Household Income, Cortisol, and Obesity During Early Childhood: A Prospective Longitudinal Study

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Objective To prospectively evaluate the relationship between household income, children's cortisol, and body mass index (BMI) trajectories over a 3-year period in early childhood.

Study design Household income, child hair cortisol levels, and BMI were measured at baseline, 12-, 24-, and 36-month follow-up visits in the Now Everybody Together for Amazing and Healthful Kids (NET-Works) Study (n = 534, children ages 2-4 years, and household income <$65 000/year at baseline). Relationships were examined between very low household income (<$25 000/year) at baseline, income status over time (remained <$25 000/year or had increasing income), cortisol accumulation from hair samples, and BMI percent of the 95th percentile (BMIp95) trajectories using adjusted linear growth curve modeling. Households with baseline income between $25 000 and $65 000/year were the reference group for all analyses.

Results Children from very low-income households at baseline had annual changes in BMIp95 that were higher ($P < .001$) than children from reference group households (0.40 vs −0.62 percentage units/year). Annual increases in BMIp95 were also greater among children from households that remained very low income ($P < .01$, .34 percentage units/year) and among those with increasing income ($P = .01$, .51 percentage units/year) compared with the reference group (−0.61 percentage units/year). Children from households that remained very low income had higher hair cortisol accumulations (0.22 pg/mg, $P = .02$) than reference group children, whereas hair cortisol concentrations of children from households with increasing income (0.03 pg/mg) did not differ significantly from the reference group. Cortisol was not related to BMIp95.

Conclusions The economic circumstances of families may impact children's BMI trajectories and their developing stress systems, but these processes may be independent of one another. *(J Pediatr 2023;252:76-82)*.

Childhood obesity is a significant public health challenge with implications for long-term health and well-being.[1-3] Low household income is often cited as a risk factor for childhood obesity.[4-6] Studies have found that recurrent poverty during the school-aged years is associated with faster body mass index (BMI) growth trajectories[7] and greater likelihood of obesity in adolescence.[7,8] Increases in family income over time have also been associated with decreased child BMI.[9] Although socioeconomic disparities in childhood obesity are well-documented,[10] the mechanisms that underlie this relationship are less clear. Chronic stress associated with poverty is one factor that may influence biological processes favoring weight gain. An important pathway in the biological stress response system is the hypothalamic-pituitary-adrenal (HPA) axis, often measured by its end product, the steroid hormone, cortisol. Although brief and relatively infrequent activations of the HPA axis support adaptive functioning, chronic or frequent activations can be harmful to health.[11] Examination of hair cortisol, which reflects cortisol secretion over an extended time window (eg, months), may be particularly useful for gauging the cumulative impact of chronic stressors such as poverty.[12,13] Longitudinal studies are scarce and there are limited data about the possible relationship between hair cortisol levels and weight trajectories in relation to early childhood poverty and household income dynamics.

The present study fills this gap in the literature by prospectively examining changes in household income with children's cortisol levels and BMI trajectories using longitudinal data from the Minnesota Now Everybody Together for Amazing and Healthful Kids (NET-Works) trial (U01HD068890), a childhood obesity prevention study of predominantly low-income families. We hypothesized that very low household income during the early childhood years

BMI	Body mass index
BMIp95	BMI percent of the 95th percentile
HCC	Hair cortisol concentration
HPA	Hypothalamic-pituitary-adrenal

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Supported by the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development of the National Institutes of Health under award numbers U01HD068990 (PIs: S.F., N.S.) and R01HD090059 (PI: A.K.-B.). The content is solely the responsibility of the authors and does not necessarily represent the official views of the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development. A.K. serves as an unpaid consultant for Novo Nordisk, Vivus, EliLilly, and Boehringer Ingelheim and receives donated drug/placebo from Vivus for an NIDDK-funded clinical trial. The other authors declare no conflicts of interest.

https://doi.org/10.1016/j.jpeds.2022.09.007

(baseline, ages 2-4 years) and households that remained very low in subsequent years would be associated with higher hair cortisol concentrations (HCCs), higher mean BMI percentiles, and greater increases in children's BMI trajectories than children from relatively higher income households at baseline. We further hypothesized that chronic stress, measured using HCCs, would mediate the relationship between low household income and children's BMI trajectories.

