Coloring the "Boys Will Be Boys" Chronicle: Race, Gender, and Behavior Problems across Two Decades

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Keywords: Race, Education, Gender, Family, Developmental Psychology

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Abstract

ADHD diagnoses and school suspensions have increased ten- and two-fold in the U.S. since the 1970s. Boys' higher baseline levels of ADHD symptoms and suspension have led to growing gender gaps in absolute prevalence over three decades. Gender gaps are largest among black children. Despite implications for black boys' growing childhood behavioral disadvantage, neither the extent nor the origins of their growing disadvantage are understood. This study helps fill this gap by leveraging behavioral scales that consistently measure across two decades the self-regulation problems and social problems correlated with ADHD symptoms and school suspension. Conditional quantile regression results show that growing gender gaps in ADHD symptomatology and school suspension – most widespread among black children – are consistent with changes in mothers' reports of worsening self-regulation problems and social problems among even the "best-behaved" black boys, not just those with near-clinical levels of behavior problems. Neither gender differences in exposure nor response to salient, racially-patterned changes in families and health explain black boys' growing disadvantage. Findings carry significant consequences for black males' future rates of delinquency, school drop-out, unemployment, and incarceration, which may result from heightened scrutiny and policing of even the best-behaved black boys' behaviors beginning in early childhood.

1 Race and a Growing Gender Gap in Children's Behavior Problems

Both medical and educational researchers have carefully documented increases in gender disparities in childhood prevalence of attention deficit and hyperactivity disorder (ADHD) diagnosis and in out-of-school school suspensions since the 1980s (Bertrand and Pan 2011; Mendez 2003; Olfson et al. 2003). ADHD is the most commonly-diagnosed mental health disorder among American children (Froehlich et al. 2007). The *Diagnostic and Statistical Manual of Mental Disorders* (DSM) is used to diagnose ADHD based on clinician assessment of "a persistent and frequent pattern of developmentally atypical inattention and/or hyperactivity-impulsivity that impairs functioning in at least two settings (e.g., school and home)," with onset by age seven (American Psychiatric Association Task Force on DESM-IV 1994: 85-93). School suspension is a disciplinary tactic that entails removing students from school for up to 10 school days on the basis of disruptive social problems and conduct problems (Skiba and Peterson 2000). Although distinct outcomes, ADHD and school suspension are both correlated with a number of the same root symptoms: inattention, hyperactivity, impulsivity, and social/conduct disorder.

Prevalence of diagnosed ADHD and school suspensions also share a number of common patterns at the population level. Prevalence has increased by roughly 400% (for diagnosed ADHD) and 100% (for school suspensions) for both boys and girls over the past two or three decades, as shown in Panel A of Figure 1 (Bertrand and Pan 2011; Cuffe et al. 2005; Mendez 2003; Olfson et al. 2003). Today, 10%-11% of American children are diagnosed with ADHD; the same percentage of children are ever suspended by the end of secondary school (Alvarez 2013; Lanphear 2012; National Center for Education Statistics 2007).

Because baseline prevalence of diagnosed ADHD and suspension are at least two times greater among boys than girls, the "gender gap" in absolute prevalence has roughly doubled. In the case of ADHD diagnosis, child age and type of sample (e.g., clinic-referred or community-referred) are predictive of the magnitude of the gender gap: boys are between two and nine times more likely than girls to be seen, diagnosed, and treated for ADHD (Biederman et al. 2002; Morgan et al. 2013). Changes in the DSM criteria for ADHD diagnosis that were made in the early 1990s resulted in higher ADHD diagnosis rates for

boys, but not for girls (Biederman et al. 2002).[1] Once diagnosed, boys are more likely than girls to be treated (Rowland et al. 2002).

[FIGURE 1 ABOUT HERE]

Strikingly, rising prevalence of ADHD symptomatology and school suspension is most pronounced among black children (Epstein et al. 2005; KewalRamani 2007; Losen and Skiba 2010; Wallace Jr. et al. 2008).[2] Because black boys have historically had higher rates of ADHD symptomatology than black girls, rising prevalence among black children has led to the largest growth of the gender gap in clinical levels of ADHD behaviors among blacks compared to Hispanics, whites, and Asians. The magnitudes of the gender gaps by race as of 2001 are shown in Panel B of Figure 1 (Cuffe et al. 2005; Reid et al. 1998, 2001; Waschbusch and Willoughby 2008). The gender gap in school suspensions also is of larger magnitude among blacks than among whites, as shown in Panel C of Figure 1 (Snyder and Pratt 1997; Aud et al. 2010).

With diagnosed ADHD having reached epidemic levels and the school suspension rate also at a post-1970s high, medical practitioners, teachers, social workers, parents, and journalists have widely debated one key question: Is rising prevalence the result of true shifts in behavior? Some researchers and practitioners argue that the rise in ADHD diagnoses and school suspensions over the past three decades results primarily from true changes in symptomatology. They point to environmental shifts, such as the rise in lead, tobacco, and mercury exposures, among a variety of other potential factors (Froehlich et al. 2011; Lanphear et al. 2005; Lanphear 2012; Mayes et al. 2009; Swanson et al. 2007). By contrast, an alternate explanation is that the rise in ADHD diagnoses and school suspensions is explained in part by heightened social awareness and a growing proclivity toward the medicalization and disciplinary treatment of socioemotional problems. This latter group of changes may have led to a rise in ADHD diagnoses and school suspensions without necessarily involving changes in underlying behaviors.

Distinguishing whether real or perceived changes in behavioral symptomatology drive the rising prevalence of behavioral disorders like ADHD and school suspension is important for parents, teachers, medical providers and insurers, and employers concerned with maximizing the health, education, and productivity of future generations (Park 2013). This

question is also important for addressing the causes of gender disparities in schooling and for confronting the early life-course origins of racial inequality in America. Nevertheless, rigorous empirical investigation of the factors that may explain why the growing gender gap in these inattentive, hyperactive, and antisocial behaviors is concentrated among black children is limited. In the case of ADHD diagnosis, this is largely because research has often been conducted by medical researchers interested in clinical as opposed to social stratification processes. Research on school suspensions has focused on racial and socioeconomic disparities, but attention has been placed on the magnitude of race and gender gaps at the means (Mendez 2003; Skiba et al. 2011). However, research suggests that the race and gender dynamics of stratification processes often vary tremendously at different points of a sample distribution (Budig and Hodges 2010).

This paper aims to help fill these gaps in extant research. This paper examines the dynamics and causes of cohort change in race-specific gender gaps across the distribution of behavior problems of two national samples of children. A segment of black boys may be reported as behaving worse than black girls relative to white boys and girls without true shifts in symptomatology even before they enter schooling age. If so, this would point to the emergence of intensified racial bias that takes hold beginning in early childhood, setting up a growing segment of black boys for disadvantages in a wide range of key outcomes. Early life behavior problems are key predictors of educational achievement and attainment and labor market earnings (Alexander et al. 1997; DiPrete and Jennings 2012; Farkas 2003; Heckman and Rubinstein 2001). The emergence across cohorts of a larger gender gap in externalizing problems among an increasing number of minority children would carry important implications for many forms of social inequality across the life-course of minority boys, and possibly affect inequality in future generations. These social inequalities could impact child well-being and delinquency now, and, as children age, labor markets, marriage markets, family stability, and economic inequality.

Moving Beyond Binary Classifications of Problem Behavior

One way to gain empirical traction on whether the rising prevalence of behavioral disorders results from true changes in symptomatology is to examine shifts in the reported

frequencies of the behaviors used to make a diagnosis or a suspension (Kraemer 2007; Swanson et al. 2007, 2013). Although diagnosis and suspension are binary outcomes, the behaviors themselves occur along a continuous spectrum with the DSM criteria or school disciplinary codes guiding the assessment of whether frequency and severity surpass the threshold necessary for intervention. For example, through the DSM, psychiatrists assess the numbers of behaviors exhibited "frequently" across settings in a way that "imposes significant impairment," as shown in Table 1 (American Psychiatric Association Task Force on DSM-IV 1994:85-93).

Implicit measures of frequency and severity are reported explicitly through "externalizing problems" scales like the Child Behavior Checklist (CBCL) and the Pre-Kindergarten Behavioral Skills-2nd Edition (PKBS-2) (Kraemer 2007). Externalizing problems scales may be compartmentalized into subsets that capture the same constructs used to diagnose ADHD and which are correlated with school suspension: hyperactivity, impulsivity, antisocial problems, and inattention (Kraemer 2007). Items in the two main externalizing problems sub-scales, self-regulation problems and social problems, overlap with all three subtypes of ADHD (Eisenberg et al. 2000; Hill et al. 2006).[3] Self-regulation problems include lower levels of attention and concentration (Kraft and Nickel 1995; Rutter et al. 2004). Social problems include lesser ability to get along with others and avoid temperamental outbursts (DiPrete and Jennings 2012; Zill and West 2001).

[TABLE 1 ABOUT HERE]

Importantly, the developmental psychological instruments used to measure externalizing problems have high diagnostic accuracy as screens for ADHD and for the conduct disorders that lead to school suspension (Breslau et al. 2000; Hack et al. 1995; Hudziak et al. 2004).[4] Although parent reports of children's externalizing problems are imperfect, research suggests that reporting bias is minimized when questions ask about the frequency with which specific behaviors (as opposed to undefined "ADHD behaviors") were exhibited in the past week or month (Elder 2010; Grimm et al. 2010).

In line with this work, this study assumes the rising prevalence of diagnosed ADHD and school suspensions and the growing absolute magnitude of the gender gaps therein are linked to shifts in underlying externalizing behavior problems. Concordant with patterns

of ADHD behaviors and school suspension, the CBCL and PKBS-2 externalizing problems measures reveal higher levels of "externalizing problems" among boys than girls, beginning in early childhood and persisting into adolescence and adulthood (Gullo and Burton 1992; Tanner 1990). Boys' higher mean levels scale up to produce a "gender gap" in externalizing problems at the group level.

Perhaps because of the long-standing gender gap in levels of self-regulation problems and social problems, researchers have not examined gender-specific shifts in externalizing problems distributions across birth cohorts. Shifts in the gender distribution of externalizing behavior problems at the group level may carry important consequences for the gender gap in ADHD diagnosis and school suspension. For example, although looking at the case of differences in blood lead exposure, Lanphear (2012) documented that "...small shifts in the frequency of ADHD symptoms in a population have a substantial influence on the prevalence of ADHD" (1183). For example, the difference between exhibiting 1 and 3 ADHD symptoms has been shown to translate into over a two-fold difference (5% versus 13%) in the population prevalence of children who meet the criteria for ADHD (Lanphear 2012).

Two Potential Population Dynamics Underlying A Growing Gender Gap in Externalizing Problems

> **Hypothesis 1:** Reflecting racial differences in the growth of gender gaps in the prevalence of clinical levels of ADHD symptomatology and school suspension, the gender gap in externalizing problems between white and Asian boys and girls appears in the middle of their respective distributions and follows the first population scenario described below. By contrast, the gap between black boys and girls extends all the way to the low-end of the behavior problems distributions, following the second population scenario described below.

Population shifts in clinical levels of ADHD behaviors and the behaviors that lead to school suspension may largely arise from one of two shifts in underlying externalizing problems distributions, displayed in Figure 2. The first scenario – shown in the top panel of Figure 2 – entails a threshold effect, whereby a disproportionate number of boys with externalizing problems scores that would have placed them just below the diagnostic threshold according to 1980s behavioral standards now fall over the threshold by 2000s

standards. In this scenario, a growing gender gap in diagnosis or suspension would require the types of girls with externalizing problems profiles that would have previously placed them just below the threshold to also enter the clinical or suspended populations, but less so than boys.[5] Note that the scores of boys and girls with low levels of behavior problems remain virtually unchanged and the gender gap in externalizing problems scores appears primarily in the top half of the distribution, between the boys and girls with higher levels of behavior problems.

[FIGURE 2 ABOUT HERE]

The second scenario – shown in panel 2 of Figure 2 – entails an upward shift in externalizing problems scores across a wider cross-section of the distribution than in the first scenario, with a larger upward shift in boys' than girls' distribution. This would mean that 2001-born boys would have received lower scores had they been born in the 1980s (i.e., assessed by 1980s standards). In this scenario, the gender gap in externalizing problems scores would appear across a wider cross-section of boys and girls, not just between those with higher externalizing problems scores. This is because as prevalence of a condition increases, a wider cross-section of the population becomes exposed (King and Bearman 2011). Unlike in the first scenario, even the types of boys who would have been reported to have relatively low levels of behavior problems in the 1980s would be judged more negatively in the 2000s than girls who also display low levels of behavior problems. This would produce a gender gap in reported behavior problems across a wider cross-section of the distribution, rather than only among the boys and girls with the highest levels of reported behavior problems.

For the first scenario to be the driving force underlying the growing gender gap, changes in the causes across cohorts would have to be concentrated among children with relatively high levels of externalizing behavior problems. Research shows that, beginning in early childhood, low socioeconomic status (SES) children receive significantly higher externalizing problems scores than middle- and high- socioeconomic status children (Bradley and Corwyn 2002; Duncan et al. 1994). Due to the correlation between race and SES, many minority children come from low SES families and are disproportionately represented in the high externalizing problems range. Not coincidentally, many of the environmental

changes discussed earlier (e.g., rise in lead and mercury exposure, pre-term and low birth weight, chronic health problems like asthma, and father absence in households) also disproportionately affect low SES and black children (Adler and Newman 2002; Lanphear et al. 1996; Lanphear 2012).

For the second scenario to help explain the growing gender gap, explanatory factors must affect a wide cross-section of children (as opposed to being concentrated among low-SES and minority children, like those environmental factors discussed earlier). Some plausible drivers of changing standards of parental behavior reports include changes in social contexts (e.g., the rise of zero-tolerance disciplinary tactics in daycares and pre-Kindergarten settings), in diagnostic procedures for socioemotional disorders, and in awareness of behavioral disorders through marketing and advertising campaigns (Gilliam and Shahar 2006; Schwarz 2013). While the effects of these factors may still be concentrated largely in minority communities, exposure to them spans virtually the entire socioeconomic spectrum – and, therefore, virtually the entire externalizing behavior problems spectrum as well. Although not definitive, empirical support of the second scenario would suggest that, at least for the children with low levels of externalizing problems, changes in perceptions of behavior problems (rather than true changes in symptomatology) may help explain the growing gender gap in early childhood externalizing problems.

Because black boys historically have received the highest baseline externalizing problems ratings, the rightward shift in their externalizing problems scores would produce the largest gender gaps between black boys and girls (Brooks-Gunn and Markman 2005; Epstein et al. 2005). An increase in reported levels of externalizing problems may extend to an even wider and less behaviorally-troubled cross-section of black boys. The gender gap in externalizing problems may then appear between black boys and girls even at the low-end of their respective behavior problems distributions.

Environmental Explanations for a True Rise in Behavior Problems through the Middle of Black and White Boys' Externalizing Problems Distribution

> **Hypothesis 2a:** The expected growth of the gender gap in externalizing problems among black and white children in the middle of their respective externalizing distributions is explained by boys' greater externalizing response to the rise in single-

parent households and early childhood health problems, like pre-term birth and low birthweight, and in chronic conditions like childhood asthma.

Because the growing absolute gender gap in ADHD- and suspension-related behavior problems is most pronounced between black boys and girls, potential population-level environmental explanations must: 1) be linked to the behavior problems implicated in ADHD and suspension; 2) incite a more negative externalizing response among boys amid rising prevalence across recent cohorts, and; 3) be concentrated among or disproportionately adversely affect black children, especially black boys.[6]

Although far from an exhaustive list of potential environmental explanations, some changes in early childhood health and in families meet each of these criteria and are testable through available longitudinal data. The last three decades have witnessed a rise in prenatal and early childhood chronic health problems, including ascending rates of low birth weight (under 5.5 pounds), infant mortality, pre-term birth (under 37 weeks gestation), and chronic conditions like asthma (CDC 2007; Demissie et al. 2001; Horbar et al. 2002; Moorman et al. 2007). Single-parent households have also been on the rise over the same period (McLanahan 2004; McLanahan and Percheski 2008). Some research shows that the poverty status or changes in economic circumstances that often accompany divorce or single-parenthood may be linked to behavior and conduct problems (Thomson et al. 1994). This association may operate through parental stress, abuse, maltreatment, or social disorganization, which produces the high cortisol levels typical in children with ADHD and social misconduct problems (Froehlich et al. 2007; Webb 2013). Each of these health and family factors is linked to the inattentive and antisocial behaviors that may lead to an ADHD diagnosis and/or a school suspension (Bhutta et al. 2002; Elsmen et al. 2004; Froehlich et al. 2007; Gillaspy et al. 2002; Mick et al. 2002; Mogensen et al. 2011).

For certain factors, within-race gender differences in exposure to these changes in early childhood health and family structure are small in scale or non-existent. However, some research shows that boys exhibit more problem behavior in response to these exposures than do girls (see Appendix for a detailed explanation of trends by gender and race) (Alexander et al. 1997; Cooper et al. 2011; Davies and Lindsay 2004; Demo and Acock 1988; Elsmen et al. 2004; Pharoah et al. 1994). Exposure to these changes in early

health and families has been concentrated in black communities. Given that the effects of exposure may be largest among boys, these changes suggest that the rise in exposure would have the largest effects on the behavior problems of black boys in particular. From this perspective, true changes in behavioral symptomatology drive the rise in ADHD and school suspension and their concentration among minority boys.

Not coincidentally, these environmental changes in families and in health disproportionately affect low-SES and black children (Adler and Newman 2002; Lanphear et al. 1996; Lanphear 2012; Reichman 2005). Due to the correlation between low-SES and high levels of behavior problems, black and white children from low-SES families are disproportionately represented among those in the top half of the externalizing problems distribution (Buchmann et al. 2008; Farkas 2003).

