Essays on Health and Development Economics - Focusing on Early Stages in Life

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Abstract

This thesis presents three essays on health development when exposed to adverse shocks and first-year vaccinations and uses data from 35 African countries and the United States.

Chapter 1 examines the impact of drought exposure in utero on sex ratio and child health at birth using data from 33 African countries between 1959 and 2019 to shed new light on the consequences of in-utero shocks on selection at birth. We find that drought exposure during the first month in utero increases the probability of female birth, in other words, this indicates that boys are more likely to be 'lost' when a heavy drought occurs during pregnancy. To strengthen this finding, we test for a causal effect on the sex ratio based on the cluster-month level dataset. The estimates confirm that drought exposure during the first month in utero results in a skewed sex ratio towards females. Additionally, we also find that drought exposure in utero decreases birth height, especially for girls.

Chapter 2 explores the impact of wildfire exposure in utero on the birth–sex ratio and birth weight in a strongly institutionalised setting. We find that exposure to wildfires during the second trimester of pregnancy reduces birth weight by 0.3% and that boys are more sensitive to such adverse environments than girls. Although the average effect of wildfires exposure on the birth–sex ratio is insignificant, our heterogeneity analysis shows that wildfire exposure during the second trimester of pregnancy tends to increase the female–male ratio among less educated mothers, which indicates that the mother's level of education is an essential mediating factor in determining the birth–sex ratio. This latter result aligns with the Trivers–Willard hypothesis, suggesting that exposure to negative shocks while in utero results in a female-biased birth–sex ratio.

Chapter 3 studies vaccination hesitancy and vaccination rates among Muslims in African countries using data for children born between 1999 and 2019. We first find that newborn Muslims are less likely to be vaccinated compared to other religious groups. Second, we study the mechanism behind this lower vaccination incidence by examining the hate crimes against Muslims recorded in the US. Our empirical results imply that one SD increase in hate crimes against Muslims leads to a decrease in the fraction of first-year vaccinations among the Muslim population by up to 3%, and this pattern is common across different types of first-year vaccines. A series of heterogeneity analysis tests identify that trust in institutions and mobile phone penetration are important mediating factors that contribute to vaccination hesitancy, which in turn decreases child immunisation rates.

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Author's Declaration

I declare that this thesis is an original work, and I am the sole author of the first two chapters. Chapter three is co-author with Prof Giacomo De Luca.

Chapter one was presented in the 2023 European Health Economics Association (EuHEA) Supervisor-PhD conference at the University of Bologna as a full paper presentation. It has also been presented in the 2022 Workshop on Labour and Family Economics (WOLFE) at the University of York as a Poster presentation.

Chapter two has not previously been presented for an award at this, or any other, University. Chapter three has been submitted to the CSAE Conference 2024: Economic Development in Africa at the University of Oxford, but has not yet been presented. All sources are acknowledged as References.

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Introduction

This paper contains three chapters on health development with a specific focus on the effects of exposure to adverse shocks and child vaccinations during the early stages of life using data from 35 African countries and the United States. Chapter 1 contributes to the existing literature on the impacts of shocks while in utero on selection at birth, focusing on whether exposure to droughts at different times during pregnancy plays an important role in determining the birth–sex ratio.

Chapter 1 examines the effect that drought exposure at different times in utero has on the birth–sex ratio and birth health outcomes. Exposure to negative shocks in early life contributes to both higher female-male sex ratios at birth and adverse health outcomes either at birth or later in life.

The mechanism behind the hypothesis by Trivers and Willard (1973) explains that the underlying reason for maternal exposure to adverse shocks potentially leading to a skewed sex ratio at birth is the variance of reproduction success for males and females. That is, women who are in a bad condition and having a daughter will potentially have more surviving grandchildren than the women having a son, while women who are in a good condition and having a son will potentially have more surviving grandchildren than those having a daughter (Trivers and Willard, 1973). Following this, Kraemer (2000) proposed the fragile male theory, which hypothesises that male foetuses are more vulnerable than female foetuses.

In addition, the idea behind the mechanism of the foetal origins hypothesis states that the in-utero condition is closely associated with birth health outcomes, i.e. exposure to adverse shocks in utero contributes to negative health outcomes such as low birth weight, cardiovascular disease, abnormal foetus and stillbirth, which in turn may result in a skewed sex ratio with more female than male births (Almond and Mazumder, 2011, Dinkelman, 2017, Maccini and Yang, 2009, Majid, 2015, Yeung et al., 2014).

The sex ratio at birth is mainly determined by fetal death due to the biological nature of a person's sex which will not be changed once the embryo is produced after conception. Biological and socioeconomic literature suggests that timing matters in determining selection at birth and health outcomes at birth. For example: negative shock exposure in the 1st trimester during pregnancy generally shows a much stronger effect on birth sex ratios as well as lower birth weight (Dagnelie et al., 2018, Foureaux Koppensteiner and Manacorda, 2016). Moreover, weather shock exposure in utero on a child's health at birth has different effects by gender. Females who were exposed to a

positive weather shock while in utero tend to show better health outcomes either at birth or later in life, have better socioeconomic status and perform better in school compared to males who experienced a similar shock while in utero (Maccini and Yang, 2009).

Substantial literature shows that exposure to adverse environmental shocks while in utero decreases the number of male births, which leads to a birth sex ratio skewed towards females (Catalano et al., 2006*a*, Fukuda, Fukuda, Shimizu and Møller, 1998, Sanders and Stoecker, 2015). In addition, studies show that exposure to drought during pregnancy is linked to adverse birth outcomes, including lower birth weight, a higher infant mortality rate and an increased probability of being disabled in adulthood (Dinkelman, 2017, Kumar et al., 2016, Rocha and Soares, 2015)

The results discussed in Chapter 1 indicate that exposure to drought while in utero has a significant impact on the birth sex ratio. More specifically, our findings show that exposure to drought during the first month of pregnancy results in a decreased number of male births relative to female births, i.e. a one standard deviation (SD) increase in the SPEI drought index, in which an increased SPEI drought index indicates less drought, correlates to a decrease in the probability of female birth by 0.3 percentage points. In other words, if more drought occurs in the first month of pregnancy, more girls than boys are likely to be born. When estimating the effects of drought on the birth–sex ratio at the cluster-month level, based on panel-level regressions, our results show that one SD increase in the SPEI drought index reduces the sex ratio by 0.4 percentage points, in which the sex ratio is defined as $\frac{female}{male+female}$ (Number of female births divided by the sum of all births for each cluster and month).

Moreover, the results discussed in Chapter 1 also show that exposure to drought while in utero is significantly linked to health outcomes at birth. For example, a one SD increase in the SPEI drought index correlates to an increase in the Height-for-age Z (HAZ) score by 1.2 percentage points. The effects are different for each gender, i.e. a one SD increase in the SPEI drought index correlates to higher HAZ scores of 1.6% and 1.2% for females and males, respectively.

Chapter 2 examines the effect of exposure to wildfires at different times in utero on the birth–sex ratio and a child's weight at birth at both the cluster and individual levels. Environmental disasters have been widely studied as exogenous shocks that impact the birth outcomes of children either at birth or later in life (Holstius et al., 2012, Maccini and Yang, 2009, Torche, 2011).

Substantial literature claims that prenatal exposure to environmental shocks (e.g. earthquakes or wildfires) increases the female-biased sex ratio at birth and reduces the

health of infants in terms of birth weight, preterm birth and the incidence of abnormal conditions (Currie and Rossin-Slater, 2013, Holstius et al., 2012, Noghanibehambari et al., 2020, Torche, 2011). To test whether our hypothesis in Chapter 1, which states that drought exposure in utero is linked with an increased number of female births and worsened birth health outcomes, holds in developed settings, where drought is typically not an issue. We use wildfire as an exogenous shock. The occurrence of wildfires has increased in recent decades due to climate change (Bowman et al., 2020). Wildfire-induced stress and air pollution are harmful to vulnerable groups of people, such as pregnant women. When pregnant women are exposed to wildfires, the health of newborns is affected through the maternal immune function or changes in the neuroendocrine system (Dunkel Schetter, 2011) and potential damage to the foetus' DNA through the placenta (Perera et al., 1999).

Most current studies tend to investigate the effects of negative shocks by focusing on low- and middle-income countries. Similarly, as discussed in Chapter 1, using data from 33 African countries, we find that exposure to drought results in a female-biased sex ratio at birth. To further test the Trivers–Willard's (TW) hypothesis and the findings documented in Chapter 1, in this Chapter, we first look at the impact of exposure to wildfires during pregnancy on the birth–sex ratio, with a focus on strongly institutionalised settings. Second, we study the impacts of exposure to wildfires while in utero on the health of newborns and explore the differences based on gender.

Since the sex of an embryo is determined at conception, the birth–sex ratio can only be changed because of social effects or exogenous environmental effects. A study by Hansen et al. (1999) indicates that external maternal stress induced by exposure to natural shocks such as smog, floods or earthquakes before and at the time of conception is linked to a reduced male-to-female sex ratio, showing that male embryos are more fragile than their female counterparts. Additional literature on health and environmental economics discusses that the mechanism through which prenatal exposure to natural disasters affects the birth–sex ratio is related to stress-related health issues for pregnant women, which may result in miscarriage or pre-term birth, in turn resulting in male foetal loss (O'Donnell and Behie, 2013).

Wildfire exposure has increasingly been studied as a natural exogenous shock to the birth–sex ratio and the health of newborns. A branch of literature provides evidence that shows that exposure to wildfires while in utero significantly reduces the birth weight of newborns and increases pre-term birth (Holstius et al., 2012, Mccoy and Zhao, 2021). Regarding the birth–sex ratio, some studies find that wildfire exposure significantly

reduces the male–female sex ratio at birth, while other studies do not find this correlation (O'Donnell and Behie, 2013). Using data from the US, we explore how exposure to wildfires while in utero changes the sex ratio and child health at birth in places where mothers have access to high-quality healthcare.

The results discussed in Chapter 2 suggest that exposure to wildfires during pregnancy does not seem to have a significant impact on the birth–sex ratio. Similarly, when exploring the impact of wildfires on the birth–sex ratio using different cutoffs of PM2.5 as alternative measures, we find that the effects are insignificant. Although our results do not show significant effects on the birth–sex ratio, we still find that exposure to wildfires while in utero is significantly associated with reduced birth weight, suggesting that in-utero conditions play an important role in determining the health of newborns. Our estimated results show that a one SD increase in the number of days exposed to the wildfire during a 9-month gestation period correlates to a decrease in birth weight of all newborns by 0.3%. By further looking at the impacts by trimester, our results show that wildfire exposure in the second trimester significantly affects the health of newborns. More specifically, a one SD increase in the number of days exposed to a wildfire correlates to a reduction in the birth weight of all newborns by 0.3% but a 0.5% reduction for male newborns and no significant effect for female newborns.

Chapter 3 explores the first-year vaccination rates for children in Muslim households across Africa, in particular, the fact that Muslim households are less likely than other religious households to vaccinate or fully vaccinate their children in the first year of life. Moreover, the chapter also analyses the potential causal effect between vaccination hesitancy in Muslim households and hate crimes against Muslims.

Although vaccines are considered one of the most effective ways of preventing the spread of diseases and saving lives, vaccination rates still remain relatively low among certain groups (e.g. Muslims) and countries (e.g. Nigeria). A sizable portion of the literature argues that the social-economic factors of income level, media environment, religious beliefs and trust in institutions are crucial determinants of vaccine hesitancy (Dubé et al., 2013, Falagas and Zarkadoulia, 2008, Larson et al., 2014, Stoop et al., 2021). Moreover, lower household income (Guay et al., 2019), financial constraints in purchasing health products (Dupas, 2011), less reliable healthcare services (Banerjee et al., 2010), a lack of trust in local institutions (Acemoglu et al., 2020) and the circulation of fake news on social media regarding vaccines (Hansen and Schmidtblaicher, 2019) were found to be significantly associated with reduced vaccination rates.

Religious doctrines are detrimental to the vaccination rates among religious

households. For example, vaccines are produced with animal tissue, such as pig fat, which is forbidden to Muslims as indicated in the Qur'an (Maravia, 2020, Pelčić et al., 2016, Rochmyaningsih, 2018). Other than Muslim religious doctrines, anti-Western sentiments or policies also contribute to the high vaccine hesitancy, particularly among Muslim groups who argue that Western countries use vaccines as a cover to reduce the population of Muslim people. Following the disclosure of a fake vaccination campaign that was organized by the CIA to trace the DNA of Osama Bin Laden's family in Pakistan, the rumour of Western countries using vaccines to reduce the population of Muslims has become more pronounced in countries with a high proportion of Muslims, such as Nigeria, where the vaccination rates (for BCG, DPT and Polio) for children born to Muslim mothers significantly decreased after disclosure of the Pfizer drug trails (Archibong and Annan, 2021).

The data on vaccination trends across religions between 1999 and 2019 shows that first-year vaccination rates among children born to Muslim mothers are significantly lower than those children born to mothers with other religious backgrounds. More specifically, our analysis shows that the fraction of vaccination doses given was 2% lower for Muslim children than other children of the same age. Additionally, by performing the same analysis with respect to other religious groups, such as Catholic, traditional, and no religion, we do not find a pattern similar to that observed in Muslim groups. By including trust in the analysis, the results indicate that in places where trust in local institutions is low, there is a decrease in child first-year vaccination rates.

Using the data from the US on hate crimes against Muslims, we further explore whether the intensity of cultural clashes between Islam and Western countries that developed over the last two decades contributed to vaccination refusal among Muslims in Africa. The results show that the intensity of anti-Muslim hate crimes significantly and negatively impacts the vaccination rates among Muslim children in Africa in their first year of life. Specifically, a one SD increase in hate crimes against Muslims correlates to a reduction in the fraction of first-year vaccination doses given to Muslim children by 3%. Moreover, a set of heterogeneity analyses was conducted to explore whether our baseline results are different for different groups of interest, such as groups with local mobile phone penetration, anglophone countries and places where Western NGOs are involved in the provision of health services. Our heterogeneous analysis results show that first-year vaccination rates are even lower for Muslim children from these groups.

Chapter 1

The effects of early-life drought exposure on the sex ratio and child health outcomes: Evidence from 33 African countries. *(Solo-authored chapter)*

1.1 Introduction

Life starts before birth, and influential contributions to prenatal shocks suggest that more attention should be devoted to pre-birth exposure to adverse shocks to interpret later outcomes correctly. For instance, Barker (1995)'s foetal origins hypothesis suggests that in-utero conditions can significantly affect children's health during early childhood and adulthood. Moreover, the hypothesis by Trivers and Willard (1973) states that shocks while in utero may result in a skewed sex ratio with more girls being born than than boys, so women should favour girls when experiencing poor conditions and boys when in good conditions. The idea behind this mechanism is related to the variance in reproduction success for males and females. A good example is that successful marriage typically depends on the family's wealth and socioeconomic status in polygynous societies (Valente, 2015). Low-income families would prefer to have girls, as these families' sons are less likely to marry if they are not rich(Valente, 2015). If this was the case, the population of newborns would be selected, and the average health across genders may be different. This, in turn, matters when interpreting later outcomes.

A substantial portion of the literature explores the effects of shocks in early life on individual economic and health-related outcomes later in life and finds that maternal exposure to adverse shocks, such as severe droughts and floods, can adversely affect people's mental and physical health (Maccini and Yang, 2009). These shocks have repeatedly been reported to affect each gender differently (Catalano et al., 2006*b*, Dagnelie et al., 2018, Valente, 2015). For instance, Drevenstedt et al. (2008) find that male infants have a higher mortality rate than female infants, and male infants who did not die in utero due to better perinatal conditions tend to be born prematurely or have a low birth weight compared to female. Moreover, several biological and social studies investigate the impact of exposure to adverse shocks while in utero on birth–sex ratios and find that male

infants are more fragile than female infants, which results in higher male foetal death and contributes to the overall gender differences as shown in the sex ratio (Dagnelie et al., 2018). An important addition to this literature is to explore the potential heterogeneity of these effects based on gender.

Given the biological condition that the sex of a human being does not change after conception, the birth–sex ratio is determined either by the sex-selective embryonic process or foetal death that results from miscarriage or stillbirth (James and Grech, 2017). Mocarelli et al. (2000) point out that prenatal exposure to dioxin, a poisonous chemical substance that is generated when forests burn or from household trash (Centers for Disease Control and Prevention (CDC), 2017), is known to be linked to a decreased male birth rate. Many studies also find that exposure to adverse environments while in utero is associated with a higher risk of having an abnormal foetus and is also more likely to be correlated to lower birth weight, higher infant mortality and a higher incidence of cardiovascular disease when in old age (Almond and Mazumder, 2011, Dinkelman, 2017, Maccini and Yang, 2009, Majid, 2015, Yeung et al., 2014), and mothers exposed to adverse shocks during pregnancy are also more likely to have an unexpected miscarriage (Davis et al., 2007). A growing body of published work provides evidence that the birth–sex ratio is correlated to exposure to negative shocks (e.g. natural disasters, conflict, pollution events and economic collapse) while in utero. Most of these studies find that exposure to adverse shocks while in utero results in fewer male births than female births, while other studies find that prenatal shocks may result in more boys than girls being born. For instance, Bobonis et al. (2016) find that a newborn was more likely to be a boy if exposed to the shock of bombing actives while in utero in Vieques, Puerto Rico.

To figure out why different studies have different findings of the relationship between the birth–sex ratio and in-utero shocks, this paper uses an extensive individual-level dataset that includes 33 African countries and about 1,766,087 children born between 1959 and 2018 compiled by the demographic and health surveys (DHS) program and combines this dataset with the standardised precipitation–evapotranspiration index (SPEI) data collected from the SPEI website for all 33 African countries at the cluster level. The SPEI was used in the study as a measure of drought to study the effect of exposure to drought at different times during pregnancy on birth health outcomes and the birth–sex ratio in hopes of shedding new light on the consequences of shock exposure while in utero on selection at birth. We first assess the impact of exposure to drought while in utero on the probability of female birth by month using the SPEI as a measure of drought. Second, we explore the effects of drought exposure on the birth–sex ratio based on panel-level

regressions. Finally, we study the impacts of drought exposure on the health of newborns and explore the differences between genders.

Our finding indicates that male fetuses are more fragile to drought. Experiencing more rain during the first month in utero is associated with a decrease in female birth. This shows that if there is less rain during the first month of pregnancy, more girls will be generated, thus resulting in a skewed sex ratio with more girls than boys. We do not find any heterogeneity effect on the sex ratio regarding wealth index, the mother's education and work. By examining the impact of SPEI on child health, we show that exposure to higher SPEI in utero positively correlates with Height-for-age Z-scores (HAZ) and that it has a differential effect on birth height among boys and girls. Girls seem slightly more sensitive to better weather conditions (i.e., higher SPEI) than boys. Our heterogonous analysis tests show that the child's gender and household wealth index are two crucial mediating factors in determining child health at birth.

The contribution of this paper is twofold: First, using a clearly exogenous shock and an extensive individual survey dataset which consists of around one million births from 33 African countries that allows us to test the fine-grained effect of in utero shocks, we confirm the existence of gender bias on the impact of drought exposure in utero on the birth sex ratio and the health outcomes at birth, all vulnerable to climatic shocks. Second, we stress the importance of early shocks during pregnancy, where the source of the gender-specific effects stems. We utilise a more sophisticated measure of drought - the Standardized Precipitation Index (SPEI) – at different timespan as a measure of droughts to examine the effects of in utero conditions on the sex ratio and its impact on birth health outcome among countries that generally rely on rain-fed agriculture. We exploit how exposure to drought in utero is likely to change the sex ratio by focusing on different timing in utero. Unlike the Standardized Precipitation Index (SPI), the Standardized Precipitation Evapotranspiration Index (See Appendix A1 and A2 for a detailed explanation of SPEI) provides information on precipitation and potential evaporation.

This chapter is organised as follows. In Section 1.2, we provide a broad review of the existing literature on the impact of shocks while in utero on the birth–sex ratio and child health at birth. Section 1.3 then discusses the individual-level child health data and the drought data. How we construct the sample data for the analysis is also explained in detail in that section. Section 1.4 describes the empirical strategy, and section 1.5 discusses the estimated results. Finally, Section 1.6 provides a conclusion.

1.2 Literature review

This study relates to two broad strains of literature: the first focuses on the effects of shocks while in utero on the birth–sex ratio and the second investigates the consequences of shocks while in utero on health outcomes at birth. We discuss the two topics sequentially.

1.2.1 In-utero shocks on sex ratio

Many studies provide solid evidence that indicates that environmental and social factors are essential in determining the female survival advantage.

Exposure to conflict, violence and war are often considered factors that adversely impact the health of newborns and the birth–sex ratio. Valente (2015) assesses the impact of prenatal exposure to the civil conflict in Nepal on health outcomes, foetal loss and the birth–sex ratio. Her findings indicate that negative prenatal shock is associated with an increased risk of miscarriage and a higher share of female births, which is in line with the Trivers–Willard hypothesis. Dagnelie et al. (2018) use cross-sectional data from 2007 of children born between 1997 and 2004 in the Democratic Republic of Congo and document that in-utero mortality is different between the genders depending on the in-utero conditions. Their study explores the effect of exposure to conflict while in utero on the probability of giving birth to a male using mineral prices as an instrument for conflict. The study finds that exposure to conflict while in utero is significantly associated with a reduction in the number of live male births. Additionally, by estimating the impact of conflict exposure while in utero by trimester, their findings show that the effect tends to be stronger when the exposure happens during early pregnancy (first and second trimesters). Similarly, Catalano et al. (2006*b*) find that the adverse shock to maternal health caused by the 9/11 attack in New York City resulted in an increased proportion of female births.

A small group of studies have explored the impact of exposure to adverse environmental shocks in the prenatal phase on newborn health and the birth–sex ratio. Sanders and Stoecker (2015) investigate the effect of air pollution on foetal health and find that boys are generally more affected than girls when exposed to air pollution while in utero, which in turn indicates that male foetal death is more likely, leading to a decrease in male births. Other literature on the impact of adverse environmental shocks (e.g. earthquakes, extreme weather) in the prenatal phase on the birth–sex ratio provides evidence that indicates that maternal exposure to negative shocks results in a birth–sex ratio skewed towards girls (Catalano et al., 2008, Fukuda, Fukuda, Shimizu and Møller,

1998).