Methods

Data were collected between 2012 and 2017 as part of the NET-Works (ClinicalTrials.gov, NCT01606891) study, a randomized controlled obesity prevention intervention, part of the Childhood Obesity Prevention and Treatment consortium.[14,15] Children were eligible if they were 2-4 years of age, had no medical problems (eg, chronic illness) that would preclude study participation, did not use medications that could affect growth (eg, oral corticosteroids), had a BMI >50th percentile for age/sex, a family income of <$65 000/year at baseline, and a parent who spoke either English or Spanish and did not have plans to move out of the state during the 3-year study period. Children (n = 534) were recruited from pediatric primary care clinics in Minneapolis-St. Paul Minnesota and randomized between July 2012 and January 2014. Data were collected in the participant's home by bilingual staff who were blinded to group assignment. Retention was 92% at the 36-month follow-up visit. The University of Minnesota and HealthPartners Institute Institutional Review Boards approved the study. All parents consented to participate.

BMI percent of the 95th percentile (BMIp95) was used as the outcome because BMIp95 performs better than other BMI metrics (eg, *z* score or percentile) for longitudinal analyses and samples like ours contain individuals with high BMI values that extend into the severe obesity range (**Table I**).[16-18] Weight and height were measured with the child participant in light clothing without shoes at baseline, 12-, 24- and 36-month follow-up visits. Weight was measured to the nearest 0.1 kg using calibrated, digital scales and height to the nearest 0.1 cm using a free-standing stadiometer (Seca Corp). BMI was calculated as weight in kilograms divided by the square of height in meters, and percentiles were calculated according to Centers for Disease Control and Prevention age and sex referenced guidelines.[19]

Cortisol measurement from hair provides an estimate of integrated cortisol secretion over extended periods of time and if elevated can serve as a biologic marker of chronic stress. This approach to cortisol measurement was selected for this study to provide a cumulative index of hypothalamic-pituitary-adrenal axis activity that is not subject to the situational confounds of salivary or plasma cortisol (eg, time of day[20]) and was expected to reflect the impact of chronic stressors in the lives of these children. Hair samples of 3 cm in length were collected from the posterior vertex of the child's head for cortisol assay at baseline and 12-, 24- and 36-month follow-up visits. As hair grows roughly 1 cm per month, 3 cm of hair provides information about the last 3 months of cortisol production. HCC values were missing at visits when children had hair that was too short to collect a 3 cm sample or parents declined. Wash and steroid extraction procedures have been described in detail elsewhere.[21] HCCs in pg/mg were determined using a commercially available immunoassay with chemiluminescence detection (CLIA; IBL-Hamburg). Intra-assay and interassay coefficients of variation are <8%.

At baseline and each follow-up visit (12, 24, and 36 months), the child's participating parent/caregiver reported their annual household income using income categories (<$15 000; $15 000-<$25 000; $25 000-<$35 000; $35 000-<$50 000; $50 000-<$75 000; $75 000-<$150 000; $150 000-<$200 000; ≥$200 000). Two approaches to measuring low income were taken to answer the research questions (**Table II**). One approach considered whether reported family income was <$25 000/year when the

Table I. Descriptive statistics for key outcomes by time point

N	Baseline 534	12 m 503	24 m 483	36 m 493
Hair cortisol concentrations				
n	383	325	307	292
ln(cortisol), pg/mg, M (SD)	2.24 (1.04)	2.07 (1.05)	2.00 (1.04)	1.87 (1.00)
BMI				
n	534	503	483	493
BMI, M (SD)	17.58 (1.78)	17.46 (2.17)	17.76 (2.56)	18.31 (2.96)
BMI %, M (SD)	81.71 (14.32)	80.86 (17.78)	81.62 (17.81)	81.50 (18.38)
BMIp95, M (SD)	96.53 (9.84)	96.77 (12.06)	96.93 (13.99)	96.65 (15.46)
Normal weight 5th -<85th percentile, %	51.7	48.3	45.8	44.6
Overweight 85th -<95th percentile, %	25.7	27.2	25.9	25.0
Obesity 95th-<120 95th percentile, %	20.0	19.7	21.7	22.7
Severe obesity ≥120 of the 95th percentile, %	2.6	4.8	6.6	7.7

