

US Infant Mortality under Slavery and after Emancipation: New Evidence from Childhood Sex Ratios¹

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Abstract

I use childhood sex ratios to characterize infant mortality rates among the US Black population 1850–1880, until now a matter of speculation due to a lack of birth and death records. Because of the biological survival advantage of infant females, high rates of infant mortality tend to skew the surviving population toward females. Building on this well-known fact, I use vital statistics data from contemporary Europe to quantify the empirical relationship between infant mortality and childhood sex ratios. Applying this relationship to the 19th century US, I compare infant mortality between the Black and white populations under slavery, and infant mortality among US Blacks before and after emancipation. Circa 1850 to 1860, the infant mortality rate among the Black population was around 300 deaths per 1,000, while the rate among whites was likely below 100. Infant mortality for US Blacks improved substantially after emancipation, dropping nearly 100 points to around 200 deaths per 1000, while white infant mortality remained roughly the same, cutting the Black-white disparity in half.

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Highlights:

- I use childhood sex ratios to provide new evidence on infant mortality and population health for the US Black population in the 19th century, overcoming the lack of data which has limited prior research.
- I find that infant mortality for the Black population was extremely high under slavery (circa 1850–1860), some 200 points higher than for contemporary whites.
- I find that infant mortality for the Black population improved substantially after emancipation (circa 1870–1880), although it still remained well above that of the white population.

Keywords: population health, infant mortality, US slavery, sex ratios

Introduction

Throughout US history, debates over slavery have occupied both the public and scholarly discourse.³ Patterns of population health — and living standards — remain an open question for the 19th-century US, because of the late development of vital statistics (birth and death records).⁴ Social historians (e.g., Morgan 2001; Jones 2010; Baptiste 2014) cite work from the 1960s to 1980s (e.g., Farley 1965; Eblen 1974; Steckel 1986) as providing estimates of infant mortality (IMR) for the enslaved. However, as discussed below, these demographic studies are deeply speculative, and often have no basis in actual data on child survival. Put simply, while we may presume the “social death” (Patterson 1982) of slavery translated into premature death, IMR among US Blacks in the 19th century remains unknown.

The lack of evidence on US infant mortality in the 19th century has allowed an idea to persist: that Black and white populations shared broadly similar demographic rates under slavery. This view is largely based on the fact that the enslaved Black population grew at a similar rate as native-born whites (Engerman 1976), and were tall by contemporary standards (Schneider 2017). At the extreme, Fogel and Engerman (1974, 1:123–26) claimed that infant mortality among Southern whites and enslaved Blacks was “virtually the same”,⁵ and that the poor health of US Blacks under slavery was due to “the primitive nature of medical knowledge and practices in the antebellum era,” rather than the actual social structure of slavery (ibid).⁶ This view continues

³ For a recent example, see the controversy, both scholarly and public, surrounding the *1619 Project* (Hannah-Jones 2019); see Serwer (2019) for a discussion.

⁴ For illustration of this point, look no further than the recent controversy over the ‘antebellum puzzle’, where researchers tried to determine a basic trend in US heights in the 19th century. See Margo and Steckel (1983) and Komlos (1987) for classic statements of the ‘puzzle’, Bodenhorn et al. (2017) for a critique, and Komlos and O’Hearn (2019) and Zimran (2019) for rebuttals. This debate is only possible because of the paucity of sources on population health in the 19th century US.

⁵ This statement was, in any case, an error. Their Black infant mortality rate was taken from Evans (1962) as being around 180 deaths per 1000. But as discussed below, Evans reported the incorrect value from his life tables, and his actual value should have been around 250.

⁶ See Postell (1952, 538) for a similar view, writing in *Pediatrics* that the medical care provided to enslaved mothers was “still far superior to anything the Negro had known in Africa, and compared favorably with the medical knowledge of the antebellum period.” Such views, evoking slavery apologists, should be discarded. See Schwartz

today, as Schneider (2017, 15) concludes from height data that “adult female slaves experienced fairly good conditions relative to other historical populations.” Furthermore, existing demographic studies claim that Black mortality rates failed to improve after emancipation (Farley 1965; Eblen 1974), downplaying the impact of slavery on population health.

I provide the first population-level evidence against these views, drawing on a novel basis for estimating infant mortality: childhood sex ratios. Because of the female survival advantage in gestation and infancy, insults to maternal and infant health are reflected in female-skewed sex ratios. Building on McDevitt-Irwin and Irwin (2024), I use a range of data from Europe and settler colonies circa 1840–1960 as a training data set, where we observe both IMR and CSR, in order to verify the empirical relationship between the two variables. Then I use this estimated relationship to infer infant mortality for US Blacks in the 19th century, based on sex ratios from the decennial US census.

I find that infant mortality among US Blacks was extremely high under slavery, approaching 300 deaths per 1000. IMR among US whites was relatively low throughout the 19th century, likely under 100. I further find, in contrast to existing research, that Black IMR improved substantially after emancipation, dropping to around 200 deaths per 1000.

Literature Review

Little is known about infant mortality among the US Black population in the 19th century. This reflects a lack of data, not a lack of interest, as academic and popular writers alike regularly allude to the deep historical roots of racial health inequities.⁷ Historians of slavery have

(2010) for an example of recent scholarship on reproductive health under slavery, and Owens and Fett (2019) for discussion of the troubling relationship of medicine and public health to slavery and race in US history.

⁷ For examples of popular discussion of the historical roots of racial health inequities, see Chakraborty (2017), Villarosa (2018), Joyner and Lee (2020), Kyere (2020), and Craven and Snipe (2022). For examples of academic discussion, see Esenwa et al. (2018), Owens and Fett (2019), Thomas and Casper (2019), Hammonds and Reverby (2019) and the Harvard School of Health’s “400 Years of Inequality” event.

offered varied examples of slave infant mortality rates in specific times and places, based on (rare) plantation records with births and infants of the enslaved. Unfortunately, no representative data on mortality are available at the population level (for Blacks or whites) until the 1900 census. The US was a laggard in collecting vital statistics, and birth and death records did not cover much of the US until the 1920s ([US-CDC 1954: 12-14](#)).⁸ Absent requisite data for direct or indirect techniques (UN 1984), 20th-century researchers stretched limited source materials into conjectural estimates of infant mortality rates.