Social Explanations for a Perceived Rise in Externalizing Problems among the "Best-Behaved" Black Boys

> **Hypothesis 2b:** The expected growth of the gender gap among the black boys and girls with the fewest behavior problems (i.e., those in the left tail of their respective behavioral distributions) is not explained by the changes in families and early childhood health described above.

Another set of explanations highlight that social shifts may have lead to heightened ADHD diagnoses or school suspensions without necessarily involving changes in underlying behaviors. For example, the 1990s saw a striking rise in zero-tolerance policies not only in primary and secondary education but also in daycares and preschools (Gilliam and Shahar 2006; Losen and Skiba 2010; Skiba and Peterson 2000). The rise in single-parent and dual-income households means that many more children enter daycares and pre-kindergartens at younger ages in the 2000s compared to the 1980s (Brooks-Gunn et al. 2002). Once there, minority children, particularly minority boys, are most-sanctioned by zero-tolerance policies (Mendez 2003; Skiba et al. 2011). One study of randomly selected preschools in Massachusetts found that classes with a higher proportion of minority children experienced increased likelihood of the use of suspension and expulsion as a disciplinary tactic relative to classrooms with fewer minority children, with minority boys most likely to be suspended or expelled (Gilliam and Shahar 2006).

Parents' awareness of behavioral disorders may be an important component of social explanations for the rise in ADHD diagnoses. Parents' awareness of behavioral disorders is likely heightened through factors like the rise of advertising campaigns marketing pharmaceutical treatments for ADHD and related behavioral disorders (Conrad 2005; Olfson et al. 2003; Schwarz and Cohen 2013). The rise in marketing campaigns, among other factors, may increase parents' awareness of their children's behavior problems even before their children enter kindergarten (Schwarz 2013). In light of the higher rates of disciplinary treatment of minority boys compared to all other groups in daycares and schools, the parents of minority boys may be most attuned to and likely to report the largest increases in their behavior problems without necessarily involving true changes in their behavioral displays.

With heightened awareness of ADHD and available pharmacological treatments, a wider cross-section of parents – including more affluent parents whose children are, on average, better-behaved – are likely to bring their children in for assessment at the first sign of a problem (Park 2013; Schwarz 2013). Once in the doctor's office, children today are more likely to be diagnosed than those 30 years ago (Froehlich et al. 2007). The expansion of Medicaid in the 1990s and the three revisions to the DSM criteria between 1980 and 2000 also may have contributed to a marked rise in ADHD diagnoses. With the transition from the DSM-III-Revised to the DSM-IV criteria in the early-to-mid 1990s, one study revealed an overall 57% increase in ADHD diagnoses with the DSM-IV compared to the DSM-III-Revised criteria (Wolraich et al. 1996). Some research also shows that, once in the doctor's office, only 38% of pediatricians used the DSM criteria to make their ADHD diagnosis (Wasserman et al. 1999). From this perspective, changes in norms and expectations about behavior take precedence over changes in symptomatology.

2 Data and Measures

Data

This study uses the National Longitudinal Survey of Youth-Child Supplement (NLSY-C) and the Early Childhood Longitudinal Study: Birth Cohort (ECLS-B) to compare two national samples of children followed from birth in the mid-1980s or in 2001 until at

least kindergarten. Based on maternal interviews or surveys, both datasets cover early childhood health, cognitive and behavioral development, home environments, and parents' relationship statuses, social class, and demographic characteristics. Data structure and timing of variables collection are shown by year, age, and birth cohort in Figure 3. The X's indicate the chronological progression of each birth cohort by age.

[FIGURE 3 ABOUT HERE]

Restrictions: National Longitudinal Survey of Youth-Children (NLSY-C)

This study uses the 1983-1986 birth cohorts of the NLSY-C because externalizing problems at ages 4-5 were not collected in the NLSY-C until 1986. These cohorts also allow me to maximize the period between studies.[7] Table 2 shows that, of the roughly 3,000 NLSY-C children born between 1983 and 1986, 506 (19%) had siblings in the sample. Because conditional quantile regression – the primary analytic method – does not permit clustered standard errors for siblings, I randomly selected one child from these families, for a total of roughly 2,600 children (a similar strategy is employed in Thomson et al. (1994)). Behavior problem measures were available for roughly 2,000 of these children.

[TABLE 2 ABOUT HERE]

Restrictions: Early Childhood Longitudinal Study-Birth Cohort (ECLS-B)

After 15-20 years, the ECLS-B collected similar developmental and family information on a nationally-representative sample of about 11,000 babies born in 2001. Based on direct observations and parental interviews/surveys, the ECLS-B tracked children at 9 months, 2 years, 4 or 5 years (preschool), and 6 years (kindergarten), as shown in Figure 3. ECLS-B mothers spanned the child-bearing ages at childbirth. For comparability across datasets, the ECLS-B sample was restricted to the roughly 6,100 children born to mothers aged 18-29 at their child's birth in 2001, as shown in Table 2.[8] Approximately 10% of the sample consisted of (oversampled) twin or higher order births. Because the quantile regressions used in these chapters cannot cluster standard errors for siblings, I randomly selected one child per mother. Of these roughly 5,700, externalizing problems at ages 4 or 5 were available for the roughly 4,600 children in the working sample.

Measures

Dependent Variable

The externalizing problems scale includes two sub-scales: self-regulation problems and social problems, as shown in Table 3 (Peterson and Zill 1986). Items used in each dataset were measured when children were the same age and collected from the same reporting party.[9] In both datasets, mothers report the child's frequency of: (1) impulsiveness, (2) restlessness, (3) trouble getting along with/difficulty getting invited to play by other children, (4) destructiveness, (5) likability (reverse-coded), and (6) temper.[10,11]

In the ECLS-B, mothers report frequency of the child's externalizing behavior on a scale from 1 ("*never*") to 5 ("*always*").[12] In the NLSY-C, mother reports are measured on a scale of 1 ("*never/rarely*"), 2 ("*sometimes*"), and 3 ("*often*") (reverse-coded). In light of research showing that differences in response categories shape reports of behavior frequencies (Schwarz 1999), there is concern that the availability of two additional extreme categories (i.e., "*never*" in addition to "*rarely*" and "*always*" in addition to "*often*") changes respondent anchoring in the ECLS-B scale relative to the NLSY-C scale. Conceptually, respondents in the ECLS-B may be more likely to avoid responses in the most extreme categories, or they may treat the scale as semi-continuous and linear and be equally likely to choose any of the categories (Schwarz 1999). Analysis of changes in the distribution and variance of the original ECLS-B 5-point scale to one concordant with the NLSY-C 3-point scale was conducted in order to examine the effects of a range of possible assumptions about response tendencies. Based on the results of this analysis (discussed in Appendix A), the ECLS-B items are rescaled from x=1 ("*never*") to x=5 ("*very often*") to 1 ("*rarely*") to 3 ("*often*") using two methods: (1) merging of extreme categories ("*very often*" with "*often*" and "*never*" with "*rarely*"), and; (2) a linear rescaling using the formula: x*0.5 + 0.5. The resulting scale ranges from 6 to 18. The ECLS-B scale has a Cronbach's Alpha of 0.75; the NLSY-C, 0.70. Because results did not differ substantively between the rescaling methods, results from the first method – the merging of extreme categories – are reported throughout the paper. Comparability of externalizing problems items across datasets and related sensitivity analyses are discussed in Appendix A.

[TABLE 3 ABOUT HERE]

Potential Mechanisms of a Widespread Gender Gap among Black Children

Recall that the family and health factors of interest are most concentrated among black children, with evidence suggesting a larger association with the externalizing problems of boys than girls. To begin to disentangle the role of these interrelated mechanisms, I differentiate among three broad sets of predictors: family socioeconomic and cultural resources, family structure, and early childhood health. Family socioeconomic and cultural resources include mother's years of schooling at time of child's birth and per capita household income at age 4 (in $1,000s) in 2011 dollars.[13] Family structure includes family composition at birth (a dummy for father absent at birth) and family composition at age 4 (indicators for single mother and social [i.e., non-biological, residential] father). Early childhood health factors include indicators for pre-term birth (fewer than 37 weeks gestation), low birth weight (fewer than 5.5 pounds), and asthma diagnosis by age 4.[14]

Demographic Context and Internalizing Problems

Models also adjust for mother's age at birth, child birth order, child year of birth, child internalizing behavior at ages 4 or 5 (discussed below), and indicators for race/ethnicity (when not stratifying by race). Internalizing problems include a range of anxiety disorders linked to depression and are comorbid with externalizing problems, but because comorbidity is much higher for girls than boys, internalizing behaviors are an important control in order to isolate the externalizing behaviors linked to ADHD and school suspension (Zahn-Waxler et al. 2000). Internalizing problems are measured by maternal report on two items that overlap across the Pre-Kindergarten Behavioral Skills (2^{nd} ed.) (PKBS-2) and the CBCL's Behavior Problems Index (BPI): (1) child seems unhappy, sad, or depressed and (2) child is too fearful or anxious. These items cover both main components of internalizing (social withdrawal and anxiety/somatic problems) while also using items deemed to be valid measures of internalizing across both the PKBS-2 and BPI scales.[15] The internalizing items are scaled (and re-scaled in the case of the 1-5 point ECLS-B items) in the same way as the items used to measure externalizing problems.

Cognitive Development

Prior research has focused on the relationship between behavioral problems and test scores

(Buchmann et al. 2008; McLeod and Kaiser 2004; Whitmire 2010). But, it is not clear whether behavior predicts cognitive development, cognitive development predicts behavior, or both are the product of some other factor associated with both (Cunha and Heckman 2010). Understanding the role of gender and racial differences in cognitive skills development in early childhood requires a level of theoretical and empirical treatment beyond the scope of the present study. Given this study's focus on behavior and the family and health factors that explain a growth of the externalizing problems gap, results in the main text do not control for cognitive development. However, supplementary analyses adjust for variation in cognitive ability and are discussed briefly in the conclusion as an avenue for further research. Empirical results from the present study that include the Peabody Picture Vocabulary Test scores as a control for receptive vocabulary are available from the author upon request.

Treatment of Missing Data and Sensitivity Analyses

Multiple imputation of 20 datasets using the built-in multiple imputation procedure in Stata 11 was used to deal with item-missingness on key predictors (Royston 2004). Externalizing problems at ages 4 or 5 (missing for roughly 24% of cases in the NLSY-C and 21% of cases in the ECLS-B) were included in imputation. Observations with imputed dependent variables were dropped prior to analyses based on the strategy of multiple imputation then deletion (Von Hippel 2007). Imputed predictor variables for which missingness was highest included per capita household income at age 4 (22% in the NLSY-C and 33% in the ECLS-B) and cognitive support at age 4 (18% in the NLSY-C and 10% in the ECLS-B). The working sample consisted of 6,400 observations after carrying out the imputations (rounded to the nearest 100 for restricted data reporting purposes). Given the extent of item missingness, three sets of sensitivity analyses were conducted: (1) Replication with complete cases only; (2) Replication with a second multiply-imputed dataset in which the variances of imputed items were increased by 10% to partially test violation of the missing-at-random assumption (Allison 2000); and (3) Assessment of systematic biases in item-missingness by regressing a binary indicator for missingness on the dependent variable on observed covariates. This tests whether the dependent variable is

missing-at-random as a function of observed predictors.

Internal and Predictive Validity Checks for the Behavioral Scales

A number of sensitivity analyses were conducted to examine the validity of constructed scales. First, the item in the constructed externalizing problems scale that asks mothers how frequently "the child has trouble getting along with other kids" is somewhat inconsistent in its wording between datasets. As a robustness check on the internal validity of the externalizing problems scale, I estimated all models without this item, drawing instead on the other 5 items. Results did not change substantively.

I also examined correlations between my constructed externalizing problems and internalizing problems scales and the complete scale provided in the respective dataset. I found high correlations (above .90) between each of my constructed scales and the more complete scale available within each dataset. For example, in the NLSY-C, the externalizing behaviors scale comes from the Behavior Problems Index, which is a subset of 10 items taken from the Achenbach Child Behavior Checklist. In the ECLS-B, the externalizing behaviors items come from the PKBS-2 and consist of a subset of 8 of the original 27 PKBS-2 externalizing problems items.

3 Analytic Strategy

By focusing on the gender difference in each group's mean level of externalizing problems, a method such as ordinary least squares (OLS) regression obscures important changes across time in other parts of boys' and girls' externalizing problems distributions. To address this limitation, the present study draws on a statistical method called conditional quantile regression. Linear conditional quantile regression is useful when the association between covariates and the outcome vary at different points in the distribution of the outcome variable, as is often the case in non-normal distributions like those of externalizing problems scales. Linear conditional quantile regression identifies differences across covariate values on the location of a given conditional quantile (or percentile) of the dependent variable (Koenker and Bassett 1978). For example, a covariate indicator for "male" in a 25^{th} quantile (i.e., percentile) regression identifies if there is a gender difference on the

location of the conditional 25th percentile of behavior problems. Substantively, this is important because boys and girls are socialized differently and their behavior is assessed as "good" or "bad" in distinct ways (Buchmann et al. 2008; West and Zimmerman 1987).

In this study, I examine race and gender differences in the locations of the 10th, 25th, 50th, 75th, and 90th percentiles of externalizing problems scores. Bootstrapped standard errors are based on 500 independent draws with replacement. Analyses use the "bsqreg" package in Stata 11 and the "quantreg" package in R. In the presentation of results in the main text, I focus on the middle and left tail of the externalizing problems distribution because this is where a gender gap in externalizing behavior problems emerges for the 2001 birth cohort where it did not exist for the 1980s birth cohorts. Given this study's focus on gender-within-race gaps in externalizing problems, the estimation sample is stratified by race before conditional quantile regression models are estimated. Because the difference in the gender gap in externalizing problems is most pronounced between blacks and whites/Asians, I focus on these results. Results for Hispanics are available upon request.

After identifying gender-within-race differences in the location of key externalizing problems quantiles, I examine potential mechanisms that may help explain the growth of the gender gap in externalizing problems. Specifically, I test the hypothesis that boys' greater exposure to and/or more negative externalizing response to increasing prevalence of single-parent households and early childhood chronic health problems helps account for the increasingly widespread gender gap across the distribution of externalizing problems. Because absolute prevalence of these family and health factors has become most widespread within black communities, the first hypothesis posited that the within-race gender gap would become most widespread among black children.

To test the second hypothesis about the factors explaining the growing gender gap hypothesized to be most widespread among black children, I introduce into the previous quantile regressions additional covariates for SES, family structure, and early childhood health. Covariates are introduced in order from the most distal to the most proximate in relation to the measure of behavior:[16,17] (1) no demographic controls beyond indicators

for male, the 2001 birth cohort, and the interaction for male*2001 birth cohort, (2) demographic controls and internalizing behavior, (3) SES main effects, (4) interactions between SES variables and gender, time period, and gender and time period, (5) family structure main effects, (6) interactions between family structure variables and gender, time period, and gender and time period, (7) health main effects, (8) interactions between the health variables and gender, time period, and gender and time period, (9) SES and family structure main effects together as "economic and cultural resources," (10) interactions between SES and family structure and gender, time period, and gender and time period, (11) SES, family structure, and health main effects, and (12) interactions between SES, family structure, and health and gender, time period, and gender and time period. Note that when included in a given model, mother's years of schooling, per capita household income in 2011 dollars (in $1,000s), internalizing, mother's age at birth, and receptive vocabulary are overall sample mean-centered.

4 Results

Racial Disparities in the Spread of the Gender Gap in Externalizing Problems

The boxplot shown in Figure 4 displays the externalizing problems distributions for the 1983-86 and the 2001 birth cohorts by gender and race.[18] The first two plots show that, among blacks in the 1980s, a gender gap appears only on the location of the 75th and 90th percentiles. The third and fourth boxplots reveal a rightward shift in the entire externalizing problems distribution of boys that is consistent with hypothesis 1, and with the second scenario presented earlier. The result is the appearance of a gender gap on the location of the 10^{th}, 25^{th}, and 50^{th} (in addition to the 75^{th} and 90^{th}) percentiles of externalizing problems of black children in the 2001 birth cohort. This is due to the heavily right-skewed distribution of black boys externalizing problems scores in the 2000s.

The results for white and Asian children are also consistent with the first hypothesis and the second population scenario. They reveal a more pronounced rightward shift in the bottom half of the distribution of white and Asian girls than in that of white and Asian boys or black girls. In the 1980s, white and Asian girls' scores are weighted toward the lowest score of 6 in the externalizing problems scale, creating gender gaps

on the location of the 25th percentiles in addition to the locations of the 75th and 90th percentiles. Comparatively, in the 2000s, there is an upward shift in the scores of the white and Asian girls in the lowest quartile of externalizing problems along with rightward shift in the top half of the male distribution. The result is gender gaps on the location of the 50th percentile in addition to those on the locations of the 75th and 90th percentiles among white and Asian girls and boys born in the 2000s. Conceptually, this 1 point is the difference between a mother reporting that a child "*sometimes*" versus "*often*" displays one of the six externalizing behavior problems assessed in the present study. Although this may seem to be a modest difference in practical terms, a 1 point difference between boys and girls in the top half of externalizing problems scores equates to a roughly 0.4-0.5 standard deviation increase (depending on whether one divides by the standard deviation of girls or boys in the early or late period).

