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Early Maternal Sensitivity and Markers of Physical Health: Enduring or Transient Associations from Childhood to Adulthood?

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Abstract

Individual differences in the quality of early experiences with primary caregivers have been reliably implicated in the development of socioemotional adjustment and, more recently, physical health. However, few studies have examined the *development* of such associations with physical health into the adult years. To that end, the current study used prospective, longitudinal data from the NICHD Study of Early Child Care and Youth Development (N = 1,306, 52% male, 77% White/non-Hispanic) to investigate whether associations between direct observations of maternal sensitivity in the first three years of life and repeated assessments of two commonly used, objective indicators of physical health (i.e., body mass and mean arterial blood pressure) remained stable or diminished in magnitude over time. Associations between early maternal sensitivity and lower body mass remained relatively stable from age 54 months to 26 years and were robust to the modeling of autoregressive and second order stability processes as well as the inclusion of potential demographic confounders. In contrast, although associations between early caregiving and lower mean arterial pressure remained relatively stable from Grade 4 to age 15 years (the oldest age for which MAP was assessed thus far), these associations were not robust to the inclusion of covariates and the modeling of second order stability processes.

Keywords

Development; caregiving; health; trajectories; embedding; sensitivity

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Developmental scientists have long been interested in the role of parent-child relationship experiences in predicting later-life outcomes (e.g., Bowlby, 1969/1982; Cassidy & Shaver, 2018). Among the most well-documented of these experiences in the early life course is *maternal sensitivity*, the degree to which a mother responds to a child's cues for caregiving in a timely and effective manner (Ainsworth et al., 1978). According to many developmental accounts (e.g., attachment theory; see Cassidy & Shaver, 2018), sensitive early-life experiences with primary caregivers lay a foundation for effective engagement with subsequent salient developmental tasks (at least in part via internal working models; see Bowlby, 1973). A good deal of prior evidence supports this claim. For example, using the NICHD Study of Early Child Care and Youth Development (SECCYD), a large, normative risk sample of over 1000 families studied prospectively from infancy, Fraley and his colleagues reported that direct observations of sensitive maternal care predicted higher levels of cognitive and social skills, and lower levels of externalizing and internalizing symptoms from childhood through mid-adolescence (Fraley et al., 2013; Haltigan et al., 2013). Drawing on data from the Minnesota Longitudinal Study of Risk and Adaptation (MLSRA), a higher-risk sample of approximately 180 participants born into poverty, Raby and colleagues (2015) similarly found that maternal sensitivity observed in the first years of life predicted positive academic outcomes (e.g., educational attainment) and better social skills (e.g., the quality of romantic relationships) into the adult years.[1]

One burgeoning line of research has extended such work to examine the potential role of early maternal sensitivity in *physical* health outcomes later in life. Just as developmental theories of early caregiving (e.g., Bowlby, 1969/1982; Cassidy & Shaver, 2018) predict that sensitive parenting supports healthy socioemotional development, such accounts may be applicable to understanding the childhood antecedents of physical health as well. Consider, for instance, the case of obesity development: sensitive caregiving in the context of feeding behaviors involves the caregiver recognizing hunger/satiety cues from their child and, accordingly, either providing food or desisting from feeding. Some caregiver feeding practices that may be connected to insensitivity include missing the child's cues of hunger/satiation (Bergmeier et al., 2020; Patel et al., 2002) and excessive use of controlling behavior during feeding (Bergmeier et al., 2020; Haycraft & Blissett, 2008).

In addition to obesity, this theoretical framework (e.g., Bowlby, 1969/1982; Cassidy & Shaver, 2018) may be valuable in understanding the development of other health outcomes, including cardiovascular health markers and inflammation. Other work has supported the notion that psychosocial factors, including parenting, contribute to health outcomes through the lifespan. The biological embedding of childhood adversity model (Miller et al., 2011), for example, posits that psychosocial stressors occurring during sensitive periods (including

[1]There is evidence that other aspects of caregiving, in addition to maternal sensitivity, are associated with offspring outcomes. For instance, parental warmth, the degree to which a parent accepts, cares for, and supports their child (McKee et al., 2008) has been found to serve as a protective factor against internalizing and externalizing symptoms into adolescence (Rothenberg et al., 2020). Similarly, Rothenberg and colleagues (2022) found evidence that children's perceived parental rejection (i.e., a lack of warmth) is associated with later internalizing/externalizing symptoms. Notably, maternal sensitivity (as assessed here) includes parental warmth (i.e., quality and frequency of mother's expressions that connote positive feelings toward the child) as well as other aspects of parenting that are not captured by parental warmth alone, such as sensitivity to non-distress (i.e., mother's prompt and appropriate responses to child's behavior). As such, and given the wealth of theoretical and empirical support for the role of sensitivity in development (e.g., Cassidy & Shaver, 2018), we focus here on the association between early maternal sensitivity and later child outcomes.

early childhood) calibrate biological systems to prepare for comparable environments in the future. The immune system is most directly implicated in Miller and colleagues' (2011) model: They posit an increased inflammatory response to adverse/risky environments may be functionally beneficial in the short-term (e.g., promoting wound healing), while also conferring increased risk of inflammation-related disease over the long haul (e.g., hypertension).

Miller and colleagues' (2011) biological embedding model provides an attractive framework for understanding ways in which early experience can "get under the skin" to influence health outcomes. Consistent with this model, prior work suggests that individuals raised in households with low socioeconomic status (which is associated with harsher and more inconsistent parenting; Leventhal & Brooks-Gunn, 2000; McLoyd, 1990) develop a pro-inflammatory phenotype, including exaggerated inflammatory response to immune challenges (Miller et al., 2009), elevated levels of circulating inflammatory biomarkers such as C-Reactive Protein and Interleukin-6 (e.g., Danese et al., 2009; Schreier & Chen, 2010), and increased resistance to cortisol-mediated anti-inflammatory signaling in circulating immune cells (Miller et al., 2009). Of note, associations between early caregiving and health markers are not necessarily limited to non-normative (i.e., severe or atypical) experiences. The quality of parenting within the normative range has also been implicated in such associations. Buchmann and colleagues (2010), for instance, found that adverse interactions with parents at 3 months of age were associated with lower levels of high-density lipoprotein (i.e., "good" cholesterol) at age 19 years. Importantly, this model can be extended to non-immune markers of health, such that early sensitive caregiving may contribute to the development of anthropometric indicators of health (e.g., body mass index) as well as cardiovascular markers such as blood pressure.