One group of estimates of US Black infant mortality in the 19th century comes from life-table exercises using decennial census data. Farley (1965) uses stable population analysis (UN 1984, chapter 7) and Eblen (1974) uses inter-censal survival methods (UN 1984, chapter 9) to fit model life tables to 19th-century US census data, in order to generate mortality rates by age, including infant mortality.⁹ These decadal estimates range from around 200 deaths per 1000 births (Eblen 1974, table 7) to roughly 300 (Farley 1965, 395).¹⁰ Both of these studies also argue that infant mortality rates failed to improve in the decades following the Civil War and the abolition of slavery. Eblen (1974, table 7) finds that infant mortality for the Black population stayed nearly constant across the 19th century, while Farley (1965, 396) finds that it increased after emancipation.

⁸ We similarly lack the requisite data for indirect estimates of infant mortality for the 19th century, although they are available from the 1900 and 1910 census (Preston and Haines 1991).

⁹ Note that Farley (1970, 65) also applies inter-censal survival to the US Black population in the 19th century, but refrains from offering infant mortality rates from this exercise. In my view, this abstention is correct, as such methods are inappropriate for estimating child mortality, a point I discuss further below.

¹⁰ Evans (1962) was often cited for an IMR of 183 in the US slave population, but that value is mistaken, reflecting an erroneous application of Jacobson's (1957) life table for US whites in 1850. Evans (p. 212) uses values for infant death rates which are in fact the averages of the age-0 and age-1 mortality rates (qx) in Jacobson's life table (1957:198). Fogel and Engerman (1974) was a prominent work to rely on Evans for infant mortality.

Another source for infant mortality rates during the Antebellum period is plantation records on births and deaths. Although better than census populations counts for measuring infant mortality, plantation records are extremely limited in scope, and are in no sense representative of the enslaved population as a whole. Steckel (1979, table 2) uses records from 11 plantations in order to make direct estimates of IMR, finding a rate of 233 deaths per 1000. Campbell (1984, p. 797) finds an IMR of 353 on one plantation over 24 years.

Evidence on heights by age offers another perspective on population health. Steckel (1986) shows that enslaved children (ages 1-6) were generally stunted, suggesting poor maternal-infant health. Steckel then extrapolates from child height to birthweight, and then from birthweight to neonatal mortality. Combining this result with plantation records on infant mortality, Steckel (1986, 193) concludes that “numbers in the range 300–350 per thousand, and possibly higher, are reasonable conjectures for slave infant mortality”. The high end of this “conjecture” (340 deaths per 1000) appears as the 1850 value of infant mortality for US Blacks in the latest *Historical Statistics of the US* (2006, Ab923),¹¹ and is thereafter cited in popular and academic works like Villarosa (2018) and Owens and Fett (2019).

In the background of these attempts to estimate Black mortality patterns in the 19th century is a well-established fact: in the Antebellum US, the enslaved Black population grew at a similar rate as the native-born white population (Engerman 1976). This rate of natural increase was notable, as nearly all other enslaved populations in the Americas were population sinks (Klein and Engerman 1978). As Fogel (1989, 1:119) discusses, the natural increase of US Blacks was interpreted by contemporaries as evidence of relatively similar living conditions of enslaved

¹¹ In HSUS, this estimate is sourced as “Richard H. Steckel, “A Dreadful Childhood: The Excess Mortality of American Slaves,” *Social Science History* 10 (4) (1986): 427-65.” However, in that 1986 paper, the number 340 does not appear. Instead, infant mortality is placed “in the neighborhood of 350” with a citation to Steckel’s (1986) *Explorations in Economic History* paper. It is unclear exactly how the number 340 ended up in *HSUS*.

US Blacks and free whites. Such arguments have continued to the more recent times, as Eblen (1972, p. 286) judges his own estimates of Black IMR under slavery to be “very high, perhaps unreasonably extreme” in light of Black population growth. Taking all of these sources of evidence into account, Fogel (1992, 286) suggests 289 deaths per 1000 as an infant mortality rate for enslaved US Blacks.

All of these estimates are deeply speculative. Direct estimates of infant mortality can only be made for a tiny, non-representative sample of the Black population. The indirect estimates of Black infant mortality, while representative, are highly conjectural, as they come from methodologies which are designed to estimate *adult*, not infant, mortality.¹² Any IMR generated by these methods is an extrapolation from indirect estimates of mortality at older ages. As discussed at length by Wood (1993), the historical relationship of infant mortality and mortality at older ages is highly variable: infant mortality and mortality at older ages are each “indispensable indices” (p. 217).¹³ Steckel’s (1986) conjecture-sum-estimate is even more problematic, as he extrapolates from child height to birthweight to infant mortality. None of these are stable relationships; there is no structural relationship between child height and infant mortality.¹⁴ These existing ‘estimates’ should be discarded as *a-priori* uninformative. Simply put, we need a new source of evidence on infant mortality in the 19th-century US Black population: enter childhood sex ratios.

¹² This is made explicit in the UN Manual X (1984) chapters on each method: stable population analysis (chapter 7) generates indirect estimates of mortality over age 5, and intercensal survival mortality age 10 and over. In some sense, this is obvious, as most infants who die within the first year of life are not recorded in census data.

¹³ Moreover, as previously noted by McDaniel and Grushka (1995), even for estimating *adult* mortality the methods applied by Farley and Eblen require strong assumptions on immigration and enumeration, which cannot be verified for the 19th-century US Black population.

¹⁴ There is no stable relationship between child height and infant mortality, particularly because of the role of selection; see Alter (2004) and Deaton (2007) for discussion. Consider, for example, that India has a rate of child stunting over 35% and child mortality is roughly 30 deaths per 1000. Nigeria has a child stunting rate under 32%, yet the child mortality rate is 114 deaths per 1000 (2020 estimates from Our World in Data).

Childhood Sex Ratios Reveal Infant Mortality¹⁵

It has long been known that, biologically, females are less vulnerable than males to infant mortality.¹⁶ Therefore, high rates of infant mortality tend to skew childhood sex ratios toward girls, unless the female biological advantage is negated by gender discrimination. Childhood sex ratios thus can offer a basis for characterizing infant mortality when data for conventional demographic methods are unavailable.¹⁷

With low infant mortality, a healthy population of young children will have some 5-6% more males than females, reflecting a healthy sex ratio at birth (Maconochie and Roman 1997; Grech, Savona-Ventura, and Vassallo-Agius 2002). However, high rates of infant death tend to leave a surviving population distinctly more female than at birth. For example, in England circa 2000, infant mortality was around 5 per 1000 (Office of National Statistics data) and there were about 5% more boys than girls under the age of 5 (Office for National Statistics 2001, tbl. P3). In 1900, England's infant mortality was around 150 per 1000, and girls outnumbered boys (see data appendix).