Table II. Two approaches to categorizing low household income status using parent-reported household income at baseline and 12-, 24-, and 36-month follow-up visits

Measurement approach	Category labels	Income at baseline	Income at 12, 24, and 36 months	n
Baseline income	Baseline very low income	<$25 000		336
	Reference	$25 000 to $65 000		198
Income over time	Remained very low income	<$25 000	<$25 000 at one-half or more	240
	Increasing income	<$25 000	<$25 000 at fewer than one-half	96
	Reference	$25 000 to $65 000		198

child was 2-4 years of age (baseline). A second approach considered whether postbaseline household income remained <$25 000/year or increased. The granularity at which income was measured did not permit precise assessment of household income relative to the federal poverty level. More detail is provided in the Analysis Plan.

The child's parent/primary caregiver reported sociodemographic information during a structured interview. Variables relevant for the current analyses included child age (years), sex, race/ethnicity (non-Hispanic White, non-Hispanic Black, Hispanic of any race, all others), and NET-Works treatment group (intervention, control).

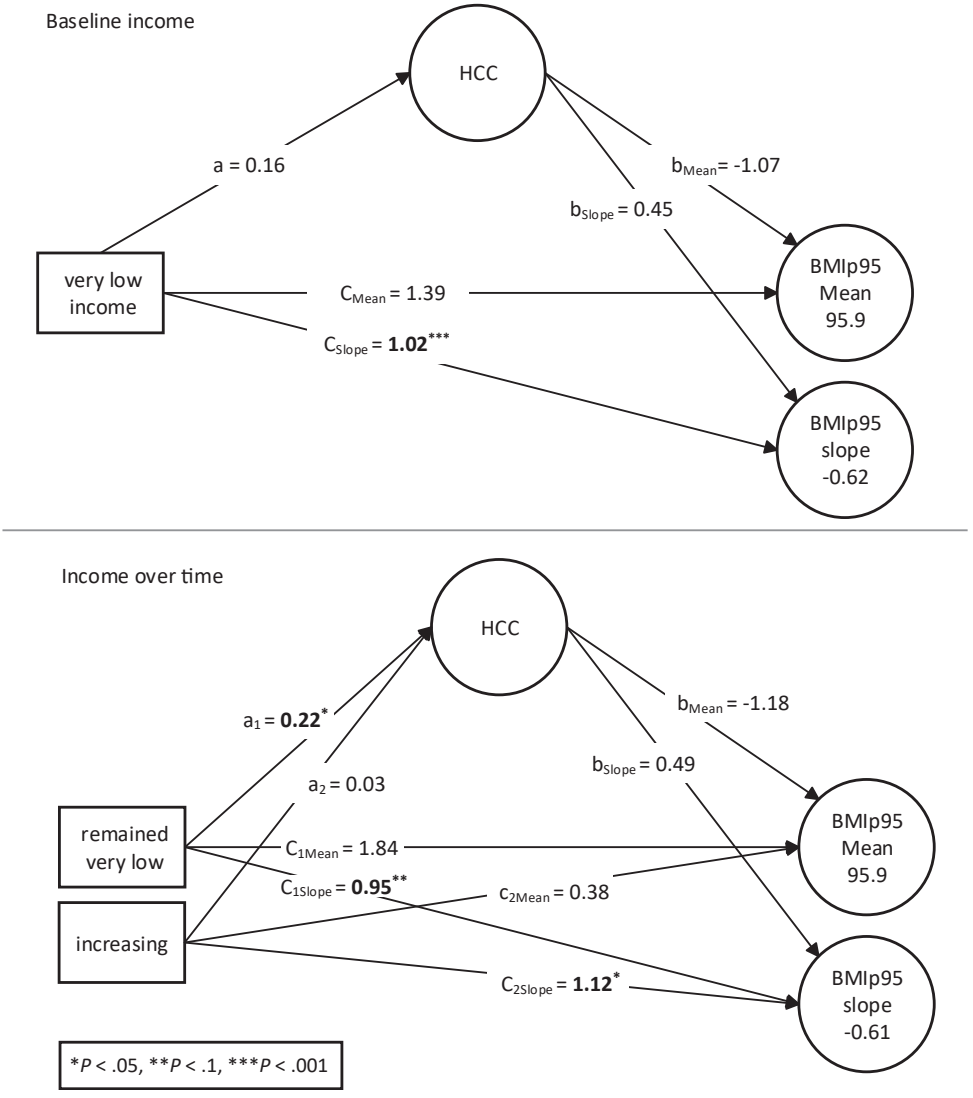

Figure. Mediated path model estimating relationships among low income, HCC, and BMIp95.

A structural equation modeling approach was used to estimate and test the significance of the relationships between income, HCC, and BMIp95. Measurement models quantified each of these constructs in a manner that supported the study hypotheses, which were tested by sequentially adding structural paths that estimated relationships between (1) income and BMIp95, (2) income and HCC, and (3) HCC and BMIp95, as illustrated in the **Figure**.

A linear growth curve model quantified the BMIp95 outcome. Baseline BMIp95 was specified as the model intercept. The slope was estimated from baseline and 12-, 24-, and 36-month measures and parameterized in years. The BMIp95 intercept and slope were both adjusted for NET-Works treatment group. All children contributed at least one BMIp95 measure (93.4% contributed 3 or 4 measures) and were therefore included in the BMIp95 measurement model.

The HCC mediator was modeled as a latent factor with 4 indicators, ln(cortisol) measured at baseline, 12, 24, and 36 months; 84% of children contributed at least one HCC measure (54.9% contributed 3 or 4 measures) to the estimation of the HCC factor. Each ln(cortisol) indicator was adjusted for Black race and Hispanic ethnicity to account for higher likelihoods of HCC missingness among these groups that varied across visits. The HCC factor was adjusted for NET-Works treatment group, baseline age, and sex.