Consistent with this framework, prior work using prospective, longitudinal data from the SECCYD reported that higher maternal sensitivity at 6 months of age (a composite variable that included maternal sensitivity/responsivity to child non-distress, warmth, and low intrusiveness) was associated with a lower risk of being overweight or obese at ages 5 to 12 years (Wu et al., 2011). Using data from the same study, O'Brien and colleagues (2007) found that children who first became overweight in preschool had mothers who were less sensitive compared to those who never became overweight by age 12 years. Likewise, Anderson and colleagues (2012) found that lower early relationship quality, using a combination of measures assessing maternal sensitivity and secure attachment, predicted higher BMI at age 15 years. Notably, Anderson and colleagues (2012) found that maternal sensitivity was a stronger predictor of BMI than was attachment classification.

Although BMI can be a valuable predictor of health outcomes, physical health is certainly more than a coarse summary of height and weight (e.g., Tomiyama et al., 2016). As such, it is vital to consider additional indicators of health that go beyond anthropometric markers. One such important objective marker is blood pressure, which is associated with a number of cardiovascular outcomes, including risk for stroke, coronary heart disease, and heart failure (Kane et al., 2014). As in the case of BMI, Miller and colleagues' (2011) framework can be extended to associations between early caregiving and blood pressure through the lifespan. Prior work with the SECCYD, for instance, reported that early (i.e., at age 54-months)

supportive parenting was associated with lower blood pressure from Grades 4 to 6 (Bell & Belsky, 2008). Similarly, work using comparable prospective, longitudinal datasets has found early maternal sensitivity to be associated with a cardiometabolic risk index that included BMI, mean arterial pressure, and C-reactive protein levels at ages 37 and 39 years (Farrell et al., 2019).

Enduring Effects versus Revisionist Models

Prior research supports the idea that maternal sensitivity in early life is associated with later indicators of health, including BMI and blood pressure. However, previous studies have not addressed whether these associations remain relatively stable (i.e., endure over time) or wane into adulthood (with relevant associations approaching zero as the temporal interval grows between the assessments of early experiences and later health). These alternative patterns, discussed in detail below, have significance for our understanding of the influence of early caregiving on life-long outcomes as well as the claim that early experiences might become increasingly reflected in physical health via accumulating wear and tear on biological systems. A number of theories of development posit that early caregiving carries special significance for later outcomes, such that the effects of early caregiving persist through the lifespan. However, it is presently unknown if the association between early caregiving and traditional health indicators, in particular, weakens or remains stable into adulthood, approaching some non-zero value at the limit. As such, in the present study, we leverage longitudinal, prospective data from the SECCYD to address this gap in the literature.

To this end, we adopted the framework proposed by Fraley, Roisman, and their colleagues (2013) for differentiating evidence for Enduring Effects and Revisionist models of human development (see also Haltigan et al., 2013; Magro et al., 2020; Raby et al., 2015). Briefly, the Revisionist model posits that early maternal sensitivity only correlates with later health to the extent that health itself is stable over time (see “c” paths in Figure 1). In contrast, the Enduring Effects model assumes that early maternal sensitivity has impacts on later functioning (e.g., health, see “b” paths in Figure 1a) that are not fully mediated through indirect pathways (i.e., stability in the outcome domain). One important consequence of the Enduring Effects model is that associations between a focal predictor and an outcome measured multiple times across development will not approach zero asymptotically. Moreover, both models can be expanded to include covariates and second-order stability in the outcome domain (i.e., transactional processes; Roisman et al., 2016), as well as other extensions (e.g., measured mediators of Enduring Effects; Magro et al., 2020), to more completely and realistically reflect the relevant developmental processes operative in a given domain. Crucially, the relative fit of these models can be tested against each other to determine which model provides a better account of the data.

In sum, this paper builds on a programmatic set of studies focusing on the predictive significance of direct observations of maternal sensitivity for a variety of indicators of social competence (Raby et al., 2015), academic skills (Raby et al., 2015), and symptoms of psychopathology (Haltigan et al., 2013) using prospective, longitudinal data. More specifically, the present study aims to describe associations between early maternal

sensitivity and objective physical health outcomes into adulthood and to determine whether these associations are better accounted for by an Enduring Effects or Revisionist model of human development. Evidence that such associations are consistent with an Enduring Effects model would be consistent with claims from attachment theory and models of biological embedding that early caregiving carries special significance for physical health outcomes, at least through the early adult years. Of note, we leverage both BMI and blood pressure data from the NICHD SECCYD here for two reasons. First, as reviewed above, there is reliable evidence for an association between maternal sensitivity and both BMI and blood pressure. Second, BMI and blood pressure are the two objective health markers most consistently assessed in the NICHD SECCYD, thus making them attractive candidates for modeling the development of such associations over time.

Method

Participants

The SECCYD began in 1991 when mothers of participating families were recruited from 24 US hospitals located near 10 research sites (Charlottesville, VA; Little Rock, AR; Irvine, CA; Lawrence, KS; Madison, WI; Morganton, NC; Philadelphia, PA; Seattle, WA; Pittsburgh, PA; and Wellesley, MA). To be included in the study, families were required to meet the following criteria: the mother was at least 18 years old, spoke English, was not engaging in substance abuse, was not ill, was not intending to place their child for adoption, and was intending to stay in the area for at least one year. Additionally, the child could not be enrolled in another study or born with serious medical complications. Finally, a small number of families from one site were not sampled due to concerns about the safety of the neighborhood in which they resided (NICHD Early Child Care Research Network, 2005).

Of the 8,996 mothers screened, 5,416 met the selection criteria, of which 1,364 were randomly selected for participation in the study. The average annual income of participating families was $37,947, which was comparable to the concurrent U.S. average ($36,875). More than half (56%) of the families were living above the poverty line, 23% near poverty, and 21% below the poverty line (NICHD Early Child Care Research Network, 2005). Data collection took place across five waves, beginning when the child participants were 1 month old. When the child participants were 15 years of age, parental consent and adolescent assent were obtained from 946 families for future projects. When the child participants were in 12th grade, 779 (of 888 contacted) adolescents agreed to complete an online survey (M_{age} = 18.38 years, SD = .31). At age 26, 875 participants were contacted and 814 agreed to participate and completed the online surveys. The present study used data collected from mothers and children from the inception of the SECCYD through this adult (i.e., age 26 year) assessment in 2019. The present analyses were conducted with participants who had any available maternal observational data collected at 6, 15, 24, or 36 months (N = 1,306). The analytic sample was 52% male and 77% were White/non-Hispanic. Descriptive statistics and correlations between all study variables are presented in Tables 1 and 3. All procedures were approved by the University of Minnesota Institutional Review Board (IRB) under protocol “Continuing Review for Study ‘Follow-up of the NICHD Study of Early Child Care and Youth Development’” (IRB 1207S16927).

