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MEDICAL INNOVATION AND RACIAL HEALTH DISPARITIES: EVIDENCE FROM A BREAKTHROUGH TREATMENT

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ABSTRACT

Whether medical innovation exacerbates or reduces racial health disparities remains an open question. We study surfactant replacement therapy (SRT), a life-saving intervention for premature infants with respiratory disorders. Before its approval by the FDA in 1989, premature Black infants were much less likely than their White counterparts to die from respiratory-related causes. Within a few years of FDA approval, the Black-White gap in respiratory-related neonatal mortality had essentially disappeared. Using 1980-2000 vital statistics data and non-respiratory-related mortality as a counterfactual outcome, we find that both Blacks and Whites benefited from the introduction of SRT, but White neonates experienced larger and more immediate reductions in mortality. We estimate that, by 1993, SRT had reduced respiratory-related mortality among White neonates by 46 percent, compared to 30 percent for Black neonates. These results are not explained by differences in health care access, as proxied by socioeconomic status or distance to the nearest neonatal intensive care unit. We conclude that racial differences in fetal pulmonary maturation, rather than barriers to access, likely drove the uneven impact of SRT on neonatal mortality.

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1. INTRODUCTION

Medical innovation is widely regarded as a fundamental driver of infant health and mortality (Cutler and McClellan 2001; Barfield et al. 2013; Taha et al. 2023). Since the turn of the 20th century, the infant mortality rate in the United States has declined by nearly 95 percent (Centers for Disease Control and Prevention 1999; Kochanek et al. 2023). Over this same period, there has been remarkable technological progress in neonatal and pediatric medicine (Vidyasagar 2011). From the first use of incubators in the early 1900s to the recent development of AI-powered diagnostics (Baker 1991; Siolos et al. 2025), medical innovation has undoubtedly contributed to these gains in infant survival.

By contrast, far less is known about how medical innovation affects infant health disparities, especially along racial lines. Historically, across a range of indicators, the health of Black infants has lagged that of their White counterparts (Bediako et al. 2015; Singh and Yu 2019; Kennedy-Moulton et al. forthcoming). In the absence of barriers to care, advances in medical technology have the potential to narrow these gaps (Levine et al. 2011). If, however, there are race-based differences in access and adoption, the introduction of new technologies can exacerbate health disparities (Goldman and Lakdawalla 2005; Glied and Lleras-Muney 2008). The "fundamental causes" hypothesis (Link and Phelan 1995; Link et al. 1998) posits that, as medical knowledge and technology advance, health disparities can widen because socially disadvantaged groups do not have the material resources and information to access these advances.

In this study, we use data from the National Vital Statistics System (NVSS) to estimate the effects of surfactant replacement therapy (SRT) on Black versus White neonatal mortality in the United States. Approved by the Food and Drug Administration (FDA) in 1989, SRT is a treatment for respiratory-related issues that frequently occur among premature and very low birthweight (VLBW) infants such as respiratory distress syndrome (RDS). Prior to its approval by the FDA,

respiratory-related problems accounted for approximately one fourth of all neonatal deaths (i.e., deaths occurring during the first 28 completed days of life).[1] Although medical experts have labeled SRT as a "major breakthrough" and the "single most important advance in neonatal medicine" (Cummings 2007, para. 2; Hallman and Herting 2023, p. 4), only a few descriptive studies have explored its association with infant mortality by race (Hamvas et al. 1996; Ranganathan et al. 2000; Frisbie et al. 2004; Cutler et al. 2012).

Figure 1, which is based on data from the NVSS Multiple Cause-of-Death Mortality Files, shows the evolution of neonatal mortality from respiratory-related causes by race for 1980–2000.[2] Through most of the 1980s, the respiratory-related mortality rate among Black neonates was 0.3 to 0.4 log points lower than the rate among White neonates. After 1989, when SRT became widely available outside of clinical trials, the Black-White gap in respiratory-related neonatal mortality narrowed markedly. By 1993, it had almost vanished; the respiratory-related mortality rate among Black neonates was within 0.05 log points of the White rate.

Can the introduction of SRT account for the dramatic narrowing of the Black-White gap in respiratory-related neonatal mortality? To answer this question, we use neonatal mortality from non-respiratory causes as a counterfactual outcome to account for the effects of other medical innovations and practices that likely contributed to the steady decline in neonatal mortality during the period under study, including better ultrasound imaging (Campbell 2013), advances in prenatal care (Peahl and Howell 2021; Briceño-Pérez et al. 2019), and increased receipt of prenatal care

[1] In the United States, more than 60 percent of infant mortality occurs during the neonatal period (Ely and Driscoll 2025). See Lee et al. (1990) for U.S. neonatal mortality rates by cause in 1985 and 1988.

[2] The neonatal respiratory-related mortality rate is calculated per 100,000 VLBW births. VLBW infants, who weigh less than 1,500 grams at birth, made up over 70 percent of neonatal deaths during the period under study. Following Hamvas et al. (1996), we defined respiratory-related causes of death as respiratory distress syndrome, pulmonary interstitial emphysema, pulmonary hemorrhage, primary atelectasis, chronic respiratory disease arising in the perinatal period, and other respiratory disorders.

(Lewis et al. 1996). Event-study estimates provide evidence that both Blacks and Whites benefited from the introduction of SRT, but White neonates experienced larger and more immediate reductions in respiratory-related mortality. Within a few months of FDA approval, we estimate that SRT had led to a 27 percent reduction in respiratory-related mortality among White neonates and a (statistically insignificant) 8 percent reduction in respiratory-related mortality among Black neonates. Four years after FDA approval, we estimate that respiratory-related mortality among White neonates had fallen by as much as 46 percent because of SRT; respiratory-related mortality among their Black counterparts had fallen by as much as 30 percent; and the Black-White respiratory-related neonatal mortality gap had narrowed by 92 percent.

There is evidence that Black fetuses mature at lower gestation ages and birth weights than do White fetuses (Wilcox and Russell 1990; Berman et al. 1996; Patel et al. 2004; Loftin et al. 2012), and medical experts have argued that, before the introduction of SRT, premature Black infants were at a lower risk of dying from respiratory-related causes than their White counterparts due to more rapid lung maturation (Hulsey et al. 1993; Hamvas et al. 1996). This basic difference in the physiological need for treatment could explain why White neonates experienced a relatively steeper reduction in respiratory-related mortality with the introduction of SRT (Hamvas et al. 1996; Frisbie et al. 2004), although barriers to access could also explain why the Black-White mortality gap essentially disappeared.[3] In an effort to distinguish between these two competing explanations, we use data from the NVSS Linked Birth and Infant Death Files to estimate the effects of SRT on respiratory-

[3] It is also possible that Black VLBW infants respond less favorably to SRT than White VLBW infants (Ranganathan et al. 2000), although Hamvas et al. (1996) found no racial differences in neonatal mortality conditional on receiving SRT. Because racial differences in fetal pulmonary maturation could reflect environmental and social factors rather than fixed biological determinants (Newberry et al. 2025), we remain agnostic as to why Black fetuses exhibit faster pulmonary maturation than their White counterparts. As noted by David and Collins (2007), genetic explanations of racial disparities in birth outcomes are difficult to reconcile with the fact that foreign-born Black mothers have, on average, better birth outcomes than U.S.-born Black mothers. This advantage erodes across generations as birth outcomes among the descendants of foreign-born Black mothers deteriorate (Collins et al. 2002).

related neonatal mortality by mother's educational attainment and marital status, two factors that, in theory, could determine access to SRT. The results of this exercise are not consistent with a barriers-to-access explanation: there is little evidence that the estimated effects of SRT depend on mother's educational attainment or marital status for either Black or White infants.

When first introduced, SRT was only available in neonatal intensive care units (NICUs).[4] To assess whether proximity to the nearest NICU explains why White neonates experienced a steeper reduction in respiratory-related mortality than their Black counterparts, we obtained information from the American Hospital Association on the location of NICUs. Merging this information with restricted-use NVSS Linked Birth and Infant Death Files, we find little evidence that distance to a NICU mattered. For Black neonates whose mother lived within 25 miles of a NICU, SRT is associated with an 8 percent reduction in respiratory-related mortality; for those living farther away, the estimate is a statistically insignificant 10 percent reduction. While not statistically distinguishable at conventional levels, these estimates suggest that a counterfactual movement of Black mothers nearer to NICUs would, if anything, widen the Black-White gap in respiratory-related neonatal mortality. More broadly, we interpret our NICU proximity estimates as providing additional evidence that racial differences in fetal pulmonary maturation, rather than barriers to access, likely drove the uneven impact of SRT on neonatal mortality. These estimates reinforce the point, made by Weiss et al. (2018) and other researchers studying health equity (e.g., Press et al. 2021, Haimi 2023, and Ndugga et al. 2024), that providing access to the latest medical technology does not guarantee the elimination of racial health disparities.

We view our research as making a general contribution to the surprisingly sparse quasi-experimental literature on how medical innovation affects racial health disparities. The emergence

[4] When first introduced, SRT was administered using procedures (e.g., endotracheal intubation and mechanical ventilation) that were not typically available outside of NICUs.

of new gene therapies for diseases such as sickle cell anemia and breakthrough medicines such as Leqembi (for Alzheimer's disease) and Ozempic (for obesity) has raised concerns about access and equity (Ndugga et al. 2024), yet little credible evidence is available to assess whether these concerns are warranted.[5] Beyond contributing to the quasi-experimental literature on medical innovation and health equity, we provide new evidence on how the FDA approval of SRT affected newborn health. High-profile medical studies have shown that the introduction of SRT coincided with a reduction in neonatal mortality among VLBW infants (Horbar et al. 1993; Schwartz et al. 1994; Hamvas et al. 1996; Lee et al. 1999). These studies, however, did not account for the influence of other difficult-to-observe factors, including advances in neonatal ventilation technologies.

Between 1989 and 1994, the use of high-frequency ventilation rose sharply among extremely low birth weight (ELBW) infants (i.e., infants born weighing less than 1,000 grams), while its use among VLBW infants weighing at least 1,000 grams remained at negligibly low levels (Richardson et al. 1998). In Section 4, we estimate the effects of SRT on respiratory-related neonatal mortality by birthweight category. For both Black and White neonates, we find that FDA approval of SRT is associated with substantially smaller reductions in respiratory-related mortality below the ELBW cutoff than above it, suggesting that the introduction of high-frequency ventilation technologies is not driving our estimates.

In the developed world today, SRT is firmly established in the armamentarium of neonatal intensive care, while in many low-resourced countries it remains costly and largely inaccessible to the average patient (Vidyasagar et al. 2011). Despite its inclusion on the World Health Organization's "Model List of Essential Medicines," surfactant was available in less than 40 percent of the best-equipped African hospitals as of 2020 (Tooke et al. 2021). Amid severe cuts to global health

[5] The quasi-experimental literature on medical technology and racial health disparities, including Jayachandran et al. (2010) and Miller and Tucker (2011), is described in Section 2.4.

funding (Cavalcanti et al. 2025; CGTN 2025; Mahase 2025), access to surfactant is likely to become even more constrained in the years ahead, highlighting the urgency of identifying which interventions in neonatal care should be prioritized. Our estimates suggest that the widespread adoption of SRT can yield profound health benefits for infants, but these gains may exacerbate disparities—a trade-off especially salient for developing countries grappling with ethnic, racial, and ethno-racial gaps in child survival (Brockerhoff and Hewett 2000; Burgard and Treiman 2006; Schellenberg and Berhanu 2020; Rebouças et al. 2024).

The remainder of the paper is organized as follows. In Section 2, we provide background information, including a brief history of SRT and a description of previous quasi-experimental studies on medical innovation and racial health disparities. We also show that White neonatal mortality among VLBW infants declined faster than Black neonatal mortality during the period under study and discuss the potential drivers of this phenomenon. In Section 3, we use data from the NVSS Multiple Cause-of-Death Mortality Files for the years 1980-2000 to estimate the effect of SRT on respiratory-related neonatal mortality by race. In Section 4, we use data from the NVSS Linked Birth and Infant Death Files for the years 1983-1991 and 1995-2000 to address the potentially confounding influence of new ventilation technologies and to estimate the effects of SRT by maternal characteristics. In Section 5, we explore whether NICU access can explain why White neonates experienced larger and more immediate reductions in mortality after the introduction of SRT than their Black counterparts. Section 6 concludes.

2. BACKGROUND

2.1. Surfactant replacement therapy

Surfactant is a lipid–protein complex that reduces surface tension in the lungs and is essential for normal respiratory function (Serrano and Pérez-Gil 2006). Production of pulmonary

surfactant usually begins at 26 weeks of gestation and continues through the 35th week (Khawar and Marwaha 2023), leaving premature infants vulnerable to severe respiratory disorders. By lowering surface tension, surfactant stabilizes the alveoli (i.e., the lung sacs), preventing them from collapsing during exhalation and enabling normal breathing (Mathew 2011). Avery and Mead (1959) first linked surfactant deficiency to neonatal RDS, and Fujiwara et al. (1980) showed that administering a modified bovine surfactant to preterm infants with RDS markedly improved their respiratory function.[6] Randomized controlled trials (RCTs) have since confirmed that SRT substantially reduces neonatal mortality (Soll and Özek 1997; Polin et al. 2014).

SRT first became available outside of clinical trials in July 1989, when the FDA approved Investigational New Drug (IND) protocols for Exosurf, a synthetic surfactant (Horbar et al. 1993).[7] Under these protocols, hospitals could apply to the FDA for permission to treat critically ill newborns with Exosurf (Del Giudice 1989; Colimore 1989).[8] A spokesperson for Burroughs Wellcome, the producer of Exosurf, estimated that, by October 1989, 200 hospitals across the country were using its product (Nevada 1989). The next month, Burroughs Wellcome announced that 280 U.S. hospitals were using Exosurf (Wofford 1989).[9]

[6] Avery and Mead (1959, p. 518) demonstrated the absence of a "surface-active material" in the lungs of premature infants with RDS, a substance later identified by researchers as pulmonary surfactant. Appendix Figure A2 illustrates normal versus collapsed alveoli. Surfactant was traditionally administered via an endotracheal tube (Polin et al. 2014). While this remains the most widely used technique, other less-invasive methods of administration have been developed (Ng and Shah 2021).

[7] A treatment IND is submitted for experimental drugs for immediately life-threatening conditions while the final clinical work is being conducted and the FDA review is underway (U.S. Food and Drug Administration 2024).

[8] The 50 hospitals that participated in the Exosurf clinical trials were immediately eligible to administer Exosurf (Del Giudice 1989; McInnis 1989). Clinical trials of surfactants, including Exosurf, began in the mid-1980s (Butler 1989; Hallman and Herting 2023). The Exosurf clinical trials involved approximately 2,000 premature newborns in the United States and Canada (Butler 1989; Corbet et al. 1991; Long et al. 1991).

[9] Based on the American Hospital Association data, described below, we estimate that in 1990 there were 936 NICUs operating in the United States.

Exosurf was approved for commercial release in August 1990 (Horbar et al. 1993) and Survanta, a natural surfactant derived from bovine lung, became commercially available one year later (Horbar et al. 1993).[10] By 1991, nearly every NICU in the United States was administering SRT to premature infants with respiratory-related disorders (Horbar and Lucey 1995).[11] As RCTs provided evidence that natural surfactant preparations such as Survanta were more effective than synthetic surfactants, the use of Exosurf was gradually phased out (Halliday 1995; Hudak et al. 1997; Moya et al. 2023; Raj et al. 2024), but SRT remains the standard of care for preterm infants suffering from respiratory-related disorders.

2.2. Surfactant replacement therapy and the Black VLBW survival advantage

Figure 2 shows the evolution of all-cause neonatal mortality by race for VLBW infants. It is constructed using information from the NVSS Linked Birth and Infant Death Files for the years 1985-1991 and 1995-2000. The linked files include every infant death that can be matched to a birth certificate, allowing us to focus on infants weighing less than 1,500 grams at birth. Due to resource constraints, no linked files were produced by the NVSS for the years 1992-1994 (Weed 1995).

Before SRT became widely available, Black VLBW infants had a distinct survival advantage as compared to their White VLBW counterparts. Within two years of the FDA authorizing the use of Exosurf for critically ill newborns, the Black survival advantage had entirely disappeared. By 1995, the neonatal mortality rate among White VLBW infants had fallen to 22,591 per 100,000 VLBW births, nearly 6 percent less than the Black neonatal mortality rate. By 2000, the neonatal

[10] Survanta received FDA approval under an IND in October 1989 and became commercially available in July 1991 (Horbar et al. 1993).

[11] Investigators of the Vermont-Oxford Trials Network Database Project (1993) and Horbar et al. (2002) examined the use of SRT at 36 NICUs participating in the Vermont Oxford Network Database. In 1990, 49 percent of VLBW newborns at these NICUs received SRT. One year later, 53 percent of VLBW newborns at these NICUs received SRT. By 1999, 63 percent of VLBW newborns at 325 NICUs participating in the Vermont Oxford Network database were receiving SRT (Horbar et al., 2002).

mortality rate among White VLBW infants had fallen to 20,365 per 100,000 VLBW births, nearly 10 percent less than the Black rate.

Only a few descriptive studies have explored whether SRT could have contributed to the reversal of the Black VLBW survival advantage. Hamvas et al. (1996) analyzed the clinical records of VLBW infants at four NICUs in St. Louis. The neonatal mortality rate among White infants at these NICUs fell by 41 percent between 1987 and 1992. During the same period, there was a small (and statistically insignificant) increase in the neonatal mortality rate among Black infants (Hamvas et al. 1996, p. 1637). Frisbie et al. (2004) used the NVSS Linked Birth and Infant Death Files for the years 1989-1990 and 1995-1998 to estimate the association between race and RDS mortality. In 1989-1990, Black infants were less likely to die from RDS than their White counterparts, but by 1995-1998 they had lost this survival advantage.[12] Finally, Ranganathan et al. (2000) and Cutler et al. (2012) compared pre- versus post-SRT RDS mortality by race among VLBW infants. They showed that VLBW White infants experienced steeper declines in RDS mortality than Black VLBW infants after 1990, when SRT became widely available.[13]

2.3. Other efforts to improve neonatal respiratory care: antenatal steroids and ventilation

Medical studies such as Hamvas et al. (1996) and Ranganathan et al. (2000) demonstrate that VLBW White infants experienced steeper declines in RDS mortality after 1989 than Black VLBW infants. Aside from SRT, two other major technological advances in perinatal care could, in theory,

[12] In 1989-1990, Black infants had 19 percent lower odds of dying from RDS than White infants. By 1995-1998, Black infants had 10 percent higher odds of RDS mortality, leading Frisbie et al. (2004) to conclude that SRT had exacerbated racial health disparities.

