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"Toxic" Schools? How School Exposures During Adolescence Influence Trajectories of Health Through Young Adulthood

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Article

"Toxic" schools? How school exposures during adolescence influence trajectories of health through young adulthood

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ABSTRACT

A large body of research identifies the critical role of early-life social contexts such as neighborhoods and households in shaping life course trajectories of health. Less is known about whether and how school characteristics affect individual health and contribute to population health inequality. However, recent scholarship argues that some school environments are so stressful due to high levels of violence, disorder, and poverty that they may be "toxic" to student health, but this hypothesis has not been tested using population data. Integrating insights from the life course perspective and stress process model, we use rich longitudinal data from the National Longitudinal Study of Adolescent to Adult Health (n = 11,382), diverse markers of physiological functioning and psychological well-being, and multilevel regression models to examine whether and how school characteristics shape trajectories of physiological dysregulation and depressive risk from adolescence through early adulthood. Findings reveal that, across multiple measures of physiological functioning and psychological well-being, the social and structural characteristics of schools play an essential role in shaping health risk from adolescence through young adulthood—long after students left school. In particular, indicators of school-level violence and perceptions of safety and school social disconnectedness had especially strong associations with health risk in both the short- and long-term. School socioeconomic composition was also strongly associated with physiological dysregulation in young adulthood, net of individual and neighborhood socioeconomic exposures. Together, findings from this study suggest that school environments can serve as early-life stressors in the lives of young people that unequally shape health trajectories and contribute to broader patterns of health inequality.

Introduction

Given striking levels of population health inequality, a large and growing body of research assesses the mechanisms underlying health disparities. Inequities in population health, including socioeconomic and racial health inequities, stem from group differences in social and economic conditions, including differential contextual exposures (Link & Phelan, 1995; Phelan & Link, 2015). In the U.S., unequal contextual exposures are rooted in historical legacies of racism—including residential redlining and school segregation—as well present day discrimination and inequalities in the housing, lending, and labor markets (Massey & Denton, 1993; Oliver & Shapiro, 1997; Orfield & Lee, 2005; Pager, 2007; Pager & Shepard, 2008). These processes have created unequal patterns of resources and risks across neighborhoods and communities, as well as unequal schools whose inequalities largely parallel those of local neighborhood contexts.

A growing number of studies examine how school contexts affect student health and contribute to population health inequality (Bernell, Mijanovich, & Weitzman, 2009; Goosby & Walsemann, 2012; Walsemann, Bell, & Goosby, 2011; Walsemann, Bell, & Maitra, 2011; Walsemann, Gee, & Geronimus, 2009). In *Toxic Schools,* Bowen Paulle (2013) makes a particularly powerful case for the link between schools and health, arguing that some school environments can be so stressful for students that they can damage their physical and mental health enough to be "toxic" to young people. Drawing on his experience teaching in high poverty schools in the South Bronx and Amsterdam, Paulle describes in detail the chaotic, stressful, and sometimes dangerous environments inside these schools. He asserts, "… the anxiety related to

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stress and episodic outbursts of brutal violence was always both 'out there,' in the educational settings and 'in there,' beneath the flesh of the exposed" (2013:100).

Paulle is not the first to describe scenes of threatening, frightening, violent, or even deadly conditions inside some of America's schools (see Devine, 1996; Fullilove et al., 2003, pp. 198–246; Hagan, Hirschfield, & Shedd, 2003, pp. 163–197; Kozol, 1991). However, his focus on how schools produce physical and emotional stress raises underexplored questions about the extent and degree of disorder, violence, and stress in American schools and how these factors shape health. Since young people spend many of their waking hours at school (Allard, 2008), and because early-life exposures play a particularly critical role in shaping life course trajectories of health (Ben-Shlomo & Kuh, 2002), examining the contextual influences of schools on individual health and population health inequality may provide new insights into social forces leading to the emergence and divergence of health disparities, particularly early in the life course.

Using nationally-representative, longitudinal data from the National Longitudinal Study of Adolescent to Adult Health (Add Health), we examine whether and how school characteristics shape trajectories of health from adolescence through early adulthood. Integrating insights from the life course perspective and stress process model and merging school-, family-, and individual-data, this study has four overarching objectives. First, building upon the work of scholars who have connected school characteristics to health (Bernell et al., 2009; Goosby & Walsemann, 2012; Walsemann, Bell, & Goosby, 2011; Walsemann, Bell, & Maitra, 2011; Walsemann et al., 2009), we assess which characteristics of schools are particularly "toxic" for student health. We pay particular attention to dimensions of toxicity described by Paulle (2013), including school-level measures of violence, perceptions of safety, disorder, and social disconnectedness. While Paulle's (2013) observations of the schools are illustrative, no study to date has theorized an operational definition of school toxicity using a nationally representative sample of schools.

Second, we examine the relationship between school toxicity and sociodemographic features of schools traditionally connected to inequality. Institutional policies and practices, both historical and more contemporary, have concentrated poor, Black, and Latino families into communities with fewer resources and greater risks (Massey & Denton, 1993; Wilson, 1996). Because public schools draw their populations from local neighborhoods, school toxicity is therefore also most likely to be found in schools with high percentages of Black and Latino students, as well as students of low socioeconomic status (SES) backgrounds. Studies of the effects of attending high poverty, high minority schools find that students fare worse academically as a result of attending those schools (Logan, Minca, & Adar, 2012; Reardon, 2016; Rumberger & Palardy, 2006), but students who attend such schools may also have worse health outcomes due to the stress of these environments. School toxicity is not a natural or inevitable phenomenon, but results from policies and institutional practices that systematically segregate and disinvest in schools that serve poor students and students of color. In this way, exposures associated with toxicity, which stem from discriminatory policies and practices, may contribute to population-level racial and socioeconomic health inequities.

Third, we document schools' impact on a diverse range of outcomes derived from multiple psychophysiological systems. Much of the extant work on contextual health effects uses self-rated health as the outcome of interest. However, people's perceptions of their own health can be inaccurate, especially if they become conditioned to long-term discomfort or stress. We therefore analyze markers of physiological and psychological well-being to elucidate how school conditions may affect health, even when students may not perceive or report those impacts.

Finally, while previous work on the links between schools and health generally relies on data from child and adolescent samples, we assess whether school exposures relate to individual trajectories of health risk in both the short- and long-term. Childhood and adolescence are sensitive periods for health, when exposure to stressful environments can have long-lasting and even irreversible impacts on adult health and well-being (Fuhrmann, Knoll, & Blakemore, 2015; Sawyer et al., 2012). In this way, the health impacts of school exposures may not only be present in childhood and adolescence but could persist well into young adulthood. By examining how school characteristics shape individual health trajectories from adolescence through adulthood, we provide new insights into the role of early-life social exposures in shaping young adult health.

Findings from this study reveal that schools matter for markers of physiological dysregulation and depressive risk. The social and structural characteristics of schools played an essential role in shaping physiological and psychological well-being from adolescence through young adulthood. Importantly, some aspects of school "toxicity" maintained associations with health net of potential confounders, such as neighborhood socioeconomic conditions and school demographic characteristics, and continued to shape health risk long after students left school. Our findings therefore suggest that school environments can serve as early-life stressors that can unequally shape health trajectories from adolescence through adulthood. As such, this study provides new insights into the role of school policies, practices, and environments in contributing to the early-life emergence and divergence of population health inequality.

Background

What makes schools "toxic?"

Paulle's (2013) cross-national comparative ethnography offers a compelling argument for the role of schools in shaping young people's stress exposure and well-being. Stress, violence, and disorder characterize the schools Paulle (2013) describes in *Toxic Schools*. Between incidents of extreme violence and the constant threat of such violence, a perpetual state of stress and fear was present among students and teachers in the schools Paulle observed. Other ethnographic studies of urban American schools describe similar scenarios. For example, in a study of a Chicago high school, Flores-Gonzalez (2002:60) reports that students complained about being on edge at school because of a "charged" atmosphere: "They are always looking out of the corners of their eyes to anticipate trouble, and feel uneasy and sometimes outright scared. Fear is ever-present because trouble lurks behind every corner, and students have to be prepared to defend themselves."