Attrition Analysis.—We compared demographic characteristics of our analytic sample (N = 1,306) to those who were included in the original SECCYD sample but did not have sufficient data to include in our analyses (hereafter "attriters"; N = 58). The analytic sample was not significantly different than the attriters in percentages of male ($t(1362) = .53$, $p = .60$) nor White/non-Hispanic participants ($t(60.98) = -1.77$, $p = .08$). However, the analytic sample had significantly higher levels of maternal education ($M = 14.28$, $SD = 2.50$) than the attriters ($M = 13.16$, $SD = 2.49$; $t(1361) = -3.32$, $p < .001$, $d = .45$). The analytic sample also had higher income-to-needs ratios ($M = 3.45$, $SD = 2.71$) than the attriters ($M = 1.70$, $SD = 1.61$; $t(61.62) = -7.36$, $p < .001$, $d = .65$). Analyses were conducted using full information maximum likelihood estimation as this approach, when compared to listwise deletion, optimizes model fit, minimizes bias, and provides better standard error estimates (Enders & Bandalos, 2001; Newman, 2003). Sample sizes for each outcome variable are presented in Table 1 and 3 and missingness of each of the analytic variables can be derived from these Ns.

Early Maternal Sensitivity

Maternal parenting behavior was assessed when the study children were 6, 15, 24, and 36 months old ($N_{6months} = 1272$, $N_{15months} = 1240$, $N_{24months} = 1172$, $N_{36months} = 1161$). Children were filmed during a series of 15-minute semi-structured activities that required their mother's assistance to complete (NICHD Early Child Care Research Network, 1999). At the 6-, 15-, and 24- month assessments, videotapes were rated on 4-point scales for positive regard (i.e., quality and frequency of mother's expressions that connote positive feelings toward the child), sensitivity to non-distress (i.e., mother's prompt and appropriate responses to child's behavior), and intrusiveness (i.e., degree to which the mother imposes her own will on the child). At the 36-month assessment, videotapes were rated on 7-point scales for respect for autonomy (i.e., the degree to which the mother provides a sense of control to the child), supportive presence (i.e., the extent to which the mother provided a secure base for the child during the assessment), and hostility (i.e., the frequency and intensity of negative affect directed toward the child). The intrusiveness and hostility scales were then reverse-scored so that higher scores represented more sensitivity. Intercoder reliability scores were .87, .83, .84, and .84 and internal consistency ratings were .75, .70, .79, and .78 for the 6-, 15-, 24-, and 36-month assessments, respectively (NICHD Early Child Care Research Network, 1999). These internal consistency scores are above minimum values considered acceptable (Hammill et al., 1992). Therefore, scores were summed to create a composite measure of maternal sensitivity at each of the four time points. These four sums were standardized and averaged to create a composite measure of observed maternal sensitivity over the first 3 years of life ($\alpha = .73$). We have used this composite "early maternal sensitivity" variable in all of our prior work testing Enduring Effects and Revisionist models in the SECCYD (e.g., Fraley et al., 2013, Magro et al., 2020).

Body Mass Index (BMI)

Height and weight were collected when the study participants were 15, 24, 36, and 54 months old, as well as in Grades 1, 3, 5, 6, 7, and 8; at age 15; at the end of high school (EOHS, ~18 years of age); and at age 26. Height and weight from 15 months through 6th grade were assessed during laboratory visits and during clinic visits (health and pubertal

development assessments) from 7th grade through 15 years. Each assessment of height, with the exception of age 15 months, was conducted while the participant was standing. To assess height at age 15 months, participants were measured using an infantometer. While laying down, participants' legs were held flat against the infantometer's surface and a foot board was pushed up to the participants feet, allowing for a measurement from head to flat foot. Given that age 15-month length was measured as height is, as well as the high internal consistency of the 15-, 24-, and 36-month assessments of height (α = .92), we averaged these values to create a composite "early BMI" measure. The EOHS and age 26 assessments of height and weight were, in contrast, self-reported. Although self-reported and measured height and weight are highly correlated, self-reports tend to be less accurate at the extreme low and high ends of BMI (Stommel & Schoenborn, 2009). Height and weight were transformed into a body mass index (BMI) score by dividing weight in kilograms by height in meters squared. BMIs from the 15-, 24-, and 36-month assessments were averaged to create a composite early BMI variable due to their high stability (α = .89).

Mean Arterial Pressure (MAP)

Blood pressure (systolic and diastolic) were assessed during clinic visits when study participants were in Grades 4, 5, 6, 7, and 8 and age 15 years. Measurements were taken while the child was seated. A cuff and stethoscope were used on the non-dominant arm. MAP represents the average pressure in an individual's arteries during one cardiac cycle (DeMers & Wachs, 2020), which offers a broader assessment of cardiovascular function than either systolic or diastolic pressure alone. The normal range of MAP depends on an individual's body size and age, such that older children and taller children tend to have higher MAP (US Department of Health and Human Services, 2005). MAP was calculated for each assessment using the following formula (Ogunleye et al., 2012):

$$MAP = \frac{2}{3}DBP + \frac{1}{3}SBP$$

Covariates

We selected four control variables that have been used frequently in other research with the SECCYD data and are known correlates of maternal sensitivity (e.g., Fraley et al., 2013). At 1 month, mothers provided information on each child's sex, race/ethnicity, her own education level, and household income during an in-home interview. Child gender was coded in a binary fashion (1 = female, 2 = male). Because the majority of study children (76.9%) were not from a US racial or ethnic minority group, race and ethnicity information were collapsed to create a dummy variable (1 = White/non-Hispanic, 0 = other). Maternal education was recoded to represent the approximate number of years of education (e.g., "High school graduate or GED" = 12, "Bachelor's degree from college or university" = 16, etc.). Years of education ranged from 7 to 21. Additionally, household income was reported at ages 6, 15, 24, and 36 months. Poverty thresholds were determined for each participant by considering the year in which income was earned, number of members in the household, and number of children living full-time in the household. Household income was then divided by the poverty threshold to calculate an income-to-needs ratio for each of the five time points. These values were then averaged to create an index for early childhood (α = .94).