[13] Using NVSS data for the years 1988 and 1991, Ranganathan et al. (2000) found that the odds of death caused by RDS decreased by 34 percent among VLBW White infants. Among VLBW Black infants, it decreased by 16 percent. Ranganathan et al. (2000) were careful to note that the observed reductions in RDS mortality, although suggestive, "cannot be interpreted as strictly reflecting the beneficial effects of surfactant therapy alone" (Ranganathan et al. 2000, p. 458). Plotting RDS mortality by race for VLBW infants for the periods 1983-1991 and 1995-1998, Cutler et al. (2012, p. 466) observed that "there were greater survival improvements for Whites than blacks" after the introduction of SRT.

have contributed to the steeper post-SRT mortality reductions among White infants: the widespread use of antenatal corticosteroids and the introduction of high-frequency ventilation.

Antenatal corticosteroids accelerate fetal lung development and reduce the risk of RDS (Liggins and Howie 1972; Crowley et al. 1990; McGoldrick et al. 2020). Despite these benefits, physicians were slow to embrace antenatal steroid therapy (National Institutes of Health Consensus Statement 1995). In the late 1980s and early 1990s, only 12-20 percent of women who delivered preterm infants were being treated with antenatal steroids (Meadow et al. 2003). Then, in 1994, a National Institutes of Health (NIH) consensus panel recommended antenatal steroid therapy for pregnant women at risk of preterm delivery. Within a year of this recommendation being issued, more than 50 percent of VLBW infants born in the United States were being exposed to antenatal steroids and weekly steroid courses for pregnant women at risk of premature delivery had become routine (Planer et al. 1996; Meadow et al. 2003; Mathew 2011).

Race-based differences in exposure to antenatal steroids could explain steeper post-SRT mortality reductions among White infants. Indeed, Hamvas et al. (1996) and Carlo et al. (2011) provide evidence that premature Black infants were less likely to be exposed to antenatal steroids than their White counterparts.[14] In Section 3.3, we report event-study estimates produced using high-frequency mortality data. These estimates show sharp reductions in respiratory-related neonatal mortality immediately after Exosurf became available in July 1989: within 4-5 months, White mortality had fallen by 27 percent; within 6-7 months, Black mortality had fallen by 24 percent. We argue that antenatal steroids almost certainly did not contribute to these almost

[14] Carlo et al. (2011) examined the use of antenatal steroids by race at 23 NICUs belonging to the Neonatal Research Network for the period 1993-2009. Sixty-eight percent of Black ELBW infants were exposed to antenatal steroids, while 79 percent of White ELBW infants were exposed. Hamvas et al. (1996) examined exposure to antenatal steroids by race at four NICUs in St. Louis. In 1991-1992, 22 percent of Black VLBW infants were exposed to antenatal steroids, while 36 percent of White VLBW infants were exposed. Because there were racial differences in antenatal steroid exposure, Hamvas et al. (1996, p. 639) acknowledged the difficulty of isolating the effects of SRT on Black versus White neonatal mortality from the effects of antenatal steroids.

immediate reductions in respiratory-related neonatal mortality. Their use gradually increased from the mid-1980s through 1994, when the NIH consensus panel issued its recommendation (Meadow et al. 2003), and there is no evidence, anecdotal or otherwise, that antenatal steroid use increased in the second half of 1989.

Mechanical ventilation became widespread in the 1960s and 1970s, reducing morbidity and mortality among VLBW infants (Boyle et al. 1983; Henderson-Smart et al. 2002). However, it relied on high pressures and oxygen concentrations, which can injure the lung tissue of newborns (Haney and Allingham 1991). High-frequency ventilation (HFV) was developed to mitigate these risks, delivering rapid, low-pressure pulses of air to maintain adequate gas exchange and prevent lung overdistention (Ramanathan 2011). High-frequency jet ventilation (HFJV), one of three main HFV modalities, received FDA approval for commercial use in 1989; flow interruption (HFFI) received FDA approval in 1990; and oscillatory ventilation (HFOV) received FDA approval in 1991 (Cronin 1994; Ackermann et al. 2023).

The timing of these FDA approvals poses a clear threat to identification. To address this concern, we provide separate estimates of the effect of SRT for neonates above and below the 1,000-gram ELBW cutoff. By the mid-1990s, it was not unusual to use HFV in the treatment of ELBW infants suffering from respiratory failure, but its use among VLBW infants weighing at least 1,000 grams remained at negligibly low levels (Richardson et al. 1998).[15] In Section 4.1, we show that FDA approval of SRT is associated with substantially smaller reductions in respiratory-related mortality for neonates below the 1,000-gram ELBW cutoff than for those above it. For example, SRT is associated with a 42 percent reduction in respiratory-related mortality among Black neonates

[15] Horbar et al. (2002) analyzed trends in clinical practice for infants weighing between 501 and 1,500 grams at birth in 39 NICUs belonging to the Vermont Oxford Network Database (38 NICUs in the United States and one in Canada). They found that 6.7 percent of these infants were treated with high-frequency ventilation in 1991. Three years later, in 1994, 13.6 percent were treated with high-frequency ventilation.

weighing 750 to 999 grams at birth and a 60 percent reduction in respiratory-related mortality among those weighing 1,000 to 1,499 grams. These results indicate that the introduction of HFV in the late 1980s and early 1990s is not driving our estimated effects of SRT. They are also consistent with the results of several meta-analyses of RCTs comparing HFJV and HFOV with conventional ventilation (Bhuta and Henderson-Smart 1998; Cools et al. 2015; Rojas-Reyes and Orrego-Rojas 2015). According to the results of these meta-analyses, HFV does not reduce the risk of neonatal mortality, although HFOV may reduce the risk of chronic lung disease.[16]

2.4. Medical innovation and racial health disparities: quasi-experimental evidence

As noted above, there is little quasi-experimental evidence on medical innovation and racial health disparities. Indeed, only two quasi-experimental studies have examined the relationship between medical innovation and the Black-White mortality gap. Jayachandran et al. (2010) estimated the effect of sulfa drugs, the first medicine to effectively treat potentially lethal bacterial infections, on mortality by cause and race. Using tuberculosis deaths as a counterfactual outcome, they found that the rapid diffusion of sulfa drugs in the mid-1930s reduced mortality due to pneumonia, scarlet fever, and childbirth. These reductions were much larger for Whites than for Blacks, a pattern of results likely due to segregationist policies and other barriers to access (Jayachandran et al. 2010, p. 143).[17] Miller and Tucker (2011) estimated the impact of keeping electronic medical records (EMRs) on neonatal mortality by race and ethnicity. Exploiting the staggered adoption of privacy regulations

[16] HFJV and HFOV are more commonly used than HFFI (Ganguly et al. 2020). Pardou et al. (1993) and Craft et al. (2003) compared HFFI with conventional ventilation and found no significant differences in several outcomes among ELBW infants, including air leaks, bronchopulmonary dysplasia, and mortality.

[17] Jayachandran et al. (2010) used tuberculosis as a counterfactual outcome because it was not treatable with sulfa drugs. In the mid-1930s, most Blacks lived "in lower-income Southern states and in predominantly rural areas, often far distances from hospitals and physicians" (Jayachandran et al. (2010, p. 140). Jayachandran et al. (2010, p. 140) also noted that hospital segregation likely made it more difficult for Blacks in the South to access sulfa drugs. See Anderson et al. (forthcoming) for a description of hospital segregation in the South during the Jim Crow era.

to isolate plausibly exogenous variation in the use of EMRs, they found larger effects on Black and Hispanic neonatal mortality than on White neonatal mortality.[18]

A recent working paper by Callison et al. (2025) explored how the introduction of direct-acting antivirals (DAAs) for the treatment of Hepatitis C affected liver transplants by race.[19] Introduced in 2014, DAAs increased the number of donor livers available for patients suffering from alcohol-related liver disease and liver cancer. Callison et al. (2025) found that direct-acting DAAs led to a 57 percent increase in liver transplants for White patients but only a 12 percent increase for Black patients, a result that can be attributed to underlying disease etiology: DAAs disproportionately benefited White patients because they were more likely to suffer from alcohol-related liver disease and liver cancer. Their research shows that medical innovation can affect racial health disparities through its interaction with pre-existing differences in disease prevalence.

Finally, Fadlon et al. (2024) studied a digital patient monitoring program at UC San Diego Health (UCSDH) designed to improve cardiovascular health. Patients with poorly managed hypertension received a Bluetooth-enabled device that automatically sent blood pressure readings to an electronic record portal. Clinical teams reviewed the readings and counseled patients on how to better manage their blood pressure. This program raised the likelihood of reaching healthy blood pressure levels by 16 percentage points among White patients and by 9 percentage points among Black and Hispanic patients.[20]

[18] See also Hollingsworth et al. (2024), who examined the effects of a hospital modernization program in North Carolina funded by the Duke Endowment. Begun in 1927, this program reduced Black infant mortality by 14 percent but only reduced White infant mortality by 5 percent (Hollingsworth et al. 2024).

[19] DAAs are designed to specifically target the Hepatitis C virus, leading to much higher cure rates (90+ percent) than had been possible with earlier treatments (Geddawy et al. 2017).

[20] There are, of course, other important drivers of racial health disparities aside from the introduction of new medical technologies. See, for instance, Wherry and Meyer (2016), Kuziemko et al. (2018), and Wherry et al. (2018) for evidence on the relationship between Medicaid eligibility and health outcomes by race.

3. SRT AND NEONATAL RESPIRATORY-RELATED MORTALITY BY RACE, 1980-2000

3.1. Baseline estimates

Our primary outcome is the natural log of respiratory-related neonatal deaths per 100,000 VLBW births. Following Hamvas et al. (1996), we define respiratory-related deaths to include those due to RDS, pulmonary interstitial emphysema, pulmonary hemorrhage, primary atelectasis, chronic respiratory disease arising in the perinatal period, and other respiratory disorders.[21] Neonatal death counts by race and cause are from the NVSS Multiple Cause-of-Death Mortality Files, made available through the data archive of the National Bureau of Economic Research (NBER). Live birth counts come from the NVSS Natality Files, also distributed through the NBER.[22]

Leveraging the fact that SRT is used to treat specific respiratory-related conditions, we estimate the following difference-in-differences (DiD) equation as a first step toward assessing the effect of SRT on neonatal mortality by race:

$$(1) \qquad \ln(Mortality_{cdt}) = \beta_0 + \beta_1 Treated_d \times Post_t + \beta_2 Treated_d + \lambda_c + \gamma_t + \varepsilon_{cdt},$$

where $\ln(Mortality_{cdt})$ is the natural log of the neonatal mortality rate in census division c for disease d in year t. The variable $Treated_d$ is equal to 1 for respiratory-related mortality and is equal to 0 for non-respiratory-related mortality.[23] $Post_t$ is equal to 1 if $t \geq 1990$ and equal to 0 if $t < 1989$. It takes

[21] There is evidence that SRT is an effective treatment for pulmonary interstitial emphysema (Engle and the Committee on Fetus and Newborn 2008), pulmonary hemorrhage (Polin et al. 2014), and primary atelectasis (Khawar and Marwaha 2023). International Classification of Disease (ICD) codes for these conditions are reported in Appendix Table A1.

[22] Figure 1 shows the evolution of respiratory-related neonatal mortality by race for 1980–2000 and Appendix Figure A2 shows the evolution of RDS neonatal mortality by race during the same period. According to both figures, Black neonates were at a lower risk of dying than their White counterparts through the 1980s. After 1989, the year in which Exosurf was approved by the FDA, the Black-White mortality gap narrows substantially.

[23] Appendix Figure A3 shows the evolution of non-respiratory mortality during the period 1980-2000. There were steady, gradual declines in the non-respiratory mortality rates for both Black and White neonates through the early 1990s. We excluded the following causes of death when constructing non-respiratory-related mortality, where 3-digit ICD-9 codes are in parentheses: acute upper respiratory infections (460-465), bronchitis and bronchiolitis (466, 490-

on the value 0.5 in 1989 because the FDA approved Exosurf in July of that year. Census division and year fixed effects are represented by λ_c and γ_t, respectively.[24]

The coefficient of interest is β_1. It captures the change in respiratory mortality between the pre- and post-treatment periods (relative to the change in non-respiratory mortality). Common shocks to respiratory and non-respiratory neonatal mortality from, for instance, better ultrasound imaging (Campbell 2013), advances in prenatal care (Peahl and Howell 2021; Briceño-Pérez et al. 2019), and increased receipt of prenatal care (Lewis et al. 1996), are captured by the year fixed effects.[25] We estimate equation (1) separately for Blacks and Whites. We use the ratio of the Black respiratory-related neonatal mortality rate to the White respiratory-related neonatal mortality rate as an additional outcome.[26] Estimates are weighted by the relevant census division live birth counts and standard errors are corrected for clustering at the disease-year level.[27] See Appendix Table A2 for variable definitions and descriptive statistics.

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491), pneumonia and influenza (480-487), remainder of diseases of respiratory system (470-478, 492-519), congenital anomalies of respiratory system (748), RDS (769), and other respiratory conditions of newborn (770).

[24] Estimating equation (1) at the national level yields estimates of β_1 that are similar to those reported below. Defining the unit of observation at the census division level allows for the inclusion of census division fixed effects, which can improve precision. It also allows us to consider weighting schemes that, for instance, place more emphasis on areas with larger Black populations.

[25] To the extent that antenatal steroids reduce deaths from non-respiratory conditions such as intraventricular hemorrhage and necrotizing enterocolitis (Bauer et al. 1984; Elimian et al. 1999), their effects are captured by the year fixed effects. In the late 1980s and early 1990s, new methods of delivering prenatal care, including group prenatal care (GPNC), were developed (Peahl and Howell 2021; Mazzoni and Carter 2017). Among low-income and Black mothers, GPNC is associated with a reduction in the likelihood of premature birth; among low-income and adolescent mothers, it is associated with an increase in the number of prenatal visits (Byerley and Haas 2017). During the period 1989-1993, receipt of first-trimester prenatal care among White mothers in the United States increased by 2.9 percentage points, from 78.9 to 81.8 percent; among Black mothers, receipt of first-trimester prenatal care increased by 6 percentage points, from 60.0 to 66.0 (Lewis et al. 1996).

[26] Similarly constructed outcomes have been used by previous researchers interested in exploring the determinants of Black-White mortality gaps (Anderson et al. 2021) or Black-White gaps in arrests, hiring, and wages (Neal 2004; LaFree et al. 2010; Quillian et al. 2017).

[27] For instance, estimates of the effect of SRT on White respiratory-related neonatal mortality are weighted by the number of White births in census division c and year t. We weight by total live births in census division c and year t when the Black-White mortality ratio is used as the outcome. Standard errors are corrected for clustering at the disease-year level because $Treated_d \times Post_t$ varies by disease and year. We also implemented the heteroskedasticity-corrected bootstrap procedure developed by Ferman and Pinto (2019), which is designed for DiD settings with a limited number

In column (1) of Table 1, we report estimates of β_1 from equation (1). FDA approval of SRT is associated with a 52 percent reduction ($e^{-0.725} - 1 = -0.516$) in respiratory-related mortality among White neonates (Panel A). This estimate translates to 8,670 fewer respiratory-related deaths per 100,000 VLBW births and explains 87 percent of the observed decline between the pre- and post-treatment periods.[28]

For Black neonates, the estimate of β_1 is also negative, large, and precisely measured (Panel B), suggesting that SRT led to a 42 percent decline ($e^{-0.544} - 1 = -0.420$) in respiratory-related mortality. However, because it is substantially smaller in absolute magnitude than the corresponding estimate for Whites, the FDA approval of SRT is associated with a 17 percent increase ($e^{0.156} - 1 = 0.169$) in the ratio of Black to White respiratory-related mortality (Panel C).[29] Based on these estimates, SRT accounts for 73 percent of the narrowing in the Black-White respiratory-related neonatal mortality gap between the pre- and the post-treatment periods.

In column (2) of Table 1, we focus on RDS mortality. In the pre-treatment period (1980-1988), 53 percent of respiratory-related deaths among Black neonates were due to RDS, while 60 percent of respiratory-related deaths among Whites were due to RDS. The FDA approval of SRT is associated with a 60 percent reduction in White neonatal mortality from RDS, a 47 percent reduction in Black neonatal mortality from RDS, and a 27 percent increase in the ratio of Black to

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of treated groups. This approach produced p-values below 0.01 for all of the estimates reported in Table 1, indicating that our conclusions are not sensitive to the choice of inference method.

[28] In the pre-treatment period (1980-1988), there were, on average, 16,673 respiratory-related deaths per year among White neonates. In the post-treatment period (1989-2000), there were, on average, 6,727 respiratory-related deaths. The difference between these averages is 9,946 and 8,670/9,946 = 0.872.

[29] In the pre-treatment period (1980-1988), the Black-White respiratory-related neonatal mortality ratio was 0.690. In the post-treatment period (1989-2000), it was 0.850.

White RDS mortality. Based on these estimates, SRT accounts for 78 percent of the narrowing in the Black-White RDS neonatal mortality gap between the pre- and post-treatment periods.[30]

In Figure 3, we report the results of several sensitivity tests. Weighting by VLBW Black births, as opposed to total births, does not appreciably change the estimated effect of SRT on the ratio of Black to White respiratory-related neonatal mortality (Row B, Figure 3). Likewise, unweighted estimates of β_1 (Row C) and estimates that use neonatal mortality due to congenital anomalies as an alternative counterfactual outcome (Row D) are similar to those discussed above. During the period under study, more than one-quarter of neonatal deaths were due to congenital anomalies such as heart, neural tube, or chromosomal defects.[31] Using influenza mortality as the counterfactual outcome increases the estimated effect of SRT on the ratio of Black to White respiratory-related neonatal mortality from 17 percent to 24 percent (Row E). Influenza primarily affects the respiratory tract but is not effectively treated by SRT (Tan et al. 2012; Ng and Shah 2021). Finally, we define the mortality rate as infant deaths per 100,000 LBW (low birthweight) births, effectively broadening the population considered at risk. The estimates of β_1 from this last exercise are slightly smaller in magnitude but remain significant at the 0.01 level.[32]

[30] In the pre-treatment period (1980-1988), the Black-White RDS neonatal mortality ratio was 0.609. In the post-treatment period (1989-2000), it was 0.819.

[31] Newborns with congenital anomalies, like those with respiratory-related issues, are more likely to be low birthweight (Mili et al. 1991).

