THE MULTIDIMENSIONAL INFLUENCE OF HEALTH ON FERTILITY OUTCOMES FOR MEN: EVIDENCE FROM SWEDISH MILITARY CONSCRIPTION DATA

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ABSTRACT. We examine how multidimensional health in early adulthood predicts men's later-life fertility using Swedish population registers linked to universal military conscription examinations. Our study follows men born 1951–1979, observed for fertility through 2024, and exploits objective measures taken at ages 18–20 across twelve indicators: muscular strength (handgrip, arm, leg, composite), cardiovascular health (resting heart rate, blood pressure, ECG pathology, maximal working capacity on cycle ergometer), and blood/urine biomarkers (hematocrit, erythrocyte sedimentation rate, U-glucose, U-albumin). Outcomes are completed parity and childlessness. We estimate linear models for all men and sibling fixed-effects models that compare full brothers, adjusting for birth cohort and age at test. Results show pronounced, monotonic gradients for strength and fitness. Men in the weakest strength deciles or lowest fitness deciles have 0.25–0.45 fewer children and a 10–20 percentage-point higher probability of childlessness than those in the strongest/highest deciles; these differences persist, with moderate attenuation, in sibling fixed-effects. High resting heart rate shows similar but slightly smaller gradients. Blood pressure categories and ECG abnormalities exhibit modest associations. Biomarkers indicative of metabolic and renal stress are consequential: positive U-glucose and U-albumin are associated with 0.10–0.20 fewer children and 5 percentage-point higher childlessness. Hematocrit and inflammation show smaller, suggestive, and potentially non-linear patterns. Findings are consistent with multiple channels—physiological fecundity, partner-market dynamics, and socioeconomic mediation—and indicate that early-adult health is an important component of male fertility stratification. Because the steepest gradients occur at the lower tail of health distributions, secular declines in youth fitness and rises in metabolic risk may materially contribute to increasing male childlessness. Investments in adolescent and young-adult health could therefore have long-run implications for family formation.

INTRODUCTION

Demographically-speaking, recent years have been characterized by a decrease in fertility across not only high-income countries, but also middle- and low-income countries. At the same time that fertility has been declining, markers of population health have also been in decline

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in many contexts: there have been steady rises in obesity, decreases in physical activity, and increases in the rates of sedentary occupations (Westerstahl et al., 2003; Bygdell et al., 2022), all of which may be linked to increased male infertility (Choy and Eisenberg, 2018). The extent to which health in early adulthood influences later fertility outcomes is under-researched. Some studies have examined how body mass index (BMI) is associated with fertility, finding that those who are underweight, overweight, or obese have lower fertility and a higher rate of childlessness (Barclay and Kolk, 2020; Lee et al., 2023). Likewise, some research indicates that physical fitness is strongly associated with fertility outcomes (Barclay and Kolk, 2020). Other research has shown that certain damaging genetic mutations contribute significantly to the risk of childlessness, and much more so amongst men than women (Gardner et al., 2022). Recent research in Sweden and Finland has found that diseases related to congenital anomalies and mental-behavioral disorders are most commonly associated with childlessness (Liu et al., 2024). However, health is multifaceted, and far from fully captured by disease diagnoses – there is a distribution of health across the undiagnosed population as well. It would be very valuable to have a much broader and prospective understanding of how different, including sub-clinical, dimensions of health are associated with fertility outcomes.

Although it is very difficult to pinpoint how population health contributes to fertility changes, a prerequisite for understanding this contribution is to identify the effects of different dimensions of health on completed fertility, childlessness, and parity progression probabilities. Furthermore, if health does affect fertility, we need a better understanding of *how*. Are there direct physiological effects of certain health issues on fecundity, or does health influence fertility primarily through partnership formation – and, if so, how much of the influence on partnership formation is mediated by health effects on socioeconomic attainment. Buss and Barnes (1986) reported that one of the most highly desired traits in a partner is good health, with woman putting a higher priority on health than men, and women ranking health higher than both physical attractiveness and socioeconomic traits. Liu et al. (2024) estimate that 37.9% of the effects of severe disease history on childlessness for men in Finland and Sweden are mediated by singlehood.

Part of the challenge of studying the relationship between health in early adulthood and later fertility is measuring health objectively, and doing so at a population-scale. In this study we use Swedish population register data for cohorts of men born 1951-1979 to examine how a range of health measures collected during military conscription examinations are associated with later fertility outcomes, focusing on total number of children and childlessness. This data contains information on the health of a very high percentage of men born over the cohorts that we study. This resource is particularly remarkable given that men in early adulthood typically have little interaction with the health care system; typically this means that usually it is only possible

to identify serious health issues, perhaps through hospitalization. In our case, thanks to the sweeping mandate of the military to conscript all young men, we have information for virtually full cohorts, and can therefore study the influence of health right across the distribution.

We present results for twelve different health measures in total: four different measures of physical strength (handgrip, arms, legs, and a composite measure), four for cardiovascular health (blood pressure, resting heart rate, physical fitness, and whether an electrocardiogram indicated any pathological signs), and four different blood- and urine-based measures of physiological function (hematocrit [a measure of red blood cell density], erythrocite sedimentation rate [ESR, a measure of inflammation], U-glucose (an indicator of potential diabetes), and U-albumin (an indicator of kidney damage). We find that health in early adulthood is a very significant predictor of later fertility outcomes, with markers of strength and cardiovascular health particularly strongly associated with eventual fertility.

Channels from Health in Early Adulthood to Male Fertility: Theory and Empirical Evidence. In this section we review research that may help us to understand the pathways by which health in early adulthood may affect later fertility outcomes. We consider several different pathways, including direct effects on fecundity, the influence on potential desirability as a romantic and childbearing partner, the influence of health on socioeconomic attainment – which may in turn affect success in the partner market – as well as potential selection effects. We consider each of these pathways holistically under the headings of the various strength measures that we use.

Strength. Few studies measure strength early in life and then follow-up reproductive outcomes over the life course, but numerous studies have examined the relationship between different measures of strength later in life and various measures of success in reproduction or partnership formation. Although these results may reflect a positive influence of parenthood or partnership on strength, they also plausibly suggest that strength affects selection into partnership and parenthood. For example, research on Hadza hunter-gatherers shows that men with greater upper-body strength, of which measured handgrip strength was one component, have higher reproductive success – a one standard deviation increase in upper-body strength was associated with 0.28 more children – and that this is likely mediated by the positive effect of upper-body strength on hunting ability (Apicella, 2014). Other research has found that handgrip strength is positively associated with parenthood in the UK (Orchard et al., 2025), and other research that men who are childless and unmarried in later mid-life have lower handgrip strength (Guralnik et al., 2009). Handgrip strength and upper-body strength are also positively associated with lifetime number of sexual partners as well as being in a current partnership in US data (Smith and Hagen, 2025). Handgrip strength is also associated with greater reproductive success amongst

women (Atkinson et al., 2012). Men who are taller, stronger, and more physically fit are generally perceived as more physically attractive by women (Tovée et al., 1999; Pawlowski et al., 2000; Sell et al., 2017). Sell et al. (2017) report that estimates of physical strength, based on photographs, accounted for over 70% of perceived male body attractiveness amongst female raters. Men with greater strength are therefore expected, on average, to have more partnering opportunities (Rhodes et al., 2005).

Cardiovascular health and fitness. Cardiovascular health is a fundamental determinant of overall vitality and biological functioning, with wide-ranging implications for reproductive capacity. Several distinct indicators – such as blood pressure, resting heart rate, electrocardiogram (ECG) abnormalities, and maximal oxygen uptake (VO_2max) – reflect different aspects of cardiovascular efficiency and regulation. Young adults with better cardiovascular fitness tend to have lower systemic inflammation, better vascular function, and more favorable lipid and hormonal profiles, all of which are associated with higher fecundity and better sexual health outcomes (Vaamonde et al., 2012; Jóźków and Rossato, 2017). Conversely, poor cardiovascular health at a young age may represent an early manifestation of metabolic dysfunction or autonomic imbalance, both of which have known links to subfertility (Nguyen et al., 2017).

Resting heart rate, in particular, provides a sensitive measure of cardiorespiratory fitness and autonomic regulation. Lower resting heart rate reflects higher parasympathetic tone and greater cardiovascular efficiency, and has been associated with improved sperm motility and concentration, as well as more balanced testosterone levels (Agostini et al., 2011). Elevated resting heart rate, by contrast, may signal poor conditioning, stress reactivity, or underlying cardiovascular strain. These physiological differences can affect male fecundity directly through hormonal pathways or indirectly through reduced sexual function, as erectile dysfunction and other reproductive impairments often share vascular etiologies (Mykoniatis et al., 2018; Rosen et al., 2005).

At the same time, cardiovascular health has strong social and behavioral correlates that may mediate its relationship with fertility. Men with higher fitness levels are more likely to engage in physical activities, participate in social networks centered on sports or outdoor activities, and exhibit greater self-confidence in social interactions (Sell et al., 2017). In contrast, early signs of cardiovascular problems – such as hypertension or pathological ECG readings – are often associated with lower energy, psychological distress, or self-perceived health limitations, which can constrain opportunities for partnership formation and reduce the likelihood of entering or maintaining stable unions. The relationship between physical fitness and fertility may therefore reflect both physiological and social selection mechanisms operating throughout the life course.

Kidney function. We have information on two biomarkers that are related to kidney function, which are measures of urinary glucose (U-glucose), an indicator of type I diabetes in men of this age, and urinary albumin (U-albumin). There is a relatively large body of evidence that suggests that diabetes is directly related to fertility outcomes in both men and women (Liu et al., 2024), though the evidence for type 2 diabetes is stronger than for type 1 diabetes (Dinulovic and Radonjic, 1990; Livshits and Seidman, 2009; Facondo et al., 2022). Diabetes is believed to affect fertility in men by disrupting the hypothalamic-pituitary–gonadal axis (i.e. affecting hormonal balance), by affecting spermatogenesis and sperm maturing (i.e. affecting sperm volume and function), and via erectile dysfunction (Huang et al., 2024).

Indicators of kidney function, such as urinary albumin (U-albumin), reflect the efficiency of renal filtration and are important markers of overall metabolic and vascular health. Albuminuria in otherwise healthy young men is often an early sign of endothelial dysfunction – a condition where the cells lining blood vessels, called the endothelium, don't function properly – and systemic microvascular damage, which are associated with a range of later-life morbidities, including hypertension, cardiovascular disease, and diabetes (Hadi et al., 2005; Biswas and Khan, 2020). These conditions are, in turn, strongly associated with reduced fecundity, both through physiological mechanisms and via reduced sexual function. Albuminuria is also an independent risk factor of erectile dysfunction in men with type 2 diabetes (Chuang et al., 2012).

Even relatively mild kidney dysfunction has been linked to hormonal imbalances, such as lower testosterone and higher levels of gonadotropins, which may directly impair male reproductive capacity (Hylander and Lehtihet, 2015). Poor kidney function can also reflect broader physiological decline, signalling poorer overall health and potentially lowering perceived mate quality. From a social perspective, chronic health conditions of this kind may reduce attractiveness and opportunities for forming long-term partnerships (Buss and Barnes, 1986). In addition, impaired kidney function has been associated with lower physical endurance and greater fatigue (Gregg et al., 2021), which can indirectly affect reproductive outcomes through socioeconomic pathways – for instance, by limiting occupational success or stability, both of which are known correlates of male fertility (Alma et al., 2023). Taken together, early signs of renal dysfunction in young adulthood may serve as indicators of both reduced biological fecundity and a broader pattern of social disadvantage relevant to fertility outcomes.

