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Academic disparities and health: How gender-based disparities in schools relate to boys' and girls' health

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ABSTRACT

Rationale: Recent research reveals that, although girls encounter some barriers in school (e.g., in science and math), on balance, boys perform worse academically. Moreover, other research has identified a correlation between exposure to a context characterized by large disparities in performance or resources and a range of negative outcomes, including negative health and well-being, among members of lower status groups.
Objective: Building on these literatures, the present research tests the relationship between gender disparities in academic performance within a school and students' health outcomes. Specifically, we investigated whether boys had worse health when they attended schools where there was a greater disparity between boys' and girls' academic performance.
Method: We tested this hypothesis in two different samples with different health outcomes. In a sample of healthy eighth graders (Study 1; 159 girls and 81 boys), we assessed two indices of metabolic syndrome, and in a sample of children with asthma (Study 2; 122 girls and 153 boys), we assessed immune function (Th1 and Th2 cytokine production) and self-reported symptoms. Participants in both samples also reported the name of the school that they attended so that we could access publicly available information about the percentage of girls and the percentage of boys in each school who met expectations for their grade level on standardized tests.
Results: In both samples, the greater the gap in a school between the percentage of girls and the percentage of boys who met expectations for their grade level on standardized tests, the worse boys' health. This pattern did not emerge among girls.
Conclusion: Results thus highlight the negative health correlates of academic disparities among members of lower-performing groups.

1. Introduction

Research has identified relationships between the nature of children's school environments and children's health outcomes. For example, in schools with respectful, supportive climates, middle and high school students behave in healthier ways and have lower rates of obesity (LaRusso et al., 2008; Richmond et al., 2014). Notably, however, even within the same school, students vary in how they experience the environment. For example, from elementary school to college, the extent to which students are supported and seen as having the potential to succeed varies with their race, gender, and other identities (Bigler and Liben, 2007; Crocker et al., 1998). In turn, children's sense of whether they are respected at school and seen as intelligent is associated with health (Goodman et al., 2003). The current research investigates how a novel aspect of the school environment—the disparity between girls' and boys' academic outcomes—relates to students' health. We focus on two sets of health outcomes. The first study focuses on early indicators of risk for cardiovascular disease, which is the leading cause of death in the United States (Centers for Disease Control and Prevention, 2017; Morrison, Friedman, Gray-McGuire, 2007). The second study focuses on immune function and clinical outcomes among children with asthma, the most common chronic condition in childhood (Bloom et al., 2012).

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1.1. Gender and school experiences

In a variety of ways, boys and girls have different school experiences and outcomes throughout their school years. Although boys are more often stereotyped as brilliant, sometimes tracked into more challenging classes and disciplines, overrepresented at the highest levels of achievement, and slightly more likely to pursue advanced professional degrees (Bian et al., 2017; Cheryan et al., 2015; Hedges and Nowell, 1995), in other ways they do not fare as well as girls. Teachers view boys as less motivated and more disruptive than girls, and discipline boys more frequently (Autor et al., 2016; Heyder and Kessels, 2015). In fact, on average, the evidence suggests that boys do less well than girls in school. One meta-analysis of studies from elementary to college-age students shows that, across all school subjects, girls earn higher grades than boys (Voyer and Voyer, 2014). This occurs, in part, because boys are overrepresented at the lower end of the academic distribution. For example, they are less likely than girls to meet kindergarten readiness standards and to graduate from high school on time (Autor et al., 2016). They also score lower on standardized tests (Autor et al., 2016). In elementary and middle school, boys' standardized test scores indicate that they are less likely than girls to meet expectations for their grade level in language arts and about equally likely to do so in mathematics (Reardon et al., 2018). Consequently, fewer boys than girls meet grade level expectations overall (i.e., across all subjects). The present work examines how this type of disparity—the extent to which more girls than boys meet or exceed grade level expectations on standardized tests—relates to outcomes a previously unexamined domain—physical health in youth.

1.2. Correlates of exposure to academic disparities

As early as elementary school, children notice disparities in which groups are valued and expected to succeed at their school (Bigler and Liben, 2007). By age seven, boys and girls both believe that their teachers see boys as academically inferior to girls (Hartley and Sutton, 2013). These disparities have negative academic consequences for children in the lower-performing group. For example, research has shown that exposure to information about disparities between the academic performance of one's ingroup and another higher performing group undermines performance and promotes academic disengagement (Hartley and Sutton, 2013; Major and Schmader, 1998; Spencer et al., 1999; Keller and Dauenheimer, 2003). Such patterns emerge in students from elementary school to college.

The aforementioned effects even extend to groups that are not traditionally considered disadvantaged. For instance, although white men are considered relatively skilled at math, when white male college students are exposed to the message that Asians outperform whites academically, their performance on a math test declines (Aronson et al., 1999). With respect to gender, and specifically to boys' underperformance relative to girls, seven- and eight-year-old boys who are told that girls typically outperform boys on an upcoming test score lower on this test (Hartley and Sutton, 2013). Furthermore, boys are less motivated and skilled at reading when their teachers think girls read better than boys (Wolter et al., 2015).

1.3. Academic disparities and health

Literature on the effect of exposure to academic disparities has largely focused on academic outcomes, such as motivation and performance. However, there is reason to suggest that academic disparities between groups (e.g., racial/ethnic groups, genders, etc.) may also be associated with worse health outcomes. No research has focused specifically on the health correlates of academic disparities between boys and girls. However, one study on race found that African-American college students' blood pressure increased when they took a test that an experimenter implied would reveal racial disparities in academic ability (Blascovich et al., 2001). These results are consistent with the more general finding that the relative deprivation experienced by one's group is associated with worse health outcomes, above and beyond absolute resources or achievement (Odgers, 2015). For example, adolescents' relative position in the socioeconomic hierarchy of their school or the geographic region in which they live correlates with their self-reported physical symptoms, controlling for their family's absolute level of financial resources (Elgar et al., 2013). Researchers have theorized that among members of lower status groups, exposure to disparities in academic outcomes or resources creates a sense of threat and vigilance that others cannot be trusted, and makes the experience of that environment more stressful, with accompanying detrimental effects on health and well-being (Oishi et al., 2011; Odgers, 2015). Similar processes may develop in boys who attend schools with greater gender disparities in academic performance.

