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Racial/ethnic disparities in preterm birth: clues from environmental exposures

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Abstract

Purpose of review—Despite advances in medical care, preterm birth and its associated racial/ethnic disparities remain major public health issues. Environmental exposures may contribute to racial disparities in preterm birth.

Recent findings—Recent work in Iran demonstrated lead levels <10 µg/dl to be associated with preterm birth and premature rupture of membranes. Data on air pollution are mixed. A study in California found exposure to nitric oxide species to be associated with preterm birth. However, results from large birth cohorts in the Netherlands found no association. Interestingly, a study in South Korea recently demonstrated that socioeconomic status modifies the association between air pollution and preterm birth. A recent promising study randomized minority pregnant women in Washington DC to cognitive behavioral therapy vs. usual care to decrease exposure to environmental tobacco smoke (ETS). The investigators reported reductions in ETS exposure and the risk of very preterm birth.

Summary—Clues about potential mechanisms underlying disparities in preterm birth can be gained from exploring differences in environmental exposures. Investigators should include environmental variables when studying birth outcomes. Such efforts should result in targeted interventions to decrease the incidence of preterm birth and its disparities.

Keywords

preterm; disparities; environment; lead; air pollution

Introduction

In 2006, 12.8% of all infants were born preterm (<37 weeks' gestation) contributing to 36.1% of all infant deaths in the US [1, 2]. Preterm birth rates varied substantially by race/ethnicity from 7.4% among Non-Hispanic white infants to 17.2% for Non-Hispanic black infants. The preterm-related infant mortality rate among Non-Hispanic black infants (6.01

per 1,000 live births) more than triples the preterm-related infant mortality rate among Non-Hispanic white infants (1.79 per 1,000). Most epidemiologic studies addressing disparities in preterm birth control for demographic and socioeconomic variables. Smoking, long recognized as a risk factor for preterm birth [3], represents the only environmental risk factor routinely included in these studies. However, recent studies reveal that other environmental toxicants such as lead [4*] and air pollution [5, 6*] may contribute to preterm birth. Because minority status and poverty often track with higher risks of toxic environmental exposures, addressing environmental risk factors may mitigate disparities in birth outcomes.

Disparities in preterm birth

Preterm birth, defined as delivery before 37 completed weeks of gestation (<37 weeks from the last menstrual period), occurs for multiple reasons including preeclampsia, fetal growth restriction, premature rupture of membranes, placental abruption, preterm labor, and cervical incompetence. These risk factors have upstream causes which include environmental chemicals. In addition to the greater risk of mortality, preterm birth infants are at risk for acute respiratory, gastrointestinal and immunologic/infectious and neurologic morbidities as well as long term motor, cognitive and sensory handicaps. The annual economic cost to society of premature birth in the U.S, was estimated to be \$26.2 billion in 2005 [7].

Infants born at the earliest gestations experience the highest rates of morbidity and mortality. The National Institute of Child Health and Human Development (NICHD) Neonatal Research Network reported that infant survival rates at discharge from the hospital from 2003 to 2007 ranged from 6% to 95% at 22 and 28 weeks' gestation respectively [8*]. Non-Hispanic black infants disproportionately bear the burden of extreme prematurity (<28 weeks' gestation) (Figure 1). In the US in 2006, births <28 weeks' gestation accounted for 0.76% of the 4.3 million births [1]. While black infants accounted for 15% of all births, they accounted for 35% of births <28 weeks' gestation. Non-Hispanic black infants were more than 3 times more likely to be born at <28 weeks' than their Non-Hispanic white counterparts (1.8% and 0.55% respectively).

Multiple hypotheses and explanations for racial/ethnic disparities in preterm birth have been proposed. These most often cite socioeconomic and demographic differences between black and white mothers: maternal age, educational attainment, household income and marital status. However, even when only low risk mothers in Illinois are analyzed disparities by race persist [9]. In one study, black women with a lifelong residence in high income Chicago neighborhoods still had a two-fold greater preterm birth rate and a six-fold increased rate of very low birth weight (<1500g) than their white counterparts [10]. Equal access to prenatal care does not eliminate these disparities either, as demonstrated by studies among women with comparable health insurance [11] and among women in the military [12] which demonstrate differential risk by race.