[FIGURE 4 ABOUT HERE]

Figure 5 more clearly highlights the locations of gender gaps within race and cohort groups. Panel 1 shows the gender gap on the location of the various externalizing problems conditional quantiles by race.[19] Panel 2 displays the change in the magnitude of the gender gap on the locations of the conditional quantiles between the 1980s and 2000s birth cohorts. Consistent with hypothesis 1, panel 2 of Figure 5 shows that, among black boys and girls in the 2000s, the gender gap in externalizing problems had spread to the locations of the lowest two quartiles (i.e., to the best-behaved children). The emergence of a gender gap on the location of the conditional 10th and 25th percentiles results from the rightward shift of the entire black male distribution, but of only the top half of the female distribution (as discussed in the second counterfactual scenario). By contrast, among whites and Asians, the gender gap only spreads to the location of the conditional 50th percentile, extending throughout the top half of the externalizing problems distribution in the 2000s cohort. This spread may be partially explained by concomitant shifts in health and families between the 1980s and 2000s cohorts.

[FIGURE 5 ABOUT HERE]

Exposures and "Effects": Families, Health, and the Growth of the Gender Gap in the Location of the Conditional 10th Percentile of Black Children's Externalizing Problems

Figure 6 displays the results of models testing the second hypothesis that the rising prevalence of single-parent households and early childhood chronic health problems helps explain the spread of the gender gap in the location of the conditional 10^{th}, 25^{th}, and 50^{th} percentiles of externalizing problems for black children and in the location of the conditional 50^{th} percentile of externalizing problems for white and Asian children. The y-axis represents the coefficient on the male * 2001 cohort interaction, or the unexplained growth of the gender gap in externalizing problems. The x-axis contains model numbers (1)-(12), which are described in detail in the analytic strategy and in Figure 6 notes. Model results are presented separately by race and behavioral quantile in order to examine whether the concentration within black communities of the family and health factors of interest helps explain the widespread gender gap among black children. The concentration of these factors within black communities may help explain the spread of the within-race gender gap on the location of a given conditional percentile of externalizing problems due to additional differences by gender in some combination of: 1) levels or types of family and health exposures or 2) the "effects" of family structures and health on boys' and girls' externalizing problems.

[FIGURE 6 ABOUT HERE]

The most salient result in Figure 6 – consistent with hypothesis 2b – is that only a small proportion of the growth of the gender gap on the location of the conditional 10^{th} percentile of black children's externalizing problems is accounted for by changes in families and health over this period (also see Appendix Table A.1). By contrast, and in support of hypothesis 2a, 100% of the 1-point growth of the gender gap in the location of the 25^{th} percentile of externalizing problems among black children and between 80% and 90% of the 1-point growth of the gender gap on the location of the 50^{th} percentile of externalizing problems for black and white and Asian children is explained by observed changes in family and health exposures, and, most importantly, their differential effects on boys' and girls' reported externalizing problems, net of controls (see also Appendix Tables A.2, A.3, and A.4).

Furthermore, the explanatory power of the mechanisms of interest follow a similar pattern in their ability to account for the unexplained proportion of the 1-point growth of

the gender gap on the location of the conditional 25th and 50th percentiles of externalizing problems for black children and the conditional 50th percentiles of externalizing problems for white and Asian children. Models on the location of the conditional 25th percentile of externalizing problems among black children show the largest fluctuations in the percent of the growth of the gender gap explained, fluctuating from 0% (models 2 and 8) to 100% (model 6; here the gap is driven by girls' higher externalizing problems scores when holding constant the differential effects of family structure, net of controls). Models on the locations of the conditional 50th percentile of externalizing problems for blacks and whites and Asians show less variance in the percent explained across the models but similarly indicate the largest percent of the growth of the gap is accounted for by father-absence on boys' and girls' externalizing problems, net of controls (model 6) or by the differential effects on boys' and girls' externalizing of lowered socioeconomic resources, net of controls (model 4). This makes the growth of the gap on the location of the conditional 10th percentile among black children stand apart even more, suggesting a distinct process is at play in the ratings of these children.

Among black children, model (1) shows the unadjusted 1-point (roughly 0.43 standard deviation) growth of the gender gap on the location of the conditional 10th percentile of externalizing problems. Models (2) and (3) reveal that neither compositional differences between boys' and girls' levels of internalizing problems, mother's age at birth, nor family economic and cultural resources account for this growth. Model (4) indicates that 11% of the growth is accounted for by the more negative relationship between mother's years of schooling at birth and males' externalizing problems (even though statistical significance is not reached on the interaction between mother's years of schooling * Male * 2001 cohort). Models (5)-(9) indicate that neither gender differences in levels of exposure to family structures and health, nor their gender-specific effects account for any of the growth in the gender gap on the location of the conditional 10th percentile of externalizing problems among black children. Model (11) shows that gender differences in low birth weight, pre-term birth, and asthma diagnosis by age 4 do not account for any of the growth. Models (10) and (12) adjust for the disproportionately beneficial effects on boys' externalizing

problems in the later period of a mother with above sample average schooling at birth. Models (10) and (12) also account for the fact that father-absence at birth and social father-presence at age 4 have a less-negative association with externalizing problems for boys than girls. Adjusting for the differential effects of these family factors on boys and girls together accounts for 11.8% (model 10) and 20.9% (holding constant gender and period differences in early health in model 12) of the growth of the gender gap on the location of the conditional 10^{th} percentile among black children.[20]

The Growth of the Gender Gap in the Locations of the Conditional 25^{th} and 50^{th} Percentiles of Externalizing Problems among Black and White and Asian Children

Consistent with hypothesis 2a about the factors accounting for the growth of the gender gap on the location of the conditional 25^{th} percentile of externalizing problems among black children, a comparison of models (1)-(2) against model (3) reveals that roughly 65% of this growth is accounted for by compositional differences by gender in family economic resources. Black girls with an externalizing problems score that places them at the conditional 25^{th} percentile are raised in households with slightly higher levels of per capita income than black boys with an externalizing problems score located at their conditional 25^{th} percentile. In practice, income differences are correlated with differences in family structures and in early childhood health experiences. Not comparing children in the same family types and/or with the same early health experiences, model (2) attributes to income what may in reality manifest as differences in number of adults in the household or early childhood health complications. The effects of compositional differences in economic resources do not account for any more of the externalizing problems gap on the location of the conditional 25^{th} percentile (model 4) nor do differences in exposure to or the effects of early health problems (models 7 and 8). Models (5) and (6) point to the importance of gender differences in exposure and effect of family structure. Girls have less exposure to single mothering at age 4 and externalize less in response to being raised by a single mother than boys. These differences account for the entire growth in the gap on the location of the conditional 25^{th} percentile among black children.

The unexplained gap on the location of the conditional median of the behavior distri-

bution among black children owes largely to the rise in black males' level of internalizing problems between the 1980s and 2000s, as shown in model 2. Comparing black girls and boys with the same internalizing problems score accounts for 50% of the growth of the gender gap on the location of the conditional 50^{th} percentile of externalizing problems.[21] Model (3) indicates that minor gender differences in socioeconomic resources, particularly income, account for an additional 24 percentage-points (for a total of 74%) of the growth of the externalizing problems gap. The gap is further reduced by 23 percentage points (a total of 97% of the growth) when accounting for the fact that more educated mothers in the later time period report higher levels of behavior problems among black boys with externalizing problems that place them in the middle of the distribution than among black girls with externalizing problems scores in the middle of the female distribution (model 4). Models (5)-(11) indicate that gender differences in family structures and early health, which are correlated with differences in SES but are not included in models (3) and (4), help explain the growth of the externalizing problems gap on the location of the conditional 50^{th} percentile among black children. This is true even though these family and health factors do not account for any more of the growth than gender differences in socioeconomic resources or their effects.

Among white and Asian children with externalizing problems scores that place them at the conditional 50^{th} percentile of externalizing problems, roughly 80% of the 1-point (0.48 standard deviation) growth of the externalizing problems gap is accounted for by compositional differences between girls' and boys' levels of internalizing problems and birth cohort. Of the remaining 20% of the growth in the gap, up to 11 percentage points (for a total of 91% of the growth) is explained by boys' tendency to externalize more than girls in response to social father presence or single mother households at age 4 (model 6). None of the family SES or health factors explains more of the gap beyond that explained by these gender differences in response to family structures.

5 Discussion

This study begins to paint a picture of trends and underlying mechanisms implicated in the evolution of the gender gap in externalizing behavior problems across two national

samples of young children born nearly two decades apart. Results support the hypothesis that gender-specific trends in externalizing problems over the past two decades mirror trends in rates of ADHD diagnosis and elementary school suspension. Like these two behavioral phenomena, the gender gap in externalizing problems also has spread to a wider cross-section of children over the past 20 years. For both blacks and whites and Asians in the 1980s, the gender gap was concentrated among the girls and boys with the highest externalizing problems scores relative to their same-gender and race peers. By the 2000s, the gender gap had spread at least to the middle half of the externalizing problems distributions of both blacks and whites and Asians.

This study is of importance to gender scholars interested in processes of early gender socialization, to demographers interested in trends in early population processes linked to later health, education, and socioeconomic attainment, and to developmental psychologists and family sociologists concerned with cross-cohort gender differences in early behavioral and cognitive development. The study is also relevant for criminologists, education researchers, and stratification scholars interested in gender gaps in educational achievement, delinquency, and the intergenerational transmission of inequality. Finally, this work is pertinent to medical researchers and epidemiologists concerned with trends in the medicalization of early childhood chronic diseases and behavior problems and the population level trends in externalizing problems that underlie trends in clinical diagnosis.

A primary contribution of the present study is to encourage attention among researchers and policy makers to the race-specific patterns of the evolution of gender differences in behavior problems. Although the gender gap in externalizing problems has spread to a wider cross-section of children across racial groups, it is markedly concentrated among black children. For black children, a statistically non-zero gender gap, equivalent to a growth of roughly 0.4 standard deviations, appears even between the "best-behaved" boys and girls – those with the lowest decile of mother-rated externalizing problems scores. This growth of the gender gap even among the "best-behaved" decile of black girls and boys results from the disproportionate upward shift among the black boys with the lowest levels of reported externalizing problems.

A pressing question concerns the practical significance of a 1-point growth of the externalizing problems gap. A 1-point increase in the gender gap in mothers' reports of externalizing problems results from a shift from a rating of "*rarely*" to "*often*" or from "*often*" to "*always*" on one of the six behaviors that comprise the externalizing problems scale. In clinical terms, a 1-point increase from not exhibiting to exhibiting a particular externalizing behavior problem is equivalent to one-sixth of the criteria necessary for an ADHD diagnosis according to the Diagnostic and Statistical Manual (DSM)-IV in effect since 1994. Although each item in the externalizing problems scale consists of a 3- (or 5-) point frequency range, the corresponding items in the DSM-IV are binary; they rely on assessments of whether or not a child exhibits a particular behavior. Furthermore, supplementary analyses show that between 25% and 35% of externalizing problems scores fall within 1-point of the mean value of externalizing problems in each racial/ethnic, gender, and cohort group. A 1-point increase translates into an upward or downward shift roughly 12 to 17 percentage points in the behavioral distribution, therefore encompassing a large amount of the variation in the ratings that lead to differentiation between "well-behaved" and "badly-behaved" children.

The 1-point growth of the magnitude of the gender gap among the best-behaved black children may have sizeable consequences when scaled up to the population level. To put this magnitude into comparative perspective, one study examining lead exposure as a leading environmental cause of the rise of ADHD found the difference of 2 points in average numbers of ADHD symptoms between children with high and low blood lead exposures translates into a 5% versus 13% prevalence of ADHD at the population level (Lanphear 2012). This 8-percentage-point difference is of a similar order of magnitude as the observed absolute gender gap in ADHD diagnosis and school suspension.

A related matter that deserves some discussion is the decision to rank children's behavior problem scores relative to their same gender, same race/ethnicity, same cohort peers. Substantively, a large body of sociological and social psychological research documents the gender-specific socialization processes through which boys and girls learn and internalize different understandings of socially-acceptable behavior (Deaux and Major

1987; Maccoby 1988; West and Zimmerman 1987). This scholarship indicates that boys' and girls' behavior is judged according to different metrics and that different meanings are assigned to the same behaviors based on a child's gender. Similarly, research suggests that behaviors are interpreted differently and assigned different meanings based on a child's race and based on the intersectionalities in the performance of racial and gender identities (Crenshaw 1991; Deater-Deckard and Dodge 1997; Miner and Clarke-Stewart 2008). Children's behaviors also are more likely to be assessed against others of their same racial/ethnic group because of the extent of persistent racial segregation within American neighborhoods and schools (Conley 1999; Massey and Denton 1993; Oliver and Shapiro 2006) and the racially-tracked nature of the incarceration system (Western 2006).

Throughout this study, I have treated mother reports of the frequency of children's behaviors as validating and unbiased measures of true behavior. However, some research points to the subjectivity of ratings of children's behavior problems based on the identities of the rater, the use of parent or teacher ratings, high-stakes testing regimes, and the percent of children exposed to disciplinary tactics such as school suspension, expulsion, retention of special services/disability referral (Elder and Lubotsky 2009; Miner and Clarke-Stewart 2008; Ouazad 2008). As high-stakes testing, the use of stringent disciplinary tactics in daycares, preschools and beyond, and the incidence of behavioral disorder diagnosis each have become more ubiquitous over the past three decades (Gilliam and Shahar 2006; Mendez 2003; Olfson et al. 2003), children may receive higher ratings of behavior problems across cohorts – with or without a true change in behaviors.

On the diagnostic front, research shows dramatic jumps in diagnosed ADHD and other externalizing-linked disorders with changes in diagnostic criteria (Wolraich et al. 1996). Even if changes in diagnostic criteria lead to an artificial jump in ADHD diagnosed prevalence, internalized perceptions of behavior problems have real consequences. Parents may view and treat their sons as worse behaved today than parents did in past decades, and millions of children may grow up with the label and associated belief in the validity of their "problem child" status.

In light of this possibility of changes in perceptions and reporting of behavior with-

out true changes in behavior symptomatology, this study also begins to investigate in a preliminary way whether the apparent growth of the gender gap in behavior problems among even the "best-behaved" black children in the 2001 cohort is due to real changes in externalizing behavior symptomatology. The changes in social contexts of reception of behavior within families, schools, and doctors' offices discussed above all suggest that reports of worsening behavior may also be due to shifts in perceptions and reporting of the frequency of externalizing behaviors in gender- and race-specific ways. True shifts in behavior may be driven by changes in environmental exposures to lead, mercury, or other toxins, which may also produce a rise in early childhood chronic health conditions, or changing behavior symptomatology may be linked to changes in families and early childhood chronic health conditions. These risk factors are concentrated within minority, especially black, communities. And although rising prevalence of these factors means a broader spectrum of children, including those with fewer externalizing problems, are affected, high SES should serve as a protective factor for the most behaviorally-advantaged children within each racial group. Exposure to these risk factors should be minimal among the children with the lowest externalizing problems scores, including those black children.

Viewed from this lens, empirical results are consistent with the possibility that changes in perceptions and reporting underlie the growth of the gender gap in externalizing behavior among the best-behaved decile of black children. Among black children, 97% of the growth of the gender gap on the location of the conditional 50^{th} percentile of externalizing problems is accounted for by observed family and health factors. Among whites and Asians, 91% is explained. For both groups, the increase across the two samples in the expected value of internalizing problems alone accounts for over 50% of the growth of the gap in the location of the 50^{th} percentile of externalizing problems.

Only black children experienced a growth of the gender gap on the location of the conditional 25^{th} and 10^{th} percentiles of externalizing problems. However, observed family and health factors explain much more of the growth in the location of the conditional 25^{th} percentile than in the location of the conditional 10^{th} percentile of externalizing problems. Roughly 65% of the growth of the gap on the location of the conditional 25^{th}

percentile of externalizing problems is accounted for by observed family and health factors, in particular by gender differences in exposure to non-traditional family structures and associated socioeconomic resource disparities. By contrast, only 20% of the growth of the gender gap on the location of the conditional 10^{th} percentile of externalizing problems is accounted for by observed family and health factors. This 20% is explained by boys' tendency to externalize less than girls when born to more highly-educated mothers and to families with more economic resources. Taken together, these factors may translate across cohorts into a shift in the very nature of the behavioral rating categories. This shift may produce a fundamental change in the behavioral scales themselves that may be imperceptible without studies designed specifically to investigate this possibility.

One way for future research to investigate this possibility empirically is to examine whether the boys with low behavior problems scores who have older brothers experience the largest increase in externalizing problems reports. The idea here is that mothers with boys already in the school system may be most aware of the crackdown on bad behavior as a result of the rise in zero-tolerance policies and other stringent disciplinary practices and most sensitive to minor changes in their younger sons' behaviors. One might expect this to be less the case for mothers who have older daughters but not older sons. And, if the story about how changes in schools have led to the greatest crackdown on behaviors among black boys is true, black boys with older brothers should experience even larger increases in mothers' reports of externalizing behavior than white or Asian boys.

Another approach to empirically understanding the social processes underlying the growing gender gap in externalizing problems concentrated among the best-behaved black children is to investigate additional environmental shifts that may explain a true rise in externalizing behaviors among these well-behaved black children. Some research shows that maternal employment and early entry into daycare are associated with increases in externalizing problems (Brooks-Gunn et al. 2002). The 1980s and 1990s witnessed a rise in dual-earner and single-parent households across a wide socioeconomic spectrum of families (Bianchi 2000). It is possible that these changes may help explain the rise in externalizing behaviors among even the best-behaved black boys.

This study also raises questions about the role of cognitive skills in shifting gender and race dynamics in externalizing behaviors. Supplementary analyses not presented in the main text also highlight a large gender difference in early childhood receptive vocabulary – a proxy for cognitive development at young ages. A key question left unanswered by the present study is that of the link between receptive vocabulary and externalizing problems. This study finds that, in some cases, gender differences in receptive vocabulary account for almost the entirety of the growth of the gender gap in externalizing problems. This raises a number of questions about the direction of causality and/or a key omitted variable in the pathways between behavior and cognitive development. Relatedly, because it is unclear to what extent behavior produces cognitive development as opposed to the other way around, issues of biological or genetic predispositions are often implicated. Research shows that cognitive development and, to some extent, behavior – primarily executive function – are both at least somewhat biologically influenced (Blair 2002).

