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Intrauterine stress and male cohort quality: The case of September 11, 2001

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ABSTRACT

Empirical research and the theory of natural selection assert that male mortality more than female mortality responds to ambient stressors *in utero*. Although population stressors may adversely damage males that survive to birth, the rival culled cohort hypothesis contends that males born during stressful times may exhibit better health than males in other cohorts because fetal loss has "culled" the frailest males. We tested these hypotheses by examining child developmental outcomes in a U.S. birth cohort reportedly affected *in utero* by the September 11, 2001 attacks. We used as outcomes the Bayley cognitive score and child height-for-age from the Early Childhood Longitudinal Study-Birth Cohort. Previous research demonstrates a male-specific effect of 9/11 on California infants born in December 2001. We, therefore, compared cognition and height of this cohort with males born prior to the 9/11 attacks. We controlled for unobserved confounding across gender, season, and region by using triple-difference regression models ($N = 6950$). At 24 months, California males born in December scored greater than expected in cognitive ability (coef = 9.55, standard error = 3.37; $p = 0.004$). We observed no relation with height. Results remained robust to alternative specifications. Findings offer partial support for the culled cohort hypothesis in that we observed greater than expected cognitive scores at two years of age among a cohort of males affected by 9/11 *in utero*. Contemporary population stressors may induce male-specific culling, thereby resulting in relatively improved development among males that survive to birth.

Introduction

An estimated fifty to seventy percent of human conceptions do not survive to birth, and much research indicates that male fetal losses far outnumber female losses (Boklage, 1990; Byrne & Warburton, 1987; Catalano, Bruckner, Anderson, & Gould, 2005; Jakobovits, 1991). Reports of excess male fetal deaths months after the terrorist attacks of September 11, 2001 (Bruckner, Catalano, & Ahern, 2010; Catalano, Bruckner, Anderson, et al., 2005) as well as economic decline (Catalano, Bruckner, Gould, Eskenazi, & Anderson, 2005) indicate that exogenous stressors may induce greater than expected male fetal loss. The literature, also consistent with the notion of male frailty *in utero*, finds lower than expected live birth sex ratios (i.e., the ratio of male to female live births) following natural and man-made disasters (Fukuda, Fukuda, Shimizu, & Moller, 1998; Mocarelli, Brambilla, Gerthoux, Patterson, & Needham, 1996; Song, 2012; Torche & Kleinhaus, 2012).

Mechanistic causes of male-specific fetal loss remain poorly established. Mammalian research indicates that male more than female fetuses appear sensitive to maternal corticosteroids produced as early as the second trimester (Giussani, Fletcher, & Gardner, 2011; Mueller & Bale, 2008; Owen & Matthews, 2003). This heightened reactivity, starting in the second trimester of human gestation, may jeopardize the viability of male fetuses. The literature, however, does not converge on this corticosteroid explanation in that recent human studies show reduced male reactivity to various stimuli *in utero* (Clifton, 2010; Glynn & Sandman, 2012).

Theory regarding excess male fetal loss and frailty *in utero* proposes that, over generations, humans conserved mechanisms by which pregnant mothers detect, and spontaneously abort, males with a low likelihood of surviving and yielding offspring (Trivers & Willard, 1973). According to theory, aborting these males increases the yield of grandchildren because, following ambient stressors, frail sons produce fewer offspring than do frail daughters. Aborting a weak male fetus presumably allows the mother to conceive other offspring with greater lifetime reproductive success (Lummaa, 2001; Rickard, Russell, & Lummaa, 2007). This line of work suggests that males born to low sex ratio cohorts would exhibit, on average, better health than males in other cohorts because spontaneous fetal loss has "culled" (i.e., selected against) the frailest

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males. Research using data from historical European populations supports the culled cohort argument in that male cohort mortality, especially before reproductive age, varies positively with the birth sex ratio of that cohort (Bruckner & Catalano, 2007).

The above literature raises the intuitive question of whether, in the contemporary context, perturbations in the sex ratio induce long-term sequelae for surviving males (Catalano, Ahern, Bruckner, Anderson, & Saxton, 2009). Secular improvements in living conditions, reductions in the dose and duration of stressors, and the close scrutiny now provided by clinicians all could serve to protect frail males in gestation that in earlier times may have been spontaneously aborted.

Alternatively, consistent with the developmental origins literature, ambient stressors that precede low sex ratios may "damage" the development of male cohorts that survived to birth (Barker, 1998). The cohort damage argument follows from the critical period model in which exposures during a specific phase of development (e.g., *in utero*) induce sequelae on the structure and function of the body (Ben-Shlomo & Kuh, 2002). Epidemiologists have invoked cohort damage to explain the relation between adverse birth outcomes and chronic disease in adulthood (Leon et al., 1998). Other population researchers forward phenotypic plasticity within the framework of cohort damage (Bateson et al., 2004). The developmental plasticity argument posits that specific birth phenotypes may induce positive or negative outcomes depending on the match between the child's realized early life environment and that predicted by the gravid mother (Bateson & Gluckman, 2012). As these explanations relate to our test, cohort damage and/or developmental plasticity could yield an inverse association between the birth sex ratio and male cohort morbidity.