[32] Corresponding estimates for White neonatal mortality and Black neonatal mortality are reported in Appendix Tables A3 and A4. In Panel G of these tables, we also show estimates in levels as opposed to taking the natural log of the mortality rate. In Appendix Table A5, we report the estimates used to produce Figure 3. In Appendix Figure A4, we report estimates of β_1 separately for the following regions: West, Midwest, South, and Northeast. Measuring the extent to which racial health disparities differ across space has become a point of emphasis among U.S. health policymakers (Radley et al. 2024). Across all four U.S. regions, SRT is associated with precisely measured increases in the ratio of Black to White respiratory-related neonatal mortality, indicating that our estimates are not being driven by any one geographic area.

3.2. Event-study estimates

The estimates reported in Table 1 do not reveal the dynamics of how SRT influenced neonatal mortality. Moreover, they risk conflating the effects of SRT and antenatal steroids, the use of which appears to have increased gradually through the mid-1990s. To address these limitations, we estimate the following event-study regression:

$$(2) \qquad \ln(Mortality_{cdt}) = a_0 + \sum_{\tau \neq 1988} \pi_\tau (1\{t = \tau\} \times Treated_d) + a_1 Treated_d + \lambda_c + \gamma_t + \varepsilon_{cdt},$$

where the event-year dummies, $1\{t = \tau\}$, are equal to 1 when the year of observation is equal to τ. We omit $\tau = 1988$ from equation (2), which normalizes the estimates of π_τ to 0 in that year.[33] The pre-treatment estimates of π_τ can be thought of as falsification tests—their patterns and statistical significance allow us to assess the plausibility of the parallel trends assumption.

In Panel A of Figure 4, we report estimates of π_τ by race for the natural log of the respiratory-related neonatal mortality rate. For the sake of comparison, we report the baseline DiD estimates of β_1 in the bottom-left corner of Panel A, which can be thought of as summarizing the event-study estimates. Consistent with the parallel trends assumption, the pre-treatment estimates are small and, with only one exception (1980 for Blacks), statistically indistinguishable from zero. Formal power calculations based on Roth (2022) indicate that any pre-existing trend large enough to explain the results reported below would be detected with high probability.[34]

[33] The event-study estimates reported below change little if we, as recommended by Miller (2023), extend the omitted category to include multiple pre-treatment years (e.g., $\tau = 1987$ and 1988, or $\tau = 1986$, 1987, and 1988).

[34] Following Roth (2022), we assessed whether we have sufficient power to detect systematic pre-treatment trends of meaningful magnitude. For White neonates, we have sufficient power to detect a linear pre-treatment trend with a slope of only 0.011 log points per year 50 percent of the time, and a slope of 0.020 log points per year 80 percent of the time, standard benchmarks in power analyses (Cohen 1988). For Black neonates, the corresponding slopes are 0.011 and 0.019 log points per year. Based on these calculations and using the 80-percent threshold, the accumulated bias in our estimated treatment effects would reach approximately 0.24 log points for Whites and 0.23 log points for Blacks by the year 2000. Even under this demanding threshold, these accumulated biases represent only 21 and 25 percent of the estimated treatment effects that we report for the year 2000, respectively. Taken alongside the large and monotonically

In 1989, the first year of the post-treatment period, there is little evidence that SRT affected Black respiratory-related neonatal mortality. The corresponding estimate for White respiratory-related mortality, although negative, is likewise small and statistically insignificant at conventional levels. There is, however, a clear break in trend after 1989. After 1989, the estimates of π_τ are consistently negative, statistically significant, and growing over time in (absolute) magnitude. Moreover, an obvious Black-White gap emerges in 1993, approximately four years after the FDA approval of Exosurf. The 1993 estimate of π_τ for Whites is -0.609, suggesting that respiratory-related mortality among White neonates fell by 46 percent because of SRT; the 1993 estimate for Blacks is -0.356, suggesting that respiratory-related mortality among Black neonates fell by 30 percent because of SRT. It is important to note that this Black-White gap in the estimates develops before 1994, when the NIH consensus panel recommended the use of antenatal steroids for mothers at risk of pre-term delivery. We calculate that, by 1993, the introduction of SRT had narrowed the Black-White respiratory-related neonatal mortality gap shown in Figure 1 by 93 percent. [35]

The Black-White gap between the estimates of π_τ is widest in 1998, approximately 9 years after the FDA approval of Exosurf, and is, in fact, large enough to explain the entire narrowing of the Black-White gap shown in Figure 1. The 1998 estimate of π_τ for Blacks is -0.631, suggesting that respiratory-related mortality among Black neonates had fallen by 47 percent relative to the omitted year (1988). By contrast, the 1998 estimate of π_τ for Whites is -0.935, suggesting that respiratory-related mortality among White neonates had fallen by 61 percent. It is important to note, however, that these 1998 estimates likely reflect the combined effects of SRT and antenatal steroids. As

growing post-treatment estimates shown below, this exercise provides strong evidence that systematic pre-treatment trends are not driving our results.

[35] The Black-White respiratory-related neonatal mortality gap narrows by 0.273 log points between 1988 and 1993 (Figure 1). The estimates of π_τ imply a narrowing of 0.253 log points over the same period.

discussed in Section 2.3, there is evidence that premature Black infants were less likely to be exposed to antenatal steroids than their White counterparts (Hamvas et al. 1996; Carlo et al. 2011). The growing gap between the Black and White estimates of π_τ could reflect the fact that antenatal steroids and SRT work synergistically (Polin et al. 2014). It could also reflect refinements in SRT dosage and the timing of administration as new evidence from RCTs emerged and as neonatologists gained experience (Horbar et al. 2002).[36]

In Panel B of Figure 4, we report estimates of π_τ using RDS mortality as the outcome. After 1989, the estimates of π_τ are, without exception, negative, but White neonates appear to have experienced larger and more immediate reductions in RDS mortality than Black neonates. Again, a Black-White gap clearly emerges in 1993. The 1993 estimate of π_τ for White neonates is -0.674, suggesting that the RDS mortality rate among White neonates fell by 49 percent because of SRT. The corresponding estimate for Black neonates is -0.328, suggesting a 28 percent reduction in the RDS mortality rate because of SRT.

3.3. Event-study estimates based on high-frequency data

Counts of neonatal deaths in the NVSS Multiple Cause-of-Death Mortality Files are sufficiently dense to estimate equation (2) at the monthly level. The dependent variable becomes $\ln(Mortality_{cdm})$, the natural log of the neonatal mortality rate in census division c for disease d in month m. For ease of visualization, we group the event-time indicators into two-month bins. Estimating equation (2) based on higher-frequency data allows us to pinpoint when SRT first had an appreciable impact on neonatal mortality.

[36] As physicians gained experience, they began administering SRT immediately after delivery, which reduces pulmonary air leaks and mortality (Ng and Shah 2021). In the early 1990s, research showed that surfactant administration could be followed by immediate extubation to (minimally invasive) continuous positive airway pressure (CPAP), reducing air leaks and lung damage. This practice, known as INSURE (INtubate-SURfactant-Extubate to CPAP), gained popularity during the period under study (Bohlin 2012).

Among White neonates, negative estimated effects become apparent almost immediately after the FDA approval of Exosurf (Figure 5, Panel A). Within 4-5 months of FDA approval, SRT is associated with a 21 percent reduction in White respiratory-related mortality. This estimate can be thought of as a pure SRT effect. Although gradually trending upwards, the use of antenatal steroids remained at relatively low levels through the early 1990s (Meadow et al. 2003). Among Black neonates, negative estimated effects clearly manifest 6-7 months after the FDA approval of Exosurf (Figure 5, Panel B). In January-February of 1990, SRT is associated with a 19 percent reduction in Black respiratory-related mortality. By January-February of 1991 it is associated with a 23 percent reduction.[37] In Figure 6, we report estimates based on high-frequency RDS mortality data.[38]

4. USING THE LINKED BIRTH AND INFANT DEATH FILES

4.1. Estimates by birthweight category

In Table 2, we report estimates of β_1 from equation (1) using the Linked Birth and Infant Death Files for the years 1983–1991 and 1995–2000. As noted above, these data are based on linkages between birth and death certificate records for all children who died within their first year of life.[39] One of the advantages to using these data is that they contain information on birthweight, which allows us to address high-frequency ventilation (HFV) technologies as a potential confounder. The primary disadvantage is that the NVSS did not produce linkages for the years 1992-1994.

[37] To facilitate comparison, we report the event-study estimates from both Panels A and B in Panel C of Figure 5. Consistent with the results based on annual data (Figure 4, Panel A), the gap between the Black and White estimates emerges in the early 1990s and is widest during the period 1996-1998.

[38] Consistent with the results based on annual data (Figure 4), the Black-White gap in estimated effects is more pronounced in the mid- to late-1990s when RDS (as opposed to respiratory-related) mortality is used as the outcome.

[39] The linked files are organized by birth-year cohorts (infants born in a given calendar year), with deaths within the first 365 days of life assigned to the infant's calendar year of birth (NCHS 2023a). For more information on the Linked Birth and Infant Death Files, see National Center for Health Statistics (2023a).

In the first three columns of Table 2, we report separate estimates of β_1 for neonates in the following birthweight categories: <750 grams, 750 to 999 grams, and 1,000 to 1,499 grams.[40] In the last column of Table 2, we report estimates for all neonates regardless of birthweight category. These estimates are very similar to those reported in Table 1, indicating that the lack of 1992-1994 data does not appreciably alter the results of our analysis.

SRT is associated with smaller reductions in respiratory-related mortality for neonates below the 1,000-gram cutoff than for those immediately above it. This pattern of results is apparent for both Black and White neonates. For example, SRT is associated with a 42 percent reduction in respiratory-related mortality among Black neonates weighing 750 to 999 grams at birth, and a 60 percent reduction among those weighing 1,000 to 1,499 grams. SRT is associated with a 54 percent reduction in respiratory-related mortality among White neonates weighing 750 to 999 grams at birth, and a 72 percent reduction among those weighing 1,000 to 1,499 grams. Between 1989 and 1994, HFV use rose sharply among ELBW infants but remained exceedingly rare among VLBW infants weighing at least 1,000 grams (Richardson et al. 1998).[41] The smaller estimated effects of SRT for neonates below the 1,000-gram cutoff suggest that confounding from the contemporaneous adoption of HFV is not an important issue. This result is consistent with those of RCTs that have

[40] These three birthweight categories were based on Richardson et al. (1998). The mortality rate is defined as neonatal deaths per 100,000 live births within a birthweight category. The sample sizes reported in Table 2 are not uniform due to a small number of cells with zero deaths.

[41] Richardson et al. (1998) used data from Brigham and Women's Hospital and Beth Israel Hospital, both of which are academic medical centers in Boston, MA with substantial delivery volumes. They compared rates of therapeutic interventions for VLBW infants delivered in 1989-1990 versus those delivered in 1994-1995. Among infants in the 1,000-1,499-gram birthweight category, the use of SRT went from 21 to 39 percent, while the use of HFV went from 0.5 percent to 1.5 percent. Among infants in the 750-1,000-gram birthweight category, the use of surfactant went from 29 to 76 percent, while the use of HFV went from zero to 10 percent. Richardson et al. (1998) did not report results for infants born over the 1,500-gram threshold.

failed to find a link between HFV and neonatal mortality (Bhuta and Henderson-Smart 1998; Cools et al. 2015; Rojas-Reyes and Orrego-Rojas 2015).[42]

4.2. Estimates by mother's educational attainment and marital status

The fundamental causes hypothesis posits that, as medical knowledge advances, health disparities can widen because disadvantaged groups are unable to access new treatments and technologies (Link and Phelan 1995; Link et al. 1998). To explore this hypothesis, we use the Linked Birth and Infant Death Files to estimate separate effects by mother's educational attainment and marital status, two factors which could, in theory, determine SRT access (Frisbie et al. 2004).[43] During the period under study, 26 percent of White VLBW infants were born to mothers without a high school degree, as compared to 31 percent of Black VLBW infants; 30 percent of White VLBW infants were born to unmarried mothers, as compared to 70 percent of Black VLBW infants.

Estimates for neonates born to mothers with less than four years, exactly four years, and more than four years of high school are reported in columns (1) through (3) of Table 3. These estimates provide no evidence of a differential impact by mother's educational attainment, suggesting that race-based differences in access to SRT do not account for the narrowing of the

[42] In column (4) of Table 2, we report estimates of the effect of SRT on respiratory-related mortality among neonates weighing above 1,500 grams at birth. SRT is associated with a 55 percent reduction in respiratory-related mortality among White neonates weighing more than 1,500 grams at birth. It is associated with a 42 percent reduction in mortality among Black neonates weighing more than 1,500 grams at birth. SRT was generally targeted at VLBW infants, but its effectiveness has been documented among newborns above the VLBW cutoff (Lotze et al. 1993; Ramanathan 2011; Polin et al. 2014; Ramaswamy et al. 2022). The estimated effects of SRT on respiratory-related mortality for neonates weighing more than 1,500 grams at birth are similar in magnitude to the estimates reported in column (2) of Table 2. It should be noted, however, that neonates weighing more than 1,500 grams at birth had much lower baseline rates of respiratory-related mortality than neonates in the lower birthweight categories. See Appendix Table A6 for estimates of the effect of SRT on RDS neonatal mortality by race and birthweight.

[43] Surfactant is a relatively low-cost medicine and administered based on need (Phibbs et al. 1993; AMA Council on Ethical and Judicial Affairs 2010). Under the Emergency Medical Treatment and Active Labor Act of 1986, protections for infants begin at the time of birth and require hospitals with emergency departments to provide stabilizing treatments for emergency medical conditions (Centers for Medicare and Medicaid Services 2019).

Black–White respiratory-related mortality gap shown in Figure 1. Among White neonates (Panel A), the SRT estimates are similar in magnitude and statistically indistinguishable across mother's educational attainment categories. This same pattern of results is also apparent for Black neonates (Panel B). Overall, there is little evidence that infants born to more educated mothers had greater access to, or disproportionately benefited from, SRT.

Estimates of β_1 for neonates born to married and unmarried mothers are reported in columns (4) and (5) of Table 3. These estimates clearly show that SRT benefited preterm infants regardless of their mother's marital status. For both White and Black neonates, the estimated coefficients are only slightly larger for those born to married as compared to unmarried mothers. These differences are, however, not statistically significant at conventional levels.[44]

5. DID NICU ACCESS MATTER?

In the previous section, we showed that race-based differences in access, as proxied by mother's educational attainment and marital status, do not explain why White neonates benefited more from the FDA approval of SRT than Black neonates. In this section, we consider an alternative measure of access. Specifically, we examine whether differences in NICU proximity can explain the steeper reductions in respiratory-related mortality among White as compared to Black neonates.

In 2004, the first year in which information on SRT use became available in the birth certificate files, 94 percent of newborns who received SRT were also admitted to a NICU.[45] The connection between SRT and NICUs was likely even stronger during the period under study, when

[44] We report the corresponding estimates for RDS mortality as the outcome in Appendix Table A7.

[45] This figure is based on our own calculations using the NVSS 2004 Natality Files. Information on SRT was added during the 2003 revision of the U.S. Standard Certificate of Live Birth (National Center for Health Statistics 2023b). States were, however, slow to adopt this revision. In 2004, only 9 states reported whether a newborn was given SRT.

surfactant therapy was only available in NICUs. Information on NICU location comes from the *Annual Survey of Hospitals*, produced by the American Hospital Association. We merge these data with the restricted-use Linked Birth and Infant Death Files, which include an identifier for the mother's county of residence.[46] Prior to 1989, these identifiers are unavailable for counties with fewer than 250,000 residents; we therefore disaggregate the data to the monthly level and define a 6-month pre-treatment period. Focusing on 1989–1991, we estimate the following equation by race and whether the mother lived within 25 miles of a NICU:

$$(3) \qquad \ln(Mortality_{dm}) = \delta_0 + \delta_1 Treated_d \times Post_m + \delta_2 Treated_d + \gamma_m + \varepsilon_{dm},$$

where $\ln(Mortality_{dm})$ is the natural log of the neonatal mortality rate (i.e., deaths per 100,000 VLBW births) for disease d in month m.[47] The variable $Post_m$ is equal to 1 if $m \geq$ July 1989 and equal to 0 if $m <$ July 1989.

Estimates of δ_1 are reported in columns (1) and (2) of Table 4.[48] They provide little evidence for a barriers-to-access story. For Black neonates whose mother lived within 25 miles of a NICU, the introduction of SRT is associated with an 8 percent reduction in respiratory-related mortality. For Black neonates whose mother lived more than 25 miles from the nearest NICU, SRT is associated with a (statistically insignificant) 10 percent reduction in respiratory-related mortality.

[46] We were able to match 96 percent of the birth certificate records with the nearest NICU. In Appendix B, we describe the data and process used to determine NICU location.

[47] If we define the unit of observation at the census division-disease-month level, we encounter a number of cells with zero deaths. To avoid this problem, we aggregate to the national level and define the unit of observation at the disease-month level.

[48] We used information from the NBER's county distance files (https://www.nber.org/research/data/county-distance-database) to produce the estimates reported in Table 4. A mother was identified as within 25 miles of a NICU if she lived in a county with a NICU or lived in a county that was within 25 miles of another county that contained a NICU, where distance between counties is measured using the Haversine formula and based on internal county points.

Although not statistically distinguishable at conventional levels, these estimates, if taken at face value, suggest that a counterfactual movement of Black mothers nearer to NICUs would not translate into reductions in respiratory-related neonatal mortality—if anything, it would widen the Black-White mortality gap.

For White neonates whose mother lived within 25 miles of a NICU, SRT is associated with a 25 percent reduction in respiratory-related mortality. For White neonates whose mother lived more than 25 miles from the nearest NICU, SRT is associated with a 23 percent reduction. Again, these estimates are not statistically distinguishable. However, if taken at face value, they suggest that a counterfactual movement of White mothers nearer to NICUs would (very modestly) exacerbate racial health disparities.

In columns (3) and (4) of Table 4, we show estimates of equation (3) by race and whether the mother lived within 25 miles of a high-level NICU.[49] These estimates are similar to, and statistically indistinguishable from, those discussed immediately above. In columns (5) through (8) of Table 4, we define access based on whether the mother lived in a county with a NICU or lived in a county with a high-level NICU. The estimates based on these definitions again provide little evidence for the barriers-to-access story. For Black neonates whose mothers lived in a county with a NICU, SRT is associated with a 9 percent reduction in respiratory-related mortality. For Black neonates whose mothers lived in a county without a NICU, SRT is associated with a 7 percent reduction in respiratory-related mortality, but this latter estimate is not statistically distinguishable from the former.[50]

[49] Following Baker and Phibbs (2002), we defined a NICU as high-level if it reported having more than 15 neonatal-intensive and neonatal-intermediate care beds. According to Baker and Phibbs (2002), this cutoff should capture technologically advanced NICUs in the country. In the late 1980s, and based on this definition, roughly one in three NICUs had a high level of capacity.