At the same time, research shows that social factors like SES and in-utero environment are key predictors of cognitive development and behavior (Bradley and Corwyn 2002). Certainly, given the growth of the gender gap in externalizing problems across cohorts born only 15-20 years apart, at least a significant component of behavior must be socially constructed and reinforced. Similarly, research shows that language development difficulties are most identifiable when paired with behavior problems (Stowe et al. 1999). The same study also finds that lower levels of language development were more strongly associated with disruptive behavior and poor peer relationships among pre-school aged boys than girls (Stowe et al. 1999). Taken together, this evidence – consistent with the notion of the social construction of cognitive ability – suggests that we would expect lower levels of language development among boys to explain much of the gender gap in behavior problems at ages 4-5. Future work should directly investigate the degree to which factors, such as gender differences in perceptions and/or reporting of behavior problems and/or changes in diagnostic criteria or medical coverage, account for the growth in the gap in externalizing problems, especially within more behaviorally and socioeconomically advantaged black communities.

Despite the possibility of a true behavioral change leading to the spread of the gender gap in early behavior problems reports to the most behaviorally and socioeconomically advantaged children in recent decades, interest in this phenomenon should not eclipse the importance of the fact that mother's reports of children's behavior problems continue to be highest among low SES children. This is important not only for social inequality in childhood since perceptions of behavior are linked to educational performance and attainment, but also for understanding the evolution of the gender gap in adult educational attainment.

Notes

[1]Whereas the DSM-III, published in 1980, declared the disorder as one of inattention, the DSM-IIIR, published in 1987, renewed pre-1980 emphasis on the hyperactive component and consolidated ADHD into a single disorder without subtypes (Wolraich et al. 1996). The DSM-IV criteria published in 1994 retained the name ADHD but created three subtypes: Primarily Inattentive, Primarily Hyperactive/Impulsive, and Combined Type.

[2]Note the important distinction between ADHD diagnosis and ADHD symptomatology. Blacks experience lower rates of ADHD diagnosis (but not symptomatology) than whites, but this is largely due to differences in health insurance coverage and doctor's visits (Currie 2005; Morgan et al. 2013).

[3]The DSM-IV classifies diagnoses of ADHD into the inattentive subtype, the hyperactive/impulsive subtype, or the combined subtype.

[4]Because these instruments are often based on parent or teacher (as opposed to clinician) reports of behavior symptoms, there has been concern over their validity. Critics assert that they overestimate population prevalence of ADHD (Getahun et al. 2013). However, other research using the same data shows that controlling for insurance status, geography, and age, parent reports are appropriate for monitoring state and national prevalence of ADHD (Visser et al. 2013). Most relevant for this study, recent work using individual-level data shows that teacher and, even more so, parent reports are considered valid for identifying children with clinical levels of problem behaviors (Elder 2010; Grimm et al. 2010).

[5]This process is exacerbated by diagnostic changes that move down the threshold for diagnosis, but for simplicity, here I assume a constant diagnostic threshold.

[6]For cases in which exposure differentials (as opposed to effect differentials) are the source of male disadvantage, there should be evidence of a growing gender gap in exposure to these factors.

[7]The NLSY-C includes children born in the 2000s but these cohorts introduce significant bias into the analysis due to maturation: the 2000s births in the NLSY-79 were to mothers much older and more economically and otherwise stable than the 1980s births.

[8]The mean age of childbearing increased from 24 to 27 years between 1983 and 2001 (Mathews and Hamilton 2002). Restricting the ECLS-B sample to children born to mothers 18-29 years may have introduced selection bias into the sample. As a robustness check, mothers through age 35 were included. The growth of the gender gap did not change significantly.

[9]Children were either age 4 or 5 because behavior was observed every other year in the NLSY-C.

[10]Most studies use a subset of the complete 27 items (and three sub-scales) in the Pre-Kindergarten Behavioral Skills (2^{nd} ed.) (PKBS-2). This study uses the subset of six items whose wording the developmental psychologists consulted by the author believe to align most closely across datasets. Fortunately, at least one item is available from each sub-scale in the complete PKBS-2 and CBCL externalizing scales: Self-centered/explosive, attention problems/overactive, and antisocial/aggressive. Despite scale limita-

tions, the study design minimizes the likelihood these would drive results because changes in wording would have to involve race and gender differences in how effects of wording changes occur. It seems unlikely that changes in item wording across datasets would differentially effect the reporting of behaviors for boys versus girls and minorities versus whites.

[11]Note that sensitivity analyses made use of the full externalizing problems scale in each dataset and found no substantial changes in results.

[12]Some research, like Mistis et al. (2000), suggests that the multidimensional measurement of externalizing behaviors through both parent and teacher reports enhances accuracy but teacher reports are not available in the NLSY-C and only on certain items in the ECLS-B. Other research suggests mother reports are valid screens for ADHD symptoms (Hudziak et al. 2004).

[13]Per capita household income is constructed by dividing total household income (adjusted to 2011 dollars) by the number of people living in the household).

[14]Pre-term and LBW are based on mother report in the first interview period after the child's birth.

[15]Models using all BPI internalizing items provided in the NLSY-C did not change substantive results.

[16]The exception is internalizing behavior problems, added with demographic controls to isolate the component of externalizing problems not co-morbid with internalizing problems.

[17]Models 1, 2, 3, 5, 7, 9, and 11 test differences in exposure to the family and health factors by gender and time period. Models 4, 6, 8, 10, and 12 investigate within-race gender and cohort differences in the influence of each factor on the location of a given conditional quantile of externalizing problems.

[18]Results pooling across race are available upon request.

[19]These quantile regressions condition on gender, a 2001 birth cohort indicator, and their interaction.

[20]Note that the three-way interaction terms between each early childhood health factor, gender, and time period are dropped from these models because of multicollinearity, indicating no differential effect of these early childhood health factors for males and females in the 2001 birth cohort.

[21]Supplementary analyses not shown control for cognitive development (i.e., receptive vocabulary). Black girls' greater receptive vocabulary in the 2000s versus the 1980s combined with the rise in males' level of internalizing problems accounts for almost 100% of the growth of the externalizing problems gap.

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Table 1: Diagnostic Criteria for the Three Subtypes of Attention-Deficit/Hyperactivity Disorder According to DSM-IV

A. "Persistent pattern of inattention and/or hyperactivity-impulsivity that is more frequently displayed and is more severe than is typically observed in individuals at comparable level of development." Individual must meet criteria for either (1) or (2):
(1) Six (or more) of the following symptoms of inattention have persisted for at least six months to a degree that is maladaptive and inconsistent with developmental level:
Inattention
(a) often fails to give close attention to details or makes careless mistakes in schoolwork, work or other activities
(b) often has difficulty sustaining attention in tasks or play activity
(c) often does not seem to listen when spoken to directly
(d) often does not follow through on instructions and fails to finish schoolwork, chores or duties in the workplace (not due to oppositional behavior or failure to understand instructions)
(e) often has difficulty organizing tasks and activities
(f) often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework)
(g) often looses things necessary for tasks or activities (e.g., toys, school assignments, pencils, books or tools)
(h) is often easily distracted by extraneous stimuli
(i) is often forgetful in daily activities
(2) Six (or more) of the following symptoms of hyperactivity-impulsivity have persisted for at least six months to a degree that is maladaptive and inconsistent with developmental level:
Hyperactivity
(a) often fidgets with hands or feet or squirms in seat
(b) often leaves seat in classroom or in other situations in which remaining seated is expected
(c) often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, may be limited to subjective feelings of restlessness)
(d) often has difficulty playing or engaging in leisure activities quietly
(e) is often "on the go" or often acts as if "driven by a motor"
(f) often talks excessively
Impulsivity
(g) often blurts out answers before questions have been completed
(h) often has difficulty awaiting turn
(i) often interrupts or intrudes on others (e.g., butts into conversations or games)
B. Some hyperactive-impulsive or inattentive symptoms must have been present before age 7 years.
C. Some impairment from the symptoms is present in at least two settings (e.g., at school [or work] and at home).
D. There must be clear evidence of interference with developmentally appropriate social, academic or occupational functioning.
E. The disturbance does not occur exclusively during the course of a Pervasive Developmental Disorder, Schizophrenia, or other Psychotic Disorders and is not better accounted for by another mental disorder (e.g., Mood Disorder, Anxiety Disorder, Dissociative Disorder, or a Personality Disorder).

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Table 2: Sample Restrictions Applied to the NLSY-C and ECLS-B Data

	NLSY-C	ECLS-B
Full Sample	11,500	10,700
Mothers 18-29 years at birth	11,500	6,100
Children born 1983-1986 (If NLSY-C)	3,000	N/A
Randomly-selected child if siblings in sample (Randomly-selected twin, if applicable)	2,600	5,700
Non-missing on externalizing	2,000	4,600

Note: In compliance with ECLS-B restricted-used reporting guidelines (and for comparability in reporting across datasets), sample sizes are rounded to the closest 50.

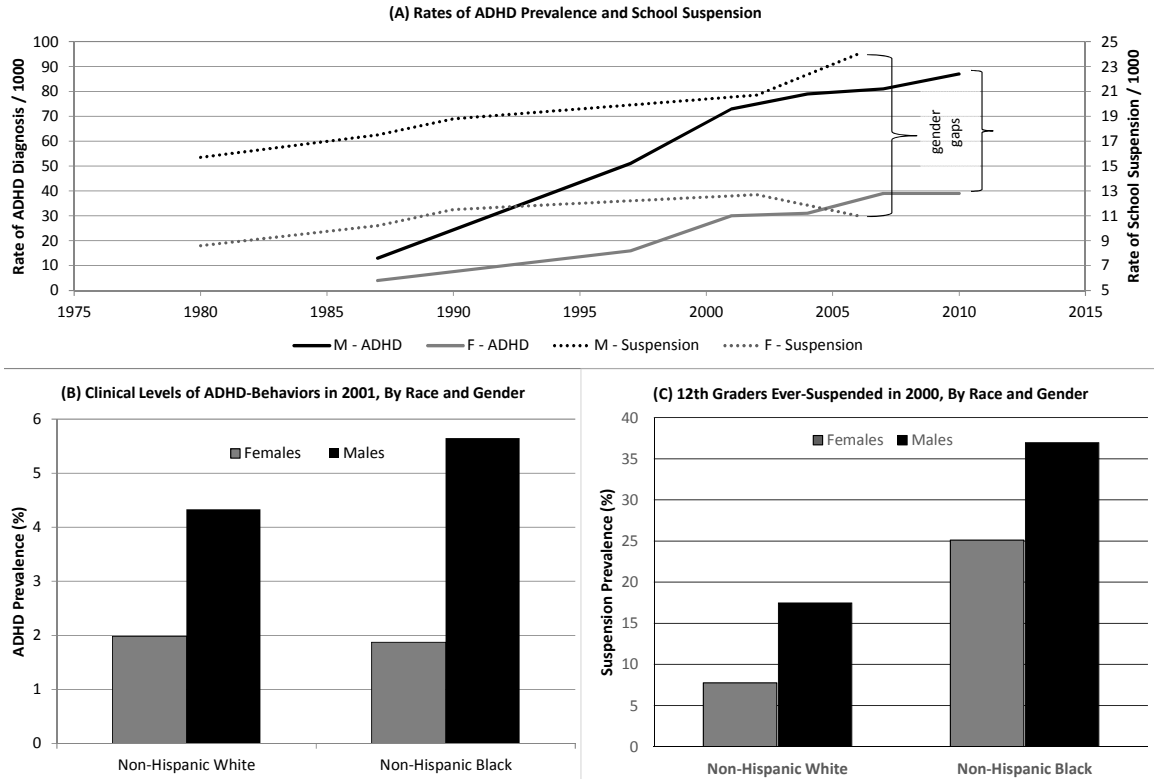
Table 3: Items in the Externalizing Problems Scale (and Self-Regulation and Social Problems Sub-Scales)

Scale/Subscale Name:	CBCL (BPI) EXTERNALIZING ITEMS AVAILABLE IN **NLSY-C**[1]	COMPARABLE PKBS-2 (PBS) EXTERNALIZING ITEMS AVAILABLE IN THE **ECLS-B**[2]
SELF-REGULATION PROBLEMS:		
Attention Problems/Overactive	Is impulsive/acts w/o thinking	Acts impulsively
	Is restless, overly active, can't sit still	Is overly active
SOCIAL PROBLEMS:		
Antisocial/Aggressive	Has trouble getting along w/ other kids	Is (not) invited to play by other children (reverse-coded)
	Breaks things on purpose	Destroys others' things
	Is not liked by other children	Is (not) liked by other children (reverse-coded)
Self-Centered/Explosive	Has strong temper and loses it easily	Has temper tantrums
	Cronbach's Alpha: 0.70	Cronbach's Alpha: 0.75

[1]Within the NLSY-C's more general Behavior Problems Index (BPI; developed by Peterson and Zill 1986) were 10 externalizing items. Of these, 6 overlapped almost identically with those available in the ECLS-B. The item listed in italics is included in the BPI-based externalizing scale, but not in the CBCL-based externalizing scale (see Guttmannova et al. 2007 for a discussion of why CBCL measures are more valid than the BPI items). However, to maximize coverage, it is included in the present externalizing scale.

[2]The ECLS-B includes a total of 8 externalizing items from the broader Problem Behaviors Scale of the Pre-Kindergarten Behavioral Skills, 2nd Ed. scale. Two PKBS-2 items (Child is physically aggressive and Child is angry) available in the ECLS-B were not used in the present scale due to non-corresponding items in the NLSY-C. However, the subset of items used in the present externalizing scale include at least one item from each of the three primary externalizing sub-scales (attention problems/overactive, etc.) listed above. In order to correspond to the NLSY-C scale of: 1=not true/rarely, 2=sometimes, 3=often, the ECLS-B items are rescaled from 1 (never)-5 (very often) to 1-3 using two approaches: (1) merging of extreme categories ("very often" with "often" and "never" with "rarely"); (2) a linear rescaling using the formula: x *.5 + .5.

Figure 1: Rates of ADHD Prevalence and School Suspension Rates, by Year and Gender or Race and Gender



Sources: **Panel A:** [1]Bertrand, M., & Pan, J. (2011). The trouble with boys: social influences and the gender gap in disruptive behavior (No. w17541). *National Bureau of Economic Research.* [2]Olfson, M., Gameroff, M. J., Marcus, S. C., & Jensen, P. S. (2003). National trends in the treatment of attention deficit hyperactivity disorder. *American Journal of Psychiatry*, 160(6), 1071-1077. [3]Centers for Disease Control. National Health Interview Surveys, 1998, 2001, 2004, 2007, 2010. **Panel B:** [4]Cuffe, S. P., Moore, C. G., & McKeown, R. E. (2005). Prevalence and correlates of ADHD symptoms in the national health interview survey. *Journal of Attention Disorders*, 9(2), 392-401. **Panel C:** In order to preserve comparability with rates of ADHD prevalence, results for school suspension are interpolated from NCES data from 1992 and 2007 using: [5]Snyder, T. D. & Pratt, R. (1997). The condition of education, 1997. *National Center for Education Statistics (NCES)* (October). Online: http://nces.ed.gov/pubsearch/pubsinfo.asp?pubid=97980. [6]Aud, S., Hussar, W. & Planty, M. (2010). The condition of education 2010. *NCES* (May). Online: http://nces.ed.gov/pubsearch/pubsinfo.asp?pubid=2010028.

Figure 2: Hypothetical Counterfactual Population Shifts in Externalizing Problems Distributions, by Gender and Cohort

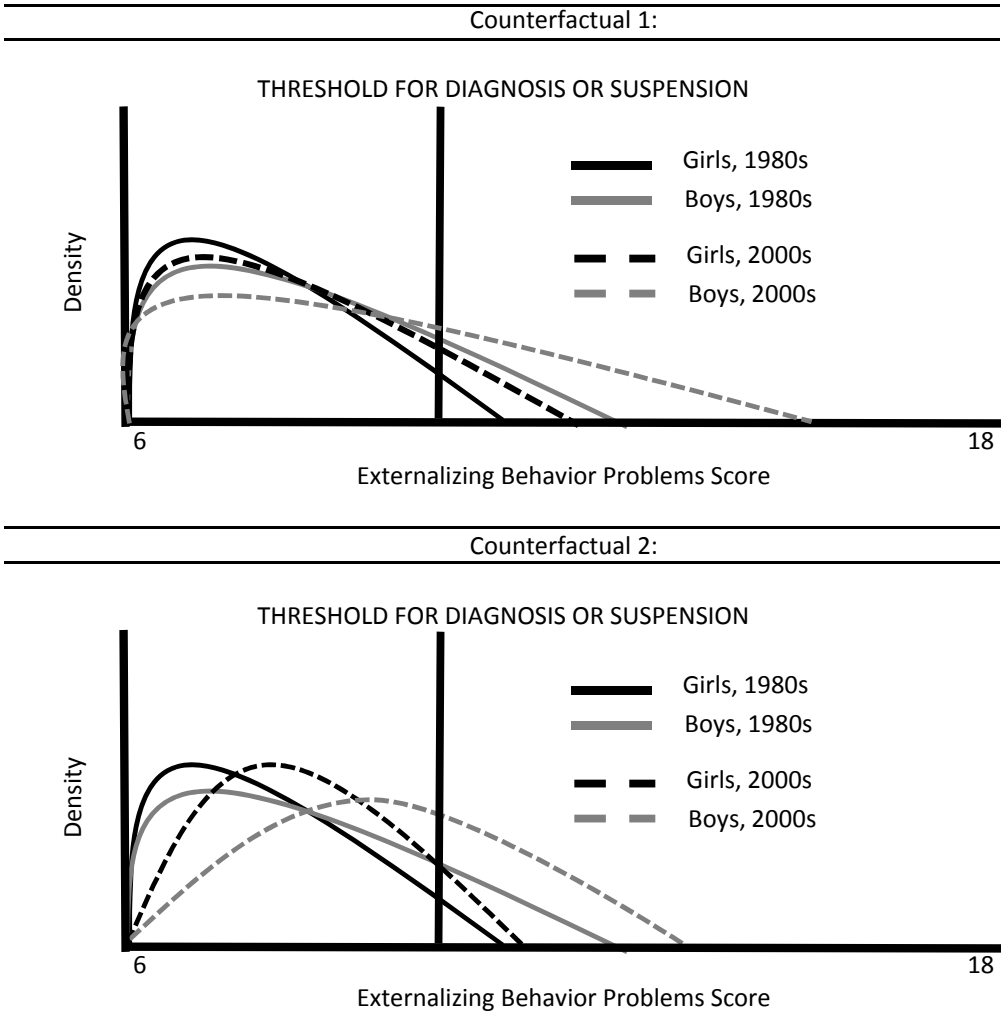


Figure 3: Data Structure and Behavioral Measures Coverage

Year	Age 0	1	2	3	4	5	6
1983	NLSY-C						
1984	NLSY-C	NLSY-C					
1985	NLSY-C	NLSY-C	NLSY-C				
1986	NLSY-C	NLSY-C	NLSY-C	NLSY-C			
1987		NLSY-C	NLSY-C	NLSY-C	NLSY-C		
1988			NLSY-C	NLSY-C	NLSY-C	NLSY-C	
1989				NLSY-C	NLSY-C	NLSY-C	NLSY-C
1990					NLSY-C	NLSY-C	NLSY-C
1991						NLSY-C	NLSY-C
1992							NLSY-C
1993							
1994							
1995							
1996							
1997							
1998							
1999							
2000							
2001	ECLS-B						
2002		ECLS-B					
2003			ECLS-B				
2004				ECLS-B			
2005					ECLS-B		
2006						ECLS-B	
2007							ECLS-B
2008							

NOTE: Grey text indicates that behavioral skills were not measured in a given year or at a given age (the complete set of behavioral skills were not collected for children under age 4).