How students manage the stress triggered by fear and violence undoubtedly affects how much of an impact it has on their health. While teachers and other adults at school may be a source of emotional support, they, too, are affected by the environment of their school. At the schools Paulle (2013) observed, teachers were not always available as a source of support, because they too often burned out from the physical and emotional toil of managing their own fear and stress. As a result, teacher turnover and institutional disorder was high, and students could not effectively develop the kind of relationships with adults that they needed to cope with and reduce their stress (Paulle, 2013).

Environments marked by threats of violence and fear can be easily described as stressful, and insights from the stress process model (Pearlin, Menaghan, Lieberman, & Mullan, 1981) tell us that the students Paulle observed—those facing immense stress without proper social supports—may be most vulnerable to the health-harming consequences of stress. Still, while research documents the health consequences of prolonged stress exposure (Cohen et al., 2012; McEwen & Stellar, 1993; Thoits, 2010), less is known about the characteristics of schools that might be particularly stressful and "toxic" to young people. One goal of this research is to theorize an operational definition of school toxicity to inform future research on school effects, stress, and health. Furthermore, although disorder and the threat of violence can induce chronic stress in students (Fowler, Tompsett, Brascizewsky, Jacques-Tiura, & Baltes, 2009; Guerra, Huesmann, & Spindler, 2003;

Heissel, Sharkey, Torrats-Espinosa, Grant, & Adams, 2017; Kirk & Hardy, 2014), it remains largely untested whether these characteristics actually harm physical and mental health.

Hypothesis 1. School "toxicity" is positively associated with physiological dysregulation and depressive risk.

Are high-poverty schools more likely to be "toxic?"

Importantly, the stress process model holds that social stratification patterns stress exposure in the population, with structurally oppressed and disadvantaged groups having higher levels of stress than more advantaged groups (Pearlin, 1989). Given the unequal patterning of school resources in the U.S., poor students and students of color may be more likely to be exposed to stressful school environments than higher SES White students. Although Paulle (2013) suggests that, in the U.S., "toxic" schools are largely composed of poor students, few studies examine how school poverty might affect health through stress-related pathways. Thus, little is known about why school poverty could produce toxic school environments for youth. However, a wide body of literature examines associations between neighborhood poverty and health (Aneshenesel & Sucoff, 1996; Ross, Reynolds, & Geis, 2000; Wickrama & Bryant, 2003; Williams and Collins 2016), and we suspect that many of the pathways linking neighborhood poverty, stress, and health also extend to schools. For example, students attending low-SES schools may have less access to health promoting resources (e.g., supportive adults at school), greater exposure to hazards (e.g., violence), and greater exposure to a range of acute and chronic stressors than students attending higher SES schools. Further, the daily stress of attending a low-SES school may produce feelings of hopelessness, frustration, and loneliness (Aneshenesel & Sucoff, 1996; Ross et al., 2000; Wickrama & Bryant, 2003), which may promote psychological distress and physiological dysregulation.

In such environments, relationships between students and teachers may also suffer. As Paulle (2013) and others have noted, the constant threat of violence is physically and emotionally taxing for adults and students, breeding mistrust between everyone. Yet students' relationships with adults and other students have important consequences for student outcomes (Blum, 2005), including health. For example, increased attachment to school and perceptions of teacher fairness are associated with positive health outcomes for students (Goosby & Walsemann, 2012). Thus, factors that inhibit the development of strong connections among students and between students and teachers may contribute to the "toxicity" of a school.

It is also possible that structural and institutional characteristics of high-poverty schools, including the quality and stability of the teaching staff, or particular programs, policies, or procedures, could produce "toxic" environments with consequences for student health. Studies find that high-poverty and high-minority schools are more likely to have less experienced teachers, higher rates of teacher turnover, and teachers who are less familiar with their student population (Battistich, Solomon, Kim, Watson, & Schaps, 1995; Clotfelter, Ladd, Vigdor, & Wheeler, 2007; Perry & Weinstein, 1998; Morris, 2005; Ladson-Billings and Gloria 1995). These factors could lead to more disorder and more stress for students and teachers.

Despite these hypothesized links, few studies have directly examined the association between school SES and health or specified how school SES may affect health. We address this by examining the direct effect of school SES on student health and assessing whether SES affects student health through the "toxicity" of the environment, as Paulle's (2013) work suggests.

Hypothesis 2a. School SES is positively associated with physiological dysregulation and depressive risk.

Hypothesis 2b. "Toxicity" is a pathway linking school SES to physiological dysregulation and depressive risk.

Are majority-minority schools more likely to be "toxic?"

Because of long histories of racism, residential inequality, and racial segregation (see Massey & Denton, 1993), in the U.S., "toxic" schools, like the ones Paulle described, are largely composed of students of color, including Black and Latino students. Previous literature suggests that the racial composition of students in schools may be associated with student health outcomes (Bernell et al., 2009; Goosby & Walsemann, 2012; Walsemann, Bell, & Goosby, 2011; Walsemann, Bell, & Maitra, 2011; Walsemann et al., 2009). Scholars hypothesize a number of reasons why school racial composition may relate to student health, including racialized health norms or expectations and relationships in schools that contribute to negative or positive health outcomes (Bernell et al., 2009; Goosby & Walsemann, 2012; Walsemann, Bell, & Goosby, 2011; Walsemann, Bell, & Maitra, 2011; Walsemann et al., 2009).

However, the evidence about the relationship between school racial composition and health is largely mixed. On the one hand, attending predominately Black and Latino schools can be associated with worse health, particularly for outcomes such as obesity (Bernell et al., 2009). On the other hand, attending schools with higher proportions of students of color may confer health *benefits* to students. For example, Black students consistently report better self-rated health and fewer depressive symptoms as the proportion of students of color in a school increases (Goosby & Walsemann, 2012; Walsemann, Bell, & Goosby, 2011; Walsemann, Bell, & Maitra, 2011; Walsemann et al., 2009). These studies suggest that attending predominantly minority schools may provide health protections to Black students by increasing their attachment to school and reducing their exposure to discrimination (Walsemann, Bell, & Goosby, 2011). However, other studies have found that perceived discrimination and loneliness, though independently associated with health outcomes, do not appear to explain the association between school racial composition and early adult health (Goosby & Walsemann, 2012).

Complicating matters further is the association between school racial and socioeconomic composition, as high-minority schools also tend to have higher proportions of low-SES students, given high levels of racial residential segregation and the strong racial patterning of socioeconomic resources, opportunities, and risks in the U.S. It is unclear, then, whether and how racial composition on its own is connected to the kind of "toxic" school environments that Paulle (2013) describes or whether school racial composition and socioeconomic context have joint associations with health. Given that school SES is more impactful than school racial composition on non-health outcomes like achievement (Logan et al., 2012), it is possible that school "toxicity" has more to do with school-level poverty than the racial make-up of schools, but this remains unknown. There may be factors uniquely associated with school racial composition—such as high levels of race-based discriminatory stress—that shape student health. We address these questions by analyzing whether school SES and school racial composition uniquely or jointly predict student health and whether school "toxicity" is associated with those relationships.

Hypothesis 3a. School racial composition is associated with physiological dysregulation and depressive risk.

Hypothesis 3b. "Toxicity" is a pathway linking school racial composition to physiological dysregulation and depressive risk.

Measuring how school toxicity "gets under the skin"

To date, most studies of schools and health focus on general indicators of health, such as self-rated health, disease diagnosis, or health behaviors (e.g. smoking or drinking). Self-rated health is among the most common outcomes utilized in studies of school effects on health (e.g., Frisvold & Golberstein, 2011; Goosby & Walsemann, 2012). However, self-rated health is criticized for being vague and imprecise and leaving questions about biological plausibility unanswered (Benyamini,

2011; Finch, Hummer, Reindl, & Vega, 2002; Franks, Gold, & Fiscella, 2003; Jylha and Heikkinen, 1998). Further, reliance on measures of self-rated health assumes that respondents' self-assessments are accurate indicators of physical and mental well-being. Existing research that relies on such measures therefore lacks more objective assessments of how exposure to chronic school-related stress may affect the body in ways that are not necessarily perceptible to those affected. Young people who experience the daily threat of violence and disorder at school may not outwardly express negative effects of that environment in self-reports of health or in risk-taking behaviors. Yet, as Paulle (2013) suggests, the effects may still be present in the bodies of those exposed.