[50] We also experimented with defining access as the mother having a NICU within 50 miles, within her county of residence or a bordering county, or within the health services area (HSA). These results were qualitatively similar to those shown in Table 4. Finally, we considered access based on proximity to a hospital (regardless of whether it had a

The fact that our SRT estimates are insensitive to whether the mother lived near a NICU should, perhaps, not come as a surprise. Seventy-five (85) percent of White (Black) infants born in 1989 either lived in a county with a NICU or within 25 miles of one. Ninety-three (97) percent of White (Black) mothers either lived in a county with a NICU or within 50 miles of one. Using data from the 1988 National Maternal and Infant Health Survey, Schwartz et al. (2000) found that high-risk Black mothers were actually more likely to deliver in a high-technology setting relative to their White counterparts, and by the late 1980s neonatal and maternal transport for specialty care was quite advanced (Perry 2021).[51]

Taken together, the results reported in Tables 3 and 4 are not consistent with the barriers-to-access hypothesis. Nevertheless, it is important to note that we cannot rule out racial prejudice within the hospital setting. There is evidence that Black premature infants were less likely to receive surfactant than their White counterparts (Howell et al. 2010), which could reflect differences in the need for treatment or could reflect discriminatory behavior on the part of hospitals or attending physicians.[52] The broader role that discrimination plays in driving racial disparities in maternal and infant outcomes has been well documented (Hill et al. 2024).

NICU), whether the mother lived in a metropolitan versus non-metropolitan county, and whether the mother lived in a rural versus urban county. Again, the results were qualitatively similar to those reported in Table 4. County metropolitan and rural statuses were determined based on the National Center for Health Statistics Urban-Rural Classification Scheme for Counties (Ingram and Franco 2012). Appendix Table A8 shows estimates of equation (3) by race and mother's proximity to the nearest NICU for RDS mortality.

[51] With increasing regionalization of perinatal care in the 1970s, the transport of infants requiring intensive care was well established by the time SRT was approved (Perry 2021). Hamvas et al. (1996) studied outcomes for 1,563 VLBW infants born in four St. Louis-area hospitals during the period 1987-1992. Of the 162 infants born outside of a tertiary-care facility, 106 were transferred to one. The 18 infants who died without transport suffered from conditions unlikely to be treated by surfactant (e.g., extreme prematurity or congenital abnormalities). Using data from five counties in Pennsylvania, Durbin et al. (1997) found that uninsured and Medicaid-covered neonates were more likely to be transferred to another hospital than similar neonates covered by private insurance.

[52] In three New York City hospitals, Howell et al. (2010) found that clinicians were less likely to administer surfactant to premature Black, as opposed to White, infants. Using data on VLBW infants born in four NICUs in St. Louis, Hamvas et al. (1996), however, found no race-based differences in surfactant administration.

6. CONCLUSION

Medical innovation has long been an important determinant of infant health and survival. Since 1900, the U.S. infant mortality rate has fallen by nearly 95 percent, a decline closely linked to advances in neonatal and pediatric care. Far less is known, however, about the relationship between medical innovation and racial disparities in infant health. For more than a century, Black infants have persistently lagged their White counterparts on a wide range of health-related outcomes, including mortality (Elder et al. 2014; Bediako et al. 2015; Singh and Yu 2019; Kennedy-Moulton et al. forthcoming). In the absence of barriers to care, advances in medical technology have the potential to address these disparities (Levine et al. 2011), but race-based differences in access and resources could mean that medical innovation widens the Black-White infant health gap. According to the "fundamental causes" hypothesis, disadvantaged groups, because they lack access and resources, may fail to benefit from medical advances even if the technology is highly effective (Link and Phelan 1995; Link et al. 1998).

Widely recognized as one of the most consequential breakthroughs in neonatal medicine (Cummings 2007; Hallman and Herting 2023), surfactant replacement therapy (SRT) is used to treat respiratory-related disorders that commonly occur among premature and very low birthweight infants. It was approved by the Food and Drug Administration (FDA) in 1989 and rapidly adopted by nearly every neonatal intensive care unit (NICU) in the country (Horbar and Lucey 1995). A large body of RCT-based evidence demonstrates that administering surfactant in a timely fashion reduces the risk of respiratory distress syndrome among premature infants and the risk of dying from respiratory-related causes within the first 28 days of life (Polin et al. 2014).

Using mortality records from the National Vital Statistics System, we estimate the effects of SRT on the Black-White neonatal mortality gap. Our results provide compelling evidence that the introduction of this breakthrough treatment reduced neonatal mortality from respiratory-related

disorders for both races. Event-study estimates suggest that within 4-5 months of FDA approval, SRT led to a 21 percent reduction in respiratory-related mortality among White neonates. Within 6-7 months of its approval, there was a 19 percent reduction in respiratory-related mortality among Black neonates.

These sharp, almost immediate, reductions in respiratory-related neonatal mortality can be interpreted as pure SRT effects. The use of antenatal steroids, which also reduces RDS and respiratory-related mortality among neonates (McGoldrick et al. 2020), remained at relatively low levels through the early 1990s (Meadow et al. 2003). In 1994, an NIH consensus panel recommended that pregnant women at risk of preterm delivery be given antenatal steroids, which led to a substantial increase in their use (Planer et al. 1996; Meadow et al. 2003). We estimate that by 1993, one year before the NIH consensus panel issued its recommendation, SRT had reduced respiratory-related mortality among White neonates by as much as 46 percent; we estimate that SRT had reduced respiratory-related mortality among Black neonates by only 30 percent.

Why did the introduction of SRT benefit White neonates more than their Black counterparts? Black fetuses mature at lower gestation ages and birth weights than do White fetuses (Patel et al. 2004; Loftin et al. 2012), which could explain why White neonates experienced a relatively steeper reduction in respiratory-related mortality. Race-based differences in access and resources offer an alternative and plausible explanation. During the period under study, Black VLBW infants were more likely to be born to a mother without a high school education and more likely to be born to an unmarried mother. Our results, however, provide scant support for a barriers-to-access hypothesis. Among both Black and White neonates, SRT estimates are similar in magnitude for married and unmarried mothers and across different levels of mother's educational attainment. Likewise, there is little evidence that proximity to the nearest NICU mattered. In fact, the estimated effect of SRT for Black mothers living within 25 miles of a NICU is slightly smaller

than (although not statistically distinguishable from) the estimated effect for those living more than 25 miles from the nearest NICU, suggesting that a counterfactual movement of Black mothers nearer to NICUs would not have reduced respiratory-related neonatal mortality. We conclude that racial differences in fetal pulmonary maturation, rather than barriers to access, likely drove the uneven impact of SRT.

More generally, our results reinforce the point, made by Weiss et al. (2018) and other health equity researchers (e.g., Press et al. 2021, Haimi 2023, and Ndugga et al. 2024), that ensuring access to care does not by itself guarantee the elimination of racial health disparities. As noted by Cutler et al. (2012), if research dollars are disproportionately directed toward conditions that are more prevalent among Whites, then medical innovation may not translate into comparable health improvements for minority versus majority groups. Surfactant replacement therapy is now standard care in high-income countries, yet it remains costly and difficult to obtain in much of the developing world (Vidyasagar et al. 2011; Tooke et al. 2021). Improving access to SRT in low-resource settings would likely save the lives of thousands of premature infants each year but could simultaneously hinder efforts to reduce health inequalities in countries already struggling to close racial and ethnic gaps in child survival.

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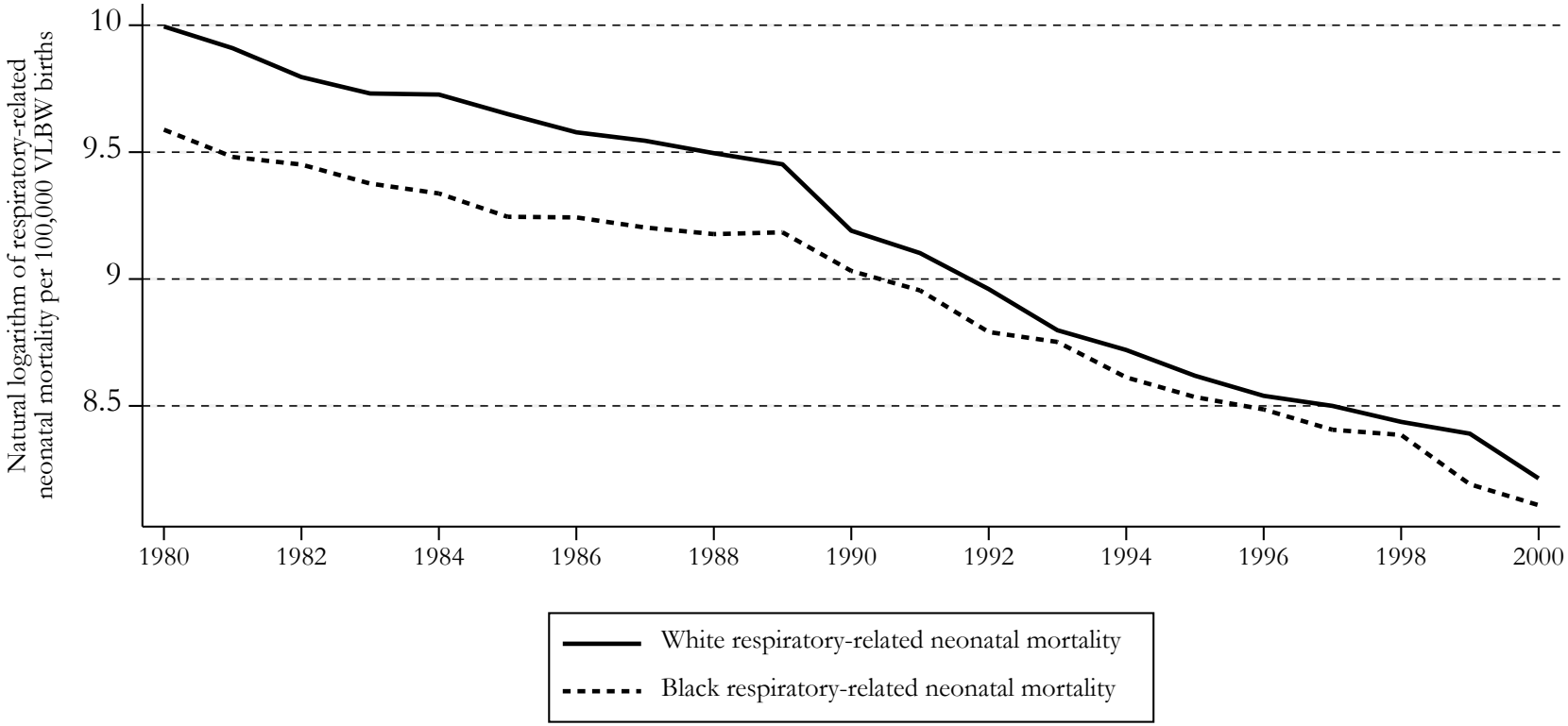
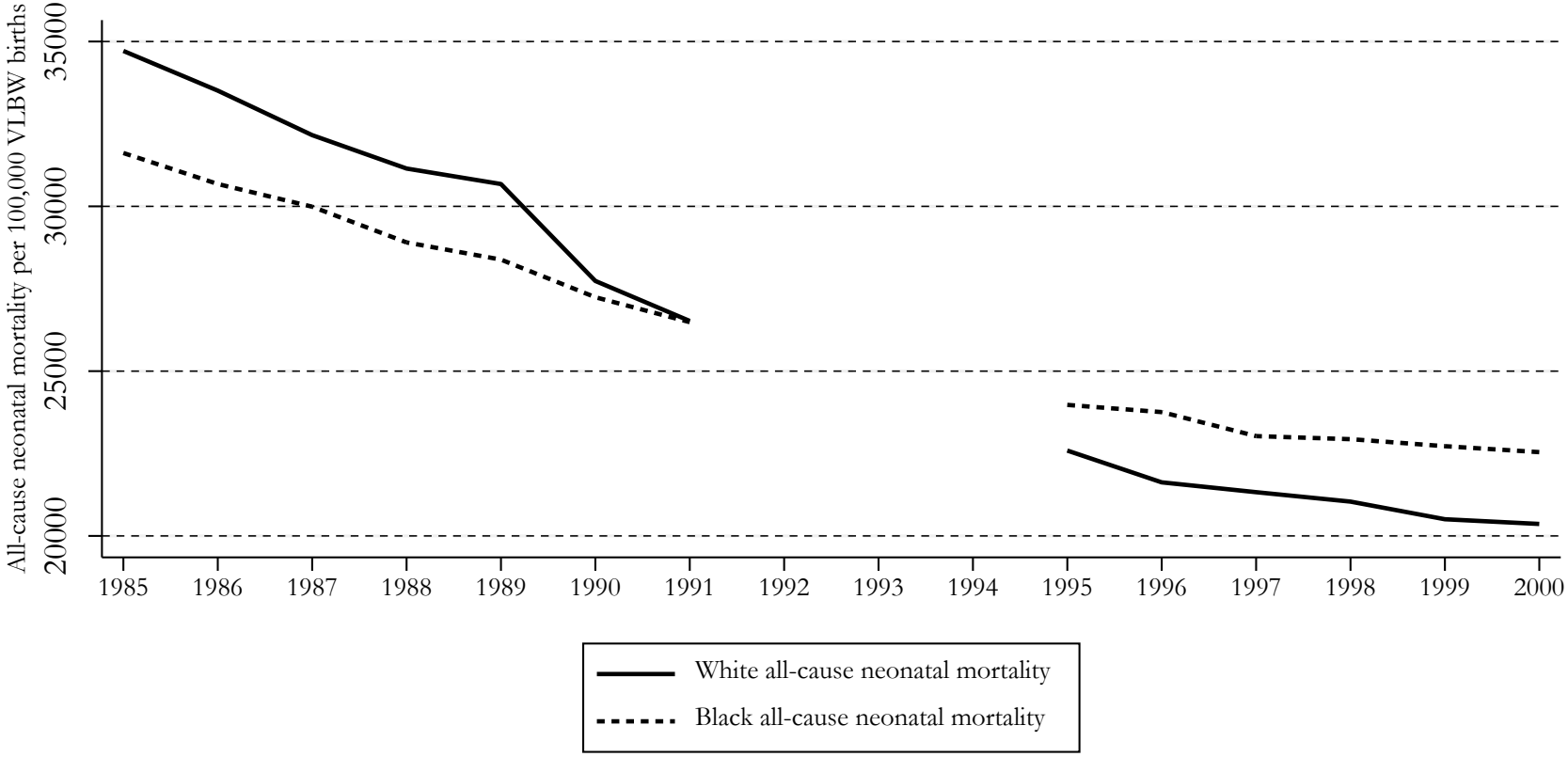
Figure 1. Respiratory-related Neonatal Mortality by Race, 1980-2000



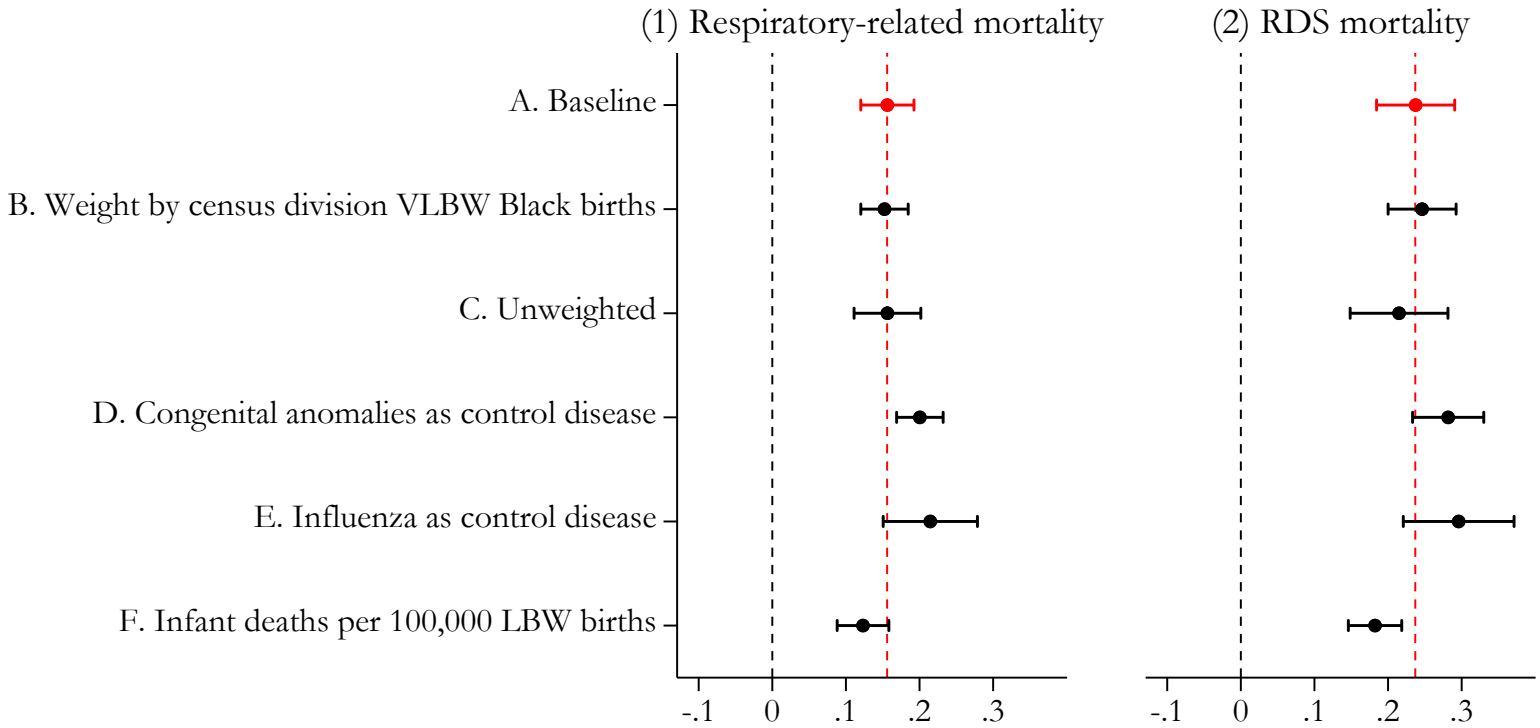
Notes: Based on annual data from the Multiple Cause-of-Death Mortality Files, published by the National Vital Statistics System.