Fasting and famine are considered adverse nutritional shocks and the later effects of exposure to malnutrition while in utero have been widely studied. Almond and Mazumder (2011) study the impact of exposure to Ramadan fasting at different times in utero on male births and showed that exposure during the first month of pregnancy was associated with a decrease in the male birth rate. Song (2012) investigates the effect of exposure to the Great Leap Forward famine (between 1959 and 1961) in China during pregnancy on the long-term trend of the birth–sex ratio using data on individuals born between 1929 and 1982. The study showed that the probability of a male birth significantly reduced by a rate of 0.001 per month between 1960 and 1963. The study also indicated that the proportion of male births mainly decreased among women whose month of conception occurred more than half a year after the famine began. These findings suggest that the number of male births declined due to the 1959–1961 Great Leap Forward famine; this argument aligns with the Trivers–Willard hypothesis that mothers in poor conditions are more likely to give birth to girls, while those in good conditions are more likely to give birth to boys. Following this, Song (2015) further reviews the existing literature on the effect of privation and stress on the human birth–sex ratio. The review concluded that studies had provided convincing and consistent evidence in support of the association between exposure to prenatal privation and stress and a decline in male births.

A few studies have reported no effect of shocks while in utero on the birth–sex ratio. For instance, Stein et al. (2004) explore the impact of exposure to acute under-nutrition while in utero on the birth–sex ratio using the evidence from the 1944–1945 Dutch Famine. Their study found that exposure to the famine while in utero had no effect on the birth–sex ratio, which suggests that exposure to acute under-nutrition during pregnancy does not reduce the number of male births or adjust to mothers being more likely to give birth to females even in poor conditions. One potential reason for exposure to the Dutch Hunger Winter not affecting the birth–sex ratio is that the duration of the famine was too short (Song, 2012). Jürges (2015) examines the impact of exposure to Ramadan fasting while in utero on both birth outcomes and the proportion of male births using the birth data of Muslim infants in Germany and finds that exposure to Ramadan fasting during pregnancy does not significantly affect birth weight or the birth–sex ratio.

Finally, Bobonis et al. (2016) explore the short-term effects of exposure to military exercises while in utero on health outcomes by examining the US Navy bombing activity in Puerto Rico and find that the effect of this shock was a birth–sex ratio that favoured males. The effect was particularly significant when the shock occurred during the first

trimester of pregnancy. This finding differs from much of the other literature, which finds that exposure to negative shocks while in utero results in a birth–sex ratio skewed towards girls. Bobonis et al. (2016)'s results indicate that shocks in the prenatal phase and health outcomes at birth are negatively correlated. They discussed the effect of exposure to bombing on foetal health by trimester and found that the results are significant when congenital anomalies, Apgar scores and the probability of premature birth are considered, but the effect differs across trimesters. There was a substantial effect shown for congenital anomalies when the exposure occurred in trimesters two and three, whereas, for both the Apgar scores and premature birth, only exposure during trimester one showed a strong effect. In addition to the impact of bombing on birth–sex ratios, their findings indicate that exposure to bombing is associated with a higher probability of a male newborn.

In sum, even though most existing studies seem to find support for the TW hypothesis, the evidence is mixed. By adopting a comprehensive dataset and using an incontrovertibly exogenous shock, we contribute to this literature. Moreover, our detailed data allows us to also study the time dimension of a shock, which is absent in the majority of the existing literature. This time dimension allows us to examine the mechanism with a higher degree of precision. To find out the difference in the findings of the effect of in-utero shocks on the birth-sex ratio in the existing studies, we assess the causal link between shocks while in utero and the birth–sex ratio by focusing on the SPEI at different times in utero to shed new light on the TW hypothesis and provide further evidence to support the association between exposure to a negative shock while in utero and fewer male births.

1.2.2 Early life shocks on health outcomes at Birth

The majority of studies document that exposure to adverse shocks while in utero is not only linked to worse health outcomes at birth (e.g. lower birth weight, stunting) and in adulthood (e.g. being disabled) but also worse school performance and a reduced ability to work in the labour market (Dinkelman, 2017, Foureaux Koppensteiner and Manacorda, 2016, Majid, 2015).

Shah and Steinberg (2017) assess the impact of early-life rainfall shocks on human capital accumulation. Their study confirms that exposure to sufficient rainfall while in utero is associated with significant positive effects on overall schooling performance. Children exposed to higher rain while in utero or during early childhood, i.e. below the age of 4, generally show a higher learning ability in reading and calculating and a higher enrolment rate (Shah and Steinberg, 2017). Hoynes et al. (2016) use the Food Stamp

Program, implemented between 1961 and 1975 across US counties, as an early life shock and assess its effects on long-term health outcomes. Their study indicates that people exposed to or with access to the Food Stamp Program both while in utero and during early childhood are at a lower risk of having metabolic syndrome-related diseases. Almond and Mazumder (2005) explore the causal effects between adverse shocks and long-term health outcomes during adulthood using the 1918 influenza pandemic as a natural experiment. Their study found that cohorts who experienced the influenza pandemic while in utero tended to be less healthy when they grew old compared to those who were not hit by the shock. Similarly, Almond (2006) further examines the long-term effects of exposure to the 1918 influenza pandemic while in utero on children's performance later in life. The study found that children exposed to the 1918 influenza pandemic while in utero were more likely to be less educated and have lower income and socioeconomic status and had an increased risk of being disabled as an adult.

Almond and Mazumder (2011) use Ramadan fasting as an exogenous nutritional shock while in utero to identify the effect on child health based on Michigan data. Their findings indicate that exposure to this negative shock before birth is correlated to lower birth weight. van Ewijk (2011) examines the effect of exposure to fasting while in utero on health later in life using cross-sectional data from Indonesia. The study indicates that exposure to Ramadan fasting while in utero is associated with poor health after birth. It further points out that those exposed to fasting while in utero are generally less healthy than those who were not and have a higher risk of living with health-related problems, such as coronary heart disease, type-2 diabetes and anaemia, when they get older.

Foureaux Koppensteiner and Manacorda (2016) explore the impact of exposure to local violence on health outcomes at birth using the Brazilian Ministry of Health dataset from 2000 to 2010. Their investigation suggests that exposure to local violence before birth is correlated to an increased risk of adverse birth outcomes, i.e. foetuses whose mothers were living in a place with a high prevalence of local violence during the time of pregnancy were more likely to have a lower birth weight and were at a higher risk of premature birth, especially for the group of children whose mothers were less educated. In addition, the study also finds that the effects are substantially significant if violence occurs during the first trimester of pregnancy. Akbulut-Yuksel (2017) uses the intensity of the destruction caused by World War II measured by the accumulated residential rubble in m3 per capita by the end of WWII as an exogenous negative shock in early life. The study uses survey data from 2002 on individuals' body mass index and obesity levels to explore the long-run impact of exposure to war in early life on health outcomes. The findings indicate that

exposure to WWII in early life is associated with metabolic syndrome diseases (diabetes, high blood pressure and cardiovascular disorders) later in life.

A subset of this literature explores the effects of exposure to weather-related shocks while in utero on a child's health outcome. Rocha and Soares (2015) assess the impact of exposure to drought while in utero on child health outcomes at birth using data collected from the semiarid region of northeastern Brazil. Their findings report that exposure to drought while in utero significantly affects birth weight and significantly increases infant mortality and decreases gestation length. Dinkelman (2017) explores the effect of exposure to random droughts of different cohorts while in utero on long-term health outcomes using the census data collected in 1996 on children born between 1948 and 1968 in South African homelands. The study finds that exposure to drought during early childhood is associated with a higher probability of being physically disabled later in life. This finding is consistent with prior literature that shows that exposure to damaging rainfall while in utero is linked to adverse health outcomes later in life.

Kudamatsu et al. (2012) study the relationship between weather fluctuations and infant mortality using individual-level data and weather data from the DHS program and re-analysis with climate models (ERA-40), respectively. The ERA-40 data was provided by the European Centre for Medium-Term Weather Forecasting from 29 African countries. The study finds that exposure to drought when in utero results in a higher probability of death after birth. Kumar et al. (2016) find that exposure to drought while in utero is associated with babies being less healthy at birth in terms of weight, which means that children exposed to drought before birth are more likely to be born underweight or severely underweight.

An influential paper by Maccini and Yang (2009) investigate the long-term effect of weather shocks at birth on later-life health outcomes using individual sample data of 4,615 girls and 4,277 boys born in the rural areas of Indonesia between 1953 and 1974. Their findings indicate that experiencing higher rainfall at the time of birth does not have a significant effect on men. In contrast, women who experienced sufficient rain during the time of birth tended to have better health outcomes, perform better in school and have better socioeconomic status and were also less likely to report poor health when asked to self-report health later in life. Following this, Bauer and Mburu (2017) adopt a different measure of drought – the standardised normalised difference vegetation index (NDVI) – to assess the impact of drought on child health. They used the NDVI remote sensing data and the index-based livestock insurance child and household panel data, and their findings suggest that the NDVI is positively correlated with child health.

Our findings, which are based on a much larger sample dataset of over 33 African countries combined with a more sophisticated measure of drought, perfectly align with the existing literature.

1.3 Data

The datasets used in this study were taken from two different data sources: (i) The Demographic and Health Surveys (DHS); and (ii) the Standardized Precipitation-Evapotranspiration Index (SPEI), which is a multi-scalar drought index based on monthly climatic data.

1.3.1 Individual-level child data: The Demographic and Health Surveys program

The DHS[1] program is a nationally representative household survey conducted in many developing countries and funded by USAID. It provides detailed and reliable health, nutrition, demographics, fertility and mortality data. The survey collected data from all eligible women aged between 15 and 49. The data for analysing child health outcomes in this study was taken from the DHS program, which was collected from 33 African countries between 1994 and 2018. Figure A2 illustrates the distribution of Height for age Z-scores, the variable we use to measure child health. Information on the child comes from two separate datasets: the children's survey, which provides information on the pregnancy and the baby's postnatal care, immunisation and health for each child born to the interviewed women as well as the mother's information, and the birth survey, which provides information on the full birth history of the interviewed women. Our study combines the children's survey and birth survey datasets to obtain a sizeable individual-level dataset. Using the GPS details provided by the DHS program, we geo-referenced all individuals in our dataset. Finally, the individual-level dataset with GPS coordinates is linked to the drought dataset to identify children exposed to shocks while in utero.

As Akombi et al. (2017) conclude that both a child's characteristics (e.g. child's age, mother's education) and a household's characteristics (e.g. wealth index) are determinants of child malnutrition in sub-Saharan Africa, our analysis includes control variables for the mother, the child and household characteristics collected by the DHS program to account for confounders in the model. The controlling variables for a child include the 'child's age

[1] Accessed in Oct 2020: https://dhsprogram.com/.

in months' and the 'birth order of the child'. These two variables are included because many studies indicate that birth order has significant adverse effects on child health outcomes (Horton, 1988), and the child's age in months also significantly affects child health outcomes (Bauer and Mburu, 2017). Moreover, the controlling variables for the mother and household include the household wealth index, the mother's occupation, the mother's education, twins and the number of siblings.

1.3.2 Drought data: Standardized precipitation-evapotranspiration index

The SPEI data is taken from the SPEI website that captures weather shocks. The SPEI is a multiscalar drought index that was first proposed by Vicente-Serrano et al. (2010) and based on the SPI (refer to Appendix A1 for detailed information on how the SPEI is derived), with a 0.5-degree spatial resolution and a monthly time resolution with different timespans (from 1 month to 48 months). An advantage of using the SPEI for drought indication rather than information on rainfall precipitation is that it captures the precipitation and includes potential evaporation information that plays a significant role in local drought conditions. The SPEI is divided into several categories based on the SPEI values (see Appendix A2). For instance, an SPEI value between -1.55 and -1.99 is defined as a severe drought. Figure A1 plots the occurrence of droughts as a percentage of the total number of the cluster over the period 1950–2015 based on the SPEI with a timespan of one month. The droughts that occurred over this period had SPEI values that ranged from 0 to 10. Over 20% of all clusters suffered drought three times, while less than 1% experienced droughts more than ten times between 1950 and 2015.

Based on the source data, we compute the SPEI for each cluster and each month, allowing us to know the exact month-by-month exposure to drought while in utero for all children in our sample. The main treatment variable we used to identify the exposure while in utero on the probability of female births, the birth–sex ratio and birth height is the SPEI, a continuous variable that captures the water balance in the period considered concerning its local long-run average.

The SPEI is provided for different timespans, ranging from 1 to 48 months, to better understand the drought of different durations (Peng et al., 2020). The SPEI with a 3-month timespan captures the average water balance in the three months preceding the month considered compared to the long-run average water balance computed over all 3-month windows available for that location. Hence, to assess the consequences of exposure

to SPEI while in utero, we utilise the SPEI data with a 9-month timespan, which captures the average water balance in the nine months preceding birth, thus roughly covering the entire duration of pregnancy.

We also explore the effects of drought exposure at specific months during pregnancy on gender differences and child health at birth. In doing so, we define the SPEI exposure by month, i.e. 'SPEI in month 1', 'SPEI in month 2', 'SPEI in month 3', 'SPEI in month 4', 'SPEI in month 5', 'SPEI in month 6', 'SPEI in month 7', 'SPEI in month 8' and 'SPEI in month 9'. We utilise the SPEI data with a timespan of one month, which captures the average water balance in the previous month.

1.3.3 Outcome variables

By merging the geo-located DHS and SPEI, we obtain a final sample dataset for the analysis. By pooling the surveys from 33 African countries and accounting for the missing observations, the final sample data for the analysis contains 1,766,087 children. For a much smaller sample (192,410 observations), the DHS program also provides the anthropometric measure for children in the age group 0–59 months whose mothers were between 15 and 49 years old at the time of the interview. We use this last sub-sample to investigate the effects of drought on later health outcomes.

The main outcome variable of interest is 'female', a dummy variable used to assess the effects of exposure to SPEI while in utero on the probability of female birth. The other outcome variable of interest in this study is the sex ratio to test the relationship between exposure to the SPEI while in utero and sex ratios. We collapsed the dataset at the cluster level and month-year of birth level and defined 'sex ratio' as the total number of females divided by the number of children born in the same month-year and place (with the same DHSID).

The third outcome variable we want to explore is the Height-for-age Z-score. Although there are studies that utilised children's weight as a proxy to identify the effects of exposure to adverse shocks in early life on the nutritional status of children, height is more often used to analyse children's early-life health outcomes as a reflection of the nutrition status of children (Currie and Vogl, 2013). The anthropometric measurements of the HAZ scores show how far a child is from the median reference value (WHO, 2018). The WHO (2018) indicated that any child whose HAZ score is lower than -200 SDs of the World Health Organization's child growth median is considered to have moderate chronic malnutrition. The child is said to have severe chronic malnutrition if their HAZ

score is lower than -300 SDs of the World Health Organization's child growth median. The variable HAZ score indicates the difference between a child's height and the median reference value of height for children of the same age and sex.

Table 1.1 presents the information of the individual-level child dataset, summarising all the variables included in the model to estimate the effects of SPEI exposure while in utero on the probability of female birth and child health outcomes. The number of observations in Table 1 is inconsistent across each variable because either missing or flagged values exist in the survey and SPEI datasets.

Table 1.2 shows the information from the cluster-month level dataset, and it summarises the statistics of the variables used to analyse the effects of exposure while in utero on the sex ratio.

1.4 Empirical strategy

We implemented three sets of regressions to assess the effects of exposure to drought while in utero on the probability of female births, the sex ratio at birth and the birth height to test whether there is any differential effect between male and female foetuses. First, we kept the unit of analysis at the individual level to run cross-sectional regressions to explore the impact of drought on the probability of female births. Second, we explored the effect on the sex ratio using a collapsed sample dataset at the cluster-month level to run panel-level regressions. Third, we ran cross-sectional regressions to study the impact of drought on birth height.

1.4.1 The probability of female birth

To test the hypothesis that exposure to drought while in utero has a significant effect on the probability of a newborn being a female, we estimated the following linear fixed-effect regressions[2]:

$$Female_{icymt} = \beta_0 + \beta_{utero} Utero_{cym} + \beta_x X_i + \omega_t + \mu_c + \delta_{ym} + \epsilon_{icymt} \tag{1.1}$$

where $Female_{icymt}$ is a dummy variable for child i living in cluster c, born in year y and month m and surveyed in year t. It takes the value of 1 if the child is female and 0 if the child is male. $Utero_{cym}$ is the main treatment variable in this equation and is measured by

[2]The survey sampling weights re-weighted method has been applied to all estimations. This is because we are combining data from multiple countries. Hence, this method should be used to make the results representative of the countries in all the analyses.

the SPEI index in cluster c in year-month ym. X_i is a vector of individual-level controls for child and household characteristics (the current child's age in months, the birth order of the child, the mother's education, the household wealth index, the mother's type of work, twins and the number of siblings). ω_t represents the survey year fixed effects that were included to control for time-varying unobserved factors throughout the survey years. μ_c is the DHS cluster fixed effects included to account for unobserved heterogeneity across each cluster and to capture local factors that may affect health outcomes. δ_{ym} represents the birth year-month fixed effects. ϵ_{icymt} is a standard error term. Cluster-robust standard errors at the DHS cluster level are presented in all the estimates. Having a cluster-robust standard error allows us to account for the possible correlation of errors within the same cluster but not between clusters. Finally, the DHS survey sampling weights are used in all estimations.

1.4.2 The sex ratio

In addition to assessing the effect of exposure to drought while in utero on the probability of female birth, we further explored the impact of relative drought on the sex ratio at birth. To evaluate this effect, we estimate the following fixed-effect panel regressions:

$$Sexratio_{cymt} = \beta_0 + \beta_{utero} Utero_{cym} + \beta_x X_{ct} + \omega_t + \mu_c + \delta_{ym} + \epsilon_{cymt} \tag{1.2}$$

where $Sexratio_{cymt}$ is a continuous outcome variable of interest surveyed in year t in each cluster c and for each year y and month m of birth. $Sexratio_{cymt}$ was obtained by collapsing the sample data at the cluster level and month-year birth level. It is defined as the total number of females divided by the total number of children born in the same cluster, the same month and the same year (i.e. $\frac{female_{cymt}}{male_{cymt}+female_{cymt}}$). The variable $Utero_{cym}$ is defined as in the previous model, and X_{ct} is a vector of cluster and month-year of birth level controls for child and household characteristics, namely, the current child's age in months, the birth order of the child, the level of mother's education, the household wealth index, the mother's type of work, twins and the number of siblings.

1.4.3 Exposure while in utero on Height for Age Z-scores

Additionally, we explored the impact of relative drought on child health using the following fixed-effects model:

$$HAZ_{icymt} = \beta_0 + \beta_{utero} Utero_{cym} + \beta_x X_i + \omega_t + \mu_c + \delta_{ym} + \epsilon_{icymt} \tag{1.3}$$

where HAZ_{icymt} is the HAZ score expressed in units equal to one standard deviation of the reference population's distribution for child i, living in cluster c, born in year y and month m and surveyed in year t. $Utero_{cym}$ is the main treatment variable in this equation and is measured by the SPEI index in cluster c, year y and month m. X_i is a vector of individual-level controls, including the gender of the child, the current child's age in months, the birth order, the mother's level of education, the household wealth index and the mother's type of work.

1.4.4 Heterogeneity analysis

It is natural to think that there may be some cases in which household factors, such as income, education and occupation, play an essential role in determining the effects of exposure to drought while in utero on child health outcomes (Kudamatsu et al., 2012). Intuitively, children from low-educated or low-income families tend to suffer more from the shocks than wealthy and well-educated families.

As such, in this study, we explore the heterogeneity of our main results across four main dimensions. The first is household wealth, a category variable in the DHS data. The second is the occupation of the mother. We defined the mother's work[3] as a dummy variable equal to 1 if the mother works for or is self-employed in the agriculture field in the survey year and 0 otherwise. About 30% of all children were born in agricultural families. The third dimension is the mother's education; we defined the mother's education as a dummy variable equal to 1 for mothers with primary or higher education and 0 otherwise. Around 53% of all children were born to an 'educated' mother. Fourth, we examined the heterogeneity of each child's health outcomes by gender.

To explore whether our results on the probability of female birth and birth weight are heterogeneous as a function of the wealth index, the mother's work, the child's gender and the mother's education level, we amended the previous model by adding an interaction term between $Utero$ and the dimensions of heterogeneity explored. The interaction terms are $Utero_{cym} * Wealth_i$, where the wealth index ranges from 1 to 5; $Utero_{cym} * MotherWork_i$, where the mother's work equals 1 if she works in the agriculture industry and 0 otherwise; $Utero_{cym} * MotherEducation_i$, where the mother's education level equals 1 if she completed primary and higher education and 0 otherwise and $Utero_{cym} * Female_i$, where Female equals 1 if the baby is a girl and 0 otherwise. These

[3]One thing to note is that by generating the dummy variable for the mother's occupation, we also bring up a measurement error we cannot eliminate. This is because other rain-fed industries exist, such as fishery and forestry (Kudamatsu et al., 2012), that we did not consider, which in turn biased our estimations for the heterogeneous effect of drought shock.

terms are used for the analysis of exposure while in utero on the HAZ scores. Regarding the cluster-level heterogeneous analysis for the sex ratio, we computed the interaction terms by taking the average of the three mediating factors (wealth index, mother's work and mother's education), which is obtained by aggregating the sample at the cluster-level and month-year of birth level.

1.5 Results

1.5.1 Results for the Probability of female birth

The estimates in Table 1.3 show the effect that exposure to drought while in utero has on the probability of female birth. In column 1, we estimated model 1.1 without adding individual controls. In columns 2 and 3, we controlled for being an only child and the mother's characteristics. Finally, in column 4, we added a complete set of controls for both the child, the mother and the household characteristics. The sign of the coefficient of the treatment variable in column 4 suggests that experiencing less drought (i.e. SPEI increases) results in fewer girls. However, the coefficients are statistically non-significant for all columns.