We draw on the stress process model (Pearlin et al., 1981) to assess how schools may impact health through stress-related pathways. Stress exposure, including exposure to stressors in the social environment, is linked to increased physiological dysregulation, higher allostatic load, and worse health overall (Cohen et al., 2012; Epel & Lithgow, 2014; Glaser & Kiecolt-Glaser, 2005; McEwen & Stellar, 1993; Thoits, 2010). In response to perceived stressors, the hypothalamic-pituitary-adrenal (HPA) axis and sympathetic nervous system (SNS) secrete hormones to up-regulate psychological and physiological functioning—a process often referred to as the "fight or flight" response (McEwen, 1998; McEwen & Stellar, 1993). While this up-regulation of bodily systems is a protective response to acute threats, prolonged activation of these systems in response to chronic stressors—such as those associated with repeated exposure to violence or disorder—can harm health by promoting physiological dysregulation and allostatic load (Cohen et al., 2012; Glaser & Kiecolt-Glaser, 2005; McEwen, 1998; McEwen & Stellar, 1993). Exposure to chronic stress diminishes the ability of bodily systems to down-regulate (Cohen et al., 2012; Miller, Cohen, & Ritchey, 2002), such that psychophysiological systems continue to operate under "threat levels" even in the absence of a direct or acute threat. This chronic, low-grade activation of the body's stress response system contributes to the malfunctioning of bodily systems and eventual increased disease and mortality risk (Friedman & Herd, 2010).

We expect that physiological and psychological stress response processes may be activated by school stressors. The sociodemographic, structural, and social characteristics of schools may chronically up-regulate physiological and psychological stress response systems in ways that could be toxic to health. In these ways, students exposed to stressors in the school environment may be at increased risk of physiological dysregulation and psychological risk. Because low-income students and students of color may be disproportionately exposed to stressful school environments given the unequal patterning of school resources in the U.S., school environments may play an important role in producing population inequities in biological and psychological risk and susceptibility. This study therefore uses a rich set of biomarker measures, including immune, cardiovascular, and metabolic functioning, as well as an indicator of depressive risk, to assess how the stressful social conditions in schools "get under the skin" to affect overall health through a nonspecific array of psychophysiological mechanisms.

School toxicity as an early-life determinant of health

Young people's early life exposures can shape health and well-being across the life span. Drawing on insights from the life course perspective (Elder, 1998), several theoretical models have been proposed to link early-life exposures to later life health outcomes. One dominant paradigm for understanding these links is the sensitive period model, which argues that exposures during particularly vulnerable life course stages, like childhood or adolescence, may induce enduring or irreversible psychological or physiological changes that continue to affect health well into adulthood (Ben-Shlomo & Kuh, 2002). Few studies have examined schools' long-term impacts on health, but given that scholars identify childhood and adolescence as sensitive periods in the life course (Fuhrmann et al., 2015; Lawlor & Chaturvedi, 2006; Sawyer et al., 2012), we speculate that school exposures during these early-life stages could impact health trajectories into adulthood, long after individuals have left school.

The life course perspective also recognizes that health and development are life-long processes (Pavalko & Willson, 2011). Individual health and population health disparities vary over the life span, as do the strength of associations between social exposures and health. It is therefore possible that school exposures affect individual health and population health inequality as individuals age, though this hypothesis has not been fully tested.

Hypothesis 4. School "toxicity" will be positively associated with markers of health risk from adolescence through young adulthood.

Gaps in the literature

In sum, despite growing interest in the relationship between schools and health, research in this area is still relatively limited, and four critical gaps remain. First, it is not yet clear what characteristics of schools are "toxic" to student health, though other ethnographies of violent and stressful school environments lead to reasonable speculation about what toxic environments might look and feel like (Caudillo & Torche, 2014; Devine, 1996; Fullilove et al., 2003, pp. 198–246; Hagan et al., 2003, pp. 163–197; Kozol, 1991). Goosby and Walsemann's (2012) analysis of survey data found that, at the individual-level, student perceptions of discrimination and school attachment are associated with self-rated health. Building on these findings and further integrating insights from Paulle (2013), we assess the dimensions of school environments that are particularly toxic for young people. Determining the characteristics of school toxicity is critically important, since it can inform policy and intervention efforts aimed at improving the health of students in such environments.

Second, it is unclear which kinds of schools are most likely to be toxic. Paulle's (2013) ethnography suggests that toxic schools are likely to be high-poverty, high-minority schools for several key reasons: high rates of violence and fear related to poverty, disconnectedness and mistrust among students and teachers in such environments, and structural disorder from high teacher turnover and other school policies (all of which stem from the structural inequality that governs the organization of neighborhoods and schools). While research suggests that such processes may undergird the relationship between school demographics and health (Aneshenesel & Sucoff, 1996; Battistich et al., 1995; Clotfelter et al., 2007; Dance, 2002; Caudillo and Torche 2015; Frisvold & Golberstein, 2011; Nolan, 2011; Ross et al., 2000; Wickrama & Bryant, 2003), these associations have not been tested directly.

Third, it is not yet clear which aspects of health are most affected by toxic school environments. Because most studies rely on subjective measures of general health, it remains unknown how school conditions "get under the skin" to affect psychological and physiological well-being and disease risk. In addition to raising concerns about biological plausibility, the over-reliance on general indicators of health also risks misclassification error, where individuals who do not yet have a disease, have not yet been diagnosed with a disease, or who are otherwise unaware of their health risk are classified as "well" (Turner, 2013). Misclassification error may be of particular concern for vulnerable populations, like adolescents, young adults, people of color, and poor individuals, many of whom are at high risk of developing disease but either do not yet have an official disease diagnosis or have not received an accurate one (Nguyen et al., 2011; Williams & Jackson, 2005). For this reason, the integration of pre-disease markers of biological and psychological function can improve understanding of the role of school contexts in disease emergence and progression, particularly early in the life course.

Finally, while a number of studies have established cross-sectional associations between school characteristics and health, few have examined the health impacts of school exposures using longitudinal data. In addition to raising concerns about causality, an over-reliance on

cross-sectional data restricts understanding of whether and how school exposures shape trajectories of health risk, even after individuals transition out of the school environment and into adulthood. Ultimately, this limits understanding of how school exposures influence the life course patterning of health inequality.

The present study

This study addresses these gaps by assessing how the sociodemographic composition of schools, as well as diverse measures of school "toxicity," impact trajectories of physiological and psychological well-being from adolescence through young adulthood. Moving beyond general measures of health, we use outcomes with documented links to the body's stress response systems: physiological dysregulation and depressive risk. Findings from this study therefore provide new evidence of the biological and psychological mechanisms undergirding schools' relationship to health and disease risk from adolescence through young adulthood, shedding new light on school factors that can ameliorate population health inequality.

Data and methods

Data and analytic sample

Data for this study come from the National Longitudinal Study of Adolescent to Adult Health (Add Health)—a nationally representative, longitudinal study of U.S. adolescents. Using a school-based complex cluster sampling frame, Add Health began in 1994-95 with an in-school questionnaire administered to a nationally-representative sample of students in grades 7–12, along with a school administrator questionnaire for information on school characteristics. Following the in-school questionnaire, a gender- and grade-stratified random sample of 20,745 adolescents (79% response rate) was selected for in-home interviews at Wave I. Follow up in-home interviews were then conducted in 1996 (Wave II; 88% response rate), 2001–02 (Wave III; 77% response rate), and 2007–08 (Wave IV; 80% response rate). At Wave IV, Add Health also collected biological specimens from study participants, including dried blood spots. For detailed information about biomarker collection procedures and protocols, see Entzel et al. (2009) and Whitsel et al. (2012). Add Health's content and longitudinal design make it a particularly rich source of data for studying the impact of schools on life course trajectories of health and well-being.

We use the in-school questionnaires, school administrator questionnaire, and in-home interviews at Wave I; the in-home interviews at Waves II and Wave III; and the in-home interviews and biomarker study at Wave IV. We also use Census tract-level data linked to respondents' residences at Waves I-IV. Our analytic sample includes respondents who have complete data on the outcomes and valid sampling weights. Supplementary analyses revealed that the greatest sources of missing data were the individual and household SES measures, the indicator for parental reason for moving to the neighborhood at Wave I, and the school toxicity measures. We used multiple imputation by chained equations (MICE) procedures to impute missing data on these measures (10 multiply imputed data sets). Final analytic samples vary by outcome: physiological dysregulation (n = 10,893 respondents) and depressive symptoms (n = 37,655 person-observations from 11,382 unique respondents).