Figure 2. All-cause Neonatal Mortality among VLBW Babies (< 1,500g) by Race



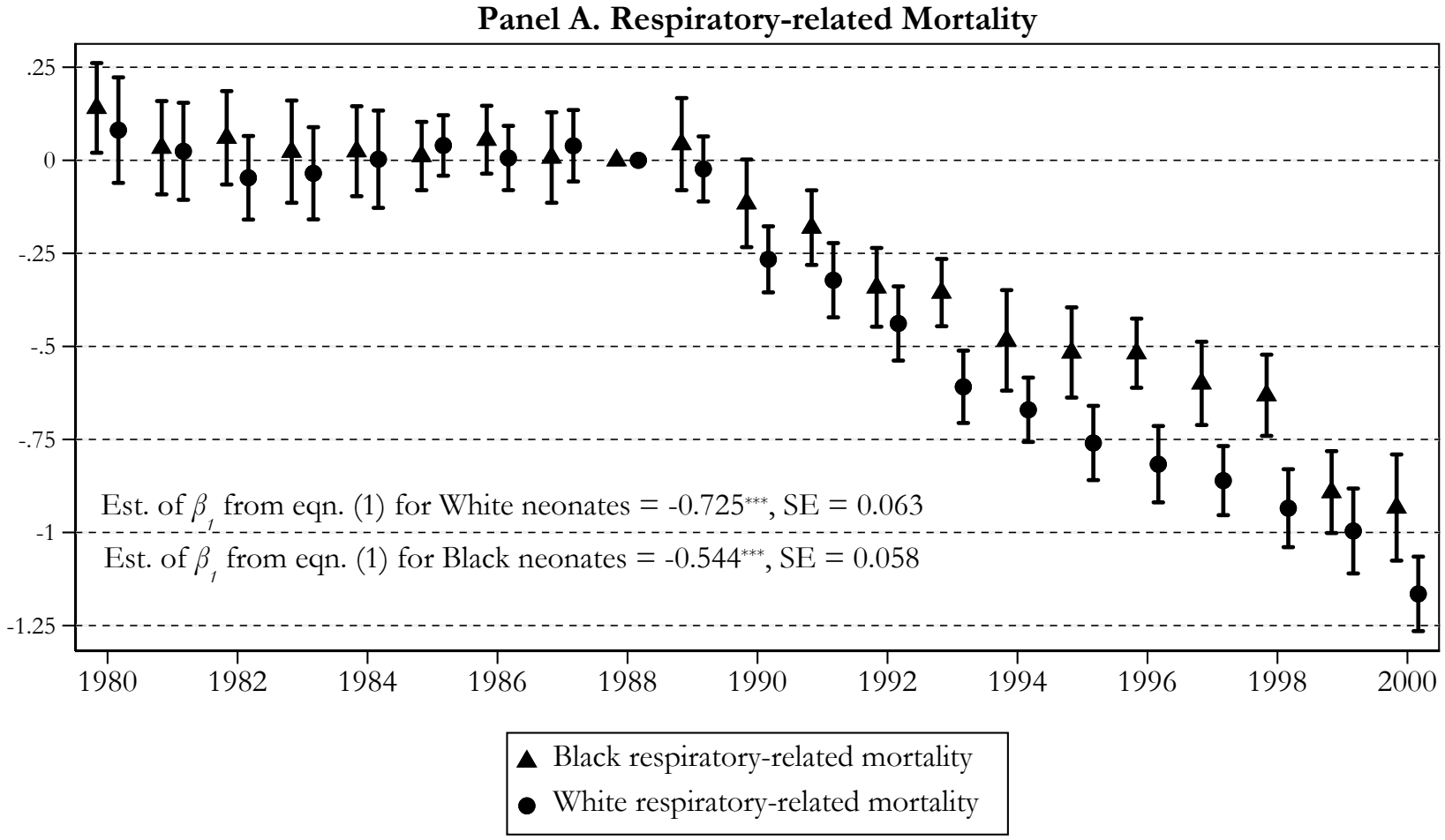
Notes: Based on annual data from the Linked Birth and Infant Death Files, published by the National Vital Statistics System. Linked files were not produced for the years 1992-1994.

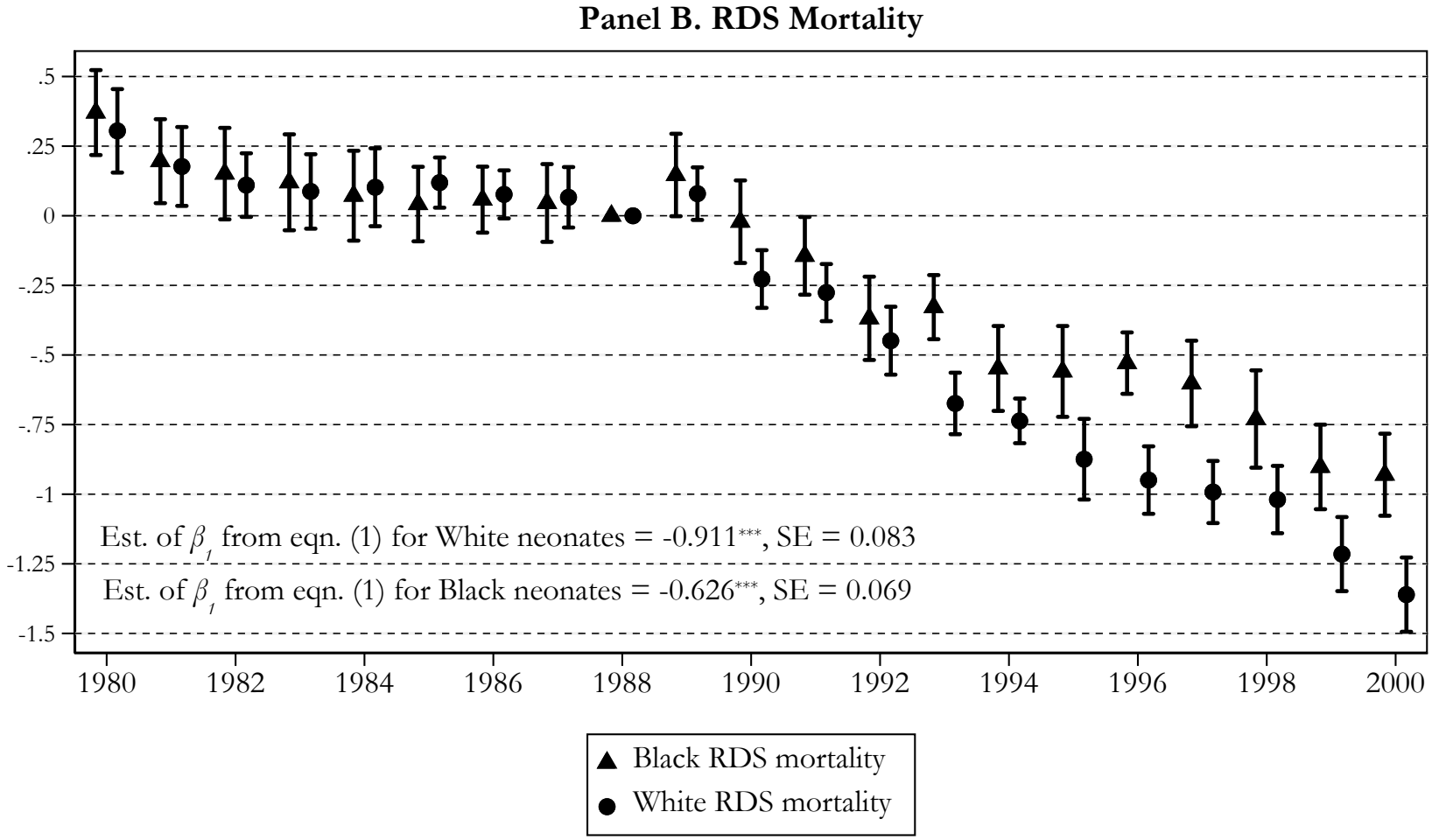
Figure 3. Surfactant Replacement Therapy and the Black-White Mortality Ratio: Sensitivity Analysis



Notes: Based on annual data from the NVSS Multiple Cause-of-Death Mortality Files, 1980-2000. Each plot represents a seperate estimate of β_1 from equation (1). Ninety-percent confidence intervals are reported. In rows A-E, the dependent variable is the natural log of the Black-White ratio of the neonatal mortality rate for disease *d* in census division *c* and year *t*. In row F, the dependent variable is the natural log of the Black-White ratio of the infant mortality rate for disease *d* in census division *c* and year *t*. All regressions include *Treated*, census division fixed effects, and year fixed effects. Unless noted otherwise, estimates are weighted by census division total births. Standard errors are corrected for clustering at the disease-year level.

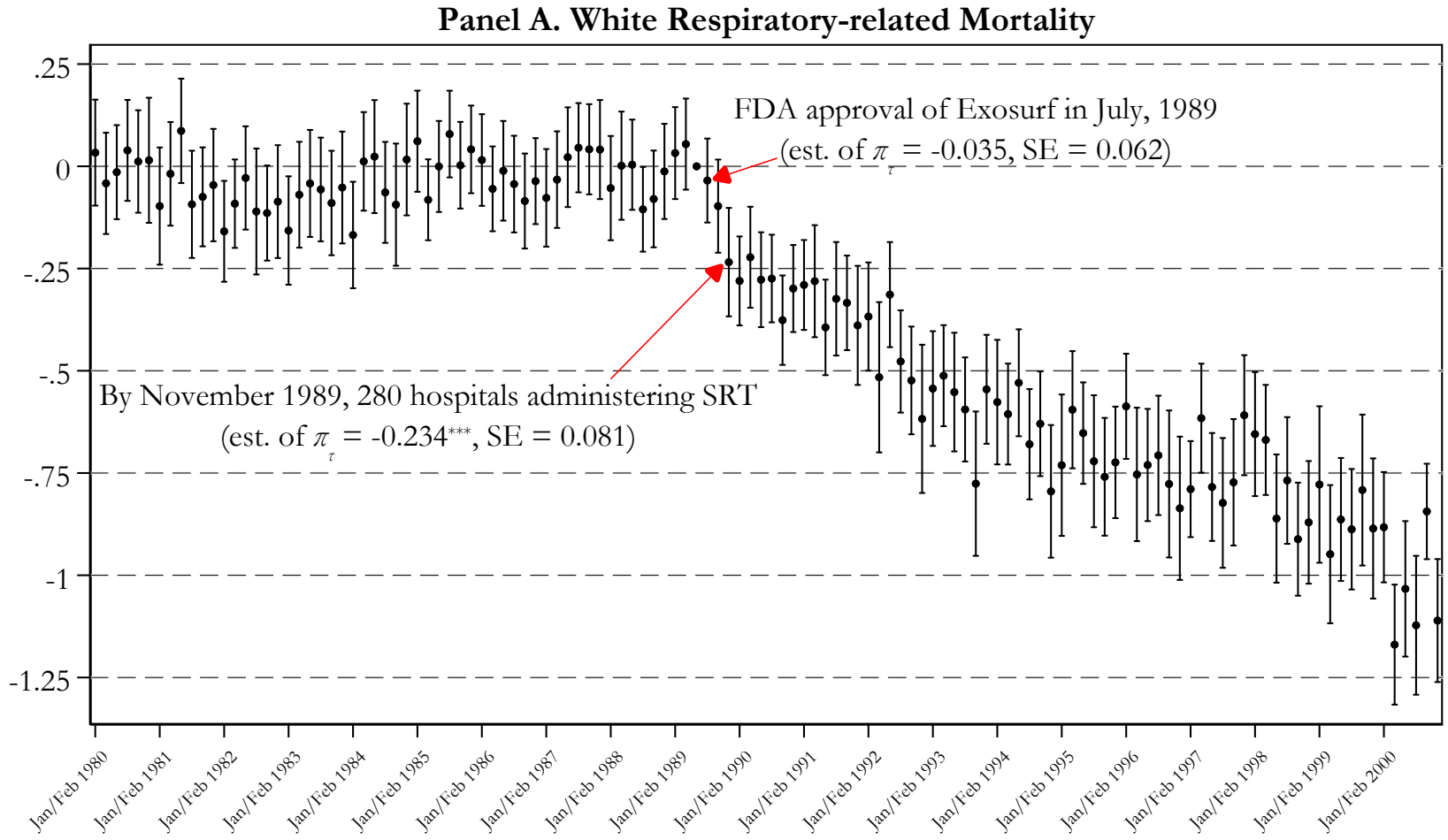
Figure 4. Surfactant Replacement Therapy and Neonatal Mortality by Race: Event-study Analysis

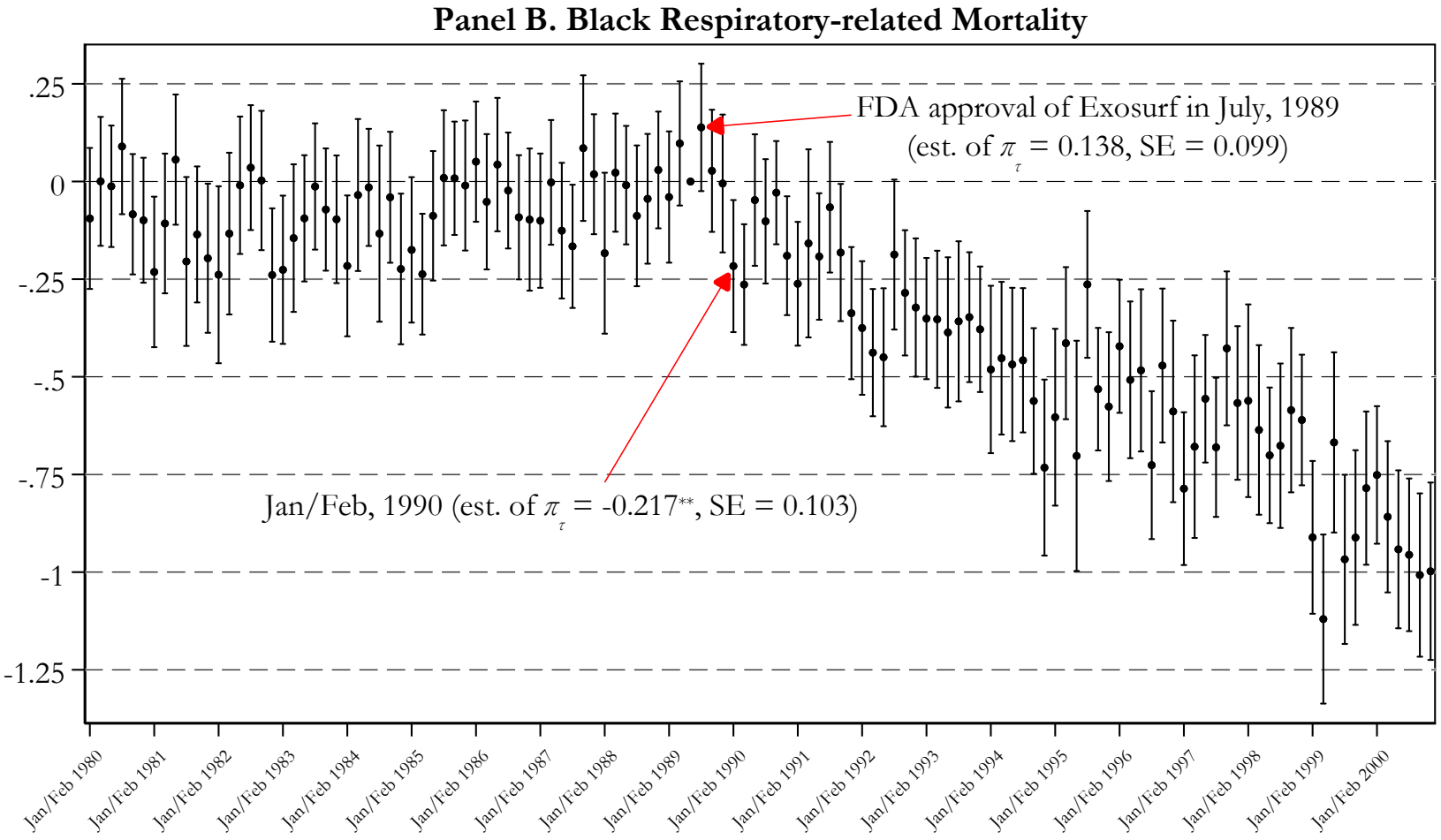


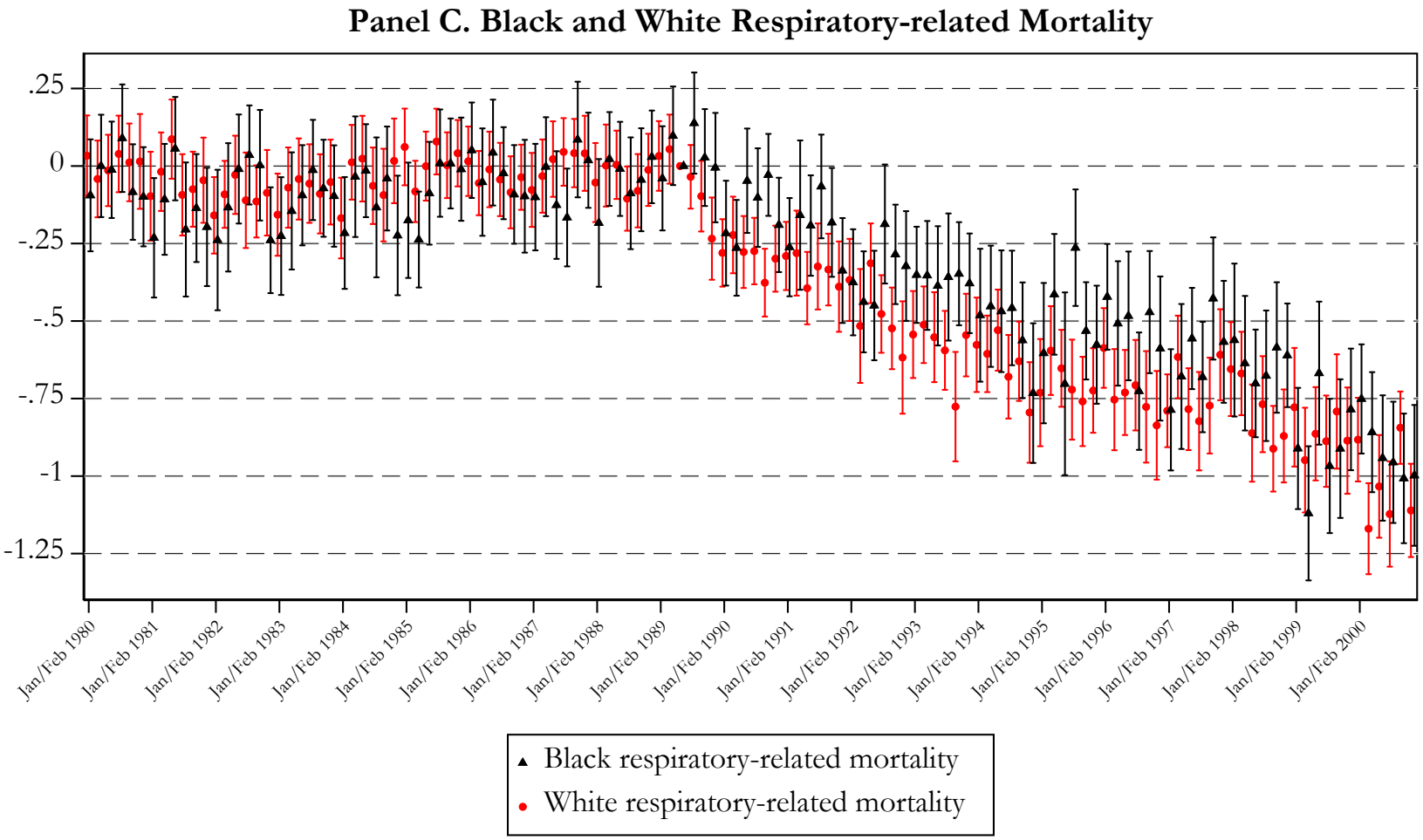


Notes: Based on annual data from the NVSS Multiple Cause-of-Death Mortality Files, 1980-2000. OLS event-study estimates (and their 90% confidence intervals) are reported, where the omitted year is 1988. The dependent variable is the natural log of the race-specific neonatal mortality rate for disease *d* in census division *c* and year *t*. All regressions include *Treated* (= 1 for respiratory-related/RDS mortality, = 0 for non-respiratory-related mortality), census division fixed effects, and year fixed effects. Estimates are weighted by census division race-specific births. Robust standard errors are used to construct the confidence intervals.