There are also other reasons, specific to gender, to believe that disparities in academic performance between boys and girls in a school might be relevant to health. The underperformance of boys relative to girls has been linked to school classroom cultures where teachers and students see adolescent boys as having traditionally masculine traits, such as the tendency to be disruptive, aggressive, and stoic (Heyder and Kessels, 2015; Santos et al., 2013). These masculine stereotypes are themselves linked to less healthy behavior among men and boys, including a lower likelihood of seeking out healthcare when necessary (Himmelstein and Sanchez, 2016).

Building on these literatures, we propose that when boys attend schools where there is a greater disparity between boys' and girls' academic performance, boys will have worse health. We focus on health outcomes related to two common chronic diseases: cardiovascular disease and asthma. Cardiovascular disease is the most common cause of death in the United States (Centers for Disease Control and Prevention, 2017). Furthermore, risk factors for cardiovascular disease begin to develop in childhood, with children in negative psychosocial environments exhibiting higher risk (Morrison et al., 2007; Tomayo, Herder and Rathman, 2010). We assess cardiovascular disease risk using metabolic syndrome, a cluster of signs that can be assessed in youth and have been prospectively linked to the development of cardiovascular disease in adulthood (Morrison et al., 2007). In addition, asthma is the most common chronic condition in childhood and can be exacerbated by stressful psychosocial environments (Chen and Miller, 2007). Hence, we also present findings from a second sample focused on asthma outcomes in youth.

1.4. Current research

The current research explores the relationship between the percentage of girls and boys who met or exceeded grade level expectations on standardized tests in their school and the health outcomes of children in that school. This research question was tested across two different samples. Consistent with other research (Autor et al., 2016; Reardon et al., 2018), in nearly every school in both samples (87.6% in Study 1, 93.3% in Study 2), more girls than boys met this standard. We therefore hypothesized that among boys, but not girls, this gap would be associated with worse health. The hypothesis that this relationship would be specific to boys was based on previous research showing that disparities and disadvantage are typically more salient to and consequential for members of lower performing or lower status groups (Crocker et al., 1998). For example, exposure to a message about group differences in academic abilities impairs academic performance among members of the lower, but not higher, performing group (Keller and Dauenheimer, 2003; Spencer et al., 1999). We examined these hypotheses in two separate datasets for replication purposes. The first was a sample of healthy eighth graders (Study 1) with metabolic syndrome as the outcome, and the second was a sample of children with asthma (Study 2).

2. Study 1

2.1. Method

Participants. We first tested our hypothesis using data from a larger study on cardiovascular disease risk in adolescents. Participants from the Chicago area were recruited via advertisements in schools, public transportation, and mailings between 2015 and 2017. Children were eligible to participate if they were in eighth grade, free of acute or chronic medical conditions, and had no metal in their body (due to an fMRI component of the larger study that is not relevant here).

Of the 277 participants in the full sample, data on gender disparities in academic performance in schools (i.e., on the gap in percentage of boys and girls who met or exceeded grade level expectations on standardized tests given to public school students in Illinois) were available for 240 children. Of the 37 participants who were missing school data, most ($N = 27$) attended private school or were homeschooled. Other excluded participants attended Illinois public schools on which the state did not report data ($N = 8$), lived in another state ($N = 1$), or did not report their school name ($N = 1$). Thus, the sample had 159 girls and 81 boys ($M_{age} = 13.92$, $SD_{age} = .54$, 35.00% White, 40.42% Black/African-American, 0.42% Native Hawaiian/Pacific Islander, 33.33% Hispanic, 6.67% Asian, and 2.50% American Indian/Alaska Native). Participants provided written assent, and their parents provided written consent. The Northwestern University Institutional Review Board approved the protocol.

Procedure. Participants visited the lab, provided health measures, reported what school they attended, and reported demographic characteristics.

Gender Disparities in Academic Performance in Schools. For participants who attended public school in Illinois, data on the standardized test performance of eighth graders were obtained from the Illinois State Board of Education (available at https://illinoisreportcard.com). Relevant data were available for 97 schools (mean number of participants per school = 2.43, range = 1–15). For each school, the state provides data on the percentage of eighth grade girls and the percentage of eighth grade boys who met or exceeded grade level expectations on the Partnership for Assessment of Readiness for Colleges and Careers test during the 2015-16 school year. A disparity score was computed by subtracting the percentage of boys who met this standard from the percentage of girls who met it. The average gender disparity in academic performance in a school was 11.31 ($SD = 12.72$), indicating that the percentage of girls who met or exceeded grade level expectations on the test was, on average, 11.31 percentage points higher than that of boys. Consistent with other research (Autor et al., 2016; Reardon et al., 2018), in nearly all schools (87.6%) in this sample, more girls than boys met or exceeded expectations on standardized tests. Disparities in boys' and girls' performance in schools ranged from −18.75 (i.e., the percentage of girls in this school that met or exceeded grade level expectations was 18.75 percentage points *lower* than the percentage of boys who did so) to 55.56 (i.e., the percentage of girls in this school that met this standard was 55.56 percentage points *higher* than the percentage of boys who did so).

Metabolic syndrome. This study's measure of health was metabolic syndrome, a cluster of signs linked to the development of cardiovascular disease and type 2 diabetes (International Diabetes Federation, 2007; Morrison et al., 2007). According to the International Diabetes Federation (2007) definition, 11–16 year olds are diagnosed with metabolic syndrome if they have central adiposity (waist circumference ≥ 90th percentile for their demographic group) and at least two of the following: HDL cholesterol < 40 mg/dL, triglycerides ≥ 150 mg/dL, fasting glucose ≥ 100 mg/dL, and systolic blood pressure ≥ 130 mmHg and/or diastolic blood pressure ≥ 85 mmHg. Reflecting the fact that the sample was relatively young and healthy, only eight participants met the criteria for diagnosis. Therefore, we also calculated two continuous variables reflecting metabolic risk, which were the outcomes of interest in our analyses. The first was the number of components (out of five) for which participants met the clinical cutoff. Second, in acknowledgement of concerns about the validity of dichotomizing children into risk categories when variables are continuous (Goodman, 2008), we created a composite that was the sum of the z-scores of each component. For one participant from whom no blood was drawn, scores on the two available components were averaged and multiplied by five to be equivalent to other participants' values. Results are similar if this participant is excluded.