Measures

Dependent variables

The outcomes of interest include indicators of physiological dysregulation and depressive symptoms. The measure of physiological dysregulation was available at Wave IV, while depressive symptoms are measured at Waves I, II, III, and IV. We selected these outcome measures because they reflect diverse aspects of well-being that represent important risk factors for future morbidity and mortality. Unlike measures of disease status, our outcomes reflect bodily function across metabolic, cardiovascular, immune, and psychological systems and provide a comprehensive assessment of health risk.

We construct a composite indicator of *physiological dysregulation*, which indicates overall levels of biological burden across metabolic, cardiovascular, inflammatory and immune systems using clinical markers: systolic blood pressure, diastolic blood pressure, Hba1c, waist circumference, body mass index, HDL cholesterol, LDL cholesterol, total cholesterol, C-reactive protein, and Epstein-Barr virus. For each individual measure, we construct a dummy indicator where "1" indicates high risk according to clinical guidelines or, when clear clinical cut-points are not available, by top quartile of the distribution. We then sum the scores from each of the markers to construct the index of overall physiological dysregulation (range: 0–10). Similar to the concept of allostatic load (McEwen, 1998; Seeman, Singer, Rowe, Horwitz, & McEwen, 1997), physiological dysregulation captures the "wear and tear" across bodily systems that occurs over time as the body attempts to respond to stressors in the social environment (McEwen, 1998; Seeman et al., 1997). Studies find that levels of physiological dysregulation are predictive of health and mortality risk from a host of causes (Seeman et al., 1997).

Depressive symptoms is a continuous measure indicating respondents' score on the Center for Epidemiological Studies-Depression (CESD) scale. We used 9 items that were consistently-measured across waves and that asked respondents how often during the past week they: were bothered by things that didn't usually bother them; felt they could not shake the blues; were just as good as other people (reverse coded); had trouble keeping their mind on what they were doing; felt depressed; were too tired to do things; enjoyed life (reverse coded); felt sad; and felt that people disliked them. Alternative operationalizations yielded substantively similar results. In addition to being highly predictive of future major depression (Pine, Cohen, Cohen, & Brook, 1999), depressive symptoms have also been linked to declines in physical health (Kiecolt-Glaser & Glaser, 2002; Penninx et al., 1998). Depressive states can directly stimulate the production of physiological stress response systems, which in turn influences a host of diseases and conditions, including cardiovascular disease, diabetes, and cancer (Kiecolt-Glaser & Glaser, 2002). Depressive states can also down-regulate the body's immune function, making individuals with high levels of depressive symptoms more prone to prolonged infection and delayed wound healing (Kiecolt-Glaser & Glaser, 2002). Because of a skewed distribution, the measure of depressive symptoms is log transformed.

Key explanatory variables

The key explanatory variables include several school-level measures. *School socioeconomic composition* is a composite measure that reflects the average parental education level and household income of students. To calculate school SES, we first calculated a composite SES score for each student, which is the mean of standardized (z-score) measures of parental education and household income. Using the students' SES scores, we then calculated the mean school SES. Higher values indicate higher SES. *School racial composition* indicates the percentage of students of color in the school. Alternative operationalizations of the school SES and racial composition measures yielded substantively similar results.

We examine whether school sociodemographic characteristics are related to health through school "toxicity." To create our measures of school toxicity, we use principal component analysis (PCA). A full list of the variables used in the PCA, with additional details about variable measurement, is in Table 1. It is worth noting that all factor loadings were moderate to strong, and the item-rest correlations for each of the individual measures used in the factor measures well exceeded 0.20, which is considered satisfactory for reliability (Kline, 1986).

The first factor (Eigen value = 1.63), which we call *school safety and violence*, is a school-level measure that reflects students' perceptions of safety and average exposure to violence and capturesstudents' fears and

Table 1
Dimensions of School Toxicity: Results of confirmatory factor analysis.

Factors	Description of Measures	Data Source	Coding of Measures
Factor 1: School safety and violence	Don't feel safe at school	Wave I in-home interview	1 = disagree or strongly disagree that student feels safe in school
	High exposure to violence	Wave I in-home interview	1 = reported exposure to violence
Factor 2: Teacher turnover	High percentage of new teachers[a]	Wave I school administrator questionnaire	1 = ≥11% of teachers in school are new
	Low percentage of teachers at school for ≥5 years[a]	Wave I school administrator questionnaire	1 = ≤46% of teachers in school have been at school for 5+ years
Factor 3: Low school connectedness	Low closeness to people at school	Wave I in-home interview	1 = disagree or strongly disagree that student feels close to people at school
	Feel teachers don't care	Wave I in-home interview	1 = disagree or strongly disagree that teachers care about students
	Feel teachers aren't fair	Wave I in-home interview	1 = disagree or strongly disagree that teachers are fair

Note: All measures are school-level variables. For variables originally measured at the individual-level, school-level measures are calculated as the average of student responses in each school.
a: "high" or "low" based on top/bottom quartile of the distribution.

concerns about safety in school. The second factor (Eigen value = 1.18) includes two measures of *teacher turnover*, another institutional characteristic that may reflect school disruption and disorder. Teachers are more likely to leave schools with high levels of disorder (Allensworth and Mazzeo 2009; Ingersoll, 2001), particularly student misbehavior and conflict, but the heavy rotation of teachers in and out of a school is itself disruptive, as it negatively impacts students' ability to form relationships with adults at school. The third factor (Eigen value = 2.03) includes three school-level measures of *school connectedness and cohesion*, which indicates overall relationship quality among students and between students and teachers in a school. We assume that more cohesive environments, where students feel cared for and connected to one another, provide a buffer from other kinds of stressors.

We also created a measure of overall toxicity, which encompasses all three factors. Together, these dimensions of school toxicity reflect many of the aspects of school-level disorder, violence, and stress described by Paulle (2013). It is important to note that all of our measures of school toxicity are at the school-level. For variables originally measured at the individual-level, we generate school-level measures as the average of student responses for students in the school. In supplementary analyses we also adjusted for individual levels of school safety and violence and school connectedness, and results were substantively consistent with the results presented here.

To assess the extent to which school exposures shape trajectories of depressive risk across Waves I-IV, we also include interaction terms for our school level measures by survey wave (e.g., school SES X wave, school safety and violence X wave, etc.).

Covariates

Our models also adjust for individual-level covariates, including race/ethnicity (1 = White, 2 = Black, 3 = Hispanic, 4 = Asian), age (years), gender (1 = female), and individual/family SES. Coefficient estimates for race/ethnicity capture the racial/ethnic disparities in the outcomes. Our measure of SES in adolescence (Waves I and II) is the mean of standardized measures of parental education and household income. Our measure of socioeconomic attainment at Waves III and IV is the mean of standardized (z-score) measures of respondent completed education and household income.

To reduce confounding school and neighborhood conditions, we also adjust for neighborhood economic deprivation using a composite index of four Census tract measures of neighborhood economic conditions: proportion of residents who are unemployed, proportion of residents over the age of 25 years without a high school degree, proportion of families living in poverty, and proportion of families receiving public assistance. For each individual measure, we created a dummy variable indicating the top quartile of all Census tracts. We then summed the four dummy measures, producing an index of neighborhood economic deprivation ranging from 0 (low deprivation) to 4 (high deprivation). To better account for student selection into schools, we also include parental responses to questions about why they chose to live in their neighborhood at Wave I (responses included: near old workplace; near current workplace; had outgrown previous housing; affordable good housing; less crime; less illegal activity; close to friends or relatives; better schools; more children of similar age to children in the household; born here). In models of prospective associations between school exposures and physiological dysregulation, all covariates, except individual SES in young adulthood, are measured at baseline (Wave I). In the longitudinal models, individual/family SES and neighborhood economic disadvantage are included as time-varying measures.

Analytic methods

We begin our analysis with descriptive statistics, paying particular attention to the bivariate associations between our key explanatory measures—school SES and overall school toxicity—and the outcomes.