Figure 4: Distribution of Externalizing Problems by Gender, Race, and Time Period

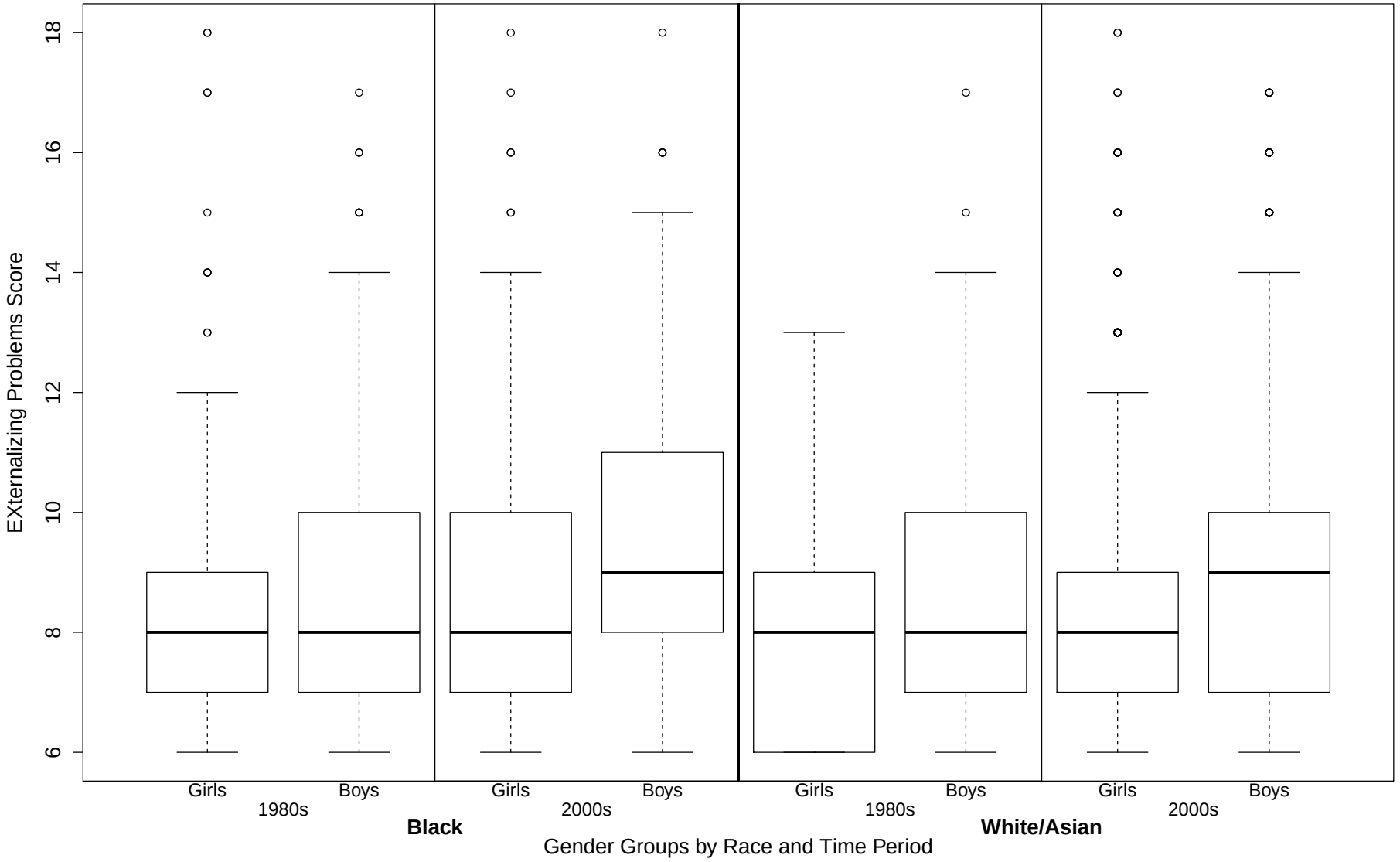


Figure 5: Gender Gap in Externalizing Problems at the 10th, 25th, 50th, 75th, and 90th Percentiles Within Girls' and Boys' Respective Behavioral Distributions and Change in Gender Gaps Between the 1980s and 2001 Cohorts, by Race

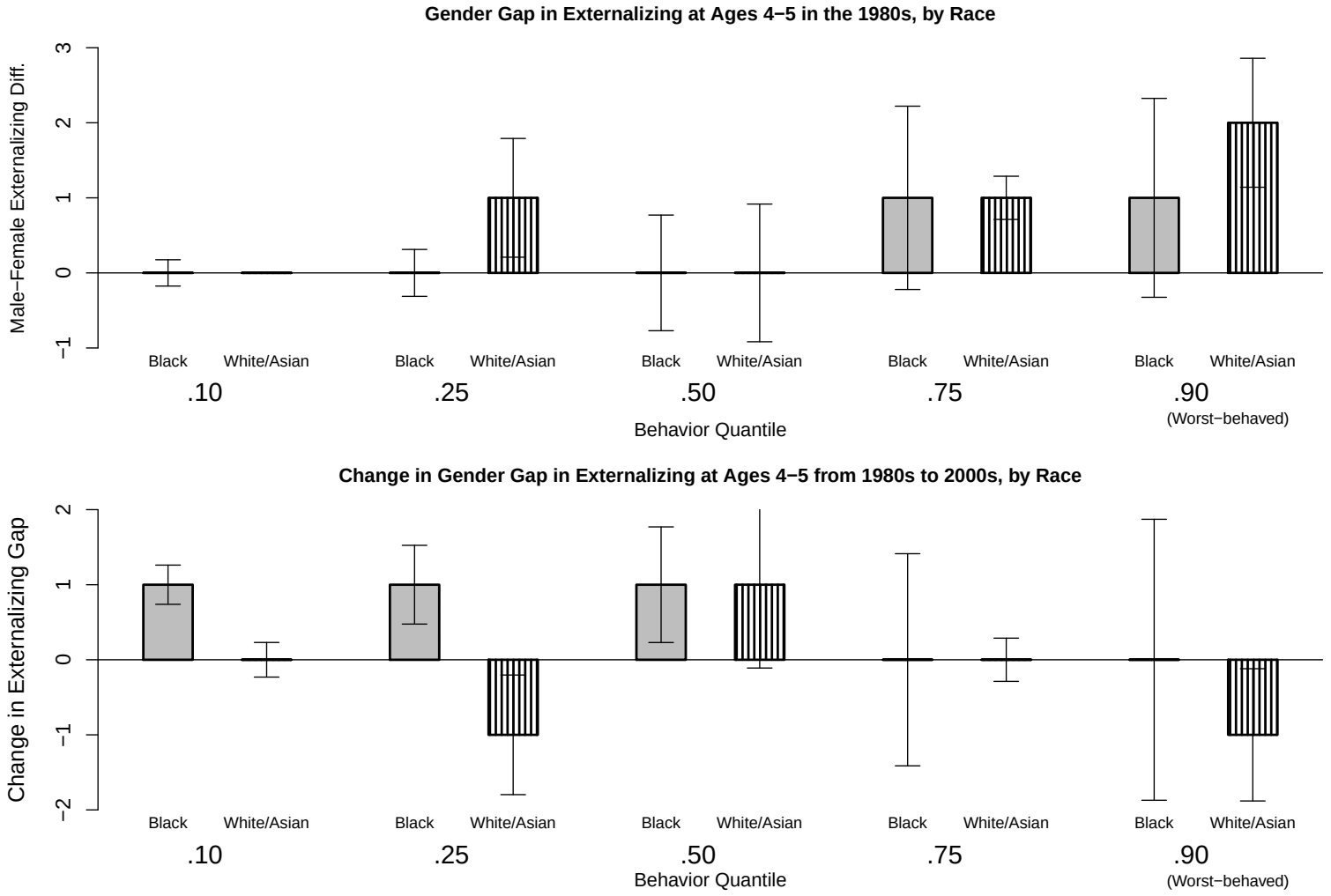
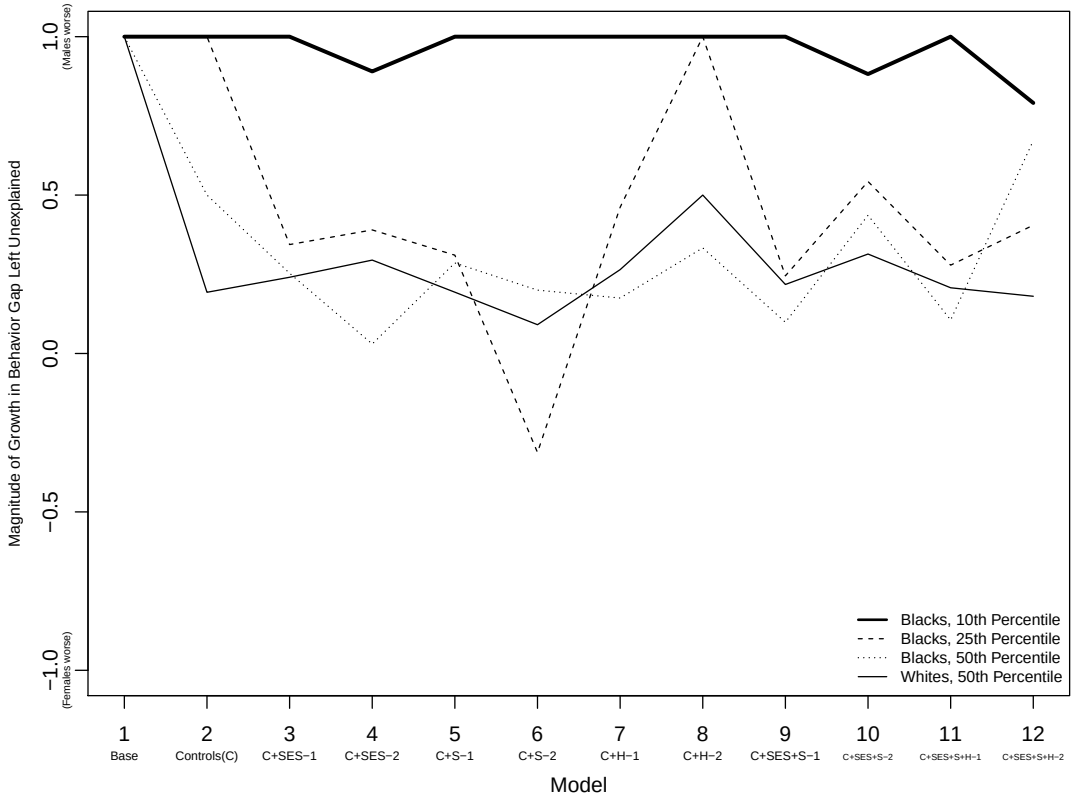


Figure 6: Potential Family and Health Mediators of the Growth of the Gender Gap in the Location of the 10th, 25th, and 50th Percentiles of Externalizing Behavior Problems at Ages 4-5, by Race



Notes: See the Appendix for the full table of results. Model labels refer to: Controls (C): Internalizing problems (centered), mother's age at birth (centered), birth order, dummies for year of birth (1984-1986); SES (I): mother's years of schooling at birth (centered) and per capita household income at child age 4, in $1,000s, adjusted to 2011 dollars (centered); SES (II): mother's years of schooling and per capita household income separately interacted with each of the following: male, time period (2001 cohort indicator variable), and male*time period; Family Structure (S) (I): father absent at birth dummy and dummies for single mother at age 4 or social father at age 4 (two biological parents at age 4 is the reference); S (II): interactions between each of the S (I) variables and male, time period, and male*time period; Early Childhood Health (H) (I): a dummy for low birth weight, a dummy for pre-term, and a dummy for asthma diagnosis by age 4 or 5; H (II): interactions between each H (I) variable and male, time period, and male*time period.

Appendix A

Trends in Potential Environmental Drivers of the Growing Gender Gap in Early Childhood Externalizing Problems

This section provides a detailed look at trends in family structure and early childhood health, with an eye toward gender and racial gaps in each, where data are available.

Family Structure

Research documents the relationship between family structure and child psychological well-being across a variety of externalizing behavior problems-linked outcomes, including suspension and drop-out (Sigle-Rushton and McLanahan 2002; Thomson et al. 1994).[22] Rates of non-marital childbearing and single motherhood in the U.S. have increased from roughly 4% and 7% in 1960 to 17% and nearly 25% in the early 2000s, as shown in Panel A of Figure A.1 (McLanahan and Percheski 2008).

[FIGURE A.1 ABOUT HERE]

Boys raised in single-parent families tend to have significantly higher levels of externalizing problems than girls (Alexander et al. 1997; Demo and Acock 1988), as do boys who experience mother partnership instability (Cooper et al. 2011). As single mother households become more widespread, boys may externalize more frequently and/or more severely than girls (Davies and Lindsay 2004). This may be because fathers serve as important male role models at a time when boys are first learning to self-regulate and express emotions in socially-acceptable ways (Buchmann et al. 2008).[23]

In 1970, just over 30% of black children lived with a single parent, compared to just under 10% of white children (Sigle-Rushton and McLanahan 2002). By 2005, the percentage-point difference had increased from roughly 20 percentage-points to roughly 30 percentage-points amid a steady upward trend for all children (Child Trends 2013). In light of boys' higher levels of externalizing problems compared to girls in single-parent families, the increasing rate of black children in single parent households predicts a growth of the absolute magnitude of the externalizing problems gap. With rising prevalence, the gender gap may have emerged among black children with lower behavior problems scores.

Some research suggests that family SES explains much of the family structure-externalizing

problems relationship (Thomson et al. 1994). Children raised in higher status and higher-income families receive lower behavior problems scores, as do children during periods of relative increase in family income (Dearing et al. 2006; Hill et al. 2013; Miller and Chen 2013; Raver 2004; Thomson et al. 1994). Boys may externalize more than girls in response to economic hardship (Shaw et al. 1994, 1998). An important concern, then, is to differentiate between these effects on children's externalizing problems.

Low Birth Weight (LBW)

Low birth weight (LBW; weighing less than 2,500 grams or 5.5 pounds at birth) is linked to hyperactivity, attention problems, and ADHD diagnosis (Pharoah et al. 1994). One study shows that LBW children were roughly three times more likely to be diagnosed with ADHD, controlling for demographic and health factors (Mick et al. 2002).

Although LBW is more common among females (McGregor et al. 1992) and the gender gap in rates of LBW has held steady in recent decades (Panel B of Figure A.1), the gender gap in birth weight among at-term births (37-41 weeks gestation) has decreased from about 135 grams in 1980 to 125 grams in 2000 (Van Vliet et al. 2009). Because birth weights are trending downwards more for males than females, rates of pre-term birth are rising faster for males than females. As a result, the risk for behavior problems may have increased more among males. Some researchers call this the "the paradox of LBW": even though a higher proportion of female than male births are LBW, infant mortality rates are higher among males than females (Rothman et al. 2008). Despite lower prevalence, LBW males are more likely than LBW females to experience externalizing behavior problems in early childhood (Pharoah et al. 1994).

Rates of LBW also are almost twice as high among black than white children (Paneth 1995; Reichman 2005). And, LBW is more strongly linked to attention problems, a key component of externalizing problems, among black compared to white children (Breslau et al. 2000). The greater prevalence of behavior problems among LBW males than females combined with the larger proportion of LBW black males than white males, suggests the gender gap in externalizing problems may be larger among black than white children.