Inflammation. Systemic inflammation is increasingly recognized as a critical factor linking general health, chronic disease, and reproductive outcomes. Elevated erythrocyte sedimentation rate (ESR) is a non-specific marker of inflammation and may capture latent infections, autoimmune processes, or general physiological stress. Chronic low-grade inflammation can disrupt

the hypothalamic-pituitary-gonadal axis, leading to reduced testosterone levels, impaired spermatogenesis, and diminished libido (Ma et al., 2025). From a life course perspective, higher levels of inlammation in early adulthood may indicate an individual tendency towards higher levels of inflammation that may in turn be associated with accelerated biological aging, lower energy levels, and poorer resilience to stress – all factors that could influence partnership formation and reproductive timing. Moreover, inflammation is linked to metabolic disorders such as obesity, which is in turn associated with lower fertility (Barclay and Kolk, 2020). Although there is much research that suggests that lower socioeconomic status increases inflammatory responses (Pollitt et al., 2008; Milaniak and Jaffee, 2019), consistent with a fundamental cause perspective (Link and Phelan, 1995), there is seems to be little research that investigates whether inflammation can itself prospectively influence later socioeconomic attainment. If this were the case, however, then a negative influence of inflammation on educational and socioeconomic attainment might in turn explain part of the influence on fertility, via partnership formation.

Hematocrit. Hematocrit reflects the proportion of blood volume composed of red blood cells and serves as a key indicator of oxygen-carrying capacity. Optimal hematocrit levels are essential for endurance, energy metabolism, and overall physical performance – factors likely to influence both health and mating success. Low hematocrit, often used clinically to diagnose anemia (World Health Organization, 2011), can result from nutritional deficiencies (e.g. Iron, Vitamin B_{12}), or chronic disease (Brigety and Pearson, 1970), and is associated with fatigue (Sobrero et al., 2001). In contrast, unusually high hematocrit may indicate dehydration, smoking, or conditions such as polycythemia, all of which are linked to increased cardiovascular risk (Wakabayashi, 2022). Because testosterone stimulates erythropoiesis – the production of red blood cells in bone marrow – hematocrit can also serve as an indirect biomarker of androgenic status (Bachman et al., 2010, 2014). Men with healthier hematocrit levels may thus enjoy both biological and behavioral advantages relevant to fertility, including greater endurance, stronger libido, and higher perceived vigor. Empirically, one would therefore expect a non-linear association, where both unusually low and unusually high hematocrit levels are linked to reduced fertility.

Data and Methods

Data. We use Swedish population register data to examine the relationship between four different measures of strength (handgrip, arms, legs, and a composite measure), four measures of cardiovascular health (blood pressure, resting heart rate, physical fitness, and whether an electrocardiogram indicated any pathological signs), and four blood- and urine-based measures of physiological function (hematocrit, erythrocite sedimentation rate, U-glucose, and U-albumin,

and two different dimensions of male fertility. Using population registers we are able to capture the full population of men born in the cohorts that we study, including institutionalised individuals. This large data also allows us to study less common dimensions of fertility, such as higher parity transitions, and the tails of the distribution of our anthropometric measures, for which it would not otherwise be possible to obtain stable estimates from survey data. Register data with monthly event histories of vital events are available from 1968 to 2012. Using personal identification numbers, we combine data from military conscription, fertility, education, and tax registers. As the vital events are based on birth records, we can only link fathers to children that are known by the authorities, though these represent over 99% of all births (SCB, 2011). As such our data are superior to self-reported information, and particularly so for assessing male fertility.

We use the Swedish Multigenerational Register to link individuals to their parents, which allows us to link them to their siblings for our sibling fixed effects analyses. We also use this information on the sibling group to construct variables for sibling group size and birth order, both of which have been linked to the anthropometric measures that we study as well as fertility (Jelenkovic et al., 2013; Myrskylä et al., 2013; Barclay and Myrskylä, 2014; Morosow and Kolk, 2019). We define our starting population as all men born in Sweden from 1951 to 1979, and whose siblings were also born in Sweden. We define a sibling group through a shared biological mother and father.

Age at Measurement of Fertility. With data up to 2024 we are able to observe fertility for these cohorts well past the ages where most fertility occurs for men. For example, for the 1979 cohort we observe fertility up to age 45, while for the 1951 cohort we observe fertility up to age 73. This assures that we have a highly complete count of fertility.

Anthropometric Measures. Each of our anthropometric measures are taken from the military conscription registers. Sweden had universal military conscription for most of the 20th century, in which all men were obliged to spend approximately one year with the military, with $> 95\%$ examined at ages 18-20. We categorize age at conscription as $\leq 17, 18, 19, 20, \geq 21$. To assess eligibility, and more importantly to select people into various branches and jobs within the military, all men in Sweden had to participate in a one to two day examination before the beginning of their conscription. During these tests, men were subject a battery of tests to assess their suitability for the armed forces, and to determine their assignment.

Strength Measures. Muscular strength was assessed through standardized dynamometer tests of handgrip, leg extension, and arm flexion strength, measured in Newtons. A composite index was calculated at the time of examination using this formula: $1.7\times$handgrip $+1.3\times$leg extension

$+0.8\times$arm flexion. This combined measure reflects overall muscular strength and was delivered to us as a single standardized score, which we divided into deciles.

Resting Heart Rate. Resting heart rate was measured in beats per minute after several minutes of rest, typically while seated or lying down. The measure reflects baseline cardiovascular function and autonomic regulation and was split into deciles for analysis. We present inverse deciles, where the highest resting heart rates are in decile 1.

Physical Fitness. Our measure for physical fitness is based upon a measure of maximal working capacity, measured in watts (*fysisk arbetsförmåga i watt*). Maximal working capacity (MWC) is measured as the maximum resistance attained in watts when riding on a stationary bike during a time period of 5 to 10 minutes, and is correlated at approximately 0.9 with maximal oxygen uptake ($V0_2$max) (Patton et al., 1982). We split this measure into deciles.

Blood Pressure at Rest. Resting blood pressure was measured in millimeters of mercury (mmHg) using a sphygmomanometer after several minutes of rest. Both systolic and diastolic pressures were recorded. We categorized blood pressure according to clinical guidelines: normal (systolic <120 and diastolic <80), elevated (120–129 and <80), hypertension stage 1 (130–139 or 80–89), hypertension stage 2 ($\geq$140 or $\geq$90), and hypertensive crisis ($>$180 or $>$120).

Electrocardiogram at Rest. A resting electrocardiogram (ECG) was used to assess cardiac electrical activity and identify potential abnormalities. Examiners coded results as nothing pathological, no clear pathology, suspected pathology, or pathological, reflecting increasing degrees of abnormality in cardiac function.

Hematocrit. Hematocrit measures the proportion of red blood cells in the blood and provides an indicator of oxygen-carrying capacity. Low hematocrit values may indicate anemia or reduced erythrocyte production, whereas high values can reflect dehydration or increased red cell mass. We categorized hematocrit as low (40), normal (41–50), or high (51).

Inflammation. Inflammation was assessed using the erythrocyte sedimentation rate (ESR), which captures the tendency of red blood cells to settle in plasma over time. Higher values indicate acute or chronic inflammatory activity or infection. We categorized ESR values as low (0–10 mm/h), moderate (11–14 mm/h), or high (15 mm/h).

U-glucose. Urinary glucose was measured with a dipstick test and recorded as negative, trace, or positive. The presence of glucose in urine indicates glycosuria, a potential sign of impaired glucose metabolism or undiagnosed diabetes.

U-albumin. Urinary albumin was measured with a dipstick test and recorded as negative, trace, or positive. Elevated albumin levels indicate albuminuria, reflecting increased permeability of the renal glomeruli and possible early signs of kidney dysfunction or vascular stress.

Statistical Analyses.

Regression Analyses. We conduct regression analyses to examine how our anthropometric measures are associated with total number of children as well as childlessness by the end of the follow-up period. To study childlessness, we apply linear probability models. We present linear regressions where we use all men in the population, as well as fixed effects models in which we examine differences between full biological siblings. For each fertility outcome that we analyse, whether that is total number of children, or a given parity transition, we present the results from two different models:

$$(1) \qquad y_i = \beta_1 Anthropometric_i + \beta_2 BirthYear_i + \beta_3 ConAge_i + \alpha + \varepsilon_i$$

$$(2) \qquad y_{ij} = \beta_1 Anthropometric_{ij} + \beta_2 BirthYear_{ij} + \beta_3 ConAge_{ij} + \beta_3 BO_{ij} + \alpha_j + \varepsilon_{ij}$$

where y denotes our outcome variable, whether that is total number of children, or a binary variable for childlessness, for an individual i, with constant α and error term ε. We run separate models for each of our three anthropometric measures. In Model 1 we control for birth cohort, *BirthYear*, using individual-year dummy variables (e.g. $1951, 1952, ..., 1979$), and *ConAge*, age at time of the conscription test ($< 17, 17, 18, 19, 20, > 20$). In Model 2 we introduce additional control variables for birth order, *BO* $(1, 2, ..., 6+)$, and we introduce a sibling fixed effect, denoted by α_j, indicating that the estimates are calculated for each individual i in sibling group j.

Our sibling comparison models allow us to hold constant all factors that are shared by siblings, such as parental educational level and parental income, parental behaviour and personality, and genetic similarity between brothers. These models therefore allow us to examine the importance of our anthropometric measures for fertility net of important shared genetic and environmental factors that influence the various dimensions of health that we study as well as fertility preferences.

RESULTS

Descriptives. Figure 1 shows the distribution of completed parity amongst these men born 1951-1979, where 25% are childless, and the most common number of children is two – approximately 37% of men have two children. Having four or more children is relatively uncommon. Descriptive graphs for each of the 12 health measures that we study can be found in the

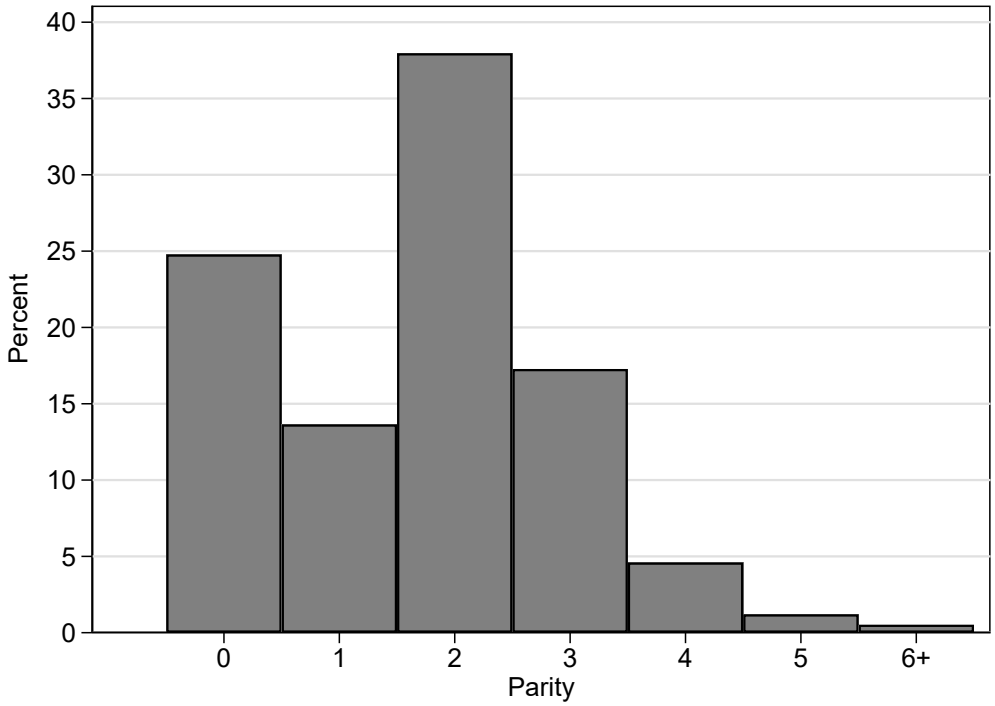

FIGURE 1. Distribution of completed parity for men born in Sweden, 1951–1979.