The effect of infant mortality on the sex composition of the surviving population is well known in demography. Familiar model life tables, such as Coale-Demeny (1983), illustrate that

¹⁵ This section draws on McDevitt-Irwin and Irwin (2024), which establishes the validity of sex ratios as an indicator for infant mortality. For a preprint of this working paper, which is currently in the revise and resubmit phase, see <https://jrmeirwin.github.io/sr1/USimr.pdf>.

¹⁶ Current knowledge is conveniently summarized by the editors of PLOS Medicine in their summary of Sawyer (2012): "Newborn girls survive better than newborn boys because they are less vulnerable to birth complications and infections and have fewer inherited abnormalities. Thus, the ratio of infant mortality among boys to infant mortality among girls is greater than one, provided both sexes have equal access to food and medical care." Knowledge of excess male infant mortality dates back at least to the 18th century, for example, Struyck (1740), Wargentin (1755), and Clarke (1786); for discussion, see Théré and Rohrbasser (2006). The female survival advantage in infancy is attributed to multiple factors: females have fewer congenital diseases owing to their redundant X chromosome, and they are also more resistant to infectious disease. For an authoritative review see Waldron (1998, 64–83).

¹⁷ Our approach to characterizing infant mortality will be of limited value for societies with missing women, where the "social vulnerability" of young girls outweighs the "biological vulnerability" of infant males (Thompson 2021, 467). In such societies, extremes of gender discrimination offset the biological survival advantage of female infants (e.g. D'Souza and Chen 1980; Das Gupta 1987), and there will be no simple relationship between the level of infant mortality and childhood sex ratios.

as infant mortality falls, the surviving child population moves towards males.¹⁸ Estimates of ‘missing women’ take into account the role of infant mortality on sex ratios (see Klasen 1994 in particular). More recent research has even used the expected relationship between infant mortality and childhood sex ratios in order to identify societies with ‘missing women’ (Beltrán Tapia and Raftakis 2021, figs. 2 & 3). However, the potential for childhood sex ratios to reveal infant mortality has remained unexploited, up until McDevitt-Irwin and Irwin (2024).

The relationship between infant mortality and childhood sex ratios can be modeled concisely with life-table concepts. Let q_0^i be the infant mortality rate of sex i , l_1^i the population surviving to age one, and B^i births:¹⁹

$$(1) \log\left(\frac{l_1^f}{l_1^m}\right) \approx \log\left(\frac{B^f}{B^m}\right) + \frac{q_0^m - q_0^f}{q_0} * q_0$$

The childhood sex ratio is determined by two additively separable terms: the sex ratio at birth ($\log\left(\frac{B^f}{B^m}\right)$), and the relative survival of males and females ($\frac{q_0^m - q_0^f}{q_0} * q_0$). The effect of the level of infant mortality (q_0) on the sex ratio of survivors is determined by the degree of excess male mortality ($\frac{q_0^m - q_0^f}{q_0}$). This value is typically in the range of 15-30% (Hill and Upchurch 1995; Drevenstedt et al. 2008). The level of infant mortality thus interacts with the degree of excess

¹⁸ For example, in the Coale-Demeny West model, moving from level 11 to level 22 infant mortality plummets from 159 to 27 (per 1000) and the sex ratio among survivors to age five (510) shifts 2.14 percentage points away from girls (Coale and Demeny 1983, 47,52).

¹⁹ See appendix for derivation; notation adapted from (Preston, Heuveline, and Guillot 2001). Observed sex ratios reflect an age interval (Lx) but we model populations at exact age (lx) for illustration of the general point. Moreover, because infant mortality is much greater and more male-skewed than later mortality (Hill and Upchurch 1995), the childhood sex ratio is mainly determined by infant mortality.

male mortality to pull childhood sex ratios away from the sex ratio at birth. The greater the female survival advantage, the more infant mortality skews the sex ratio among survivors.

Moreover, maternal-infant health will also be reflected in the sex ratio at birth, as a growing body of work demonstrates that insults to maternal well-being push the sex ratio at birth towards females (e.g., Almond and Edlund 2007; Fukuda et al. 1998; Catalano 2003).²⁰ Male frailty, in utero and in early infancy, means that poor maternal-infant health will be reflected both in terms of fewer males being born and fewer males surviving infancy. The direct effect of infant mortality on childhood sex ratios will, in most cases, be reinforced by a female-tilted sex ratio at birth, as infant mortality and maternal health are closely linked (e.g., Kramer 1987).²¹

Childhood sex ratio should thus reflect the infant mortality, and broader matrix of maternal-infant health within a population. The relationship between the sex ratio of surviving children and the level of infant mortality should be roughly linear. In a regression of childhood sex ratios on infant mortality, the intercept coefficient should reflect a healthy sex ratio at birth (5-6% male), and the slope coefficient the level of excess male mortality (15-30%).

Data and Methods

In order to characterize the empirical relationship between childhood sex ratios and infant mortality, we assemble data from historical vital statistics.²² Our data come from Europe, and the

²⁰ The apparent mechanism is maternal stress hormones, which increase the probability of miscarriages, which are disproportionately male (James and Grech 2017, 51). The sex ratio at birth has been used as an indicator for maternal health and fetal loss (Davis, Gottlieb, and Stampnitzky 1998; Grech and Masukume 2016; Shifotoka and Fogarty 2013; Sanders and Stoecker 2015; Valente 2015; Guimbeau, Menon, and Musacchio 2020).

²¹ Klasen (1994, 1064–66) noted this relationship between sex ratio at birth and infant mortality in the context of ‘missing women’. A similar pattern can be seen today in the US between the white, Black, and indigenous populations. Black women have the highest rates of infant mortality, followed by indigenous, and then whites (Ely and Driscoll 2021); sex ratios of birth follow the same pattern, with Black women giving birth to the most females (Mathews and Hamilton 2005).