Two sets of low-income status predictor variables were calculated as very low household income at baseline (when children were ages 2-4 years) and very low household income that remains low or increases post baseline (**Table II**). With the first approach, a single indicator denoted that a household was baseline very low income if the parent reported income under $25 000 on the baseline survey. The second, dynamic approach resulted in 2 indicators that split the baseline very-low-income group into households that remained very low income if income remained below $25 000 at one-half or more of the postbaseline visits or that had very low but increasing income if income was below $25 000 in fewer than half of postbaseline visits. Both measurement approaches treated households with baseline income between $25 000 and $65 000/year as the reference category. All households provided sufficient data to calculate the low-income status indicators.

Model 0. The BMIp95 measurement model functioned as an empty model that estimated the average baseline (intercept) and rate of change (slope) in BMIp95 among all children. Structural paths with the intercept component of BMIp95 added to this model assessed whether children in different income groups or with different HCC values already differed in BMIp95 at 2-4 years of age. Relationships with the rate of change component assessed whether income group or HCC was associated with how BMIp95 changed in subsequent years.

Model 1. Model 1a included structural paths to estimate the relationships between very low income and BMIp95. Two direct paths compared the baseline (c_{Int}) and rate of change in (c_{Slope}) BMIp95 of children in baseline very-low-income households relative to those in the reference group. Model 1b replaced the baseline very-low-income indicator with the set of indicators denoting remained very low income and increasing income and therefore included 2 sets of c_{Int} and c_{Slope} paths. One set compared baseline and rate of change in BMIp95 of households that remained very low-income to reference group households and the other set made these same comparisons between increasing income households and reference group households.