Figure 5. Surfactant Replacement Therapy and Respiratory-related Neonatal Mortality by Race: Monthly Event-study Analysis

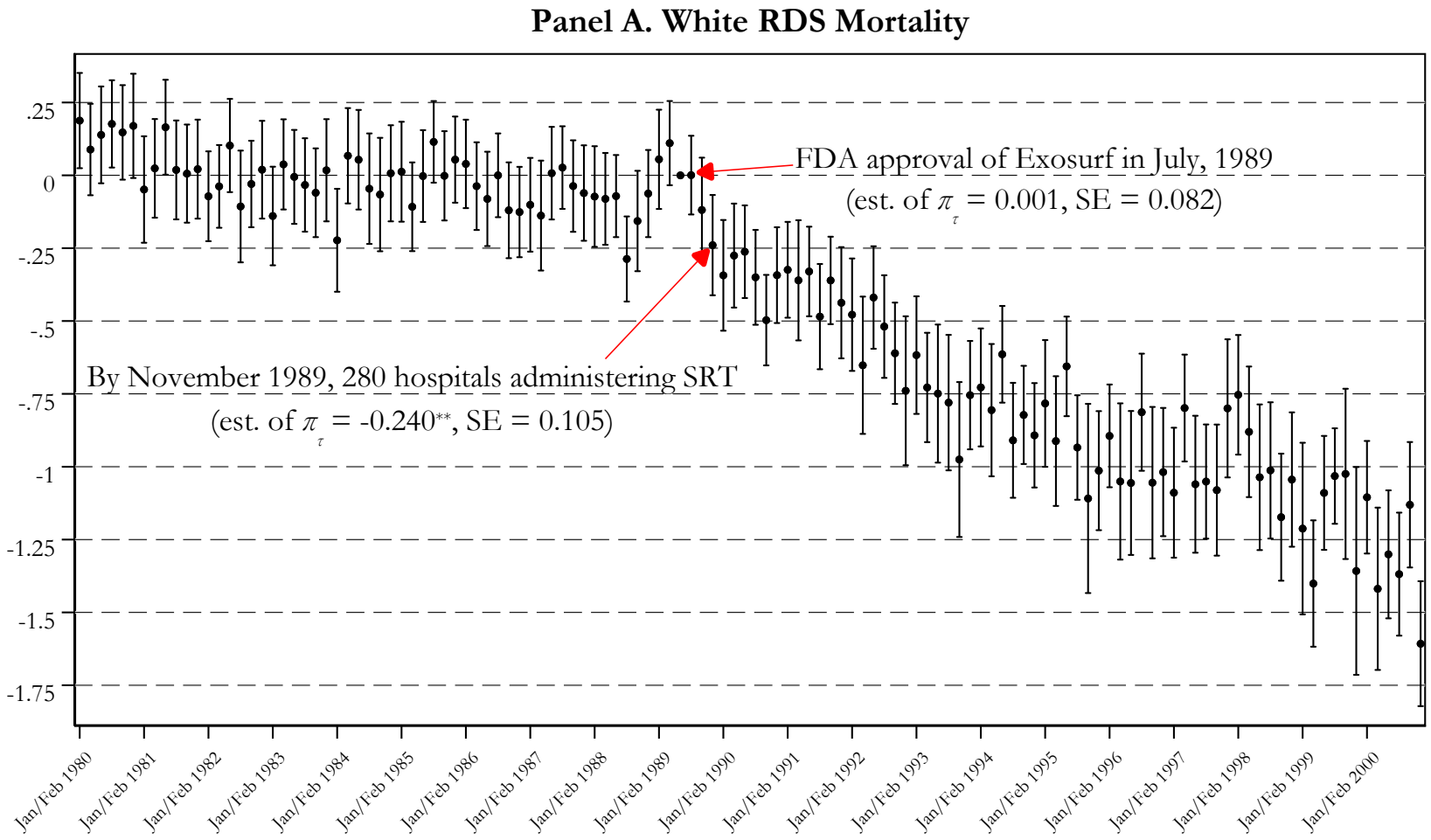



Panel B. Black Respiratory-related Mortality
.25
0
-.25
-.5
-.75
-1
-1.25
FDA approval of Exosurf in July, 1989
(est. of π_τ = 0.138, SE = 0.099)
Jan/Feb, 1990 (est. of π_τ = -0.217**, SE = 0.103)
Jan/Feb 1980
Jan/Feb 1981
Jan/Feb 1982
Jan/Feb 1983
Jan/Feb 1984
Jan/Feb 1985
Jan/Feb 1986
Jan/Feb 1987
Jan/Feb 1988
Jan/Feb 1989
Jan/Feb 1990
Jan/Feb 1991
Jan/Feb 1992
Jan/Feb 1993
Jan/Feb 1994
Jan/Feb 1995
Jan/Feb 1996
Jan/Feb 1997
Jan/Feb 1998
Jan/Feb 1999
Jan/Feb 2000

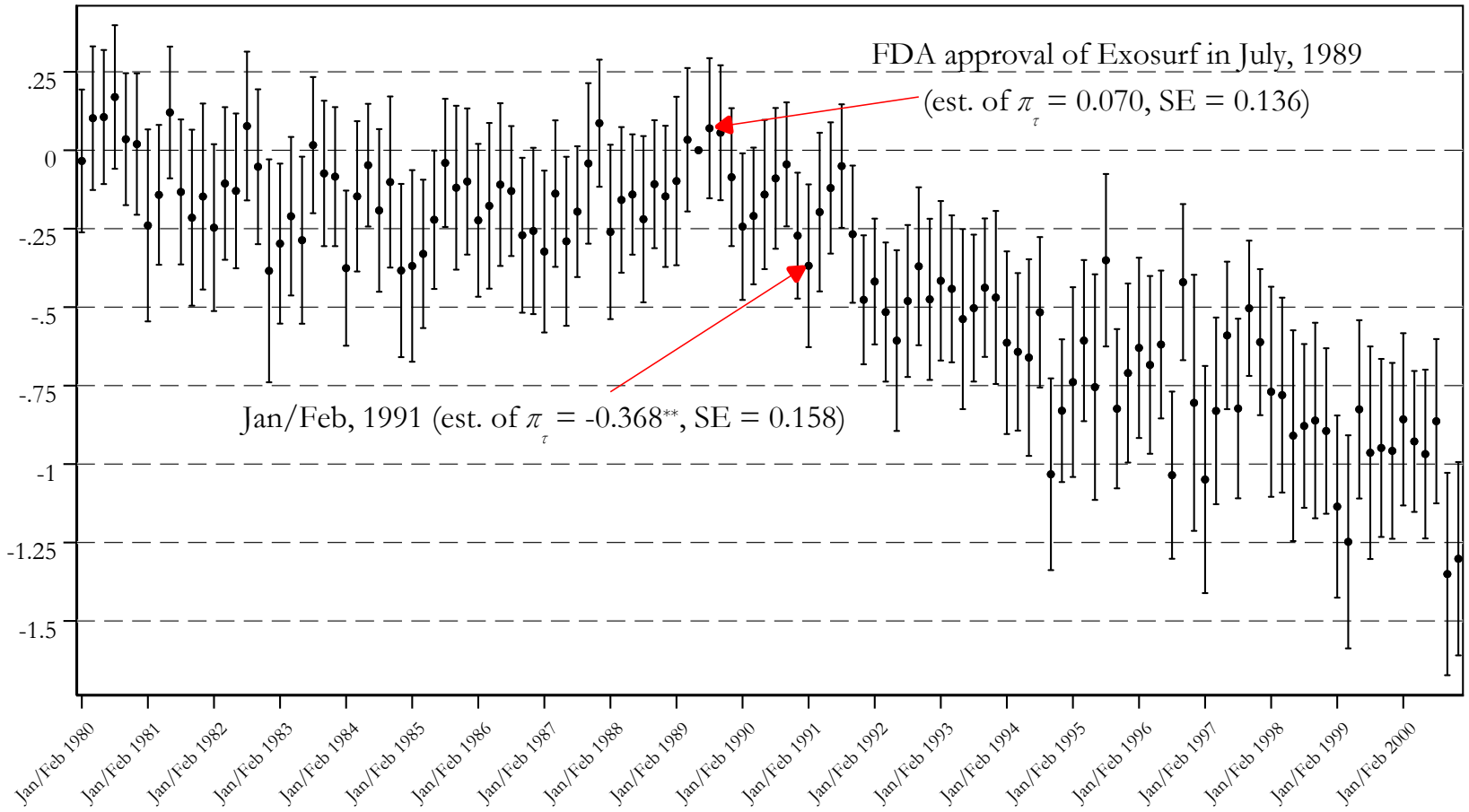


Notes: Based on monthly data from the NVSS Multiple Cause-of-Death Mortality Files, 1980-2000. OLS event-study estimates (and their 90% confidence intervals) are reported, where the omitted category is May/June 1989. The dependent variable is the natural log of the race-specific respiratory-related neonatal mortality rate for disease *d* in census division *c* and month *m*. All regressions include *Treated* (= 1 for respiratory-related mortality, = 0 for non-respiratory-related mortality), census division fixed effects, and month fixed effects. Estimates are weighted by census division race-specific births. Robust standard errors are used to construct the confidence intervals.

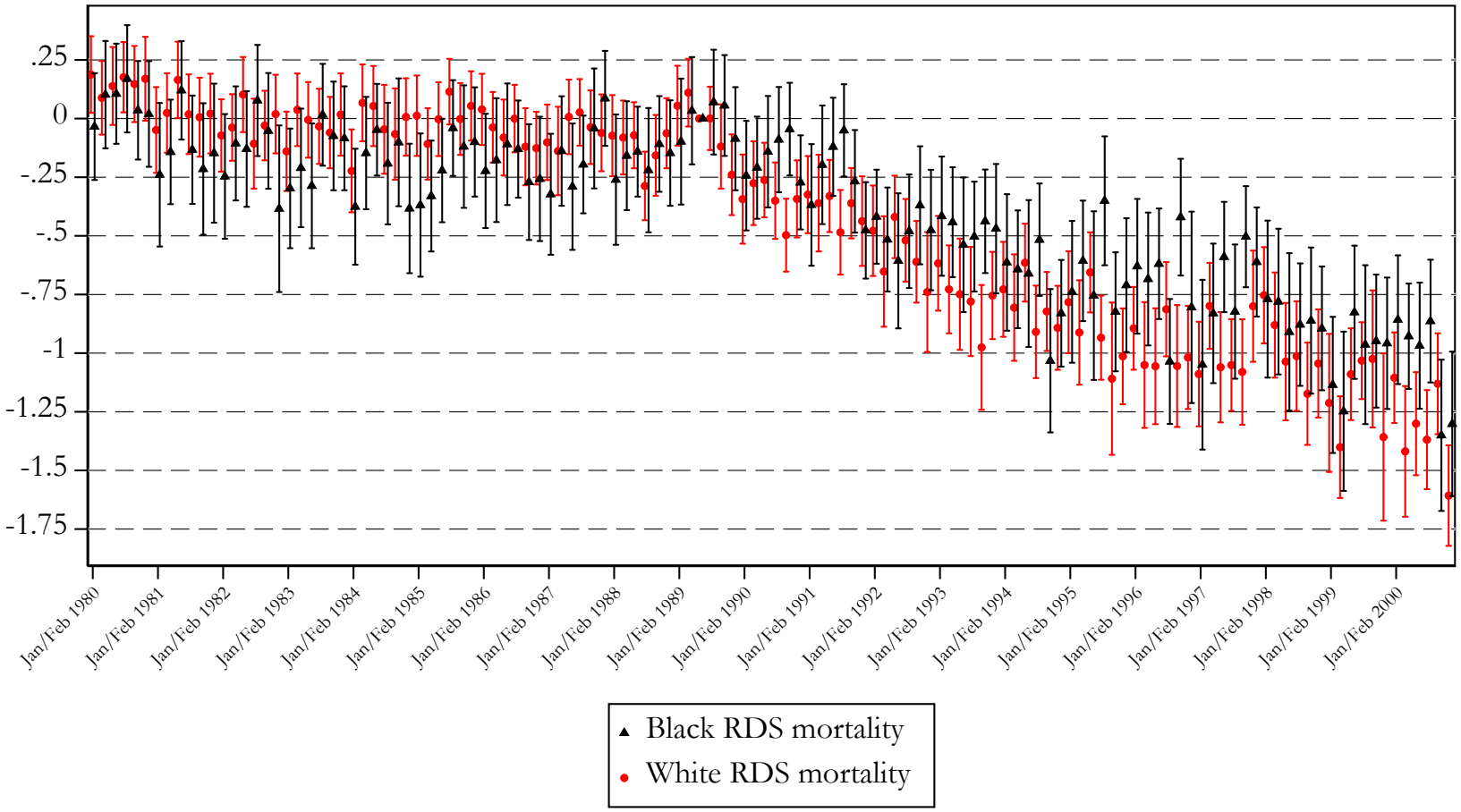
Figure 6. Surfactant Replacement Therapy and RDS Neonatal Mortality by Race: Monthly Event-study Analysis



Panel B. Black RDS Mortality



Panel C. Black and White RDS Mortality



Notes: Based on monthly data from the NVSS Multiple Cause-of-Death Mortality Files, 1980-2000. OLS event-study estimates (and their 90% confidence intervals) are reported, where the omitted category is May/June 1989. The dependent variable is the natural log of the race-specific RDS neonatal mortality rate for disease *d* in census division *c* and month *m*. All regressions include *Treated* (= 1 for RDS mortality, = 0 for non-respiratory-related mortality), census division fixed effects, and month fixed effects. Estimates are weighted by census division race-specific births. Robust standard errors are used to construct the confidence intervals.

Table 1. Surfactant Replacement Therapy and Neonatal Mortality by Race

	(1)	(2)
	Respiratory-related mortality	RDS mortality
Panel A. Whites		
Treated × *Post*	-0.725***	-0.911***
	(0.063)	(0.083)
Panel B. Blacks		
Treated × *Post*	-0.544***	-0.626***
	(0.058)	(0.069)
Panel C. Black-White ratio		
Treated × *Post*	0.156***	0.237***
	(0.021)	(0.032)
N	378	378

***Statistically significant at 1% level.

Notes: Based on annual data from the NVSS Multiple Cause-of-Death Mortality Files, 1980-2000. Estimates from a separate OLS regression are reported in each cell. In panels A and B, the dependent variable is the natural log of the race-specific neonatal mortality rate (i.e., deaths per 100,000 VLBW births) for disease *d* in census division *c* and year *t*. In panel C, the dependent variable is the natural log of the Black-White ratio of the neonatal mortality rate for disease *d* in census division *c* and year *t*. The variable *Post* is equal to 1 if $t \geq 1990$ and equal to 0 if $t < 1989$. It takes on the value 0.5 in 1989 because the FDA approved Exosurf in July of that year. All regressions include *Treated* (= 1 for respiratory-related/RDS mortality, = 0 for non-respiratory-related mortality), census division fixed effects, and year fixed effects. Estimates are weighted by census division White births, Black births, and total births in panels A, B, and C, respectively. Standard errors corrected for clustering at the disease-year level are in parentheses.

Table 2. Surfactant Replacement Therapy and Respiratory-related Neonatal Mortality by Race and Birthweight

	(1)	(2)	(3)	(4)	(5)
	Birthweight < 750g	Birthweight 750 to 999g	Birthweight 1,000 to 1,499g	Birthweight ≥ 1,500g	All neonates
Panel A. Whites					
Treated ×*Post*	-0.489***	-0.783***	-1.287***	-0.801***	-0.742***
	(0.071)	(0.081)	(0.104)	(0.062)	(0.075)
Pre-treatment mean	20,927	17,986	5,026	18.26	11,973
Panel B. Blacks					
Treated × *Post*	-0.447***	-0.553***	-0.925***	-0.546***	-0.548***
	(0.080)	(0.100)	(0.114)	(0.081)	(0.085)
Pre-treatment mean	18,492	10,596	2,239	23.42	9,207
Panel C. Black-White ratio					
Treated × *Post*	-0.010	0.163***	0.294***	0.309***	0.137***
	(0.021)	(0.032)	(0.068)	(0.058)	(0.021)
N	270	270	262	267	270

***Statistically significant at 1% level.

Notes: Based on annual mortality counts from the NVSS Linked Birth and Infant Death Files, 1983-1991 and 1995-2000. Estimates from a separate OLS regression are reported in each cell. In panels A and B, the dependent variable is the natural log of the race-specific neonatal mortality rate for disease *d* in census division *c* and year *t*. In panel C, the dependent variable is the natural log of the Black-White ratio of the neonatal mortality rate for disease *d* in census division *c* and year *t*. The variable *Post* is equal to 1 if $t \geq 1990$ and equal to 0 if $t < 1989$. It takes on the value 0.5 in 1989 because the FDA approved Exosurf in July of that year. All regressions include *Treated* (= 1 for respiratory-related mortality, = 0 for non-respiratory-related mortality), census division fixed effects, and year fixed effects. Estimates are weighted by census division White births, Black births, and total births in panels A, B, and C, respectively. Standard errors corrected for clustering at the disease-year level are in parentheses.