²² When I refer to infant mortality ‘data’, I mean *direct estimates*. These are measures of infant mortality calculated from records of infant births and deaths. These are distinct from *indirect estimates* of infant mortality, which are typically derived from maternal recall of births and deaths using model life tables (see Hill 1991 for discussion). I

US and other settler societies, mostly from the mid-nineteenth century onward. I pair the under-5 sex ratio (children aged 0–4) with the rolling mean of infant mortality.²³ Each observation is a geographic unit in a particular census year.²⁴

In Figure 1, I plot under-5 sex ratios against infant mortality. Their empirical correspondence is striking. Populations with low infant mortality have childhood sex ratios approaching 5% male, a typical sex ratio at birth in a healthy population. As we move to the right on the graph, infant mortality increases and childhood populations become more female, with parity occurring around 200 deaths per 1000. Populations with very high rates of infant mortality (250+) almost exclusively have more females than males in the under-5 population.

treat these direct estimates as ‘facts’, and use them as ‘training’ or ‘validation’ data for characterizing an empirical relationship between infant mortality and childhood sex ratios.

²³ We use the under-5 sex ratio for several reasons: it is widely available in published census data, pooling the under-5 population increases the sample sizes (small samples are of concern for sex-ratios, see Visaria 1967 for discussion), and pooling across ages reduces the impact of sex-biased age heaping. We have one exception, Prussia from 1895 to 1910 (4 observations), where we use under-6 sex ratios, as the under-5 population is not available for our geographic units of interest.

²⁴ See the appendix below for the geographic and temporal scope of our data. Our data go no further than 1961; by then infant mortality in our sample populations was low enough that patterns of sex-ratio variation were largely independent of infant mortality, and ultrasound, which spread in the 1970s (Campbell 2013), was not yet a factor in sex-ratio patterns. Ultrasound and sex-selective abortion complicates the interpretation of childhood sex ratios.

Under-5 sex ratios by infant mortality

US 1850-1860 - Black - - white

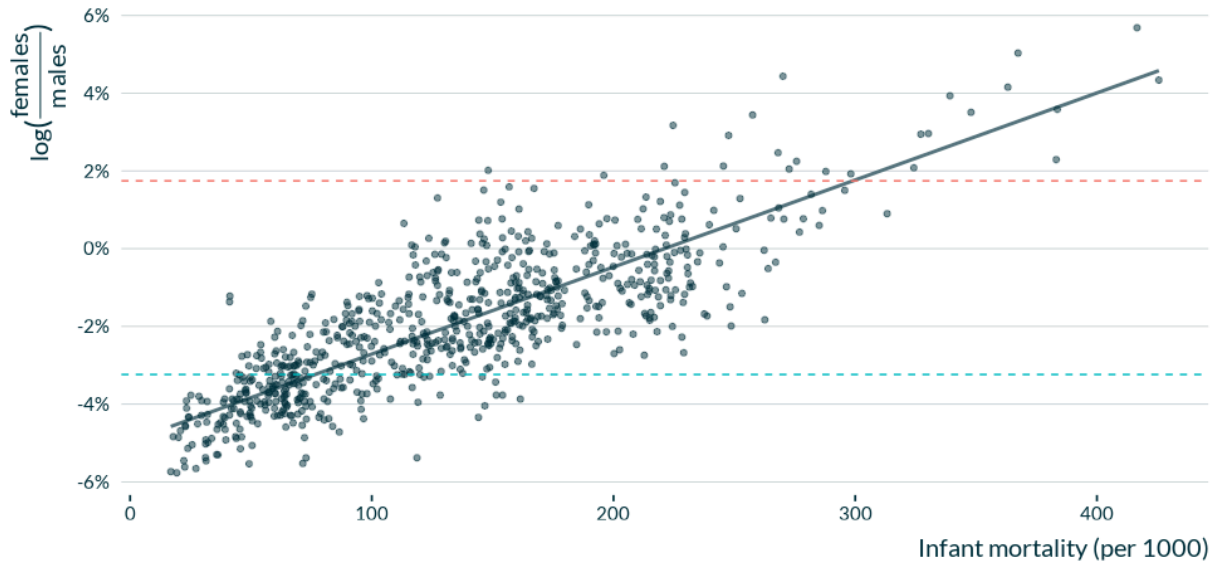


Figure 1: Under-5 Sex ratios by infant mortality, Europe and settler societies in the 19th and 20th centuries. Scatter plot and regression line, data from vital statistics and censuses, see McDevitt-Irwin and Irwin (2024) for discussion and a detailed list of sources. Dashed horizontal lines are US under-5 sex ratios by race, averaged across the 1850 and 1860 censuses; data from the IPUMS and published census volumes.

I use this observed relationship to infer broad patterns of infant mortality in the 19th-century US, where we lack vital statistics but have sex-ratio data from the census. Among many possible approaches, a simple regression of childhood sex ratios on infant mortality provides robust results. The least-squares fitted relationship is:²⁵

$$(2) \quad \hat{SR} = -4.95 + 0.224 \cdot q_0$$

This estimated relationship closely conforms to the theoretical relationship above. The regression intercept (-4.95%) corresponds to a healthy sex ratio at birth, with about 5% more boys than girls. The regression slope coefficient estimates the magnitude of the excess male mortality regression, and the 22.4% value falls well within the usual range for 19th-century European

²⁵ For clarity of exposition, we have scaled both infant mortality and sex ratios to be percentages (multiplied by 100).

populations (Hill and Upchurch 1995; Drevenstedt et al. 2008). I invert this regression in order to provide point estimates of infant mortality based on observed sex ratios:

$$(3) \quad \hat{IMR}_i = \frac{SR_i + 4.95}{.224} .$$

Point estimates fail to convey the uncertainty in inferring IMR from CSR. Therefore, following McDevitt-Irwin and Irwin (2024), I also estimate a bivariate Bayesian regression model, which characterizes IMR conditional on CSR. This model can account for several key features of the CSR-IMR relationship, including the non-negativity of IMR and the statistical noise inherent to sex ratios.²⁶ The full details of this Bayesian model is given in the appendix. Once estimated, the model produces Bayesian posterior predictive intervals, based on the estimated probability of IMR values conditional on observed sex ratios. These posterior predictive intervals take into account three distinct forms of uncertainty: sample uncertainty (standard errors on coefficients), model regression residuals, and the statistical noise inherent to finite-population sex ratios. Given an under-5 sex ratio and a population size, I report the 50% Bayesian posterior predictive interval as an estimate of IMR.