Biologic mechanisms such as genetic variation and infection/inflammation have been proposed as explanations for disparities in preterm birth. It is difficult to conceive how genetics in the absence of environmental interactions could produce such disparities, as this would be counter to accepted evolutionary theory which would posit genetic variants that cause pre-term delivery to be reduced over time rather than increased. Indeed, most studies have not supported genetic variation as the underlying cause of disparities in preterm birth [13]. When infants born to African-born black mothers, US-born African American mothers and US-born white mothers were compared, infants born to African-born black mothers had birth weights more similar to white infants [9]. Furthermore, race has been shown to be more of a social construct than an genetic one as there is more within race variation in

genotype than between race variation [14]. Genitourinary tract colonization and infection have often been implicated in preterm birth since rates of infections such as bacterial vaginosis have been shown to be higher in black versus white women. However, black women without lower genital tract infection still have a higher risk of preterm delivery than their white counterparts [15].

Smoking status differences between white and black mothers have also been implicated in disparities in preterm delivery. However, while some studies report higher rates of smoking among black mothers [16], black non-smokers continue to have higher rates of preterm delivery than white smokers and non-smokers [17].

Lead exposure

While studies examining the association between lead exposure and preterm birth [18] have had mixed results [7], a recent prospective cohort study of 348 non-smoking pregnant women in Iran found that early pregnancy blood lead levels were significantly higher among women who delivered preterm compared to women who delivered at term (4.53 $\mu\text{g/dl}$ vs. 3.72 $\mu\text{g/dl}$ respectively) ($P<0.05$) [4*]. Women with higher lead levels had higher odds (1.41, 95% CI 1.08–1.84) of delivering preterm after adjusting for maternal age, education, passive smoking, pregnancy weight gain, parity, hematocrit, mode of delivery and infant sex. The reason for preterm delivery was not proposed in their study. The same Iranian cohort was studied to examine the association between lead exposure and premature rupture of membranes (PROM) [19]. Blood lead levels were significantly higher among women who subsequently developed PROM 4.61 $\mu\text{g/dl}$ vs. 3.69 $\mu\text{g/dl}$ ($P=0.03$). These studies highlight that low levels of lead ($<10 \mu\text{g/dl}$) may still affect reproductive outcomes, despite being in the “acceptable range.”

Air Pollution Exposure

Multiple studies have addressed the association between air pollution and preterm birth. Sulfur dioxide and particulate matter demonstrate the most consistent associations [7]. A recent study of residential exposure to traffic-generated air pollution in Southern California and 81,186 singleton births from 1997 to 2006 demonstrated that exposure to nitric oxide species were associated with increased odds of preterm delivery and very preterm delivery (<30 weeks) (adjusted interquartile odds ratio 1.06, 95% CI 1.03–1.15 and 1.25, 95% CI 1.17–1.33 respectively) [20]. A time-series analysis performed in Atlanta revealed that preterm birth rates were associated with exposure to NO_2 in the 6 weeks prior to delivery and $\text{PM}_{2.5}$ in the week prior to delivery [5].

In contrast, there have been two recent negative studies relating air pollution to preterm birth. Results from the Generation R Study, a population-based prospective pregnancy/birth cohort in the Netherlands of 7,339 pregnant women and their children delivered between 2002 and 2008, reported no association between residential proximity to traffic and preterm birth [21*]. Similarly, the results from the Dutch ABCD birth cohort study of 7600 singleton births revealed no increase in preterm birth among highly exposed women to NO_2 [22*].

Environmental tobacco smoke exposure

While active maternal smoking increases the risk of preterm delivery, the relationship between environmental tobacco smoke (ETS) and preterm delivery is unclear. A recent, extensive meta analysis (seventy-six articles and over 48,000 exposed women), of ETS and perinatal outcomes revealed that while birth weights were lower in exposed subjects, the duration of gestation was similar between exposed and unexposed (weighted mean difference 0.02 weeks) [23**]. The authors of this analysis admit its limitations include

inadequate accounting for preeclampsia which is crucial since active maternal smoking substantially decreases the risk of preeclampsia and thus may decrease the risk of medically indicated preterm birth [24]. It is not known whether this relationship exists for ETS exposure. There is some evidence associating ETS with the incidence of very preterm birth, but not overall gestational age [25]. Since preeclampsia generally presents later in gestation, it would not contribute substantially to the rate of very preterm birth (<28 weeks' gestation). Thus, this finding supports the hypothesis that ETS, like active smoking, decreases preeclampsia and by so doing may mask an inherent risk of ETS to increase spontaneous preterm birth.