Income-to-needs ratios ranged from 0.14 to 18.76, with higher values indicating greater income relative to economic need.

Analytic Models

All analyses were conducted in R, version 4.0.2, using the package *lavaan* (Yves, 2012), with the bootstrapping option utilized for the estimates of standard errors (B = 1,000). We estimated the parameters of a series of models, depicted in Figure 1, using early maternal sensitivity to predict: (1) BMI and (2) MAP over time. Note that the Enduring Effects model estimates b pathways from early maternal sensitivity to later health outcomes. To test a Revisionist model, we fixed these paths to 0, thus assuming no direct effect of early maternal sensitivity on later health outcomes. The Revisionist models implies correlations between early caregiving and physical health indicators that , approach an effect size of zero asymptotically as the temporal lag grows between the predictor and outcome. We also compared two alternative specifications of the Enduring Effects model (as we have done in prior work; Fraley et al., 2013): one that allowed the b paths to vary (i.e., unconstrained) and another that constrained them to be equal at each time point (i.e., equality constrained).

In contrast to the Revisionist model, the Enduring Effects model implies associations between early caregiving and physical health indicators that approach a non-zero asymptotic value (see Fraley et al., 2013, for simulation evidence). Because the formal models that result from the Enduring Effects and Revisionist perspectives are nested, we were able to compare their respective fits with differences in chi-square tests (Fraley et al., 2013). We calculated additional estimates of model fit, including the comparative fit index (CFI), root mean square error of approximation (RMSEA), and standardized root mean squared residual (SRMR) for each model. Values that indicate acceptable model fit for these measures are: CFI > .95, RMSEA < .06, and SRMR < .08 (Hu & Bentler, 1999).

After comparing the basic Revisionist models to the basic Enduring Effects models, we increased the complexity of the models to include covariates (Figure 1b). All covariates were included and retained, regardless of their effect size, consistent with prior work (Fraley et al., 2013). Finally, we estimated models that included covariates *and* transactional pathways (Figure 1c; Roisman et al., 2016). Transactional processes reflect developmental perspectives positing that individuals shape the environment around them while also being shaped by the environment that they are shaping (e.g., Gottlieb, 2007). These processes can be modeled in a structural equation framework by including second-order stability paths (i.e., X_T to X_{T+2} paths in addition to X_T to X_{T+1} paths). These paths capture any indirect effects of the earlier outcome on the later one that are not due to autoregressive stability alone. Although these second-order stabilities do not necessarily capture the exact nature of the transactional processes within the observed model, they do serve as an omnibus method of detecting transactional processes via any number of unmeasured environmental mechanisms (Roisman et al., 2016).”These increasingly complex models represent more conservative tests of the basic model comparisons and served as an important robustness check given the non-experimental nature of the SECCYD.

Data Availability

Data and study materials are publicly available through age 15 years through the ICPSR at the University of Michigan (https://www.icpsr.umich.edu//web/pages/). Although data from subsequent assessments (i.e., EOHS and age 26 years) are not publicly available, variables needed to reproduce the analyses presented here are available from the authors upon request by qualified researchers. The present work was not preregistered.

Results

Body Mass Index

Basic Model.—Table 1 presents the correlations between early maternal sensitivity and body mass index. Early maternal sensitivity was negatively associated with BMI at each time point. These associations were generally small to moderate in magnitude (*r*s = −.09 to −.24). Notably, the smallest correlation was the contemporaneous one (i.e., the composite early BMI that overlapped with the assessments of maternal sensitivity) with correlations modestly increasing in magnitude thereafter. Model fit statistics and model comparisons are presented in Table 2 and the path estimates for all models tested can be found in the Supplemental Materials.

In the basic model, the equality-constrained Enduring Effects *b* paths were −0.11 (standardized β estimates ranged from −0.01 to −0.05, M_β = −0.02, $p < .001$). Additionally, the non-constrained Enduring Effects *b* paths ranged from −1.03 to 0.02, with an average effect size of −0.20 (standardized β estimates ranged from −0.13 to 0.00, M_β = −0.03). However, the only effects that reached significance (i.e., $p < .050$) were those at Grades 1 and 5, age 15 years, and age 26 years (Supplemental Table 1). This unconstrained model showed a significantly better fit compared to the equality constraint model ($\Delta\chi^2 = 36.29$, $p < .001$). Fixing the *b* paths to 0 (i.e., as in the Revisionist model) resulted in a significant decrement in fit compared to both Enduring Effects models (Table 2).

Basic Model + Covariates.—After including all covariates, both Enduring Effects models continued to show improved fit over the Revisionist model (Table 2). When including all covariates, the unconstrained Enduring Effects model continued to show improved fit over the equality constrained Enduring effects model ($\Delta\chi^2 = 22.70$, $p = .007$).

The equality constrained Enduring Effects model's estimated *b* paths were estimated to be −0.08 (standardized β estimates ranged from −0.01 to −0.04, M_β = −0.02, $p = .003$). In this equality constrained model, sex positively predicted BMI at 54 months ($b = 0.14$, $p = .044$), Grade 7 ($b = 0.25$, $p = .012$), age 15 ($b = -0.27$, $p = .045$), and end of high school ($b = -0.64$, $p = .001$). Maternal education also significantly predicted BMI at Grade 7 ($b = -0.05$, $p = .037$), age 15 ($b = -0.07$, $p = .029$), and end of high school ($b = 0.12$, $p = .010$). Additionally, childhood income-to-needs ratio significantly predicted BMI at Grade 1 ($b = -0.04$, $p = .038$) and age 26 ($b = -0.18$, $p = .005$). Race/ethnicity did not uniquely predict BMI at any of the observed time points in the equality constrained Enduring Effects model.

The unconstrained Enduring Effects *b* paths ranged from −0.77 to 0.07, with an average effect size of −0.15 (standardized β estimates ranged from −0.10 to 0.02, M_β = −0.02). In

this unconstrained model, the only effects that reached significance (i.e., $p < .050$) were those at ages 15 and 26 years (Supplemental Table 1). Sex significantly predicted BMI at Grade 7 ($b = 0.23$, $p = .021$) and end of high school ($b = -0.64$, $p = .001$), as did income-to-needs ratio at Grade 1 ($b = -0.04$, $p = .049$), Grade 3 ($b = -0.05$, $p = .024$), and age 26 years ($b = -0.15$, $p = .011$). Maternal education also significantly predicted BMI at Grade 8 ($b = -0.06$, $p = .008$) and end of high school ($b = 0.12$, $p = .014$). Race/ethnicity did not uniquely predict BMI at any of the observed time points in the unconstrained Enduring Effects model.