Supplementary Materials, in Figures S1-S12. Each of these 12 figures includes two panels. The top panel shows missing data by birth year, and distinguishes between those who attended the conscription test but do not have a measure for a given health dimension, and those for whom data is missing and they did not attend the conscription examination (based on whether a date is recorded for the conscription examination). Generally speaking, for our analysis of each health measure, we focus on cohorts where we have information for 80 to 90% of men born in Sweden in those years. Thus, the exact birth cohorts examined differ by the health measure.

Total Number of Children. In this section we present the results from analyses where we examine the relationship between the various health measures and completed parity, i.e. the total number of children that the index persons have had, by the end of the follow-up period. Figure 2 shows the results for the four strength measures, each split into deciles. These results show a very strong association between each of these measures of strength and completed fertility. We observe a negative monotic association between strength and fertility, where men in the weakest decile have approximately 0.25 to 0.5 fewer children than the strongest decile of men, depending on the measure of strength, while the handgrip measure of strength show that men in the weakest decile have approximately 0.4 fewer children than men in the strongest decile. Remarkably, these results persist with very little difference in the sibling comparison analysis

that hold constant all shared family background factors. Although there is a negative monotonic pattern, we observe a particularly large difference between the weakest (1st decile) and second-weakest (2nd decile) deciles of men – in the case of handgrip strength, for example, there is a difference of more than 0.15 children just between these two categories.

Figure 3 shows the results by the various measures of cardiovascular health in our data. The results for resting heart rate and physical fitness, based on a stationary bike test, are shown by deciles. Men with highest resting heart rates have approximately 0.34 fewer children than men with the lowest resting heart rates without applying the sibling comparison, and 0.25 fewer with the sibling comparison; men with the lowest physical fitness have approximately 0.45 fewer children than men with the lowest resting heart rates without applying the sibling comparison, and 0.35 fewer with the sibling comparison – large differences in both cases. The results for blood pressure show smaller differences, where men with blood pressure classified as elevated, hypertension category 1, or hypertension category 2 have, respectively, 0.01, 0.05, and 0.11 fewer children than men with normal blood pressure. The results from the sibling comparison analysis are slightly smaller, but remain statistically significant and follow the same pattern.

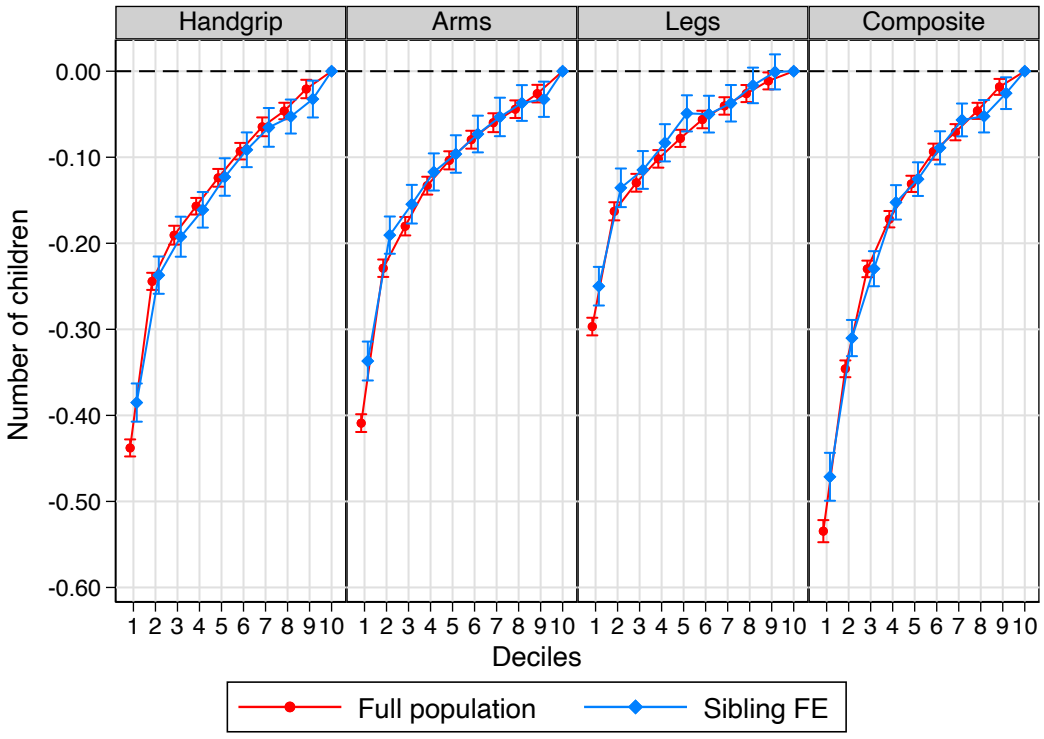

FIGURE 2. Total number of children by physical strength measured in early adulthood for men born in Sweden, 1951–1975 (1951-1979 for composite measure). Error bars are 95% confidence intervals

Men who exhibit some kind of pathological, or potentially pathological, pattern on the electrocardiogram examination also have up to 0.05 fewer children than men who exhibited no signs of pathology.

Figure 4 shows the results for the relationship between four different blood- or urine-based tests and completed parity. Abnormally high levels of hematocrit are associated with approximately 0.06 fewer children without applying the sibling comparison, but there is no difference when comparing brothers. Individuals who exhibit high levels of inflammation are also expected to have approximately 0.04 fewer children than men with low inflammation. The difference is statistically significant in models where we do not apply sibling comparisons, but not when we do; however, the point estimate remains the same, and given the small cell size of this group, this is suggestive evidence that high inflammation may reduce fertility slightly. The results for U-glucose, indicative of diabetes, shows that men who have a positive test, or trace evidence, of glucose in the urine sample have approximately 0.15 to 0.20 fewer children than men who test negative. Finally, men who test positive for U-albumin, an indicator of kidney damage, are also expected to have approximately 0.10 fewer children than men who test negative.

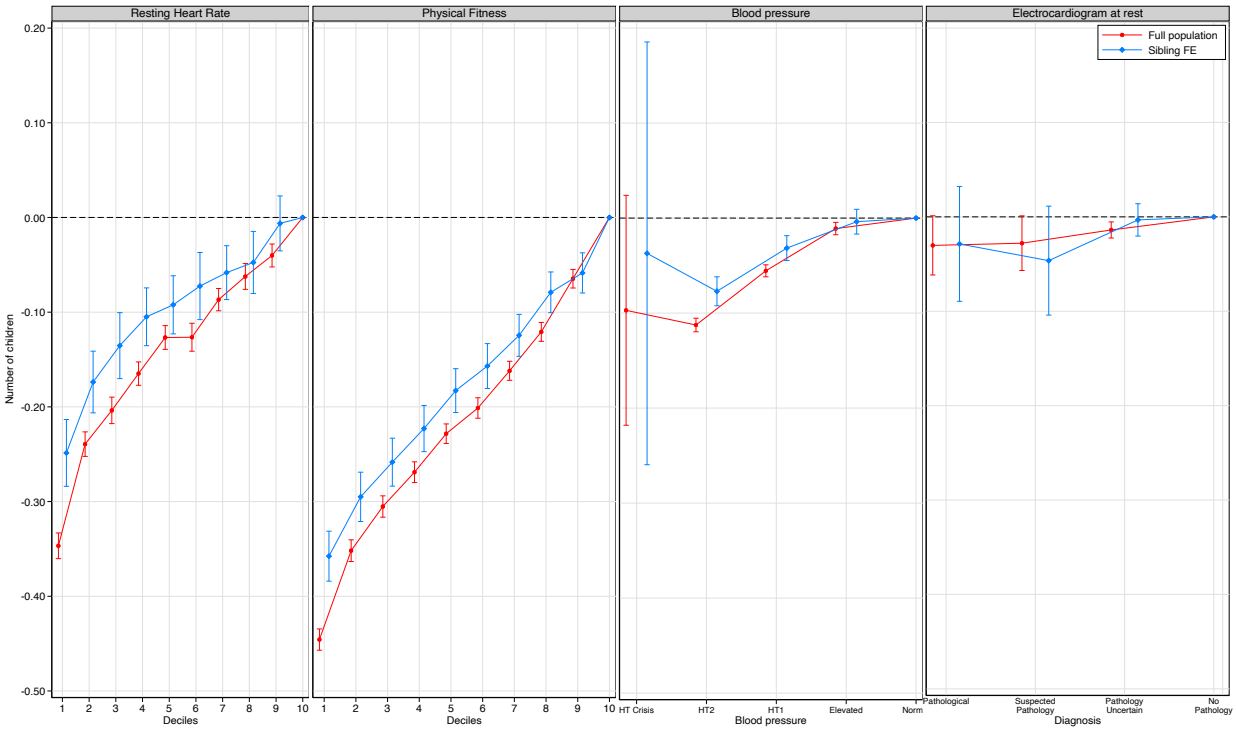

FIGURE 3. Total number of children by resting heart rate (1951-1964, 1977-1979) and physical fitness (1951-1975) measured in early adulthood for men born in Sweden. Error bars are 95% confidence intervals

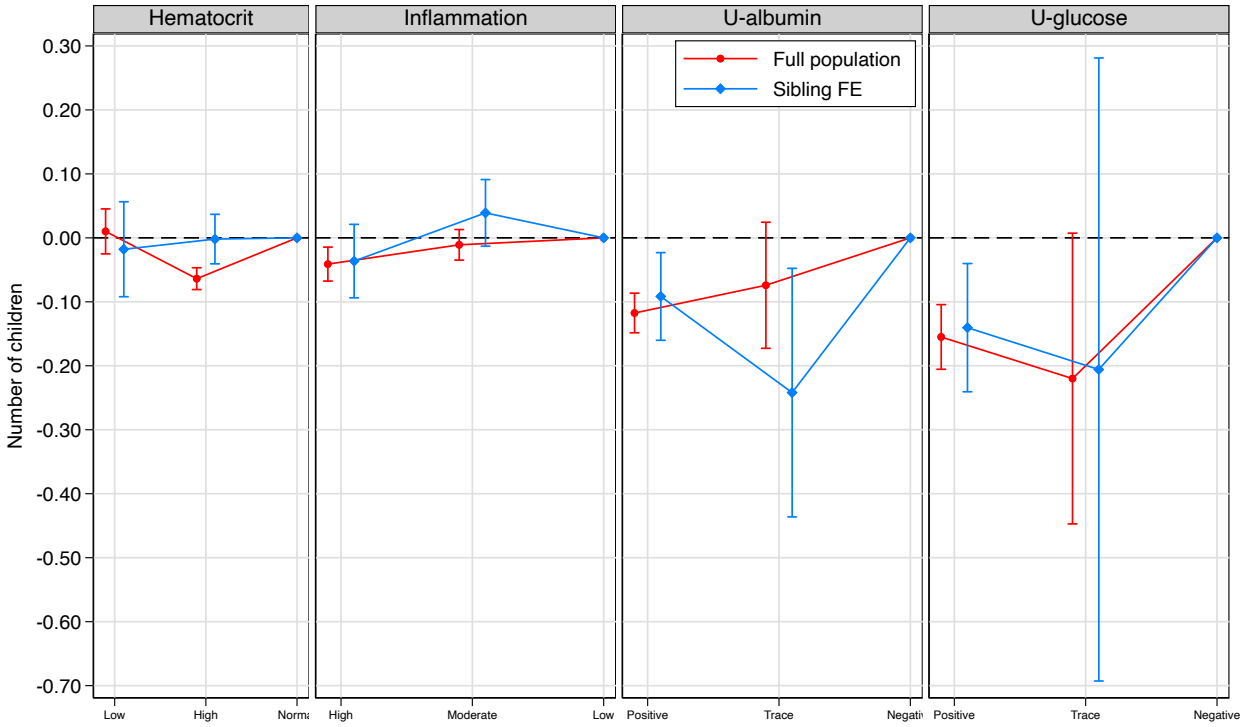

FIGURE 4. Total number of children by measures for hematocrit, inflammation, U-glucose, and U-albumin, measured in early adulthood for men born in Sweden, 1951–1964 . Error bars are 95% confidence intervals

Childlessness. We now turn to childlessness. Figure 5 shows the results by the various measures of strength. These results are based on linear probability models, and the point estimates are interpreted as percentage point differences relative to the reference category. When reading these results and evaluating the magnitude of the associations, it is worth bearing in mind that, as is shown in Figure 1, the baseline probability of childlessness in this broader cohort of men is 0.25.