Valente (2015) has found that exposure to a negative shock both in the first trimester and third trimester shows a significant positive effect on the probability of female birth, and the impact of being exposed during the first trimester is slightly higher than during the third trimester in terms of resulting in a higher likelihood of female birth. We, therefore, moved to a more granular analysis. Table 1.4 shows the estimated results based on the SPEI data with a 1-month timespan preceding the 1-month bracket period considered. The estimated results indicate that exposure to SPEI while in utero during the first month of pregnancy significantly affects the probability of female births. Our results are consistent with Valente (2015)'s study: the effect on the probability of female birth is most significant if exposed during the first trimester. Moreover, experiencing more rain during the first month of pregnancy, for example, a one SD increase in the SPEI, is associated with a decrease in the probability of a newborn being a girl by 0.3% ($=\frac{-0.0015\times0.984}{0.499}$)[4]. In other words, more girls will be born if less rain occurs during the first month of pregnancy. For instance, compared to a place where the SPEI is 0 for the year and the probability of a female birth is 50%, if there is a heavy drought, i.e. the SPEI

[4]Standardized regression coefficient, i.e., Standardized regression coefficient $= \beta_i \times \frac{Standard deviation (SD) of X_i}{Standard deviation (SD) of Y}$

decreases from 0 to -2, the probability of a newborn being a girl would increase by 0.6% ($=\frac{-0.0015\times0.984}{0.499}\times-2$). The estimated results are also consistent with the TW hypothesis that exposure to adverse shocks while in utero increases female birth.

Table 1.5 replicates the previous estimations, keeping only the first month of pregnancy in the analysis, to provide a relevant reference point to compare with the later heterogeneity analysis where we focus on the first month of pregnancy for simplicity.

1.5.2 Results of Sex Ratio

To strengthen the confidence in our results, we ran a set of panel-level regressions using the dataset aggregated at the cluster-month level to further assess the effect of exposure to drought while in utero on the sex ratio. Table 1.6 shows the estimated results for the birth–sex ratio. Again, in column 1, we excluded all controls. In columns 2 and 3, we controlled for being an only child and the mother's characteristics. In column 4, we included a complete set of controls. The estimates are insignificant in all columns, indicating that drought exposure throughout the nine months in utero does not seem to strongly affect the birth–sex ratio. However, the sign of the coefficient of the treatment variable suggests that experiencing less drought (i.e. a higher SPEI) decreases the birth–sex ratio. Intuitively, the estimates in Table 1.6 are again similar to our previous results that more relative drought leads to more girls and fewer boys being born. Moving to the granular analysis, Table 1.7 shows that drought exposure during the first month of pregnancy significantly decreases the birth–sex ratio. According to the coefficient in column 4, increasing the SPEI by one SD during the first month in utero reduces the birth–sex ratio by about 0.4%.

As we did for the individual level analysis, Table 1.8 reports the results of a simplified model that focuses only on the first month of pregnancy to simplify the discussion of the heterogeneity results.

1.5.3 Results of heterogeneous effects

Table 1.9 presents the estimated results for the interaction terms between drought exposure while in utero and the wealth index, the mother's type of work and the mother's education level and how each factor affects the probability of female birth and the sex ratio, while Table 1.10 shows the estimates for the interaction coefficients of drought exposure during the first month of pregnancy on both the probability of female birth and the sex ratio. Although none of the interaction variables shows statistically significant results,

some of the signs of the coefficients in the table confirm the intuitive theory previously mentioned: a higher wealth index may reduce the effect that drought exposure during the first month of pregnancy has on the probability of female birth and the birth–sex ratio.

1.5.4 Results for exposure to drought while in utero on HAZ scores

Table 1.11 presents the results of estimating model 1.3. It shows the causal relationship between exposure to drought while in utero and child health at birth. Since drought is treated as a negative shock and measured by the SPEI in our sample data, the estimates indicate that less exposure to drought while in utero positively correlates with a higher HAZ score for the child at birth.

Column 1 shows the estimated results for the whole sample, and the estimates show that a one SD increase in the SPEI increases the HAZ score by 1.2%. Columns 2 and 3 are the estimated results for the girl and boy sub-samples, respectively. Experiencing less drought (i.e. a higher SPEI) while in utero significantly affects the health of both girls and boys. However, girls seem to be stronger than boys. In particular, a one SD increase in the SPEI is associated with a 1.6 percentage point increase in HAZ score above the average of the reference population for girls but with only a 1.2 percentage point increase in the HAZ score for boys. In line with our intuitive theory, we found that the wealth index has a strong positive effect on child health outcomes. As such, the HAZ score increases by 8.5 percentage points when the wealth index increases by one SD. Additionally, whether a child is one of twins also significantly affects birth height.

Table 1.12 presents a set of heterogeneity analysis tests to examine whether our baseline results on child health are heterogeneous by household wealth, the mother's type of work, the mother's education level and the child's gender. Column 2 and column 3 show that no differential effects on child height at birth were found based on the mother's type of work and the mother's education level. However, column 1 and column 4 show that the effect of drought exposure while in utero on the HAZ score is different for the wealth index level and the child's gender, indicating that both the household wealth index and the child's gender are critical mediating factors that contribute to child health outcomes at birth. The results for all the other non-interacted variables are consistent with the previous estimations for girls, boys and the full samples.

We further assessed the effects of being exposed to drought during each month of pregnancy. The estimates in Table 1.13 indicate that girls seem more likely to be healthy

when exposed to relatively good conditions during the last month of pregnancy. With a one SD increase in the SPEI during the ninth month in utero, the HAZ score for girls increased by around 2%. There is evidence that part of the difference found in the impact of drought on HAZ scores is due to the differential impact that drought has at different times during pregnancy. While shocks early in pregnancy have a strong effect on the birth–sex ratio, shocks later in pregnancy have a strong effect on the HAZ score.

1.6 Conclusions

This chapter explores the effect of exposure to the drought (measured by SPEI) while in utero on the birth–sex ratio and child health by using a rich dataset on births in 33 African countries between 1959 and 2018. In this study, we define the sex ratio as the total number of females divided by the total number of children born in the same cluster and in the same month and year. We used the SPEI as a measure of drought to represent the weather shocks experienced while in utero, then modelled the SPEI at the cluster level.

Our finding indicates that prenatal exposure to a negative shock significantly impacts both the birth–sex ratio and child health outcomes at birth. By exploring the effect of drought exposure while in utero on the probability of female birth and the sex ratio, we find that exposure to less drought (i.e. a higher SPEI) during the first month of pregnancy in particular, negatively correlates with the probability of female birth. That is, experiencing less drought (i.e. a higher SPEI) while in utero results in a lower probability of a newborn being female. In other words, boys are more fragile when exposed to heavy drought in utero. Additionally, tests of the effect of SPEI exposure while in utero on the sex ratio based on the cluster-month level dataset also confirm the idea that being exposed to heavy drought during the first month of pregnancy leads to a sex ratio skewed towards girls, thus suggesting that the selection mechanism occurs at birth.

When assessing the effects of drought exposure on child health outcomes at birth, our findings show that experiencing less drought while in utero results in better child health outcomes. This finding is in line with Barker's hypothesis, which states that exposure to adverse shocks while in utero can lead to a baby being born malnourished. To determine whether there is a differential effect on overall child health between genders, we tested the impact of SPEI exposure while in utero on boys and girls separately. The estimated results indicate that girls tend to be healthier when exposed to less drought during pregnancy than boys. Such a difference is potentially due to the differential impact

that drought has in the first month of pregnancy. To some extent, our findings are in line with both Barker's hypothesis and the Trivers-Willard hypothesis.

This study allowed us to derive an estimate of the effect of SPEI exposure while in utero on both the sex ratio and child health at birth with a higher level of precision by using a rich dataset from African countries where droughts are frequent, which also allows us to shed new light on the effects of prenatal shocks on selection at birth.

From a policy perspective, this paper emphasises the need to consider public health policies that help improve maternal and foetal health given the high frequency of droughts in African countries. Support programmes such as supplying food to pregnant women should also be considered during droughts to prevent the foetus from being born malnourished. In addition, this study provides further evidence that negative shocks while in utero result in the selected population at birth, which carries significant consequences for the increasing literature that studies the impact of shocks in early life on health and socioeconomic outcomes.

Tables

Table 1.1: Descriptive statistics – Cross sectional level regressions on probability of female birth and birth height

Variable	N	Mean	SD	Min	Max
Dependent and independent variables:					
Height for age	192410	-136.628	173.081	-600	600
SPEI in utero	1766087	-0.104	0.962	-5.159	4.267
SPEI in month1	1766087	-0.053	0.982	-4.802	5.077
SPEI in month2	1766087	-0.051	0.982	-4.783	5.077
SPEI in month3	1766087	-0.052	0.982	-4.802	5.077
SPEI in month4	1766087	-0.055	0.981	-4.802	5.756
SPEI in month5	1766087	-0.058	0.980	-4.802	5.077
SPEI in month6	1766087	-0.056	0.981	-4.802	5.077
SPEI in month7	1766087	-0.060	0.979	-4.802	4.874
SPEI in month8	1766087	-0.061	0.978	-5.591	4.956
SPEI in month9	1766087	-0.058	0.979	-5.591	5.756
Additional control variables:					
Female	1766087	0.491	0.499	0	1
Age in month	1766087	124.986	90.885	0	480
Birth order	1766087	3.223	2.209	1	22
Mother's education	1766087	0.585	0.493	0	1
Mother's work	1766087	0.354	0.478	0	1
Wealth index	1674282	2.820	1.386	1	5
Twin	1766087	0.017	0.129	0	1
Number of siblings	1766087	1.851	1.946	0	18

Note: The individual child data comes from the demographic and health surveys program for children born between 1959 and 2018 and was collected from the interviewed mothers aged between 15 and 49. All surveys were conducted between 1994 and 2018.

Table 1.2: Descriptive statistics – Panel-level regressions on the sex ratio

Variable	N	Mean	SD	Min	Max
Dependent and Independent variables:					
Sex ratio	1488160	0.253	0.241	0	0.5
SPEI in utero	1488160	-0.104	0.965	-5.159	4.267
SPEI in month1	1488160	-0.053	0.984	-4.802	5.077
SPEI in month2	1488160	-0.052	0.984	-4.783	5.077
SPEI in month3	1488160	-0.052	0.984	-4.802	5.077
SPEI in month4	1488160	-0.056	0.983	-4.802	5.756
SPEI in month5	1488160	-0.058	0.983	-4.802	5.077
SPEI in month6	1488160	-0.057	0.982	-4.802	5.077
SPEI in month7	1488160	-0.06	0.981	-4.802	4.874
SPEI in month8	1488160	-0.06	0.981	-5.591	4.956
SPEI in month9	1488160	-0.058	0.981	-5.591	5.756
Additional control variables:					
Age	1488160	129.545	92.885	0	480
Birth order	1488160	3.157	2.087	1	22
Mother's education	1488160	0.598	0.48	0	1
Mother's age	1488160	0.352	0.469	0	1
Wealth index	1410979	2.854	1.371	1	5
Twin	1488160	0.009	0.064	0	1
Number of siblings	1488160	1.795	1.83	0	15.875

Note: The individual child data comes from the demographic and health surveys program for children born between 1959 and 2018 and was collected from the interviewed mothers aged between 15 and 49. All surveys were conducted between 1994 and 2018. Table 2 is a collapsed dataset obtained by collapsing the individual-level dataset at the cluster level and month level. The sex ratio is defined as females/(females+males).

Table 1.3: The effect of SPEI in utero on the probability of female birth

Dependent Variable:	Female (1)	Female (2)	Female (3)	Female (4)
SPEI09 in utero	-0.0004	-0.0004	-0.0004	-0.0004
	(0.0008)	(0.0008)	(0.0008)	(0.0008)
Observations	1766034	1766034	1674239	1674239
Child controls		✓		✓
Household controls			✓	✓
DHS Cluster FE	✓	✓	✓	✓
Survey year FE	✓	✓	✓	✓
Month-Year Birth FE	✓	✓	✓	✓

Notes: All regressions were estimated using a multi-way fixed effects estimator. The robust standard errors clustered by DHSID are in parentheses. *** p<0.01, ** p<0.05 and * p<0.1.

Table 1.4: The effect of SPEI on the probability of female birth by month.

Dependent Variable	Female (1)	Female (2)	Female (3)	Female (4)
spei01 in month 1	-0.0015**	-0.0015**	-0.0015**	-0.0015**
	(0.0007)	(0.0007)	(0.0007)	(0.0007)
spei01 in month 2	0.001	0.001	0.0008	0.0009
	(0.0007)	(0.0007)	(0.0007)	(0.0007)
spei01 in month 3	0.0003	0.0003	0.0003	0.0003
	(0.0007)	(0.0007)	(0.0007)	(0.0007)
spei01 in month 4	-0.0005	-0.0005	-0.0004	-0.0004
	(0.0007)	(0.0007)	(0.0007)	(0.0007)
spei01 in month 5	0.0003	0.0003	0.0003	0.0003
	(0.0007)	(0.0007)	(0.0007)	(0.0007)
spei01 in month 6	0.0011	0.0011	0.0011	0.0011
	(0.0008)	(0.0008)	(0.0008)	(0.0008)
spei01 in month 7	-0.0005	-0.0005	-0.0004	-0.0004
	(0.0007)	(0.0007)	(0.0007)	(0.0007)
spei01 in month 8	-0.001	-0.001	-0.001	-0.001
	(0.0007)	(0.0007)	(0.0007)	(0.0007)
spei01 in month 9	0.0000	0.0000	0.0001	0.0001
	(0.0007)	(0.0007)	(0.0008)	(0.0008)
Observations	1766034	1766034	1674239	1674239
Child controls		✓		✓
Household controls			✓	✓
DHS Cluster FE	✓	✓	✓	✓
Survey year FE	✓	✓	✓	✓
Month-Year Birth FE	✓	✓	✓	✓

Notes: All regressions were estimated using a multi-way fixed effects estimator. The robust standard errors clustered by DHSID are in parentheses. *** p<0.01, ** p<0.05 and * p<0.1.

Table 1.5: The effect of SPEI on the probability of female birth - the first month only.

Dependent Variable	Female (1)	Female (2)	Female (3)	Female (4)
SPEI01 in month1	-0.0014**	-0.0014**	-0.0014*	-0.0014*
	(0.0007)	(0.0007)	(0.0007)	(0.0007)
Observations	1766034	1766034	1674239	1674239
Child controls		✓		✓
Household controls			✓	✓
DHS Cluster FE	✓	✓	✓	✓
Survey year FE	✓	✓	✓	✓
Month-Year Birth FE	✓	✓	✓	✓

Notes: All regressions were estimated using a multi-way fixed effects estimator. The robust standard errors clustered by DHSID are in parentheses. *** p<0.01, ** p<0.05 and * p<0.1.

Table 1.6: The effect of SPEI in utero on sex ratio

Dependent Variable	Sex ratio (1)	Sex ratio (2)	Sex ratio (3)	Sex ratio (4)
SPEI09 in utero	-0.0003	-0.0003	-0.0003	-0.0003
	(0.0004)	(0.0004)	(0.0004)	(0.0004)
Observations	1488103	1488103	1410933	1410933
Child controls		✓		✓
Household controls			✓	✓
DHS Cluster FE	✓	✓	✓	✓
Survey year FE	✓	✓	✓	✓
Month-Year Birth FE	✓	✓	✓	✓

Notes: All regressions were estimated using a multi-way fixed effects estimator. Robust standard errors clustered by DHSID are in parentheses. *** p<0.01, ** p<0.05 and * p<0.1.

Table 1.7: The effect of SPEI in utero on sex ratio

Dependent Variable	Sex ratio (1)	Sex ratio (2)	Sex ratio (3)	Sex ratio (4)
spei01 in month 1	-0.0008**	-0.0008**	-0.0008**	-0.0008**
	(0.0004)	(0.0004)	(0.0004)	(0.0004)
spei01 in month 2	0.0004	0.0004	0.0004	0.0004
	(0.0004)	(0.0004)	(0.0004)	(0.0004)
spei01 in month 3	0.0000	0.0000	0.0000	0.0000
	(0.0004)	(0.0004)	(0.0004)	(0.0004)
spei01 in month 4	-0.0002	-0.0002	-0.0001	-0.0001
	(0.0004)	(0.0004)	(0.0004)	(0.0004)
spei01 in month 5	0.0000	0.0000	0.0000	0.0000
	(0.0004)	(0.0004)	(0.0004)	(0.0004)
spei01 in month 6	0.0006	0.0006	0.0006	0.0006
	(0.0004)	(0.0004)	(0.0004)	(0.0004)
spei01 in month 7	-0.0002	-0.0002	-0.0002	-0.0002
	(0.0004)	(0.0004)	(0.0004)	(0.0004)
spei01 in month 8	-0.0004	-0.0004	-0.0004	-0.0004
	(0.0004)	(0.0004)	(0.0004)	(0.0004)
spei01 in month 9	0.0000	0.0000	0.0001	0.0001
	(0.0004)	(0.0004)	(0.0004)	(0.0004)
Observations	1488103	1488103	1410933	1410933
Child controls		✓		✓
Household controls			✓	✓
DHS Cluster FE	✓	✓	✓	✓
Survey year FE	✓	✓	✓	✓
Month-Year Birth FE	✓	✓	✓	✓

Notes: All regressions were estimated using a multi-way fixed effects estimator. Robust standard errors clustered by DHSID are in parentheses. *** p<0.01, ** p<0.05 and * p<0.1.

Table 1.8: The effect of SPEI on sex ratio at birth - the first month only

Dependent Variable	Sex ratio (1)	Sex ratio (2)	Sex ratio (3)	Sex ratio (4)
SPEI01 in month1	-0.0007**	-0.0007**	-0.0007*	-0.0007*
	(0.0004)	(0.0004)	(0.0004)	(0.0004)
Observations	1488103	1488103	1410933	1410933
Child controls		✓		✓
Household controls			✓	✓
DHS Cluster FE	✓	✓	✓	✓
Survey year FE	✓	✓	✓	✓
Month-Year Birth FE	✓	✓	✓	✓

Notes: All regressions were estimated using a multi-way fixed effects estimator. Robust standard errors clustered by DHSID are in parentheses. *** p<0.01, ** p<0.05 and * p<0.1.

Table 1.9: The interaction estimation on the probability of female and the sex ratio: 9-month in utero

Dependent Variable	female (1)	female (2)	female (3)	Sex ratio (4)	Sex ratio (5)	Sex ratio (6)
Independent:						
SPEI09 in utero	-0.0015	0.0002	-0.0003	-0.0006	0.0001	0.0002
	(0.0017)	(0.001)	(0.0013)	(0.0009)	(0.0005)	(0.0007)
Interaction:						
SPEI09 in utero x wealth	0.0004			0.0001		
	(0.0005)			(0.0003)		
SPEI09 in utero x mother work		-0.0021			-0.0013	
		(0.0016)			(0.0008)	
SPEI09 in utero x mother education			-0.0002			-0.0007
			(0.0016)			(0.0008)
Observations	1674239	1674239	1674239	1410933	1410933	1410933
Child controls	✓	✓	✓	✓	✓	✓
Household controls	✓	✓	✓	✓	✓	✓
DHS Cluster FE	✓	✓	✓	✓	✓	✓
Survey year FE	✓	✓	✓	✓	✓	✓
Month-Year Birth FE	✓	✓	✓	✓	✓	✓

Notes: All regressions were estimated using a multi-way fixed effects estimator. Robust standard errors clustered by DHSID are in parentheses. *** p<0.01, ** p<0.05 and * p<0.1.

Table 1.10: The interaction estimations on the probability of female and the sex ratio: in the first month

Dependent Variable	female (1)	female (2)	female (3)	Sex ratio (4)	Sex ratio (5)	Sex ratio (6)
Independent:						
SPEI01 in month1	-0.0003	-0.0015*	-0.0014	0.0000	-0.0007	-0.0005
	(0.0016)	(0.0009)	(0.0012)	(0.0009)	(0.0005)	(0.0006)
Interaction:						
SPEI01 in month 1 x wealth	-0.0004			-0.0002		
	(0.0005)			(0.0003)		
SPEI01 in month 1 x mother work		0.0004			0.0000	
		(0.0014)			(0.0007)	
SPEI01 in month 1 x mother education			-0.0001			-0.0003
			(0.0015)			(0.0008)
Observations	1674239	1674239	1674239	1410933	1410933	1410933
Child controls	✓	✓	✓	✓	✓	✓
Household controls	✓	✓	✓	✓	✓	✓
DHS Cluster FE	✓	✓	✓	✓	✓	✓
Survey year FE	✓	✓	✓	✓	✓	✓
Month-Year Birth FE	✓	✓	✓	✓	✓	✓

Notes: All regressions were estimated using a multi-way fixed effects estimator. Robust standard errors clustered by DHSID are in parentheses. *** p<0.01, ** p<0.05 and * p<0.1.

Table 1.11: The effect of SPEI in utero on child health: Full sample, Girls and Boys.

Dependent Variable	Height (HAZ)	Height (HAZ)	Height (HAZ)
	(1)	(2)	(3)
	All	Girls	Boys
Independent:			
SPEI09 in utero	2.423***	3.047***	2.244*
	(0.84)	(1.121)	(1.194)
Observations	191765	93664	94356
Child controls	✓	✓	✓
Household controls	✓	✓	✓
DHS Cluster FE	✓	✓	✓
Survey year FE	✓	✓	✓
Month-Year Birth FE	✓	✓	✓

Notes: All regressions were estimated using a multi-way fixed effects estimator. Robust standard errors clustered by DHSID are in parentheses. *** p<0.01, ** p<0.05 and * p<0.1.

