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Early external-environmental and internal-health predictors of risky sexual and aggressive behavior in adolescence: An integrative approach

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Abstract

External predictive adaptive response (PAR) models assume that developmental exposures to stress carry predictive information about the future state of the environment, and that development of a faster life history (LH) strategy in this context functions to match the individual to this expected harsh state. More recently internal PAR models have proposed that early somatic condition (i.e., physical health) critically regulates development of LH strategies to match expected future somatic condition. Here we test the integrative hypothesis that poor physical health mediates the relation between early adversity and faster LH strategies. Data were drawn from a longitudinal study (birth to age 16; N = 1,388) of mostly African American participants with prenatal substance exposure. Results demonstrated that both external environmental conditions early in life (prenatal substance exposure, socioeconomic adversity, caregiver distress/depression, and adverse family functioning) and internal somatic condition during preadolescence (birthweight/gestational age, physical illness) uniquely predicted the development of faster LH strategies in adolescence (as indicated by more risky sexual and aggressive behavior). Consistent with the integrative hypothesis, the effect of caregiver distress/depression on LH strategy was mostly mediated by worse physical health. Discussion highlights the implications of these findings for theory and research on stress, development, and health.

KEYWORDS

conduct symptoms, early life stress, life history strategy, longitudinal research, physical health, predictive adaptive response, risky sexual behavior

1 | INTRODUCTION

Decades of research has linked early life stress to both poor health outcomes (e.g., Evans & Kim, 2007; Shonkoff et al., 2012) and more risky and aggressive behavior (e.g., Belsky, Schlomer, & Ellis, 2012; Doom, Vanzomeren-Dohm, & Simpson, 2016; Kerig, 2019). These two effects, however, are often conceptualized in different theoretical frameworks. The effects of childhood adversity on health have typically been interpreted within an allostatic load framework (Lupien et al., 2006; McEwen & Stellar, 1993), which proposes that the wear and tear on multiple tissues and organ systems resulting from repeated physiological adaptations to stress predisposes the individual to disease. The emphasis of the allostatic load model is thus on the effects of early life stress on deterioration of internal somatic condition. In contrast, the effects of childhood adversity on risky and aggressive behavior have often been interpreted within a developmental life history framework (e.g., Doom et al., 2016; Ellis et al., 2012), which proposes that early exposures to harsh, unpredictable environments regulates development toward life history strategies that are (or at least once were) adaptive in such

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environments. A key feature of developmental life history models is that they are forward looking; the developing person uses early experiences to predict and prepare for future conditions they are likely to encounter. In this framework, developmental adaptations to adversity, including shifts toward a *faster* life history strategy (characterized by more risky and aggressive behavior), enable the individual to "make the best of a bad job" (i.e., to mitigate the inevitable fitness costs), even though "the best" may still constitute a high-risk strategy that jeopardizes health and survival, as per models of allostatic load.

Although the allostatic load framework, rooted in biological and neuroscience approaches to *how* disease develops, and the life history framework, based on evolutionary models of *why* development occurs that way that it does, have largely operated independently, recent theory and research (e.g., Hartman, Zhi, Nettle, & Belsky, 2017; Nettle, Frankenhuis, & Rickard, 2013; Rickard, Frankenhuis, & Nettle, 2014) have brought these perspectives closer together. This has led to new ways of thinking about the relations between early life stress, health outcomes, and the development of life history strategies. In this paper we explicate this integrated perspective, focusing on the hypothesis that poor physical health mediates the relation between early adversity and faster life history strategies. We provide an empirical test of this hypothesis in a longitudinal study of socially and economically disadvantaged youth.

1.1 | The life history framework: External and internal predictive adaptive response models

In evolutionary biology, a major framework for explaining coordinated patterns of variation in development is *life history theory* (Roff, 2002; Stearns, 1992). Life history theory deals with the way organisms allocate their limited time and energy to the various activities (including growth, maintenance of bodily tissues, mating, and parenting) that comprise their life cycle. Since all these activities ultimately contribute to the organism's fitness, devoting time and energy to one will typically involve both benefits and costs, engendering trade-offs between different fitness components. Natural selection favors organisms that schedule developmental activities in ways that optimize resource allocation. This chain of resource-allocation decisions over the lifespan—expressed in the development of an integrated suite of physiological and behavioral traits—constitutes the individual's *life history strategy*. An organism's life history strategy coordinates morphology, physiology, and behavior in ways that maximizes expected fitness in a given environment (Del Giudice, 2020; Ellis, Figueredo, Brumbach, & Schlomer, 2009).

At the broadest level of analysis, life history-related traits appear to covary along a dimension of *slow* versus *fast* life history, reflecting the different trade-offs that individuals face in different environmental contexts. Although there is ongoing debate about the robustness of the slow-fast continuum across species (for a review, see Del Giudice, 2020), and about the best ways to characterize human life history variation (e.g., Copping, Campbell, & Muncer, 2014; Figueredo et al., 2015), substantial empirical evidence supports a slow-fast continuum in humans. Specifically, some people adopt slower strategies characterized by later reproductive development (especially in girls) and delayed sexuality, preferences for stable pair bonds and high investment in parenting, an orientation toward future outcomes, low impulsivity, and allocation of resources toward enhancing long-term survival; others display faster strategies characterized by the opposite pattern (Belsky, 2012; Belsky, Steinberg, & Draper, 1991; Del Giudice, Gangestad, & Kaplan, 2015; Ellis et al., 2009; Figueredo et al., 2006).

Fast life history strategies are comparatively high risk, focusing on mating effort (including more risky and aggressive behavior), reproducing at younger ages, and producing a greater number of offspring with more variable outcomes. From a life history perspective, the clear clustering of aggressive, antisocial behavior with earlier onset of sexual activity and reproduction, greater sexual promiscuity and short-term mating, and lower-quality parental investment (e.g., harsh parenting, low involvement) reflects allocation of resources toward a fast strategy (reviewed in Del Giudice, 2018).

Two key dimensions of the environment that regulate the development of life history strategies are extrinsic morbidity–mortality (external sources of disability and death that are relatively insensitive to the adaptive decisions of the organism) and predictability of environmental change (Ellis et al., 2009). In stressful environments characterized by relatively high age-specific rates of morbidity and mortality and/or high unpredictability, fast life history strategies may maximize short-term gains (such as through risky and aggressive behaviors that leverage positions in status hierarchies and access to mates) and, through it, reduce the risk of disability or death prior to reproduction (e.g., Ellis et al., 2012; Yao, Långström, Temrin, & Walum, 2014). Consistent with this theoretical perspective, environmental cues indicating higher extrinsic morbidity–mortality and unpredictability generally promote faster strategies (e.g., Belsky et al., 2012; Doom et al., 2016; Simpson, Griskevicius, Kuo, Sung, & Collins, 2012).

Trade-offs incurred by faster strategies include reduced health, vitality, and longevity. A large body of research now indicates that the development of faster life history strategies comes at the cost of increasing allostatic load (for a review, see Ellis & Del Giudice, 2014). Indeed, both cross-sectional and longitudinal studies have shown that individuals who pursue faster life history strategies tend to display diminished levels of immunocompetence (Gassen et al., 2019; Hill, Boehm, & Prokosch, 2016) and suffer from more mental health problems, medical ailments (e.g., thyroid disease, high blood pressure or hypertension, ulcers), and physical health symptoms (e.g., sore throat or cough, dizziness) (Brumbach, Figueredo, & Ellis, 2009; Chua, Lukaszewski, Grant, & Sng, 2017; Figueredo, Vasquez, Brumbach, & Schneider, 2004; Gibbons et al., 2012; Mell, Safra, Algan, Baumard, & Chevallier, 2018; Sefcek & Figueredo, 2010).