We observe large differences in eventual childlessness by strength in early adulthood. The difference between the weakest decile of men and the strongest decile is approximately 20 percentage points; for handgrip, arm, and leg strength, the differences are approximately 15, 12, and 10 percentage points respectively, considering the estimates from models both with and without adjustment for shared family background factors. Once again, we observe large differences between the weakest and second-weakest decile of men, but we nevertheless observe substantial differences in childlessness between deciles 2 to 10.

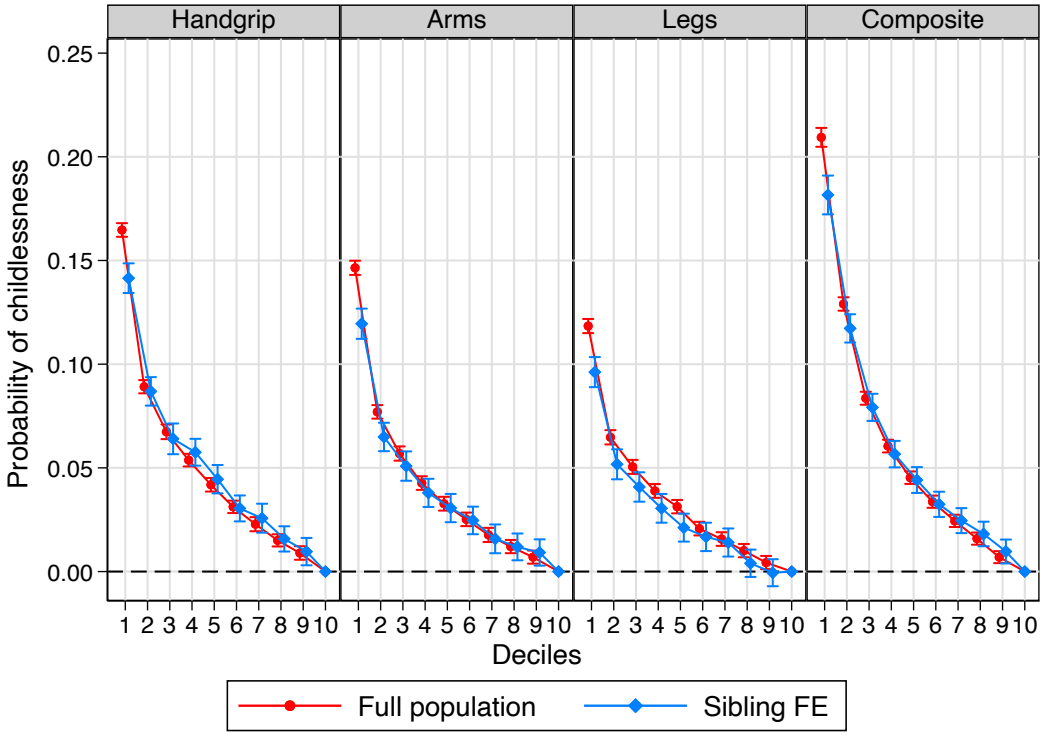

FIGURE 5. Childlessness by physical strength measured in early adulthood for men born in Sweden, 1951–1975 (1951-1979 for composite measure). Error bars are 95% confidence intervals

Figure 6 shows differences in childlessness by the four measures of cardiovascular health and fitness that we employ. The results for resting heart rate show that the probability of childlessness amongst men with the highest resting heart rates are approximately 13 percentage points higher than the probability of childlessness amongst men with the lowest resting heart rates in models without sibling comparisons, and 9 percentage points in models with sibling comparisons. The differences between the highest and lowest deciles for physical fitness are even larger, at approximately 17 percentage points in models without sibling comparisons, and 14 percentage points in models with sibling comparisons. Men whose blood pressure could already be classified as being in hypertension level 2 in early adulthood are approximately 4 percentage points more likely to be childless than men with normal blood pressure in models without sibling comparisons, and 2.5 percentage points more likely in models with sibling comparisons. We observe no statistically significant differences in childlessness by the results of the electrocardiogram examination, but the point estimates suggest a slightly higher chance of childlessness if there were indications of pathology.

Finally, Figure 7 shows the results for childlessness for the four measures derived from blood- and urine-tests. Men with abnormally high hematocrit are approximately 2.5 percentage points more likely to be childless than men with normal levels of hematocrit in models that do no apply sibling comparisons, but the differences are smaller and not statistically significant in

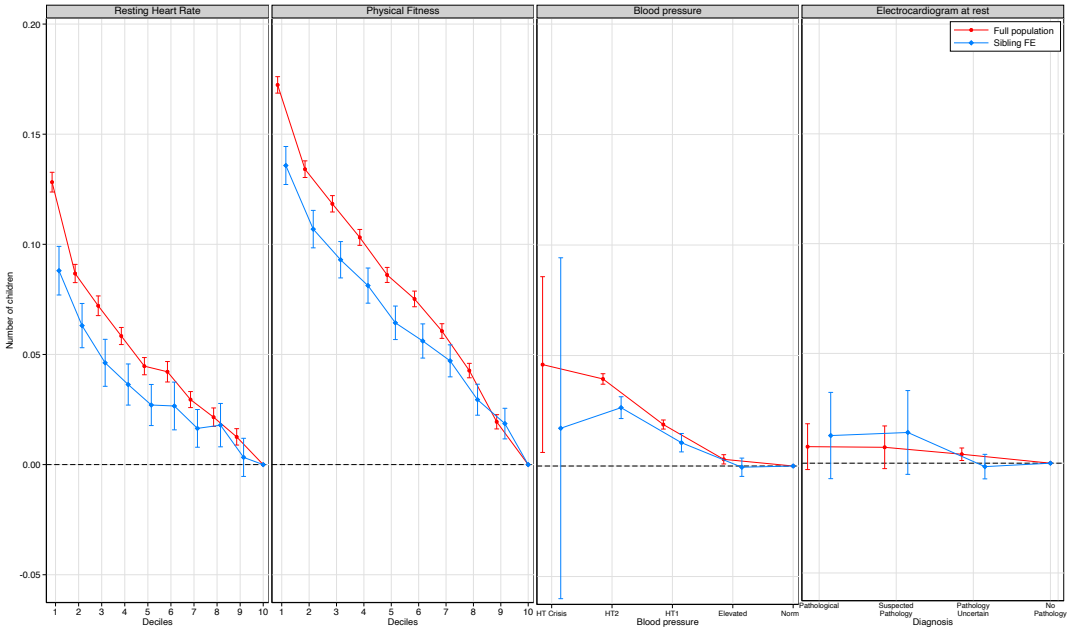

FIGURE 6. Childlessness by resting heart rate (1951-1964, 1977-1979) and physical fitness (1951-1975) measured in early adulthood for men born in Sweden. Error bars are 95% confidence intervals

models that do compare brothers. More or less the same can be said for men with high levels of inflammation relative to men with low levels of inflammation. Men who test positive for U-albumin or U-glucose are both approximately 5 percentage points more likely to be childless than men who test negative for those two markers of kidney function.

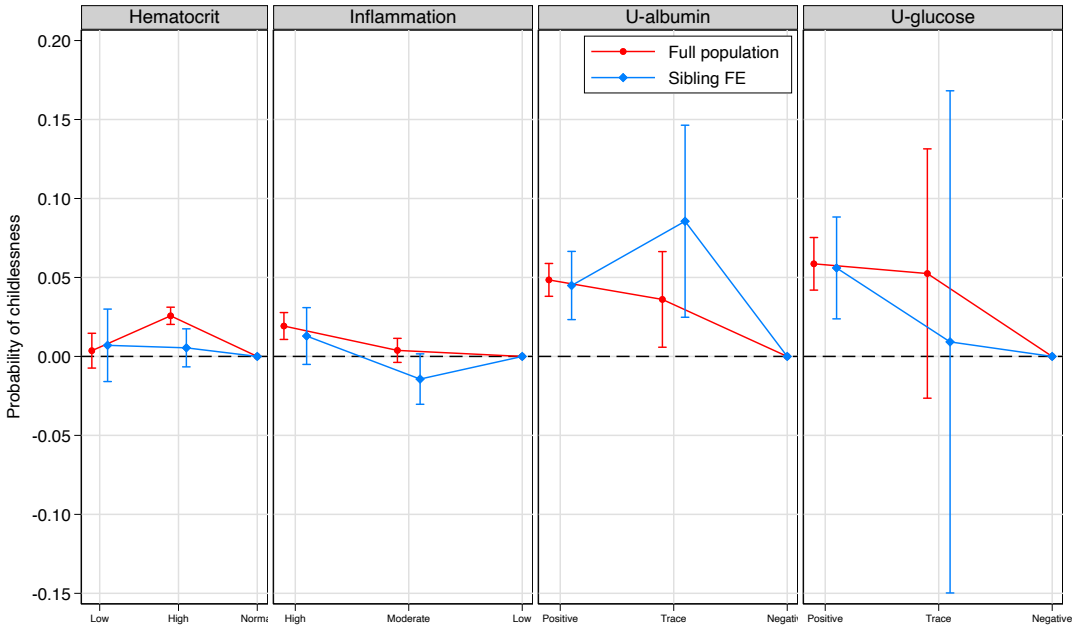

FIGURE 7. Childlessness by measures for hematocrit, inflammation, U-glucose, and U-albumin, measured in early adulthood for men born in Sweden, 1951–1964 . Error bars are 95% confidence intervals

DISCUSSION

This study leverages unusually rich population data linked to universal military conscription examinations to show that health measured in early adulthood is strongly associated with men's subsequent fertility outcomes in Sweden. Across twelve indicators spanning muscular strength, cardiovascular function, and circulating or urinary biomarkers, we find particularly pronounced associations for strength and cardiovascular fitness: men in the weakest strength deciles and the lowest fitness deciles have substantially fewer children and a markedly higher probability of remaining childless, even when comparing brothers and thereby holding constant all factors shared within families. By contrast, blood pressure categories and electrocardiogram abnormalities display smaller differences, and the blood- and urine-based markers show modest but directionally consistent associations, especially for U-glucose and U-albumin. Although we have no measure of fertility preferences, we find it unlikely that fertility preferences would align

with the patterns of childlessness that we observe for men with relatively worse health markers; our results almost certainly reflect a high degree of unwanted childlessness for Swedish men with relatively poor health. This is consistent with recent research from the UK showing that poor health leads to underachieving fertility goals (Trappolini et al., 2025). Intended childlessness in Sweden was estimated at approximately 11% amongst 18-40 year-olds in the 2011 Eurobarometer survey, but was as low as 3.7% in the equivalent 2001 survey (Miettinen and Szalma, 2014).