Below, I apply these methods to under-5 sex ratios from the 19th century US. These sex ratios come from the decennial US census. For the white population, I draw on published census volumes and the full-sample IPUMS microdata (Ruggles et al. 2021), averaging these two sources. For the Black population of 1850 and 1860, the data differ slightly, as the full-sample microdata are not available. Instead, I have published census counts of population by age-race-county, which I then aggregate to the nationwide level. From 1870 onward, I use the

²⁶ Sex ratios are the result of a binomial draw on some underlying probability, as discussed in Visaria (1967). Gelman and Weakliem (2009) highlight the problems of using sex-ratio data without accounting for this inherent noise.

average of the published census volume values and the full-sample microdata, for both the Black and white population.

Results

In Table 2, we present US under-5 sex ratios by race for 1850–1880, along with inferred infant mortality rates. US whites have relatively male-skewed sex ratios, with some 3% more boys than girls in the under-5 population. This compares favorably with contemporary European cases, which are generally in the range of parity to 2% male in the 19th century. Indeed, only the healthiest (Norway, New Zealand and Scotland) of contemporary populations have sex ratios comparable to US whites. The US Black population, on the other hand, had roughly 2% more *girls* than boys circa 1850–1860. Such female-skewed sex ratios are found in contemporary Europe, but only among the highest-mortality populations (like Bavaria and Austria). The 5 percentage-point difference between under-5 sex ratios among US Blacks and whites is clear evidence of stark difference in the demographic regime which each lived under.²⁷

²⁷ The racial disparity in childhood sex ratios is even larger if we restrict attention to the South, suggesting little role for geography.

Table 2: Under-5 sex ratios and implied infant mortality rates by race, US 1850–1880. Data from the Decennial Census. Point estimate of IMR is from equation (3); the interval estimate is the 50% Bayesian posterior predictive interval.

Year	Black			White		
	Under-5 sex ratio log(girls/boys)	IMR point estimate per 1000 births	IMR interval estimate per 1000 births	Under-5 sex ratio	IMR point estimate per 1000 births	IMR interval estimate per 1000 births
1850	1.92%	307	240–300	-3.47%	66	54–102
1860	2.34%	325	255–312	-3.18%	79	63–113
1870	-0.464%	200	155–212	-3.12%	82	66–115
1880	-0.418%	202	157–215	-3.37%	71	57–107

These differences in sex ratios translate into enormous differences in infant mortality. The white population likely had IMR under 100 deaths per 1000, making it one of the healthiest of contemporary populations (see McDevitt-Irwin and Irwin 2024 for more discussion of the white population). The Black population had IMR some 3-4 times higher, at least 250 deaths per 1000, and potentially greater than 300. Applying equation (3) to the sex-ratio disparity suggests an enormous 250-point racial disparity in IMR circa 1850–1860.

After emancipation, the racial gap in childhood sex ratios fell in half (comparing 1870-1880 to 1850-1860). White sex ratios were roughly constant across the period, at around 3.3% male (see Figure 2). Black sex ratios moved some 2.8 points toward males, from 2.2% female to 0.5% male. This change is powerful evidence of a substantial improvement in Black infant mortality, and of maternal and infant health more generally. Based on our regression results, Black infant mortality fell 100 points from nearly 300 deaths per 1000 to around 200.²⁸

²⁸ This value of 200 lines up well with Du Bois (1899, 150), who notes that Black IMR in Philadelphia was just over 170 deaths per 1000 births.

Despite this improvement, Black infant mortality circa 1870--1880 was still twice that of white – a relative gap that has persisted to today, even as infant mortality rates collapsed over the course of the 20th century.

Robustness

The substantial racial differences in under-5 sex ratios of the mid-19th-century US is powerful evidence of an enormous racial disparity in infant mortality (and maternal-infant health more generally). However, there are several robustness concerns to address. The most important is about data quality, both in terms of age heaping and under-enumeration, particularly for the enslaved population.

I next plot the under-10 sex ratios, in order to check for patterns of age heaping. The same pattern of racial differences is found in the sex ratios of the under-10 populations of 1850 to 1880. Looking at Figure 2, there is a remarkably close correspondence between the under-5 and under-10 sex ratios; this correspondence serves to stave off a potential concern that sex-biased age-heaping might be responsible for our results.

US Under-5 sex ratios by race

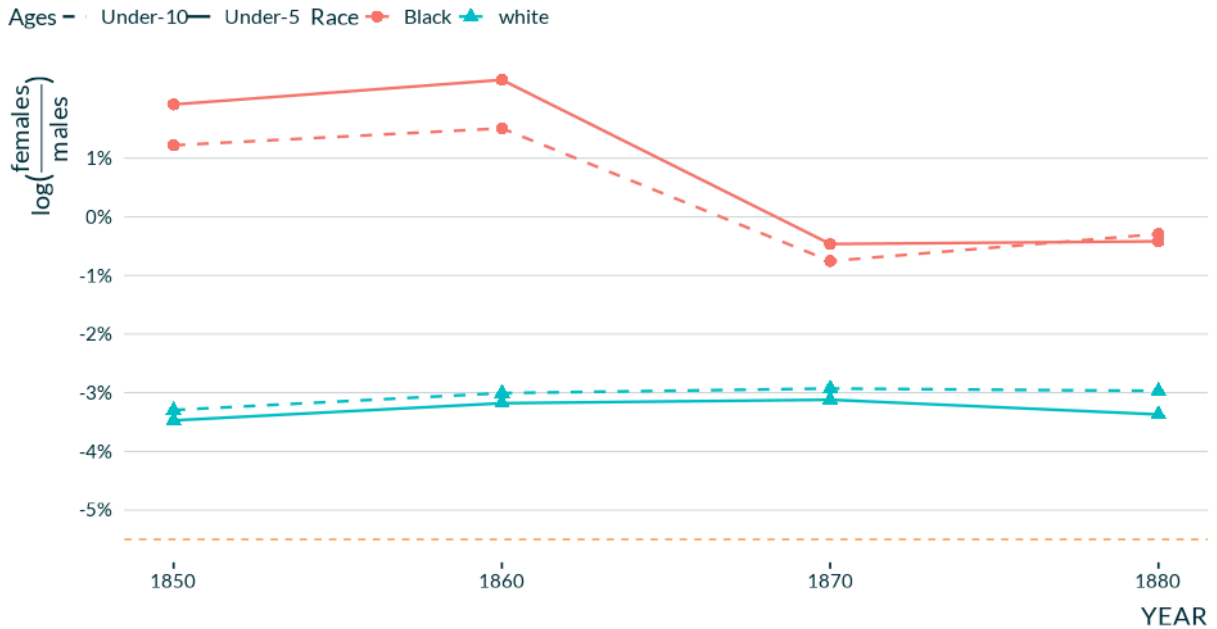


Figure 2: US childhood sex ratios by census benchmark year and race, 1850–1880. Ages under-5 and under-10. Data from the decennial US census, both the full-count PUMS and published census volumes. The yellow dashed line is for reference, and represents a healthy sex ratio at birth (5.5% more males than females).