Table 1.12: The effect of SPEI in utero on child health: Full sample

Dependent Variable	Height (HAZ) (1)	Height (HAZ) (2)	Height (HAZ) (3)	Height (HAZ) (4)
Independent:				
SPEI09 in utero	-1.497	2.308**	1.842	1.267
	(1.838)	(0.97)	(1.463)	(1.044)
Interaction:				
SPEI09 in utero x wealth	1.321**			
	(0.527)			
SPEI01 in utero x mother work		0.394		
		(1.436)		
SPEI01 in utero x mother education			0.809	
			(1.632)	
SPEI01 in utero x female				2.315*
				(1.198)
Observations	191765	191765	191765	191765
Child controls	✓	✓	✓	✓
Household controls	✓	✓	✓	✓
DHS Cluster FE	✓	✓	✓	✓
Survey year FE	✓	✓	✓	✓
Month-Year Birth FE	✓	✓	✓	✓

Notes: All regressions were estimated using a multi-way fixed effects estimator. Robust standard errors clustered by DHSID are in parentheses. *** p<0.01, ** p<0.05 and * p<0.1.

Table 1.13: SPEI expsoure on child health by month: Full sample, Girls and Boys

Dependent Variable	Height (HAZ)	Height (HAZ)	Height (HAZ)
	(1)	(2)	(3)
	All	Girls	Boys
spei01 in month 1	0.585	1.511	-0.096
	(0.662)	(0.951)	(0.954)
spei01 in month 2	0.462	0.544	0.869
	(0.632)	(0.96)	(0.958)
spei01 in month 3	0.965	0.616	0.662
	(0.678)	(0.983)	(0.984)
spei01 in month 4	2.108***	1.136	3.544***
	(0.698)	(0.951)	(1.025)
spei01 in month 5	1.261*	0.986	1.798*
	(0.661)	(0.98)	(0.991)
spei01 in month 6	1.727**	1.48	1.768*
	(0.684)	(0.961)	(1.031)
spei01 in month 7	1.353**	1.245	1.979**
	(0.667)	(0.963)	(0.999)
spei01 in month 8	2.291***	1.693*	2.566**
	(0.679)	(0.999)	(1.013)
spei01 in month 9	1.773***	2.682***	1.405
	(0.662)	(0.949)	(0.965)
Observations	191765	93664	94356
Child controls	✓	✓	✓
Household controls	✓	✓	✓
DHS Cluster FE	✓	✓	✓
Survey year FE	✓	✓	✓
Month-Year Birth FE	✓	✓	✓

Notes: All regressions were estimated using a multi-way fixed effects estimator. Robust standard errors clustered by DHSID are in parentheses. *** p<0.01, ** p<0.05 and * p<0.1.

Chapter 2

The effect of prenatal exposure to wildfire pollution on child health and the sex ratio at birth: Evidence from California *(Solo-authored chapter)*

2.1 Introduction

The fetal origins hypothesis (Barker, 1995) indicates that conditions while in utero are associated with the life-long health of newborns. A growing body of literature investigates the effect of adverse shocks on birth outcomes and documents that maternal exposure to adverse shocks negatively impacts the health of newborns in the short and long run. Trivers and Willard (1973) hypothesise that under certain well-defined conditions, women can vary their offspring's sex ratio, favouring girls in bad conditions and favouring boys in good conditions. Male embryos are also hypothesised to be more sensitive when exposed to external shocks while in utero, which leads to a higher probability of male foetal death (Noghanibehambari et al., 2020). Exposure to environmental disasters, such as wildfires and earthquakes, during pregnancy is shown to have significant effects on birth outcomes, including lower birth weight, preterm birth and an increased risk of newborns having abnormal conditions (Currie and Rossin-Slater, 2013, Holstius et al., 2012, Mccoy and Zhao, 2021, O'Donnell and Behie, 2013, Torche, 2011, Torche and Kleinhaus, 2012).

These shocks can affect females and males differently, as shown in a few related studies (Catalano et al., 2006*a*, Dagnelie et al., 2018, Valente, 2015). For instance, Maccini and Yang (2009) find that exposure to high levels of rainfall at the time of birth has no significant effect on the health of men later in life but is positively correlated with the health of women. In Chapter 1, we investigate the effect of drought exposure at different times during pregnancy and find that exposure to drought decreases birth height and that this decrease tends to be higher among female infants than male infants.

Wildfires have been considered one of the most destructive and dangerous disasters that occurs unexpectedly and uncontrollably in forests and grassy plains. Wildfires are usually caused by changes to the natural environment (e.g. high temperatures) but are sometimes associated with human

activities. In recent decades, climate change has significantly increased the global temperature, contributing to an increased probability of wildfire ignition (Bowman et al., 2020). The impacts of wildfires are particularly pronounced in populated flammable temperate and tropical zones, such as Western USA and the Amazon Rainforest (Bowman et al., 2020). Wildfires threaten humans' physical health through fire-related injuries and loss of life (Amjad et al., 2021) and mental health through the stress and anxiety of relocation, unemployment and disruption in connection to families and friends (Xiong et al., 2008). Pregnant women are one of the most sensitive groups to these wildfire-related health risks (Centers for Disease Control and Prevention (CDC), 2022*b*), resulting in children born to mothers who were exposed to external stressors during pregnancy usually having worse birth health outcomes. Exposure to external stressors induced by wildfires lowers birth weight and height and increases foetal and infant mortality (Dunkel Schetter, 2011, Hobel et al., 1999*a*, O'Donnell and Behie, 2013). The biological mechanisms behind these adverse birth outcomes may be due to maternal stress, which can affect child health outcomes through changes in immune function and neuroendocrine and behavioural mechanisms (Dunkel Schetter, 2011).

Air pollution induced by wildfires can adversely affect human health both before and after birth (Amjad et al., 2021). It has been documented that burning wood or biofuels can generate smoke that consists of extremely fine substances, such as particulate matter (PM). PM are considered harmful to human health if the particles are less than 10 micrometres (PM10) and considered extremely harmful if the particles are less than 2.5 micrometres (PM2.5), as these pose the greatest risk of being inhaled (United States Environmental Protection Agency, 2017). Exposure to fine particles while in utero is found to be associated with adverse birth outcomes. This exposure can affect foetal health indirectly through maternal health, as fine particles can get into the mother's bloodstream and pose a risk to her health. This, in turn, interferes with the nutritional intake needed for foetal development(Hobel et al., 1999*a*). Such impacts may be due to increased levels of polycyclic aromatic hydrocarbons (PAHs)–DNA adducts, a class of chemicals released when burning wood, tobacco, etc. (Centers for Disease Control and Prevention (CDC), 2022*a*), that can bind to DNA and cause genetic damage. This disrupts foetal development by interfering with the activity of the endocrine system and binding to the placenta, which reduces the flow of oxygen and nutrients (Perera et al., 1999).

Although many early studies have shown that natural disasters are one of the main contributors to a reduced male-to-female baby ratio, the effects of maternal exposure to wildfires on the birth-sex ratio remain unclear. In contrast, some research finds that wildfire exposure during pregnancy does not significantly affect the birth–sex ratio (O'Donnell and Behie, 2013). Even though the

determinants of changes in the sex ratios and birth health outcomes remain elusive both biologically and psychologically, it is evident that prenatal exposure to environmental disasters, which result in stress-related and pollution-related health issues for pregnant women, is associated with increased preterm birth and reduced birth weight (O'Donnell and Behie, 2013).

Most studies on the impact of negative shock exposure in utero focus on less institutionalised settings. To further test whether our hypothesis in Chapter 1 that drought exposure in utero results in a female-biased sex ratio at birth holds in a well-institutionalised setting where drought is not a significant concern. This study, however, uses a credibly exogenous source of pollution/stress on pregnant mothers to study the impact of shocks in early life on the sex ratio and child health in a context in which most mothers can rely on well-functioning institutions that provide timely services. To find out whether there is a difference between highly institutionalised and less institutionalised settings in the impacts of maternal exposure to adverse shocks on the birth–sex ratio and to explore how this exposure affects child health at birth in institutionalised settings, we assess the effects of wildfire exposure at different times during pregnancy on the sex ratio and child health at birth using wildfire data and the Natality birth data from California between 2000 and 2004, in which the individual-level birth data includes detailed demographic and geographic information for each birth in the sample. The United States has been through several severe wildfire disasters over the last few decades, and the prevalence of wildfires is particularly pronounced in Western USA, such as in California. For instance, in late 2003, Southern California experienced one the most devastating wildfire disasters that comprised a series of severe wildfires that happened on the same day and resulted in 3,710 homes destroyed and more than 750,000 acres of land burned (Blackwell and Tuttle, 2003).

Our results suggest that wildfire exposure during pregnancy is associated with reduced birth weight. When assessing the effects by gender, we notice that wildfire exposure tends to affect boys more strongly than girls when it occurs during the second trimester of pregnancy and within a total of whole nine months being in utero. Regarding the birth–sex ratio, in which the sex ratio is defined as the proportion of female births which is computed by the number of female births over the sum of all births for each city and month i.e. $\frac{female}{female+male}$, we do not find any significant effects of wildfire exposure while in utero on the birth–sex ratio. However, when exploring the heterogeneous effects of wildfire exposure on the sex ratio, such as the mother's education level, we observe that the sex ratio at birth increases among less educated mothers. This implies that among women with less education, relatively bad environmental conditions lead to a higher share of female births. This, to some extent,

is in line with the Trivers and Willard (1973) hypothesis.

Our study contributes to the current literature in two different ways. First, to the best of our knowledge, this is the first study to jointly examine the impact of environmental disaster exposure on the sex ratio and child health outcomes using wildfires as a natural exogenous shock. Second, we assess the effects of wildfire exposure during pregnancy in a strongly institutionalised setting to test the generality of the sex ratio findings documented in Chapter 1.

The rest of the chapter is organised as follows. Section 2.2 provides a broader review of the existing related literature. Section 2.3 describes the data. Section 2.4 presents the empirical strategy. Section 2.5 reports the estimated results. Finally, Section 2.6 provides a conclusion.

2.2 Literature review

This study relates to two broad branches of literature: the first focuses on the effects of exposure to shocks while in utero on the sex ratio and the second focuses on the effects of exposure to air wildfire/ air pollution while in utero on child health. We discuss the two branches sequentially.

2.2.1 The effect of exposure to shocks while in utero on the sex ratio

A substantial amount of studies show that exposure to adverse shocks, such as natural disasters, while in utero has been found to be correlated with an increased sex ratio, i.e. more female births than male births, and the effects are likely to depend on the timing of the exposure (Fukuda, Fukuda, Shimizu and Møller, 1998). Torche and Kleinhaus (2012) utilise the Tarapacá earthquake as a plausibly exogenous shock to evaluate the association between early life exposure to the earthquake at different times during pregnancy on birth outcomes and the birth–sex ratio. They find that exposure to the earthquake in the second and third months of pregnancy increased the probability of preterm birth, and this impact tended to affect females more significantly than males. In addition, the study finds that exposure to the earthquake in the third month of pregnancy reduced the male-to-female ratio by 5.8%.

Following this, Wilde et al. (2017) use the census and demographic and health survey data for sub-Sahara Africa to explore the effect of an ambient temperature shock at conception on the sex ratio and find that higher temperature nine months before birth is significantly associated with a higher female birth rate; more specifically, a one-degree increase in the average monthly temperature caused a 0.249% increase in the fraction of female births.

Fukuda, Fukuda, Shimizu and Møller (1998) explore the association between natural catastrophes and the sex ratio based on the data of newborns born nine months after the 1995 Kobe earthquake in Japan. Their study indicates that exposure to the Kobe earthquake significantly reduced the birth of male babies from 0.516 to 0.501. Noghanibehambari et al. (2020) use the great Tambora eruption in Indonesia in 1815 as a naturally exogenous acute stressor to examine the long-term effects of maternal exposure. The explosion caused significant environmental disruption and generated pollution, such as ash columns. Using a difference-in-difference approach, the study finds that exposure to the aftermath of the great Tambora eruption reduced literacy rates by 1.4% and increased the probability of female birth, leading to a 1% increase in the female-to-male sex ratio. Moreover, SAADAT (2008) explores the association between the sex ratio and the severe earthquake that happened in 2003 in southern Iran. The study's result shows that being born after the earthquake increased the female-biased birth–sex ratio.

In addition to utilising natural disasters as exogenous experiments to explore the effects of adverse shock exposure while in utero on the sex ratio at birth, many existing studies have investigated the correlation between social conflict events (e.g. terrorist attacks and war) and the sex ratio. For example, Bruckner et al. (2010) examine the impact of the 9/11 terrorist attacks on the sex ratio in the US and finds that male births declined following the 9/11 attacks. Furthermore, Kemkes (2006) uses the French Revolutionary War (1787–1802) as an exogenous experiment to assess how the sex ratio would change during stressful times and finds that prenatal exposure to the war significantly reduced the proportion of male births. In contrast to the many studies that have suggested that events skew the sex ratio at birth towards girls, Helle et al. (2009) jointly study the association between war, famine, ambient temperature, economic development, and total mortality rate on the sex ratio. Their study finds that male birth increased when the ambient temperature increased during World War II. In contrast, they did not find any significant association between the Finnish Civil War or the Great Finnish Famine and the sex ratio at birth.

The evidence in favour of a female-biased sex ratio in response to exposure to negative shocks while in utero is, however, not unanimous. Previous studies have shown that maternal exposure to natural disasters does not significantly skew the sex ratio towards girls. O'Donnell and Behie (2013) were the first to study the joint association between maternal stress at different times of pregnancy and birth and reproductive outcomes. They use the 2009 Victorian Black Saturday bushfires in Australia as a natural source of acute stress to assess the effects of exposure to bushfires while in utero on birth weight and the sex ratio at birth. They use the population cohort method, which

compared the outcomes in 2009 and 2006–2008 for babies born in the fire-affected and non-affected areas and find that bushfire exposure in the second and third trimesters significantly increased the probability of preterm birth and reduced birth weight. Regarding the sex ratio, they do not detect any significant effects on the sex ratio at birth if exposed to the bushfire.

2.2.2 The effects of exposure to wildfires or air pollution while in utero on child health

A strand of literature investigates the correlation between wildfire exposure and birth outcomes. A recent review of a study that evaluated several existing pieces of evidence on the association between wildfire exposure and birth outcomes concluded that prenatal wildfire exposure during late pregnancy can lead to adverse birth outcomes through air pollution and psycho-social stress induced by wildfires, resulting in low birth weight and preterm birth (Amjad et al., 2021). It has been hypothesised that prenatal stress is associated with foetal health because it can increase maternal cortisol, one of the potential mediating factors that can disrupt foetal development through the placenta (Glover, 2015). The mother and the foetus are physically connected through the placenta, which plays a central role in determining foetal health. Exposure to wildfires during pregnancy has been associated with the mother experiencing prenatal stress and depression, leading causes of increased cortisol. The cortisol then travels through the placenta to disrupt foetal development and, in turn, results in low birth weight (Kertes et al., 2016).

Research on the potential impacts of exposure to wildfires on birth outcomes in high-income countries has indicated that exposure to pollution while in utero increases the risk of adverse birth outcomes, including decreased birth weight, preterm birth and developing gestational diabetes or gestational hypertension (Abdo et al., 2019). Following this, O'Donnell and Behie (2015) explore the potential effects of wildfire exposure on child health using the sample population influenced by the 2003 Canberra wildfires in Australia. Their findings show that prenatal exposure to wildfires is associated with a higher birth weight, whereas many previous studies find the opposite effect on birth weight. They find that male foetuses exposed to wildfire tended to have a higher birth weight than those born in a non-affected year or a less exposed area.

Some studies have examined the impacts of wildfires in Colorado between 2007 and 2015. These studies explored the association between wildfires and birth outcomes either by focusing on PM2.5 exposure, in which PM2.5 is a significant source of ambient particulate matter generated by wildfires

(Abdo et al., 2019), or by comparing individuals living in the area affected by the smoke plumes with children who were born in an unaffected area under the assumption that those born in an unaffected area were less affected (Mccoy and Zhao, 2021). These studies obtained similar results, concluding that exposure to wildfires while in utero is correlated with low birth weight and the risk of preterm birth.

Holstius et al. (2012) assess the effects of exposure to wildfires in the year 2003 in Southern California on the birth outcomes of pregnant women before and after the wildfires. They find that exposure to wildfires while in utero reduced the average birth weight. The effect differs according to the gestational timing of the exposure, with the most substantial effect observed (a 9.7 g lower average birth weight) when the exposure happened during the second trimester of pregnancy.

In addition to previous studies, Malley et al. (2017) study the correlation between maternal fine particulate exposure and child health outcomes and find that exposure to PM2.5 during pregnancy was substantially associated with preterm birth. A systematic review of studies on infant health and air pollution conducted by Glinianaia et al. (2004) has indicated that several epidemiological studies confirm that maternal exposure to air pollution strongly correlates with adverse health outcomes for the child (e.g. preterm birth or low birth weight). Currie and Neidell (2005) assess the impact of exposure to air pollution on infant death using individual-level data and pollution data of three different pollutants (ozone, carbon monoxide and particulate matter 10) measured weekly in California over the 1990s. Their study finds that a reduction in carbon monoxide resulted in a significant decrease in infant mortality.

Perera et al. (1999) examine the effect of exposure to environmental contaminants while in utero and find that ambient air pollution is associated with low birth weight. Their study indicates that exposure to air pollution is linked to the level of PAH-DNA adducts. PAHs can bind to DNA, resulting in genetic damage, and are highly transplacental from mother to foetus, therefore affecting foetal development. Animal experiments that explore the effects of exposure to PAHs during pregnancy also confirm the correlation i.e. exposure to PAHs while in utero is correlated with low birth weight, stillbirth and abnormal foetal births (Barbieri et al., 1986, Bui et al., 1986, Rigdon and Rennels, 1964).

Beyond wildfires and air pollution, an extensive portion of literature investigates the effects of exposure to shocks while in utero on child health using a variety of exogenous shocks, such as fasting during Ramadan (Awwad et al., 2012, Chaudhry and Mir, 2021, Dikensoy et al., 2008, Malihe and Shahla, 2001, Savitri et al., 2014, Somayeh et al., 2013), earthquakes (Torche, 2011), famine (Chen

and Zhou, 2007, Dercon and Porter, 2014, Lumey, 1992, Roseboom et al., 2011, 2000), floods and conflicts (Nasir, 2021, Rosales-Rueda, 2018). The vast majority of these studies report detrimental effects on children's health, generally measured by the HAZ score or birth weight; they document that exposure to negative shocks while in utero is adversely correlated with birth outcomes, and such effects tend to be more significant when exposed during early pregnancy. A subset of these studies explores the effect of prenatal exposure to air pollution on child health and has shown a causal relationship between exposure to air pollution while in utero and health outcomes at birth.

Most of the existing literature provides strong evidence for prenatal exposure to adverse shocks (e.g. famine, conflict, earthquakes, wildfires and air pollution) being correlated with adverse birth outcomes, such as low birth weight. However, some studies argue that maternal exposure to adverse shocks has no significant effect on birth outcomes. Additionally, few or no studies jointly examine the impacts of maternal exposure on both the health outcomes of the child and the birth sex ratio using wildfires as a stress event. This study aims to provide appropriate solutions in addressing the exposure timing issues to evaluate the impacts of adverse shocks in utero on child health outcomes and sex ratios at birth. We attempt to fill the study gap by investigating the effects of wildfires using individual-level data from California.

2.3 Data

The datasets used in this study were taken from three different data sources: (i) the National Bureau of Economic Research, (ii) Monitoring Trends in Burn Severity (MTBS) and (iii) the California Air Resources Board.

2.3.1 Data on births

We used the Vital statistics Natality Birth Data[1] from the National Bureau of Economic Research (NBER) website, on which the National Center for Health Statistics at the California Department of Health Care Service initially provided the natality data on individuals born between 2000 and 2004 in the state of California for public use. Due to the data restrictions imposed by the state, the birth data from 2005 onward does not include any geographic information (e.g. county). Hence, this study focuses only on the individual-level birth data from 2000 to 2004. The birth records contain detailed

[1]Accessed in Oct 2020: Vital Statistics Natality Birth Data. https://www.nber.org/research/data/vital-statistics-natality-birth-data.

demographic information on the children (e.g. sex of the child, birth weight and birth order) and the mothers (e.g. the mother's age, race, marital status and education).

2.3.2 Data on wildfires

We study the wildfires in the state of California from 2000 to 2004. The detailed data on wildfires was collected by MTBS[2], an interagency programme that consistently maps the burn severity and extent of large fires across all lands of the United States from 1984 to the present. The data on each wildfire downloaded from MTBS contains information on the fire ignition date, extension (in acres) and location (in longitude and latitude). To assign the wildfires to each individual in the sample at the city level, we first loaded the wildfire data to ArcGIS[3], which shows each wildfire's exact location and burned boundary directly through the web map services provided by MTBS. Following this, we aggregated and restricted the individual-level data to the city level. We then assigned each city in the dataset its closest wildfire by measuring the distance between each city and the corresponding closest wildfire using the measure tool in ArcGIS and included the wildfires that may also have affected each city if the wildfire plumes covered the city. The wildfire plumes were constructed in ArcGIS according to the spatial extent of the visible smoke, which was obtained by overlaying the daily satellite images for each fire produced by the Terra satellite at a resolution of 250 m within the first five days following the start of each wildfire on a map of the US. The satellite images were created using the worldview snapshots tool from NASA Earth data. Thus, each individual in the data was assigned its corresponding wildfires at the city level, and each wildfire had its unique name.

Figures B1 and B2 are maps showing 22 wildfires that hit California between 2000 and 2004 and their corresponding smoke plumes. Figure B1 shows the wildfires in the northern part of California, while Figure B2 shows the wildfires in the south.

As a measure of wildfire exposure, we calculate the total number of days that each wildfire lasted in each month from 2000 to 2004 based on the ignition date and end date of each wildfire. The end date of the wildfire was determined from the daily satellite images created by NASA Earth data[4]. Figure B3 presents the geographic distribution of the city-level average number of days exposed to wildfires and the location of wildfires in 2003. Finally, we merged the wildfire dataset, which includes the number of days of wildfire exposure in each month between 2000 and 2004, with the birth data by city name, wildfire name and the child's month-year of birth. The resulting main analysis

[2]Accessed in Oct 2020: MTBS. https://www.mtbs.gov.
[3]ArcGIS is a complete online mapping and analysis tool that allows us to transform data into interactive web maps.
[4]Accessed in Oct 2020: NASA Earth data. https://earthdata.nasa.gov/faq/worldview-snapshots-faq#base-layers.

sample consisted of 952,165 births. Given the month-year of birth and the wildfire occurrence, we calculated whether a child was exposed to a wildfire during a total of nine months in utero (Utero) and whether the exposure happened during the first (T1), second (T2) or third (T3) trimesters by counting forwards from the date of conception.