Intrauterine effects of September 11th, 2001

The terrorist attacks of September 11, 2001 (hereafter referred to as 9/11) induced widespread social and economic disruption as well as elevated levels of stress and anxiety in the United States (Schuster et al., 2001). In January 2002, New York City witnessed a five percent drop in the sex ratio (Catalano, Bruckner, Marks, & Eskenazi, 2006). California, moreover, yielded excess male fetal loss in October and November 2001 and two percent fewer live male births than expected in December 2001 (Catalano, Bruckner, Gould, et al., 2005). The California fetal death file defines fetal loss as non-elective fetal deaths at or greater than 20 weeks of gestation. In California, the majority of losses recorded in the fetal death file occur between the 20th and 28th week of gestation. The temporal lag, therefore, between observed male fetal loss and drop in male live and male very low weight births in California has led researchers to infer that the majority of males lost *in utero* (i.e., 20–28 weeks of gestation on 9/11) may have been pregnancies whose full term due dates fell in December 2001 and January 2002 (Bruckner et al., 2010).

The heightened sensitivity of birth outcomes among California women may arise in part from anxiety and economic stress due to more 9/11-related mass layoffs than any other state in the US (Catalano, Ahern, & Bruckner, 2007). A central component of California's economy—tourism—also suffered an estimated 30% decline in revenue between 9/11 and December 2001 (RAND, 2002). The literature, moreover, documents adverse birth outcomes in California after 9/11 (Lauderdale, 2006).

Study and hypotheses

Also in 2001, the National Center for Education Statistics initiated the Early Child Longitudinal Study Birth Cohort (ECLS-B), a nationally-representative study of the 2001 U.S. birth cohort. The excess male fetal loss observed in California in 2001 thus provides a rare opportunity to examine the effect of excess intrauterine mortality on cohort quality of males that survived to birth.

We use the ECLS-B to test whether male infants born to low sex ratio cohorts after 9/11 exhibit different trajectories of health relative to males born in other calendar months of 2001. We focus our test on California males born to the low sex ratio cohort in December. We examine height-for-age (WHO Multicentre Growth Reference Study Group, 2006) and Bayley cognitive score (Hillemeier, Farkas, Morgan, Martin, & Maczuga, 2009), two validated measures of child growth and development that reportedly vary with ambient exposures *in utero* (Deaton, 2007; Laplante et al., 2004) and predict mental and physical health in adulthood (Batty, Deary, & Gottfredson, 2007; Calvin et al., 2011; Jousilahti, Tuomilehto, Vartiainen, Eriksson, & Puska, 2000; Osler et al., 2003; Vagero & Leon, 1994).

Culling, damage, or a combination of these factors could affect the developmental trajectory of California males born after 9/11. A positive relation between male height and cognitive ability and membership in a birth cohort with a low sex ratio would support culling, whether or not damage also occurs to a lesser extent. Similarly, an inverse relation would support cohort damage.

Methods

ECLS-B data

The ECLS-B is a U.S. nationally representative, longitudinal cohort study that targeted 14,000 births and successfully enrolled 10,600 children born in 2001. A detailed description of the ECLS-B survey methodology and cohort characteristics appears elsewhere (Nord, Edwards, Andreassen, Green, & Wallner-Allen, 2006). At 9 months and 24 months after the child's birth, trained staff conducted developmental child assessments as well as parent interviews. The institutional review boards at the University of Wisconsin, Madison and the University of California, Irvine approved the use of restricted ECLS-B data.

The literature finds fewer than expected male births, following the terrorist attacks of September 11, 2001, in California and New York City (Catalano, Bruckner, Gould, et al., 2005; Catalano et al., 2006). California exhibited 20 percent more male fetal deaths than expected in October and November 2001 and a two percent reduction in live male births in December 2001. New York witnessed a five percent reduction in live male births in January 2002. Given the focus of ECLS-B on births in 2001, we could not measure child development among males born in New York City in January 2002. We, therefore, focused our test on California males. Restriction of the ECLS-B sample to California births yielded 1100 children at the 9 month assessment and 900 children at the 24 month assessment. Our comparison group consists of children born in all other states: 8650 children at the 9 month assessment and 7600 children at the 24 month assessment.

Outcome variables

Consistent with theory, child mortality rates serve as the optimal measure of culling or damage among California males born in December 2001. Given the rarity of death in the ECLS-B cohort and the limited statistical power to detect cohort differences in child mortality, we sought other ECLS-B variables to gauge cohort health. We selected candidate variables using three criteria. First, the literature reports that the candidate variable responds to ambient exposures *in utero*. Second, the risk of death varies positively with the candidate variable. Third, the candidate variable's strong measurement and construct validity allows for its