It is important to note, however, that human research on the relations between early life stress, health outcomes, and life history strategy is correlational; causation may in fact be bidirectional. On the one hand, developmental exposures to stress may induce

faster life history strategies at a cost to mental and physical health (as per standard developmental life history models). This approach assumes that early stress carries predictive information about the future state of the environment (e.g., danger and consequent high mortality), and that development of a faster life history strategy in this context functions to match the individual to this expected harsh state, despite the costs (including potential mismatch if early environmental cues prove unreliable). This hypothesized developmental trajectory has been referred to as an "external predictive adaptive response" (e.g., Nettle et al., 2013).

On the other hand, early life stress may first cause damage to the soma (i.e., erode phenotypic condition in a manner that reduces health and longevity), and the damaged soma itself may function to induce a faster life history strategy (see Nettle et al., 2013; Rickard et al., 2014; Wells, 2012). In this view, developmental mechanisms respond to the individual's compromised internal state (e.g., allostatic load) and not to probabilistic cues about its future environment. This proposed developmental trajectory has been referred to as an "internal predictive adaptive response" (Nettle et al., 2013). The internal predictive adaptive response model assumes that internal somatic states are stable over individual lifetimes (i.e., that compromised somatic condition in childhood predicts compromised somatic condition in adulthood), thus reducing the possibility of mismatch when using childhood somatic condition as a basis for calibrating life history strategy (Frankenhuis, Nettle, & McNamara, 2018).

To date, the most compelling human evidence that both external and internal predictive adaptive responses occur simultaneously comes from a study of the British Birth Cohort (Waynforth, 2012). Childhood adversity (i.e., low parental occupational status, father absence) and chronic health conditions known to reduce life expectancy (excluding conditions that cause severe physical or mental disablement or disfigurement) were assessed in this study at age 10. Each of these variables independently predicted earlier timing of first reproduction. The effect of chronic health conditions on earlier age at reproduction concurs with a larger literature demonstrating elevated levels of risky sexual behavior (and substance use) among adolescents with chronic health conditions (e.g., Miauton, Narring, & Michaud, 2003; Nylander, Seidel, & Tindberg, 2014; Suris & Parera, 2005).

A surprising result from the British Birth Cohort study, which makes the larger pattern of results difficult to interpret, is that the indicators of adversity were simply uncorrelated with chronic health conditions ($rs < 0.02$). On the one hand, this suggests that chronic childhood illnesses, independent of demographic indicators of early adversity, can shift the individual toward earlier reproduction—an indicator of a faster life history strategy. The other studies cited above regarding adolescents with chronic health conditions (Miauton et al., 2003; Nylander et al., 2014; Suris & Parera, 2005) further support this general conclusion. This research literature provides support for the internal predictive adaptive response model. On the other hand, the absence of any relations in the British Birth Cohort between early external conditions and health, as has been recurrently found in other research (e.g., Adler, Boyce, Chesney, Folkman, & Syme, 1993; Chen, Matthews, & Boyce, 2002), suggests that either the demographic indicators of early adversity failed to adequately capture variation in childhood stress exposures, or there was a restriction of range. As such, Waynforth (2012) is not well-positioned to address questions regarding the direction of relations between early life stress, health, and life history strategy.

Another relevant body of theory and research centers on the "weathering hypothesis" (Geronimus, 1992), which proposes that socially disadvantaged groups (African American women in the original formulation) experience accelerated health deterioration as a result of the cumulative impact of repeated, interlocking experiences of adversity and marginalization. Geronimus (1996) demonstrated that among African American women, especially those of lower socioeconomic status, maternal age above 15–16 years was positively associated with bearing low birthweight and very low birthweight offspring, and that this effect was accounted for by deteriorating health. These data may be most consistent with the hypothesis that the effects of repeated exposure to stress on early reproduction are mediated by damage to the soma, rather than vice versa. This mediational hypothesis integrates the external and internal prediction models by targeting poor physical health as a mechanism through which developmental exposures to stress induce faster life history strategies.[1]

This mediational hypothesis was directly tested in a recent longitudinal study. Using data from the NICHD Study of Early Child Care and Youth Development, Hartman et al. (2017) assessed early exposures to adversity (based on a composite of socioeconomic adversity, family unpredictability, and harsh/insensitive parenting) over the first 5 years of life and general health over the first 12 years of life. The study employed two measures of life history-relevant traits: age of menarche (girls only) and a composite of number of sexual partners, substance use, and aggressive-antisocial behavior at age 15. Hartman et al. (2017) did not find statistically significant effects of general health on either of these life history strategy indicators (though better health was marginally associated with later age at menarche). As in much previous research (reviewed in Ellis & Del Giudice, 2019), early adversity was significantly associated with worse overall health, earlier age of menarche, and higher scores on the behavioral measure of fast life history-relevant traits. These findings are consistent with the external prediction model. Nonetheless, it is worth noting that Hartman et al. (2017) also included a measure of overall adolescent adjustment (i.e., better social skills and impulse control, less depression, loneliness, and aggression). Although it is unclear how this measure relates to life history strategy, they did find that the effect of early adversity on adolescent adjustment was partially mediated by general health. Major limitations of Hartman et al. (2017) included the sample (largely middle class) and the general health measure (single item: caregiver rating of global health). The null effects of general health on the life history strategy indicators may reflect a lack of variation across the full range of health (i.e., more serious health problems that are typically found in disadvantaged populations may have been underrepresented in the NICHD

study) and/or a failure of the general health measure to adequately capture deterioration of the individual's somatic state.

1.2 | The current study

The current study was designed to test the mediational hypothesis originally tested by Hartman et al. (2017): that physical health deterioration operates as an intervening mechanism through which developmental exposures to stress induce faster life history strategies. Whereas the external prediction model emphasizes direct effects, the internal prediction model specifies indirect effects. The current integrative approach tests for both direct and indirect effects in a structural equation model.

Our approach to testing the mediational hypothesis both paralleled and extended Hartman et al. (2017). We paralleled Hartman et al. in terms of sample size, use of a longitudinal research design that prospectively followed children from birth through age 16, and careful assessments of both early life adversity (described below) and life history-relevant traits in adolescence. The current life history strategy indicators converged with Hartman et al. in terms of assessing risky sexual behavior, aggressive-antisocial behavior, and delinquency (in peers). We extended Hartman et al. by addressing its two major limitations. First, we employed a high-adversity sample (Maternal Lifestyles Study; Lester et al., 2002). The overwhelming majority of our participants were African American, had prenatal substance exposure, and were born to unmarried parents living in poverty. Thus, consistent with the weathering hypothesis (discussed above), our participants were likely to have experienced multiple forms of early adversity and marginalization that are known to accelerate biological aging and undermine health (e.g., Goosby & Heidbrink, 2013). Second, the current study included detailed assessments of chronic and acute health problems. In total, the current study was well positioned to test for both external and internal predictive adaptive responses, and especially their integration within the proposed mediational hypothesis.