Waist circumference was measured at the narrowest point between the ribs and iliac crest. Resting systolic and diastolic blood pressure (SBP and DBP) were recorded continuously for 10 min while participants sat in a chair watching a nature video. We used a Continuous Non-invasive Arterial Pressure (CNAP) Monitor 500 (CNSystems, Graz, Austria). This monitor non-invasively records beat-to-beat arterial pressure using finger arterial sensors, which are automatically calibrated to brachial pressures via an upper arm-cuff. Blood pressure data were then scored using Mindware Technology software (BP 3.1, Gahanna, OH), which computed average SBP and DBP values across the 10-min period.

To assess HDL cholesterol, triglyceride, and fasting glucose levels, blood samples were collected via antecubital venipuncture. Participants fasted for 8 h prior to the blood draw. Samples were collected in serum separator tubes (Becton-Dickinson, Franklin Lakes, NJ), which were spun for 10 min at 1200 RCF 60–120 min after the blood draw. The serum was harvested, divided into aliquots, and stored at −20 °C until being sent to the NorthShore University HealthSystem laboratory (Evanston, IL) for analysis. HDL cholesterol and triglycerides were measured on a Roche/Hitachi cobas c701 analyzer. The average intra- and inter-assay coefficients of variation for these assays were below 1.6% and 2.4%, respectively. The assay's detection ranges are 8.85–885 mg/dL (triglycerides) and 3–120 mg/dL (HDL).

Covariates. At the child level, covariates included age, race/ethnicity (three variables that were not mutually exclusive: whether the child was white, African-American, and Hispanic [0 = no, 1 = yes for each]), family's savings (assets that their family could easily convert to liquid cash in an emergency, on a nine-point scale from “less than $500” to “$500,000 or higher,” reported by children's parents), and pubertal stage (Physical Development Scale; Petersen, Crockett, Richards and Boxer, 1988). At the school level, covariates included the percentage of students overall who met or exceeded grade level expectations on standardized tests; dummy-coded school level (elementary school: 0 = no, 1 = yes; junior high school: 0 = no, 1 = yes; high school was the reference group); percentage of students who were white, African-American, and Hispanic (three variables); percentage of the students who were “low income” (eligible for free/reduced price lunch, in alternative care, or receiving government assistance), and percentage of students who were English language learners. Values for eight participants who did not report their family's savings and four participants who did not complete the pubertal stage measure were mean imputed.

Analytic Approach. Because the data had participants nested within schools, we tested our hypotheses using Hierarchical Linear Modeling (HLM) 7 software (Raudenbush et al., 2011), with separate models for each outcome. Child gender (female = 0, male = 1) and the child-level covariates were entered at Level 1. At Level 2, the gender disparity in test scores and the school-level covariates were entered to predict the intercept and the child gender slope. Continuous predictor variables were grand mean centered, and dummy-coded variables were uncentered. The coefficient of interest was the cross-level interaction between child gender and the gender disparity in test scores in the school.

2.2. Results

Preliminary Analyses. Table 1 provides descriptive statistics for

Table 1
Descriptive statistics for Study 1 participants.

Variable	Boys		Girls		*p*
	N	Mean (SD) or %	*N*	Mean (*SD*) or %	
Age (years)	81	13.89 (0.50)	159	13.93 (0.56)	0.579
White (%)	81	33.33	159	35.85	0.699
Black (%)	81	40.74	159	40.25	0.942
Hispanic (%)	81	37.04	159	31.45	0.385
Pubertal stage (5 point scale)	79	3.14 (0.67)	157	4.08 (0.59)	< .001
Savings (9 point scale)	80	3.83 (2.55)	152	3.53 (2.44)	0.385
Waist circumference (in)	81	31.13 (5.19)	159	29.51 (4.98)	0.020
Systolic Blood Pressure (mmHg)	81	117.68 (10.13)	159	114.00 (9.50)	0.006
Diastolic Blood Pressure (mmHg)	81	69.24 (6.19)	159	68.57 (6.69)	0.452
Fasting glucose (mg/dL)	81	85.63 (7.23)	158	84.13 (8.20)	0.166
HDL cholesterol (mg/dL)	81	48.09 (12.35)	158	51.25 (11.15)	0.047
Trigylcerides (mg/dL)	81	71.80 (36.58)	158	77.14 (41.68)	0.330
Number of metabolic syndrome signs	81	0.62 (0.85)	159	0.47 (0.79)	0.187
Continuous metabolic syndrome composite	81	0.57 (2.87)	159	−0.33 (2.80)	0.021

the participants. Boys had higher waist circumferences, SBP, and continuous metabolic syndrome composite scores, while girls were at a higher pubertal stage and had higher HDL cholesterol levels. They did not differ on the other variables.

At the school level, greater test score disparities were associated with a smaller percentage of the student body being African-American, $r(96) = -.21$, $p = .042$. There were no relationships between the gender test score disparities in schools and the percentage of students overall who met or exceeded grade level expectations on standardized tests or other available demographic characteristics of the student body, *ps* > .14. See Supplementary Materials for details.

Primary Analyses. To address our hypotheses, we first tested how child gender, the school's gender disparity in academic performance, and their interaction were related to the number of clinically elevated metabolic syndrome signs. There was a marginal main effect of gender ($b = -0.681$, $SE = 0.356$, $p = .059$, 95% *CI* [-1.388, .026]), but not of schools' gender disparities in academic performance ($b = -0.003$, $SE = 0.005$, $p > .561$, 95% *CI* [-0.013, 0.007]). As Fig. 1 depicts, the expected interaction emerged ($b = 0.023$, $SE = 0.010$, $p = .029$, 95% *CI* [0.002, 0.043]). Specifically, among girls, there was no relationship between schools' gender disparities in academic performance and the number of metabolic syndrome signs ($b = -0.003$, $SE = 0.005$, $p > .561$, 95% *CI* [-0.013, 0.007]). However, boys in schools with greater gender disparities had more clinically elevated metabolic syndrome signs ($b = 0.020$, $SE = 0.009$, $p = .023$, 95% *CI* [0.003, 0.037]).

