was marginally significant ($p < 0.10$) and predicted increased serum cortisol levels. In this model, pubertal stages 1, 2 and 3 retained significance. Post-hoc analyses indicated that children in pubertal stage 2 had significantly lower serum cortisol relative to children in pubertal stage 3 ($p < 0.01$). There were no significant differences in the neighbourhood disorder regression coefficient across racial/ethnic groups ($p > 0.05$).

Separate mixed models by gender were also conducted. While both models showed that the neighbourhood index operated negatively on serum cortisol, neither indicated significant relationships between neighbourhood disorder and serum cortisol (data not presented).

DISCUSSION

This is the first longitudinal study that evaluates the relationship between neighbourhood social and physical characteristics

Table 4 Mixed model for longitudinal trends in serum cortisol levels among European-American children

	Model 1***			Model 2***		
	B	SE	β	B	SE	β
Age (Tanner 1)	-0.046*	0.022	-2.09	-0.051*	0.022	-1.56
Age (Tanner 2)	-0.049**	0.019	-2.57	-0.053**	0.019	-2.00
Age (Tanner 3)	-0.037*	0.018	-2.05	-0.041*	0.018	-1.76
Age (Tanner 4 and 5)	-0.022	0.017	-1.29	-0.026	0.017	-1.33
Total fat mass	-0.009	0.047	-0.19	-0.001	0.047	-1.03
Gender, female	-0.005	0.073	-0.06	0.015	0.073	-0.085
Neighbourhood index				0.042†	0.031	1.64
Log likelihood	170.9			174.2		
χ^2	67.23			64.71		
p Value	0.001			0.001		

Age (Tanner) refers to Tanner stage nested in age.

Neighbourhood index consisted of z-score tabulations of percentage of unemployment, poverty, single female-headed households with dependent children and percentage of vacant housing.

Significance levels were set at * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

B, unstandardised coefficient; β , standardised coefficient.

and cortisol. The findings suggest that children in disordered neighbourhoods exhibit lower serum cortisol levels than their counterparts who reside in socially ordered neighbourhoods. Initial research indicated that low SES and mother's depressive state are related to the hypersecretion of salivary cortisol among children.^{6, 13} Salivary cortisol represents the biologically active fraction of cortisol.^{21, 22} While the overall results report neighbourhood effects on serum cortisol levels for the full sample, subgroup analyses indicate that the relationship between neighbourhood disorder and serum cortisol operates in a race/ethnic-specific manner. Results indicate that neighbourhood disorder is related to lower total serum cortisol among African-American children. This supports the research of Chen and Paterson³, in which children in lower SES neighbourhoods have lower basal salivary cortisol levels. These findings support the hypothesis of Lupien and colleagues⁶ that the mechanism between neighbourhood SES and altered cortisol lies in the characteristics of low SES neighbourhoods (that is increased vacant housing, higher rates of unemployment and poverty).

Because African-American children were more likely to live in disordered neighbourhoods, it was important to determine whether neighbourhood indicators were surrogates for ethnicity. Although not significant, neighbourhoods were predictive of higher serum cortisol among European Americans. These findings are in line with Lupien and colleagues⁶ and with research that suggests ethnic-specific neighbourhood pathways that are pertinent to health.³⁵ However, Chen and Paterson³ posit that the accumulation of stressors will result in blunted cortisol among all children as they transition into adulthood.

While research demonstrates that cortisol reactivity, as assessed by social stress tests, is different between men and women,²² the current study found no significant gender differences. Research has shown that cortisol secretion is suppressed by estradiol;^{43, 44} however, the HPA axis does not develop fully until pubertal stage 3,⁴³ which may explain why there were no significant sex differences. Serum cortisol levels were significantly lower for children in pubertal stage 2 when compared with children in pubertal stage 3. This finding corresponds with the literature, indicating a positive relationship between pubertal maturation and cortisol output.⁴⁵ Although not significant, greater fat mass affected serum cortisol such that children with greater fat mass exhibited lower total serum cortisol, which is consistent with Hanrahan and colleagues.²⁰ Cortisol is intimately tied with adipose tissue, particularly abdominal fat, as it is related to an increased clearance of cortisol.¹⁴ The increased clearance results in comparable or lower levels of cortisol in obese individuals, when compared with non-obese individuals.^{14, 22, 46}

Limitations

To our knowledge, there are no established guidelines for healthy cortisol ranges for a bi-ethnic sample;^{9, 22} therefore, the extent of altered serum cortisol is unknown. Second, only one serum cortisol measurement was obtained at each time point; it is known that cortisol follows a diurnal pattern, and salivary cortisol may be a better indicator.^{22, 47} While the neighbourhood measure was developed using theory and findings from the literature, it is possible that important indicators of neighbourhood disorder were not taken into account. The inclusion of these indicators may have resulted in stronger observed relationships between neighbourhood disorder and serum cortisol. Also, ZCTA were used to assess neighbourhoods; smaller units of analysis may be more preferable. Because the neighbourhood index was obtained from the decennial US Census, there may have been neighbourhood changes over the study period.

What is already known on this subject

A source of chronic stress such as low neighbourhood SES has been associated with dysregulation of the HPA axis, resulting in either blunted or elevated levels of cortisol among children.

What this study adds

This study is the first to evaluate long-term trends in serum cortisol levels among children and the extent to which neighbourhood stressors affect serum cortisol among a bi-ethnic sample of children. This study extends the neighbourhood literature to include other stressors aside from SES. In addition, this is the first study to adjust for important covariates that result from clinical observations such as pubertal status and total fat mass. These current findings demonstrate that characteristics associated with low neighbourhood SES (eg, percentage of unemployment, poverty, single female-headed households with dependent children and percentage of vacant housing) are related to lower serum cortisol levels.

However, research indicates that neighbourhoods remain relatively stable over time and can be 'identified by patterned behaviour and reproducibility of patterns across space and time' from which certain cultural values and beliefs are developed and maintained.^{1 48} Based on these conclusions we propose that the neighbourhoods did not experience significant changes in exposures over the study period.

Individual level SES is not accounted for in the present analyses and while researchers have found significant relationships between individual level SES and cortisol in young children,⁶ there has been no evidence of this relationship during adolescence.^{3 6 23} Also, there are no measures that account for parental stress levels; research has established that maternal stress is related to cortisol levels among young children.^{8 10} Finally, important mediators such as social support are not included.^{24 26 49–51} A strength of this study is the use of DXA to measure adiposity. While body mass index has been used as a surrogate of adiposity for cortisol research, this may be inaccurate because body mass index underestimates/overestimates adiposity prevalence in a multi-ethnic sample.

This study underscores the need to measure aspects of the social and physical environments above and beyond traditional measures of SES. Future studies should assess longitudinal relationships to cortisol using frequent sampling techniques and include other potential sources of chronic stress.

Acknowledgements The author would like to thank Tena Hilario-Hailey and the VICTORY study team for recruitment, study coordination and data management, and all the children and parents who participated. They also wish to thank Betty Darnell and the nurses and staff of the GCR.

Funding This work is supported by NIH grants R01-DK49779, R01-DK-51684, General Clinical Center Grant M01-RR00032, Clinical Nutrition Research Unit Grant P30-DK56336 and the Diabetes Research Training Center P60-DK079626.

Competing interests None.

Patient consent Obtained.

Ethics approval This study was conducted with the approval of the University of Alabama at Birmingham institutional review board.

Provenance and peer review Not commissioned; externally peer reviewed.

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