Pre-Term Birth

Even moderately pre-term birth (delivery prior to 37 weeks of gestation) is linked to behavioral disorders like ADHD, autism, and increased levels of externalizing problems (Bhutta et al. 2002; Potijk et al. 2012). Pre-term births are on the rise in the United States (Demissie et al. 2001). As shown in Panel B of Figure A.1, national rates of pre-term birth are roughly 1% higher for males than females (Department of Health and Human Services 2005; Ingemarsson 2003; Zeitlin et al. 2002). In 1983, the rate of pre-term birth was 0.78 percentage-points higher for males than females; by 2001 this difference had grown to 1.05 percentage points, or 35% over these two decades (Department of Health and Human Services 2005). Although modest, the growth of the pre-term birth rates gap equates to tens of thousands more males than females born pre-term in the 2000s compared to the 1980s.

Pre-term and LBW babies are at higher risk for asthma (Bhutta et al. 2002), slow behavioral development, and behavioral disorders (Elsmen et al. 2004). The precise social and biological mechanisms are unclear, but some studies show that pre-term boys are at higher risk than comparable pre-term girls (Elsmen et al. 2004).

Pre-term birth rates are highest among blacks. Between 1989 and 1997, pre-term births rose from 8.8% to 10.2% of live births among whites, fell by 7.6% among blacks, but were still 17.5% of black births (down from 19.0% in 1989) (Demissie et al. 2001). Research on trends in pre-term birth by race and gender is limited, but the significantly larger male-to-female ratio of pre-term births among blacks compared to whites suggests pre-term birth is declining faster among black females than males. In this case, the pre-term gap may be growing more among blacks than whites, even amid declining pre-term rates for blacks. The externalizing problems gap may similarly appear among a more behaviorally-advantaged cross-section of children, especially minority children.

Asthma

Results from the Child Health Supplement of the National Health Interview Survey (NHIS) documents that asthma diagnosis in early childhood is linked to a behavioral outcome closely related to externalizing problems: first grade retention (Byrd and Weitzman 1994). Panel D of Figure A.1 shows that rates of diagnosed asthma prevalence among

children 2-5 years increased fastest for boys between the 1980s and the 2000s.

Asthma diagnosis is highest overall among black children, with 2001-2003 *National Surveillance of Asthma* estimates indicating prevalence rates of roughly 16% for black boys and 10% for black girls, compared to 5% for white boys and under 4% for white girls (Moorman et al. 2007). These data, collected by the National Center for Health Statistics, indicate that the absolute magnitude of the asthma gap for children ages 0-4 in 2001-03 is significantly larger among black compared to white and Hispanic children. The growing absolute gender gap in asthma diagnosis, largest among black children, predicts a growing gender gap in externalizing problems also concentrated among black children.

Changes in the Externalizing Problems Distribution with Rescaling from a Five-Point to Three-Point Scale

The difference in scaling between the three-point NLSY-C items and the five-point ECLS-B items raise the potential for response bias based on psychological processes of item anchoring (Schwarz 1999). In order to understand what effects this may have on the rescaling of the ECLS-B externalizing problems scale for concordance across datasets, Figure A.2 displays the NLSY-C 18-point scale in panel 1, the rescaled ECLS-B 18-point scale based on the merging of extreme categories with its nearest neighbor category, and the original 30-point ECLS-B scale. These plots indicate that the nearest neighbor merging strategy changes the shape of the distribution of the 30-point ECLS-B scale to a non-normal distribution that mirrors that of the 18-point NLSY-C scale.

Because the focus of the study is on gender differences in reports of frequencies of externalizing problems, another first-order concern is whether the rescaling of the ECLS-B externalizing problems scale from 30-points to 18-points through the nearest neighbor merging strategy introduces bias along lines of gender. To examine the extent to which this may be happening, Figure A.3 displays sample size-adjusted differences in the frequencies with which females and males receive a given score on both the 18-point and 30-point externalizing problems scale in the ECLS-B data. Results indicate a similar pattern across scales whereby females are overrepresented at the lowest problem scores (i.e., 6-8 in the

18-point scale and 6-13 in the 30-point scale) while the opposite is true at the higher problem scores.

Finally, Figure A.4 presents sample size-adjusted differences across datasets in the frequencies with which females and males receive each externalizing problems score on the 18-point scale. Results indicate that both scales follow the same general pattern discussed above, but with some changes in the female and male distributions across cohorts.

Table A.1: Potential Family and Health Mediators of the Gender Gap in the Location of the Conditional 10th Percentile of Externalizing among Black Children Ages 4-5

VARIABLES	(1) NC:	(2) C:	(3) C + SES (I):	(4) C + SES (II):	(5) C +S (I):	(6) C + S (II):	(7) C + H (I):	(8) C + H (II):	(9) C + SES + S (I):	(10) C + SES + S (II):	(11) C + SES + S + H (I):	(12) C + SES + S + H (II):
2001 Birth Cohort	0.000	-0.000	-0.000	-0.000	-0.000	-0.000	-0.000	-0.000	-0.000	0.001	-0.000	-0.064
	(0.000)	(0.253)	(0.214)	(0.222)	(0.231)	(0.185)	(0.223)	(0.265)	(0.224)	(0.599)	(0.208)	(0.200)
Male	-0.000	-0.000	-0.000	-0.000	0.000	0.000	0.000	0.000	0.000	-0.015	0.000	0.074
	(0.109)	(0.080)	(0.133)	(0.174)	(0.126)	(0.102)	(0.096)	(0.180)	(0.128)	(0.659)	(0.147)	(0.175)
2001 Birth Cohort * Male	1.000***	1.000***	1.000***	0.890***	1.000***	1.000**	1.000***	1.000**	1.000***	0.882	1.000***	0.791*
	(0.140)	(0.160)	(0.186)	(0.221)	(0.181)	(0.384)	(0.190)	(0.313)	(0.192)	(0.826)	(0.280)	(0.340)
Socio-Economic Status (SES):												
Mother's Years of Schooling at Birth[1]			-0.000	0.000					-0.000	-0.004	-0.000	0.011
			(0.009)	(0.186)					(0.018)	(0.515)	(0.026)	(0.041)
Per Capita Household Income at Age 4 (in $1,000s, 2011 dollars)[1]			-0.000	0.000					0.000	0.004	0.000	0.004
			(0.001)	(0.013)					(0.002)	(1.145)	(0.003)	(0.005)
Mother's School * Male				-0.000						0.137		0.092
				(0.193)						(0.096)		(0.088)
Household Income * Male				0.000						-0.004		0.002
				(0.018)						(0.014)		(0.014)
Mother's School * 2001 Cohort				-0.000						0.004		-0.019
				(0.184)						(1.321)		(0.064)
Household Income * 2001 Cohort				0.000						-0.004		-0.004
				(0.013)						(0.068)		(0.010)
Mother's School * Male * 2001 Cohort				-0.121						-0.403		-0.332*
				(0.155)						(1.075)		(0.139)
Household Income * Male * 2001 Cohort				0.007						0.022		0.018
				(0.016)						(0.343)		(0.020)
Family Structure:												
Father Absent at Birth					-0.000	0.000			-0.000	0.004	-0.000	0.032
					(0.016)	(0.073)			(0.026)	(1.645)	(0.054)	(0.167)
Single Mother at Age 4					0.000	-0.000			0.000	0.046	0.000	0.058
					(0.035)	(0.144)			(0.057)	(0.155)	(0.091)	(0.186)
Social Father at Age 4					0.000	-0.000			0.000	0.019	0.000	-0.041
					(0.039)	(0.399)			(0.065)	(0.444)	(0.089)	(0.450)
Two Biological Parents at Age 4 (=reference)					ref	ref			ref	ref	ref	ref
Father Absent at Birth * Male						-0.000				0.113		0.124
						(0.398)				(0.329)		(0.353)
Single Mother at Age 4 * Male						1.000*				0.558		0.498
						(0.471)				(0.376)		(0.359)
Social Father at Age 4 * Male						1.000				1.186+		1.037
						(0.625)				(0.718)		(0.780)
Father Absent at Birth * 2001 Birth Cohort						-0.000				-0.004		-0.008
						(0.113)				(2.525)		(0.259)
Single Mother at Age 4 * 2001 Birth Cohort						0.000				-0.046		-0.070
						(0.163)				(2.934)		(0.280)
Social Father at Age 4 * 2001 Birth Cohort						0.000				-0.019		0.036
						(0.456)				(5.947)		(0.548)
Father Absent at Birth * Male * 2001 Birth Cohort						0.000				-0.903		-0.842
						(0.596)				(1.081)		(0.527)
Single Mother at Age 4 * Male * 2001 Birth Cohort						-1.000				0.131		0.103
						(0.657)				(0.680)		(0.566)
Social Father at Age 4 * Male * 2001 Birth Cohort						-1.000				-0.719		-0.687
						(0.910)				(0.831)		(0.952)
Early Childhood Health:												
Low Birth Weight (<5.5 pounds, 2,500 g)							-0.000	-0.000			-0.000	-0.247
							(0.053)	(0.593)			(0.105)	(0.441)
Pre-Term Birth (<37 weeks gestation)							0.000	0.000			0.000	0.237
							(0.040)	(0.513)			(0.137)	(0.342)
Asthma Diagnosis by Age 4 or 5							-0.000	-0.000			-0.000	-0.084
							(0.017)	(0.250)			(0.075)	(0.189)
Low Birth Weight * Male								0.000				0.037
								(0.338)				(0.346)
Pre-Term Birth * Male								-0.000				0.112
								(0.430)				(0.336)
Asthma Diagnosis * Male								0.000				-0.083
								(0.286)				(0.251)
Low Birth Weight * 2001 Cohort								0.000				0.272
								(0.548)				(0.434)
Pre-Term Birth * 2001 Cohort								0.000				-0.208
								(0.523)				(0.364)
Asthma Diagnosis * 2001 Cohort								-0.000				0.070
								(0.261)				(0.217)
Controls:												
Internalizing[1]		-0.000	-0.000	0.000	-0.000	0.000	-0.000	-0.000	-0.000	0.118	-0.000	0.149
		(0.103)	(0.104)	(0.521)	(0.111)	(0.130)	(0.110)	(0.125)	(0.107)	(2.316)	(0.134)	(0.111)
Mother's Age at Birth[1]		0.000	0.000	-0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.006
		(0.000)	(0.003)	(0.006)	(0.004)	(0.006)	(0.002)	(0.003)	(0.008)	(0.309)	(0.015)	(0.019)
Birth Order		-0.000	-0.000	0.000	-0.000	0.000	-0.000	-0.000	-0.000	-0.000	-0.000	-0.013
		(0.000)	(0.010)	(0.013)	(0.007)	(0.008)	(0.009)	(0.016)	(0.019)	(2.937)	(0.047)	(0.050)
Child Born 1983		ref	ref	ref	ref	ref	ref	ref	ref	ref	ref	ref
Child Born 1984		-0.000	-0.000	0.000	-0.000	-0.000	-0.000	-0.000	-0.000	-0.017	-0.000	-0.087
		(0.263)	(0.231)	(0.237)	(0.258)	(0.184)	(0.238)	(0.268)	(0.235)	(0.182)	(0.232)	(0.167)
Child Born 1985		-0.000	-0.000	-0.000	-0.000	-0.000	-0.000	-0.000	-0.000	-0.015	-0.000	-0.124
		(0.271)	(0.250)	(0.259)	(0.241)	(0.183)	(0.252)	(0.272)	(0.243)	(0.191)	(0.218)	(0.196)
Child Born 1986		-0.000	-0.000	-0.000	-0.000	-0.000	-0.000	-0.000	-0.000	-0.102	-0.000	-0.199
		(0.254)	(0.217)	(0.236)	(0.233)	(0.207)	(0.224)	(0.272)	(0.229)	(0.203)	(0.224)	(0.195)
Constant	6.000	6.000***	6.000***	6.000***	6.000***	6.000***	6.000***	6.000***	6.000***	6.077***	6.000***	6.179***
	(0.000)	(0.260)	(0.231)	(0.476)	(0.237)	(0.199)	(0.233)	(0.281)	(0.238)	(0.430)	(0.248)	(0.215)
Observations	1,450	1,450	1,450	1,450	1,450	1,450	1,450	1,450	1,450	1,450	1,450	1,450

[1]Specified variables (including within interactions) are centered at the overall sample mean.

Standard errors in parentheses. *** p<0.001, ** p<0.01, * p<0.05, + p<0.10. Model estimates are based on multiple imputation of 20 datasets. NC = No Controls, C = Controls, SES = Family Socioeconomic and Cultural Resources, S = Family Structure, H = Early Childhood Health. Model (I) refers to main effects models, Model (II) refers to models with interactions by gender, time period, and gender * time period.

Table A.2: Potential Family and Health Mediators of the Gender Gap in the Location of the Conditional 25th Percentile of Externalizing among Black Children Ages 4-5

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
VARIABLES	NC:	C:	C + SES (I):	C + SES (II):	C +S (I):	C + S (II):	C + H (I):	C + H (II):	C + SES + S (I):	C + SES + S (II):	C + SES + S + H (I):	C + SES + S + H (II):
2001 Birth Cohort	0.000	-0.000	0.156	0.030	0.224	0.438	-0.154	-0.500	0.290	0.139	0.136	-0.015
	(0.078)	(0.250)	(0.233)	(0.239)	(0.235)	(0.475)	(0.278)	(0.345)	(0.222)	(0.421)	(0.247)	(0.367)
Male	-0.000	-0.000	0.469+	0.387	0.414	0.750+	0.462+	0.500+	0.484*	0.524	0.458*	0.496
	(0.109)	(0.307)	(0.255)	(0.632)	(0.253)	(0.397)	(0.238)	(0.277)	(0.220)	(0.373)	(0.201)	(0.366)
2001 Birth Cohort * Male	1.000***	1.000*	0.344	0.390	0.310	-0.313	0.462	1.000*	0.245	0.543	0.279	0.405
	(0.228)	(0.451)	(0.371)	(0.297)	(0.354)	(0.589)	(0.324)	(0.414)	(0.307)	(0.515)	(0.285)	(0.512)
Socio-Economic Status (SES):												
Mother's Years of Schooling at Birth[1]			-0.125*	0.008					-0.113*	0.019	-0.096*	0.016
			(0.060)	(0.247)					(0.050)	(0.055)	(0.045)	(0.060)
Per Capita Household Income at Age 4 (in $1,000s, 2011 dollars)[1]			0.000	0.001					0.003	0.003	0.001	0.003
			(0.006)	(0.020)					(0.005)	(0.006)	(0.005)	(0.006)
Mother's School * Male				0.009						0.039		0.052
				(1.960)						(0.102)		(0.097)
Household Income * Male				-0.006						0.004		0.003
				(0.505)						(0.019)		(0.019)
Mother's School * 2001 Cohort				-0.169						-0.130		-0.176+
				(0.178)						(0.085)		(0.097)
Household Income * 2001 Cohort				-0.003						0.013		0.006
				(0.020)						(0.019)		(0.017)
Mother's School * Male * 2001 Cohort				-0.171						-0.249+		-0.218
				(0.148)						(0.138)		(0.135)
Household Income * Male * 2001 Cohort				0.007						-0.013		0.002
				(0.026)						(0.028)		(0.027)
Family Structure:												
Father Absent at Birth					0.103	-0.062			0.141	0.018	0.222	-0.053
					(0.190)	(0.258)			(0.157)	(0.242)	(0.157)	(0.241)
Single Mother at Age 4					0.345	0.625+			0.202	0.502	0.158	0.514+
					(0.225)	(0.344)			(0.198)	(0.333)	(0.185)	(0.285)
Social Father at Age 4					-0.241	-0.125			-0.133	-0.172	-0.298	-0.363
					(0.350)	(0.793)			(0.282)	(0.839)	(0.259)	(0.815)
Two Biological Parents at Age 4 (=reference)					ref	ref			ref	ref	ref	ref
Father Absent at Birth * Male						-0.063				-0.121		-0.072
						(0.530)				(0.478)		(0.500)
Single Mother at Age 4 * Male						-0.375				0.082		0.048
						(0.580)				(0.534)		(0.517)
Social Father at Age 4 * Male						0.938				1.287		1.502
						(1.032)				(1.145)		(1.112)
Father Absent at Birth * 2001 Birth Cohort						1.062*				0.860*		0.915*
						(0.414)				(0.378)		(0.372)
Single Mother at Age 4 * 2001 Birth Cohort						-1.187*				-0.720		-0.959*
						(0.558)				(0.510)		(0.426)
Social Father at Age 4 * 2001 Birth Cohort						-0.437				-0.022		0.041
						(0.900)				(0.907)		(0.898)
Father Absent at Birth * Male * 2001 Birth Cohort						-1.000				-1.158+		-1.322*
						(0.773)				(0.629)		(0.632)
Single Mother at Age 4 * Male * 2001 Birth Cohort						1.812*				0.537		1.065
						(0.875)				(0.754)		(0.729)
Social Father at Age 4 * Male * 2001 Birth Cohort						-0.375				-1.282		-1.352
						(1.240)				(1.200)		(1.196)
Early Childhood Health:												
Low Birth Weight (<5.5 pounds, 2,500 g)							0.231	0.000			0.036	-0.417
							(0.231)	(0.859)			(0.188)	(0.769)
Pre-Term Birth (<37 weeks gestation)							0.615*	0.500			0.489*	0.448
							(0.269)	(0.374)			(0.196)	(0.378)
Asthma Diagnosis by Age 4 or 5							-0.231	0.000			-0.060	-0.138
							(0.265)	(0.279)			(0.195)	(0.262)
Low Birth Weight * Male								-0.500				0.018
								(0.458)				(0.322)
Pre-Term Birth * Male								0.000				-0.288
								(0.458)				(0.376)
Asthma Diagnosis * Male								-0.500				0.327
								(0.426)				(0.374)
Low Birth Weight * 2001 Cohort								0.500				0.533
								(0.837)				(0.745)
Pre-Term Birth * 2001 Cohort								0.500				0.348
								(0.424)				(0.430)
Asthma Diagnosis * 2001 Cohort								-0.000				-0.149
								(0.431)				(0.355)
Controls:												
Internalizing[1]		-0.000	0.438**	0.425**	0.414**	0.438**	0.462**	0.500***	0.419***	0.430***	0.448***	0.458***
		(0.204)	(0.158)	(0.145)	(0.145)	(0.152)	(0.141)	(0.148)	(0.124)	(0.103)	(0.096)	(0.091)
Mother's Age at Birth[1]		0.000	0.031	0.023	0.017	-0.000	-0.000	-0.000	0.030	0.031	0.026	0.030
		(0.023)	(0.028)	(0.085)	(0.023)	(0.019)	(0.023)	(0.022)	(0.025)	(0.026)	(0.026)	(0.026)
Birth Order		-0.000	0.000	0.003	0.086	0.063	0.077	0.000	0.012	0.045	0.020	0.038
		(0.066)	(0.066)	(0.177)	(0.075)	(0.072)	(0.061)	(0.058)	(0.066)	(0.077)	(0.077)	(0.078)
Child Born 1983		ref	ref	ref	ref	ref	ref	ref	ref	ref	ref	ref
Child Born 1984		-0.000	-0.219	-0.219	-0.155	-0.000	-0.154	0.000	-0.120	-0.181	-0.115	-0.193
		(0.291)	(0.245)	(0.245)	(0.275)	(0.277)	(0.248)	(0.284)	(0.263)	(0.260)	(0.252)	(0.233)
Child Born 1985		-0.000	-0.250	-0.135	-0.138	-0.062	-0.077	0.000	-0.102	-0.220	-0.137	-0.279
		(0.309)	(0.288)	(0.281)	(0.283)	(0.280)	(0.275)	(0.307)	(0.266)	(0.265)	(0.262)	(0.265)
Child Born 1986		-0.000	-0.219	-0.183	-0.121	-0.125	-0.385	0.000	-0.031	-0.286	-0.184	-0.366
		(0.369)	(0.309)	(0.307)	(0.311)	(0.324)	(0.307)	(0.326)	(0.289)	(0.296)	(0.277)	(0.294)
Constant	7.000***	7.000***	6.920***	7.041***	6.476***	6.352***	6.767***	6.831***	6.621***	6.592***	6.578***	6.707***
	(0.045)	(0.246)	(0.236)	(0.402)	(0.318)	(0.379)	(0.229)	(0.252)	(0.276)	(0.373)	(0.280)	(0.353)
Observations	1,450	1,450	1,450	1,450	1,450	1,450	1,450	1,450	1,450	1,450	1,450	1,450

[1]Specified variables (including within interactions) are centered at the overall sample mean.