Another potential robustness issue is the under-enumeration of infants and younger children, a concern often raised in the context of historical US censuses (Coale and Zelnik 1963; Hacker 2013). I address this problem by following cohorts across multiple censuses. In Table 2, I compare young adult (age 9-27) sex ratios in 1870 and 1900 by race.²⁹ There I see much the same patterns as in contemporary childhood sex ratios: the Black cohort born under slavery (age 9-27 in 1870) were much more female-skewed than the corresponding white population; and the Black population born after emancipation (age 9-27 in 1900) were less female-skewed than those

²⁹ I use 1900 because the 1890 Census records were lost in a fire, so there is no PUMS data available. Also, age reporting in the 1890 census was not consistent with practices in the other censuses. As discussed in 1900 and 1910 census reports, only the 1890 census asked for “age at nearest birthday” instead of “age at last birthday”, which was used from 1850 to 1880, and from 1900 forward (page xlviii of Twelfth Census (1900), Census Reports Volume II, Population Part II, Washington: GPO, 1902). Therefore we exclude 1890 from our analysis.

born under slavery.³⁰ These two findings support our broad points: (1) that the US Black population in the 19th century had much more female-skewed sex ratios than the white population; and (2) that childhood sex ratios among the Black population become less female-skewed (more male-skewed) after the abolition of slavery.

Table 3: US sex ratios for ages 9-27. Data from the 1870 and 1900 US Census full-count PUMS.

Year	Black		White	
	Males per 1000 females	Log-terms F/M	Males per 1000 females	Log-terms F/M
1870	926 per 1000	7.67%	983 per 1000	1.74%
1900	954 per 1000	4.67%	1006 per 1000	-0.60%

Discussion

Childhood sex ratios paint a stark picture of the 19th-century US. Black infant mortality under slavery was roughly 4 times that of whites, with an absolute gap upwards of 200 deaths per 1000. Nearly 1-in-3 Black infants died in their first year of life, compared to fewer than 1-in-10 white infants. This is a much larger racial disparity in infant mortality than found in previous research. Indirect estimates of infant mortality pointed towards a clear racial disparity under slavery (Farley 1965; Eblen 1974), but all of these accounts agree on the basic characterization of generally high infant mortality in the 19th-century US, albeit somewhat worse for Blacks than whites.

After emancipation, Black infant mortality improved substantially, pointing to the institution of slavery as responsible for the uniquely large health disparity described above.

³⁰ The same basic pattern is observed with other relevant age-groupings.

Between 1860 and 1870 Black childhood sex ratios moved 2.8 percentage points towards males, suggesting a decrease in infant mortality of over 100 points: from above 300 to around 200 deaths per 1000. This improvement contrasts with previous views, which argued that Black health remained the same—or even deteriorated—after emancipation (Farley 1965; Fogel and Engerman 1974, 1:261; Eblen 1974; Meeker 1976). Left to their own devices, Black families were able to achieve much lower infant mortality after emancipation than they had experienced under slavery.³¹

The everyday violence of enslavement likely played a major role in determining Black reproductive health under slavery. Recent scholarship in social history points towards experiences of extreme stress for the enslaved, especially women (e.g., Hartman 1997), under the threat of sexual violence (Jones 2003). Medical research has demonstrated the important role of stress on maternal-infant health (Oths, Dunn, and Palmer 2001; Loomans et al. 2013), particularly for the Black population (Collins and David 2009). High levels of chronic stress among mothers would have contributed to high rates of fetal loss (Qu et al. 2017) and infant mortality (e.g., through preterm birth, Shaikh et al. 2013). Thus the violence, and threat of violence, experienced by enslaved women might alone account for a large part of the enormous racial disparity in maternal-infant health under slavery, in addition to previously noted factors like overwork (Campbell 1984) and malnutrition (Steckel 1986).

One concern in this approach is that sex ratios at birth have persistent racial patterns in the US, with the Black women giving birth to relatively more females than white women do. This observation, that in the US the Black and white population tend to have different sex ratios at birth, dates back to at least De Jastrzebski (1919), and the pattern persists today. In the 21st

³¹ The improvement in Black health with emancipation is particularly striking in light of the large decline in per-capita income witnessed in the US South over the same period (Easterlin 1961).

century US, roughly 5% more males than females (1055 M per 1000 F) are born to white women, but that value was only 3.5% (1036 M per 1000 F) for Black women (Mathews and Hamilton 2005, table 4). Although some have suggested a genetic basis for this disparity, it is most likely due to differences in maternal health and well-being, such as chronic stress, as recently argued by Grech (2023).³² Thus, racial differences in sex ratios at birth reflect broader health disparities, and not some inheritable biological factor.³³ Moreover, childhood sex ratios broadly line up with known patterns of maternal-infant health for the Black population in the 20th century, as childhood sex ratios move towards boys after 1960.

This attention to the sex ratio at birth illustrates that differences in childhood sex ratios may not only reflect infant mortality, but also maternal health. Because I only observe childhood sex ratios, and not sex ratios at birth, I cannot disentangle the role of fetal loss as opposed to infant death. Therefore, the results should be interpreted as speaking to a broad matrix of maternal-infant health, rather than only infant mortality.

Conclusion

As discussed above, sex ratios reflect infant mortality because of the biological survival advantage of infant females. Drawing on McDevitt-Irwin and Irwin (2024), I use vital statistics data from contemporary populations in order to quantify the empirical relationship between CSR and IMR. I then use this relationship to estimate infant mortality from under-5 sex ratios for the US 1850–1880, drawing on the decennial census. Circa 1850–1860, under slavery, infant mortality among US Blacks was extremely high, likely around 300 deaths per 1000. During the

³² This explanation is strengthened by the fact that Native Americans – another group facing systemic racism – have a similarly feminized sex ratio at birth (Mathews and Hamilton 2005, table 4).