To examine the associations between school exposures and physiological and psychological well-being, we utilize multilevel regression models to account for the nesting of individual students within schools. In the multilevel models, individual outcomes are predicted by individual- and school-level variables. All multilevel models also include random intercepts at the school level.

We estimate models for physiological dysregulation and depressive symptoms separately. Add Health began collecting comprehensive biomarker data at Wave IV, so the physiological dysregulation models regress the Wave IV outcome on the covariates. The physiological dysregulation models therefore indicate the prospective associations between school exposures in adolescence and overall biophysiological burden in young adulthood. Model 1 regresses the outcome on school socioeconomic conditions and racial composition, adjusting for age, gender, race/ethnicity, family SES in adolescence, neighborhood economic deprivation in adolescence, and parental reason for moving to the neighborhood. Model 2 builds on Model 1 by also adjusting for individual SES in young adulthood. Models 3–5 build on Model 2 by including each of the measures of school toxicity in a stepwise fashion. Model 6 includes all dimensions of school toxicity simultaneously. Finally, in Model 7, we include the composite measure of overall toxicity. In the physiological dysregulation models, all covariates are measured during adolescence, with the exception of SES in young adulthood, which was measured at Wave IV.

The depressive symptoms models examine how school exposures in adolescence shape trajectories of mental health using longitudinal outcome data. In these models, the data has a three level structure, where observations (level 1) are clustered within individuals (level 2), who are nested within schools (level 3). Because Add Health does not include three-level weights, we estimate average level health trajectories, including random intercepts at the school level. Supplementary analyses utilizing unweighted three-level models with random intercepts at both the individual and school levels, as well as marginal models that do not fully account for the clustering of individuals within schools, produced substantively similar results. Together, results from these supplementary analyses indicate that our results are robust to

alternative model specifications. Our longitudinal models for CESD take the following generic form:

$$y_{tij} = \alpha + \beta_1 wave_{tij} + \beta_2 age_{tij} + \beta_3 gender_{ij} + \beta_4 race_{ij} + \beta_5 SES_{tij} + \beta_6 neighborhood\ SES_{tij} + \beta_7 school\ sociodemographics_{ij} + \beta_8 school\ sociodemographics_{ij} * wave_{tij} + \beta_9 school\ toxicity_{ij} + \beta_{10} school\ toxicity_{ij} * wave_{tij} + e_{0j}$$

In addition to examining the relationship between schools' sociodemographic characteristics, school toxicity, and mean levels of depressive symptoms, these models also assess how school exposures in adolescence relate to changes in the outcomes from Wave I through Wave IV. In the multilevel mixed effects depressive symptoms models, we use a similar covariate adjustment strategy to the physiological dysregulation models. Model 1 regresses the outcomes on school socioeconomic conditions and racial composition, adjusting for age, gender, race/ethnicity, neighborhood economic deprivation, and parental reason for moving to the neighborhood. Model 2 builds on Model 1 by also adjusting for time-varying individual/family socioeconomic status. Models 3–5 build on Model 2 by including each measure of school toxicity in a stepwise fashion, and Model 6 includes all three dimensions of toxicity simultaneously. Finally, we include the overall measure of school toxicity in Model 7. In the longitudinal CESD models, the measures of race, gender, parental reason for living in neighborhood, school SES, school racial composition, and school toxicity are time constant, while the measures of age, survey wave, neighborhood economic disadvantage, and individual/family SES are time varying.

All analyses use sample weights to ensure the representativeness of the respondents. We model both outcomes as linear measures, but alternative model specifications produced substantively similar results. Supplemental analyses examined interactions between race, family SES in adolescence, and the measures of school context (e.g., race X school SES, race X school toxicity, family SES at Wave I X school SES, etc.), but we found no evidence of moderating effects.

Results

Descriptive statistics

Descriptive statistics for the outcomes and key explanatory variables are presented in Table 2. In addition to full sample statistics, we also present descriptive statistics by school SES and overall level of school toxicity. Though both of these measures are included as continuous measures in multivariate models, for descriptive purposes, we dichotomize school SES and toxicity into "low" and "high" categories. "Low" school SES indicates being in the lowest quartile of school SES, and "high" school SES indicates being in the highest three quartiles of school SES. "High" school toxicity indicates being in the top quartile of school toxicity, whereas "low" school toxicity indicates being in the bottom three quartiles of school toxicity. Results in Table 2 reveal that, compared to individuals who attended high SES schools in adolescence, individuals who attended low SES schools had higher levels of physiological dysregulation at Wave IV ($p < 0.001$) and more depressive symptoms across waves. Further, individuals who attended high toxicity schools in adolescence had higher levels of physiological dysregulation ($p < 0.001$) and depressive risk than individuals who attended low toxicity schools. Table 2 also reveals a strong bivariate relationship between toxicity and school SES. Both "low" SES schools and "high" toxicity schools had greater proportions of students of color than "high" SES schools and "low" toxicity schools.

Multilevel models

Physiological dysregulation

Results from the multilevel physiological dysregulation models are in Table 3. These models estimate the prospective associations between school exposures in adolescence and biophysiological risk in young adulthood. Model 1 indicates that school socioeconomic context in adolescence is negatively associated with physiological risk; low school SES in adolescence is associated with higher levels of physiological dysregulation in young adulthood (−0.43, $p = <0.001$), net of family SES in adolescence. Across all models we find no relationship between school racial composition and the outcome. In Model 2, we include the measure of individual socioeconomic attainment in young adulthood, which is negatively associated with physiological dysregulation. Including the measure of Wave IV SES partially attenuates the association between school SES and physiological dysegulation, which suggests that individual socioeconomic attainment may partially mediate the association between school SES and physiological risk. Across models, Black and Hispanic individuals have especially high levels of physiological dysregulation compared to Whites, and the racial gaps are largely consistent across models, providing little evidence that the measures of school toxicity mediate racial gaps in physiological risk.

In Models 3–6, we include measures of school toxicity in a stepwise fashion. Results from Model 3 indicate that students who attended schools with higher levels of safety concerns and violence in adolescence

Table 2
Descriptive statistics (n = 11,382).

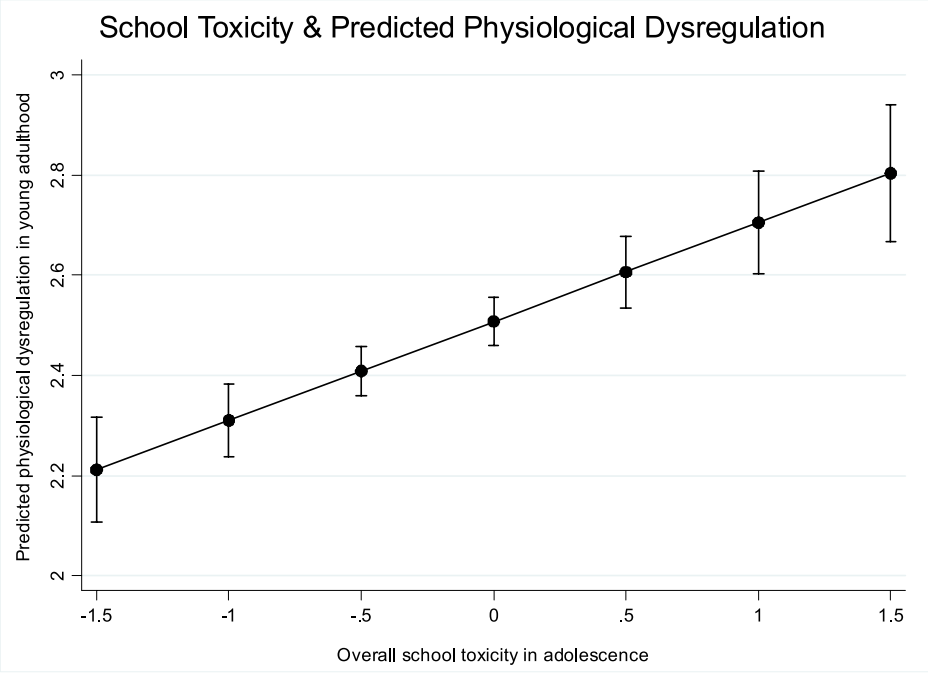
	Full Sample	By School SES			By Level of School "Toxicity"		
		High SES	Low SES		Low Toxicity	High Toxicity	
	Mean/Prop.	Mean/Prop.	Mean/Prop.	*p*-value	Mean/Prop.	Mean/Prop.	*p*-value
Outcomes							
Physiological dysregulation (at Wave IV)	2.49	2.37	2.83	<0.001	2.38	2.80	<0.001
Depressive symptoms (log CESD score)							
Wave I	1.68	1.67	1.73	0.069	1.66	1.73	0.052
Wave II	1.67	1.63	1.78	<0.001	1.65	1.72	0.036
Wave III	1.42	1.39	1.51	<0.001	1.42	1.43	0.708
Wave IV	1.60	1.56	1.68	<0.001	1.57	1.66	0.003
School Characteristics							
School socioeconomic status	0.02	0.16	−0.36	<0.001	0.80	−0.13	0.001
School racial composition (% students of color)	0.35	0.28	0.56	<0.001	0.27	0.56	<0.001
School toxicity (overall)	−0.06	−0.19	0.29	<0.001	−0.37	0.76	<0.001
School safety and violence	−0.20	−0.46	0.53	<0.001	−0.53	0.69	<0.001
Teacher turnover	0.09	0.12	0.00	0.542	−0.12	0.67	<0.001
Low school connectedness	−0.06	−0.21	0.36	0.010	−0.42	0.91	<0.001