Model 2. Model 2a added a structural path (a) from the baseline very-low-income indicator to the HCC factor to compare HCC of children from baseline very-low-income households to reference group households. Model 2b included 2 income-HCC paths that compared HCC of children from households that remained very low income and from households with increasing income to reference group households.

Model 3. Models 3a and 3b enabled a test of mediation by adding structural paths from the HCC factor to baseline (b_{Int}) and rate of change (b_{Slope}) in BMIp95. Model 3a added these paths to model 2a so the components of a mediated income-HCC-BMIp95 relationship could be compared between baseline very-low-income and reference group households. Model 3b used the remained very-low-income and increasing income indicators so that the components of the income-HCC-BMIp95 relationship could be compared between each of these low-income groups and the reference group.

All models were estimated in MPlus7 using maximum likelihood, which assumes data are missing at random conditional on covariates, so that all observations are included in the covariance matrix from which model parameters are estimated.

Results

The children whose data are included in these analyses (N = 534) were M = 3.35 (SD = 0.65) years old at baseline. About one-half (50.9%) were female, over one-half (54.3%) were non-Black Hispanic, 29.2% were Black, and 12.6% were non-Hispanic White. The children were evenly divided by treatment arm (49.6% NET-Works intervention). At baseline, 62.9% of families reported annual household income below $25 000. The proportion of households with annual income below this threshold declined in postbaseline follow-up years (48.4% at 12 months, 44.0% at 24 months, and 38.5% at 36 months) such that 44.9% remained very low income and 18.0% had very low but increasing income.

HCCs declined from baseline (ln[cortisol] M = 2.24) through 36 months (ln[cortisol] M = 1.87; see **Table I**). The means of the BMI measures were stable and their SDs increased over the 36-month measurement period. The increase in the proportion of children categorized as having obesity and decrease in those with a healthy weight suggest that the increased variability in weight measures was not solely regression to the mean following the eligibility requirement that children have BMI >50th percentile for age/sex at baseline.

The BMIp95 growth curve model established that the baseline BMIp95 intercept is 96.66 and that prior to accounting

Table III. Path coefficients and intercept and slope estimates [95% CI] in structural models estimating relationships between very-low-income status, HCC, and BMIp95

Structural models	Income - HCC (a)	HCC—baseline BMIp95 (b_{Int})	HCC—change BMIp95 (b_{Slope})	Income—baseline BMIp95 (c_{Int})	Income—change BMIp95 (c_{Slope})	Baseline BMIp95 (intercept)	Change BMIp95 (slope)
BMIp95 growth curve							
Model 0						96.66 [95.80, 97.52]	0.07 [−0.23, 0.38]
Models comparing baseline very low income with the reference group							
Model 1a				1.26 [−0.49, 3.00]	**1.06 [0.46, 1.65]**	95.93 [94.55, 97.31]	**−0.59 [−1.04, −0.14]**
Very low							
Model 2a	0.15 [−0.02, 0.33]			1.25 [−0.49, 3.00]	**1.06 [0.47, 1.66]**	95.88 [94.51, 97.25]	**−0.59 [−1.04, −0.14]**
Very low							
Model 3a	0.16 [−0.01, 0.33]	−1.07 [−3.19, 1.06]	0.45 [−0.33, 1.24]	1.39 [−0.33, 3.12]	**1.02 [0.42, 1.61]**	95.90 [94.52, 97.27]	**−0.62 [−1.07, −0.17]**
Very low							
Models comparing remained very low-income and increasing income with the reference group							
Model 1b				1.60 [−0.34, 3.54]	**1.06 [0.41, 1.71]**	95.86 [94.49, 97.23]	**−0.61 [−1.06, −0.17]**
Remained							
Increasing				0.25 [−1.83, 2.34]	**1.11 [0.26, 1.97]**		
Model 2b	**0.22 [0.03, 0.41]**			1.58 [−0.36, 3.52]	**1.05 [0.40, 1.70]**	95.90 [94.53, 97.28]	**−0.60 [−1.05, −0.16]**
Remained							
Increasing	0.03 [−0.16, 0.22]			0.28 [−1.81, 2.36]	**1.12 [0.26, 1.98]**		
Model 3b	**0.22 [0.04, 0.41]**	−1.18 [−3.36, 0.99]	0.49 [−0.31, 1.28]	1.84 [−0.10, 3.78]	**0.95 [0.30, 1.60]**	95.91 [94.53, 97.29]	**−0.61 [−1.05, −0.16]**
Remained							
Increasing	0.03 [−0.16, 0.22]			0.38 [−1.72, 2.49]	**1.12 [0.27, 1.97]**		