Consistent with both external and internal predictive adaptive response models, locally optimal life history strategies can be expected to vary as a function of at least two overarching factors. First, the costs and benefits of different strategies depend on the physical and social parameters of an organism's environment, especially extrinsic morbidity–mortality and unpredictability. In research in industrialized populations, extrinsic morbidity–mortality is often operationalized in terms of *socioeconomic adversity* (e.g., Belsky et al., 2012; Simpson et al., 2012) because of the relationship between poverty and higher levels of virtually all forms of morbidity and mortality (e.g., Adler et al., 1993; Chen et al., 2002). Cues to environmental unpredictability have typically been operationalized as stochastic changes in ecological and familial conditions (e.g., Belsky et al., 2012; Simpson et al., 2012). Recent research suggests that the most important aspect of environmental unpredictability in relation to adolescent development is number of *parental transitions* (Hartman, Sung, Simpson, Schlomer, & Belsky, 2018). Accordingly, in the current research, we operationalized extrinsic morbidity-mortality in terms of socioeconomic adversity and unpredictability as number of parental transitions (caregiver changes). We also assessed the internal condition of the mothers/primary caregivers in the study, based on their levels of depression and psychological distress. Diminished internal condition in parents may foster lower parental investment and less stable pairbonds, thus promoting faster life history strategies that result in greater exposure of the child to parental transitions (i.e., unpredictability). Finally, following Lester and Padbury (2009), we conceptualized prenatal substance exposure as an intrauterine stressor that, like socioeconomic adversity and parental instability, can be expected to regulate development toward faster life history strategies.

The other key factor in determining locally optimal life history strategies is the organism's internal condition and competitive abilities relative to other members of the population (e.g., age, body size, health, history of wins and losses in agonistic encounters; see Del Giudice & Ellis, 2016). As per the internal predictive adaptive response model, the current study focused on internal somatic condition. Following Waynforth (2012), we assessed physical illnesses and injuries in early adolescence as an index of morbidity, while excluding conditions that cause severe disablement or disfigurement (and could thus impede development of alternative life history strategies). In addition, to get an early baseline for physical condition, we assessed birthweight, and its close correlate gestational age, which are known to predict later health outcomes (e.g., Boulet, Schieve, & Boyle, 2011; Valdez, Athens, Thompson, Bradshaw, & Stern, 1994).

According to Bronfenbrenner's (Bronfenbrenner & Morris, 2006) bioecological theory and other related developmental frameworks (e.g., Belsky, 1984; Conger, Ge, Elder, Lorenz, & Simons, 1994; McLoyd, 1990), socioeconomic adversity, parental instability, and caregiver depression/psychological distress all influence more proximal family relationships and, through it, child development. Thus, we included a measure of adverse family functioning as a potential mediator of both external ecological factors and internal condition of the mother/primary caregiver.

In total, we tested a structural equation model in which (a) physical and psychosocial parameters of the child's environment were assessed in early childhood, (b) life history-relevant traits and behavior were assessed in adolescence, and (c) proposed mediators (family functioning and health) were assessed in middle childhood to preadolescence. Specifically, we tested whether prenatal substance exposure, caregiver psychological distress/depression, socioeconomic adversity, and caregiver instability in early childhood predict the development of faster life history strategies in adolescence (as indexed by more risky sexual and aggressive behavior). We hypothesized that these effects would operate both *directly* (as per external predictive adaptive response models) and *indirectly* through (a) adverse family functioning in middle childhood and (b) poor health in preadolescence (as per internal predictive adaptive response models). Finally, we tested whether indicators of physical and psychosocial stress in the child's very early environment (prenatal substance exposures, caregiver psychological distress/depression, socioeconomic adversity) were already linked to the child's somatic condition

TABLE 1 Descriptive Statistics for Study Variables (Prior to Transformations)

Observed variable	Scale range	Min	Max	Mean	*SD*	*N*
Sex[a]	0–1	0	1	0.48	0.50	1,388
Race[b]	0–1	0	1	0.77	0.42	1,388
Prenatal polysubstance exposure[c]	0–5	0	5	1.89	1.38	1,388
Gestational age (weeks)	21–42	21	42	36.25	4.03	1,385
Birthweight (grams)	519–4880	519	4,880	2,629.82	818.53	1,388
Caregiver psychological distress, 4m–5.5y	0–4	0	3	0.56	0.48	1,290
Caregiver depression, 4 m–5.5 y	0–63	0	45	7.46	6.38	1,297
Socioeconomic status[d], 1 y	8–66	8	66	27.82	9.94	1,364
Socioeconomic status[d], 2 y	8–66	8	66	27.72	9.68	1,372
Socioeconomic status[d], 3 y	8–66	8	66	27.82	9.51	1,375
Socioeconomic status[d], 4 y	8–66	10	66	27.93	9.42	1,377
Socioeconomic status[d], 5 y	8–66	11	66	28.08	9.31	1,378
Affective responsiveness[e], 7 & 9 y	1–4	1	3.17	1.97	0.43	1,109
Affective involvement[e], 7 & 9 y	1–4	1	3.64	2.15	0.43	1,109
General functioning[e], 7 & 9 y	1–4	1	3.17	1.84	0.36	1,109
Physical illness, 11 y	1–5	1	3.35	1.43	0.34	911
Physical illness, 12 y	1–5	1	2.98	1.42	0.33	933
Physical illness, 13 y	1–5	1	3.30	1.42	0.33	952
Conduct disorder symptoms (count), 14 y	0–27	0	14.50	2.59	2.47	911
Peer delinquency, 15 y	0–2	0	1.82	0.31	0.31	945
Adolescent risky sexual behaviors, 16 y	0–1	0	1	0.32	0.34	1,043

[a]0 = Male; 1 = Female.

[b]0 = Non-black/Other, 1 = Black/African American.

[c]Prenatal polysubstance exposure coded as a count of substances used during pregnancy.

[d]Original coding for Hollingshead Two-Factor Index of Social Position (ISP) is shown here. In the analyses, ISP was reverse-scored so that higher scores indicated greater socioeconomic adversity.

[e]Higher scores indicate lower levels of family functioning.

at birth (birthweight/gestational age), whether this indicator of somatic condition at birth predicated later somatic condition (poor health in preadolescence), and whether poor health in preadolescence mediated the effect of birthweight/gestational age on risky sexual and aggressive behavior in adolescence. Because the internal predictive adaptive response model conceptualizes internal somatic state as a unique determinant of life history strategy, the model contends that health deterioration will independently predict faster life history strategies (above and beyond any effects of external environmental conditions).

2 | METHOD

2.1 | Participants

The present study draws on the 1,388 participants (661 female) in the Maternal Lifestyle Study (MLS; Lester et al., 2001, 2002), a multisite longitudinal study of the effects of prenatal substance exposure on child development. Enrollment in the study began at 1 month; follow-up data collections continued through 16 years

of age. Participants were recruited from 4 US cities (Detroit, MI; Memphis, TN; Miami, FL; Providence, RI). The sample was ethnically and racially diverse (77% African Americans, 16% Caucasian, 6% Hispanic, and 1% other racial or ethnic backgrounds) and socioeconomically disadvantaged. See Lester et al. (2001, 2002) for detailed description of study recruitment, enrollment, maternal characteristics, and exclusion criteria. In brief, the families were selected based on cocaine or opiate use during pregnancy. The exposed group was formed based on the maternal report of cocaine or opiate use during pregnancy or gas chromatography-mass spectrometry confirmation of presumptive positive meconium screens for cocaine or opiate metabolites. For the families in the comparison group, mothers denied any use of cocaine or opiate during pregnancy and had a negative enzyme multiplied immunoassay meconium screen for cocaine and opiate metabolites. Youths in the substance-exposed and comparison (non-substance-exposed) groups were matched on race, sex, and gestational age within each study sites. The study was approved by the institutional review board at each study site, and written informed consent was obtained from caregivers of all participants prior to participation. Each site had a certificate of confidentiality from the National Institute on Drug Abuse.