A recent interventional randomized controlled trial of cognitive behavioral therapy (n=335) versus usual care (n=356) for minority pregnant mothers in Washington DC to reduce ETS exposure and preterm delivery found that such an intervention resulted in decreased odds of ETS exposure at the time of delivery (0.50, 95% CI 0.35–0.71) as well as decreased odds of very preterm birth (OR 0.22, 95% CI 0.07–0.68) [26**]. This study demonstrates the promise of environmental interventions to decrease disparities in preterm birth.

Disparities in Environmental Exposures

Environmental exposures often track with socioeconomic status and race/ethnicity. Lead exemplifies this phenomenon. The Centers for Disease Control and Prevention (CDC) tracks data from the National Health and Nutrition Examination Survey (NHANES). In the most recent report (2005) geometric mean blood levels differed significantly among Non-Hispanic white and Non-Hispanic black females; for girls ages 6–19 years, levels were 0.9 µg/dl and 1.3 µg/dl respectively and for women ages 20–59, levels were 1.2 and 1.4 µg/dl respectively [27].

Data on air pollution demonstrate similar disparities in exposure rates. A population-based study monitoring air pollution in 264 counties from all regions of the US and natality data from the National Center for Health Statistics analyzed over four million births and found that African American women experienced significantly higher mean levels of air pollution [28]. Additionally, the investigators reported higher odds of preterm delivery with increased levels of air pollution. However, adding air pollution to models for preterm birth did not attenuate the odds ratios for preterm births for African-American mothers compared with white mothers. They concluded that air pollution differences are unlikely to explain disparities in birth outcomes. Interactions between race and air pollution were not reported. A recent study from the Republic of Korea demonstrated that exposure to PM10 during the second trimester increased the risk of preterm delivery among women living in low income areas but not high income areas [6*]. It is possible that differences in diet, occupational exposures, stress or other factors may explain this heightened effect on poorer women and underlie this interaction with ambient pollution.

Linking Environmental Exposures and Preterm Birth Disparities

Overwhelmingly, the evidence supports that the social and physical environments contribute to disparities in preterm birth. The cumulative exposures that black mothers experience throughout their early lives are likely responsible for their increased risk of delivering preterm since circumstances in adulthood (military service, college education, and adequate prenatal care) do not eliminate the disparities. The increased risk of preterm delivery with increasing maternal age among black mothers differs from the increased risk of preterm delivery among young white mothers. This association has led to the “Weathering Hypothesis” stating that with advancing age and declining health status, black women accumulate negative experiences that worsen reproductive outcomes with age [29]. A recent study supported this hypothesis finding that with each 5-year increase in maternal age, black

non-smokers experienced increased odds of delivering preterm than younger black non-smokers (adjusted odds ratio 1.11, 95% CI 1.07–1.15) [17]. This was similar to rates of preterm birth among white smokers. Additionally, when NHANES data of lead levels were analyzed among women of childbearing age, differences between black and white women were more pronounced among older women, supporting a cumulative dose-effect of lead toxicity that may be partly responsible for disparities in preterm birth that are larger among older versus younger mothers [30].

A recent statistical analysis of Virginia Vital Records utilized siblings, half-siblings and children of twins to analyze genetic and environmental contributions to disparities in preterm birth [31**]. The statisticians created models accounting for fetal genetic makeup, maternal genetic makeup, shared environment and unique environment and then created stratified models by race. They found that the variance of gestational age among black infants was almost twice that of white infants, an unexpected finding. By analyzing kinship and residential factors, they reported that fetal genetic factors explained 35.2% of variability in gestational age among white infants and just 3.7% among black infants. Maternal genetic influences contributed to 13.8% and 13.4% of the gestational age variance for black and white infants respectively. Environmental factors explained 82.5% and 51.0% of the variance for black and white infants, respectively. These models controlled for birth order, maternal age, fetal sex, source of care, smoking and maternal education, suggesting that additional social/environmental factors are responsible for differences in gestational age.

