Social Science & Medicine 72 (2011) 999–1003

Contents lists available at ScienceDirect

Social Science & Medicine

journal homepage: www.elsevier.com/locate/socscimed

Selection *in utero* contributes to the male longevity deficit

Ralph Catalano

School of Public Health, 15 University Hall, University of California, Berkeley, CA 94720, United States

ARTICLE INFO

Article history:
Available online 4 February 2011

Keywords:
Selection *in utero*
Male longevity deficit
Sweden
UK
Gender
Mortality
Natural selection

ABSTRACT

The literature offering evolutionary explanations of the male longevity deficit does not address temporal variation in the deficit. This circumstance appears attributable to the fact that natural selection intuitively explains the deficit's pervasive and persistent nature, while social processes more parsimoniously explain its temporal variability. I offer consilience of these perspectives by speculating that selection *in utero*, a mechanism both conserved by natural selection and affected by social processes, could induce deviations around trend in the male longevity deficit. I describe the mechanism and offer an empirical test of its possible effect among Swedes – a population with the longest continuous record of sex-specific longevity in annual birth cohorts. I replicate the test with data from England and Wales. Results support the hypothesis that selection *in utero* against less fit males may explain part of the difference in longevity between males and females in modern populations.

Introduction

Men, on average, live shorter lives than women. Globally, men born in 2009 can expect to live 64.3 years compared to 68 years for women (Central Intelligence Agency of the United States of America, 2010). Reliable vital statistics kept over long periods of time show, moreover, that this male longevity deficit has been persistent and pervasive (Human Mortality Database, 2010).

The nearly ubiquitous observation of the deficit suggests that it arises from fundamental biological mechanisms conserved by natural selection. The "mother" and "grandmother" hypotheses, for example, assume that women have looked after infants and "provisioned" children over much of human history (O'Connell, Hawkes, & Blurton-Jones, 1999; Peccei, 2001). Women who lived long enough to help their children and grand children survive to reproductive age presumably had more grandchildren and great-grandchildren than women who died relatively young (Lahdenpera, Lummaa, Helle, Tremblay, & Russell, 2004). These assumptions imply that natural selection would conserve mutations that confer longevity on women more than men (Madrigal & Melendez-Obando, 2008; Shanley, Sear, Mace, & Kirkwood, 2007).

Other literature claims that reproductive success among men depends on the ability to compete successfully for women (Clutton-Brock & Isvaran, 2007). Natural selection would, therefore, conserve mutations that aid this competition (Moller, Fincher, & Thornhill, 2009). Mutations that make males more competitive may, however, also reduce lifespan. Metabolism that yields rapid growth, for example, confers the advantage of size and strength but also apparently accelerates aging (Kawahara & Kono, 2010). Competition itself, moreover, can lead to trauma that shortens life (Moller, Fincher et al., 2009).

Not all mechanisms that increase fitness in males also shorten life span. Males hardy enough to father children late in life through, for example, serial monogamy should yield more grandchildren than less robust men (Jokela, Rotkirch, Rickard, Pettay, & Lummaa, 2010; Lahdenpera, Russell, and Lummaa; 2007). The literature suggests, however, that natural selection conserved more, or more powerful, life extending mechanisms in women than in men.

These arguments may parsimoniously explain the emergence and persistence of the male longevity deficit. But the deficit has exhibited frequent and sizeable changes over time. Conserved differences such as those described briefly above do not suggest intuitively appealing explanations of this temporal variability. Researchers have, therefore, typically attributed it to phenomena such as war (Elder, Clipp, Brown, Martin, & Friedman, 2009), improvements in obstetric practices (Pavard, Koons, & Heyer, 2007), gender roles (Courtenay, 2000), and social reforms (Nobles, Brown, & Catalano, 2010) that differentially affect male and female mortality and appear more in the realm of the social than biological sciences. I, however, speculate that selection *in utero*, a mechanism both conserved by natural selection and affected by social processes, could induce variability over time in the male longevity deficit. I describe the mechanism and offer an empirical test of its possible effect among Swedes – a population with the longest continuous record of sex-specific longevity in annual birth cohorts. I also

E-mail address: rayc@uclink4.berkeley.edu.

0277-9536/$ – see front matter © 2011 Elsevier Ltd. All rights reserved.
doi:10.1016/j.socscimed.2011.01.012

replicate the test in a shorter, more complex set of data from England and Wales. Results support the hypothesis that selection *in utero* may explain part of the difference in longevity between males and females in modern populations.

Selection *in utero*

The fact that at least half, and as many as 70%, of human conceptions end without live births (Boklage, 1990) makes gestation as much an opportunity for selection as for maturation (Moller, 1997; Stearns, 1987). Much theory suggests that natural selection has conserved mechanisms by which women spontaneously abort conceptuses and fetuses least likely to yield grandchildren (Forbes, 1997). Observation suggests that these mechanisms select early in gestation against female fetuses that somehow signal that they, or the eggs of their potential children (present around the 6th week of gestation), will unlikely yield offspring that survive to reproductive age (Boklage, 1990). Later in gestation, the mechanisms supposedly select against fetuses, mostly small males, least likely to survive to reproductive age (Waldron, 1983; Wells, 2000).