Bold indicates significance of at least $P < .05$.

for very-low-income status the annual rate of change in BMIp95 was not significant (slope = 0.07, $P = .65$; Table III, Model 0).

Mean BMIp95 was not significantly higher among children from households with very low income at baseline relative to children from reference group households ($c_{Mean} = 1.26$, $P = .16$; Table III Model 1a). Consistent with our hypotheses, the estimated annual rate of change in BMIp95 was significantly higher ($c_{Slope} = 1.06$, $P < .001$) among children from very-low-income households at baseline (estimated change = 0.47 percentage units/year) relative to children from reference group households (estimated change −0.59 percentage units/year). The addition of the relationship between baseline very low income and HCC was in the predicted direction over the 36-month time period (ie, relatively higher estimated HCC among children from very-low-income households compared with the reference group), though this did not reach statistical significance (a = 0.15, $P = .08$; Model 2a). Finally, adding the relationships between HCC and the BMIp95 components (Model 3a) showed that HCC over 36 months was not related to baseline BMIp95 ($b_{Mean} = -1.07$, $P = .33$) or change in BMIp95 ($b_{Slope} = 0.45$, $P = .26$). Although the higher rate of change in BMIp95 among children from baseline very-low-income households remained significant in this final model ($c_{Slope} = 1.02$, $P < .001$; estimated change of 0.40 compared with −0.62 percentage units/year), the nonsignificant relationships between income and HCC, and between HCC and BMIp95, made a significant income-HCC-BMIp95 relationship implausible.

Consistent with our hypotheses, the estimated annual rate of change in BMIp95 was significantly higher among children from households that remained very low income ($c_{1Slope} = 1.06$, $P < .001$; Model 1b) compared with children from reference group households (estimated change of 0.45 compared with −0.61 percentage units/year). In contrast to our hypotheses, the rate of change in BMIp95 was also higher in households with very low but increasing income ($c_{2Slope} = 1.11$, $P = .01$; estimated change of 0.50 percentage units/year). Adding the relationships between the low-income statuses and HCC (Model 2b) showed that children from households that remained very low income had higher HCC over 36 months than reference group households ($a_1 = 0.22$, $P = .02$), consistent with our hypothesis. Children from households with very low but increasing income did not show this relationship with HCC ($a_2 = 0.03$, $P = .77$). In Model 3b, the relationships between HCC and the BMIp95 components were not significant ($b_{Mean} = -1.18$, $P = .28$; $b_{Slope} = 0.49$, $P = .23$). The higher estimated HCC among children from households that remained very low income was still significant ($a_1 = 0.22$, $P = .02$) as were the higher estimated rates of change in BMIp95 among children from households that remained very low income ($c_{Slope} = 0.95$, $P < .01$; change = 0.34 percentage units/year) or had very low but increasing income ($c_{Slope} = 1.12$, $P = .01$; change = 0.51 percentage units/year) relative to children from reference group households (change = −0.61 percentage units/year). Again, the lack of a significant relationship between HCC and BMIp95 made significant income-HCC-BMIp95 relationships implausible.