The increased risk of preterm birth [32] and infant mortality [33] among black infants born to mothers residing in the most segregated cities and neighborhoods strongly supports the notion that environmental exposures contribute to disparities in preterm birth. Furthermore, Osypuk and Acevedo-Garcia demonstrated in an analysis of 1.9 million US births in 2000 that black-white disparities in preterm birth were not only larger in hypersegregated areas, but that this effect was even more pronounced among older black mothers [32]. This finding suggests a dose-effect of neighborhood exposures including environmental toxicants which is not surprising in light of the proximity of minority neighborhoods to industrial waste sites, factories, highways and other sources pollution[34].

Summary and future directions

In summary, environmental exposures may contribute to disparities in preterm birth and with the exception of smoking status, are omitted from current epidemiologic models of preterm birth and its causes. Much work remains if we are to further understand the root causes of these disparities. Longitudinal birth cohorts specifically designed to measure environmental toxicants and birth outcomes are needed to elucidate which exposures may be causally linked to preterm birth.

Once the causal links are clearer, there are two paths to be taken. If elimination of the toxicant is possible, public health endeavors akin to lead removal or environmental tobacco exposure avoidance must be undertaken at a massive scale to improve infant health outcomes. Additionally, biologic research into how toxicants trigger early labor, cervical incompetence, spontaneous rupture of membranes and medical conditions that lead to medically-indicated early induction of labor is needed to better understand and prevent preterm birth.

One potential mechanism is epigenetic variation. Epigenetic marks alter gene expression in the absence of DNA sequence variation. Understanding potential environmental effects on phenotype (including predisposition to preterm labor) through such mechanisms will lay the foundation for future work on interventions to mitigate the effects of toxic exposures through manipulation of epigenetic marks. While we are far from such interventions in

humans, work in animals is promising. Bisphenol A (BPA) exposure in mice has been shown to be counteracted by a high methyl-donor diet in pregnant mice [35]. Such intervention strategies may be employed in the future to improve human health outcomes including reducing the incidence of preterm birth and its racial/ethnic disparities.

Preterm birth is not just an American phenomenon. Worldwide there were over 12.9 million preterm births in 2005 [36]. Eleven million of these occurred in Africa and Asia where environmental exposures differ from the U.S. and are likely to be even more significant contributors to perinatal health. Improvements in infant mortality necessitate tackling preterm birth. Understanding the antecedents of preterm birth around the globe and the particular risk factors in differing regions can help direct public health efforts. This work requires collaborations among environmental scientists, maternal-child public health experts, epidemiologists, basic scientists, educators, clinicians and political leaders. If effective, the reward will be huge as disparities in preterm birth domestically and worldwide embody social and environmental injustices that affect our smallest, most vulnerable members of society.

Conclusion

Preterm birth disproportionately affects black families who also experience higher levels of environmental toxin exposure than white families in the United States. The extent to which exposures such as environmental tobacco smoke, lead and air pollution contribute to disparities in preterm birth is unknown, but should be evaluated in future epidemiologic studies.

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Abbreviations

ETS	environmental tobacco smoke
PROM	premature rupture of membranes
PM_{2.5}	Particulate matter <2.5 micrometers in diameter
PM₁₀	Particulate matter <10 micrometers in diameter
NHANES	National Health and Nutrition Examination Survey

References and recommended reading

Papers of particular interest, published within the annual period of review are highlighted as:

*Of special interest

**Of outstanding interest

1. Martin JA, Hamilton BE, Sutton PD, et al. Births: Final Data for 2006. Natl Vital Stat Rep. 2009; 57:1–102.
2. Martin JA, Hamilton BE, Sutton PD, Ventura SJ, Menacker F, Kirmeyer S, Mathews TJ. Births: final data for 2006. National Vital Statistics Reports. 2009; 57:1–102.
3. Savitz DA, Murnane P. Behavioral influences on preterm birth: a review. Epidemiology. 2010; 21:291–299. [PubMed: 20386169]