The pattern of results observed is consistent with the potential mechanisms for the relationship between health and fertility discussed earlier. First, measures of cardiovascular fitness, resting heart rate, and indicators of kidney function are known to have direct effects on physiological fecundity, including factors directly related to sexual function that might operate even among partnered men. Similarly, positive U-glucose and U-albumin tests are established risk signals for metabolic and vascular dysregulation, which may impair male reproductive function directly or via sexual dysfunction. Second, the large differences in fertility by measures of strength and physical fitness are consistent with a perspective that health directly affects success in terms of partnership formation. Muscular strength and fitness are visible signals of vitality and are correlated with confidence, activity participation, and physical attractiveness to women. Such traits plausibly increase opportunities for partnership formation, reduce time spent single, and facilitate repartnering after union dissolution, all of which raise the likelihood of eventual childbearing. Finally, there is also evidence that at least some of these measures of health are influential for socioeconomic attainment, which in turn affects partnership formation and union stability. Better health in early adulthood can support educational attainment, stable employment, and higher earnings, which are well-known predictors of male fertility in the Nordic context. Although we do not model any of this mediation in the current paper, our ambition is to directly investigate how much of the association between health and fertility persists after accounting for partnership formation as well as educational attainment and income.

A key factor to consider in any study examining how some dimensional of an individual profile in early adulthood affects later-life outcomes is the role of family background; it is certainly possible that early life factors, such as parental socioeconomic status and education, parental divorce, sibling group size, local contextual conditions, and even genetics, affect different aspects of health, partnership formation preferences and predispositions, and childbearing preferences. Such background factors are multitudinal, and difficult to fully control. We approximate this full control by comparing full biological siblings to one another, in order to hold constant all factors that are shared between siblings. The persistence of strong strength- and fitness-related gradients in those models indicates that the associations are not purely artifacts of shared family environments or genetics. At the same time, we acknowledge the limitations of the fixed effects

approach. Within-family comparisons do not eliminate individual-specific confounding (e.g., adolescent behaviors, injuries, or personality traits) that could affect both health and later fertility. The results we present based on sibling comparisons are therefore best interpreted as robust associations consistent with a combination of causal pathways and selection operating through individual-specific factors – something closer to the 'net effect' of the various health measures that we examine.

It is interesting to consider why strength and fitness have greater predictive power for fertility outcomes than blood pressure or resting ECG abnormalities, for example. One interpretation is that strength and fitness at ages 18–20 are integrative, high-signal measures of overall health capital and behavior: they summarize cumulative inputs from development, nutrition, physical activity, and cardiometabolic integrity. In contrast, resting blood pressure in late adolescence may have lower between-person variance, weaker predictive content for later-life disease in these cohorts, or greater measurement error in the conscription setting. Resting ECG codes capture pathology in a small subset; the resulting low prevalence and potential misclassification likely attenuate differences in fertility. The clearer patterns for U-glucose and U-albumin are consistent with these being early flags for clinically meaningful metabolic and renal stress that plausibly depress fertility through both physiological and social channels. Alternatively, one must consider that strength and physical fitness are clearer visual signals in the partner market than blood and urine markers.

Our findings underscore that men's health is a consequential component of contemporary fertility dynamics. In contexts where childbearing is concentrated in stable unions and socioeconomic resources matter for family formation, early-adult health may shape not just fecundity but also the tempo and success of partnering. Secular declines in physical fitness and increases in metabolic risk among young adults – documented in many high-income countries – could thus contribute to rising male childlessness and lower completed fertility, net of economic conditions and shifting norms. Importantly, because the strongest gradients are concentrated among men with the poorest health profiles, population-level changes in the bottom half of the health distribution may have outsized effects on the prevalence of childlessness. This is worthy of reflection given some research that suggests that although the healthiest of young men today are only somewhat less healthy than they were in earlier generations, the young men of today who dwell in the lower reaches of the health distribution are much less healthy than their counterparts in the past (Huotari et al., 2010; Albon et al., 2010; Dyrstad et al., 2012).

Although this study has numerous strengths, there are also important limitations to consider. First, while Swedish registers capture nearly all births, paternity is only observed for legally recognized fathers; misattribution is rare but not impossible. Second, we analyze cohorts conscripted between 1951 and 1979 and observe fertility through 2024; cohort differences in norms,

technology (e.g., assisted reproduction), medications (such as the recently emergent glucagon-like peptide-1 (GLP-1) drugs with proven weight loss efficacy and apparently many other positive health effects) and labor markets limit straightforward extrapolation to younger cohorts. Third, the conscription measures, though standardized, are a cross-sectional snapshot of health that may contain measurement error. Classical error would bias associations toward zero, likely making our estimates conservative. However, the military nature of the testing context may also have unpredictable influences, perhaps increasing anxiety in some individuals, for example. Fourth, we do not observe cohabiting unions without children directly, so we cannot fully separate fecundity from partnering pathways. Finally, despite sibling fixed effects, individual-specific confounding remains possible, and we stop short of causal claims.

Although this is an observational study, the patterns suggest several policy-relevant points. First, investments in adolescent and young adult health – especially interventions that raise physical activity and cardiorespiratory fitness – may yield long-run benefits that extend beyond morbidity and mortality to include partnership and family formation. Second, routine screening for early metabolic and renal dysfunction (e.g., glycosuria, albuminuria) and timely management could mitigate trajectories that ultimately depress fertility. Third, public-health messaging and clinical practice in reproductive health could more explicitly include men's health, acknowledging that male factors are a substantial share of couple infertility and that modifiable behaviors in adolescence and early adulthood matter. Alternatively, if the effects of health on fertility primarily flow through partnership formation, the benefits of such investments – at least for fertility, since the benefits for population health are clear – are rather unclear.

Men's health in early adulthood is clearly linked to later fertility outcomes. Strength and cardiovascular fitness stand out as particularly strong predictors, with metabolic and renal indicators also showing meaningful associations. These patterns are robust to stringent controls for shared family background and are consistent with multiple, potentially reinforcing, pathways – physiological fecundity, partner-market dynamics, and socioeconomic mediation. Recognizing men's early-adult health as part of the fertility equation may help us to improve our understanding of lower fertility and increasing rates of childlessness and inform public-health strategies aimed at supporting family formation.

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SUPPLEMENTARY MATERIALS

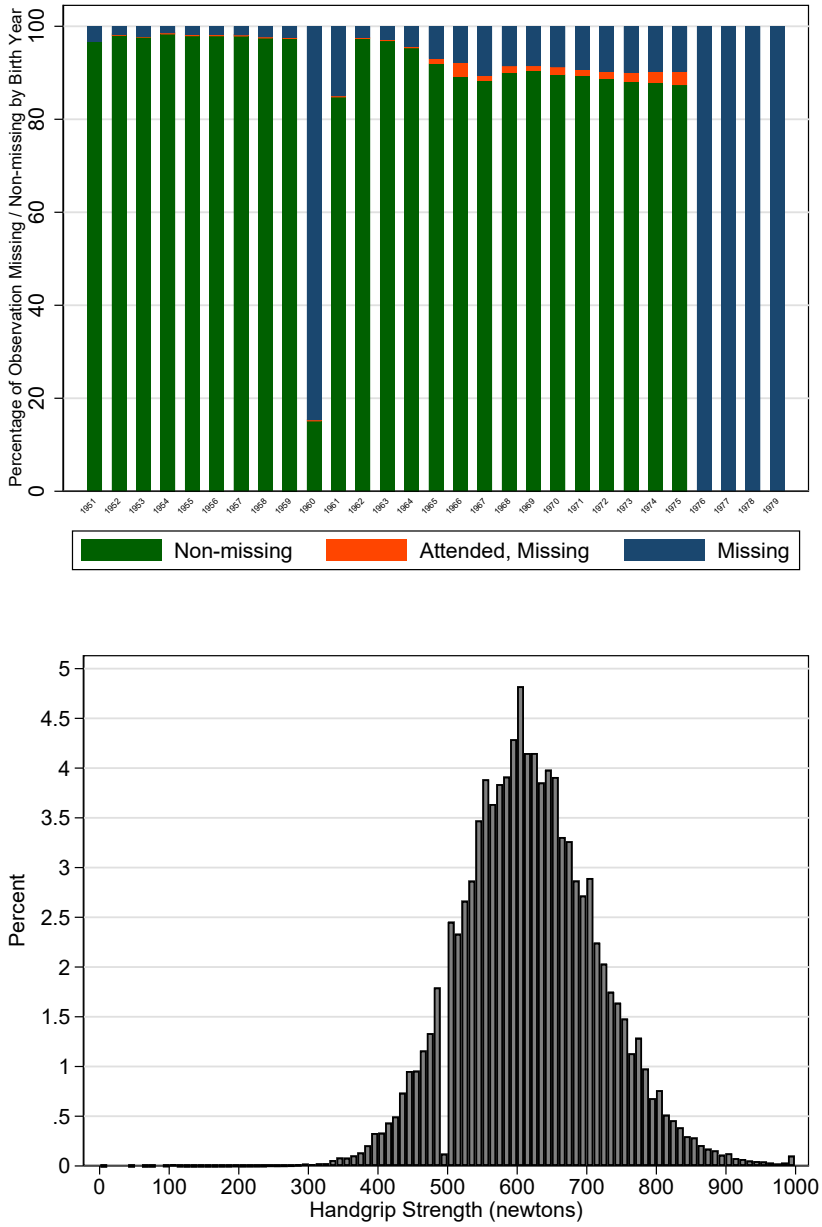

FIGURE S1. Percent data missing (%) by birth year (top) and distribution of Handgrip strength (newtons).

Handgrip strength (newtons).

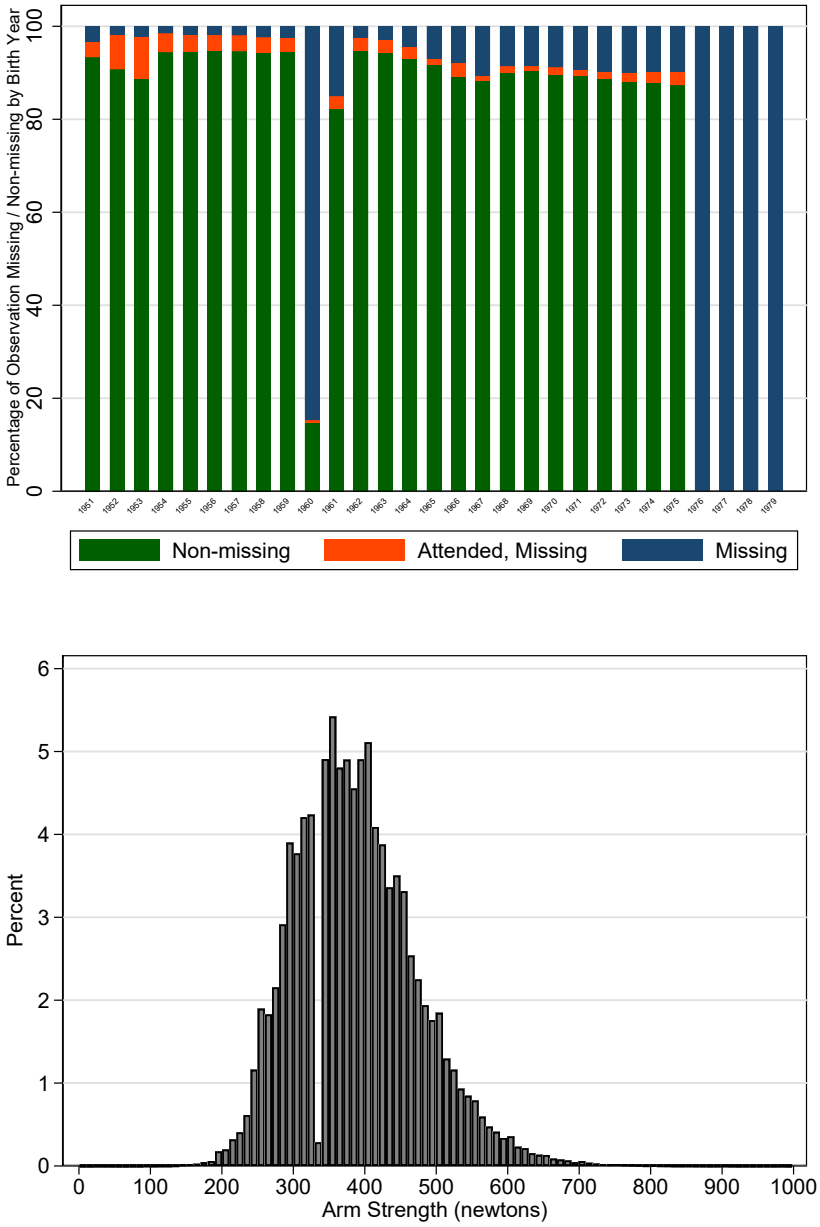

FIGURE S2. Percent data missing (%) by birth year (top) and distribution of Arm strength (newtons).