Table 3. Surfactant Replacement Therapy and Respiratory-related Neonatal Mortality by Race, Mother's Education, and Mother's Marital Status

	Mother's education			Mother's marital status	
	(1)	(2)	(3)	(4)	(5)
	< 4 years of HS	= 4 years of HS	> 4 years of HS	Married	Unmarried
Panel A. Whites					
Treated ×*Post*	-0.737***	-0.680***	-0.754***	-0.763***	-0.693***
	(0.078)	(0.085)	(0.072)	(0.081)	(0.071)
Pre-treatment mean	12,197	11,684	11,524	12,114	11,539
Panel B. Blacks					
Treated × *Post*	-0.540***	-0.515***	-0.576***	-0.616***	-0.525***
	(0.091)	(0.092)	(0.094)	(0.094)	(0.085)
Pre-treatment mean	9,060	9,021	9,444	9,628	9,008
Panel C. Black-White ratio					
Treated × *Post*	0.129***	0.121***	0.153***	0.124***	0.077**
	(0.044)	(0.039)	(0.035)	(0.030)	(0.037)
N	265	270	268	270	270

Statistically significant at 5% level; * at 1% level.

Notes: Based on annual mortality counts from the NVSS Linked Birth and Infant Death Files, 1983-1991 and 1995-2000. Estimates from a separate OLS regression are reported in each cell. In panels A and B, the dependent variable is the natural log of the race-specific neonatal mortality rate for disease *d* in census division *c* and year *t*. In panel C, the dependent variable is the natural log of the Black-White ratio of the neonatal mortality rate for disease *d* in census division *c* and year *t*. The variable *Post* is equal to 1 if $t \geq 1990$ and equal to 0 if $t < 1989$. It takes on the value 0.5 in 1989 because the FDA approved Exosurf in July of that year. All regressions include *Treated* (= 1 for respiratory-related mortality, = 0 for non-respiratory-related mortality), census division fixed effects, and year fixed effects. Estimates are weighted by census division White births, Black births, and total births in panels A, B, and C, respectively. Standard errors corrected for clustering at the disease-year level are in parentheses.

Table 4. Surfactant Replacement Therapy and Respiratory-related Neonatal Mortality by Race and NICU Access

	(1) NICU within 25 miles	(2) No NICU within 25 miles	(3) High-level NICU within 25 miles	(4) No high-level NICU within 25 miles	(5) NICU in county	(6) No NICU in county	(7) High-level NICU in county	(8) No high-level NICU in county	(9) All neonates
Panel A. Whites									
Treated ×*Post*	-0.289***	-0.267***	-0.304***	-0.255***	-0.317***	-0.220***	-0.330***	-0.247***	-0.285***
	(0.026)	(0.084)	(0.034)	(0.048)	(0.031)	(0.062)	(0.039)	(0.039)	(0.026)
Panel B. Blacks									
Treated × *Post*	-0.083*	-0.106	-0.061	-0.189	-0.093**	-0.066	-0.051	-0.179*	-0.088*
	(0.047)	(0.161)	(0.042)	(0.136)	(0.044)	(0.161)	(0.044)	(0.106)	(0.050)
Panel C. Black-White ratio									
Treated × *Post*	0.206***	0.161	0.243***	0.066	0.224***	0.154	0.278***	0.068	0.196***
	(0.039)	(0.159)	(0.042)	(0.151)	(0.038)	(0.157)	(0.050)	(0.110)	(0.050)
N	72	72	72	72	72	72	72	72	72

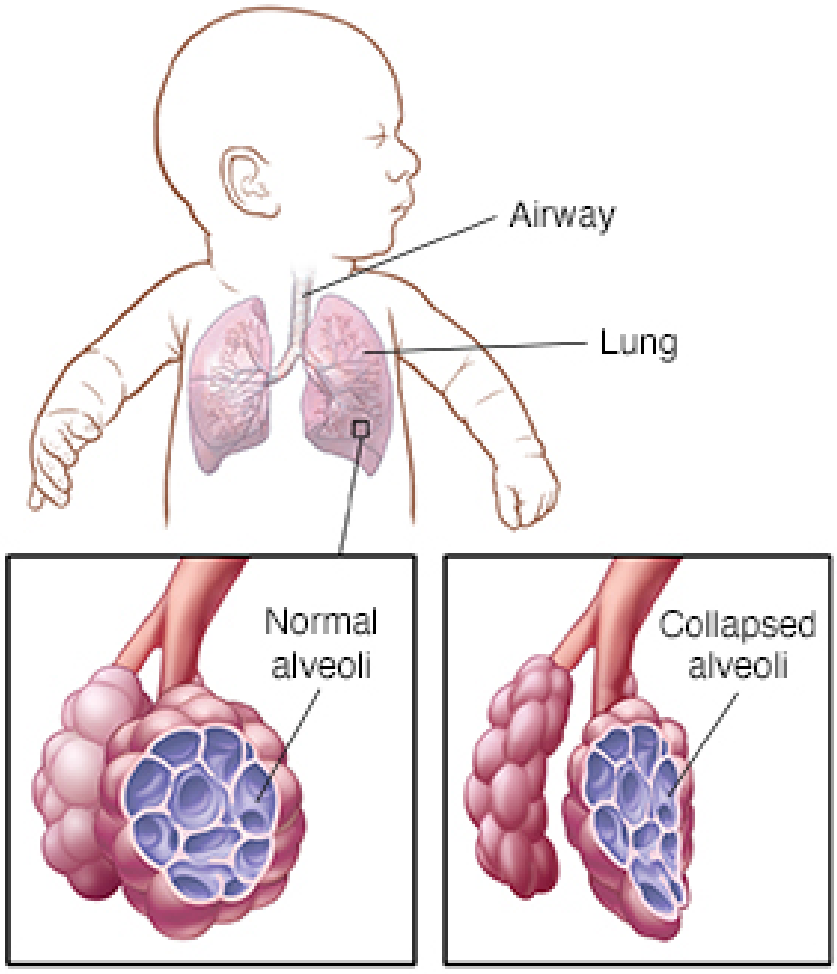
*Statistically significant at 10% level; ** at 5% level; *** at 1% level.

Notes: Based on monthly mortality counts from the restricted-use NVSS Linked Birth and Infant Death Files, 1989-1991. Estimates from a separate OLS regression are reported in each cell. In panels A and B, the dependent variable is the natural log of the race-specific neonatal mortality rate for disease *d* in month *m*. In panel C, the dependent variable is the natural log of the Black-White ratio of the neonatal mortality rate for disease *d* in month *m*. The variable *Post* is equal to 1 if $m \geq$ July 1989 and equal to 0 if $m <$ July 1989. All regressions include *Treated* (= 1 for respiratory-related mortality, = 0 for non-respiratory-related mortality) and month fixed effects. Robust standard errors are in parentheses.

Appendix A

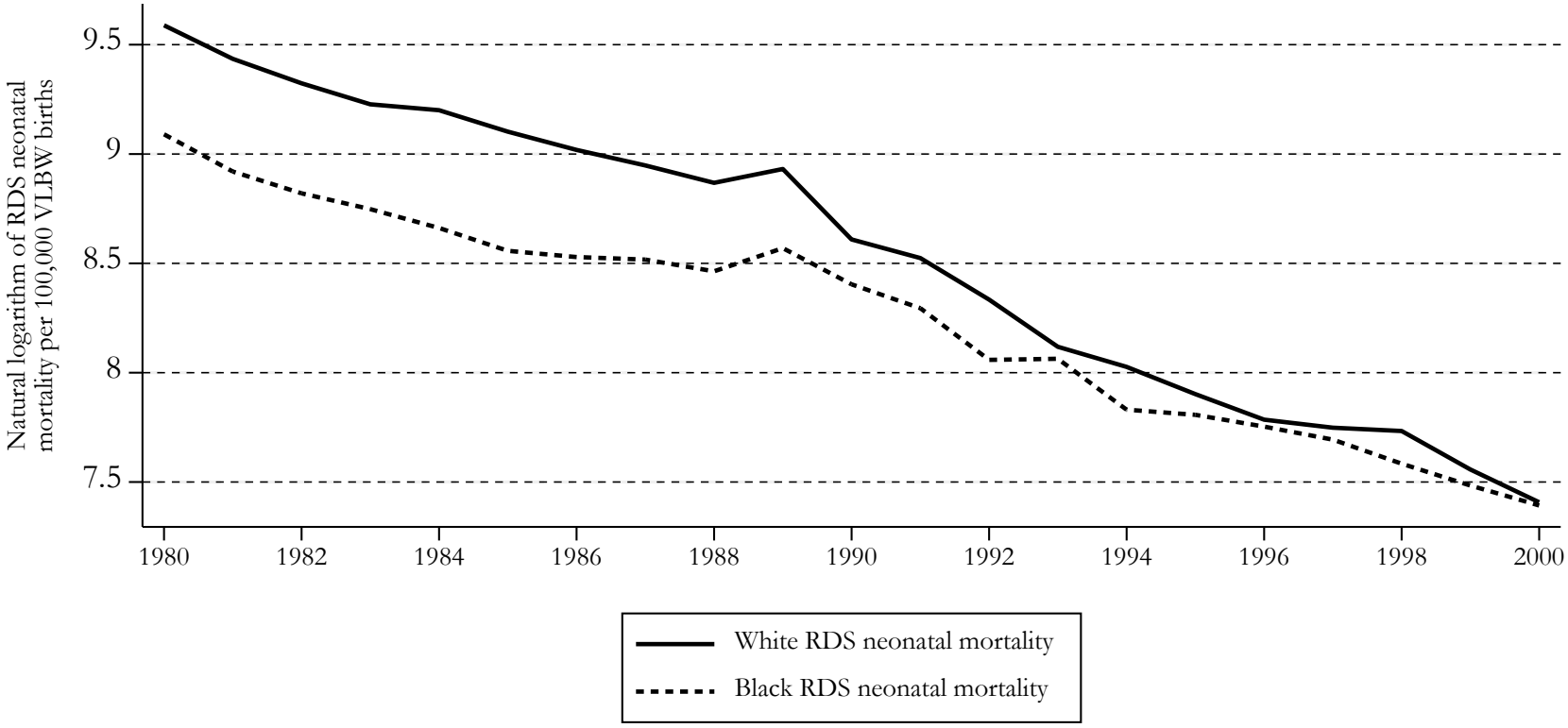
For Online Publication

Appendix Figure A1. Respiratory Distress Syndrome: Normal Versus Collapsed Alveoli



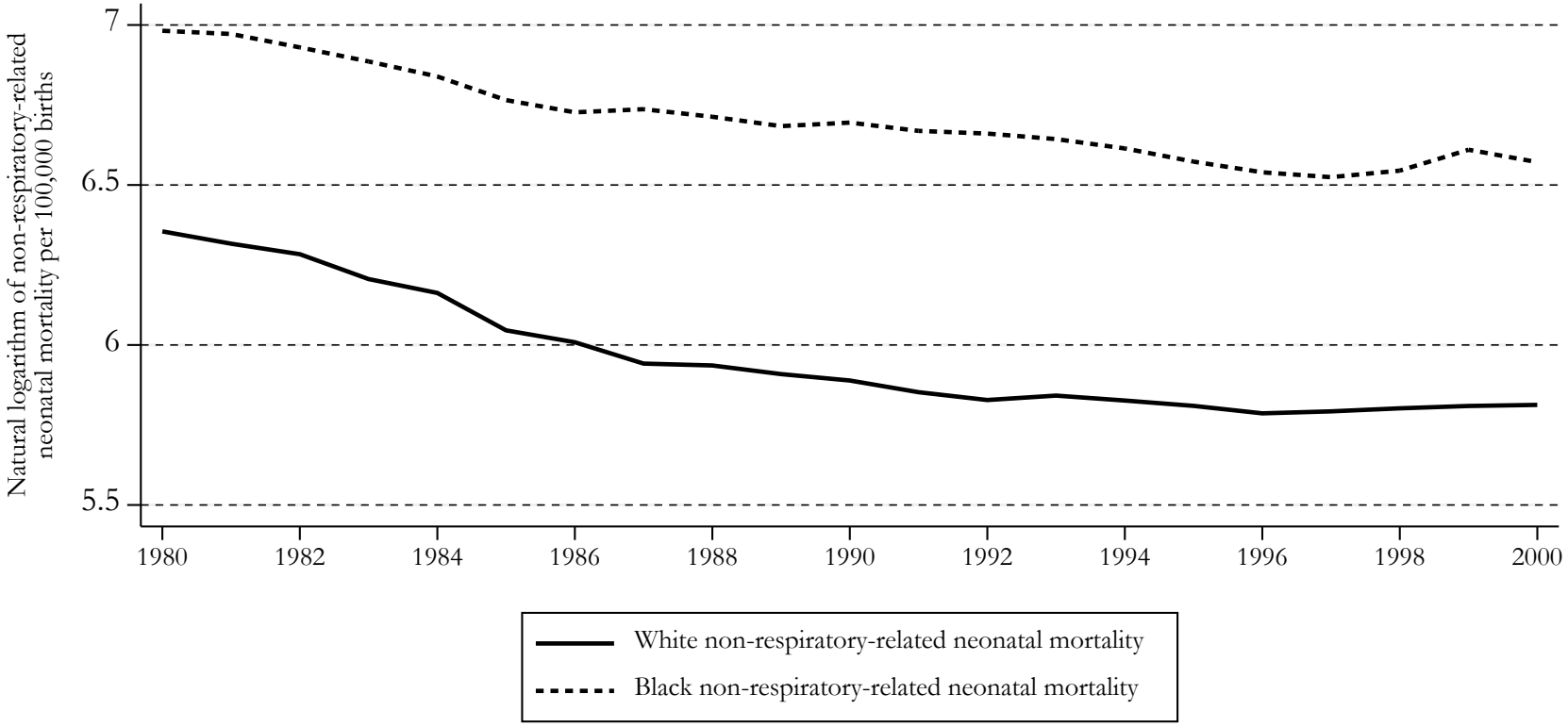
Notes: Alveoli are small air sacs in the lungs that lie at the end of the bronchioles. Their purpose is to facilitate gas exchange between the lungs and the blood. Surfactant deficiency leads to alveolar collapse and compromised pulmonary function. Source: https://bravebeginnings.org/respiratory-distress-in-infants-and-adults/.

Appendix Figure A2. RDS Neonatal Mortality by Race, 1980-2000



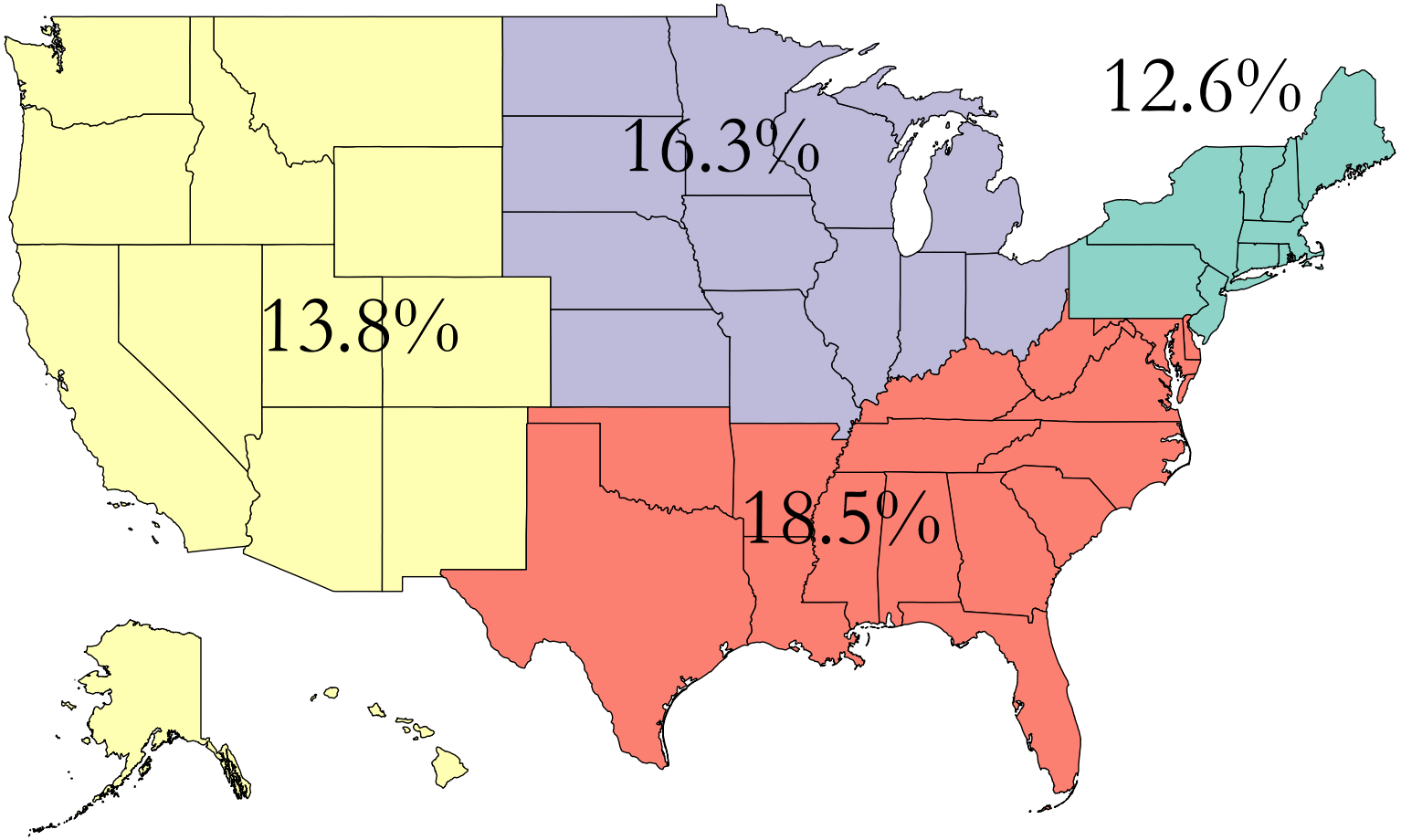
Notes: Based on annual data from the Multiple Cause-of-Death Mortality Files, published by the National Vital Statistics System.

Appendix Figure A3. Non-respiratory-related Neonatal Mortality by Race, 1980-2000



Notes: Based on annual data from the Multiple Cause-of-Death Mortality Files, published by the National Vital Statistics System.