Standard errors in parentheses. *** p<0.001, ** p<0.01, * p<0.05, + p<0.10. Model estimates are based on multiple imputation of 20 datasets. NC = No Controls, C = Controls, SES = Family Socioeconomic and Cultural Resources, S = Family Structure, H = Early Childhood Health. Model (I) refers to main effects models, Model (II) refers to models with interactions by gender, time period, and gender * time period.

Table A.3: Potential Family and Health Mediators of the Gender Gap in the Location of the Conditional 50th Percentile of Externalizing among Black Children Ages 4-5

VARIABLES	(1) NC:	(2) C:	(3) C + SES (I):	(4) C + SES (II):	(5) C +S (I):	(6) C + S (II):	(7) C + H (I):	(8) C + H (II):	(9) C + SES + S (I):	(10) C + SES + S (II):	(11) C + SES + S + H (I):	(12) C + SES + S + H (II):
2001 Birth Cohort	-0.000	0.500+	0.548*	0.540*	0.429	0.400	0.225	0.167	0.541*	0.351	0.297	-0.002
	(0.100)	(0.288)	(0.248)	(0.251)	(0.266)	(0.363)	(0.283)	(0.299)	(0.215)	(0.340)	(0.248)	(0.369)
Male	-0.000	0.500+	0.570*	0.661**	0.571*	0.600+	0.642*	0.667*	0.676**	0.478	0.620*	0.437
	(0.406)	(0.281)	(0.242)	(0.254)	(0.240)	(0.357)	(0.249)	(0.262)	(0.212)	(0.364)	(0.243)	(0.351)
2001 Birth Cohort * Male	1.000*	0.500	0.253	0.030	0.286	0.200	0.175	0.333	0.099	0.436	0.106	0.672
	(0.406)	(0.333)	(0.293)	(0.306)	(0.303)	(0.517)	(0.309)	(0.359)	(0.278)	(0.470)	(0.284)	(0.463)
Socio-Economic Status (SES):												
Mother's Years of Schooling at Birth[1]			-0.139*	0.066					-0.146*	0.046	-0.122+	0.056
			(0.066)	(0.133)					(0.062)	(0.138)	(0.063)	(0.128)
Per Capita Household Income at Age 4 (in $1,000s, 2011 dollars)[1]			-0.003	-0.003					-0.002	-0.003	-0.002	-0.004
			(0.005)	(0.013)					(0.005)	(0.013)	(0.006)	(0.013)
Mother's School * Male				-0.120						-0.081		-0.092
				(0.153)						(0.166)		(0.166)
Household Income * Male				0.003						0.009		0.010
				(0.021)						(0.025)		(0.025)
Mother's School * 2001 Cohort				-0.238						-0.197		-0.183
				(0.157)						(0.161)		(0.155)
Household Income * 2001 Cohort				0.002						0.003		0.008
				(0.018)						(0.018)		(0.020)
Mother's School * Male * 2001 Cohort				0.012						-0.097		-0.094
				(0.220)						(0.222)		(0.224)
Household Income * Male * 2001 Cohort				-0.011						-0.022		-0.023
				(0.031)						(0.033)		(0.032)
Family Structure:												
Father Absent at Birth					-0.000	0.200			-0.014	0.129	0.051	0.050
					(0.179)	(0.348)			(0.208)	(0.336)	(0.220)	(0.316)
Single Mother at Age 4					0.286	-0.000			0.162	-0.027	0.102	-0.086
					(0.188)	(0.350)			(0.199)	(0.341)	(0.196)	(0.322)
Social Father at Age 4					-0.286	-0.200			-0.298	-0.110	-0.271	-0.023
					(0.299)	(0.743)			(0.272)	(0.784)	(0.324)	(0.797)
Two Biological Parents at Age 4 (=reference)					ref	ref			ref	ref	ref	ref
Father Absent at Birth * Male						0.000				0.106		0.150
						(0.512)				(0.525)		(0.527)
Single Mother at Age 4 * Male						0.000				0.184		0.225
						(0.503)				(0.581)		(0.532)
Social Father at Age 4 * Male						-0.800				-0.642		-0.624
						(0.935)				(1.067)		(1.115)
Father Absent at Birth * 2001 Birth Cohort						0.800				0.628		0.564
						(0.524)				(0.511)		(0.525)
Single Mother at Age 4 * 2001 Birth Cohort						-0.600				-0.502		-0.143
						(0.552)				(0.508)		(0.522)
Social Father at Age 4 * 2001 Birth Cohort						-0.800				-0.800		-0.474
						(0.965)				(0.887)		(0.985)
Father Absent at Birth * Male * 2001 Birth Cohort						-1.600*				-1.612*		-1.408+
						(0.722)				(0.745)		(0.748)
Single Mother at Age 4 * Male * 2001 Birth Cohort						1.400+				0.874		0.446
						(0.736)				(0.807)		(0.778)
Social Father at Age 4 * Male * 2001 Birth Cohort						2.000				1.468		0.833
						(1.239)				(1.204)		(1.321)
Early Childhood Health:												
Low Birth Weight (<5.5 pounds, 2,500 g)							0.108	0.500			0.067	0.599
							(0.187)	(0.840)			(0.177)	(0.842)
Pre-Term Birth (<37 weeks gestation)							0.558*	0.333			0.509+	0.442
							(0.265)	(0.701)			(0.262)	(0.744)
Asthma Diagnosis by Age 4 or 5							0.075	0.167			0.106	-0.237
							(0.200)	(0.429)			(0.224)	(0.419)
Low Birth Weight * Male								-0.333				-0.201
								(0.415)				(0.396)
Pre-Term Birth * Male								-0.333				-0.516
								(0.537)				(0.559)
Asthma Diagnosis * Male								0.000				0.274
								(0.469)				(0.455)
Low Birth Weight * 2001 Cohort								-0.167				-0.461
								(0.820)				(0.804)
Pre-Term Birth * 2001 Cohort								0.500				0.450
								(0.651)				(0.701)
Asthma Diagnosis * 2001 Cohort								-0.167				0.070
								(0.412)				(0.418)
Controls:												
Internalizing[1]		0.500***	0.571***	0.600***	0.571***	0.600***	0.642***	0.667***	0.593***	0.561***	0.621***	0.578***
		(0.122)	(0.105)	(0.100)	(0.107)	(0.124)	(0.127)	(0.123)	(0.096)	(0.101)	(0.111)	(0.103)
Mother's Age at Birth[1]		-0.000	0.030	0.031	0.000	-0.000	0.008	0.000	0.037	0.033	0.044	0.030
		(0.013)	(0.025)	(0.024)	(0.020)	(0.021)	(0.025)	(0.024)	(0.027)	(0.024)	(0.037)	(0.029)
Birth Order		-0.000	0.062	0.101	0.143+	0.200*	0.192**	0.167*	0.122+	0.147*	0.110+	0.111+
		(0.094)	(0.064)	(0.066)	(0.081)	(0.082)	(0.071)	(0.071)	(0.063)	(0.062)	(0.060)	(0.063)
Child Born 1983		ref	ref	ref	ref	ref	ref	ref	ref	ref	ref	ref
Child Born 1984		0.000	-0.341	-0.225	-0.286	-0.200	-0.150	-0.167	-0.380	-0.274	-0.346	-0.290
		(0.316)	(0.325)	(0.298)	(0.325)	(0.326)	(0.342)	(0.320)	(0.299)	(0.310)	(0.304)	(0.320)
Child Born 1985		0.500	0.331	0.153	0.143	0.200	0.167	0.167	0.163	0.168	0.206	0.247
		(0.337)	(0.345)	(0.326)	(0.319)	(0.315)	(0.320)	(0.319)	(0.322)	(0.309)	(0.321)	(0.338)
Child Born 1986		0.000	0.093	-0.086	-0.143	-0.000	0.092	-0.000	0.005	-0.145	-0.004	-0.151
		(0.286)	(0.305)	(0.296)	(0.303)	(0.327)	(0.295)	(0.282)	(0.306)	(0.336)	(0.344)	(0.366)
Constant	8.000***	7.831***	7.683***	7.641***	7.521***	7.397***	7.406***	7.441***	7.558***	7.612***	7.566***	7.693***
	(0.100)	(0.297)	(0.245)	(0.245)	(0.272)	(0.318)	(0.240)	(0.265)	(0.225)	(0.287)	(0.260)	(0.321)
Observations	1,450	1,450	1,450	1,450	1,450	1,450	1,450	1,450	1,450	1,450	1,450	1,450

[1]Specified variables (including within interactions) are centered at the overall sample mean.

Standard errors in parentheses. *** p<0.001, ** p<0.01, * p<0.05, + p<0.10. Model estimates are based on multiple imputation of 20 datasets. NC = No Controls, C = Controls, SES = Family Socioeconomic and Cultural Resources, S = Family Structure, H = Early Childhood Health. Model (I) refers to main effects models, Model (II) refers to models with interactions by gender, time period, and gender * time period.

Table A.4: Potential Family and Health Mediators of the Gender Gap in the Location of the Conditional 50th Percentile of Externalizing among White and Asian Children Ages 4-5

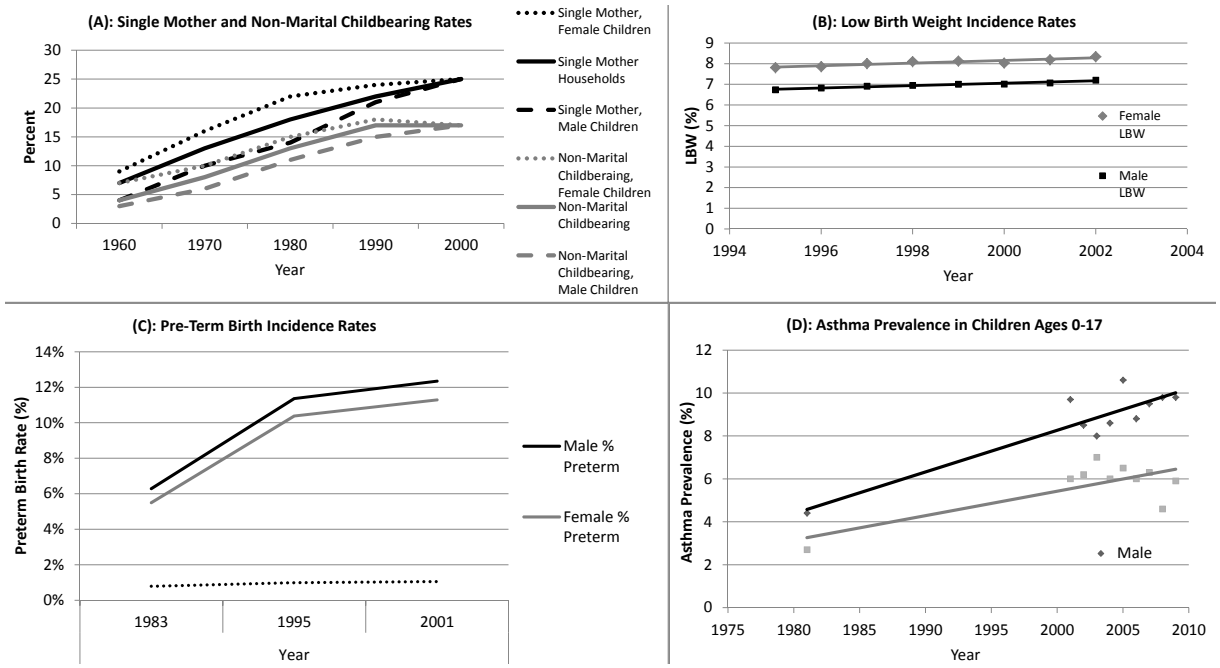
VARIABLES	(1) NC:	(2) C:	(3) C + SES (I):	(4) C + SES (II):	(5) C +S (I):	(6) C + S (II):	(7) C + H (I):	(8) C + H (II):	(9) C + SES + S (I):	(10) C + SES + S (II):	(11) C + SES + S + H (I):	(12) C + SES + S + H (II):
2001 Birth Cohort	-0.000	0.034	0.241	0.260	-0.011	0.212	-0.147	-0.500*	0.176	0.240	0.072	0.206
	(0.448)	(0.186)	(0.204)	(0.201)	(0.240)	(0.241)	(0.244)	(0.254)	(0.221)	(0.197)	(0.192)	(0.217)
Male	-0.000	0.591**	0.545***	0.559***	0.559**	0.636**	0.529**	0.500**	0.533**	0.520***	0.553***	0.541**
	(0.450)	(0.195)	(0.157)	(0.154)	(0.185)	(0.226)	(0.184)	(0.175)	(0.170)	(0.157)	(0.141)	(0.171)
2001 Birth Cohort * Male	1.000+	0.193	0.240	0.295	0.194	0.091	0.265	0.500*	0.218	0.314	0.193	0.181
	(0.541)	(0.256)	(0.220)	(0.211)	(0.261)	(0.309)	(0.227)	(0.226)	(0.216)	(0.215)	(0.172)	(0.239)
Socio-Economic Status (SES):												
Mother's Years of Schooling at Birth[1]			-0.126***	-0.155**					-0.123***	-0.128*	-0.131***	-0.126*
			(0.033)	(0.057)					(0.033)	(0.062)	(0.027)	(0.060)
Per Capita Household Income at Age 4 (in $1,000s, 2011 dollars)[1]			-0.004	0.000					-0.004	0.000	-0.001	0.000
			(0.003)	(0.002)					(0.003)	(0.002)	(0.003)	(0.003)
Mother's School * Male				-0.101						-0.091		-0.103
				(0.083)						(0.091)		(0.090)
Household Income * Male				-0.004						-0.006		-0.005
				(0.008)						(0.008)		(0.010)
Mother's School * 2001 Cohort				0.093						0.065		0.039
				(0.075)						(0.074)		(0.074)
Household Income * 2001 Cohort				-0.016*						-0.017**		-0.014*
				(0.006)						(0.006)		(0.006)
Mother's School * Male * 2001 Cohort				0.053						0.036		0.050
				(0.107)						(0.106)		(0.116)
Household Income * Male * 2001 Cohort				0.006						0.011		0.011
				(0.011)						(0.012)		(0.013)
Family Structure:												
Father Absent at Birth					0.204	0.303			0.092	0.392	0.122	0.282
					(0.197)	(0.381)			(0.193)	(0.343)	(0.159)	(0.351)
Single Mother at Age 4					0.280	0.424			0.185	0.171	0.107	0.267
					(0.178)	(0.352)			(0.168)	(0.320)	(0.122)	(0.334)
Social Father at Age 4					0.226	1.667*			0.167	1.426*	0.047	1.601*
					(0.225)	(0.676)			(0.231)	(0.691)	(0.170)	(0.677)
Two Biological Parents at Age 4 (=reference)					ref	ref			ref	ref	ref	ref
Father Absent at Birth * Male						0.515				0.453		0.471
						(0.576)				(0.566)		(0.583)
Single Mother at Age 4 * Male						-0.212				0.014		-0.117
						(0.495)				(0.416)		(0.473)
Social Father at Age 4 * Male						-0.515				-0.715		-0.731
						(1.146)				(1.104)		(1.180)
Father Absent at Birth * 2001 Birth Cohort						-0.333				-0.428		-0.356
						(0.494)				(0.441)		(0.453)
Single Mother at Age 4 * 2001 Birth Cohort						-0.182				-0.025		-0.210
						(0.453)				(0.388)		(0.387)
Social Father at Age 4 * 2001 Birth Cohort						-1.667*				-1.544*		-1.689*
						(0.735)				(0.762)		(0.708)
Father Absent at Birth * Male * 2001 Birth Cohort						-0.424				-0.521		-0.512
						(0.712)				(0.683)		(0.721)
Single Mother at Age 4 * Male * 2001 Birth Cohort						0.182				-0.137		0.245
						(0.630)				(0.527)		(0.584)
Social Father at Age 4 * Male * 2001 Birth Cohort						0.818				1.193		0.900
						(1.234)				(1.187)		(1.236)
Early Childhood Health:												
Low Birth Weight (<5.5 pounds, 2,500 g)							0.294+	0.000			0.247*	0.023
							(0.176)	(0.342)			(0.114)	(0.323)
Pre-Term Birth (<37 weeks gestation)							0.118	0.500			0.116	0.201
							(0.206)	(0.392)			(0.156)	(0.398)
Asthma Diagnosis by Age 4 or 5							0.265	-0.000			0.233*	-0.042
							(0.174)	(0.274)			(0.117)	(0.249)
Low Birth Weight * Male								0.000				-0.019
								(0.325)				(0.243)
Pre-Term Birth * Male								-0.500				-0.394
								(0.357)				(0.312)
Asthma Diagnosis * Male								0.500				0.407
								(0.305)				(0.248)
Low Birth Weight * 2001 Cohort								0.500				0.266
								(0.309)				(0.304)
Pre-Term Birth * 2001 Cohort								0.000				0.125
								(0.394)				(0.421)
Asthma Diagnosis * 2001 Cohort								-0.000				0.084
								(0.297)				(0.269)
Controls:												
Internalizing[1]		0.580***	0.513***	0.498***	0.548***	0.545***	0.559***	0.500***	0.520***	0.488***	0.530***	0.524***
		(0.101)	(0.067)	(0.064)	(0.073)	(0.071)	(0.058)	(0.062)	(0.055)	(0.060)	(0.055)	(0.054)
Mother's Age at Birth[1]		-0.034	0.011	0.017	-0.022	-0.030	-0.029	-0.000	0.016	0.016	0.016	0.026+
		(0.023)	(0.015)	(0.015)	(0.018)	(0.021)	(0.019)	(0.020)	(0.018)	(0.015)	(0.015)	(0.015)
Birth Order		0.091	0.002	-0.020	0.097	0.091	0.088	0.000	0.017	-0.029	0.010	-0.033
		(0.068)	(0.051)	(0.054)	(0.067)	(0.068)	(0.061)	(0.062)	(0.049)	(0.051)	(0.047)	(0.047)
Child Born 1983		ref	ref	ref	ref	ref	ref	ref	ref	ref	ref	ref
Child Born 1984		0.068	0.093	0.203	0.118	0.182	0.059	0.000	0.083	0.061	0.068	0.106
		(0.205)	(0.212)	(0.200)	(0.200)	(0.227)	(0.184)	(0.208)	(0.197)	(0.195)	(0.190)	(0.212)
Child Born 1985		-0.284	-0.325+	-0.309	-0.312	-0.212	-0.353	-0.500*	-0.312	-0.418*	-0.396*	-0.345+
		(0.222)	(0.197)	(0.202)	(0.218)	(0.256)	(0.223)	(0.216)	(0.201)	(0.209)	(0.193)	(0.200)
Child Born 1986		-0.034	-0.013	0.008	-0.140	-0.030	-0.176	-0.500*	-0.115	-0.052	-0.153	0.013
		(0.218)	(0.243)	(0.180)	(0.246)	(0.241)	(0.236)	(0.247)	(0.195)	(0.198)	(0.216)	(0.201)
Constant	8.000***	7.551***	7.675***	7.627***	7.518***	7.368***	7.583***	7.831***	7.645***	7.672***	7.639***	7.607***
	(0.448)	(0.218)	(0.175)	(0.181)	(0.220)	(0.230)	(0.223)	(0.217)	(0.189)	(0.195)	(0.176)	(0.192)
Observations	3,700	3,700	3,700	3,700	3,700	3,700	3,700	3,700	3,700	3,700	3,700	3,700