Arm strength (newtons).

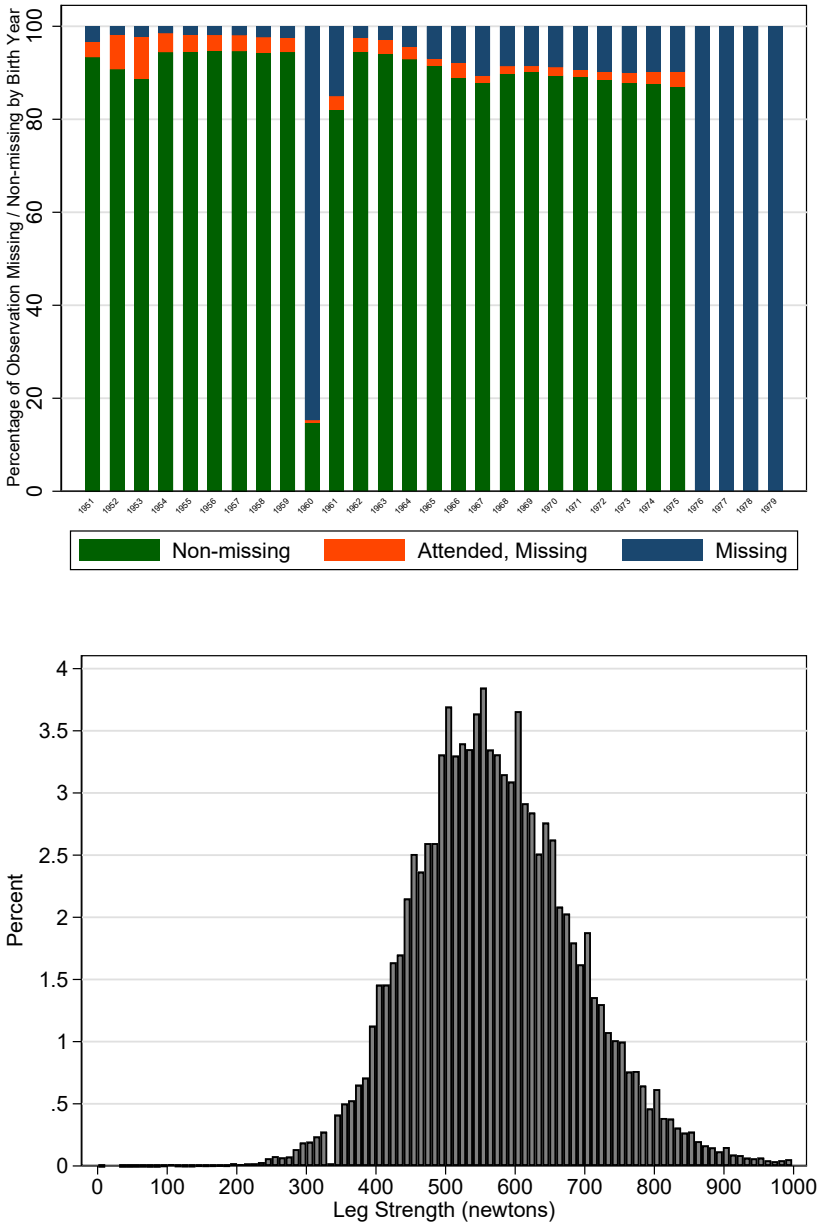

FIGURE S3. Percent data missing (%) by birth year (top) and distribution of Leg strength (newtons).

Leg strength (newtons).

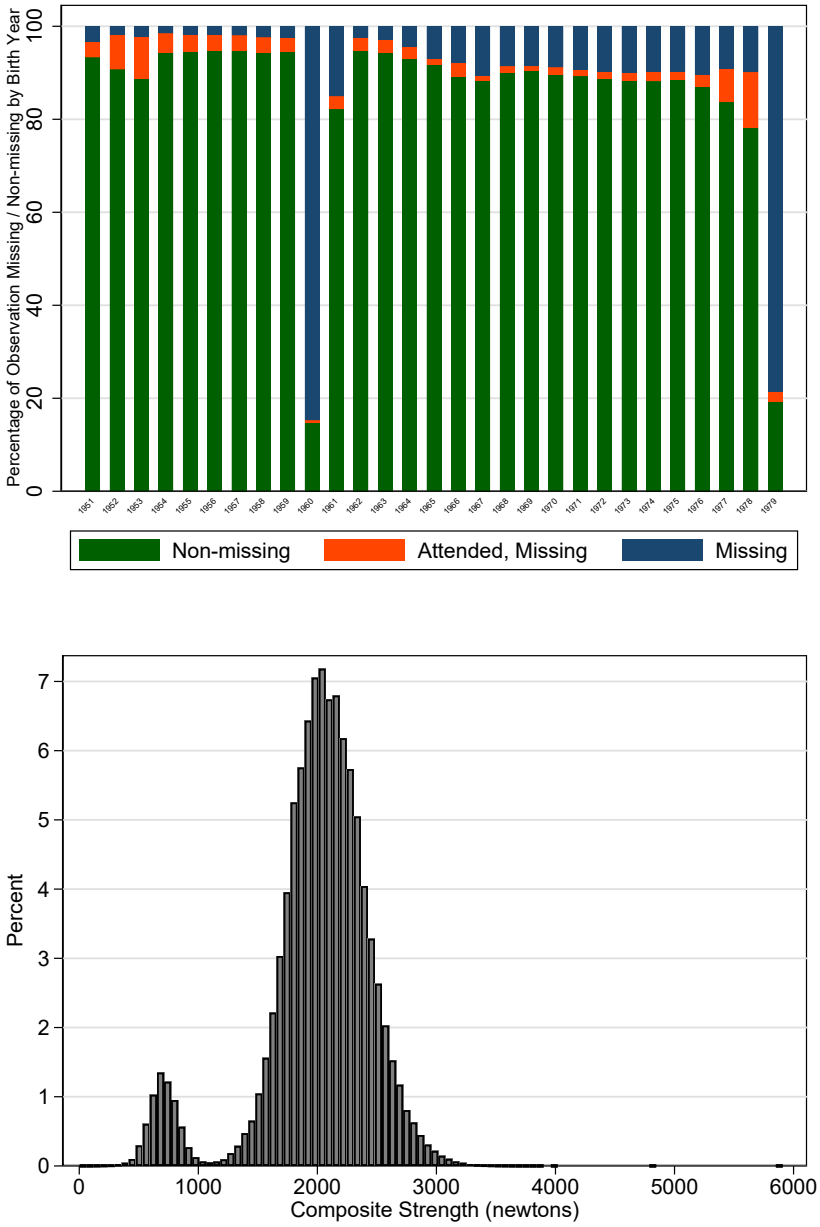

FIGURE S4. Percent data missing (%) by birth year (top) and distribution of Composite strength measure (newtons).

Composite strength measure (newtons).

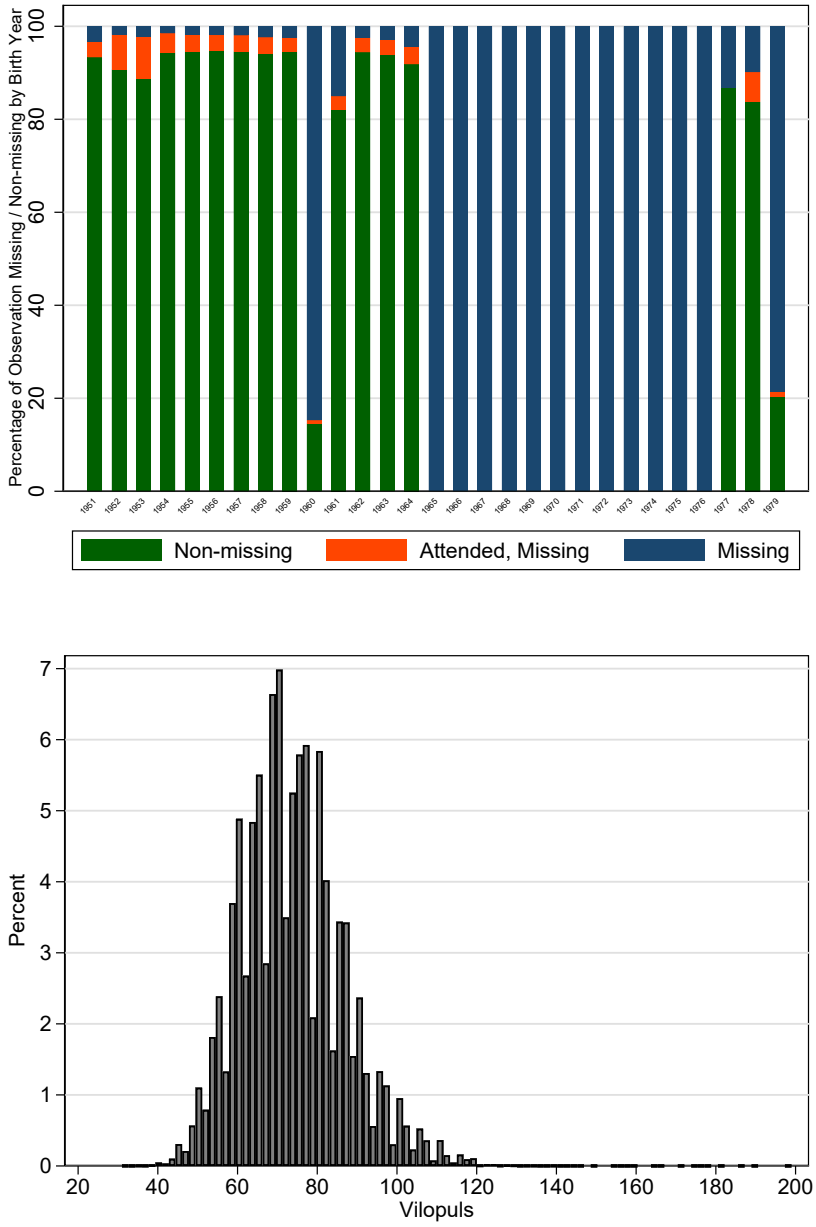

FIGURE S5. Percent data missing (%) by birth year (top) and distribution of Resting heart rate.

Resting heart rate.