Appendix Figure A4. Surfactant Replacement Therapy and the Black-White Neonatal Respiratory-related Mortality Ratio by Region



Notes: Based on annual data from the NVSS Multiple Cause-of-Death Mortality Files, 1980-2000. Percent changes shown on map are based on four separate estimates of β_1 from equation (1). The dependent variable is the natural log of the Black-White ratio of the neonatal mortality rate for disease *d* in census division *c* and year *t*. All regressions include *Treated*, census division fixed effects, and year fixed effects. Estimates are weighted by census division total births and standard errors are corrected for clustering at the disease-year level. All estimates are statistically significant at the 1 percent level.

Appendix Table A1. International Classification of Disease (ICD) Codes Used to Generate Respiratory-related and RDS Neonatal Mortality Rates

Cause of death	ICD codes, 9th revision (1980-1998)	ICD codes, 10th revision (1999-2000)
Respiratory-related	Respiratory distress syndrome (769)	Respiratory distress syndrome of newborn (P220)
	Pulmonary interstitial emphysema and related conditions (7702)	Other respiratory distress of newborn (P228)
	Pulmonary hemorrhage (7703)	Respiratory distress of newborn, unspecified (P229)
	Primary atelectasis (7704)	Interstitial emphysema originating in the perinatal period (P250)
	Chronic respiratory disease arising in the perinatal period (7707)	Pneumothorax originating in the perinatal period (P251)
	Other respiratory problems after birth (7708)	Pneumomediastinum originating in the perinatal period (P252)
		Pneumopericardium originating in the perinatal period (P253)
		Other conditions related to interstitial emphysema originating in the perinatal period (P258)
		Tracheobronchial hemorrhage originating in the perinatal period (P260)
		Massive pulmonary hemorrhage originating in the perinatal period (P261)
		Other pulmonary hemorrhage originating in the perinatal period (P268)
		Unspecified pulmonary hemorrhage originating in the perinatal period (P269)
		Wilson-Mikity syndrome (P270)
		Bronchopulmonary dysplasia originating in the perinatal period (P271)
		Other chronic respiratory diseases originating in the perinatal period (P278)
		Unspecified chronic respiratory disease originating in the perinatal period (P279)
		Primary atelectasis of newborn (P280)
		Cyanotic attacks of newborn (P282)
		Primary sleep apnea of newborn (P283)
		Other apnea of newborn (P284)
		Respiratory failure of newborn (P285)
		Other specified respiratory conditions of newborn (P288)
		Respiratory condition of newborn, unspecified (P289)
RDS	Respiratory distress syndrome (769)	Respiratory distress syndrome of newborn (P220)
		Other respiratory distress of newborn (P228)
		Respiratory distress of newborn, unspecified (P229)

Notes: ICD codes are in parentheses. While ICD-10 codes were not officially adopted in the United States until 2015 (Hirsch et al. 2016), the National Vital Statistics System switched from reporting ICD-9 to ICD-10 codes in 1999. Respiratory-related causes of death were defined based on Hamvas et al. (1996).

Appendix Table A2. Descriptive Statistics for Neonatal Mortality Outcomes, 1980-2000

	Mean, 1980-1988 (SD)	Mean, 1989-2000 (SD)	Description
Treated outcomes			
White respiratory-related mortality	16,673	6,727	White respiratory-related neonatal deaths per 100,000 VLBW White
	(3,510)	(2,689)	births in census division *c* and year *t*.
Black respiratory-related mortality	11,449	5,857	Black respiratory-related neonatal deaths per 100,000 VLBW Black
	(2,494)	(2,066)	births in census division *c* and year *t*.
Black-White respiratory-related mortality ratio	0.690	0.850	Black-White ratio of the respiratory-related neonatal mortality rate in
	(0.093)	(0.166)	census division *c* and year *t*.
White RDS mortality	9,963	3,510	White RDS neonatal deaths per 100,000 VLBW White births in census
	(2,789)	(1,815)	division *c* and year *t*.
Black RDS mortality	6,058	2,919	Black RDS neonatal deaths per 100,000 VLBW Black births in census
	(1,819)	(1,239)	division *c* and year *t*.
Black-White RDS mortality ratio	0.609	0.819	Black-White ratio of the RDS neonatal mortality rate in census division
	(0.115)	(0.225)	*c* and year *t*.
Control outcomes			
White non-respiratory-related mortality	465.6	340.8	White non-respiratory-related neonatal deaths per 100,000 White births
	(90.28)	(27.27)	in census division *c* and year *t*.
Black non-respiratory-related mortality	931.5	745.7	Black non-respiratory-related neonatal deaths per 100,000 Black births
	(159.3)	(115.5)	in census division *c* and year *t*.
Black-White non-respiratory-related mortality ratio	1.983	2.092	Black-White ratio of the non-respiratory-related neonatal mortality rate
	(0.212)	(0.257)	in census division *c* and year *t*.

Notes: Weighted means are reported (with standard deviations in parentheses). Based on annual data from the NVSS Multiple Cause-of-Death Mortality Files. See Appendix Table A1 for the cause of death codes used to generate respiratory-related and RDS mortality.

Appendix Table A3. Surfactant Replacement Therapy and White Mortality: Sensitivity Analysis

	(1)	(2)
	Respiratory-related mortality	RDS mortality
Panel A. Baseline estimates from Table 1		
Treated × *Post*	-0.725***	-0.911***
	(0.063)	(0.083)
Panel B. Weight by census division VLBW White births		
Treated × *Post*	-0.744***	-0.932***
	(0.063)	(0.083)
Panel C. Unweighted		
Treated × *Post*	-0.741***	-0.933***
	(0.062)	(0.082)
Panel D. Congenital anomalies as control disease		
Treated × *Post*	-0.649***	-0.834***
	(0.050)	(0.072)
Panel E. Influenza as control disease		
Treated × *Post*	-0.459***	-0.644***
	(0.038)	(0.052)
Panel F. Define mortality rate as infant deaths per 100,000 LBW births		
Treated × *Post*	-0.558***	-0.762***
	(0.039)	(0.060)
Panel G. White neonatal mortality in levels		
Treated × *Post*	-10,393***	-6,714***
	(786.0)	(615.1)
Pre-treatment mean	16,673	9,963

***Statistically significant at 1% level.

Notes: Based on annual data from the NVSS Multiple Cause-of-Death Mortality Files, 1980-2000. Estimates from a separate OLS regression are reported in each cell. In panels A-E, the dependent variable is the natural log of the White neonatal mortality rate (i.e., deaths per 100,000 VLBW births) for disease *d* in census division *c* and year *t*. In panel F, the dependent variable is the natural log of White infant deaths per 100,000 LBW births for disease *d* in census division *c* and year *t*. In panel G, the dependent variable is the White neonatal mortality rate for disease *d* in census division *c* and year *t*. The variable *Post* is equal to 1 if $t \geq 1990$ and equal to 0 if $t < 1989$. It takes on the value 0.5 in 1989 because the FDA approved Exosurf in July of that year. All regressions include *Treated*, census division fixed effects, and year fixed effects. Unless noted otherwise, estimates are weighted by census division White births. Standard errors corrected for clustering at the disease-year level are in parentheses.

Appendix Table A4. Surfactant Replacement Therapy and Black Mortality: Sensitivity Analysis

	(1)	(2)
	Respiratory-related mortality	RDS mortality
Panel A. Baseline estimates from Table 1		
Treated × *Post*	-0.544***	-0.626***
	(0.058)	(0.069)
Panel B. Weight by census division VLBW Black births		
Treated × *Post*	-0.544***	-0.622***
	(0.058)	(0.069)
Panel C. Unweighted		
Treated × *Post*	-0.584***	-0.718***
	(0.064)	(0.076)
Panel D. Congenital anomalies as control disease		
Treated × *Post*	-0.432***	-0.514***
	(0.042)	(0.056)
Panel E. Influenza as control disease		
Treated × *Post*	-0.161***	-0.243***
	(0.037)	(0.036)
Panel F. Define mortality rate as infant deaths per 100,000 LBW births		
Treated × *Post*	-0.382***	-0.505***
	(0.048)	(0.059)
Panel G. Black neonatal mortality in levels		
Treated × *Post*	-5,791***	-3,183***
	(509.0)	(353.2)
Pre-treatment mean	11,449	6,058

***Statistically significant at 1% level.

Notes: Based on annual data from the NVSS Multiple Cause-of-Death Mortality Files, 1980-2000. Estimates from a separate OLS regression are reported in each cell. In panels A-E, the dependent variable is the natural log of the Black neonatal mortality rate (i.e., deaths per 100,000 VLBW births) for disease *d* in census division *c* and year *t*. In panel F, the dependent variable is the natural log of Black infant deaths per 100,000 LBW births for disease *d* in census division *c* and year *t*. In panel G, the dependent variable is the Black neonatal mortality rate for disease *d* in census division *c* and year *t*. The variable *Post* is equal to 1 if $t \geq 1990$ and equal to 0 if $t < 1989$. It takes on the value 0.5 in 1989 because the FDA approved Exosurf in July of that year. All regressions include *Treated*, census division fixed effects, and year fixed effects. Unless noted otherwise, estimates are weighted by census division Black births. Standard errors corrected for clustering at the disease-year level are in parentheses.

Appendix Table A5. Surfactant Replacement Therapy and the Black-White Mortality Ratio: Sensitivity Analysis

	(1)	(2)
	Respiratory-related mortality	RDS mortality
Panel A. Baseline estimates from Table 1		
Treated × *Post*	0.156***	0.237***
	(0.021)	(0.032)
Panel B. Weight by census division VLBW Black births		
Treated × *Post*	0.152***	0.246***
	(0.019)	(0.027)
Panel C. Unweighted		
Treated × *Post*	0.156***	0.215***
	(0.027)	(0.039)
Panel D. Congenital anomalies as control disease		
Treated × *Post*	0.200***	0.282***
	(0.019)	(0.029)
Panel E. Influenza as control disease		
Treated × *Post*	0.215***	0.296***
	(0.038)	(0.045)
Panel F. Define mortality rate as infant deaths per 100,000 LBW births		
Treated × *Post*	0.123***	0.182***
	(0.021)	(0.022)
Panel G. Black-White neonatal mortality difference in levels		
Treated × *Post*	4,331***	3,363***
	(302.4)	(262.6)
Pre-treatment mean of Black-White difference	-5,177	-3,881

***Statistically significant at 1% level.

Notes: Based on annual data from the NVSS Multiple Cause-of-Death Mortality Files, 1980-2000. Estimates from a separate OLS regression are reported in each cell. In panels A-E, the dependent variable is the natural log of the Black-White ratio of the neonatal mortality rate for disease *d* in census division *c* and year *t*. In panel F, the dependent variable is the natural log of the Black-White ratio of the infant mortality rate for disease *d* in census division *c* and year *t*. In panel G, the dependent variable is the difference between the Black and White neonatal mortality rates for disease *d* in census division *c* and year *t*. The variable *Post* is equal to 1 if $t \geq 1990$ and equal to 0 if $t < 1989$. It takes on the value 0.5 in 1989 because the FDA approved Exosurf in July of that year. All regressions include *Treated*, census division fixed effects, and year fixed effects. Unless noted otherwise, estimates are weighted by census division total births. Standard errors corrected for clustering at the disease-year level are in parentheses.

Appendix Table A6. Surfactant Replacement Therapy and RDS Neonatal Mortality by Race and Birthweight

	(1)	(2)	(3)	(4)	(5)
	Birthweight < 750g	Birthweight 750 to 999g	Birthweight 1,000 to 1,499g	Birthweight ≥ 1,500g	All neonates
Panel A. Whites					
Treated ×*Post*	-0.394***	-0.907***	-1.531***	-1.424***	-0.851***
	(0.066)	(0.105)	(0.151)	(0.158)	(0.089)
Pre-treatment mean	9,049	11,878	3,739	11.36	6,821
Panel B. Blacks					
Treated × *Post*	-0.275***	-0.647***	-1.010***	-0.780***	-0.536***
	(0.065)	(0.135)	(0.134)	(0.138)	(0.086)
Pre-treatment mean	7,618	6,958	1,625	12.92	4,660
Panel C. Black-White ratio					
Treated × *Post*	0.056**	0.199***	0.414***	0.635***	0.243***
	(0.026)	(0.042)	(0.061)	(0.086)	(0.025)
N	270	265	251	238	270

Statistically significant at 5% level; * at 1% level.

Notes: Based on annual mortality counts from the NVSS Linked Birth and Infant Death Files, 1983-1991 and 1995-2000. Estimates from a separate OLS regression are reported in each cell. In panels A and B, the dependent variable is the natural log of the race-specific neonatal mortality rate for disease *d* in census division *c* and year *t*. In panel C, the dependent variable is the natural log of the Black-White ratio of the neonatal mortality rate for disease *d* in census division *c* and year *t*. The variable *Post* is equal to 1 if $t \geq 1990$ and equal to 0 if $t < 1989$. It takes on the value 0.5 in 1989 because the FDA approved Exosurf in July of that year. All regressions include *Treated* (= 1 for RDS mortality, = 0 for non-respiratory-related mortality), census division fixed effects, and year fixed effects. Estimates are weighted by census division White births, Black births, and total births in panels A, B, and C, respectively. Standard errors corrected for clustering at the disease-year level are in parentheses.

Appendix Table A7. Surfactant Replacement Therapy and RDS Neonatal Mortality by Race, Mother's Education, and Mother's Marital Status

	Mother's education			Mother's marital status	
	(1)	(2)	(3)	(4)	(5)
	< 4 years of HS	= 4 years of HS	> 4 years of HS	Married	Unmarried
Panel A. Whites					
Treated ×*Post*	-0.920***	-0.781***	-0.894***	-0.886***	-0.781***
	(0.105)	(0.099)	(0.099)	(0.098)	(0.083)
Pre-treatment mean	7,180	6,773	6,461	6,930	6,484
Panel B. Blacks					
Treated × *Post*	-0.563***	-0.514***	-0.535***	-0.633***	-0.515***
	(0.102)	(0.102)	(0.087)	(0.096)	(0.090)
Pre-treatment mean	4,819	4,612	4,583	4,790	4,599
Panel C. Black-White ratio					
Treated × *Post*	0.229***	0.238***	0.309***	0.235***	0.154***
	(0.047)	(0.044)	(0.039)	(0.038)	(0.043)
N	258	267	265	270	270

***Statistically significant at 1% level.

Notes: Based on annual mortality counts from the NVSS Linked Birth and Infant Death Files, 1983-1991 and 1995-2000. Estimates from a separate OLS regression are reported in each cell. In panels A and B, the dependent variable is the natural log of the race-specific neonatal mortality rate for disease *d* in census division *c* and year *t*. In panel C, the dependent variable is the natural log of the Black-White ratio of the neonatal mortality rate for disease *d* in census division *c* and year *t*. The variable *Post* is equal to 1 if $t \geq 1990$ and equal to 0 if $t < 1989$. It takes on the value 0.5 in 1989 because the FDA approved Exosurf in July of that year. All regressions include *Treated* (= 1 for RDS mortality, = 0 for non-respiratory-related mortality), census division fixed effects, and year fixed effects. Estimates are weighted by census division White births, Black births, and total births in panels A, B, and C, respectively. Standard errors corrected for clustering at the disease-year level are in parentheses.

Appendix Table A8. Surfactant Replacement Therapy and RDS Neonatal Mortality by Race and NICU Access

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
	NICU within 25 miles	No NICU within 25 miles	High-level NICU within 25 miles	No high-level NICU within 25 miles	NICU in county	No NICU in county	High-level NICU in county	No high-level NICU in county	All neonates
Panel A. Whites									
Treated ×*Post*	-0.325***	-0.272**	-0.357***	-0.247***	-0.372***	-0.200**	-0.405***	-0.234***	-0.314***
	(0.050)	(0.105)	(0.064)	(0.064)	(0.050)	(0.089)	(0.065)	(0.057)	(0.042)
Panel B. Blacks									
Treated × *Post*	-0.125*	-0.199	-0.108*	-0.248**	-0.139**	-0.135	-0.103	-0.235***	-0.135**
	(0.063)	(0.152)	(0.059)	(0.092)	(0.058)	(0.118)	(0.063)	(0.070)	(0.055)
Panel C. Black-White ratio									
Treated × *Post*	0.200***	0.074	0.249***	-0.001	0.234***	0.065	0.303***	-0.001	0.178***
	(0.056)	(0.124)	(0.071)	(0.093)	(0.053)	(0.114)	(0.068)	(0.075)	(0.053)
N	72	72	72	72	72	72	72	72	72

*Statistically significant at 10% level; ** at 5% level; *** at 1% level.

Notes: Based on monthly mortality counts from the restricted-use NVSS Linked Birth and Infant Death Files, 1989-1991. Estimates from a separate OLS regression are reported in each cell. In panels A and B, the dependent variable is the natural log of the race-specific neonatal mortality rate for disease *d* in month *m*. In panel C, the dependent variable is the natural log of the Black-White ratio of the neonatal mortality rate for disease *d* in month *m*. The variable *Post* is equal to 1 if $m \geq$ July 1989 and equal to 0 if $m <$ July 1989. All regressions include *Treated* (= 1 for respiratory-related mortality, = 0 for non-respiratory-related mortality) and month fixed effects. Robust standard errors are in parentheses.

Appendix B

For Online Publication

APPENDIX B. DESCRIPTION OF DATA USED TO DETERMINE NICU ACCESS

To determine the location of NICUs, we purchased *Annual Survey of Hospitals* data for the period 1980-1990, produced by the American Hospital Association. Each year, hospitals were asked whether neonatal intensive care services were available in their facility, and these questions were consistent over the period 1980-1990. Hospitals providing these services were asked how many beds were set up and staffed in these units. Because the FDA approved SRT in 1989, we used the presence of a NICU in 1988 to determine a mother's access. High-level NICUs were defined as those with more than 15 beds "set up and staffed in 'neonatal intensive care' and 'neonatal intermediate care' units" (Baker and Phibbs 2002, p. 545). Importantly, the *Annual Survey of Hospitals* also contains separate indicators for pediatric intensive care units, newborn nurseries, and premature nurseries, minimizing the risk that our NICU measure is misclassified.

As noted by Baker and Phibbs (2002), hospitals do not report consistently over time, leading to missing NICU information. To address this issue, we used the following definition of NICU adoption year and type proposed by Baker and Phibbs (2002, p. 545): "Adoption year is the first year in which the hospital says it has a unit, and the type of unit adopted is the type present in that adoption year." For example, if a hospital reported having a NICU in 1985 but had missing data for 1986-1990, we coded NICU = 1 in 1988. If the same hospital reported having 16 beds in 1985, it was also classified as a high-level NICU in 1988. According to Baker and Phibbs (2002, p. 545), "hospitals seldom if ever open full high-level units and then quickly scale them back."