³³ Moreover, race, as a social construct, does not map to any simple genetic grouping, making any claims of ‘genetic’ origins of racial disparities inherently flawed. This is particularly true of the Black population of the US, who share substantial genetic ancestry with the white population (Bryc et al. 2015).

same period, IMR among US whites was low by historical standards, likely under 100. Circa 1870–1880, after emancipation, infant mortality improved substantially for the Black population, dropping some 100 points to around 200 deaths per 1000.

As discussed above, the lack of demographic indicators on the health of US Blacks under slavery has left open the notion that Blacks and whites had broadly similar demographic regimes under slavery. Childhood sex ratios baldly contradict this view of US slavery. US Blacks and whites may have shared a similar rate of natural increase, but this came from starkly different patterns of mortality and fertility. US Blacks and whites resemble populations on opposite sides of demographic transition: one experiencing an extremely ‘high-pressure demographic regime’ while the other a relatively ‘low-pressure’ one. This difference was in no way inevitable; it was due to the extreme social inequalities of slavery.

These results focus attention on the role of social structures in determining population health. Structural racism is widely acknowledged as a driver of today’s racial health disparities (Bailey et al. 2017). The antebellum US offers an even more extreme example of social structure driving mortality. Slavery in the US was responsible for a staggering number of infant deaths, with Black mothers losing infants at 3-4 times the rate of the free white population. After emancipation, Black infant mortality declined substantially, but remained about twice that of whites. One can only speculate what difference there might have been had the full promise of reconstruction—‘40 acres and a mule’—been fulfilled;³⁴ but surely Black infant mortality would have been much lower (much closer to that of contemporary rural whites) in such a case.

³⁴ The US government had suggested breaking up large plantations for Black freedmen to take as property, but never enacted this policy. See Fleming (1906) and Du Bois (1910; 1935) for early academic discussion of ‘forty acres and mule’. More recently, Darity (2008, 661) speculates that “had the promise of 40 acres been fulfilled, one can readily imagine a completely different U.S. history unfolding over the course of the subsequent century, a history in which race did not intertwine with dense inequalities.”

The US of the 1850s was a place of extremes. US whites had some of the lowest infant mortality rates in the world. US Blacks had some of the highest. Enormous progress would be seen over the following 150 years, with infant mortality plummeting for all groups in the US. However, the 20th-century decline in US infant mortality was smaller (in absolute terms) than the difference between Blacks and whites in 1850. In terms of infant mortality, US whites in 1850 were closer to today's populations than to contemporary Blacks. The decline in Black infant mortality after emancipation was larger than the decline in white infant mortality from 1850 to today. Placed in a historical perspective, the institution of slavery had a larger effect on life and death than did the following century of social and economic progress.

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Appendix

Bayesian Model of IMR Conditional on CSR

Here I reproduce the Bayesian model of IMR conditional on CSR first developed in McDevitt-Irwin and Irwin (2024). In that paper, we model the underlying data-generating process as a linear relationship between IMR and CSR, with errors distributed normally $(0, \sigma^2)$. Furthermore, we model the implicit measurement error in observed sex ratios, which are probabilistic draws of an underlying binomial distribution. Thus, we have $x_i = x_i^* + \varphi_i$ where $\varphi_i \sim N(0, g(n_i))$, where x_i is observed sex ratios, x_i^* is the sex ratio of the underlying binomial draw, φ_i is classical measurement error, and $g(n_i)$ is the variance of the log sex ratio as a function of sample size. We also place a non-negativity bound on infant mortality. The full model then becomes:

$$y_i = \alpha + \beta \cdot x_i + \epsilon_i, \text{ where } y_i \geq 0,$$

$$x_i \sim N(x_i^*, g(n_i)), \text{ and } \epsilon_i \sim N(0, \sigma^2)$$

We estimate the model using Markov Chain Monte Carlo, 4 chains with 10,000 iterations each, using the *brms* package in *R* (which calls the C++ program *Stan*). We use ‘weakly informative priors’ — i.e, prior distributions which are specific enough to regularize the estimation problem but vague enough to allow the data to dominate the resulting posterior distributions — following a growing consensus in applied Bayesian statistics (Gelman et al. 2008; Gelman, Simpson and Betancourt 2017; Lemoine 2019; Gabry et al. 2019). We follow the default priors of *Stan*, described in Gelman, Hill and Vehtari (2021, p. 124), where the variance of the priors is scaled

by the variance of the data. In the appendix, we plot the prior vs. posterior distributions, showing that our priors are sufficiently diffuse to have minimal effect on our results, as well as posterior predictive checks (Gelman, Meng and Stern 1996), which show that our model is able to roughly reproduce the observed distribution of infant mortality. As noted above, we follow the default recommended priors in *Stan* (see [here](#) for discussion from the developers of *Stan*), and scale priors by the variance of the observed variables (Gelman, Hill and Vehtari 2021, p. 124). The full priors for our Bayesian model are:

Table A2: Priors for Bayesian Model

Parameter	Family	Mean	Variance
Intercept	Normal	0	0.17
Slope	Normal	0	0.096
Sigma (Residual variance)	Exponential	14	NA
Standard Deviation (measurement error)	Exponential	1	NA
Mean (measurement error)	Normal	0	1

Here we plot prior and posterior predictive checks. In Figure A1, we plot the prior and posterior distributions of our parameters of interest. We see that the prior distributions of our parameters are an order of magnitude more diffuse than the posterior. In effect, we can see that the priors are sufficiently ‘weak’ that they are not substantially influencing the posterior distributions. Instead, they only regularize the estimation problem, as is desirable from ‘weakly informative priors’ (Gabry et al. 2019).

In Figure A4, we plot posterior predictive checks, that is the predicted IMR (\hat{y}) values from various draws of our posterior distributions against the actual observed IMR values. We see that the model is roughly able to reproduce the underlying distribution of infant mortality.