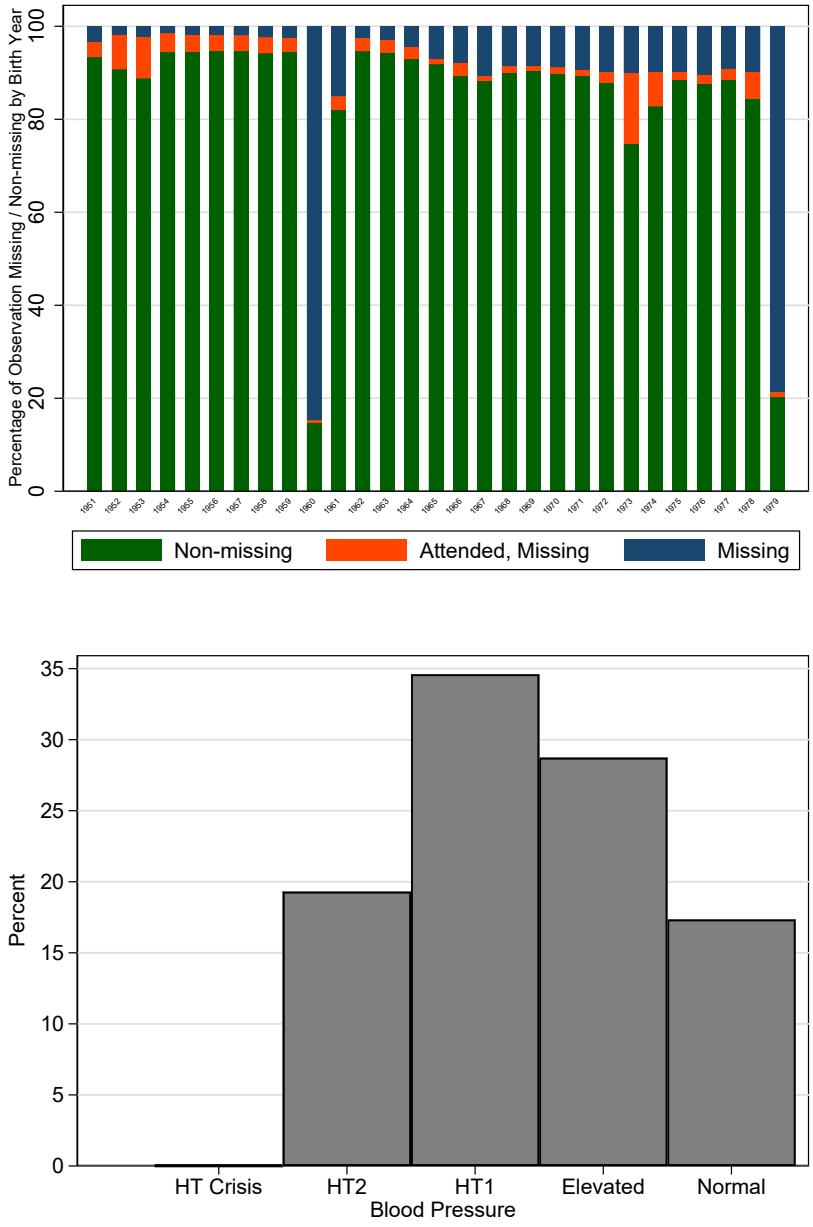

FIGURE S6. Percent data missing (%) by birth year (top) and distribution of Blood pressure.

Blood pressure.

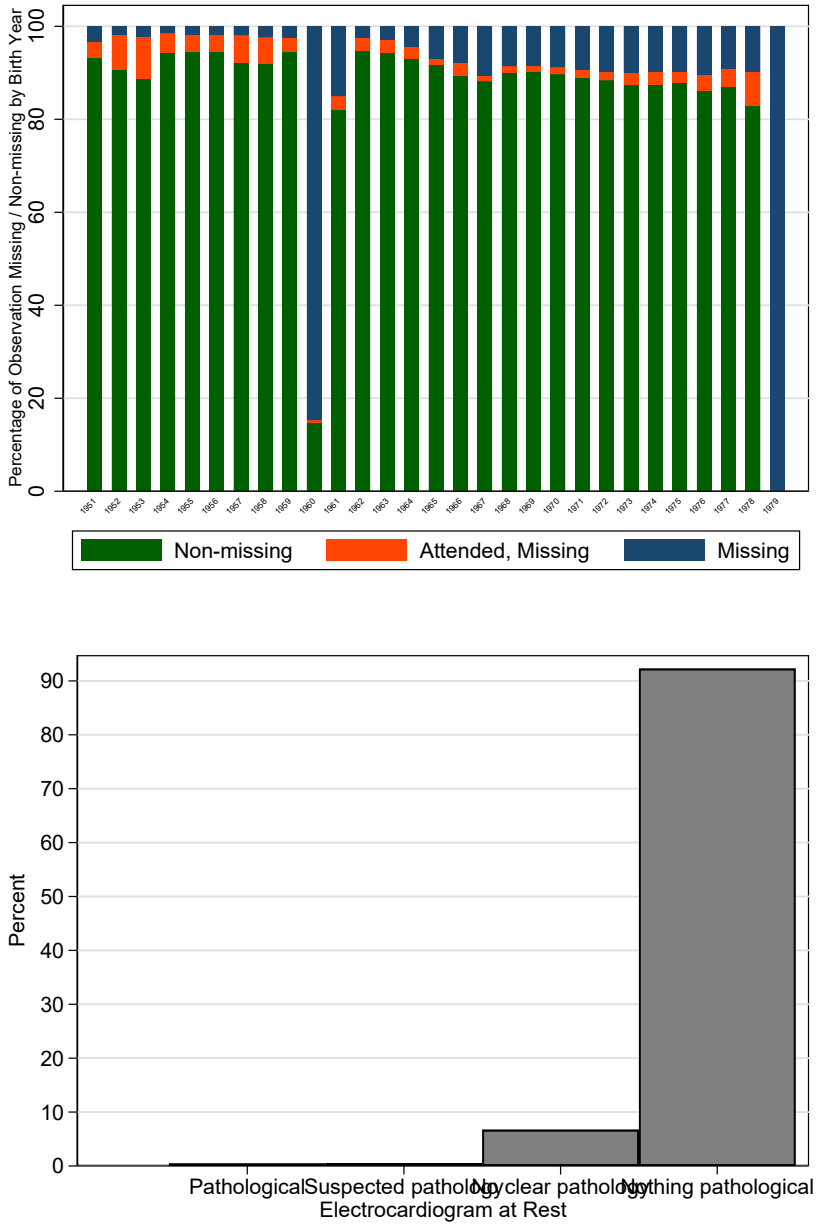

FIGURE S7. Percent data missing (%) by birth year (top) and distribution of Electrocardiogram at rest.

Electrocardiogram at rest.

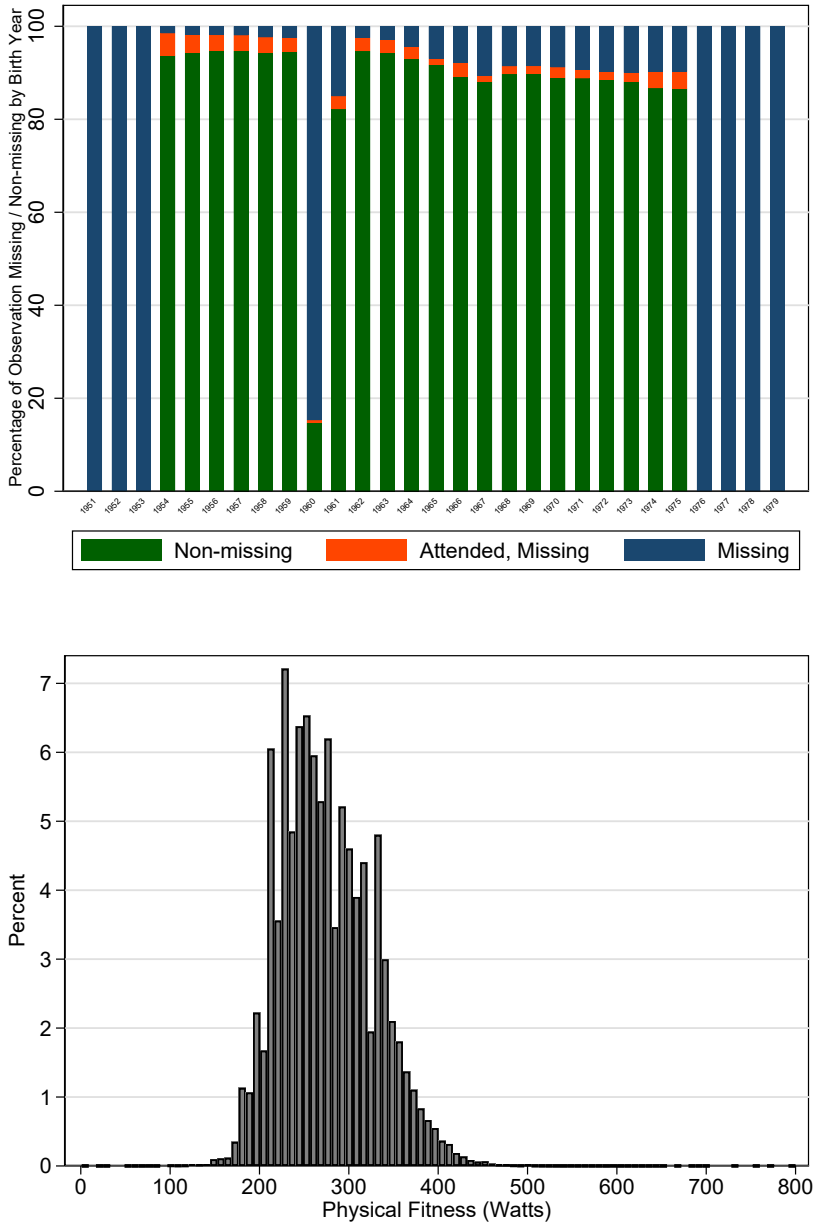

FIGURE S8. Percent data missing (%) by birth year (top) and distribution of Physical fitness (Watts).

Physical fitness (Watts).

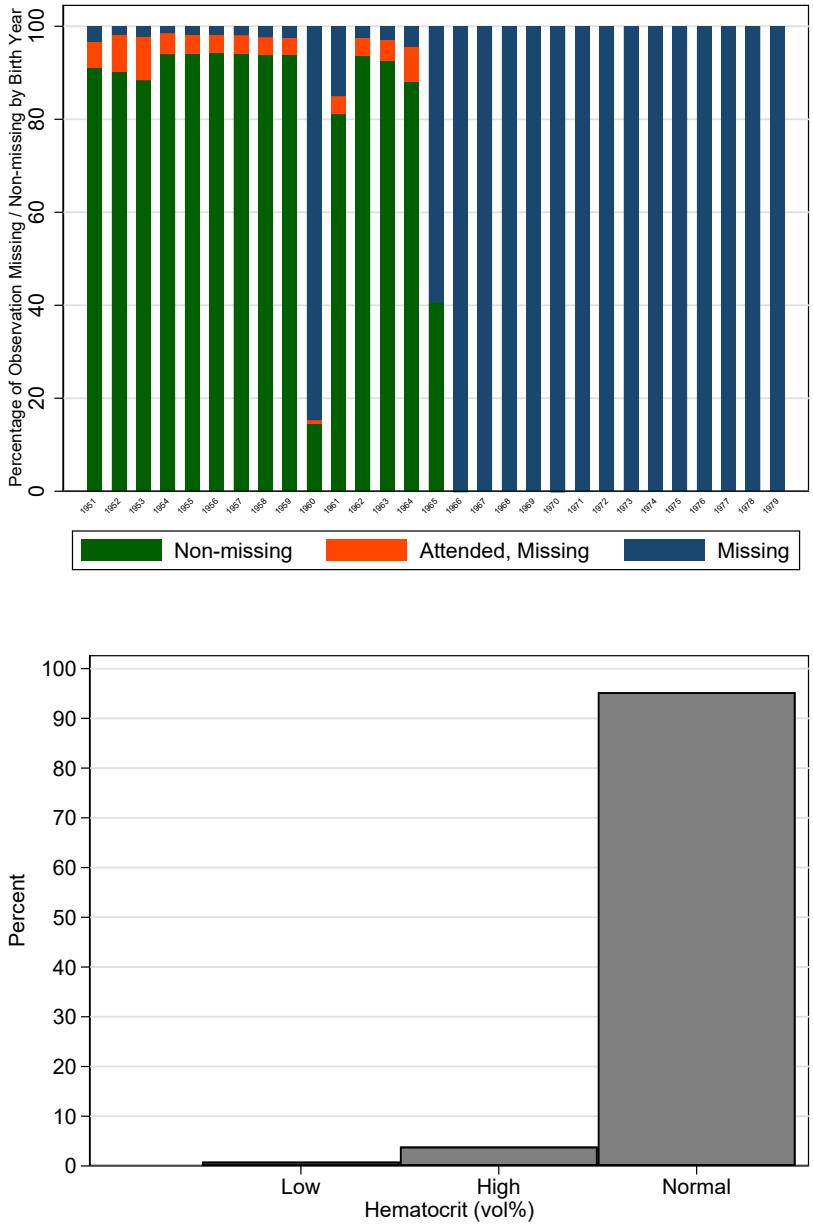

FIGURE S9. Percent data missing (%) by birth year (top) and distribution of Hematocrit (vol%.