2.3.3 Data on air pollution

Besides addressing wildfires directly, we also assess the wildfire effects on birth outcomes using data on the particles present in the air (PM2.5). Our data on PM2.5 is collected from the California Air Resources Board using the Air Quality Data Query tool [5] they provide. This is a publicly used database that provides information on air quality. The daily average PM2.5 data we downloaded includes the name of each monitor station where the PM2.5 level was captured and the longitude and latitude coordinates of each monitor station. We first located each monitor station on the map using ArcGIS based on their GPS coordinates. We then assigned each city in the data to its closest monitor station and merged this data with the birth data of the city, the monitor station name and each child's month and year of birth.

To test the effects of wildfire exposure on the birth outcomes using PM2.5, we generated new variables that captured the total number of days that the PM2.5 was above a certain threshold within the wildfire periods. According to the Air Quality Index provided by the US Environmental Protection Agency, we focused on using three different cutoffs of PM2.5 as measurements: PM2.5>12.1 (yellow), PM2.5>35.5 (orange) and PM2.5>55.5 (red). We calculated whether a child was exposed to a wildfire or bad air during their nine months in utero (Utero) and whether the exposure occurred during the first (T1), second (T2) or third (T3) trimester by counting forward from the month of conception based on the information on the children's month-year of birth and the month-year occurrence of the wildfire (Currie and Vogl, 2013).

2.3.4 Outcome Variable

The main outcome of interest is the 'Sex ratio', a continuous variable used to examine the effects of wildfire exposure during pregnancy on the sex ratio at birth. To test the correlation between wildfire exposure in utero and sex ratio at birth, we collapse the data at the city and month-year of birth level to derive the total number of female and male births in a given place and given month-year of

[5] Accessed in Sept 2020: Air Quality Data (PST) Query Tool. https://www.arb.ca.gov/aqmis2/aqdselect.php?tab=daily.

birth and defined the sex ratio as $\frac{female}{female+male}$ to get the share of female births in a given city and month-year.

We are also interested in exploring the effect of air pollution on 'birth weight', an anthropometric measure used to systematically predict the health status of newborns (Clark et al., 2021). Birth weight has been widely used as a proxy for a baby's health to identify the consequences of exposure to adverse shocks in early life on health and socioeconomic outcomes, such as infant mortality, cognitive ability and earnings in both childhood and adulthood (Almond et al., 2005, Black et al., 2007, McCormick, 1985, Orchinik et al., 2011).

Table 2.1 presents the descriptive statistics. Panel A summarises the statistics of the sex ratio data obtained by collapsing the birth data at the city level and birth month-year level. Panel B summarises the statistics for the individual-level birth data.

2.4 Empirical strategy

We implemented two separate sets of regressions. First, we explored the effect of wildfire exposure on the birth–sex ratio using collapsed sample data to run panel-level regressions, in which the collapsed data was aggregated at the city-month level. Second, we ran cross-sectional regressions to examine the correlation between wildfire exposure while in utero and birth weight.

2.4.1 The effect of wildfire exposure in utero on sex ratio

To test the effects of in utero exposure to the wildfire on the sex ratio, we fit the data to linear fixed-effects panel regressions. This is to see whether exposure to wildfire in utero would result in a skewed sex ratio. In this study, we defined wildfire exposure as the timing of exposure during pregnancy with two categories: a total of 9-month in utero and Trimesters. Formally, we start to estimate the following model (2.1)

$$Sexratio_{tc} = \beta_0 + \beta_{utero} Utero_{tc} + \beta_z Z_{tc} + \omega_t + \mu_c + \epsilon_{tc} \tag{2.1}$$

where the subscripts c and t denote the city and month-year of birth, respectively. $Sexratio_{tc}$ is a continuous outcome variable of interest that represents the sex ratio of a given city c and a given month-year of birth t. Sex ratio data was obtained by collapsing the individual-level data at the city

level and month-year of birth level. The sex ratio was defined as the total number of females divided by the total number of births in the same city, same month and same year (i.e. $\frac{female_{tc}}{male_{tc}+female_{tc}}$). $Utero_{tc}$ is the main explanatory variable of interest, measured by the number of days exposed to the wildfire during the entire nine months in utero. The corresponding coefficient β_{utero} is the impact of a one SD increase in wildfire exposure on the sex ratio at birth. Z_{tc} is a set of controls for a vector of both the mother's and the child's characteristics, including the mother's marital status, the mother's race and education level, the mother's age, the infant's sex and gestational age and the live-birth order, which are all averaged at the city level. ω_t represents the month-year of birth fixed effects included to control for time-varying unobserved factors. μ_c is a set of city fixed effects.

Month-year of birth fixed effects were included to capture common time shocks for the children born in the same month. The city fixed effects were included to account for unobserved time-invariant heterogeneity across the city and to capture the other local factors that may affect health outcomes. ϵ_{tc} is a standard error term. Cluster-robust standard errors are clustered at the city level throughout the paper.

We also present estimates by decomposing $Utero_{tc}$ by pregnancy trimester to capture the specific effects of exposure to wildfire depending on the phase it affects the pregnancy. Formally, we amend the model as follows.

$$Sexratio_{tc} = \beta_0 + \sum_{k=1}^{3} \beta_t T_{k,tc} + \beta_z Z_{tc} + \omega_t + \mu_c + \epsilon_{tc} \tag{2.2}$$

Where the values of k indicates the pregnancy trimester.

2.4.2 The effects of wildfire exposure in utero on birth weight

To further explore the association between wildfire exposure at different times of pregnancy and birth weight, we employed the following cross-sectional regressions.

$$Birthweight_{itc} = \beta_0 + \beta_{utero} Utero_{itc} + \beta_z Z_i + \omega_t + \mu_c + \epsilon_{itc} \tag{2.3}$$

where $Birthweight_{itc}$ is the outcome variable that represents the health status of child i, living in city c and born in time t. $Utero_{itc}$ is the main explanatory variable of interest measured in days

exposed to wildfires during the entire nine months of the pregnancy. The indices i and t stand for birth i at time t in city c of the exposure during pregnancy. Z_i is a set of maternal and birth characteristics controls.

Similarly, we also decompose the $Utero_{itc}$ by pregnancy trimesters, to capture the specific effects of exposure to wildfire depending on the phase it affects the pregnancy. Formally, we amend the model as follows:

$$Birthweight_{itc} = \beta_0 + \sum_{k=1}^{3} \beta_t T_{k,itc} + \beta_z Z_i + \omega_t + \mu_c + \epsilon_{tc} \tag{2.4}$$

where the value of k indicates the pregnancy trimester

2.5 Results

2.5.1 Changes to the sex ratio

Table 2.2 presents the estimated impacts of the number of days exposed to wildfire at different times during pregnancy on the sex ratio at birth. Columns 1 and 2 show the impacts of wildfire exposure on the sex ratio at birth by focusing on the total nine months in utero with and without all controls. Columns 3 and 4 are the estimated effects of the number of days exposed to wildfires during different trimesters. The estimates show that wildfire exposure has no significant effect on the sex ratio at birth. Alternatively, we replicated all regressions using PM2.5 as a measure of exposure to wildfire. We explored the impacts of wildfire exposure while in utero during different trimesters of pregnancy on the birth–sex ratio by using the different cutoffs of air pollution: pm2.5>12.1 (yellow), pm2.5>35.5 (orange) and pm2.5>55.5 (red). The estimated results shown in Table B1 and Table B2 in the appendix have similar patterns.

Our results for the change in the sex ratio are different from many early studies that show that experiencing stressful events during pregnancy is associated with a reduced male-to-female birth ratio. There are two potential explanations for this difference. One possible explanation involves the timing of the effect (O'Donnell and Behie, 2013), as the parents may have been affected even before conception. Research conducted by Lyster (1974) indicates that observed newborn sex ratio changes occurred around 12 months after the London smog. Fukuda, Fukuda, Shimizu and Møller (1998),

however, show that a reduction in the sex ratio appeared nine months after the Kobe earthquake in Japan, and they argued that such a difference between their findings and Lyster's may be due to the duration of the disasters, i.e. smog lasts longer than an earthquake. The second possible explanation may have to do with the different settings – mothers in California have steady access to medical services, which lessens the occurrence of bad outcomes. Moreover, previous studies have also shown that changes in the birth–sex ratio are associated with parental stress. Maternal stress caused by exogenous shocks changes the hormone levels of pregnant women (Hobel et al., 1999*b*), resulting in an increased probability of preterm birth and spontaneous abortions of male foetuses (Byrne et al., 1987). Following this, Fukuda et al. (1996) examine the relationship between the earthquake and the sperm motility of men. Their study concludes that an acute stressful event reduces sperm motility, which may result in changes to the birth–sex ratio. Since the birth–sex ratio seems more affected by long-term stress experienced by both mothers and fathers, evaluating the effect of wildfire exposure while in utero on the birth–sex ratio using birth data of newborns conceived after the wildfire period may generate more meaningful results. However, due to the data limitation, the birth data from 2005 onward does not contain geographic information, so it cannot be analysed.

2.5.2 Changes to birth weight

The estimated results for the specification of equations 2.3 and 2.4 are presented in Table 2.3 and Table 2.4. In each table, columns 1, 2 and 3 show the results estimated without controls for the whole sample population, boys and girls, respectively. Columns 4, 5 and 6 show the estimated results after including all sets of controls for the whole sample population, boys and girls, respectively. This study finds small but significant decreases in birth weight, and our findings are consistent with many earlier studies that concluded that exposure to environmental disasters during the in-utero period is associated with worse child health outcomes at birth.

Table 2.3 reports the estimated effects of wildfire exposure by focusing on the timing of exposure by a total of 9 months during pregnancy. The results are consistent, which shows that wildfire exposure during pregnancy significantly affects child health outcomes at birth, and boys seem to be more affected. As shown in columns 1 to 3, no significant effect was found when all controls were excluded. In contrast, the estimated results in columns 4 to 6 suggest that a one SD [6] increase in the number of days exposed to wildfire during the entire pregnancy period results in a statistically significant decrease in the birth weight of 0.3% ($=\frac{-0.2\times 6.358}{487.353}$) for the whole sample group. Similarly,

[6] A standardized regression coefficient, calculated using $\beta_i \times \frac{Standard deviation (SD) of X_i}{Standard deviation (SD) of Y}$

the effect appears to be more significant among male infants than female infants. In addition, we further tested the effect of wildfire exposure using PM2.5 as an alternative measure, and the estimated results that indicate similar patterns are shown in Table B7.

The estimates in columns 1, 2 and 3 of Table 2.4 indicate that a one SD increase in wildfire exposure during the second trimester significantly decreased the birth weight by 0.2% ($=\frac{-0.3\times3.77}{487.353}$) for the whole sample group and by 0.4% ($=\frac{-0.5\times3.77}{487.353}$) for boys, but there was no significant effect on girls. In Columns 4, 5 and 6, we controlled for a vector of the child's and the mother's characteristics. The estimates in column 4 show that with a one SD increase in fire exposure, the birth weight decreased by 0.3% ($=\frac{-0.45\times3.77}{487.353}$) for the whole sample group. Columns 5 and 6 show the estimates for the boy and girl sub-samples. Experiencing wildfire while in utero significantly affects boys' health at birth. Specifically, a one SD increase in the number of days exposed to wildfire in the second trimester is associated with a 0.5% ($=\frac{-0.7\times3.77}{487.353}$) decrease in the birth weight of boys. However, we did not find any significant effects on the birth weight of girls. The significance of the estimates increases when additional controls are added. Our results are, to some extent, in line with O'Donnell and Behie (2013)'s study, which found that exposure to fire during the second or third trimester is associated with lower birth weight.

2.5.3 Heterogeneous analysis

We assessed the heterogeneous impacts of wildfire exposure in utero on the birth–sex ratio and child health at birth by the mother's level of education and the mother's ethnicity. The estimated results of the effect on the birth–sex ratio are reported in Table 2.5 to Table 2.8, and Table 2.9 to Table 2.12 are the estimates of child health outcomes.

We first explored the effects of wildfire exposure during pregnancy on the sex ratio at birth to see whether the effects differ based on the mother's level of education. Here, we defined a less educated mother as a mother who had 12 or fewer years of education. More educated mothers were defined as having more than 12 years of education. The evidence presented in Table 2.2 shows that wildfire exposure during different trimesters has no significant effect on the birth–sex ratio. However, when we looked at the heterogeneous effects, i.e. the level of the mother's education, our findings showed that the impacts are more significant for less educated mothers. We first assessed the heterogeneous impact of wildfire exposure on the birth–sex ratio by the mother's education level based on a total of 9 months in utero. The estimates are provided in Table 2.5. However, we did not find any significant effects that differ between these two groups. Table 2.6 provides evidence that

less educated mothers are more likely to give birth to girls when they experience adverse conditions, and the effect is particularly significant in response to wildlife exposure during the second trimester of pregnancy.

Second, we assessed whether the impact of exposure to wildfires while in utero differs based on the mother's ethnicity. There were two categories for the mother's ethnicity: Hispanic and Other. The ethnicities that fall under Other include non-Hispanic Whites, non-Hispanic Blacks and non-Hispanic other races. We first examined the effects by looking at a total of nine months in utero. The evidence presented in Table 2.7 does not show any significant heterogeneous impact the of mother's ethnicity on the birth–sex ratio. We additionally explored the effects by trimester, and the estimated results in Table 2.8 indicate that exposure to wildfires during the second trimester significantly affects the birth–sex ratio among mothers from non-Hispanic groups compared to mothers from the Hispanic group. The estimates are similar when assessing the effects of air pollution at different cutoffs.

Regarding the heterogeneous effects on child health outcomes, we first investigated whether wildfire affects children born to mothers with less education and those born to mothers with more education differently. Table 2.9 shows the heterogeneous effects by a total of 9-month period in utero; the results do not indicate any significant impact for either group. Additionally, the estimates in Table 2.10 show that wildfire exposure in the second trimester of pregnancy significantly affects children's birth weight regardless of the mother's education level, but the mother's education level did not have a significant impact on birth weight for the whole sample group. However, when we assessed the impacts by gender, the estimates show that boys born to less educated mothers seem to be more affected when exposure happens in both the first and second trimesters, while no significant heterogeneous effects were found for girls.

Following the mother's education level, we further investigated the heterogeneous impacts of wildfire exposure while in utero on child health outcomes based on the mother's ethnicity. Table 2.11 reports the impacts of exposure happening within a total of 9 months in utero for children born to Hispanic and non-Hispanic mothers. The estimated results suggest that there are no significant effects when the consequences of wildfire exposure are assessed based on the mother's ethnicity for the whole sample group, boys and girls, respectively. Next, to further examine whether the heterogeneous effects change with the timing of the exposure, we explored the impact by trimester. The results in Table 2.12 suggest that no significant heterogeneous effect was found based on the mother's ethnicity for the whole sample group. However, our results indicate that wildfire exposure in the first trimester of pregnancy impacts girls born to non-Hispanic mothers.

To strengthen the confidence in the findings of this study, we conducted a set of heterogeneous analyses using the air pollution index as a measure of wildfire exposure to examine whether there were any differential effects in our baseline results based on the mothers' education levels and ethnic groups. These results are shown in Table B3 to Table B6 and Table B8 to Table B19, and the results indicate similar patterns.

2.6 Conclusions

The hypothesis by Trivers and Willard (1973) states that exposure to adverse shocks in utero is linked to reduced male births when compared to female births. In this chapter, we tested whether shocks while in utero have a relevant effect on the sex ratio for settings in which pregnant mothers can have regular medical monitoring during their pregnancy. We used detailed individual-level birth data obtained from the Public Use Data Archive of NBER for children born between 2000 and 2004 in the state of California, USA.

In general, our analysis showed that maternal exposure to wildfire events in highly institutionalised settings does not seem to impact the birth–sex ratio significantly like it does in developing countries. This may be due to either the timing of the exposure, i.e. effects on the sex ratio may be delayed and will only show up after the adverse shock occurred (O'Donnell and Behie, 2013), or due to the fact that mothers in well-institutionalized countries generally have access to advanced and steady medical care, which in turn may prevent the occurrence of negative birth outcomes. Nevertheless, when we focused on the more disadvantaged categories, i.e. exploring the heterogeneous impacts on the birth–sex ratio for more-educated mothers and less-educated mothers, we found that wildfire exposure in the second trimester increases the proportion of female births to mothers with less education. Using the air quality index as an alternative measure of wildfires to assess the heterogeneous impacts on the birth–sex ratio did not seem to change the results. Tests on the heterogeneous analysis by the mother's education on the birth–sex ratio suggest that maternal adverse shock exposure leads to a sex ratio skewed towards girls. Hence, the selection at birth tends to occur among less educated mothers. This finding, to some extent, is in line with Trivers and Willard (1973)'s hypothesis.

Although we did not find many significant impacts of wildfire exposure on the birth–sex ratio when we looked at exposure happening at different times during pregnancy, we still observed that women exposed to wildfires while pregnant are likely to have babies with lower birth weights, and these

impacts are particularly pronounced when exposure happens in the second trimester of pregnancy. By estimating the impacts on children by gender, we noticed that boys seem more affected when exposure happens in the second trimester than girls. The conclusion remains unchanged when focusing on the effects of exposure happening at any point throughout pregnancy (a total of 9-month in utero), i.e. boys are more affected. Our heterogeneity analyses also indicated that wildfire exposure tends to more strongly affect boys born to less educated mothers.

This study allowed us to investigate the impacts of wildfire exposure during the in-utero period on both the birth–sex ratio and child health outcomes using detailed birth data from the US. Specifically, this study investigated how exposure to adverse shocks while in utero affects the birth–sex ratio and birth weights in a more developed setting, which also allowed us to provide further evidence on the consequences of adverse maternal shocks on birth selection. Regarding the policy implications, our study provides evidence that policy interventions aimed at tackling the potential consequences of environmental disasters such as wildfires should be considered. In addition, health interventions, such as therapy and medication, are also needed to protect pregnant women from depression, anxiety or stress caused by wildfires.

Tables

Table 2.1: Descriptive statistics.

Variable	N	Mean	SD	Min	Max
Panel A: Panel-level regressions on sex ratio					
Sex ratio	2532	.49	.037	.286	.629
wildfire exposure					
T1	2532	.818	3.211	0	41
T2	2532	.805	3.198	0	41
T3	2532	.795	3.184	0	41
Utero	2532	2.417	5.472	0	41
PM2.5 exposure >12.1 (yellow)					
T1	2492	.34	1.509	0	27
T2	2497	.337	1.506	0	27
T3	2503	.338	1.502	0	27
Utero	2464	1.017	2.629	0	27
PM2.5 exposure >35.5 (orange)					
T1	2492	.121	.601	0	8
T2	2497	.119	.598	0	8
T3	2503	.115	.594	0	8
Utero	2464	.353	1.021	0	8
PM2.5 exposure >55.5 (red)					
T1	2492	.053	.343	0	3
T2	2497	.052	.342	0	3
T3	2503	.052	.341	0	3
Utero	2464	.159	.582	0	3
Marital status of mother	2532	.69	.117	.394	1
Maternal ethnicity					

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Table 2.1 – continued from previous page

Variable	N	Mean	SD	Min	Max
1 Hispanic	2532	.512	.203	.087	.972
2 Other races	2532	.478	.205	.028	.907
Maternal education					
1 >12 years of education	2532	.525	.181	.053	.884
2 <12 years of education	2532	.453	.179	.116	.947
Maternal age					
1 <20yrs	2532	.086	.044	0	.219
2 20-24yrs	2532	.218	.074	0	.384
3 25-34yrs	2532	.526	.07	.361	1
4 35-44yrs	2532	.167	.065	0	.458
Birth order					
1 first live birth	2532	.404	.065	.214	.676
2 second live birth	2532	.321	.043	0	.5
3 third live birth	2532	.167	.041	0	.545
Gestational weeks					
36	2532	.043	.017	0	.176
37	2532	.088	.024	0	.357
38	2532	.192	.038	0	.5
39	2532	.285	.038	0	.524
40	2532	.239	.038	.071	.429
41	2532	.116	.031	0	.273
Panel B: cross-sectional regressions on birth weight					
Birth weight	952165	3386.25	487.353	1001	5995
wildfire exposure					
T1	952165	.91	3.774	0	41
T2	952165	.904	3.77	0	41
T3	952165	.967	3.871	0	41
Utero	952165	2.782	6.538	0	41

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Table 2.1 – continued from previous page

Variable	N	Mean	SD	Min	Max
PM2.5 exposure >12.1(yellow)					
T1	936721	.505	2.185	0	27
T2	937566	.5	2.176	0	27
T3	938719	.532	2.217	0	27
Utero	927231	1.551	3.808	0	27
PM2.5 exposure >35.5(orange)					
T1	936721	.196	.866	0	8
T2	937566	.196	.868	0	8
T3	938719	.207	.892	0	8
Utero	927231	.603	1.496	0	8
PM2.5 exposure >55.5(red)					
T1	936721	.074	.435	0	3
T2	937566	.074	.435	0	3
T3	938719	.078	.449	0	3
Utero	927231	.229	.743	0	3
Marital status of mother	952165	.65	.477	0	1
Child sex	952165	.509	.5	0	1
Maternal ethnicity					
1 Hispanic	952165	.532	.499	0	1
2 Other races	952165	.468	.499	0	1
Maternal education					
1 >12 years of education	952165	.422	.494	0	1
2 <12 years of education	952165	.578	.494	0	1
Maternal age					
1 <20yrs	952165	.096	.295	0	1
2 20-24yrs	952165	.231	.421	0	1
3 25-34yrs	952165	.511	.5	0	1
4 35-44yrs	952165	.16	.366	0	1
5 45yrs+	952165	.002	.043	0	1

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Table 2.1 – continued from previous page

Variable	**N**	**Mean**	**SD**	**Min**	**Max**
Birth order					
1 first live birth	952165	.399	.49	0	1
2 second live birth	952165	.314	.464	0	1
3 third live birth	952165	.169	.375	0	1
4 fourth or more live births	952165	.118	.322	0	1
Gestational weeks					
36	952165	.044	.204	0	1
37	952165	.088	.284	0	1
38	952165	.189	.392	0	1
39	952165	.283	.45	0	1
40	952165	.241	.428	0	1
41	952165	.117	.321	0	1
42	952165	.038	.19	0	1

Note: Individual child data from the Natality Birth Data for children aged below five and born between the years 2000 and 2004 was collected from the National Bureau of Economic Research website.