2.2 | Measures

Basic descriptive data, including scale range, minimum and maximum scores, means, standard deviations, and *N* for all variables used in the analyses are shown in Table 1. To increase interpretability, Table 1 shows the raw parameters (prior to any data transformations). Reliability information for the latent variables used in the analyses is presented in the Results.

2.2.1 | Prenatal substance exposure

Child prenatal substance exposure to alcohol, cocaine, marijuana, opiate, and tobacco was identified based on maternal report of any substance exposure during pregnancy and gas chromatography–mass spectrometry confirmation of presumptive positive meconium screens for cocaine and opiate metabolites. Prenatal substance exposure was measured as a summative index ranging 0–5 (0 = no; 1 = yes) for use of cocaine, opiates, marijuana, alcohol, and tobacco during pregnancy based on maternal report/meconium screen of drug use. The large majority of children (81.3%) were exposed to at least one substance in utero.

2.2.2 | Birthweight/gestational age

Gestational age was based on the best obstetric estimate, derived from the last menstrual period and/or early sonography. Study nurses were trained to reliability on estimation of gestational age by the site study principal investigator. Birthweight and gestational age were highly correlated ($r = 0.84$) and thus were standardized and averaged together to form the composite measure of birthweight/gestational age.

2.2.3 | Caregiver psychological distress

Caregiver psychological distress was assessed at ages 4 months, 2.5 years, and 5.5 years using the mean of total psychological symptoms from the Brief Symptom Inventory (BSI; Derogatis, 1993; Derogatis & Spencer, 1993). The BSI is a 53-item self-report inventory designed to evaluate psychological stress symptoms during the past 7 days; it has high reliability and validity in adolescents and adults (Derogatis & Spencer, 1993). The scale yields a global severity index, which reflects both the number and severity of all items endorsed. To create one index of *caregiver psychological distress*, we standardized the total severity score for each time point and averaged them together. Higher scores indicated more caregiver psychological distress.

2.2.4 | Caregiver depression

Caregiver depression was assessed at ages 4 months, 2.5 years, 4 years, and 5.5 years using the Beck Depression Inventory (BDI; Beck, Steer, & Brown, 1996). The BDI is a 21-item, self-administered scale that evaluates key symptoms of depression such as mood, pessimism, sense of failure, and suicidal ideas. Past studies support the validity and other psychometrics of the BDI (e.g., Beck, Steer, & Garbing, 1988; Sanz & Vazquez, 1998). Participants reported on depressive symptoms during the past week. To create one index of *caregiver depression*, we standardized the total scores for each time point and averaged them together. Higher scores indicated more caregiver depression.

2.2.5 | Socioeconomic adversity (ages 1–5 years old)

Socioeconomic adversity was measured using Hollingshead Two-Factor Index of Social Position (ISP), based on the formula provided by Hollingshead (1975). For parental occupation, the scale ranges from 9 (e.g., higher executives) to 1 (unskilled employees). For parental education, the scale ranges from 7 (e.g., professional degree) to 1 (less than 7 years of school). If there was another contributing adult in the family, then ISP was calculated by summing the average of the primary caregiver and the contributing adult's weighted education and occupation. Following Hollingshead (1975), ISP was calculated based on the formula: (Occupation × 5) + (education × 3). As shown in Table 1, average ISP scores at each time point were 28, indicating a socioeconomically disadvantaged sample. In the analyses, the ISP scores for each time point were reverse scored so that higher values reflected greater socioeconomic adversity.

2.2.6 | Caregiver instability

Caregiver instability refers to history of primary caretaker changes and was calculated by counting the number of the times that the primary caretaker changed in child's life during the first 7 years of life. The child received a score of 1 for each year in which they experienced at least one change in their primary caregiver, resulting in a maximum score of 7.

2.2.7 | Adverse family environment

Adverse family enviornment was assessed at ages 7 and 9 years old using the Family Assessment Device (FAD; Epstein, Baldwin, & Bishop, 1983). The FAD, which was completed by the primary caregiver, is a self-report scale designed to measure family members' perceptions of family functioning. Three subscales were used in the present analyses: Affective Responsiveness (six items): family members' ability to respond with the appropriate affect over a range of stimuli; Affective Involvement (seven items): the extent to which the family, as a group, values and shows interest in each other's activities and concerns; and General Functioning (12 items): overall lack of family cohesion. To create single indices of Affective Responsiveness, Affective Involvement, and General Functioning, scale scores for each time point were standardized and averaged together. All three composite scales were scored so that higher values represented a more adverse family functioning (e.g., less family involvement).

2.2.8 | Early adolescent physical illness and injury (ages 11–13)

Youth reported on their physical health using the Child Health and Illness Profile-Adolescent Edition questionnaire (CHIP-AE; Starfield et al., 1993, 1995) at ages 11, 12, and 13 years. The CHIP-AE is a self-administered health status measure designed to provide a comprehensive description of the health of adolescents. The scale comprises of 6 domains and 20 subdomains. The Disorders domain was used in the present research; it assesses injuries, impairments, and disorders over the past 12 months. To operationalize *physical illness*, we standardized the scores for acute minor disorders (10 items; e.g., colds, tonsillitis, sprains) and recurrent disorders (11 items; e.g., ear infections, asthma, allergies) and averaged them together at each time point (ages 11–13). Higher scores indicated worse physical health (more illnesses and injuries).

2.2.9 | Conduct disorder symptoms

Conduct disorder symptoms were measured at 14 years of age using the Conduct Symptom Count subscale of the Diagnostic Interview Schedule for Children (DISC-IV; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000). The subscale included 26 items that assessed whether the target child engaged in deviant acts (yes/no) such as lying, shoplifting, physical assault, vandalism, and cruelty to animals during the past year. Higher scores indicated more conduct symptoms.

2.2.10 | Peer delinquency

Peer delinquency was measured at age 15 via adolescent self-report on the Things that Your Friends Have Done interview (TYFHD; Dishion, Patterson, Stoolmiller, & Skinner, 1991; Elliott, Huizinga, & Ageton, 1985). TYFHD is a self-report scale that measures to what extent the child's friends have engaged in delinquent activities within the past 12 months. TYFHD consists of 16 items, including physical aggression, threats, trespassing, theft, drug use, gang fights, and carrying a weapon. First the child reports whether their friends have engaged in the specific activity (No = 0); if yes, then the child is asked whether some (score = 1) or most (score = 2) of his/her friends have done that behavior. Items were averaged together to form a composite score, with higher scores indicating more delinquent behaviors by peers.

2.2.11 | Adolescent risky sexual behaviors

Adolescent risky sexual behaviors were assessed using the Adolescent Sexual Behavior Assessment (ASBA; Dolezal, Mellins, Brackis-Cott, & Meyer-Bahlburg, 2006) administered via audio computer-assisted self-interviewing (ACASI) technology. The ASBA is an unpublished interview schedule that was designed to assess various specific sexual practices and is appropriate for younger children. At age 16, adolescents reported (Yes = 1, No = 0) on lifetime engagement on sexual activities, including sexual contact, pregnancy, sexual intercourse, and unprotected intercourse. The *risky sexual behaviors* construct was created by averaging the 4 items together. Higher scores indicated more risky sexual behaviors.