Covariates + Second-Order Stability.—The final models included second-order autoregressive pathways. Again, both Enduring Effects models showed improved fit compared to the Revisionist model (Table 2) and the unconstrained Enduring Effects model showed improved fit over the equality constrained Enduring effects model ($\Delta\chi^2 = 21.86$, $p = .009$).

The b paths in the equality constrained Enduring Effects model were −0.08 (standardized β estimates ranged from −0.01 to −0.04, $M_\beta = -0.02$, $p = .002$; Supplemental Table 1). In this constrained model, child race/ethnicity significantly predicted BMI at Grade 7 ($b = 0.25$, $p = .035$), as did child sex at 54 months ($b = 0.14$, $p = .042$), Grade 7 ($b = 0.31$, $p = .001$), Age 15 ($b = -0.30$, $p = .031$), and end of high school ($b = -0.66$, $p = .001$). Additionally, maternal education significantly predicted BMI at Grade 7 ($b = -0.05$, $p = .013$), Age 15 ($b = -0.06$, $p = .047$), and end of high school ($b = 0.11$, $p = .021$) and childhood income-to-needs ratio predicted BMI at Grade 1 ($b = -0.04$, $p = .037$) and age 26 ($b = -0.19$, $p = .002$).

The unconstrained Enduring Effects b paths ranged from −0.71 to 0.08, with an average effect size of −0.15 (standardized β estimates ranged from −0.09 to 0.02, $M_\beta = -0.02$). In this unconstrained model, the only effects that reached significance (i.e., $p < .050$) were those at ages 15 and 26 years (Supplemental Table 1). Sex significantly predicted BMI at 54 months ($b = 0.14$, $p = .048$), Grade 7 ($b = 0.29$, $p = .002$), age 15 years ($b = -0.27$, $p = .047$), and end of high school ($b = -0.65$, $p = .001$). Income-to-needs ratio also significantly predicted BMI at Grade 1 ($b = -0.04$, $p = .044$), Grade 3 ($b = -0.05$, $p = .033$), and age 26 years ($b = -0.16$, $p = .007$), as did maternal education at Grade 7 ($b = -0.06$, $p = .004$) and end of high school ($b = 0.12$, $p = .015$). Race/ethnicity did not uniquely predict BMI at any of the observed time points in the unconstrained Enduring Effects model. Taken together, the results above indicate that the Enduring Effects model, both with and without equality constraints, provides a better account for the observed associations between early maternal sensitivity and BMI than the Revisionist model.

Mean Arterial Pressure

Basic Model.—Table 3 reports the correlations between early maternal sensitivity and measurements of MAP from 4th grade to age 15 (the oldest age for which MAP was assessed). Early maternal sensitivity was negatively associated at each time point and these associations were generally small in magnitude (rs = −.06 to −.13). The smallest correlations were observed at the Grade 4 and Grade 5 assessments, and these associations

were not statistically significant. Model fit statistics and model comparisons are presented in Table 4. In the basic model, the equality-constrained Enduring Effects *b* paths were −0.61 (standardized β estimates ranged from −0.05 to −0.07, $M_\beta = -0.06$, $p < .001$; Supplemental Table 17).

There was not a significant difference in fit between the equality constrained and unconstrained models. As such, the equality constrained Enduring Effects model was adopted as the more parsimonious model. Conversely, fixing the *b* paths to 0 (i.e., as in the Revisionist model) resulted in a significant decrement in fit compared to both Enduring Effects models (Table 5). In sum, the equality constrained Enduring Effects model better accounted for the observed patterns than did the Revisionist or unconstrained Enduring Effects models.

Basic Model + Covariates.—After including all covariates, neither the equality-constrained nor the unconstrained Enduring Effects models showed improved fit over the Revisionist one ($\Delta\chi^2 = 2.89$, $p = .089$). The revisionist model was thus adopted as the more parsimonious model. In the Revisionist model, maternal education significantly predicted MAP at Grade 7 ($b = -0.34$, $p = .008$), as did child race/ethnicity at Grade 8 ($b = -1.89$ $p = .015$). Child sex at Grade 8 ($b = -1.52$, $p = .012$) and age 15 ($b = -2.10$, $p < .001$) also significantly predicted MAP. Income-to-needs ratio did not significantly predict MAP at any of the observed time points.

Covariates + Second-Order Stability.—The final models included second-order autoregressive pathways, as well as covariates. Once again, the equality-constrained and unconstrained Enduring Effects models did not show a better fit to the data compared to the Revisionist model ($\Delta\chi^2 = 2.52$, $p = .113$). In the Revisionist model, second-order autoregressive pathways each significantly predicted MAP, as did maternal education at Grade 7 ($b = -0.29$, $p = .026$) and child ethnicity at Grade 8 ($b = -2.20$, $p = .003$). Sex also significantly predicted MAP at Grade 8 ($b = -1.35$, $p = .016$) and age 15 ($b = -2.13$, $p < .001$). Again, income-to-needs ratio did not significantly predict MAP at any of the observed time points. Taken together, the results above indicate that the Enduring Effects model *did not* provide a better account for the association between early maternal sensitivity and MAP compared to the Revisionist one, after accounting for covariates and/or second-order autoregressive pathways.

Discussion

The extent to which early experiences are related to later outcomes is among the most enduring questions in developmental science. Some perspectives assume that early experiences, such as maternal sensitivity, are related to specific outcomes in early childhood but the long-term associations eventually fade away as individuals develop (i.e., a Revisionist account). Other perspectives assume that early experiences continue to manifest in later outcomes in ways that are sustained over time and do not continuously fade (i.e., an Enduring Effects account). The purpose of the present study was to examine which of these perspectives best accounts for the associations between early maternal sensitivity and two commonly used measures of health: body mass (i.e., BMI) and blood pressure.

Leveraging existing archival and recently acquired adult data from the large, normative risk SECCYD cohort, this study shows that early maternal sensitivity is predictive of health markers into adolescence and adulthood. The association between early maternal sensitivity and later BMI was more consistent with an Enduring Effects model (i.e., with direct effects of early sensitivity on later BMI) than with a Revisionist model (i.e., with no direct effect of early sensitivity on later BMI), even after controlling for covariates and second-order autoregressive pathways. Additionally, the unconstrained Enduring Effects model better accounted for the association between early maternal sensitivity and BMI than did the equality constrained model. In contrast to the BMI models, bivariate associations between early caregiving and lower mean arterial blood pressure appeared to remain relatively stable from Grade 4 to age 15 years, the oldest age for which this outcome was assessed. The associations between early caregiving and later MAP (i.e., in the Enduring Effects model) were generally weak and not robust to more complex model specifications.