Table 2.2: The effect of wildfire exposure on the sex ratio by nine months in utero and by trimesters

	Sex ratio (1)	Sex ratio (2)	Sex ratio (3)	Sex ratio (4)
Utero	-.00001	-.00003		
	(.00017)	(.00016)		
T1			.00033	.00031
			(.00027)	(.0003)
T2			-.00025	-.00026
			(.00024)	(.00023)
T3			-.00012	-.00015
			(.00034)	(.00033)
Observations	2532	2532	2532	2532
All Controls		✓		✓
City Fixed effects	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. The column headings represent the main outcome variable. The sex ratio is defined as female/(male+female). The controls include the marital status of the mother, the child's sex, maternal ethnicity, maternal education, maternal age, birth order and number of gestational weeks. The fixed effects include month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table 2.3: The effect of wildfire exposure during pregnancy on birth weight

	Birth weight All (1)	Birth weight Boys (2)	Birth weight Girls (3)	Birth weight All (4)	Birth weight Boys (5)	Birth weight Girls (6)
Utero fire	-.14957	-.17598	-.13739	-.23668*	-.25632**	-.21608
	(.10147)	(.13951)	(.19544)	(.13023)	(.11462)	(.20066)
Observations	952165	484798	467367	952165	484798	467367
All Controls				✓	✓	✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. The column headings represent the dependent variables. The controls include the marital status of the mother, the child's sex, maternal ethnicity, maternal education, maternal age, the birth order and number of gestational weeks. The fixed effects include the month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table 2.4: The effect of wildfire exposure on birth weight by trimester

	Birth weight All (1)	Birth weight Boys (2)	Birth weight Girls (3)	Birth weight All (4)	Birth weight Boys (5)	Birth weight Girls (6)
T1 fire	-.27443	-.16865	-.3965	-.30015	-.18639	-.41668
	(.26033)	(.28219)	(.33627)	(.28517)	(.27419)	(.35419)
T2 fire	-.30476**	-.51243**	-.12086	-.45235***	-.67867***	-.2234
	(.14665)	(.19307)	(.18839)	(.14598)	(.15051)	(.18818)
T3 fire	.11369	.1337	.08726	.02575	.07458	-.02327
	(.12951)	(.21857)	(.2817)	(.1025)	(.16548)	(.23028)
Observations	952165	484798	467367	952165	484798	467367
All Controls				✓	✓	✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. The column headings represent the dependent variables. The controls include the marital status of the mother, the child's sex, maternal ethnicity, maternal education, maternal age, birth order and number of gestational weeks. The fixed effects include the month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table 2.5: The interaction estimations based on the mother's education level during pregnancy

	Number of days to wildfire Sex ratio (1)	Number of days to wildfire Sex ratio (2)	PM2.5>12.1(Yellow) Sex ratio (3)	PM2.5>12.1(Yellow) Sex ratio (4)	PM2.5>35.5(orange) Sex ratio (5)	PM2.5>35.5(orange) Sex ratio (6)	PM2.5>55.5(Red) Sex ratio (7)	PM2.5>55.5(Red) Sex ratio (8)
Utero	-.00027	-.00041	-.00062	-.00092	-.00186	-.00219	-.00453	-.00471
	(.00038)	(.0004)	(.00099)	(.00098)	(.00271)	(.0027)	(.00585)	(.00581)
<12yrs edu	-.02598	-.02947	-.0271	-.02957	-.02764	-.02975	-.02832	-.0304
	(.02975)	(.03024)	(.03026)	(.03131)	(.03008)	(.0316)	(.0298)	(.03129)
Interactions								
Reference: >12yrs edu								
Utero x <12yrs edu	.00047	.00073	.00159	.00206	.00468	.0052	.01	.01016
	(.00066)	(.00068)	(.00201)	(.00198)	(.0047)	(.00472)	(.0099)	(.00995)
Observations	2532	2532	2464	2464	2464	2464	2464	2464
All Controls		✓		✓		✓		✓
City Fixed effects	✓	✓	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. The column headings represent the dependent variables. The controls include the marital status of the mother, the child's sex, maternal ethnicity, maternal education, maternal age, birth order and number of gestational weeks. The fixed effects include the month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table 2.6: The interaction estimations based on the mother's education level by trimester

	Number of days to wildfire Sex ratio (1)	Number of days to wildfire Sex ratio (2)	PM2.5>12.1(Yellow) Sex ratio (3)	PM2.5>12.1(Yellow) Sex ratio (4)	PM2.5>35.5(orange) Sex ratio (5)	PM2.5>35.5(orange) Sex ratio (6)	PM2.5>55.5(Red) Sex ratio (7)	PM2.5>55.5(Red) Sex ratio (8)
T1	.00059	.00041	-.00151	-.00201	-.00227	-.00295	-.00708	-.00758
	(.00072)	(.00075)	(.00166)	(.00157)	(.0047)	(.00461)	(.00967)	(.00943)
T2	-.00217***	-.00231***	-.00291	-.00317*	-.01064**	-.0111**	-.01562*	-.01596*
	(.00081)	(.00084)	(.00182)	(.00185)	(.00429)	(.00448)	(.00784)	(.00814)
T3	.00074	.00064	.00303**	.00287**	.00709*	.00716*	.00927	.00957
	(.00086)	(.00084)	(.00134)	(.00139)	(.00399)	(.00393)	(.00754)	(.00736)
<12yrs edu	-.0262	-.02948	-.02734	-.02941	-.02763	-.02967	-.02829	-.03107
	(.0301)	(.03047)	(.03025)	(.03108)	(.03014)	(.03138)	(.0297)	(.03096)
Interactions								
Reference: >12yrs edu								
T1 x <12yrs edu	-.00056	-.0002	.0033	.00422	.00616	.00773	.0165	.01774
	(.0013)	(.00129)	(.00337)	(.00314)	(.00862)	(.00833)	(.01536)	(.01493)
T2 x <12yrs edu	.00363***	.0039***	.00532	.00569	.01825**	.01884**	.02387*	.02417*
	(.00132)	(.00137)	(.00385)	(.0039)	(.00765)	(.00798)	(.01364)	(.01411)
T3 x <12yrs edu	-.00168	-.00153	-.0045*	-.00434	-.00947	-.00983	-.01033	-.01133
	(.00136)	(.00134)	(.00262)	(.00271)	(.00648)	(.00647)	(.01264)	(.0124)
Observations	2532	2532	2464	2464	2464	2464	2464	2464
All Controls		✓		✓		✓		✓
City Fixed effects	✓	✓	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. The column headings represent the dependent variables. The controls include the marital status of the mother, the child's sex, maternal ethnicity, maternal education, maternal age, birth order and number of gestational weeks. The fixed effects include the month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table 2.7: The interaction estimations based on the mother's ethnicity during pregnancy

	Number of days to wildfire Sex ratio (1)	Number of days to wildfire Sex ratio (2)	PM2.5>12.1(Yellow) Sex ratio (3)	PM2.5>12.1(Yellow) Sex ratio (4)	PM2.5>35.5(orange) Sex ratio (5)	PM2.5>35.5(orange) Sex ratio (6)	PM2.5>55.5(Red) Sex ratio (7)	PM2.5>55.5(Red) Sex ratio (8)
Utero	-.00035	-.00046	-.00103	-.00128	-.00177	-.00219	-.00555	-.00588
	(.00046)	(.00046)	(.00098)	(.001)	(.00319)	(.00316)	(.00633)	(.00631)
ethnic Others	-.01929	-.0134	-.02698	-.01969	-.0253	-.01798	-.02662	-.019
	(.02093)	(.02716)	(.02286)	(.03003)	(.0227)	(.02982)	(.02277)	(.02989)
Interactions								
Reference: ethnic Hispanic								
Utero x ethnic Others	.00063	.00081	.00259	.00289	.00463	.0052	.01262	.01291
	(.00071)	(.00073)	(.00189)	(.00194)	(.00531)	(.00532)	(.01054)	(.01059)
Observations	2532	2532	2464	2464	2464	2464	2464	2464
All Controls		✓		✓		✓		✓
City Fixed effects	✓	✓	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. The column headings represent the dependent variables. The controls include the marital status of the mother, the child's sex, maternal ethnicity, maternal education, maternal age, birth order and number of gestational weeks. The fixed effects include the month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table 2.8: The interaction estimations based on the mother's ethnicity by trimester

	Number of days to wildfire Sex ratio (1)	Number of days to wildfire Sex ratio (2)	PM2.5>12.1(Yellow) Sex ratio (3)	PM2.5>12.1(Yellow) Sex ratio (4)	PM2.5>35.5(orange) Sex ratio (5)	PM2.5>35.5(orange) Sex ratio (6)	PM2.5>55.5(Red) Sex ratio (7)	PM2.5>55.5(Red) Sex ratio (8)
T1	.00089	.00071	-.00051	-.00105	-.00029	-.00141	-.00608	-.0074
	(.00089)	(.00092)	(.0021)	(.00211)	(.00642)	(.00645)	(.01155)	(.01156)
T2	-.00257***	-.00272***	-.00384**	-.00411**	-.00958**	-.01008**	-.01472*	-.01512*
	(.00085)	(.00084)	(.00162)	(.00165)	(.00397)	(.00403)	(.00748)	(.00754)
T3	.00066	.00065	.00156	.00161	.00465	.00499	.0035	.00427
	(.00089)	(.00086)	(.00161)	(.0016)	(.00464)	(.00441)	(.00746)	(.0072)
ethnic others	-.01811	-.01076	-.02628	-.018	-.02417	-.01606	-.0255	-.01721
	(.0212)	(.02762)	(.02297)	(.0303)	(.02278)	(.03005)	(.02295)	(.03016)
Interactions								
Reference: ethnic Hispanic								
T1 x ethnic Others	-.00105	-.00072	.00133	.00224	.00241	.00453	.01515	.01783
	(.00162)	(.00165)	(.00442)	(.00443)	(.01171)	(.01171)	(.01944)	(.01945)
T2 x ethnic Others	.00429***	.00456***	.00753**	.00781**	.01619**	.01674**	.02338*	.02353*
	(.00129)	(.0013)	(.00307)	(.00313)	(.00726)	(.00745)	(.01321)	(.01344)
T3 x ethnic Others	-.00154	-.00156	-.00162	-.00194	-.00466	-.00559	.00037	-.00162
	(.00134)	(.00132)	(.00327)	(.00331)	(.00734)	(.00706)	(.01216)	(.01176)
Observations	2532	2532	2464	2464	2464	2464	2464	2464
All Controls		✓		✓		✓		✓
City Fixed effects	✓	✓	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. The column headings represent the dependent variables. The controls include the marital status of the mother, the child's sex, maternal ethnicity, maternal education, maternal age, birth order and number of gestational weeks. The fixed effects include the month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table 2.9: The interaction between wildfire exposure in days and the mother's education level while pregnant

	Birth weight All (1)	Birth weight Boys (2)	Birth weight Girls (3)	Birth weight All (4)	Birth weight Boys (5)	Birth weight Girls (6)
Utero fire	-.1849	-.21926	-.19002	-.26918	-.322**	-.21619
	(.15722)	(.15675)	(.27931)	(.17063)	(.15761)	(.27296)
1bn.edu>12 yrs						
2.edu<12 yrs	-12.68509**	-17.07537***	-7.74089	-20.19923***	-24.64007***	-15.60636***
	(5.35709)	(5.44757)	(5.33214)	(2.21725)	(2.20864)	(2.49607)
Interactions						
1bn.edu>12 yrs x utero fire						
2.edu<12 yrs x utero fire	.05944	.06872	.09834	.06394	.13008	.00022
	(.28044)	(.27952)	(.36977)	(.21416)	(.2165)	(.29707)
Observations	952165	484798	467367	952165	484798	467367
All Controls				✓	✓	✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. The column headings represent the dependent variables. The controls include the marital status of the mother, the child's sex, maternal ethnicity, maternal education, maternal age, birth order and number of gestational weeks. The fixed effects include the month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table 2.10: The interaction between wildfire exposure in days and the mother's education level by trimester

	Birth weight All (1)	Birth weight Boys (2)	Birth weight Girls (3)	Birth weight All (4)	Birth weight Boys (5)	Birth weight Girls (6)
T1 fire	-.24301	-.33558	-.16423	-.35274	-.46399*	-.22937
	(.23812)	(.23311)	(.36103)	(.25538)	(.24537)	(.35109)
T2 fire	-.28774	-.35638	-.28086*	-.36514**	-.43469*	-.30112
	(.17831)	(.30418)	(.15905)	(.1789)	(.24594)	(.19217)
T3 fire	-.03836	.01204	-.12942	-.10713	-.09592	-.12256
	(.33271)	(.26647)	(.47533)	(.26087)	(.26212)	(.40812)
1bn.edu>12 yrs						
2.edu<12 yrs	-12.68351**	-17.09878***	-7.71085	-20.20965***	-24.67822***	-15.58841***
	(5.36296)	(5.45713)	(5.33457)	(2.21899)	(2.21029)	(2.49737)
Interactions:						
1bn.edu>12 yrs x T1 fire						
2.edu<12 yrs x T1 fire	-.07874	.31604	-.47158	.10483	.56218*	-.37409
	(.32253)	(.36669)	(.42256)	(.26094)	(.28435)	(.3543)
1bn.edu>12 yrs x T2 fire						
2.edu<12 yrs x T2fire	-.04355	-.31977	.30329	-.16961	-.47272	.14775
	(.21744)	(.35967)	(.33176)	(.19241)	(.30263)	(.27864)
1bn.edu>12 yrs x T3 fire						
2.edu<12 yrs x T3 fire	.28303	.21896	.41126	.25218	.32412	.18998
	(.46079)	(.46393)	(.47553)	(.36696)	(.39686)	(.40946)
Observations	952165	484798	467367	952165	484798	467367
All Controls				✓	✓	✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. The column headings represent the dependent variables. The controls include the marital status of the mother, the child's sex, maternal ethnicity, maternal education, maternal age, birth order and number of gestational weeks. The fixed effects include the month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table 2.11: The interaction estimations between wildfire exposure in days and the mother's ethnicity

	Birth weight All (1)	Birth weight Boys (2)	Birth weight Girls (3)	Birth weight All (4)	Birth weight Boys (5)	Birth weight Girls (6)
Utero fire	-.22947	-.1494	-.30732	-.32172	-.26243	-.38135
	(.23852)	(.25536)	(.3199)	(.2338)	(.20082)	(.31842)
1bn.ethinic hisapnic						
ethinic others	-39.23097***	-34.82216***	-44.64522***	-49.95078***	-47.57837***	-52.51977***
	(6.48924)	(6.42079)	(6.82079)	(7.1463)	(6.92093)	(7.64127)
Interactions						
1bn. hisapnic x utero fire						
2. others x utero fire	.17632	-.03969	.36317	.17121	.01222	.33545
	(.33678)	(.36493)	(.34305)	(.27289)	(.28578)	(.29827)
Observations	952165	484798	467367	952165	484798	467367
All Controls				✓	✓	✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. The column headings represent the dependent variables. The controls include the marital status of the mother, the child's sex, maternal ethnicity, maternal education, maternal age, birth order and number of gestational weeks. The fixed effects include the month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table 2.12: The interaction estimations between wildfire exposure in days and the mother's ethnicity by trimester

	Birth weight All (1)	Birth weight Boys (2)	Birth weight Girls (3)	Birth weight All (4)	Birth weight Boys (5)	Birth weight Girls (6)
T1	-.47664	-.17389	-.80362*	-.46203	-.17299	-.76177*
	(.44472)	(.51434)	(.4499)	(.41665)	(.44796)	(.45312)
T2	-.38692*	-.4963**	-.25044	-.48511**	-.674***	-.29463
	(.19313)	(.1925)	(.30731)	(.23799)	(.19936)	(.33031)
T3	.14538	.19117	.09134	-.0451	.02776	-.12095
	(.19973)	(.34599)	(.39201)	(.16645)	(.24494)	(.35069)
1bn.ethinic hispanic						
ethinic others	-39.23667***	-34.82999***	-44.66092***	-49.96697***	-47.59542***	-52.54042***
	(6.49509)	(6.42554)	(6.8244)	(7.15238)	(6.93283)	(7.6419)
Interactions:						
1bn. hispanic x T1						
2. others x T1	.42862	.02889	.8498**	.32389	-.02687	.69278**
	(.46282)	(.5757)	(.40185)	(.36346)	(.45965)	(.32675)
1bn. hispanic x T2						
2. others x T2	.18209	-.00078	.26462	.06423	-.00981	.14442
	(.25954)	(.28812)	(.33753)	(.26877)	(.2673)	(.36333)
1bn. hispanic x T3						
2. others x T3	-.05969	-.12577	.01476	.14532	.0963	.20065
	(.39382)	(.49835)	(.42718)	(.30713)	(.39245)	(.38615)
Observations	952165	484798	467367	952165	484798	467367
All Controls				✓	✓	✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. The column headings represent the dependent variables. The controls include the marital status of the mother, the child's sex, maternal ethnicity, maternal education, maternal age, birth order and number of gestational weeks. The fixed effects include the month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Chapter 3

Vaccination Hesitancy in Africa: the Unintended Consequence of a Culture Clash? *(with Prof. Giacomo De Luca)*

3.1 Introduction

Vaccines save lives. Even though the vaccination rate remains relatively low in certain groups of the population and countries as reported by WHO (2021*a*), the progressive implementation of structured immunisation campaigns has been a crucial mechanism behind the global health improvement of humankind over the last decades (WHO, 2019).

Logistical issues in the implementation of the immunisation campaigns, however, are not the only reasons preventing full vaccination rates across the globe. Doubts over the effectiveness, reliability and genuine motives behind these policies, referred to as vaccination hesitancy, regularly surface in consistent strata of the population. The current COVID pandemic highlighted the saliency of this debate, with the No Vax movements around the globe revindicating the individual right to refuse the proposed immunisation, despite the potential costs the community as a whole would have to bear. Both traditional and social media featured the topic systematically, with the vast majority of the scientific community urging people to use the vaccination campaigns as a tool to shorten and loosen the alternative draconian measures needed to fence the circulation of the virus (e.g. lockdowns, curfews, homeschooling).

While the pandemic scaled up the saliency of vaccine hesitancy at the global level, the issue has been regularly affecting several countries with different degrees in the past. Where does this behaviour come from? Given the complexity of the issue, there is no unique determinant behind it. In the case of resistance to measles immunisation (which lead to the reappearance of the virus in the context of the UK and the US (Gardner et al., 2020, Thompson et al., 2023) it was the consequence of a scientific study which found a correlation between the vaccination and lack of knowledge about vaccine among parents (Krishnamoorthy et al., 2019). More often, it is the uncertainty regarding side effects and the long-term consequences of vaccination (e.g., often mentioned in the case of COVID-19 vaccines). A third potential factor is the overall lack of trust towards science, and medical science

more specifically, usually - although not exclusively - combined with low education. The existing distrust can be further nourished by religious or traditional leaders drawing their arguments either on non-scientific grounds or outright conspiracy theories. For instance, in Nigeria, rumours spread that the anti-polio vaccination effort was part of an effort by Westerners to spread HIV among Muslims and cause infertility in Muslim girls. Similar arguments were circulating in Pakistan, supported by the local religious leadership. The general idea is that immunisation campaigns are a disguised plot of Western countries (or the white man) to hit the local population.

Unfortunately, while these theories have in general no evidence, badly designed trials and or reckless usage of immunisation campaigns for political goals can provide the perfect narrative to increase their credibility and their disastrous effects.

This paper studies child first-year immunisation across Africa, the region of the world clearly lagging behind in terms of overall vaccination (UNICEF and WHO, 2020). We first document a substantially different behaviour in immunisation among Muslim households, whereby children of Muslim families are less likely to complete their first-year vaccination schedule as compared with children with other religious backgrounds. Although this divergence appears first in the early 2000s, it emerges more strongly in the 2010s. We find no similar trends among children of different religious backgrounds. Also, a series of heterogeneity tests reveals that vaccination rates are lower among Muslims, even when we look at families with educated mothers (and fathers), and that the effect is more marked in areas featuring low trust in institutions, thereby suggesting vaccination hesitancy as the underlying reason behind this anomaly.

In the second part of the paper, we therefore focus on vaccination hesitancy and propose a potential mechanism for why it increased among Muslims during the period considered. We argue that the relatively more hostile environment against Muslims unfolding in Western countries after the 9/11 terroristic attack in the US and the following "global war on terror" has generated a climate of cultural clash, to which Muslims may have reacted by increasing their refusal of policies perceived as Western-led, which include immunisation campaigns. More specifically, our difference-in-differences estimates show that the lower vaccination among African Muslims tracks the timing of hate crimes against Muslims as recorded in the US. The effect is stronger in areas better connected with global information channels, where the debate around the cultural clash circulated mostly. Crucially, the effect is stronger in anglophone countries, where we expect the intensity of Islamophobia in the US to have a wider dissemination in the local population. Finally, the effect is stronger where Save the Children, a global leading Western NGO active in children health across developing countries, is

directly involved in the provision of local health - once more suggesting that the increase in vaccination hesitancy may be directly related to the distrust towards Western led policies.

The rest of the paper is organised as follows. We first discuss the related literature. We then describe the data used in our analysis in section 3.3, and present the simple empirical strategy adopted to document the differential patterns of immunisation among Muslim children in Africa in section 3.4. Section 3.5 first discusses our main results, and then proposes and tests the cultural clash mechanism.