Selection *in utero* assumes that mothers autonomically assess the fitness of fetuses and spontaneously abort those that fall below some criterion (Moller, 1997; Stearns, 1987; Trivers & Willard, 1973). Researchers have invoked this argument to explain clinically important circumstances such as the relatively high prevalence of birth defects among infants born to older women (Neuhäuser & Krackow, 2007). More important for my purposes, the literature also uses selection *in utero* to explain variation over time in characteristics of populations. These include declines in the ratio of male to female births (i.e., the secondary sex ratio) following not only "natural" population stressors such as cold ambient temperature (Helle, Helama, & Lertola, 2009) and earthquakes (Fukuda, Fukuda, Shimizu, & Moller, 1998), but also human-induced stressors such as air pollution (Lyster, 1974) and terrorist events (Catalano, Bruckner, Marks, & Eskenazi, 2006), as well as political and economic upheaval (Catalano, 2003). The explanation posits that women autonomically manipulate the criterion for spontaneous abortion to avoid offspring unlikely to survive prevailing environmental conditions (Stearns, 1987; Trivers & Willard, 1973). More threatening environments would, presumably through the maternal stress response (Subbaraman et al., 2010), raise the criterion thereby leading to the spontaneous abortion of fetuses that mothers in more benign circumstances may have delivered live (Catalano, Saxton, Bruckner, Goldman, & Anderson, 2009).

An exception to these finding arises from the study of sex ratios during war. The work, perhaps reflecting considerable variation in the referent for "war," fails to converge on an association. Some studies report that sex ratios increased during the long, world wars of the 20th Century (Graffelman & Hoekstra, 2000; MacMahon & Pugh, 1954), but research into recent, shorter conflicts reports decreased secondary sex ratios (Ansari-Lari & Saadat, 2002; Zorn, Sucur, Stare, & Meden-Vrtovec, 2002).

Less, indeed little, controversy arises from the assumption that males disproportionately populate the lower ranks of fetal fitness. Sons require a greater investment to sustain than do daughters (Clutton-Brock, 1991; Powe, Knott, & Conklin-Brittain, 2010). Indeed, mothering sons reportedly reduced the life span of women more than mothering daughters in environmental circumstances more typical of human evolutionary history than those we have enjoyed since the industrial age (Helle, Lummaa, & Jokela, 2002). Sons, despite this extra investment, more likely die before reproducing than do daughters (Waldron, 1983; Wells, 2000). A mother's investment in a son that died before reproducing would also deplete resources that she could have used to increase the reproductive chances of her other children. So, when population stressors raise the "average" criterion among pregnant women for spontaneous abortion, the gestations of small male fetuses, given their relatively low fitness, disproportionately end. This selection *in utero* thereby lowers the secondary sex ratio of the affected birth cohort (Catalano, Saxton et al., 2009).

Research supports the argument for selection *in utero*. Secondary sex ratios reportedly rise with improving health conditions (Klasen, 1994). Swedish men from low sex ratio annual birth cohorts reportedly live longer than men from high sex ratio cohorts (Catalano & Bruckner, 2006). Males from low sex ratio monthly birth cohorts in California suffer lower infant mortality than those from high sex ratio cohorts (Catalano, Ahern, Bruckner, Anderson, & Saxton, 2009). Unusually cold weather in the Nordic countries coincides with low sex ratio birth cohorts, but men from those cohorts live longer than men in gestation during warm periods (Catalano, Bruckner, & Smith, 2008).

The above literature provides either argument or data implying that pregnant women in stressed populations autonomically raise the standard of fitness required to complete gestation and that male fetuses will disproportionately fail to reach the new standard. None of the work, however, carries the argument to its logical conclusion – that the male longevity deficit should vary over time with the sex ratio of birth cohorts. This conclusion may seem too obvious, or its implications too applied, for evolutionary theorists to pursue, but the fact remains – a reasonable explanation for an important dimension of perhaps the most pervasive and persistent health inequity in humans remains unexplored. Greater culling of male fetuses in stressful times implies that the difference between the lifespan of men and women should decline in low sex ratio birth cohorts; less culling of males in benign times implies that the difference in lifespan should increase in high sex ratio cohorts. I search for these patterns in data from Sweden describing annual birth cohorts starting in 1751, the first year for which I can obtain data, and ending in 1916, the last in which sufficient mortality has occurred to estimate cohort lifespan. More specifically, I test the hypothesis that deviations from trends in the sex ratio of annual birth cohorts correlate positively with deviations from trends in the male longevity deficit. I then replicate the test for England and Wales over the years 1841–1915.

Methods

I used data from Sweden for my primary test for several reasons. Sweden has kept dependable vital statistics longer than any other nation-state. Swedes, moreover, have fought in relatively few wars during the test period meaning that combat deaths distort estimates of Swedish lifespan less than those from other countries. Data from other societies describe shorter time periods and often reflect the influence of war on male longevity. These circumstances suggest that testing the hypothesis in other societies may require more complex tests than that described below for Sweden, and thereby reduce accessibility. I, however, attempt, as summarized below, a replication in England and Wales to determine if the Swedish findings appear in a society likely exposed to different stressors including war. The data for England and Wales describe the period 1841–1915.

I obtained vital statistics and lifespan data for Sweden as well as for England and Wales from the Human Mortality Database (Human Mortality Database, 2010). This source archives life table data that meet quality standards agreed among demographers and researchers. I used female lifespan, technically referred to as cohort life expectancy at birth, less male lifespan as my measure of the male longevity deficit; and used the odds of a male birth as the secondary sex ratio.