- *4. Vigeh M, Yokoyama K, Seyedaghamiri Z, et al. Blood lead at currently acceptable levels may cause preterm labour. *Occup Environ Med*. 2010 This prospective study of over 300 pregnant women in Iran demonstrates that even slight elevations of lead levels (<10 µg/dl) may still affect reproductive outcomes.
5. Darrow LA, Klein M, Flanders WD, et al. Ambient air pollution and preterm birth: a time-series analysis. *Epidemiology*. 2009; 20:689–698. [PubMed: 19478670]
- *6. Yi O, Kim H, Ha E. Does area level socioeconomic status modify the effects of PM(10) on preterm delivery? *Environ Res*. 2009; 110:55–61. This Korean study reveals a potential interaction between socioeconomic status and vulnerability to the effects of air pollution on birth outcomes. [PubMed: 19878932]
7. Behrman RE, Butler AS, Institute of Medicine (U.S.). Preterm birth: causes, consequences, and prevention. Washington, D.C: National Academies Press; 2007. Committee on Understanding Premature Birth and Assuring Healthy Outcomes.
- *8. Stoll BJ, Hansen NI, Bell EF, et al. Neonatal outcomes of extremely preterm infants from the NICHD Neonatal Research Network. *Pediatrics*. 2010; 126:443–456. This NICHD Neonatal Research Network study provides the most recent outcomes data among very extremely preterm infants. [PubMed: 20732945]
9. David RJ, Collins JW Jr. Differing birth weight among infants of U.S.-born blacks, African-born blacks, and U.S-born whites. *N Engl J Med*. 1997; 337:1209–1214. [PubMed: 9337381]
10. Collins JW Jr, David RJ, Simon DM, Prachand NG. Preterm birth among African American and white women with a lifelong residence in high-income Chicago neighborhoods: an exploratory study. *Ethn Dis*. 2007; 17:113–117. [PubMed: 17274219]
11. Murray JL, Bernfield M. The differential effect of prenatal care on the incidence of low birth weight among blacks and whites in a prepaid health care plan. *N Engl J Med*. 1988; 319:1385–1391. [PubMed: 3185649]
12. Barfield WD, Wise PH, Rust FP, et al. Racial disparities in outcomes of military and civilian births in California. *Arch Pediatr Adolesc Med*. 1996; 150:1062–1067. [PubMed: 8859139]
13. David R, Collins J Jr. Disparities in infant mortality: what's genetics got to do with it? *Am J Public Health*. 2007; 97:1191–1197. [PubMed: 17538073]
14. Goodman AH. Why genes don't count (for racial differences in health). *Am J Public Health*. 2000; 90:1699–1702. [PubMed: 11076233]
15. Hitti J, Nugent R, Boutain D, et al. Racial disparity in risk of preterm birth associated with lower genital tract infection. *Paediatr Perinat Epidemiol*. 2007; 21:330–337. [PubMed: 17564589]
16. Shaw RJ, Pickett KE, Wilkinson RG. Ethnic density effects on birth outcomes and maternal smoking during pregnancy in the US linked birth and infant death data set. *Am J Public Health*. 2010; 100:707–713. [PubMed: 20167891]
17. Holzman C, Eyster J, Kleyn M, et al. Maternal weathering and risk of preterm delivery. *Am J Public Health*. 2009; 99:1864–1871. [PubMed: 19696383]
18. Andrews KW, Savitz DA, Hertz-Picciotto I. Prenatal lead exposure in relation to gestational age and birth weight: a review of epidemiologic studies. *Am J Ind Med*. 1994; 26:13–32. [PubMed: 8074121]
19. Vigeh M, Yokoyama K, Shinohara A, et al. Early pregnancy blood lead levels and the risk of premature rupture of the membranes. *Reprod Toxicol*. 2010; 30:477–480. [PubMed: 20576532]
20. Wu J, Ren C, Delfino RJ, et al. Association between local traffic-generated air pollution and preeclampsia and preterm delivery in the south coast air basin of California. *Environ Health Perspect*. 2009; 117:1773–1779. [PubMed: 20049131]
- *21. van den Hooven EH, Jaddoe VW, de Kluizenaar Y, et al. Residential traffic exposure and pregnancy-related outcomes: a prospective birth cohort study. *Environ Health*. 2009; 8:59. This large birth cohort in the Netherlands demonstrated no effect of residential traffic exposure and birth outcomes. [PubMed: 20028508]
- *22. Gehring U, van Eijsden M, Dijkema MB, et al. Traffic-related air pollution and pregnancy outcomes in the Dutch ABCD birth cohort study. *Occup Environ Med*. 2010 This large Dutch birth cohort demonstrated no effect of traffic-related air pollution and birth outcomes.