3 | RESULTS

3.1 | Multivariate normality

Non-normal kurtosis is known to bias structural equation models (SEMs); thus, 5 analysis variables were transformed using the square root transformation: peer delinquency age 15, physical illness ages 11–13, and caregiver transitions by age 7. The resulting set of 21 analysis variables was approximately multivariate normal, with substantial variance and approximately normal skewness and kurtosis, and thus, highly appropriate for the linear *SEM* methods employed. Descriptive statistics for all analysis variables, including tests for normality, are reported in the Supplemental Materials (Appendix S1).

3.2 | Missing data

Nonmissing samples sizes are given in the final column of Table 1. There is a modest amount of missing data, with most variables missing < 8% of the maximum sample size (N = 1,388). The amount of missing data ranged from none (i.e., N = 1,388) for ethnicity, gender, prenatal drug exposure, caregiver transitions, and birthweight, to 34% (i.e., N = 911) for physical illness at age 11 and conduct disorder symptoms at age 14. Of the original 1,388 families, 1,043 youth participated in the final data collection at age 16; 345 did not participate. We analyzed whether there were differences in prenatal substance exposure, race, or socioeconomic status at Year 1 among youth with and without missing data at age 16. We found no significant differences in probability of missingness by prenatal substance exposure (p = .27), race (p = .12), or socioeconomic status (p = .70). Full information maximum likelihood (FIML) estimation was used to account for missing data in all model estimation and inference (Bollen, 1989; Enders & Bandalos, 2001). FIML methods have been shown to have optimal properties across a wide range of realistic scenarios (e.g., "missing at random") relative to conventional missing data methods (e.g., listwise deletion).

3.3 | Bivariate correlations

Correlations between model indicator variables are displayed in Table 2; intercorrelations for confirmatory factor analysis (CFA) indicators may be seen immediately adjacent to the diagonal. Notable patterns include extremely high correlations among the five socioeconomic adversity measures (ages 1–5: $r \geq 0.9$). There were moderate correlations between the three risk behavior indictors (peer delinquency, conduct problems, and sexual risk behavior: $0.31 \leq r \leq 0.38$), as well as large correlations for the three indicators of adverse family functioning (responsiveness, involvement, and general functioning: $0.69 \leq r \leq 0.79$). Next, physical illness is moderately-strongly correlated across ages 11–13 ($0.44 \leq r \leq 0.57$). Finally, the correlation between the two caregiver psychological distress/depression indicators (caregiver depression and distress) is large (r = 0.76). To support rigor and reproducibility, we provide the summary statistics (and correlation matrix) of the final, transformed analysis variables in the Supplemental Materials (Appendices 1 and 2) which may be used to replicate the full *SEM* analysis.

3.4 | Structural equation modeling

We employ the conventional linear (i.e., continuous outcome) structural equation model (*SEM*) approach (Bollen, 1989). *SEM* is appropriate for the current problem, which includes latent constructs and a multiple outcome system of equations. Additionally, *SEM* is preferred for its ability to formally test individual parameters, indirect (mediating) effects, and omnibus model fit (Bollen, 1989, 2002; Bollen & Pearl, 2013; Muthén & Muthén, 2007). We used a systematic model building approach, beginning with measurement modeling and separate examination of outcome equations, to minimize the risk of model misspecification (Bollen, 1989; Bollen & Curran, 2005). Finally, we used standard algebraic path analysis methods (i.e., Stata post-estimation command *estat teffects*) to calculate total, indirect, and direct effects for the *SEM*. This method calculates these estimates for all model-implied relationships, with standard errors calculated using the delta method (Bollen, 1989; Sobel, 1987). All effect sizes are standardized and, thus, interpreted as the expected *SD* change in the outcome, given one *SD* change in the predictor. Because caregiver transitions had no direct or indirect effects on any of the outcome variable, it was dropped from the *SEM* models.

Figure 1 shows CFA measurement models for all 5 of the latent constructs (A-to-E) considered in this analysis. Specifically, we derived latent measures of (A) socioeconomic adversity (higher scores indicate lower SES), (B) caregiver psychological distress/depression, (C) adverse family functioning, (D) physical illness, and (E) risky/aggressive behavior. As denoted by "$\sigma^2 = 1$" in each panel of Figure 2, all latent variable variances were specified equal to 1, for identification purposes, to allow estimation of all factor loadings. Loadings are given from individual CFA measurement models, but for all 5 constructs, the loadings were very comparable in full *SEM* (see Supplemental Materials, Appendix S3). The partial exception to this is construct B (caregiver distress/depression), which, as it has only two indicators, is not identified without fixing a factor loading (and the latent variable variance) (Bollen, 1989). In the full *SEM*, however, there are additional paths from construct B, allowing estimation of both factor loading paths.

As shown in Figure 1 and Table 2 (as discussed above), the intercorrelations between construct indicators was high-to-acceptable in all cases. For constructs B-to-E, tests of omnibus model fit are not available due to perfect identification. Despite its methodological limitations (Sijtsma, 2009), we present the Cronbach's α values for the five constructs to provide continuity with previous research. The alphas range from α = 0.99 (socioeconomic adversity), to α = 0.89 (adverse family functioning), α = 0.86 (caregiver distress/depression), α = 0.74 (physical illness), and α = 0.60 (adolescent risky/aggressive behavior), which is good-to-acceptable fit for all constructs, given that Cronbach's α is conservative for constructs with few indictors (Cortina, 1993). Additionally, as described below, the full *SEM*, encompassing all measurement models, demonstrates excellent omnibus fit across all fit indices, implying good fit for each of the individual measurement models.

The full *SEM* with measurement models and structural equations is presented, in simplified form, in Figure 2 (see Supplemental Materials, Appendix S3, for full model results). On the left side of the figure are 4 exogenous predictors: socioeconomic adversity, caregiver distress/depression, prenatal substance exposure, and birthweight/gestational age. Moving right, the figure describes a series of 3 structural equations, one for each of the two intermediate outcomes—(a) adverse family environment and (b) physical illness—and