Next, we conducted the same analyses with the continuous metabolic syndrome composite as the outcome. There was no significant main effect of gender ($b = -0.375$, $SE = 0.846$, $p = .659$, 95% *CI* [-2.055, 1.306]) or of schools' gender disparities in academic performance ($b = -0.009$, $SE = 0.015$, $p = .533$, 95% *CI* [-0.038, 0.020]). However, again the predicted interaction emerged ($b = 0.078$, $SE = 0.035$, $p = .029$, 95% *CI* [0.008, 0.148]) and is depicted in Fig. 1. Specifically, among girls, there was no relationship between a school's gender disparity in academic performance and the metabolic syndrome composite ($b = -0.009$, $SE = 0.015$, $p = .533$, 95% *CI* [-0.038, 0.020]), but boys in schools with a greater gender disparity had higher metabolic syndrome scores ($b = 0.069$, $SE = 0.032$, $p = .034$, 95% *CI* [0.005, 0.132]). See the Supplementary Materials online for additional details on these analyses.

2.3. Supplementary analyses

Individual School Experiences. One alternative explanation for these results is that boys in schools with greater gender disparities in academic performance have lower academic performances themselves or are disciplined more frequently. It is possible that this experience, rather than the inequality, is associated with boys' health outcomes. To address this question, we repeated our analyses controlling for participants' school subscale scores on the Life Stress Interview (Hammen, 1991). In this open-ended interview, participants answer questions about their academic achievement and disciplinary record. Trained interviewers give each participant a rating from one (all As, no disciplinary problems) to five (failure in multiple subjects, expulsion, and/or dropped out of school). When this score is added as a covariate, both

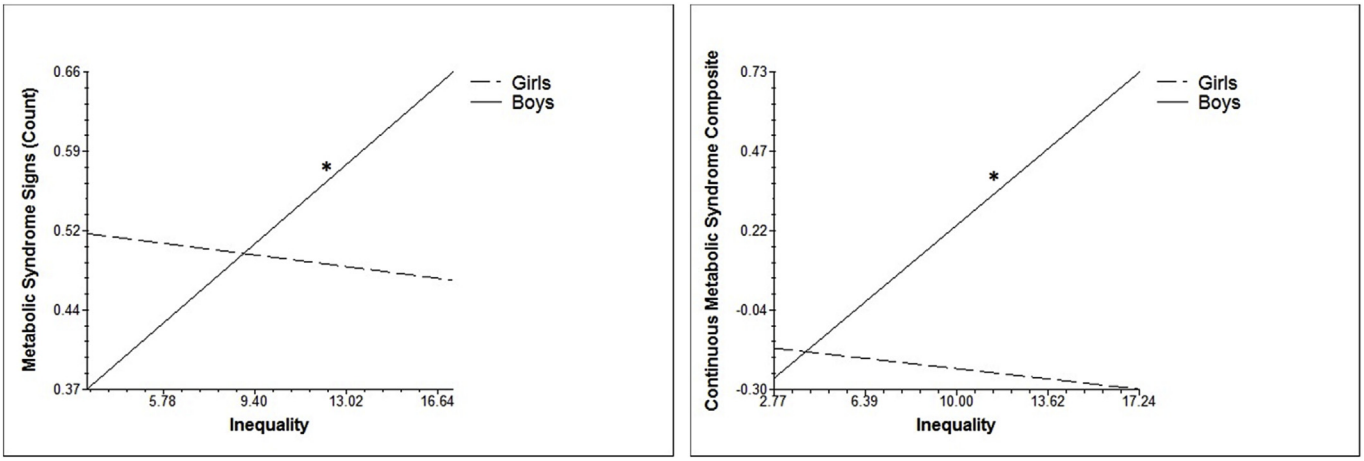

Fig. 1. Covariate adjusted estimates of metabolic syndrome signs (left panel) and continuous metabolic syndrome composite scores (right panel) among boys and girls as a function of the academic gender disparities in their schools in Study 1. *$p \leq .05$.

interactions remained significant ($p < .034$).

Exposure to Violence. Another possible alternative explanation for the results observed here is that schools with greater gender disparities in academic outcomes are in areas with higher violent crime rates and that exposure to violence has a disproportionate effect on boys' health and academic outcomes. To address this possibility, we repeated our analyses, controlling for children's self-reported exposure to violence (i.e., how frequently in the past year they experienced events such as being present when someone was shot or killed or had a friend violently hurt or killed; Thomson et al., 2002). When this violence measure was included as a covariate, both interactions remained significant ($p < .026$).

3. Study 2

Study 2 used a sample of children diagnosed with asthma to replicate these findings and explore their clinical relevance and functional implications.

3.1. Method

Participants. Data were taken from a larger study that included 308 children ages eight to seventeen. All of these children had been diagnosed with asthma by a physician. Families were recruited through one health care system (NorthShore University HealthSystem) and one federally qualified health center (Erie Family Health Center) in the Chicago area between 2013 and 2016. Data on schools' gender disparities in test scores were available for 275 children. Of the 33 participants missing school data, 28 attended private school, two attended an Illinois public school for which the state did not report data, and three lived in another state. This left a sample of 122 girls and 153 boys ($M_{age} = 12.93$, $SD_{age} = 2.52$, 57.82% White, 26.55% Black/African-American, 1.09% Native Hawaiian/Pacific Islander, 16.73% Hispanic, 13.09% Asian, and 0.36% American Indian/Alaska Native). Participants provided written assent, and their parents provided written consent. The Northwestern University, NorthShore, and Erie Institutional Review Boards approved the protocol.

Procedure. Participants visited the lab, provided a blood sample, and reported on their school name, their demographic characteristics, and their asthma medications and symptoms.