3.2 Literature review

This paper contributes to the literature on the determinants of vaccination hesitancy. The scepticism and resistance towards vaccines can be traced back to the 18th century when Dr. Edward Jenner invented the first vaccine for preventing smallpox in 1796 (Callender, 2016). Before the development of the smallpox vaccine, variolation was first induced as an effective way to immunize an individual against smallpox simply by inserting the smallpox lesions from infected individuals into non-infected individuals, but unqualified practitioners had made variolation an unsafe practice (Riedel, 2005). The invitation of the smallpox vaccine provides a revolutionary way of preventing smallpox infection. However, the vaccine has encountered opposition from different groups of people, particularly those with religious beliefs, due to its feature of containing animal matter (Durbach, 2000). In addition, mandatory vaccination laws induced in the late 19th century in Britain and the USA to mandate all infants within the first three months of birth be vaccinated against smallpox, and all the local residents in places where smallpox hit led to a widespread vaccination hesitancy and a rise in anti-vaccination movements (Callender, 2016, Durbach, 2000, Prabhu, 2021).

A recent systematic review identified research on understanding vaccine hesitancy documented that resistance to vaccination in the modern era is complex and context-specific, it varies by determinants of hesitancy (Larson et al., 2014). A strand of studies have explored the factors associated with hesitancy in different settings and show that socioeconomic factors such as parental education, income level, media environment and religious beliefs; cognitive factors, such as lack of knowledge about vaccine and diseases, fear of side effects or risk regarding the vaccine, trust in institutions, were found to be statistically significant determinants of vaccine hesitancy (Dubé et al., 2013, Falagas and Zarkadoulia, 2008, Larson et al., 2014, Stoop et al., 2021)

Guay et al. (2019) examine the key drivers of vaccine hesitancy based on extensive cross-

sectional survey data conducted between June and October 2014 in the Eastern Townships of Quebec. Applying a multivariate analysis, it finds that respondents with a household income less than 3000$ were statistically significantly associated with higher vaccine hesitancy. Those individuals were also found to be more likely linked with lower educational levels. Rahman and Obaida-Nasrin (2010) use 2004 Bangladesh Demographic and Health Survey data to evaluate the child immunisation coverage in the most demanding vaccines, including Bacille Calmette-Guérin(BCG), three doses of diphtheria, tetanus, and pertussis vaccine (DPT3), three doses of polio vaccine (Polio3) and Measles vaccine. It explores the factors that influence the full immunization status of children by utilizing the multivariate logistic model and finds that children born to mothers with higher education are significantly associated with a higher full immunization rate. Their study also shows a positive correlation between wealth index and vaccination rate, the full immunization rate among children born to the wealthiest family was 1.5 times that of children born to the low-income family.

Babalola and Lawan (2009) use detailed survey data conducted during July and August 2007 in northern Nigeria and document that community characteristics and parental and household conditions can explain the immunization rate. It exploits the contribution of different types of predictors of child immunization regarding BCG among infants by applying logistic regression analysis. They find that children whose mothers have good knowledge about vaccine-preventable diseases are around two times more likely to be vaccinated than mothers with little or no related knowledge about vaccines and diseases. The study also indicates that mothers exposed to child health-related information via the media or community sources tended to be associated with a higher BCG immunization rate.

Using detailed unit record survey data, Lim et al. (2008) estimate the child immunisation coverage in three doses of diphtheria, tetanus, and pertussis vaccine (DTP3) between 1986-2006 among 193 countries receiving support from both the Global Alliance on Vaccines and Immunisations (GAVI) and GAVI's immunisation services support (ISS). Their study finds that the actual immunisation coverage of DPT3 is significantly lower than the estimates reported by governments or the WHO and UNICEF.

Dupas (2011) reviews studies of health-seeking behaviour in developing countries and indicates that household demand for preventive health technologies and products is lower than optimal. Two stylised facts are considered as the main reasons for the low acceptance of preventable care. The first one is the financial constraint which has been proven as one of the main factors preventing households from getting health products (Ashraf et al., 2010, Cohen and Dupas, 2010, Dupas, 2011). Helen

L. Guyatt (2002) explore this path by assessing the willingness to pay (WTP) for insecticide-treated bednets (ITNs) in highland Kenya and find that an increase in the amount for ITNs significantly reduces the rate of WTP. Ashraf et al. (2010) conducted a field experiment in Zambia using door-to-door marketing to promote Clorin-a home-use water purification solution, to investigate the effects of prices on the demand for Clorin. Their study finds negative and significant impacts of prices on using Clorin. That is, a 100 Kwacha (US $0.03 in 2010) increase in the offer price of Clorin significantly reduced the purchase of Clorin by 7%. Second, the delivery of free immunisation services is also a crucial determinant of the demand for vaccination Dupas (2011). Banerjee et al. (2010) investigate the impacts of reliable immunisation services on the immunisation rates in children under the age of three. They find that full immunisation rates increased from 6% to 18% in villages with reliable vaccination camps presented, and the immunisation rates increased even more (up to 39%) with modest non-financial incentives in place. One crucial implication from this study is that providing reliable free preventive care is the key to improving child immunisation rates.

Vaccines are one of the most affected health products from scepticism in most settings (Martinez-Bravo and Stegmann, 2021). Mistrust in the local authorities or state institutions can be a crucial driving factor that affects citizens' behaviour in using public services provided by the government (León-Ciliotta et al., 2022). Acemoglu et al. (2020) exploit the effect of credible information about the delivery of improved national services on the trust in state actors of the citizens using the household survey data collected from rural areas in Punjab, Pakistan, where the mistrust in local authorities is high. Their study indicates that a lack of trust in the state institutions leads to low interaction between the citizens and state institutions, while credible and positive information about state institutions can increase the trust in state institutions of the local citizens, which in turn increases the utilisation of state institutions. van der Weerd et al. (2011) investigate the impacts of government trust in vaccination hesitancy using cross-sectional survey data conducted in the Netherlands in 2009 when the influenza A (H1N1) pandemic occurred. Change in the level of government trust is significantly and positively associated with vaccination hesitancy, a higher level of trust in the government increases the acceptance rate of vaccination. Rumours and conspiracy theories leading to mistrust are key factors resulting in the decrease in the uptake of vaccines (Freeman et al., 2022). Islam et al. (2021) explore the relationship between rumours and conspiracy theories in COVID-19 vaccine and vaccine hesitancy by conducting a content analysis using the data on rumours and conspiracy circulated online and concluding that vaccine hesitancy is correlated with the rumours and conspiracy theories.

An unreliable supply of free immunisation services may result in mistrust in the public health sector, leading to under-vaccination. Larson et al. (2018) review sizable literature on trust in vaccination among low and middle-income countries and report that low trust in vaccination programmes is considered one of the most driving reasons for vaccine hesitancy. Stoop et al. (2021) use cross-sectional data, which captures the information on child vaccination status, for 22 African countries covering 216 sub-national regions from 2004 to 2018 and document that institutional mistrust can explain the low child vaccination rate rates in low-income and middle-income countries. It explores the effects of variation in institutional trust within sub-national regions on child vaccination rates using fixed effects regression analysis. It finds that increasing the institutional mistrust index by one SD decreases the child vaccination rate for getting any of the eight vaccines by 10% and all the basic vaccines by 6%. Das and Das (2003) exploit the factors that affect the demand for vaccination using the data for children born in the Garhwal region of India. It shows that vaccination rates are positively associated with midwife visits in the region. However, the vaccination rates decreased immediately due to increased mistrust in midwives after the incidence of two mother deaths occurred during birth delivery with midwife assistance. The authors argue that lack of information on vaccination results in low vaccination rates of children among poor and low-educated mothers. Their study confirms that information constraints significantly affect the household's decision to vaccinate their children.

In addition to the reasons mentioned above, disclosing negative information resulting in long-run mistrust in health institutions also plays a crucial role in determining the household's health-seeking behaviour (Martinez-Bravo and Stegmann, 2021). In an influential work, Alsan and Wanamaker (2017) exploit the effect of the disclosure of the 1972 Tuskegee syphilis study, in which black men with syphilis were denied adequate treatment for the purpose of exploring the impact of the disease, on demand for the health of the black men. It finds adverse and significant effects of the disclosure of the Tuskegee study on the utilisation of medical services by the black men who live geographically near the study's subjects. The effects are particularly strong for black men with lower education and income. Lowes and Montero (2021) investigate the impact of 1920s-1950s French colonial campaigns on mistrust in medicine in Central Africa. They use the suitability of cassava relative to millet as an instrument for sleeping sickness prevalence. Applying an instrumental variable analysis, it finds that being exposed to the colonial campaign is associated with a 12.6 % reduction in the share of child vaccination rates. Furthermore, disinformation about the vaccination spread through social media has been identified as a crucial determinant factor in vaccine hesitancy (Wilson and Wiysonge, 2020).

Hansen and Schmidtblaicher (2019) assess the effect of social media coverage on the vaccination rate by applying a dynamic model of vaccine compliance using Danish human papillomavirus (HPV) data. Their study indicates that fake news on serious side effects of the HPV vaccination spread by the Danish media significantly reduces the HPV vaccine uptake. The effects were particularly strong after the release of a vaccine-critical TV documentary.

Vaccination refusal may also stem from religious doctrines (Hardon, 2004, Streefland, 2001). Among some Christian confessions dominates the view that divine providence should not be interfered with by human beings, and vaccination is considered exactly as a way of interfering with it (Ruijs et al., 2013). The position of Islamist groups vis-a-vis immunisation policies has been generally ambiguous. Although official declarations (Fatwa) have been repeatedly released by Muslim religious leaders, allowing Muslims to participate in them, several recurring themes have been systematically used to spread distrust among believers towards vaccines (Mushtaq et al., 2015). The first argument presented against vaccines is that they are produced with pig fat (or other substances), which cannot be assumed by faithful Muslims as indicated as impure in the Qur'an (Maravia, 2020, Pelčić et al., 2016, Rochmyaningsih, 2018).

A second argument, again directly related to the Qur'an, is that vaccines should be avoided because it is un-Islamic to take medicine before the disease is contracted. In a discussion about the 11-month boycott of polio vaccination in Kano, Nigeria, in 2003–2004, the Islamic faith and the belief that polio was caused by spirits were the regular motivations behind immunisation refusal (Ghinai et al., 2013, James Fairhead, 2007).

Finally, perhaps the most relevant idea developed in this paper, a third recurring idea is that vaccines are a covered policy to sterilize Muslim girls. Using the words of an article by Dr. Katme (2011), a spokesman for the Islamic Medical Association UK: "according to many scientific reports, there is additional concern that some vaccines cause infertility and are used for clandestine population control".[1] Hence, the overall idea is that Westerners are using immunization as a plot to depopulate countries of the Muslim world.

The origin of the distrust against Western-led policies may be deeply rooted in the colonial past, as attested by the early attempts by colonial governments to vaccinate their subjects frequently met with resistance (Arnold, 1993, Feldman-Savelsberg et al., 2000, Hardon, 2004). This specific theory in all its different variants, however, most likely stems from a historical misunderstanding around early immunisation campaigns in Nigeria. In the 1980s, under President Babangida's administration,

[1] Accessed in June 2023: www.hizbululama.org.uk/highlights/1432ah/shawwal/Islam_Vaccines_and_Health.html.

the population policy was limited to four children per woman. As the polio vaccination campaign incidentally followed this policy, it resulted in suspicions of depopulation. There were general suspicions as to why children were given free vaccination instead of basic medicines and treatment perceived as more urgently required. The 1994 Pfizer trial in the same country reinforced the general distrust in the population, which led to an 11-month halt of polio immunisation in four major Muslim states in the North of the country between 2003 and 2004 (Sulaiman, 2014). Vaccine refusal has been attributed to both political tensions within the country, but also to distrust of "American intervention" (Ghinai et al., 2013, James Fairhead, 2007, Jegede, 2007, Larson et al., 2011, Yahya, 2007). Local authorities presented the vaccination campaign as a Western Christian plot to try and reduce the Muslim population in Nigeria. For instance, on February 26, 2004, Ibrahim Shekarau, Governor of Kano State, one of the three Nigerian northerner states refused the polio vaccine in his State, saying that "it is lesser of two evils, to sacrifice two, three, four, five, even ten children (to polio) than allow hundreds of thousands or possibly millions of girlchildren likely to be rendered infertile (Olufowote, 2011, Sulaiman, 2014).

Similar ideas circulated in Malaysia, where social media were recently featuring news claiming that the vaccine is a plot to weaken Muslims and transmit diseases to non-Western communities. The news gained substantial visibility after the endorsement of a popular singer for an anti-immunization stance, posting "Allah is all powerful, vaccines have no power" on social media (Ahmed et al., 2018).

Whatever the true origin of these theories, these ideas gained momentum, particularly in politically contested countries (e.g. Afghanistan, Pakistan, Somalia), where Western intervention is filling grievances in the local population, and spread through the Muslim communities around the world (Chaturvedi et al., 2009, Hussain et al., 2016, Maravia, 2020). Interestingly, this belief goes beyond developing countries, if even the American organisation Nation of Islam fully endorses and spreads through its online platforms the rethoric that vaccinations are an attempt by the white man to depopulate the Black (Muslim) population (Farrakhan, 2010).

Against this background, any scandal or input reinforcing distrust or lending credibility to these conspiracy theories further boosts vaccination hesitancy. A recent blatant example is the CIA vaccination ruse taking place in 2011 (Andrade and Hussain, 2018, Martinez-Bravo and Stegmann, 2021). In an attempt to obtain a further confirmation of the location of Osama Bin Laden, in 2010, the CIA organised a fake immunization campaign in the northern Pakistan city of Abbottabad. With the complicity of a local doctor and a team of unaware health workers, the administration of hepatitis B vaccines to children started in March 2011. The immunisation campaign, allegedly entirely unknown

to local health authorities, was meant to cover the actual goal of the CIA, namely to gather the DNA of treated children to be compared to the one of the Bin Laden's family. The overall plan was to be able to establish with reasonable certainty that Osama Bin Laden was indeed living in the compound already under CIA observation before the implementation of a military strike to eliminate him was to be carried out. Only months after the attack, in July 2011, the British newspaper The Guardian revealed the use of the fake immunisation campaign to kill Bin Laden, leading to a compact outcry among health practitioners and beyond. The White House announced that immunisation campaigns would not be used by the CIA in the future as a cover to gather intelligence or genetic material. But the reputational damage was done.

Taliban leaders, already critical against the immunisation campaigns implemented in the country, used the disclosure of the ruse to substantiate their claims. Quoting directly the words of the Taliban leader Maulana Fazlullah (as reported by Martinez Bravo), the immunisation is nothing but a "conspiracy of Jews and Christians to make Muslims impotent and stunt the growth of Muslims" (Roul, 2014, page 18). Martinez-Bravo and Stegmann (2021) find that the vaccination rates were significantly lower in places where the support for Islamist parties was high after the disclosure of information in the 2011 fake vaccination campaign.

Following the disclosure of the CIA ruse, the rumour of immunisation as a Western plot to depopulate the Muslim population around the world has been noted in various countries. For instance, a recent survey on vaccination hesitancy in Indonesia reported the belief of a conspiracy between the government of Indonesia and particular Western countries intended to reduce the global Muslim population (Syiroj et al., 2019). According to a father interviewed: "Basic immunisation is Jew's [western countries'] program. They want to reduce population growth among Muslims, so the Muslim population will decrease".

A second example is the 2000 Pfizer drug trials that resulted in 11 deaths of children in Nigeria, whose effects are studied by Archibong and Annan (2021). Using the DHS data in the period 1990-2013 for children born between 1985-2013 in Nigeria, they document that disclosure of medical misconduct is negatively associated with vaccination rates. More specifically, it shows that the disclosure of Pfizer drug trials is associated with 9.1%, 8.9% and 5.1% in BCG, DPT and polio vaccination rates, respectively, for children born to Muslim mothers after 2000.

Our paper contributes to this late literature suggesting that the level of vaccination hesitancy characterising immunisation decisions among Muslims in Africa may be related to the intensity of the cultural clash between Islam and Western countries developing over the last two decades.

3.3 Data

To examine immunisation behaviour, we use repeated cross-sectional data from the Kid's recode (KR) on child vaccination status collected from the Demographic Health Survey program (Demographic and Health Surveys (DHS), 2020) covering the period survey years 1992-2021 for individuals born in 35 African countries. In the main analysis, to be able to include a set of relevant controls in the analysis we focus on children born after 1998. We also end our analysis in December 2019, to avoid the confounding spillover effects of Covid-19 on first-year vaccinations. To construct the baseline sample dataset, we first combine all the available kids' datasets between 1999 and 2019 from 35 African countries to obtain a sizeable individual-level dataset. Following this, we georeferenced all individuals in the dataset by merging the appended kid's dataset with the GPS dataset collected from DHS using the unique identifiers (DHSYEAR, DHSCLUST, DHSCC) for the GPS data files.

The child vaccination status was obtained from the DHS Kid's Survey (KR), which provides detailed information on postnatal care, immunisation, and health status for each child born to the interviewed women. In addition, detailed information on mothers is also included in the survey. The information on mothers' religion was also from DHS Kid's survey (KR), which also captures detailed information on respondents' religion. Based on the information about the mother's religion provided in the dataset, we defined a dummy variable that tells us whether or not the mother is Muslim. Next, we derived the share of the Muslim population by aggregating the dataset at the DHS cluster level, which identifies the intensity of Muslims in each cluster where the children were born. In total, we obtained a baseline sample, which consists of detailed information on the child and mother, of 949,313 children born between Jan 1999 and Dec 2019.

The primary outcomes of interest correspond to the three basic routine immunisation of children stated by the WHO standards: Polio, Pentavalent and BCG (tuberculosis), which are recommended by the WHO to be scheduled within the first 14 weeks of birth (WHO, 2021*b*). Our primary outcomes are dummy variables that equal to one if the child has received the following vaccines according to the vaccination card: 1st dose vaccination of the polio vaccine; 1st dose vaccination of the Pentavalent vaccine; One dose of the BCG (tuberculosis) vaccine; And a total of seven doses of all these three basic vaccines. In addition to the dummy measure of child vaccination status, we are also interested in the continuous outcomes of interest. To obtain the continuous measurement of child vaccination status, we defined each child's vaccination status as a fraction of receiving the full doses of the three basic vaccines (3 doses in Polio, three doses in Pentavalent and 1 dose in BCG).

Since, as indicated by Sheikh et al. (2011), a significant measurement error can occur when relying on recalling the vaccination status, following the standard approach in the literature, we focused on the vaccination status verified or reported on the card.

We code as Muslim children of Muslim mothers. Additionally, as a continuous treatment measure of Muslims, we aggregate the dummy variable for mothers being Muslim to obtain the share of Muslims in each cluster.

From the DHS data, we also source the following variables: child age in months, a dummy for a female child, and birth order, a set of parents' characteristics including the mother's age at the interview, and her age at her first birth, mother's and father's education, and the household wealth index.

Our data on institutional trust comes from the 2nd to 7th round of the Afrobarometer survey (Afrobarometer, 2020) conducted between 2002 and 2018 among 37 African countries. The Afrobarometer survey is publicly available data that provides information on the level of trust at the regional level (first administrative level) that people have for public institutions of their country, such as trust in parliament, local government, prime minister, courts of law council and electoral commission. We redefined each type of institutional trust variable as a dummy equal to one if people reported low trust and zero otherwise. Following this, we calculated the share of people who reported low trust in each type of trust by region and by year, and then we aggregated all types of trust at the region year level to derive the average share of people who reported low trust in all types of trust.

The data on hate crimes in the US is taken from the FBI's Uniform Crime Reporting (UCR) Program (The Uniform Crime Reporting (UCR) Program, 1990-2019) and spans from 1991 to 2020. The data on mobile broadband subscription and cellular tower density are sourced from the International Telecommunication Union (ITU) (The International Telecommunication Union (ITU), 2022) and The OpenCellID project (The OpenCellID, 2009-2023), respectively. Data on Save the Children operations are taken from the official website of Save the Children (Save the Children (StC), 2023).

The main variables used in the analysis are provided in the Table 3.1 below.

3.4 Empirical strategy

To study vaccination behaviour in Africa we estimate a simple model which explores the change in immunisation rate across children of Muslim families over time. Formally, we estimate the following

equation:

$$Vax_{itmkz} = \alpha_i + \delta_t + \theta_k + \omega_z + \sum_{year=2000}^{2019} \gamma_{year} year \times Muslim_m + X'_{itm}\beta + \epsilon_{itmkz} \tag{3.1}$$

where Vax_{itmkz} is the specific vaccination outcome for child i born in month-year t to mother m, interviewed at time k, while the child was z years old; $Muslim_m$ is a dummy variable that is equal to one if the child was born to a Muslim mother and zero otherwise; α_i are DHS cluster fixed effects; δ_t are month-year of birth fixed effects; θ_k are month-year of interview fixed effects; and ω_z age of child at interview fixed effects (expressed in years), and ϵ_{itmkz} is the error term which we allow to be heteroscedastic and correlated across municipalities; in practice, the standard errors we report are clustered at ADM1 region level. While the various time-fixed effects are meant to capture specific shocks which may be common to a specific cohort or DHS survey wave, the DHS cluster fixed effects absorbe the time-invariant unobserved heterogeneity at the local level, including geographic factors which may influence the vaccination rate. The coefficients of interest are the γs which capture the differential vaccination rates among children of Muslim mothers as compared to other same aged children year by year.

Alternative models are also estimated for children of different religious backgrounds focusing on Catholics, members of traditional religions, or individuals identifying themselves as following no religion.

In addition, we run one specification of the model above in which we include a set of child-level controls, namely child age in months, a dummy for female child, and birth order, a set of parents' characteristics including mother's age at interview, and her age at her first birth, mother's and father's education, and the household wealth index as collected by the DHS, all captured by the term X_{itm} in equation 3.1.