Of particular interest, we note that the bivariate associations between early maternal sensitivity and BMI increased somewhat in magnitude from age 15 months to age 26 years. Furthermore, when covariates and transactional processes were included in the unconstrained Enduring Effects model, the effect of early maternal sensitivity on BMI did not emerge as statistically significant until ages 15 and 26 years (although the sensitivity-end of high school association was also not statistically significant). Though any such conclusion would require additional assessments of physical health in the SECCYD, one possibility is that this pattern may reflect a “sleeper effect” of early maternal sensitivity on BMI, such that the impacts of these early experiences do not become fully apparent until later in the lifespan. Such sleeper effects may be a result of accumulating and/or compounding small effects through childhood and into adulthood. Notably, such effects may be more likely to appear in relation to health outcomes influenced by the cumulative biological embedding of early experience (e.g., Miller et al., 2011), which may take years or even decades to fully emerge in an observable manner at the population level. Importantly, it is also possible that these effects grow larger in part due to increasing variation in BMI into adolescence and adulthood.

We did not observe a similar pattern for blood pressure. However, it should be noted that MAP measurements are not currently available on the SECCYD beyond mid-adolescence, potentially limiting age-related variation and, in turn, covariation between early maternal sensitivity and MAP. This limitation of the data is notable in the case of detecting possible sleeper effects, but it is more important in the impact it may have on conclusions reached regarding the patterns of associations. As already noted, it is possible given the cumulative nature of Miller and colleagues’ (2011) hypothesized biological embedding that the effects of early maternal sensitivity on MAP may not emerge until adulthood. If this is the case, and future work focused on MAP and early maternal sensitivity find a strengthening association between the two variables past age 15 years, we might expect the Enduring Effects model to provide a better fit to such data than in the present study. That said, based on the data collected to date, we conclude that the Revisionist model is a better fit for the pattern of associations observed between early maternal sensitivity and MAP in the SECCYD.

This study is limited in several respects. First, the focus was on just two of many indicators of physical health: body mass and blood pressure. Second, this longitudinal analysis is inherently limited by the sociodemographic characteristics of its sample. The SECCYD sample was roughly representative of the population at each data collection site at its inception, per U.S. Census data (ECCRN, 2001). However, given demographic shifts, future work in this domain would benefit from more diverse and representative samples. Third, due to the nature of the SECCYD research design, we are unable to rule out potential genetic factors that may (directly or indirectly) influence height, weight, and/or blood pressure. As such, genetically informed studies (e.g., twin, adoption, and other within-family designs) would be valuable in evaluating genetic confounding of the associations studied here. Relatedly, the SECCYD sample does not have prospective measures of parental health and, as such, this potential familial influence cannot be ruled out. Fourth, the present results represent only one possible set of models representing the association between early caregiving and physical health. For instance, we present results of separate models of BMI and MAP, due to the relatively weak association between the two (Table 5) and lack of MAP measurements in early childhood and after age 15 years. Nonetheless, one could model these markers simultaneously[2]. Finally, and as noted above, there is a lack of physical health data available after ages 15 years (for MAP) and 26 years (for BMI), which limits our ability to draw conclusions about the pattern of these associations into later adulthood, where we will no doubt observe greater disparities in health outcomes. This lack of follow-up information on the health of SECCYD participants into the years of maturity underlines the need for continued data collection from the cohort so that we will be increasingly well positioned to explore questions about the patterns of associations between early experience and health outcomes. Such efforts are already underway and offer a promising opportunity to extend the current (and other) work.

The present study extends findings and methods of a programmatic line of work focused on how associations between early caregiving and subsequent child and adult characteristics are structured over time, thereby addressing a central aim of developmental science (e.g., Fraley et al., 2013; Haltigan et al., 2013; Magro et al., 2020; Raby et al., 2015). Specifically, we provide evidence here that early maternal sensitivity is associated with later BMI in a manner better accounted for by an Enduring Effects model than a Revisionist model. In contrast, we provide evidence that the associations observed thus far between early maternal sensitivity and later MAP is better accounted for by a Revisionist model. Taken together, these findings provide a starting point for future work focused on how early caregiving influences on later health are structured over the life course.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

[2]We include the results of preliminary analyses modeling BMI and MAP simultaneously in the supplemental materials (Supplemental Table 34-Supplemental Table 53). These results mirror those of the MAP models: When using the basic model specification, the Enduring Effects model provided a better account of the observed associations, although the Revisionist model provided a better account when including covariates and/or transactional processes. Notably, these represent only one way of modeling BMI and MAP simultaneously: one could also use a latent factor of health with BMI, MAP, and/or other markers of health loaded onto it at each time point.