School Disparities. We used data from the Illinois State Board of Education to compute the same measure of gender disparities in test performance as in Study 1. However, because participants in Study 2 spanned a broader age (and thus grade level) range than in Study 1, we computed this disparity score using data on standardized test performance among boys and girls in the school overall, rather than in 8th grade specifically. Relevant data were available for 134 schools (mean number of students in the sample per school = 2.07, range = 1–21). The average gender disparity in test scores was 8.09 ($SD = 4.95$), indicating the difference between the percentage of girls and the percentage of boys who met or exceeded grade level expectations on standardized tests was 8.09 percentage points higher than that of boys. As in Study 1, and consistent with other research (Autor et al., 2016; Reardon et al., 2018), in nearly all schools (93.3%) in this sample, more girls than boys met or exceeded expectations on standardized tests. Schools' gender disparities in academic performance ranged from −3.82 (i.e., the percentage of girls in this school who met or exceeded grade level expectations was 3.82 percentage points *lower* than the percentage of boys who did so) to 21.83 (i.e., the percentage of girls in this school who met this standard was 21.83 percentage points *higher* than the percentage of boys who did so).

Cytokine Production. Asthma is a disease characterized by inflammation and obstruction of the airways, which typically result from excessive immune responses to triggers, such as allergens, pollutants, and pathogens (Busse and Lemanske, 2001). As part of this immune response, T-helper (Th) lymphocytes release chemical messengers called cytokines (Busse and Lemanske, 2001). Typically, Th2 cytokines, such as interleukin (IL)-4, IL-5, IL-10, and IL-13, are released in response to allergens, whereas Th1 cytokines, such as IFN-γ and IL-2, are released in response to pathogens. Both can mobilize immune cells into the lungs, where they provoke mucus production, limit airflow, and trigger wheezing, coughing, and shortness of breath. To model these processes, we exposed children's immune cells *in vitro* to substances that stimulate Th1 and Th2 cytokine production.

We measured stimulated cytokine secretion by peripheral blood mononuclear cells (PBMCs). Antecubital blood was drawn into BD Cell Preparation Tubes (Becton Dickinson, Franklin Lakes, NJ) containing sodium heparin. PBMCs were isolated by density-gradient centrifugation according to the manufacturer's instructions and dispensed into 12-well culture plates in the presence of different mitogen configurations. To measure Th1 and Th2 cytokine production after nonspecific stimulation, we incubated 0.5 x 10^6 PBMCs with 25 ng/mL of PMA (Sigma-Aldrich, St. Louis, MO) + 1 μg/mL of INO (Sigma-Aldrich, St. Louis, MO) for 24 h at 37 °C in 5% CO2. An unstimulated well with the same number of PBMCs but no mitogen was cultured under the same conditions. After the incubation, supernatants were harvested by centrifugation and frozen at −80 °C until assayed in batch via electrochemiluminescence on a SECTOR Imager 2400A (Meso Scale Discovery, MSD, Rockville, MD). We used MSD's Human Th1/Th2 7-Plex Tissue Culture Kit, which measures both Th2 (IL-4, IL-5, IL-10, and IL-13) and Th1 (IFN-γ and IL-2) cytokines in parallel. Mean interassay coefficients of variation ranged from 1.97% to 4.14%. Cytokine responses were quantified by subtracting values in the unstimulated wells from those in the PMA/INO wells. Principal components analyses indicated that the cytokine values loaded onto two factors, which reflected Th1 and Th2 cytokines. Thus, cytokine levels were standardized and averaged into two composites, indexing Th1 and Th2 cytokines (correlated with each other, $r = .484$, $p < .001$).

Glucocorticoid Sensitivity. Glucocorticoids (cortisol in humans) play a key role in regulating cytokine responses. At high concentrations, glucocorticoids inhibit many immune responses, and hence, synthetic versions of cortisol are used as medications to treat asthma. People vary in their immune cells' sensitivity to cortisol signaling, and factors such as chronic stress are associated with decreased sensitivity (Chen and Miller, 2007). Decreased glucocorticoid sensitivity may result in less effective inhibition of asthma-related inflammation and in bronchoconstriction in the airways. To model this process, we repeated the cytokine production procedure described above, this time measuring cytokine production after exposing participants' immune cells *in vitro* to mitogens and cortisol.

Specifically, 0.5 x 10^6 PBMCs were coincubated with 25 ng/mL of PMA, 1 μg/mL INO and 1.38 x10−6 mol/L hydrocortisone (Sigma-Aldrich, St. Louis, MO) for 24 h at 37 °C in 5% CO_2. An unstimulated well was also included on the plate. Supernatants were assayed in batch using the MSD Th1/Th2 kit, as previously shown, and unstimulated values were subtracted out before analysis. Again, principal components analyses indicated that the cytokine values loaded onto two factors, which reflected Th1 and Th2 cytokines. Thus, cytokine levels were standardized and averaged into two composites, indexing Th1 cytokines and Th2 cytokines (correlated with each other $r = .452$, $p < .001$). At the dose used, cortisol suppresses production of Th1 and Th2 cytokines, so higher cytokine levels indicate less responsiveness to glucocorticoids.

Asthma Symptoms. Asthma symptoms were measured using the symptoms subscale of the Pediatric Asthma Quality of Life Scale (Juniper et al., 1996). Children rated 10 items that assessed frequency of cough, wheezing, and waking at night due to asthma symptoms ($\alpha = .88$). Higher scores indicate more symptoms.

Covariates. At the child level, analyses controlled for age, family's savings (using the same 9-point scale as Study 1), ethnicity (0 = not white, 1 = white), asthma medication use (two variables: beta-agonist use and inhaled corticosteroid use in the past week; 0 = no, 1 = yes),

asthma severity (determined from the National Asthma Education and Prevention Program/Expert Panel Report 2 guidelines based on the higher of symptom frequency and medication use; Bacharier et al., 2004), and pubertal stage (Physical Development Scale; Petersen et al., 1988). The asthma severity covariate was not included in the analyses predicting the asthma symptoms outcome, due to conceptual overlap with this dependent measure. At the school level, analyses controlled for the percentage of students overall who met or exceeded grade level expectations on standardized tests; school level (dummy-coded, as in Study 1); and the percentage of students who were white, "low income," and English language learners (see Study 1 for details). Scores were mean imputed for six participants who did not report their family's savings and three participants who did not report their pubertal stage.