Hematocrit (vol%.

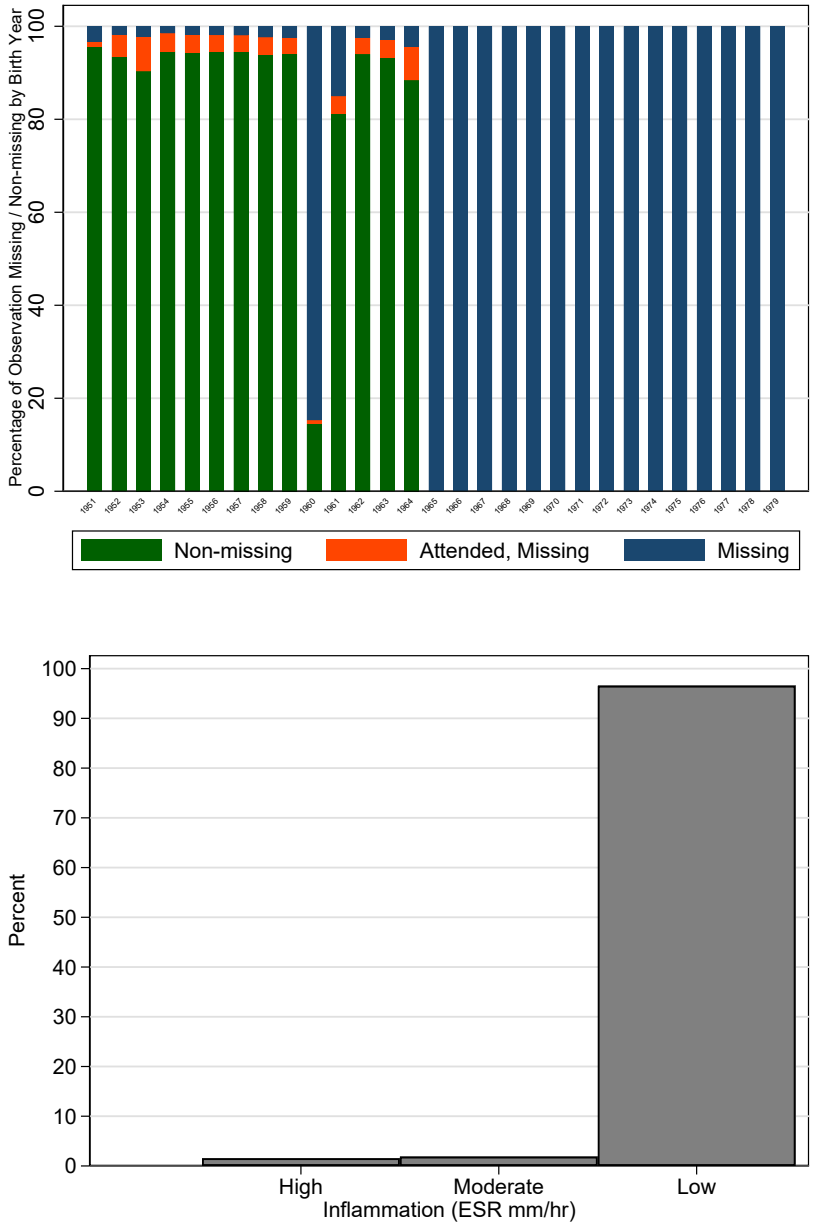

FIGURE S10. Percent data missing (%) by birth year (top) and distribution of Inflammation (ESR mm/hr).

Inflammation (ESR mm/hr).

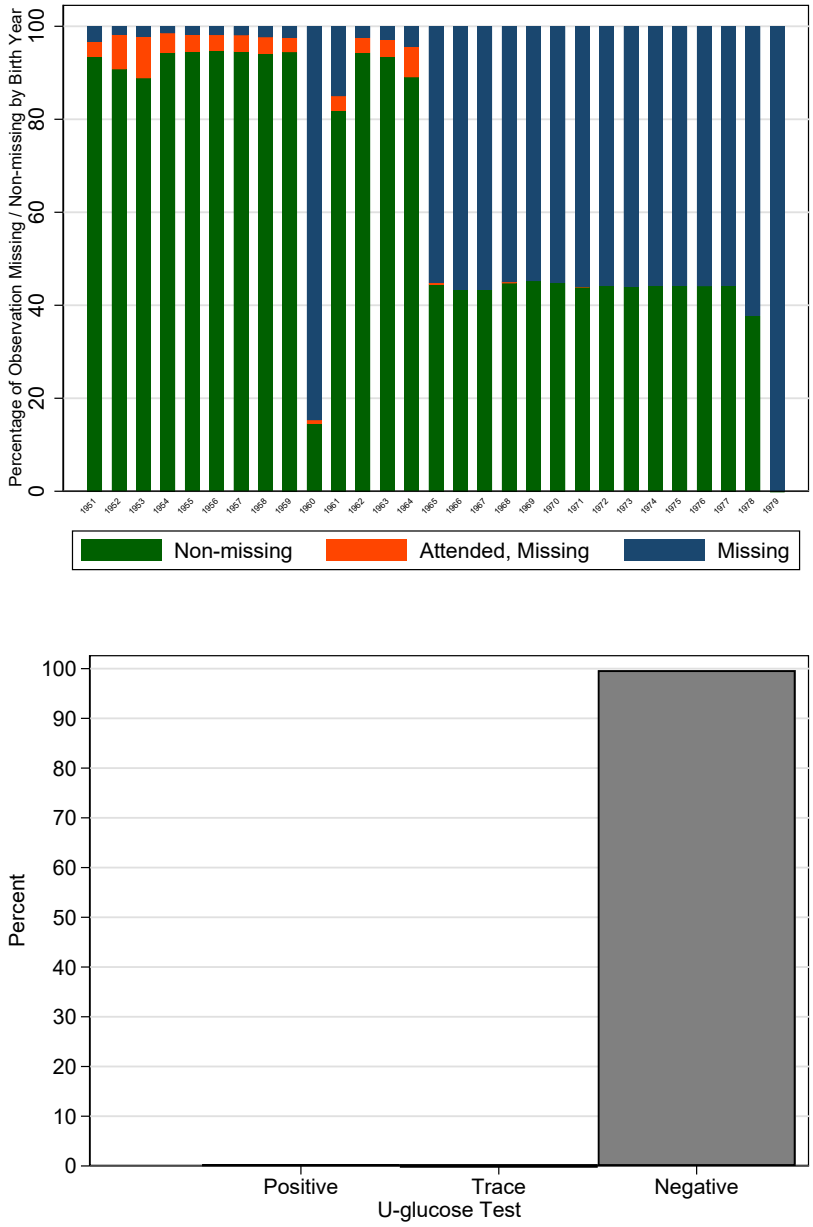

FIGURE S11. Percent data missing (%) by birth year (top) and distribution of U-glucose.

U-glucose.

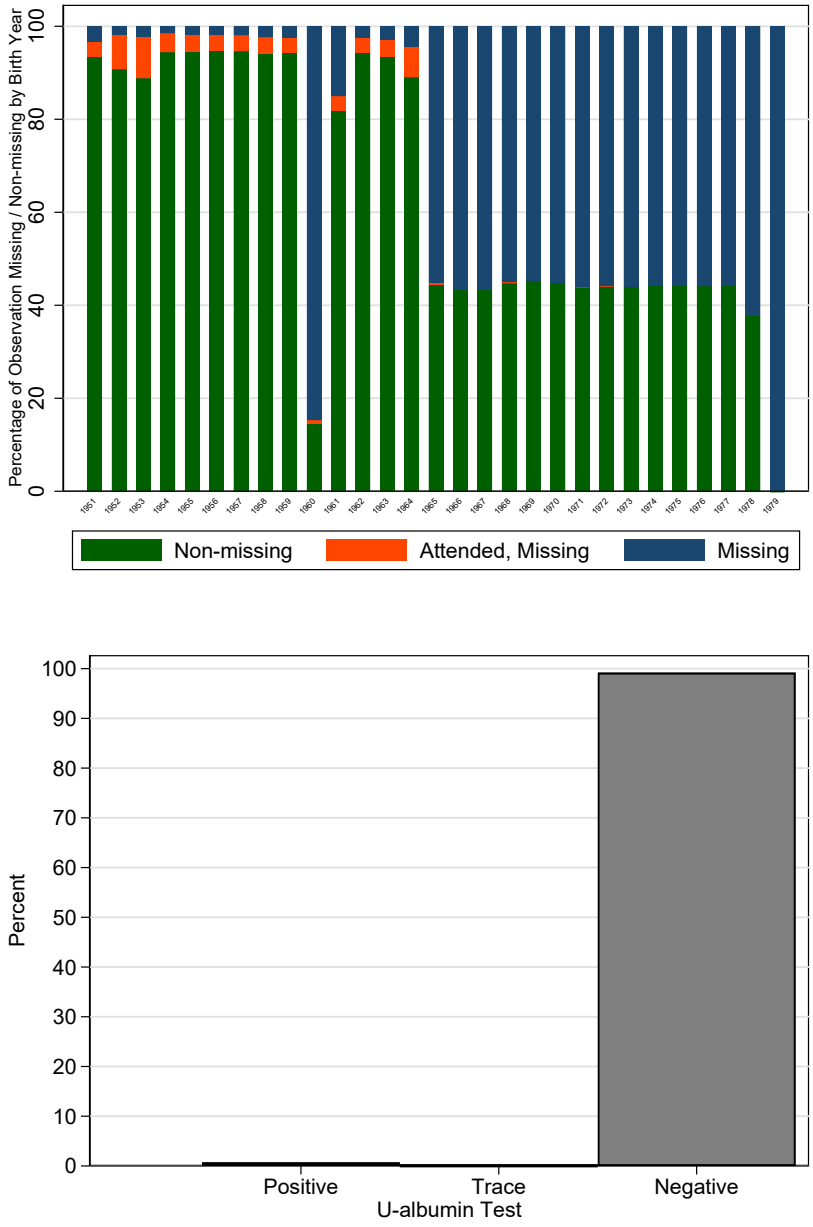

FIGURE S12. Percent data missing (%) by birth year (top) and distribution of U-albumin.

U-albumin.