[1]Specified variables (including within interactions) are centered at the overall sample mean.

Sample size are rounded to the nearest 50 in compliance with ECLS-B restricted-use data terms. Standard errors in parentheses.

*** p<0.001, ** p<0.01, * p<0.05, + p<0.10. Model estimates are based on multiple imputation of 20 datasets. NC = No Controls, C = Controls, SES = Family Socioeconomic and Cultural Resources, S = Family Structure, H = Early Childhood Health. Model (I) refers to main effects models, Model (II) refers to models with interactions by gender, time period, and gender * time period.

Figure A.1: Prevalence or Incidence Rates of Single Mother Families with Children, Non-Marital Childbearing, Low Birth Weight, Pre-Term Birth, and Asthma Diagnosis, by Gender and Year



Sources: **Panel A:** [1]McLanahan, S. and Percheski, C. 2008. Family Structure and the Reproduction of Inequalities. *American Sociological Review* 34: 257-276. **Panel B:** [2]U.S. Department of Health and Human Services, Centers for Disease Control and Prevention (CDC), National Center for Health Statistics (NCHS), Division of Vital Statistics, Natality public-use data 1995-2002, on CDC WONDER Online Database, November 2005. Accessed at http://wonder.cdc.gov/natality-v2002.html on May 21, 2013 5:34:37 PM. **Panel C:** [3]U.S. Department of Health and Human Services, Centers for Disease Control and Prevention (CDC), National Center for Health Statistics (NCHS), Division of Vital Statistics, Natality public-use data 1995-2002, on CDC WONDER Online Database, November 2005. Accessed at http://wonder.cdc.gov/natality-v2002.html on May 21, 2013 5:34:37 PM. [4]Cooperstock, M., & Campbell, J. 1996. Excess males in preterm birth: interactions with gestational age, race, and multiple birth. *Obstetrics & Gynecology*, 88(2), 189-193. **Panel D:** [5]U.S. Department of Health and Human Services, Centers for Disease Control and Prevention (CDC), National Center for Health Statistics (NCHS), Division of Vital Statistics, National Health Interview Survey public-use data 2001-2009, on CDC Online Database. Accessed at http://www.cdc.gov/nchs/nhis.htm on May 21, 2013 5:34:37 PM. [6]Weitzman, M., Gortmaker, S., & Sobol, A. (1990). Racial, social, and environmental risks for childhood asthma. Archives of Pediatrics & Adolescent Medicine, 144(11), 1189-1194.

Figure A.2: Comparison of the NLSY-C 18-Point Externalizing Problems Scale to the 18-Point Re-Scaled and 30-Point Original ECLS-B Externalizing Problems Scale

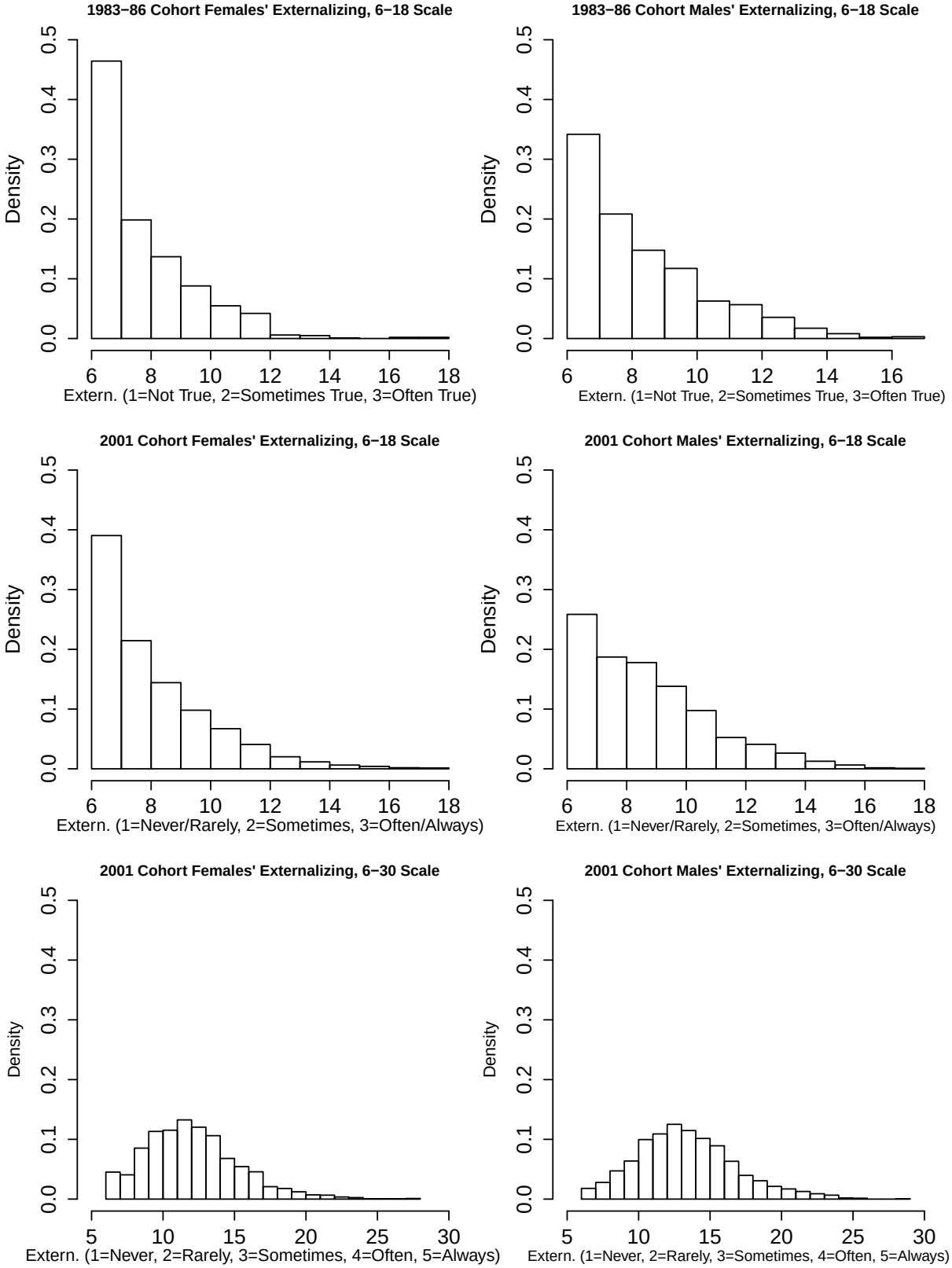
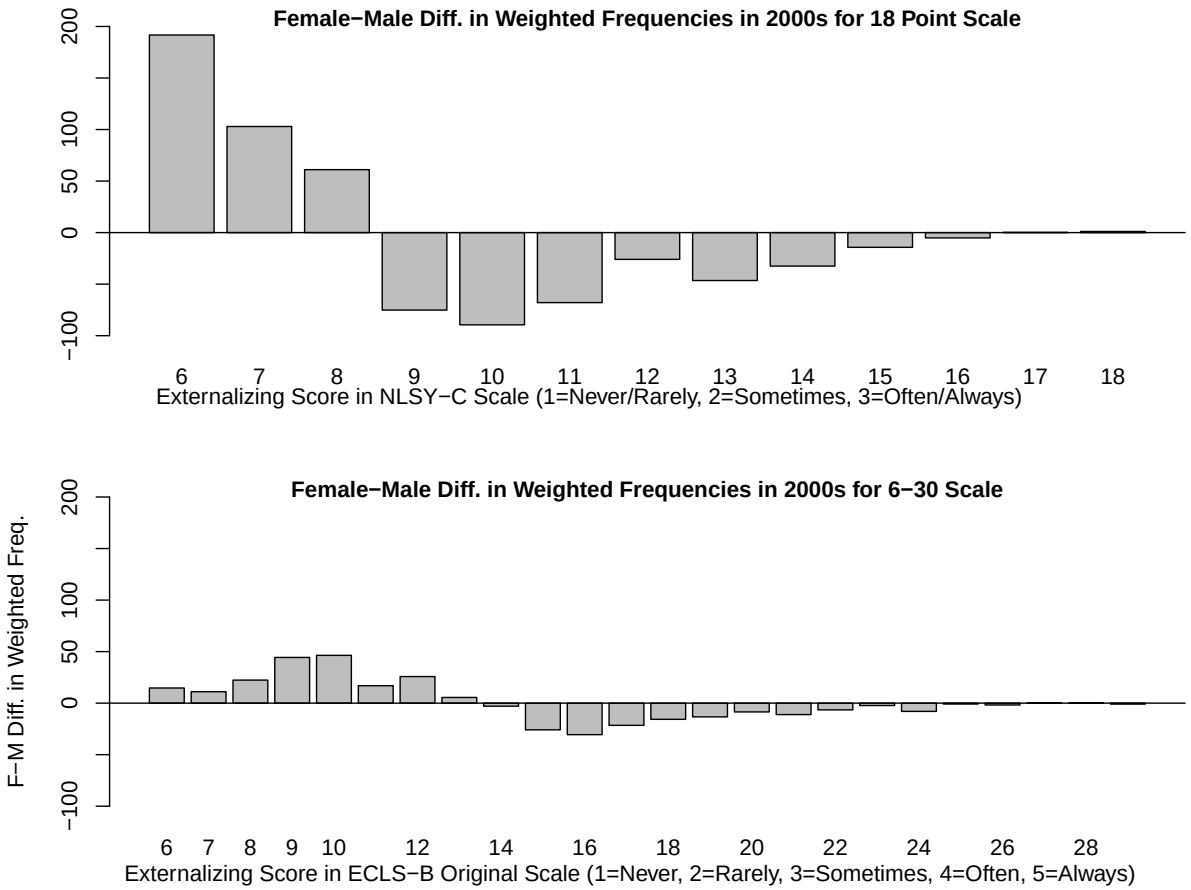
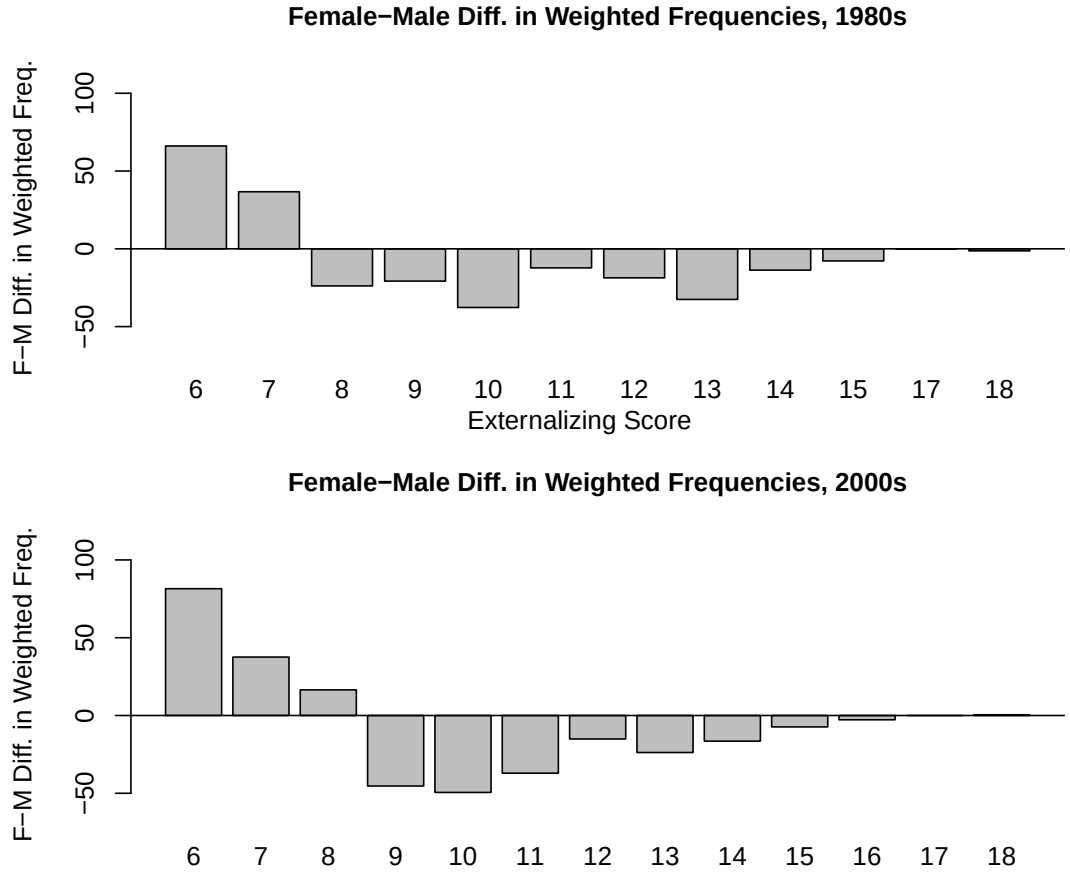


Figure A.3: Effects of Rescaling on the Female-Male Difference in Sample Size-Adjusted Frequencies across the Distribution of the ECLS-B Externalizing Problems Scale



Note that both distributions are from the ECLS-B data but the frequencies are weighted to adjust for minor differences in the size of the female and male samples.

Figure A.4: Comparison across Datasets of Female-Male Differences in Sample Size-Adjusted Frequencies across the Distribution of Externalizing Problems



Note that the distribution in the top panel is from the NLSY-C data, whereas the distribution in the bottom panel is from the ECLS-B data. As such, the frequencies are weighted to adjust for both differences in the size of the female and male samples in each dataset and for differences in sample sizes across datasets.