Analytic Approach. We again tested our hypotheses using HLM 7 software (Raudenbush et al., 2011), with separate models for each outcome. The variables were entered as in Study 1, with the addition of the asthma-relevant covariates of medication use (uncentered) and severity (grand mean centered) at Level 1.

3.2. Results

Preliminary Analyses. Table 2 summarizes descriptive statistics for the participants. Girls were at a higher pubertal stage and reported more asthma symptoms than boys. At the school level, a greater gender disparity in academic performance in a school was associated with a marginally smaller percentage of the student body being English language learners, $r(133) = -.15$, $p = .082$. There were no relationships between the gender disparity in academic performance and the percentage of students overall who met or exceeded grade level expectations on standardized tests or other available demographic characteristics of the student body, $ps > .14$. See the Supplementary Materials for details.

3.3. Primary Analyses

Cytokine Production. To address the hypotheses, we first tested how child gender, the school's gender disparity in academic performance, and their interaction related to Th1 and Th2 cytokine production. With respect to Th1 cytokine production, there was no significant main effect of gender ($b = 0.082$, $SE = 0.292$, $p = .779$, 95% CI [-0.497, 0.661]) or the gender disparity in academic performance in a school ($b = -0.016$, $SE = 0.016$, $p = .335$, 95% CI [-0.048, 0.016]). Nonetheless, the predicted interaction emerged ($b = 0.046$, $SE = 0.021$, $p = .032$, 95% CI [0.004, 0.087]). Among girls, there was no relationship between schools' gender disparities in academic performance and Th1 cytokine production ($b = -0.016$, $SE = 0.016$, $p = .335$, 95% CI [-0.048, 0.016]). In contrast, boys who attended schools with a greater gender disparity in academic performance had greater Th1 cytokine production ($b = 0.030$, $SE = 0.014$, $p = .029$, 95% CI [0.003, 0.057]).

With respect to Th2 cytokine production, there was no significant main effect of gender ($b = -0.020$, $SE = 0.314$, $p = .948$, 95% CI [-0.644, 0.602]), but a greater gender disparity in academic performance in a school was associated with marginally lower Th2 cytokine production ($b = -0.027$, $SE = 0.016$, $p = .102$, 95% CI [-.059, .005]). As Fig. 2 shows, the predicted significant interaction also emerged ($b = 0.067$, $SE = 0.024$, $p = .006$, 95% CI [0.019, 0.114]). Among girls, a school's gender disparity in academic performance was associated with marginally lower Th2 cytokine production ($b = -0.027$, $SE = 0.016$, $p = .102$, 95% CI [-0.059, 0.005]), but among boys, the gender disparity was associated with greater Th2 cytokine production ($b = 0.040$, $SE = 0.016$, $p = .016$, 95% CI [0.007, 0.072]).

Glucocorticoid Sensitivity. Turning to glucocorticoid sensitivity, there was no significant main effect of child gender ($b = -0.001$, $SE = 0.242$, $p = .995$, 95% CI [-0.482, 0.479]) or schools' gender disparities in academic performance ($b = -0.009$, $SE = 0.013$, $p = .513$, 95% CI [-0.034, 0.017]) on Th1 cytokine production. However, the expected interaction emerged ($b = 0.036$, $SE = 0.018$, $p = 0.048$, 95% CI [0.0003, 0.072]). Specifically, among girls, the gender disparity in academic performance in a school was unrelated to Th1 cytokine production ($b = -0.009$, $SE = 0.013$, $p = .513$, 95% CI [-0.034, 0.017]). However, boys who attended schools with a greater gender disparity in academic performance had greater Th1 cytokine production ($b = 0.028$, $SE = 0.013$, $p = .040$, 95% CI [0.001, 0.054]).

With respect to Th2 cytokine production in response to stimulation with PMA/INO + cortisol, there was no significant main effect of child gender ($b = -0.367$, $SE = 0.311$, $p = .241$, 95% CI [-0.984, 0.250]), but the gender disparity in academic performance in a school was associated with marginally lower cytokine production ($b = -0.029$, $SE = 0.016$, $p = .068$, 95% CI [-0.060, 0.002]). In addition, the predicted interaction emerged ($b = 0.052$, $SE = 0.019$, $p = .008$, 95% CI [0.014, 0.091]). Specifically, a school's gender disparity in academic performance was associated with marginally lower Th2 cytokine production among girls ($b = -0.028$, $SE = 0.016$, $p = .068$, 95% CI [-0.060, 0.002]) but marginally greater Th2 cytokine production among boys ($b = 0.024$, $SE = 0.013$, $p = .072$, 95% CI [-0.002, 0.049]).

Asthma Symptoms. Finally, we repeated these analyses for asthma symptoms. There was a main effect of gender, with boys reporting fewer symptoms ($b = -0.926$, $SE = 0.314$, $p = .004$, 95% CI [-1.547, -0.305]), but not of the gender disparity in academic performance in a school ($b = -0.004$, $SE = 0.018$, $p = .836$, 95% CI [-0.040, 0.033]). Again, the predicted interaction emerged ($b = 0.053$, $SE = 0.025$, $p = .036$, 95% CI [0.004, 0.103]) and is depicted in Fig. 2. The gender

Table 2
Descriptive statistics for Study 2 participants.