Notes: Sample size based on depressive symptoms analytic sample (n=11,382), except for physiological dysregulation outcome (n=10,893). School characteristics measured at Wave I. p-values of difference between low/high schools and low/high "toxicity" schools, respectively. "Low" school SES indicates being in the lowest quartile of school SES; "high" school SES indicates being in the highest three quartiles of school SES. "High" school toxicity indicates being in the top quartile of school toxicity; "low" school toxicity indicates being in the bottom three quartiles of school toxicity.

Table 3
School exposures in adolescence in physiological dysregulation in young adulthood.

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
	Coeff (SE)	Coeff (SE)	Coeff (SE)	Coeff (SE)	Coeff (SE)	Coeff (SE)	Coeff (SE)
Individual characteristics							
Age	0.09***	0.10***	0.10***	0.10***	0.08***	0.09***	0.09***
	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)
Gender (1 = female)	−0.32***	−0.31***	−0.31***	−0.31***	−0.31***	−0.31***	−0.31***
	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)
Race/ethnicity (white is reference)							
Black	0.51***	0.47***	0.48***	0.47***	0.47***	0.47***	0.48***
	(0.10)	(0.10)	(0.10)	(0.10)	(0.10)	(0.10)	(0.10)
Hispanic	0.24*	0.25*	0.26*	0.24*	0.27*	0.26*	0.26*
	(0.13)	(0.12)	(0.12)	(0.12)	(0.12)	(0.12)	(0.12)
Asian	0.40+	0.36+	0.36+	0.36+	0.36+	0.35	0.36
	(0.21)	(0.21)	(0.21)	(0.22)	(0.21)	(0.22)	(0.22)
Family SES in adolescence (Wave I)	−0.11*	−0.05	−0.06	−0.06	−0.06	−0.06	−0.06
	(0.07)	(0.05)	(0.05)	(0.05)	(0.05)	(0.05)	(0.05)
Neighborhood economic disadvantage in adolescence (Wave I)	−0.00	−0.00	−0.00	−0.00	−0.00	−0.00	−0.00
	(0.03)	(0.03)	(0.03)	(0.03)	(0.02)	(0.02)	(0.02)
Individual SES in young adulthood (Wave IV)		−0.19***	−0.18***	−0.19***	−0.18***	−0.18***	−0.18***
		(0.04)	(0.04)	(0.04)	(0.04)	(0.04)	(0.04)
School sociodemographic characteristics							
School socioeconomic composition (Wave I)	−0.43***	−0.35***	−0.25**	−0.35***	−0.27***	−0.27**	−0.26**
	(0.08)	(0.09)	(0.09)	(0.09)	(0.07)	(0.08)	(0.08)
School racial composition (% students of color) (Wave I)	0.00	0.00	0.00	0.00	0.00	−0.00	−0.00
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
School "toxicity"							
School safety and violence			0.10*			0.01	
			(0.04)			(0.04)	
Teacher turnover				0.03		0.05*	
				(0.03)		(0.02)	
Low school connectedness					0.10***	0.10***	
					(0.02)	(0.02)	
Overall toxicity							0.20***
							(0.04)
Intercept	−0.44	−0.51	−0.44	−0.62	−0.12	−0.18	−0.33
	(0.57)	(0.57)	(0.58)	(0.58)	(0.58)	(0.57)	(0.56)

Notes: Results of mixed effects models. Physiological dysregulation was measured at Wave IV. Models also control for parental reason for living in neighborhood (measured at Wave I). Only fixed effects coefficients presented. n = 10,893.
***p < 0.001, **p < 0.01, *p < 0.05, + p < 0.1.



Notes: Results based on Model 7 of Table 3. n=10,393.

Fig. 1. School Toxicity in Adolescence and Physiological Dysregulation in Young AdulthoodNotes: Results based on Model 7 of Table 3 n = 10,393.

had higher levels of physiological dysregulation in young adulthood. While we find no associations between teacher turnover and the outcome in Model 4, Model 5 shows that students reporting lower levels of school cohesion and connectedness in adolescence have higher levels of physiological risk in young adulthood (0.10, $p < 0.001$). Including the measure of school safety and violence in Model 3 further attenuates the coefficient estimate for school socioeconomic composition over Model 2, suggesting that this dimension of school toxicity may contribute to the association between school SES and physiological risk. In Model 6 we include all three dimensions of school toxicity simultaneously, and the association between low school connectedness and physiological dysregulation persists, net of the other dimensions of toxicity. In Model 6 we also find that high levels of teacher turnover are associated with the outcome. Finally, in Model 7, we find that our composite indicator of overall school toxicity is strongly associated with physiological dysregulation in young adulthood (0.20, $p < 0.001$). Fig. 1 presents the results of Model 7 and reveals a clear gradient in young adult physiological well-being resulting from adolescent school exposures.

Depressive symptoms

Table 4 presents the results from the longitudinal CESD models, where we model trajectories of depressive symptoms from adolescence through young adulthood as a function of school exposures in adolescence. In Model 1, we find a significant relationship between school socioeconomic composition and depressive risk, but this association is completely attenuated with the inclusion of the measure of family/individual SES in Model 2. Consistent with the physiological dysregulation models, we find no association between school racial composition and depressive risk. Still, all models reveal striking racial disparities in depressive risk that remain largely unchanged with the inclusion of the individual- and school-level covariates across models.

In Models 3–6, we include the measures of school toxicity in a stepwise fashion. In Model 3, we find that low levels of safety and high exposure to violence are associated with greater depressive risk (0.09, $p < 0.001$), an association that is consistent over time. In Model 4 we find evidence that teacher turnover is marginally associated with depressive risk in adolescence, but that association is fully attenuated by Wave IV. In Model 5 we include the measure of low school connectedness and find a strong positive association between low school connectedness and depressive risk (0.06, $p < 0.001$) that diminishes over time. Fig. 2 illustrates how the association between low school connectedness and depressive symptoms varies over time. In Model 6, the association between school safety and violence is no longer associated with depressive risk when the other dimensions of toxicity are controlled. Low school connectedness maintains a strong, positive association with depressive risk in Model 6 (0.05, $p < 0.001$) that fades across subsequent waves. Finally, Model 7 includes the composite indicator for overall school toxicity, which is positively associated with depressive risk.

Discussion

A wide body of research documents the critical role of contextual factors in shaping individual and population health, with most research in this area focused on neighborhood influence (Diez Roux, 2001; Diez Roux & Mair, 2010; Kawachi & Berkman, 2003; Ross & Mirowsky, 2001). Yet young people spend a large proportion of their waking time at school, and less is known about how school contexts shape the health of young people as they age. Using nationally representative, longitudinal school-, family-, and individual-level data, our study examined how exposure to a variety of school-level factors relates to markers of psychological well-being and physiological functioning from adolescence through early adulthood. By integrating diverse and nuanced measures of school context and markers of pre-disease physiological and psychological well-being, this study improves understanding of how school contexts shape the health of young people and serve as salient early-life stressors with lasting health consequences.