TABLE 2 Pairwise Pearson correlation matrix for all analysis variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
1. Sex	1																				
2. Race	0.08	1																			
3. Prenatal drug use	0.06	−0.01	1																		
4. Gestational age	−0.06	−0.13	0.02	1																	
5. Birthweight	−0.14	−0.17	−0.05	0.84	1																
6. Caregiver distress, ≤5.5 y	0.02	−0.02	0.04	0.07	0.05	1															
7. Caregiver depression, ≤5.5 y	0.02	0.01	−0.01	0.07	0.04	0.76	1														
8. SES adversity, 1 y	0.00	0.23	0.16	−0.04	−0.07	0.14	0.14	1													
9. SES adversity, 2 y	0.00	0.24	0.16	−0.04	−0.08	0.17	0.17	0.96	1												
10. SES adversity, 3 y	0.01	0.24	0.17	−0.05	−0.09	0.18	0.18	0.94	0.98	1											
11. SES adversity, 4 y	0.01	0.24	0.17	−0.04	−0.08	0.19	0.19	0.92	0.96	0.99	1										
12. SES adversity,5 y	0.01	0.24	0.17	−0.05	−0.08	0.19	0.19	0.90	0.95	0.98	0.99	1									
13. Responsiveness, 7–9 y	0.02	0.24	0.02	0.01	0.01	0.13	0.16	0.21	0.23	0.24	0.25	0.26	1								
14. Involvement, 7–9 y	0.02	0.11	0.09	0.05	0.05	0.24	0.27	0.20	0.22	0.23	0.24	0.25	0.69	1							
15. General functioning, 7–9 y	0.04	0.13	0.07	0.03	0.03	0.18	0.22	0.18	0.20	0.22	0.22	0.22	0.79	0.74	1						
16. Physical illness, 11 y (sqrt)	−0.02	−0.11	0.05	0.03	0.03	0.08	0.05	−0.02	−0.03	−0.02	−0.01	0.00	−0.09	−0.01	−0.03	1					
17. Physical illness, 12 y (sqrt)	−0.02	−0.08	−0.02	0.01	0.03	0.11	0.07	−0.05	−0.04	−0.04	−0.03	−0.02	−0.03	0.04	0.03	0.48	1				
18. Physical illness, 13 y (sqrt)	0.01	−0.12	−0.02	−0.03	−0.03	0.12	0.08	−0.06	−0.05	−0.06	−0.06	−0.05	−0.03	0.03	0.02	0.44	0.57	1			
19. Conduct Symptoms, 14 y	−0.17	0.05	0.18	0.11	0.08	0.12	0.11	0.06	0.08	0.08	0.08	0.09	0.06	0.06	0.09	0.14	0.20	0.16	1		

(Continues)

TABLE 2 (Continued)

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
20. Delinquency, 15 y (sqrt)	0.02	0.01	0.08	0.02	0.03	0.04	0.03	0.05	0.06	0.06	0.07	0.06	−0.03	0.04	0.03	0.13	0.13	0.18	0.33	1	
21. Sexual risk, 16 y	−0.19	0.05	0.18	0.08	0.06	0.03	0.04	0.11	0.12	0.12	0.12	0.12	0.08	0.12	0.12	0.09	0.13	0.11	0.38	0.31	1

Note: *N*s range from 911 to 1,388. SES = Socioeconomic status. Sex: 0 = Male; 1 = Female. Race: 0 = Non-black/Other, 1 = Black/African American.

one for the primary distal outcome, (c) adolescent risky/aggressive behavior. The diagram is arranged with measures from earlier developmental periods on the left and later developmental periods on the right. For clarity, the figure omits variances, control variables (i.e., ethnicity and gender), and most covariances (see Supplemental Materials, Appendix S2, for full model results). All structural paths are in the expected direction and are statistically significant at $p < .05$ ($p < .001$ for 5 of 8 structural paths). Omnibus model fit for the full *SEM* visualized in Figure 2 was excellent across all assessed fit indices (e.g., RMSEA = 0.035; CFI = 0.97; TLI = 0.96).

A primary objective of this study was to compare the effects of external environmental and internal health predictors on the distal outcome of adolescent risky/aggressive behavior. Toward this end, we calculated total, indirect, and direct effects for all model predictors on the distal outcome of risky/aggressive behavior (Table 3). The direct effects, as depicted in Figure 2 and the right columns of Table 3, were relevant to assessing both the internal and external predictive adaptive response models. The largest standardized effect in the model was preadolescent physical illness → adolescent risky/aggressive behavior ($\beta = 0.35$, $p < .001$), which was substantially larger than second largest effect in the model, prenatal substance exposure → risky/aggressive behavior ($\beta = 0.26$, $p < .001$). Although the large effect of physical illness supported the internal predictive adaptive response model, this effect may have been inflated by method variance. Specifically, the effect of physical illness on risky/aggressive behavior constituted a within-person association (single rater) over a relatively short developmental time period, whereas the smaller effects of the external environmental constructs on risky/aggressive behavior represented across-person associations (different raters) over longer developmental periods. Finally, contrary to the internal predictive adaptive response model, the cross-rater association between birthweight/gestational age and risky/aggressive behavior was positive ($\beta = 0.11$, $p < .01$), indicating that larger birthweight/older gestational age (a positive indicator of health) was associated with more risky/aggressive behavior. Most of the structural paths depicted in the Figure 2 were in the $\beta = 0.1$–0.25 range, indicating small/moderate effect sizes.

Consistent with the external predictive adaptive response model, there were significant direct effects for prenatal substance exposure, socioeconomic adversity, and adverse family functioning on risky/aggressive behavior (independent of physical illness and birthweight/gestational age). As per developmental life history models, there were unique, statistically significant effects for multiple indicators of early environmental stress—socioeconomic adversity and prenatal substance use, as well as adverse family environment in mid-childhood, on risky/aggressive behavior in adolescence. As shown in the left section of Table 3, of the external predictors, prenatal substance exposure had the largest total effect on risky/aggressive behavior ($\beta = 0.26$, $p < .001$). Each of the other external environmental constructs (i.e., adverse family functioning, caregiver distress/depression, and socioeconomic adversity) had significant total effects on risky/aggressive behavior, but these effects were smaller in magnitude ($0.07 \leq \beta \leq 0.11$).