Discussion

In this prospective, longitudinal study, we examined whether household income and changes in household income status over time were associated with the annual change in BMI in the early childhood years and whether cortisol mediated

this association. We found that although children from the lowest income households (<$25 000/year) in early childhood (2-4 years of age) did not have significantly higher mean BMIp95 than children from relatively higher income households ($25 000-$65 000/year), they did have greater annual increases in BMIp95. The percent increase in BMIp95 was also steeper among children from households that remained very low income, suggesting that duration of poverty may play a role in BMI trajectories. Although children from households that had very low but increasing income also demonstrated greater BMIp95 increases relative to children from low to moderate income households at baseline, it is important to note that increases in household income within the cohort were modest and did not reflect alleviation of poverty in the vast majority of cases. Thus, although income rose among participating families in the study, the degree of increase may have been insufficient to meaningfully alter BMI trajectories.

Children from very-low-income households during the early childhood years with persistent income poverty also had higher concentrations of hair cortisol over the 3-year follow-up period, suggesting higher chronic stress exposure. This is consistent with prior research documenting the impact of poverty on children's stress physiology.[22] Importantly, HCCs were not significantly higher among children with increasing income, which could suggest that even modest increases in annual household income may reduce family stress and thus reduce burden on children's developing stress systems. Although we had hypothesized that cortisol may mediate the relationship between poverty and childhood obesity risk, in our study, higher hair cortisol levels were not significantly associated with mean BMIp95 or increased rates of change in BMIp95. Prior studies have suggested that cortisol might promote fat accumulation, weight gain, and persistence of obesity in adults.[23-25] Although some studies have found high hair cortisol to be associated with obesity and higher BMI during childhood,[26,27] others have found no such association.[27,28] Our findings are consistent with a recent study that found no association between HCCs and obesity in a large sample of children and adolescents ages 4-18 years.[27] Also similar to these findings, a recent study of children and adults who experienced significant stressors in childhood (ie, maltreatment) found that although this history was related to higher cortisol levels and higher BMI, cortisol did not mediate the relationship between maltreatment and BMI.[29]

Our study has some limitations. The income and BMI criteria needed for eligibility into the obesity prevention trial (ie, <$65 000/year annual income and BMI ≥50th percentile) provided a restricted range of both income and BMI within which to detect associations. Thus, we were comparing children in families experiencing poverty to those in families that could be considered poor or near poor. However, even within the available range, our study yielded significant findings. Additionally, our analyses were limited by the examination of household income only and did not consider the complex set of environmental and psychosocial risks that could influence the association between income poverty, cortisol, and BMI trajectories, which should be evaluated in future studies. Measurement of cortisol from hair samples, although the most feasible approach for the current study, is not a thorough assessment of the stress pathway. Future studies should consider circulating stress biomarkers in addition to cortisol. Even though the current study adjusted for racial/ethnic differences in hair cortisol, this adjustment may not have fully accounted for potential differences due to hair color.[30] Additionally, inhaled or topical steroid use was not an exclusion, and use of oral steroids (although excluded at baseline) was not known at subsequent time points. The absence of normative values for hair cortisol among this age range also limits the conclusions that can be made regarding the clinical significance of cortisol levels and relative elevations.

Our findings suggest that the experience of household poverty and the persistence of poverty over time impact young children's cortisol levels and BMI trajectories, but these processes may be independent of one another. For example, poverty may have a more proximal impact on child obesity through limiting access to nutritious food or physical activity opportunities. In contrast, the downstream impact of chronically elevated cortisol on BMI could potentially be more applicable later in the life course. Alternatively, high cortisol levels could exert its impact on specific fat depots such as the visceral depot, which were not measured in our study but should be examined in future research. Our finding that poverty income was associated with higher hair cortisol levels is consistent with other studies examining the impact of household economic circumstances on children's developing stress systems, which have tied such elevations to long-term physical and mental health risks and future susceptibility to illness and chronic disease.[31,32] That very low household income was found to be associated with steeper BMIp95 increases in early childhood is also concerning for future health risk, as children who have obesity are more likely to become adults with obesity,[33] ultimately placing them at increased risk of heart disease, type 2 diabetes, and cancer.[34] ■

Submitted for publication Mar 18, 2022; last revision received Sep 2, 2022; accepted Sep 9, 2022.

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