This study makes four key contributions. First, our study is the first to use survey data to conceptualize and operationalize Paulle's (2013) notion of "school toxicity" and further test its association with diverse markers of health. A growing body of studies links school characteristics to health (Bernell et al., 2009; Goosby & Walsemann, 2012; Walsemann, Bell, & Goosby, 2011; Walsemann, Bell, & Maitra, 2011; Walsemann et al., 2009). Using confirmatory factor analyses, we built on this work to consider multiple dimensions of "toxicity" as described by Paulle (2013) and generated three individual measures of school toxicity, each relating to a different dimension of toxicity: perceptions of school safety and indications of violence; teacher turnover; and perceptions of school cohesion and connectedness. These measures in many ways reflect diverse social and structural characteristics described by Paulle (2013). The use of factor analysis allowed us to comprehensively consider how latent constructs reflecting school environment relate to student well-being.

Second, consistent with Paulle's (2013) arguments and adding to the growing body of research on schools and health and in support of Hypothesis 1, we found evidence that schools can, indeed, be "toxic" places that can have strong, immediate, and lasting impacts on markers of physiological functioning and mental health. While we found weak and rather mixed evidence of the link between teacher turnover and the markers of health, our findings revealed that school safety and violence, low school connectedness, and overall levels of school toxicity were strongly associated with markers of physiological dysregulation and psychological well-being. School safety and violence, as indicated by school-level measures reflecting student concerns about safety and exposure to violence, was associated with both physiological functioning and psychological well-being in Model 3 of Tables 3 and 4. Just as exposure to neighborhood-level violence has been found to impact children's sleep and cortisol patterns (Heissel et al., 2017), our findings suggest that violence and safety threats in one's social environments may harm health through stress-related pathways. Based on evidence from other neighborhood-level studies (Gooding, Milliren, Austin, Sheridan, & McLaughlin, 2015; Heissel et al., 2017; Kirk & Hardy, 2014; McCoy, Raver, & Sharkey, 2015), we suspect that perceptions of safety and violence may promote worry and hypervigilance among students in ways that chronically activate stress response systems to ultimately impact both acute and future health and well-being.

Low levels of school connectedness were also strongly and consistently associated with higher risk of physiological dysregulation and greater numbers of depressive symptoms from adolescence through young adulthood. This is consistent with other documented associations between student connection to teachers and well-being (Goosby & Walsemann, 2012) and extends a wide body of literature documenting the critical role of social connectedness and social support in maintaining and protecting physiological and mental health across the life course (Berkman, Glass, Brissette, & Seeman, 2000; Holt-Lunstad, Smith, & Layton, 2010; Yang et al., 2016). Importantly, our measures indicating school safety and violence and school connectedness were measured at the school level, suggesting that school environments characterized by high levels of concern about safety and violence and low levels of closeness, care, and trust are detrimental for student health. The composite indicator of overall toxicity was related to both physiological dysregulation and depressive risk, as indicated in Model 7 of Tables 3 and 4 and illustrated in Fig. 1.

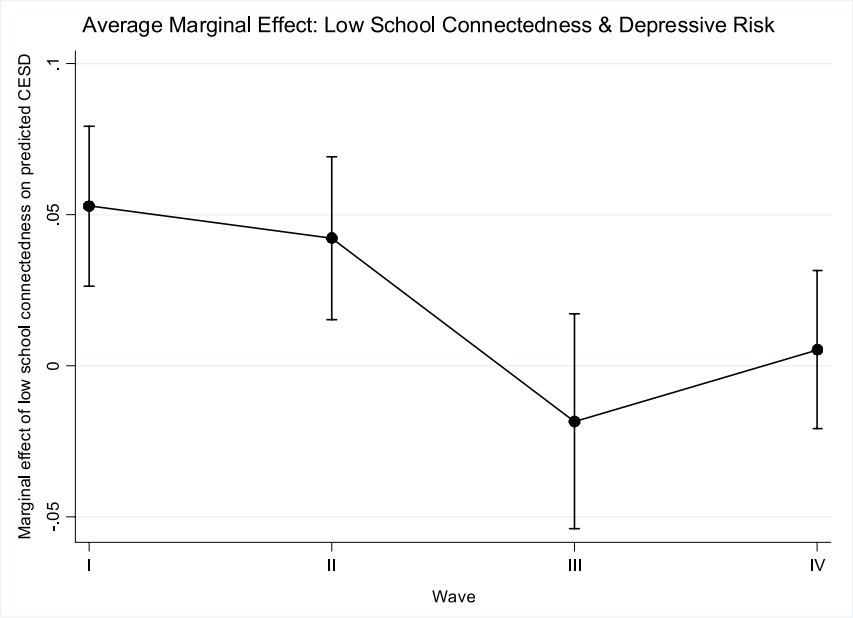
Taken together, results indicate that measures of school toxicity had clear associations with objective markers of physiological and psychological health. This finding is an important contribution to our understanding of contextual effects, and particularly those of schools, on health, since much of the extant research on school effects relies upon general measures of self-reported health.

Third, we found mixed evidence of the role of school toxicity in linking school SES and school racial composition to health. The "toxic" school Paulle (2013) studied in the U.S. was majority poor and

Table 4
School exposures in adolescence in trajectories of depressive risk through young adulthood.

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
	Coeff (SE)	Coeff (SE)	Coeff (SE)	Coeff (SE)	Coeff (SE)	Coeff (SE)	Coeff (SE)
Individual Characteristics							
Age	0.03***	0.02***	0.03***	0.02***	0.02***	0.02***	0.02***
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
Gender (1 = female)	0.19***	0.19***	0.19***	0.19***	0.19***	0.19***	0.19***
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
Race/ethnicity (white is reference)							
Black	0.15***	0.14***	0.14***	0.14***	0.14***	0.14***	0.14***
	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)
Hispanic	0.13***	0.11***	0.11***	0.11***	0.11***	0.11***	0.11***
	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)
Asian	0.05	0.04	0.04	0.04	0.04	0.04	0.04
	(0.05)	(0.05)	(0.05)	(0.05)	(0.05)	(0.05)	(0.05)
Neighborhood economic disadvantage	0.02**	0.01+	0.01+	0.01+	0.01+	0.01+	0.01+
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
Individual/Family SES		−0.12***	−0.12***	−0.12***	−0.12***	−0.12***	−0.12***
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
School sociodemographic characteristics							
School socioeconomic composition	−0.12**	−0.03	−0.06	−0.01	0.02	0.04	−0.00
	(0.04)	(0.04)	(0.05)	(0.04)	(0.03)	(0.04)	(0.04)
School socioeconomic composition by wave (Wave I is reference)							
School SES X Wave II	0.02	−0.05	−0.02	−0.04	−0.05	−0.01	−0.04
	(0.05)	(0.05)	(0.05)	(0.05)	(0.05)	(0.05)	(0.05)
School SES X Wave III	0.03	−0.02	−0.04	−0.02	−0.05	−0.01	−0.03
	(0.06)	(0.05)	(0.06)	(0.06)	(0.05)	(0.05)	(0.06)
School SES X Wave IV	0.04	0.02	0.01	0.01	−0.01	0.00	0.02
	(0.05)	(0.05)	(0.05)	(0.05)	(0.04)	(0.05)	(0.05)
School racial composition (% students of color)	−0.00	−0.00	−0.00	0.00	−0.00	−0.00	−0.00
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
School racial composition by wave (Wave I is reference)							
School racial composition X Wave II	0.00	0.00	−0.00	0.00	0.00	−0.00	0.00
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
School racial composition X Wave III	0.00	0.00	0.00	0.00	0.00	−0.00	0.00
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
School racial composition X Wave IV	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
School "toxicity"							
School safety and violence			0.09***			0.02	
			(0.02)			(0.02)	
School safety and violence by wave (Wave I is reference)							
School safety and violence X Wave II			0.03			0.04	
			(0.02)			(0.03)	
School safety and violence X Wave III			−0.03			0.06	
			(0.04)			(0.04)	
School safety and violence X Wave IV			−0.04			0.03	
			(0.03)			(0.03)	
Teacher turnover				−0.03+		−0.12	
				(0.02)		(0.01)	
Teacher turnover by wave (Wave I is reference)							
Teacher turnover X Wave II				−0.00		0.00	
				(0.01)		(0.01)	
Teacher turnover X Wave III				0.03		0.03	
				(0.02)		(0.02)	
Teacher turnover X Wave IV				0.04+		0.03+	
				(0.02)		(0.02)	
Low school connectedness					0.06***	0.05***	
					(0.01)	(0.01)	
Low school connectedness by wave (Wave I is reference)							
Low school connectedness X Wave II					0.00	−0.01	
					(0.01)	(0.02)	
Low school connectedness X Wave III					−0.05**	−0.07**	
					(0.02)	(0.02)	
Low school connectedness X Wave IV					−0.04*	−0.05**	
					(0.02)	(0.02)	
Overall toxicity							0.06*
							(0.03)
Overall toxicity by wave (Wave I is reference)							
Toxicity X Wave II							0.01
							(0.03)
Toxicity X Wave III							−0.04
							(0.05)
Toxicity X Wave IV							−0.02
							(0.03)
Intercept	1.07***	1.09***	1.18***	1.10***	1.18***	1.18***	1.13***
	(0.10)	(0.10)	(0.10)	(0.10)	(0.10)	(0.10)	(0.01)