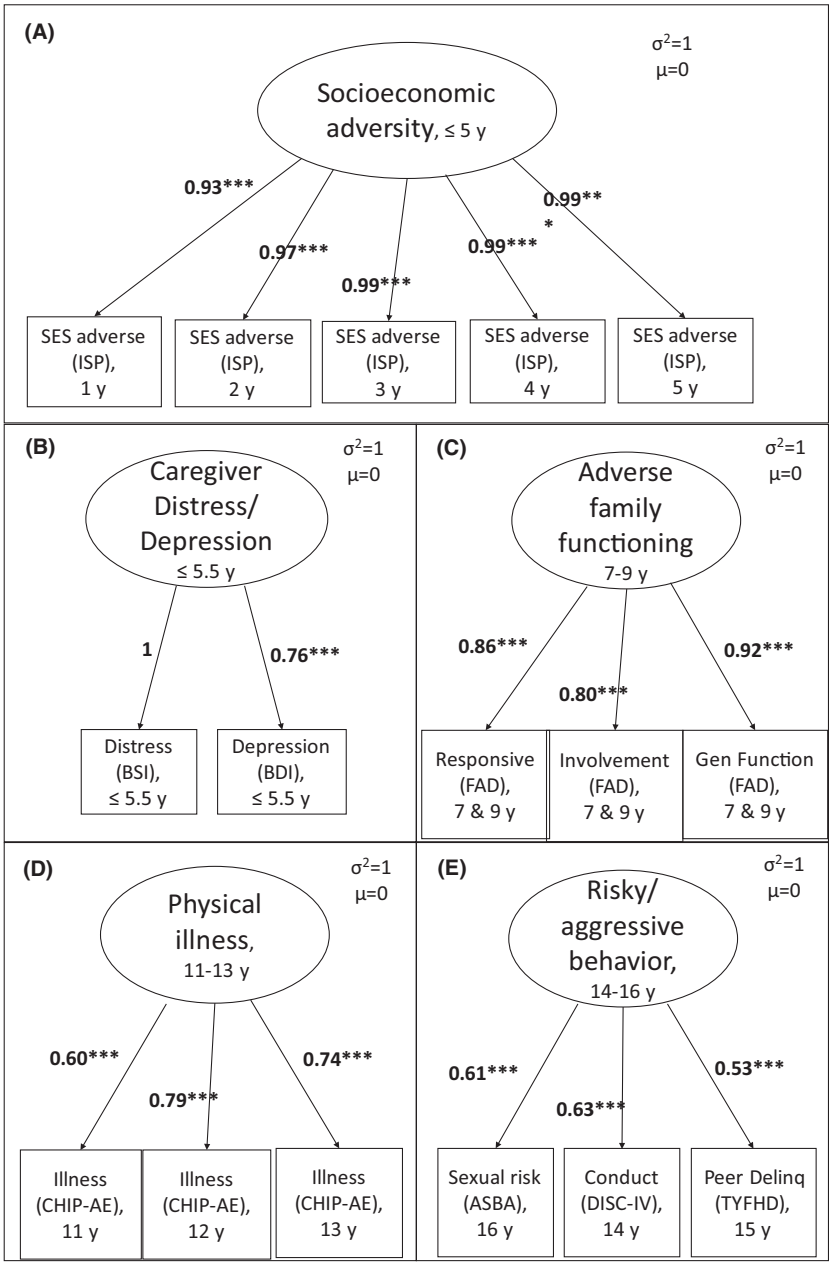

FIGURE 1 Measurement models for five primary constructs. Loadings are given from CFA models, but the loadings were very comparable in full *SEM* (see Supplemental Materials, Appendix S2). $^{*}p < .05$, $^{**}p < .01$, $^{***}p < .001$

As shown in the middle section of Table 3, two of the four exogenous predictors (i.e., caregiver distress/depression and socioeconomic adversity) had significant indirect effects on risky/aggressive behavior, acting through more proximate mediators. Specifically, early life caregiver distress/depression (≤age 5.5) exhibited the largest indirect effect ($\beta = 0.07$, $p < .001$) on risky/aggressive behavior in adolescence (ages 14–16); this effect was significantly mediated by both adverse family functioning at ages 7–9 ($\beta = 0.02$, $p < .05$) and physical illness at ages 11–13 ($\beta = 0.05$, $p < .01$). Early life socioeconomic adversity (ages 1–5) exhibited a smaller, indirect effect on risky/aggressive behavior, which was significantly mediated by adverse family functioning at ages 7–9 ($\beta = 0.02$, $p < .05$). Prenatal substance exposure and birthweight/gestational age did not show evidence of indirect effects. The statistically significant indirect effect of caregiver distress/depression on risky/aggressive behavior through physical illness is consistent with our central hypothesis—that poor physical health operates, in part, as an intervening mechanism through which developmental exposures to stress induce faster life history strategies.

4 | DISCUSSION

The goal of the current research was to test an integrative model of stress, health, and development, pulling together core assumptions of both internal and external predictive adaptive response models. This integrative approach led us to evaluate the hypothesis that that poor physical health operates as an intervening mechanism through which developmental exposures to stress induce faster life history strategies.

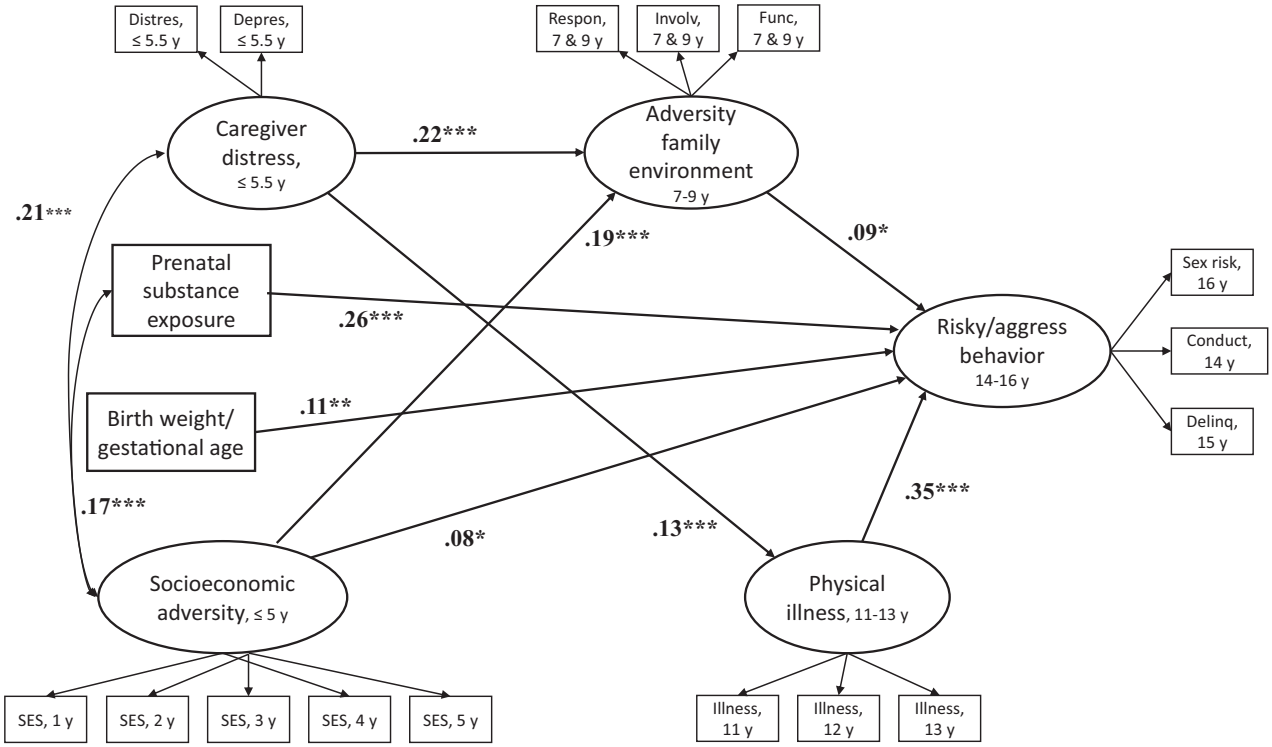

FIGURE 2 Simplified structural equation model path diagram with standardized coefficients. Omits variances and control variables. See Figure 1 for measurement model path loadings. SES: Socioeconomic Status. $^{*}p < .05$, $^{**}p < .01$, $^{***}p < .001$

TABLE 3 Indirect, direct and total effects of predictors on adolescent risky/aggressive behavior

	Total effects			Indirect effects			Direct effects		
Predictor	**β**	**t-stat**	***p*-val**	**β**	**t-stat**	***p*-val**	**β**	**t-stat**	***p*-val**
Adverse family environment (latent)	0.09*	2.14	.03				0.09*	2.14	.03
Physical illness (latent)	0.35***	6.12	<.001				0.35***	6.12	<.001
Prenatal polysubstance exposure (observed)	0.26***	6.31	<.001				0.26***	6.31	<.001
Birthweight/gestational age (observed)	0.11**	2.91	.004				0.11**	2.91	.004
Caregiver distress (latent)	0.07***	3.54	<.001	0.07***	3.54	<.001			
Socioeconomic adversity (latent)	0.10*	2.50	.01	0.02*	2.01	.04	0.08*	2.02	.04

$^{*}p < .05$,
$^{**}p < .01$,
$^{***}p < .001$.