Variable	Boys		Girls		p
	N	Mean (SD) or %	*N*	Mean (*SD*) or %	
Age (years)	153	12.78 (2.46)	122	13.11 (2.59)	0.293
White (%)	153	58.17	122	57.38	0.895
Pubertal stage (on 5 point scale)	152	2.89 (1.01)	120	3.68 (1.12)	< .001
Savings (on 9 point scale)	152	5.06 (2.82)	117	4.75 (2.62)	0.362
Use inhaled corticosteroids (%)	153	73.20	122	72.13	0.843
Use beta-agonist (%)	153	97.39	122	97.54	0.935
Asthma severity (on 4 point scale)	153	2.41 (0.92)	122	2.43 (0.93)	0.897
Cytokine Production					
Stimulation with PMA/INO: Th 1 cytokines	141	0.01 (0.90)	102	0.002 (0.94)	0.975
Stimulation with PMA/INO: Th 2 cytokines	141	0.04 (0.94)	102	−0.08 (0.96)	0.324
Glucocorticoid Sensitivity					
Stimulation with PMA/INO + Cort: Th 1 cytokines	131	−0.01 (0.94)	93	0.03 (0.85)	0.742
Stimulation with PMA/INO + Cort: Th 2 cytokines	131	−0.02 (0.89)	93	0.03 (0.94)	0.705
Asthma Quality of Life - Symptoms	153	2.96 (1.14)	122	3.30 (1.20)	0.016

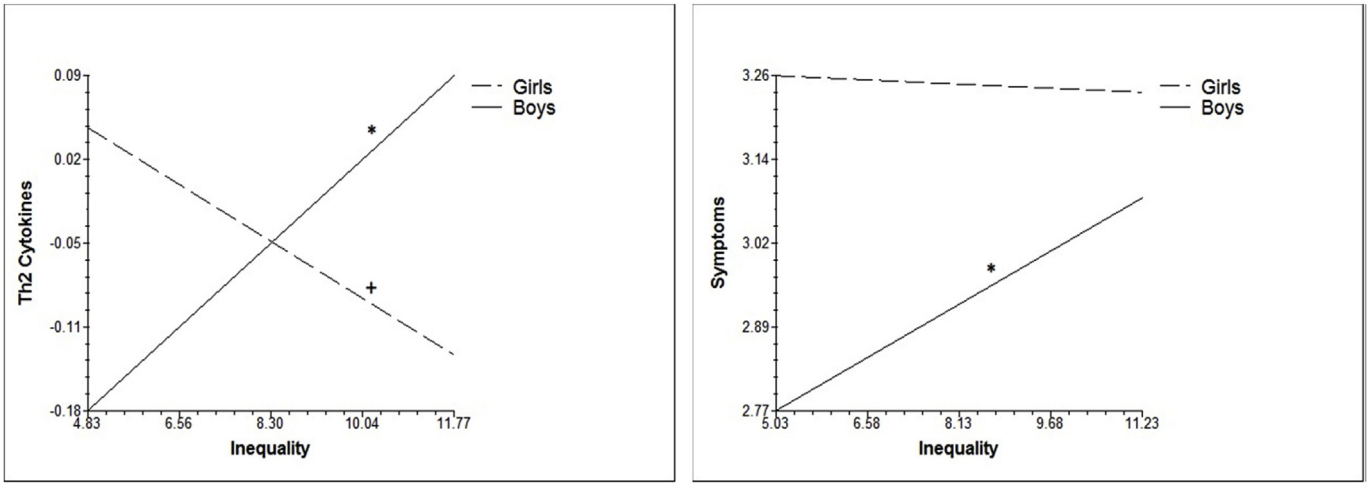

Fig. 2. Covariate adjusted estimates of Th2 cytokine production in response to stimulation of PBMCs with PMA/INO (left panel) and asthma symptoms (right panel) among boys and girls as a function of the academic gender disparities in their schools in Study 2. $^{*}p \leq .05$, $^{+}p \leq .10$.

disparity in academic performance in a school was unrelated to symptoms among girls ($b = -0.004$, $SE = 0.019$, $p = .836$, 95% CI [-0.040, 0.033]), but among boys, the greater the disparity, the more frequent their asthma symptoms ($b = 0.049$, $SE = 0.018$, $p = .008$, 95% CI [0.013, 0.086]). See the Supplementary Materials online for additional details on these analyses.

3.4. Supplementary analyses

Individual School Experiences. As in Study 1, we repeated our analyses with the school subscale of the Life Stress Interview (Hammen, 1991) included as a covariate. The interaction for the production of Th1 cytokines after exposure to PMA and INO and cortisol became marginal ($p = .055$), and the rest remained significant ($ps < .049$).

Exposure to Violence. Also as in Study 1, we repeated our analyses with exposure to violence (Thomson et al., 2002) as a covariate. All interactions remained significant ($ps < .048$).

4. Discussion

Across two studies, we show parallel patterns whereby boys who attend schools where there is a larger gender gap in academic performance have worse health. This disparity in schools is associated with cardiovascular risk in healthy adolescents (see Fig. 1) and immune and clinical outcomes in children with asthma (see Fig. 2). These findings extend previous research on the relationship between disparities and health, which has largely focused on socioeconomic disparities (e.g., income gaps, rather than academic outcomes) at a larger scale (e.g., in the state or country, rather than school; Wilkinson and Picket, 2006). The current research also extends previous work on the school context and child health outcomes, which had focused more on health behaviors than on physical health markers (e.g., LaRusso et al., 2008).

Notably, while the gender-based disparity in academic performance in a school was related to boys' health across multiple health measures in both studies, the results for glucocorticoid sensitivity in Study 2 were weaker. Specifically, the relationship between a schools' gender disparity and boys' Th2 cytokine production in response to mitogen stimulation in the presence of cortisol (i.e., a measure of glucocorticoid sensitivity) was marginal, whereas the relationship between a school's gender disparity and boys' health was significant for all other outcomes. This raises the possibility that glucocorticoids may help to regulate children's immune functioning in a way that attenuates the association between gender-based disparities in academic outcomes in a school and boys' immune functioning.

4.1. Possible mechanisms

There are multiple explanations for why a gap between girls' and boys' academic performance in a school might relate to boys' health. Psychologically, just as socioeconomic disparities undermine social trust among those with lower status (Oishi et al., 2011), disparities in boys' and girls' academic performance in a school could undermine boys' trust that teachers and classmates will judge their abilities fairly or support them academically. Boys might also feel a reduced sense of school belonging or see themselves as lower status in the school, particularly with respect to academic performance. All of these concerns are associated with worse health (Goodman et al., 2003; Elovainio et al., 2002; Hale et al., 2005; Schwartz, 2017). Behaviorally, given that disparities in economic resources increase risk-taking (Payne et al., 2017), academic disparities might prompt riskier and unhealthier behavior in boys. Finally, biologically, stress from attending schools with greater gender disparities in academic performance might increase HPA-axis dysregulation among boys, in turn giving rise to metabolic syndrome and poor immune functioning in children with asthma.