- **23. Salmasi G, Grady R, Jones J, McDonald SD. Environmental tobacco smoke exposure and perinatal outcomes: a systematic review and meta- analyses. *Acta Obstet Gynecol Scand*. 2010; 89:423–441. The authors of this extensive systematic review and meta-analysis of 76 articles conclude that a link between environmental tobacco smoke (ETS) and decreased birth weight exists but that the evidence supports no connection between ETS and preterm delivery. [PubMed: 20085532]
24. Conde-Agudelo A, Althabe F, Belizan JM, Kafury-Goeta AC. Cigarette smoking during pregnancy and risk of preeclampsia: a systematic review. *Am J Obstet Gynecol*. 1999; 181:1026–1035. [PubMed: 10521771]
25. Windham GC, Hopkins B, Fenster L, Swan SH. Prenatal active or passive tobacco smoke exposure and the risk of preterm delivery or low birth weight. *Epidemiology*. 2000; 11:427–433. [PubMed: 10874550]
- **26. El-Mohandes AA, Kiely M, Blake SM, et al. An intervention to reduce environmental tobacco smoke exposure improves pregnancy outcomes. *Pediatrics*. 2010; 125:721–728. This impressive interventional trial among minority pregnant women in Washington DC comparing usual care to cognitive behavioral therapy to avoid environmental tobacco smoke exposure led to a decreased risk of very preterm birth in the intervention group compared to controls. [PubMed: 20211945]
27. Blood lead levels--United States, 1999–2002. *MMWR Morb Mortal Wkly Rep*. 2005; 54:513–516. [PubMed: 15917736]
28. Woodruff TJ, Parker JD, Kyle AD, Schoendorf KC. Disparities in exposure to air pollution during pregnancy. *Environ Health Perspect*. 2003; 111:942–946. [PubMed: 12782496]
29. Geronimus AT. The weathering hypothesis and the health of African-American women and infants: evidence and speculations. *Ethn Dis*. 1992; 2:207–221. [PubMed: 1467758]
30. Geronimus AT, Hillemeier MM. Patterns of blood lead levels in US black and white women of childbearing age. *Ethn Dis*. 1992; 2:222–231. [PubMed: 1467759]
- **31. York TP, Strauss JF 3rd, Neale MC, Eaves LJ. Racial differences in genetic and environmental risk to preterm birth. *PLoS One*. 2010; 5. This statistical analysis of kinship connections and residential relationships by vital records in Virginia demonstrated that environmental factors are responsible for more of the variance in gestational age among black infants compared to white infants.
32. Osypuk TL, Acevedo-Garcia D. Are racial disparities in preterm birth larger in hypersegregated areas? *Am J Epidemiol*. 2008; 167:1295–1304. [PubMed: 18367470]
33. Polednak AP. Trends in US urban black infant mortality, by degree of residential segregation. *Am J Public Health*. 1996; 86:723–726. [PubMed: 8629726]
34. American Academy of Pediatrics Committee on Environmental Health. Environmental Disparities. In: Etzel, RA.; Balk, SJ., editors. *Pediatric environmental health*. 2. Elk Grove Village, IL: American Academy of Pediatrics; 2003. p. 585-595.
35. Dolinoy DC. The agouti mouse model: an epigenetic biosensor for nutritional and environmental alterations on the fetal epigenome. *Nutr Rev*. 2008; 66 (Suppl 1):S7–11. [PubMed: 18673496]
36. Beck S, Wojdyla D, Say L, et al. The worldwide incidence of preterm birth: a systematic review of maternal mortality and morbidity. *Bull World Health Organ*. 2009; 88:31–38. [PubMed: 20428351]

Key Points

- Environmental exposures such as tobacco smoke, lead and air pollution may increase the risk of preterm birth.
- Black women in the United States have higher levels of environmental toxicant exposures deliver preterm infants more often than white women.
- Epidemiologic studies evaluating disparities in preterm birth should account for environmental exposures.

Figure 1.
All US 2006 births, and births <28 weeks' gestation by race/ethnicity.[1]
Data from [1]