First, consistent with the internal predictive adaptive response model, we found that preadolescents who reported more acute minor physical disorders (e.g., colds, sprains) and recurrent physical disorders (e.g., asthma, allergies) subsequently engaged in more risky sexual and aggressive behavior in adolescence (e.g., early sexual debut, criminal behavior, violence, peer delinquency). A large body of previous research employing cross-sectional designs has established that (a) serious, chronic health conditions are concurrently associated with more health-risking behaviors in adolescents (e.g., Miauton et al., 2003; Nylander et al., 2014; Suris & Parera, 2005) and (b) adults who display faster life history strategies tend to have more mental health problems, medical ailments, and physical health symptoms (e.g., Chua et al., 2017; Figueredo et al., 2004; Mell et al., 2018; Sefcek & Figueredo, 2010). The current results are consistent with this past cross-sectional work.

At the same time, few studies have prospectively examined whether normative variation in childhood physical health predicts later variation in life history-relevant traits—a key assumption of

the internal predictive adaptive response model, which assumes that deteriorating phenotypic condition promotes faster life history strategies. Whereas Waynforth (2012) found that chronic health conditions at age 10 predicted earlier age at first reproduction, Hartman et al. (2017) did not find significant effects of general health over the first 12 years of life on any of their indicators of life history strategy in adolescence (including a measure of risky/aggressive behavior that was comparable to that used in the current study). As noted above, the current research extends Hartman et al. (2017) by employing a more disadvantaged sample (with substantially greater adversity exposure and, presumably, more physical health problems) and by conducting a more detailed assessment of physical health. We presume that these differences in research design, which are particularly relevant to testing for internal predictive adaptive responses, accounts for why we found robust effects of earlier physical illness on later risky and aggressive behavior.

One anomalous finding was that individuals who had higher birthweight/older gestational age, which is known to predict positive future health outcomes (e.g., Boulet et al., 2011; Valdez et al., 1994), displayed higher levels of risky sexual and aggressive behavior. Further, we did not find support for the hypothesis, drawn from the internal predictive adaptive response model, that adversity exposures (e.g., prenatal substance exposures) have immediate negative effects on internal somatic condition (as indicated by birthweight/gestational age) (Rickard et al., 2014). Finally, we did not find an effect of our measure of early somatic condition (birthweight/gestational age) on later somatic condition (physical illness), which is another key prediction of the internal predictive adaptive response model. As birthweight/gestational age was not associated with any study variables other than risky/aggressive behavior, it is difficult to interpret its meaning. Thus, we are not prepared to offer any substantive interpretation of these anomalous finding.

We also found support for external predictive adaptive response models. There were unique direct and indirect effects of prenatal substance use, socioeconomic adversity, caregiver instability, caregiver distress/depression, and adverse family functioning on risky/aggressive behavior in adolescence, with all of these effects occurring independently of physical health. These findings concur with a large body of past research linking early life stress to more risky and aggressive behavior (e.g., Belsky et al., 2012; Doom et al., 2016; Kerig, 2019).

There was also some support for the mediational hypothesis. Caregiver distress/depression had indirect effects on risky/aggressive behavior in adolescence, which operated through both adverse family functioning (e.g., lower family cohesion) and more physical health problems. This latter indirect effect supported our central integrative hypothesis: that poor or deteriorating somatic condition operates as an intervening mechanism through which early life stress regulates development toward faster life history strategies. To the best of our knowledge, this is the first successful test of this hypothesis using prospective, longitudinal data. It is important to note, however, that only our measure of caregiver distress/depression, and not our other measures of early adversity, predicted preadolescent physical health; thus, the integrative hypothesis was not supported in relation to other early adversity measures.

These findings have implications for theory and research on stress, development, and health. In the developmental and health sciences, there is a growing consensus that substantial stress exposures early in life increase the probability of negative cognitive and behavioral outcomes (e.g., insecure attachments, mistrustful internal working models, opportunistic interpersonal orientations, excessive vigilance to threat, substance use, oppositional–aggressive behavior), and that that these outcomes in turn increase the probability of morbidity and premature mortality as the organism ages (e.g., Aafjes–van Doorn, Kamsteeg, & Silberschatz, 2019; Miller, Chen, & Parker, 2011; Wideman et al., 2016). The internal predictive adaptive response model suggests that causation may be bidirectional, or even fundamentally reversed. Many aspects of a faster life history strategy, including more risky and aggressive behavior and early age of sexual development and reproduction, may be driven by earlier somatic deterioration. It is now well-established that various forms of early life stress are associated with accelerated biological aging (e.g., Belsky, 2019; Colich, Rosen, Williams, & McLaughlin, 2020). This early health deterioration may be the horse that pulls the cart of faster life history strategies, as per the integrative hypothesis guiding the current research. The current findings thus highlight childhood health as an important early intervention target.

4.1 | Strengths, limitations, and future directions

Strengths of the current research include use of a prospective, longitudinal design; use of an appropriate sample for testing the internal and external predictive adaptive response models (in terms of substantial adversity exposures and variation in physical health problems); sample attrition that met the assumption of "missing completely at random"; use of multiple data sources to assess key constructs; and use of *SEM* with latent variables to reduce measurement error. A key limitation of the current research was the self-reported health measurement. Future research could benefit from clinical and biological assessments of health (e.g., telomere-length and erosion, epigenetic clocks, increased inflammatory tone). The goal would be to obtain increasingly precise measures of physical deterioration of the soma. Another potential limitation of the study was a restriction of range on socioeconomic adversity, given our socioeconomically disadvantaged sample. This restriction may have limited our power to detect direct and indirect effects of socioeconomic adversity on target outcomes.

It is also important to note that the current research was based on descriptive, longitudinal data. Although both internal and external predictive adaptive response models focus on the causal effects of early exposures to psychosocial stress, our data cannot directly test for causation because they are not experimental. Unmeasured third variables, such as allelic variations, could explain the observed correlations. Furthermore, stability in lifecourse adversity could potentially explain the outcomes (i.e., life history strategies could be

programmed by greater cumulative stress exposures rather than early stress exposures per se, as earlier exposure statistically translates into more exposure). Finally, associations we documented between early adversity exposures and development of life history-relevant traits could be confounded by unmeasured current adversity exposures.

5 | CONCLUSION

In conclusion, conceptualizing stress, health, and development from an evolutionary-developmental perspective has generated both external and internal predicative adaptive response models. The current research suggests the utility of integrating these two approaches, to move us toward a fuller understanding of the processes underlying relations between early life exposures to adverse conditions, physical illness, and life history strategies. Such strategies appear to be calibrated by both external and internal cues, with compromised internal state potentially operating as a key signal that the individual employs to adaptively regulate development toward faster strategies.

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ENDNOTE

[1] Although this mediational hypothesis is advanced by Rickard et al. (2014) as an auxiliary prediction of the internal prediction model, this hypothesis does not define the internal prediction model. Rickard et al. state that applications of the internal prediction model to life history outcomes need not invoke psychosocial stress. They cite Waynforth (2012) as providing unique support for the internal prediction model by showing that childhood illness, even though uncorrelated with childhood psychosocial stress, still predicted earlier age at reproduction (i.e., no mediation). In addition, the external prediction model (e.g., Del Giudice, Ellis, & Shirtcliff, 2011) conceptualizes autonomic, neuroendocrine, metabolic, and immune system factors as intervening mechanisms in the relation between early adversity and development of life history strategies. In the external prediction model, however, these physiological mediators may or may not index damage to the soma. In total, the current integrative mediational hypothesis is consistent with both the internal and external prediction models, but it is not integral to either model.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

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