Notes: Results of longitudinal mixed effects model. Only fixed effects coefficients presented. Models also adjust for survey wave and parental reason for living in neighborhood (measured at Wave I). n = 37,655 person-observations (11,382 unique individuals).
***p < 0.001, **p < 0.01, *p < 0.05, + p < 0.1.



Notes: Results based on Model 5 of Table 4. n=37,655 person-observations (11,382 unique individuals).

Fig. 2. Low School Connectedness in Adolescence and Trajectories of Depressive RiskNotes: Results based on Model 5 of Table 4 n = 37,655 person-observations (11,382 unique individuals).

predominantly Black—a result of urban disinvestment (Newman, 2004; Squires 1992) and other discriminatory housing, lending, and school zoning and student assignment policies that contribute to segregation (Massey & Denton, 1993; McDermott and Diem 2014; Rothwell, 2012). In the U.S., those who are poor, Black, and Latinx disproportionately attend segregated and underfunded schools, suggesting that poor young people and adolescents of color may be disproportionately exposed to the types of "toxic" school environments described by Paulle (2013). As such, we hypothesized that "toxicity" would be a key mechanism through which high poverty and majority-minority schools transmitted health disadvantages to students. Instead, we found more evidence that dimensions of school toxicity were associated with physiological dysregulation and depressive risk *net* of the sociodemographic characteristics of schools.

Descriptive statistics in Table 2 showed strong bivariate associations among school socioeconomic composition, school racial composition, and school toxicity. In general, high toxicity schools were composed of greater proportions of students of color and lower SES students. In Table 3, we found strong evidence that school SES shaped physiological dysregulation, but little evidence of a link between school SES and trajectories of depressive risk net of individual and family SES. In this way, we found mixed evidence for Hypothesis 2a. Results from Models 3–6 of Table 3 further revealed some evidence that the measures of school toxicity may mediate the relationship between school SES and physiological risk, as the coefficient estimate for school SES in Model 2 was attenuated with the inclusion of our school toxicity measures. In this way, results suggest that school toxicity may be one pathway linking school SES to physiological risk, providing support for Hypothesis 2b. Still, including both the measures of school SES and school toxicity revealed that these characteristics of schools are also unique related to physiological risk and that school SES maintained an association with physiological dysregulation after adjusting for toxicity.

School SES likely operates through multiple mechanisms to affect student health. For example, school SES is associated with nutritional value of school-provided meals, which can have an impact on students' physiological health, net of the stress induced by fear of violence, chaos, and disorder. School SES also does not just capture deprivation and disadvantage; it is a continuous measure that reflects school SES across the distribution. There may be features of high SES schools that contribute to the association between school SES and health but are not studied here (e.g. robust physical and mental health curriculum; more school psychologists, counselors, and student mental health support resources; etc.). It is therefore possible that school SES maintains its own independent association because our measure of toxicity, which is meant to capture some of the disadvantages of low-income schools described by Paulle (2013), does not account for the health advantages high-SES schools may confer to students. It is also possible, of course, that our measures of toxicity do not include some of the factors implicated in the school SES-health link (e.g., issues related to food access or the physical environments within schools), so more research in this area is needed. Still, our findings may also reflect the relatively independent influences of these characteristics of schools on health. Though high-poverty schools, on average, are characterized by higher levels of toxicity than higher SES schools, not all high-poverty schools are characteristically "toxic"; conversely, higher SES schools are not immune from concerns about safety (see DeJong, Epstein, & Hart, 2003, pp. 70–100; Newman & Fox, 2009) or challenges to connectedness and thus may also be toxic to student health. More research in this area is needed to assess whether and how health impacts of school socioeconomic context are largely independent of toxicity, and vice versa.

Our results provided no evidence of a link between school racial composition and the markers of health risk, net of school SES. Further, we find little evidence that school exposures help to explain racial-ethnic disparities in physiological dysregulation or depressive risk. In these ways, we found no support for Hypotheses 3a or 3b. The Black-White and Hispanic-White disparities in health documented in Tables 3 and 4 are both striking and persistent. They persist after adjusting for adolescent and young adult SES, neighborhood socioeconomic

exposures, and a host of school characteristics. Other studies find that the unequal patterning of discriminatory stress across schools may contribute to the links between school environments and health (Goosby & Walsemann, 2012; Walsemann, Bell, & Goosby, 2011). Our study did not include measures capturing this aspect of the school environment. Given that racial-ethnic disparities in health stem from structural racism and reflect the unequal distributions and accumulations of social, environmental, and psychological resources and risks, research should continue to interrogate how schools might unequally pattern material and psychosocial exposures in ways that shape racial-ethnic health inequities.

In sum, our findings speak to the critical role of stress-related processes ungirding associations between school environments and markers of physical and mental health, with school-related stress increasing depressive risk in the short term and physiological dysregulation in the long term. While lower SES schools with greater proportions of students of color tend to have higher levels of toxicity, schools are not toxic for student health *because* they are poor or comprised disproportionately of students of color. Perceptions of violence and social disconnectedness—whatever the composition of the student body—may be stressful enough to students that their mental and physiological health can be impacted, even long after leaving or graduating from school.

Our study is not without limitations. First, though we integrate individual- and family-level longitudinal data on several key measures, longitudinal biomarker and school-level data are not available in Add Health data, which limits our ability to make causal inferences. Second, though we are able to assess population average trajectories and estimate random school-level intercepts, we are unable to estimate random slopes or intercepts at the individual-level because there are no three-level sampling weights in Add Health data. Finally, though Add Health includes a number of school-level measures, it is possible that our analyses do not include several of the dimensions of school "toxicity," as originally hypothesized by Paulle (2013), such as the frequency of in-school fights or the presence of gangs in schools. Fear of or trauma associated with school shootings, although relatively rare when the Add Health students were in school in the mid-90s (and not available in the data set), may also contribute to school toxicity. Other characteristics that shape later life SES, such as the academic performance of schools, may also disparately affect health outcomes in early adulthood. As such, future research should build on our results to continue examining the school features and characteristics that affect student health and contribute to population health inequality.

Together, findings from this study revealed that, across multiple measures of biological and psychological well-being, schools matter for health. The school environment shaped the physical and psychological well-being of young people, net of their home or neighborhood environments. Schools may therefore serve as essential early-life social contexts that contribute to the patterning of adult health risk. As such, future research should continue to examine the relationship between schools and health, paying particular attention to mechanisms through which school characteristics "get under the skin" to affect individual and population health inequality. The findings presented here suggest that early investments in improving the social, safety, and socioeconomic contexts of schools may reduce population health inequality from adolescence through adulthood.

Ethics

This study does not include human subjects, as the data used for the research are not obtained through intervention or interaction with living individuals or through identifiable private information. Neither intervention nor interaction with living individuals is a part of the proposed research. The proposed project involves the analysis of previously collected and de-identified data. As a result, ethics approval is not required for this study.

CRediT authorship contribution statement

Courtney E. Boen: Conceptualization, Methodology, Formal analysis, Writing - original draft. **Karen Kozlowski:** Conceptualization, Methodology, Writing - original draft. **Karolyn D. Tyson:** Conceptualization, Methodology, Writing - original draft.

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