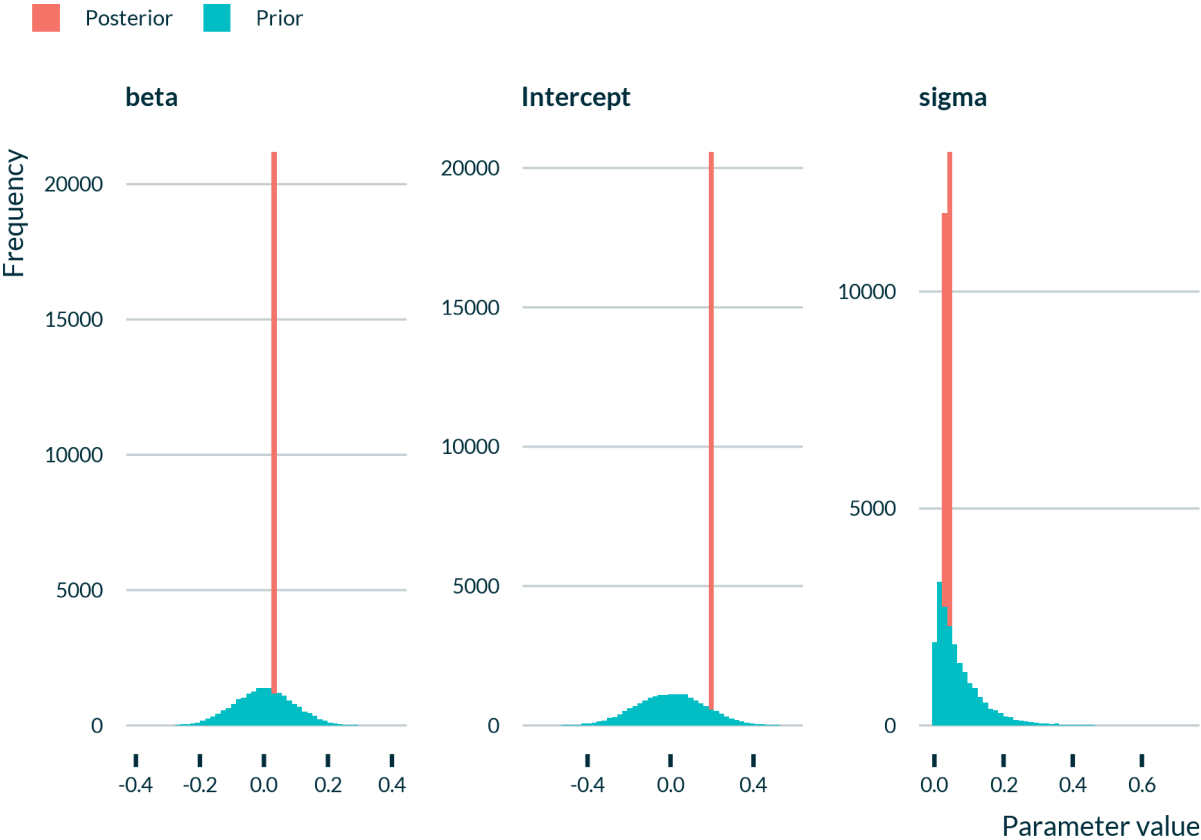


Figure A1: Prior vs. Posterior Distributions. The blue histograms are for the prior distributions for each parameter; the red are for the posterior. Beta is the slope parameter, and sigma is the residual variance parameter.

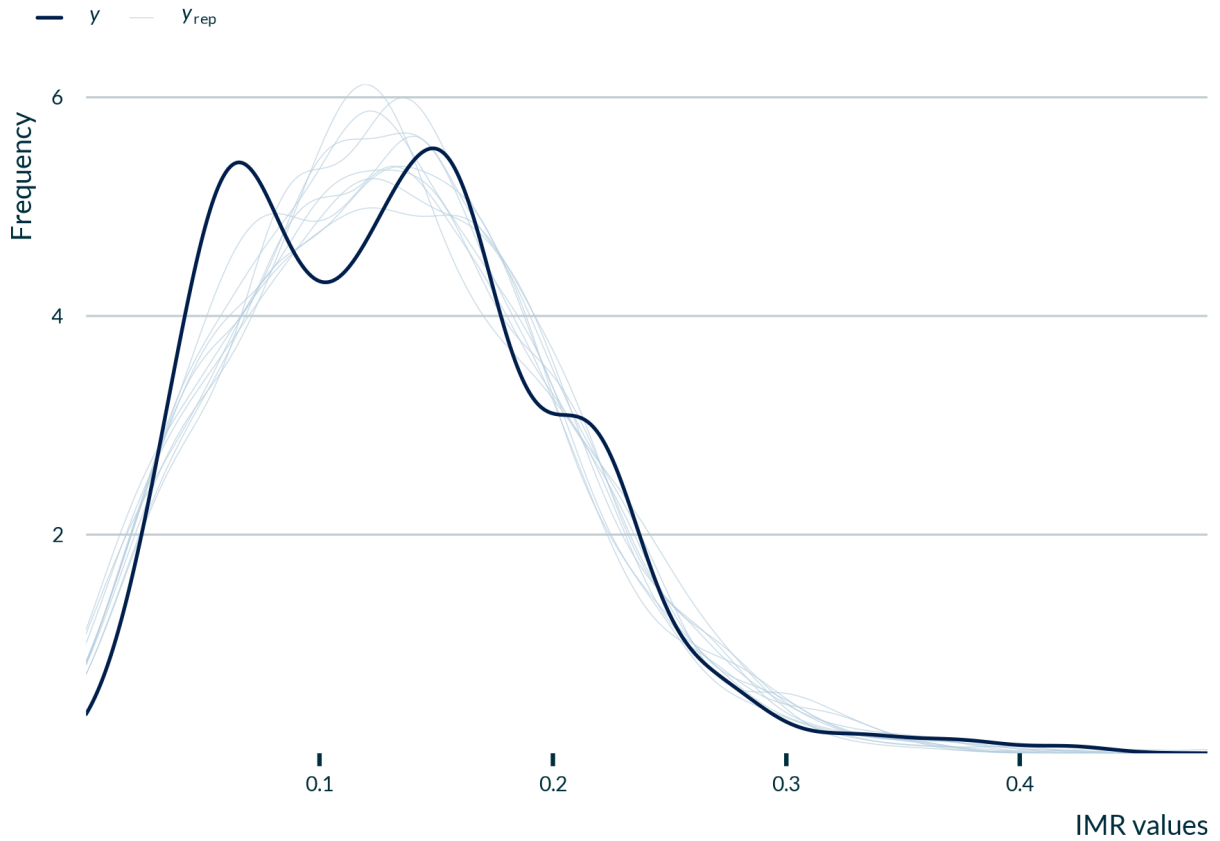


Figure A2: Predictive posterior check for Bayesian model. The black line is the distribution of the observed IMR data from Figure 3. The shaded blue lines are the distributions of predicted values from 10 draws of our posterior distributions.