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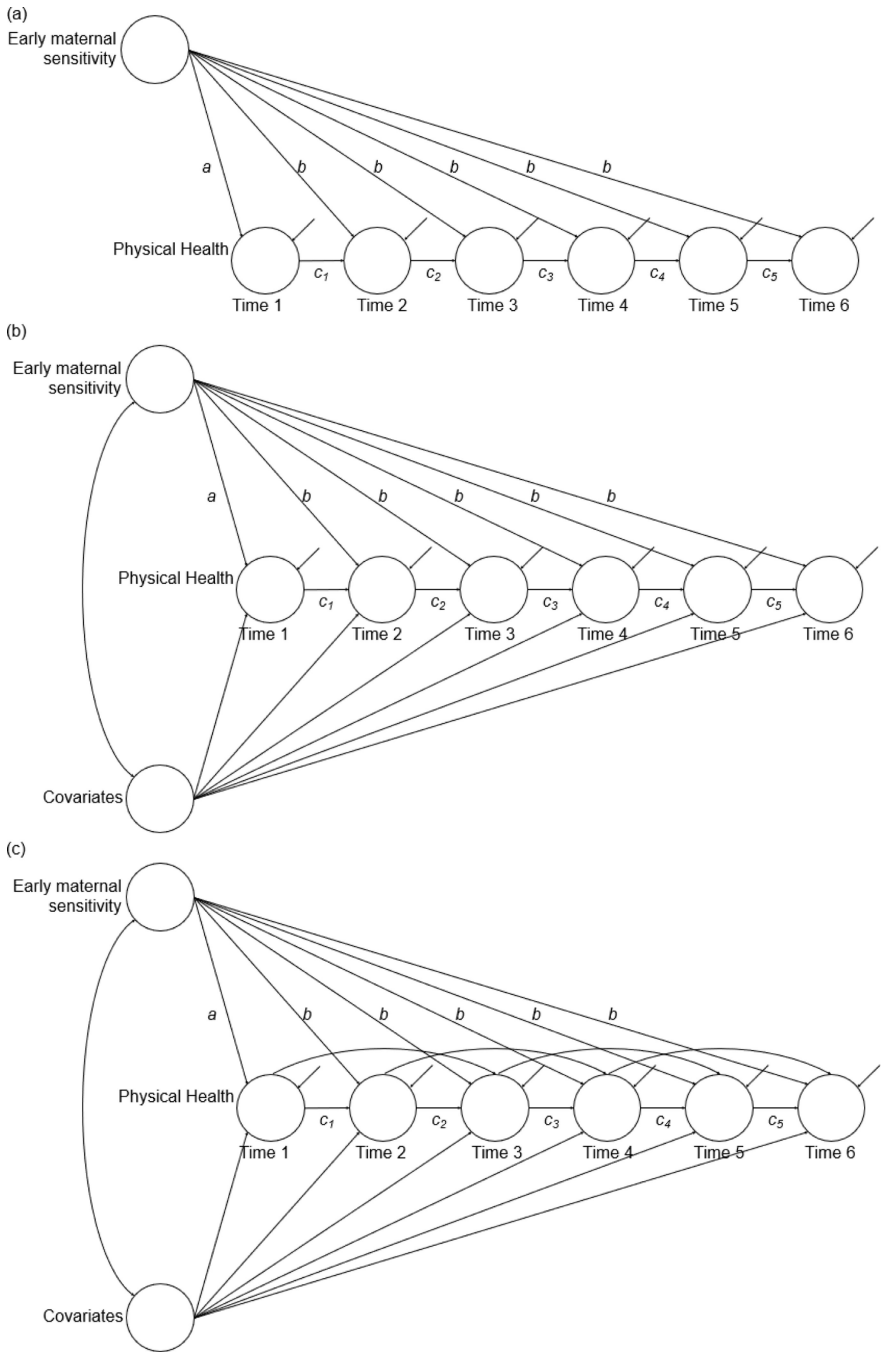

Figure 1.

Conceptual models of the influence of early maternal sensitivity on physical health markers. *Note:* The path models above represent the Enduring Effects model. However, fixing the *b* paths to zero results in the Revisionist model. As such, the Revisionist model is nested within the Enduring Effects model, allowing for model comparisons to be made. The X_T to X_{T+2} paths in Model "c" represent an omnibus method for detecting transactional processes. Complete models may be found in the Supplemental Materials.

Table 1

Correlations, Means, and Standard Deviations of Early Maternal Sensitivity, Body Mass Index, and Covariates

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. Early maternal sensitivity	—															
2. Early body mass index	**−.09**	—														
3. Body mass index, 54 months	**−.10**	**.65**	—													
4. Body mass index, G1	**−.12**	**.52**	**.83**	—												
5. Body mass index, G3	**−.13**	**.46**	**.76**	**.92**	—											
6. Body mass index, G5	**−.17**	**.43**	**.70**	**.86**	**.94**	—										
7. Body mass index, G6	**−.17**	**.44**	**.69**	**.84**	**.92**	**.95**	—									
8. Body mass index, G7	**−.17**	**.40**	**.67**	**.83**	**.90**	**.95**	**.96**	—								
9. Body mass index, G8	**−.14**	**.41**	**.64**	**.80**	**.88**	**.92**	**.93**	**.96**	—							
10. Body mass index, age 15	**−.20**	**.40**	**.63**	**.77**	**.84**	**.86**	**.87**	**.90**	**.93**	—						
11. Body mass index, EOHS	**−.17**	**.37**	**.58**	**.68**	**.73**	**.77**	**.76**	**.90**	**.83**	**.85**	—					
12. Body mass index, age 26	**−.24**	**.31**	**.48**	**.58**	**.61**	**.66**	**.67**	**.69**	**.69**	**.72**	**.77**	—				
13. Child gender	**.09**	**−.16**	−.06	−.05	−.06	−.04	−.02	−.01	−.04	−.04	**−.11**	**−.07**	—			
14. Child ethnicity	**.38**	**−.09**	−.04	−.05	**−.08**	**−.13**	**−.12**	**−.11**	**−.12**	**−.11**	−.04	**−.07**	.00	—		
15. Maternal education	**.50**	**−.07**	**−.10**	**−.10**	**−.13**	**−.14**	**−.15**	**−.16**	**−.14**	**−.20**	**−.14**	**−.17**	.04	**.22**	—	
16. Income-to-needs ratio	**.41**	**−.08**	**−.06**	**−.11**	**−.15**	**−.18**	**−.20**	**−.20**	**−.17**	**−.20**	**−.16**	**−.21**	.04	**.23**	**.54**	—
N	1306	1230	1031	991	933	925	914	797	738	841	760	796	1306	1306	1306	1304
M	−.02	16.76	16.05	16.82	18.43	20.11	20.86	21.39	22.04	23.10	23.95	26.45	1.48	.77	14.28	3.45
SD	.77	1.43	1.56	2.54	3.75	4.71	4.95	5.15	5.15	5.17	5.03	6.10	.50	.42	2.50	2.71

Note: Gender was coded as 1 = female, 2 = male. Ethnicity was coded as 1 = White/non-Hispanic, 0 = non-White. Bolded values are associated with $p < .05$. $N = 1306$.

Table 2

Model Fit Estimates for the Influence of Early Maternal Sensitivity on Body Mass Index Across Childhood and Into Adulthood

	Model fit						Nested model comparisons			
Model	χ^2	*df*	*p*	**CFI**	**RMSEA**	**SRMR**		$\Delta\chi^2$	*df*	*p*
Basic Model										
A. Revisionist	330.62	55	< .001	.98	.06	.06	A – C	64.14	10	< .001
B. Enduring: Constrained	302.75	54	< .001	.98	.06	.05	A – B	27.88	1	< .001
C. Enduring: Unconstrained	266.48	45	< .001	.99	.06	.05	B – C	36.26	9	< .001
Basic Model with Covariates										
A. Revisionist	300.90	55	< .001	.98	.06	.04	A – C	31.14	10	< .001
B. Enduring: Constrained	292.47	54	< .001	.98	.06	.04	A – B	8.43	1	.004
C. Enduring: Unconstrained	269.77	45	< .001	.99	.06	.04	B – C	22.70	9	.007
Transactional Model with Covariates										
A. Revisionist	104.38	46	< .001	1.00	.03	.02	A – C	30.75	10	< .001
B. Enduring: Constrained	95.49	45	< .001	1.00	.03	.01	A – B	8.89	1	.003
C. Enduring: Unconstrained	73.63	36	< .001	1.00	.03	.01	B – C	21.86	9	.009

Note. CFI = comparative fit index. RMSEA = root mean-square-error of approximation. SRMR = standardized root-mean-square residual. $N = 1306$.