4.2. Strengths and limitations

These studies have both strengths and limitations, some of which stem from the sample. The present analyses were conducted using data from two larger studies that examined a variety of different hypotheses related to the psychosocial correlates of health disparities. The participants thus reflect those from racially and socioeconomically diverse backgrounds who were willing and able to visit the lab and undergo a blood draw. The samples were not recruited with the initial aim of testing hypotheses related to the school context. Rather, we took advantage of existing samples of a large number of children who attended many different schools. As a result, we were able to find evidence for our hypotheses in large, diverse samples of participants looking at multiple health outcomes. We consider the present work an initial test of the novel hypothesis that inequality in the school environment has any relationship to students' health but recognize that follow-up research using samples collected with this hypothesis in mind would be necessary. Because schools and participants were not systematically sampled, in many cases, we had only one or two participants in our sample per school. Recent work suggests that the number of participants per group has little effect on multilevel estimates when the number of groups is large, or if anything fails to detect group level effects (Theall et al., 2011). Nevertheless, it would be important to replicate the present results with a larger sample of participants per school.

Another limitation is the correlational design, which prevents us from knowing the directionality or causality of the relationships. We have theorized that gender disparities in academic performance harm boys' health. An alternative explanation is that less healthy boys underperform academically or are absent more often, contributing to academic disparities. Although we cannot rule out this possibility, we suggest that it is unlikely to fully explain our results. First, the argument that health disparities create academic disparities assumes that boys have worse health than girls in schools where boys' test scores are lower than girls'. Our data do not support this claim. Although girls outperform boys in nearly all the schools in our samples, we did not find any main effects of gender on health in which boys were worse off than girls. Analyses looking at most outcomes yielded no main effect of gender, and girls had marginally more clinically elevated metabolic syndrome signs than boys in Study 1 and worse asthma symptoms than boys in Study 2. Furthermore, the argument that gender differences in health give rise to gender differences in academic outcomes may explain why individual boys in our sample with worse health attended schools with greater academic disparities, but it cannot explain why individual girls' health was often unrelated to academic disparities in their schools. Finally, it seems unlikely that the health of the individual boys assessed in our sample affected test scores across whole schools because there were just a few students in our sample from any given school.

It is also possible that a third variable contributes to both gender disparities in test scores and to boys' worse health. One possible third variable is parents' beliefs. For example, parents who believe that boys are disruptive, aggressive, and stoic might be more likely to send their boys to schools where teachers and students also hold such beliefs (i.e., schools where boys underperform relative to girls; Santos et al., 2013). These same parents might also treat their sons in ways that are detrimental to their sons' health (e.g., provide less emotional support to their sons).

A final limitation is that there were half as many boys as girls in the Study 1 sample, which reflects the fact that girls were more likely to respond to advertisements and express an interest in participating. Although Study 2, which included more boys than girls, helped to address this limitation, it is possible that the boys in Study 1 were atypical in some respect. Hence, future studies on this topic in other samples would be useful.

4.3. Future directions

The current research has established that there is a relationship between gender disparities in academic outcomes and boys' health. An important next step will be to test the causal relationships between these variables, especially before drawing policy-relevant conclusions about how to improve boys' health. To do this, researchers might manipulate whether students are told (or not) about the extent of these disparities in their school and measure outcomes such as blood pressure in the lab.

In addition, the current analyses could be extended by examining what other characteristics of schools correlate with gender disparities in test scores, with particular attention to understanding what policies or practices in a school might contribute to test score disparities and be relevant to health. In a recent study of school districts across the country, Reardon et al. (2018) found that while a small portion of the variance in gaps between girls' and boys' standardized test score performance in districts was explained by student demographic characteristics (i.e., socioeconomic composition of district residents), over 80% of the variance in gender disparities in test scores remained unexplained. We propose that future research could test how gender disparities in test scores in a school and students' health outcomes vary as a function of other factors, such as disparities in grades or teachers' differential beliefs about and treatment of boys and girls. It might be especially fruitful to simultaneously assess many of these additional factors to understand how they relate to each other. We speculate that schools with greater gender test score disparities are those where teachers treat boys and girls differently or boys and girls earn different grades and that students' health is affected when they observe these differences. Future research could test such possibilities.

Future research might also explore whether equivalent patterns emerge among girls in academic contexts where girls are disadvantaged and among adults in professional domains characterized by gender disparities. For example, women might have worse health when they pursue science, math, engineering, or technology (STEM) degrees at universities with large gender disparities in STEM grades or course enrollments. Outside of the school context, both men and women might have worse health when they work in professional fields where people of their gender lack opportunities to succeed. Moving beyond gender to other demographic characteristics, future research could explore whether racial disparities in academic or professional outcomes in a school or workplace are associated with health outcomes among members of lower status racial groups.

Finally, future research could test whether the relationships observed here extend to other health outcomes. We suggest that our patterns will replicate with other health outcomes because our findings emerged across multiple health measures that included both biomarkers and self-reported symptoms and across markers for two different diseases (i.e., cardiovascular disease and asthma). Given that we observed effects for inflammatory processes (i.e., cytokine production), we suggest that these patterns might be especially likely to replicate among people with other diseases linked to inflammation (e.g., rheumatoid arthritis, lupus, or other autoimmune disorders).

5. Conclusion

There has been persistent interest in how gender affects children's experiences in schools, and increasing evidence suggests that boys often perform worse than girls academically (Voyer and Voyer, 2014). The present work highlights one previously unrecognized correlate of these disparities. Although additional experimental work is necessary, the present work raises the possibility that attending schools where male classmates underperform relative to their female classmates may put boys' health at risk. If future work confirms that this is the case, building such more equitable, inclusive educational environments has the potential to improve not only academic outcomes among students from lower performing groups, but possibly also their health as well.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.socscimed.2019.02.036.

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