Table 3

Correlations, Means, and Standard Deviations of Early Maternal Sensitivity, Mean Arterial Pressure, and Covariates

	1	2	3	4	5	6	7	8	9	10	11
1. Early maternal sensitivity	—										
2. Mean arterial blood pressure, G4	−.06	—									
3. Mean arterial blood pressure, G5	−.06	**.36**	—								
4. Mean arterial blood pressure, G6	**−.08**	**.35**	**.44**	—							
5. Mean arterial blood pressure, G7	**−.13**	**.24**	**.34**	**.42**	—						
6. Mean arterial blood pressure, G8	**−.10**	**.28**	**.42**	**.43**	**.46**	—					
7. Mean arterial blood pressure, Age 15	**−.08**	**.21**	**.23**	**.24**	**.21**	**.31**	—				
8. Child gender	**.09**	−.05	−.03	−.04	−.04	**−.11**	**−.18**	—			
9. Child ethnicity	**.38**	.38	−.04	−.02	**−.08**	**−.11**	**−.07**	.00	—		
10. Maternal education	**.50**	**−.07**	−.04	**−.07**	**−.13**	−.03	**−.07**	.04	**.22**	—	
11. Income-to-needs ratio	**.41**	.01	.01	−.03	**-.07**	.01	−.06	.04	**.23**	**.54**	—
N	1306	874	829	802	791	737	854	1306	1306	1306	1304
M	−.02	74.49	75.36	76.09	76.54	78.87	79.74	1.48	.77	14.28	3.45
SD	.77	8.03	8.59	8.49	8.62	9.10	7.01	.50	.42	2.50	2.71

Note: Gender was coded as 1 = female, 2 = male. Ethnicity was coded as 1 = White/non-Hispanic, 0 = non-White. Bolded values are associated with $p < .05$.

Table 4

Model Fit Estimates for the Influence of Early Maternal Sensitivity on Mean Arterial Pressure Across Childhood and Into Adulthood

	Model fit						Nested model comparisons			
Model	χ^2	*df*	*p*	**CFI**	**RMSEA**	**SRMR**		$\Delta\chi^2$	*df*	*p*
Basic Model										
A. Revisionist	218.47	15	< .001	.76	.10	.12	A – C	18.36	5	.003
B. Enduring: Constrained	202.57	14	< .001	.78	.10	.11	A – B	15.90	1	< .001
C. Enduring: Unconstrained	200.11	10	< .001	.78	.10	.11	B – C	2.46	4	.651
Basic Model with Covariates										
A. Revisionist	201.95	15	< .001	.79	.10	.07	A – C	4.57	5	.470
B. Enduring: Constrained	199.06	14	< .001	.79	.10	.07	A – B	2.89	1	.089
C. Enduring: Unconstrained	197.37	10	< .001	.79	.12	.07	B – C	1.68	4	.794
Transactional Model with Covariates										
A. Revisionist	66.86	11	< .001	.94	.06	.03	A – C	4.31	5	.506
B. Enduring: Constrained	64.35	10	< .001	.94	.07	.03	A – B	2.52	1	.113
C. Enduring: Unconstrained	62.56	6	< .001	.94	.09	.03	B – C	1.79	4	.774

Note. CFI = comparative fit index. RMSEA = root mean-square-error of approximation. SRMR = standardized root-mean-square residual. $N = 1306$.

Table 5

Correlations Between Body Mass Index and Mean Arterial Pressure

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1. Mean arterial blood pressure, G4	—																
2. Mean arterial blood pressure, G5	**.36**	—															
3. Mean arterial blood pressure, G6	**.35**	**.44**	—														
4. Mean arterial blood pressure, G7	**.24**	**.34**	**.42**	—													
5. Mean arterial blood pressure, G8	**.28**	**.42**	**.43**	**.46**	—												
6. Mean arterial blood pressure, Age 15	**.21**	**.23**	**.24**	**.21**	**.31**	—											
7. Early body mass index	**.12**	**.17**	**.13**	**.13**	**.20**	**.17**	—										
8. Body mass index, 54 months	**.27**	**.28**	**.30**	**.28**	**.30**	**.21**	**.65**	—									
9. Body mass index, G1	**.32**	**.31**	**.34**	**.34**	**.35**	**.26**	**.52**	**.83**	—								
10. Body mass index, G3	**.35**	**.34**	**.40**	**.39**	**.38**	**.26**	**.46**	**.76**	**.92**	—							
11. Body mass index, G5	**.36**	**.37**	**.42**	**.39**	**.39**	**.30**	**.43**	**.70**	**.86**	**.94**	—						
12. Body mass index, G6	**.35**	**.32**	**.39**	**.40**	**.37**	**.28**	**.44**	**.69**	**.84**	**.92**	**.95**	—					
13. Body mass index, G7	**.40**	**.35**	**.37**	**.39**	**.36**	**.31**	**.40**	**.67**	**.83**	**.90**	**.95**	**.96**	—				
14. Body mass index, G8	**.35**	**.37**	**.36**	**.38**	**.42**	**.35**	**.41**	**.64**	**.80**	**.88**	**.92**	**.93**	**.96**	—			
15. Body mass index, age 15	**.32**	**.37**	**.38**	**.37**	**.42**	**.34**	**.40**	**.63**	**.77**	**.84**	**.86**	**.87**	**.90**	**.93**	—		
16. Body mass index, EOHS	**.26**	**.32**	**.31**	**.33**	**.38**	**.34**	**.37**	**.58**	**.68**	**.73**	**.77**	**.76**	**.90**	**.83**	**.85**	—	
17. Body mass index, age 26	**.22**	**.23**	**.24**	**.30**	**.27**	**.26**	**.31**	**.48**	**.58**	**.61**	**.66**	**.67**	**.69**	**.69**	**.72**	**.77**	—

Note: Bolded values are associated with $p < .05$.