3.5 Results

3.5.1 Vaccination trends across religions

We report the results of estimating model 3.1 in a series of graphs (all regression results are reported in the appendix as additional tables). Figure 3.1 reports the coefficients of all interaction terms between years and the dummy for Muslim children, for five different outcomes related to vaccination

as dependent variables in our most complete specification of the model (mother's age at interview, and her age at her first birth, mother's and father's education, and the household wealth index as collected by the DHS). More specifically, in panel (a) we consider the fraction of first-year vaccines received; in panel (b) a dummy for children receiving all doses prescribed for their first year. The last three panels report the results for the decision of receiving the first dose of the three main vaccination routines aggregated in our main vaccination measures, namely Polio (c), Pentavalent (d), and BCG (e).

For all dependent variables considered, there is a negative and statistically significant coefficient for 2002, suggesting that in 2002 children of Muslim mothers experienced a lower vaccination rate than same aged children with different religious backgrounds. In terms of magnitude, panel (a) suggests that the fraction of vaccination doses received in their first year by Muslim children was, on average, 2% lower than other children with the same age. Starting from 2012, coefficients are again negative and significant, with a larger magnitude of the effect towards the end of the period considered. Alternative specifications of the model produce similar estimates, and crucially, none of the controls included in the specification adopted is determinant for the pattern displayed in Figure 3.1.

We next replicate the same analysis for three alternative large groups along the religious dimension: Catholics, traditional religions, and no religion. Results are reported in Figure 3.2, 3.3, and 3.4, all adopting an identical structure as Figure 3.1.[2]

Overall, the pattern found for Muslims does not emerge for any other large religious group. In fact, even though some coefficients are statistically significant for some of the years and religions considered, no clear pattern nor trend seems to characterise vaccination rates over time for children of these groups.

Hence, the results for Muslims seem to be unique and deserve closer scrutiny. In an attempt to narrow down the potential explanations, we run a series of heterogeneity tests, aiming at identifying mediating factors which may have co-determined the relative decrease in the vaccination rate observed. Following the related literature, we investigate in particular two potential mediating factors: the mother's (and father's) education and the level of trust in the local institutions. Formally, we are estimating three augmented specifications of model 3.1, in which, along with the full set of controls, we also add the interaction terms between our $year \times Muslim$ term with a dummy for

[2]We remove Muslims from the sample before running the regressions underlying Figure 3.2, 3.3, and 3.4 to avoid positive effects due to having Muslim in the control group. The corresponding figures with the full sample, however, are qualitatively very similar.

Educated mother, a dummy for *Educated father* and a dummy for *Low trust* (and their respective non interacted terms), respectively.

In Figure 3.5, we report the results of the test involving the mother's education on the fraction of first-year vaccines received. To facilitate the interpretation, we report in panel (a) the same coefficients of panel (a) of Figure 3.1. Panel (b) reports the coefficient of the $year \times Muslim$ terms obtained from estimating the augmented model, to be interpreted as the year specific difference in vaccination among children of low educated mothers. Panel (c) reports the coefficients of the triple interaction term $year \times Muslim \times$ *Educated mother*, which estimates the average additional effect for children of educated mothers (defined as mothers with at least primary school degree). Surprisingly, the usually beneficial effect of educated mothers on decisions regarding children's welfare (Cui et al., 2019) does not seem to operate among Muslim women, at least for what concerns vaccinations. The corresponding results adopting the father's education (*Educated father* is a dummy equal to one for fathers with at least primary school degree) as mediating factor are reported in Figure 3.6. As for mothers' education, we find no consistent significant beneficial effect. Figures 3.7 and 3.8 replicate the above exercise for the dummy for children receiving all doses prescribed for their first year as the dependent variable. The overall pattern is very similar.[3]

In Figure 3.9, adopting the same structure as Figure 3.5, we report the results of the test involving trust on the fraction of first-year vaccines received. Again panel (a) reports the same coefficients of panel (a) of Figure 3.1. Panel (b) reports the coefficient of the $year \times Muslim$ terms obtained from estimating the augmented model, to be interpreted as the year specific difference in vaccination among children in areas with a level of trust in local institutions above the median. Panel (c) reports the coefficients of the triple interaction term $year \times Muslim \times$ *Low trust*, which estimates the average additional effect for children of Muslim mothers in areas with relatively low trust (defined as a dummy for areas with average trust in local institutions below the median).

Even though the pattern is not consistent throughout the period considered, the change in vaccination is more pronounced in areas featuring lower trust.

Figure 3.10 replicates the heterogeneity test along the trust dimension for the dummy for children receiving all doses prescribed for their first year as the dependent variable. The overall pattern is very similar.[4]

Overall, the above results suggest that, indeed, at least part of the decrease in vaccination

[3]The corresponding figures for the first doses of Polio, Pentavalent and BCG are reported in the appendix.
[4]The corresponding figures for the first doses of Polio, Pentavalent and BCG are again reported in the appendix.

experienced by children of Muslim mothers is occurring in low-trust areas. This is consistent with vaccination hesitancy being a mechanism behind it. In the next section, we pursue this line of reasoning proposing a potential explanation and testing it formally with additional data.

3.5.2 Cultural clash?

Whereas grievances against Western countries have always been present in developing countries, due mainly to the legacy of the colonial experience, a series of notorious events taking place in the early 2000s increased significantly the saliency of the tension mounting among radical Islamic movements around the world against the US administration and the spread of the Western culture in general. The terroristic attack on the Twin Towers in New York City on 9/11, 2001, clearly marks a structural break in the perception of the problem among the Western population. The following invasions in Iraq and Afghanistan further exacerbated the perceived divide leading to more frequent violent demonstrations against the US in several Muslim-majority countries on one side and anti-Muslim hate crimes in Western countries. We conjecture that against this background, an environment of cultural clash developed, whereby the level of trust towards Western countries may have eroded among Muslims around the world. This widespread distrust would have lent support to already circulating conspiracy theories concerning policies which stem from the West, including immunisation campaigns - feared to be an instrument of rich Western elites to depopulate Muslim countries.

To test empirically our conjecture, we gather data on hate crimes against Muslims in the US and estimate whether the decrease in vaccination rates among Muslims in Africa closely tracks the intensity of the cultural clash which developed in the background. More formally, we estimate the following model:

$$Vax_{itmkz} = \alpha_i' + \delta_t' + \theta_k' + \omega_z' + \gamma' HC_{y-1} \times Muslim_m + X_{itm}'\beta' + \epsilon_{itmkz}' \tag{3.2}$$

where all variables are identical to equation 3.1 but HC, which is the number of hate crimes against Muslims in the previous year $(y-1)$ in the US. The parameter of interest is γ which captures the specific effect that hate crimes against Muslims in the US have on vaccination decisions among the Muslim population in Africa in the following year.

Table 3.2 reports the results of this exercise. Panel (a) features the results of estimating model 3.2 controlling for the large set of fixed effects, but not including the children and household level controls, which are then included in the models reported in panel (b). Each of the five columns

adopts a different dependent variable concerning vaccination decisions. Throughout the table, the coefficient of the interaction term of interest is negative and statistically significant, suggesting that, indeed, children of Muslim households experience a differential decrease in vaccination following a period of more intense friction with the Western culture. In terms of magnitude, panel (a) suggests that the fraction of vaccination doses received in their first year by Muslim children was, on average, 4% lower than other children of the same age following a one standard deviation increase in hate crimes in the US against Muslim in the previous year. Adding the rest of our controls does not alter the main finding (column (1) of Panel (b)), and the impact on the other vaccination variables are comparable in term of magnitude.

To discern our mechanism better, we develop our analysis in two ways. First, in Table 3.3, we report the results of a more demanding model, in which an additional Muslim-specific linear trend and a set of country-specific linear trends are included in the model.[5] Table 3.3 has an identical structure as Table 3.2. Whether we include the child and household controls (Panel (b)) or not (Panel (a)) the results stay largely unaffected with the magnitude remaining very similar. According to column (1) of panel (b) the fraction of vaccination doses received in their first year by Muslim children was, on average 3% lower than other children of the same age following a one standard deviation increase in hate crimes in the US against Muslim in the previous year.

Next, we explore the heterogeneity of our results along several dimensions, which allow us to narrow down the channel through which the impact is operating. If our conjecture is correct, the Muslim population would respond to a mounting hostile environment by changing their vaccination decisions. If this is correct, we should observe a more direct reaction in areas which are better connected with global information channels (e.g., the Internet and social media). We pursue this intuition and estimate an alternative model in which we include in equation 3.2 an additional interaction term between our $HC \times Muslim$ term with a measure of local mobile phone penetration MPP (and their respective non interacted terms), respectively. We measure local mobile phone penetration with two alternative variables: per capita mobile phone subscriptions (Table 3.4) and local cellular tower density (Table 3.5).

The coefficient of the triple interaction term is mostly negative and significant in Table 3.4 and Table 3.5, confirming that indeed in areas better connected with the global information channels, the reaction of Muslim households is more pronounced. Relatedly, one could argue that the specific trends in hate crimes against Muslims in the US may be mostly relevant for anglophone countries,

[5]Results are qualitatively identical if we include more granular ADM1 region-specific trends.

which are more directly linked to the events unravelling in English-speaking Western countries. We test directly whether our findings are consistent with this intuition. Once more, the heterogeneity test consists in augmenting model 3.2 with an interaction term between our $HC \times Muslim$ term and a dummy for anglophone African countries. The results of this test, reported in Table 3.6, largely confirm that it is exclusively anglophone countries which experience the decrease in vaccination rates in response to the intensity of hate crimes against Muslims in the US.

Finally, we propose an alternative strategy to pin down whether it is the distrust against Western policies which underlies our findings. We argue that distrust against Western policies would emerge more clearly where Western NGOs are directly involved in the provision of local health services, including immunisations. For this test, we identify African countries in which Save the Children (StC), a global leader in the provision of children's health across the developing world, is operating. They then explore whether countries in which StC is active are experiencing a more pronounced effect in reaction to hate crimes against Muslims. We report the results of this test in Table 3.7. The coefficient of the triple interaction term is negative, although statistically significant only for some of the vaccination related dependent variables, suggesting that, indeed, having Western organisations directly involved in the provision of local health services may have amplified the distrust towards immunisation campaigns among Muslims, thereby contributing to the overall decreasing pattern documented.

3.5.3 Further tests

In this section, we run three additional tests to bolster our interpretation of the previous results. First, we estimate an augmented version of model 3.2, which includes mother fixed effects. In this specification, we exploit the different intensity of hate crimes in the US experienced by mothers across their pregnancy of different children. The results, reported in the Appendix in Table C5, broadly confirm the previous findings, even though some coefficients for the interaction term $Muslim \times HC$ in the specification adding controls are not statistically significant according to the standard cutoff levels. [6]

Next, we explore the intuition according to which it is not the religious background of the mother shaping the vaccination decision, but perhaps the overall proportion of Muslims in the area in which the mother is located at the time in which such decision is taken. Accordingly, we adopt

[6]Notice that with mother fixed effect, all household controls are not included in the model as they do not vary across children of the same mother.

a slightly alternative approach: we first measure the fraction of the Muslim population at the DHS cluster level (*Muslim proportion*) and then use this measure as a measure of Muslim religion exposure for each child located in that cluster at birth to be interacted with the intensity of hate crimes against Muslims in the US. The results of this exercise are reported in the Appendix in Table C6. While the interpretation of the coefficient changes slightly, the overall message remains unaffected. A standard deviation increase in hate crimes against Muslims in the US in the previous year is associated with a decrease in the fraction of first-year vaccination doses by 5% in a cluster characterized by the average presence of Muslims in our sample.

A potential concern is that our analysis is simply picking up a late impact of the 1994 Pfizer controversial trial implemented in Nigeria, which has been studied recently by Archibong and Annan (2021). To address this concern we replicate the estimation of model 3.2 while dropping Nigeria from our sample. Dropping any other country from the sample does not affect the result either. The results, reported in the Appendix in Table C7, show that while Nigeria is clearly a crucial part of the story, given its region composition and its anglophone nature, our results do not depend on its inclusion.

3.6 Conclusions

Muslim children have experienced a substantial decrease in vaccination rates over the last 20 years. In this chapter, we explore child first-year immunisation using the DHS survey data for individuals born between 1999 and 2019 across African countries to find out whether the intensity of cultural clashes increased in recent decades between Islam and Western countries contributes to vaccination hesitancy among African Muslim households. Conspiracy theories regarding vaccination, such as the depopulation of Muslims, circulated online or by religious leaders can leave newborn Muslims unvaccinated due to increased levels of vaccine hesitancy. Mistrust in Western-led policies on vaccines which were considered as a cover to depopulate or sterilize Muslim girls, also contributes to the vaccination refusal among the Muslim population. However, the determinants of vaccine hesitancy among Muslim groups are diverse.

Our analysis documents that the overall trends of the child's first-year vaccination rates are lower among Muslim households in comparison to other religious groups across African countries. Such difference was particularly pronounced after 2010 when vaccination trends among Muslims in African countries started going down rapidly. A series of heterogeneity tests were conducted to reveal

whether our baseline results are heterogeneous by different groups, and it finds that the effect is larger on Muslim households among low-trust communities.

This chapter highlights an important way in which Islamophobia and vaccination hesitancy interact. We propose a potential explanation which relies on the recent culture clash related to the increase of terrorist attacks by Islamic groups and the following wave of Islamophobia in Western countries. The culture clash substantiated already circulating fears of vaccinations being used as policies serving the interests of Western countries against the Muslim population. Using the recorded anti-Muslim hate crimes data in the US between 1990 and 2019 and applying a difference-in-differences approach, we find that an increased level of hate crimes against Muslims is linked with a reduced vaccination rate among African Muslims.

Moreover, we provide further empirical evidence focusing on the heterogeneous effects by anglophone countries, mobile penetration and areas with StC involved and find that the effects of hate crimes against Muslims in the US on the first-year child vaccination rate among African Muslims are much stronger in anglophone countries and in areas better connected to global Internet information channels are consistent with the interpretation proposed. Additionally, our heterogeneous analysis also indicates that vaccination rates tend to be lower among Muslims in areas in which StC is involved in local health provision, which is consistent with the interpretation proposed.

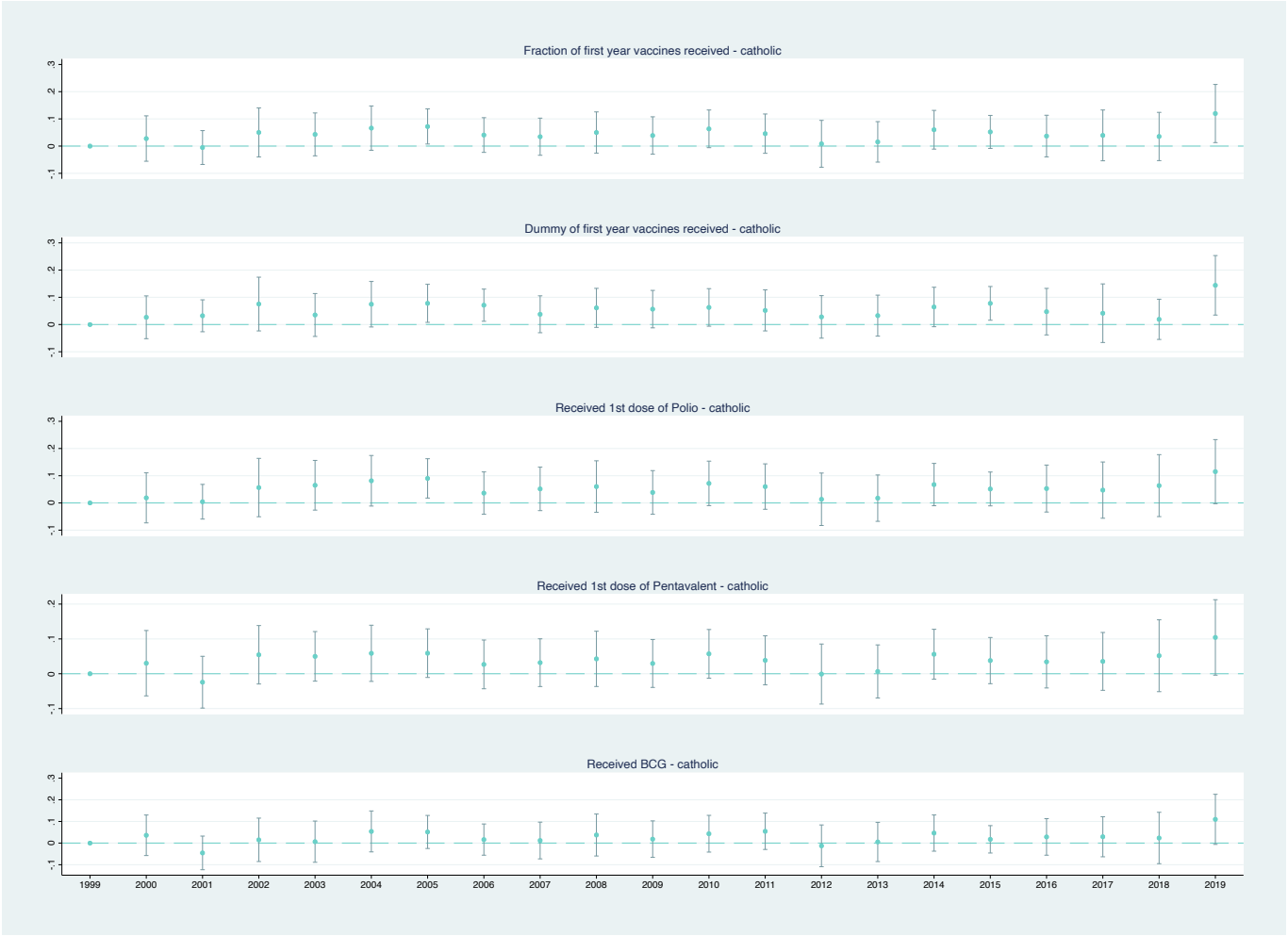
Although we find additional evidence that Islamophobia related to the terrorist attacks perpetrated by Muslims developing in recent decades in the US is a potential reason contributing to the vaccination hesitancy among African Muslims, there is no unique determinant identified. More studies are needed to further investigate the underlying reasons behind it.

Figures

Figure 3.1: Vaccination trends among Muslims

Notes: Year by year coefficients and 95% confidence interval of the event-study regressions.

Figure 3.2: Vaccination trends among Catholics



Notes: Year by year coefficients and 95% confidence interval of the event-study regressions.

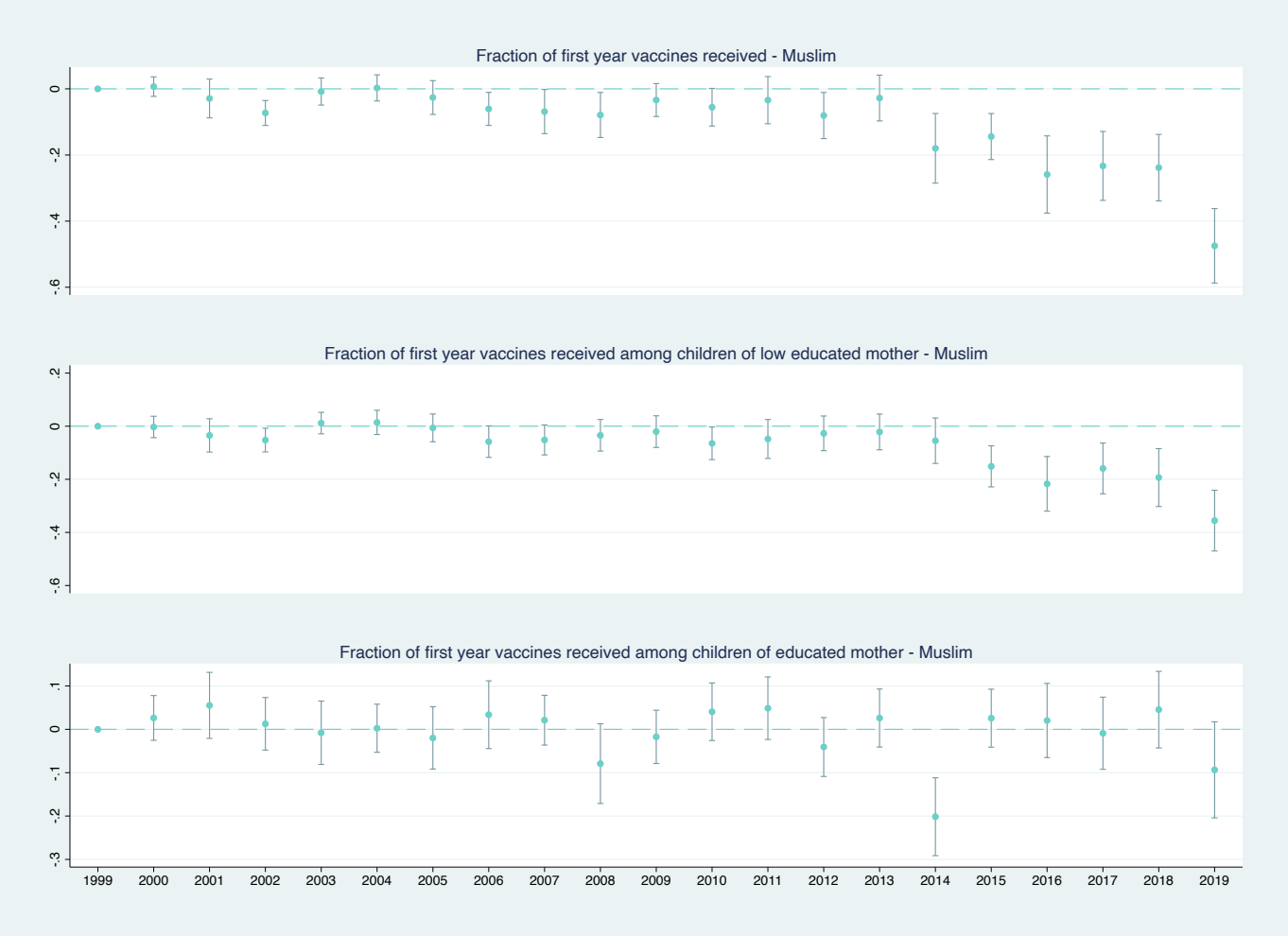
Figure 3.3: Vaccination trends among households of traditional religions

Notes: Year by year coefficients and 95% confidence interval of the event-study regressions.

Figure 3.4: Vaccination trends among households with no religion

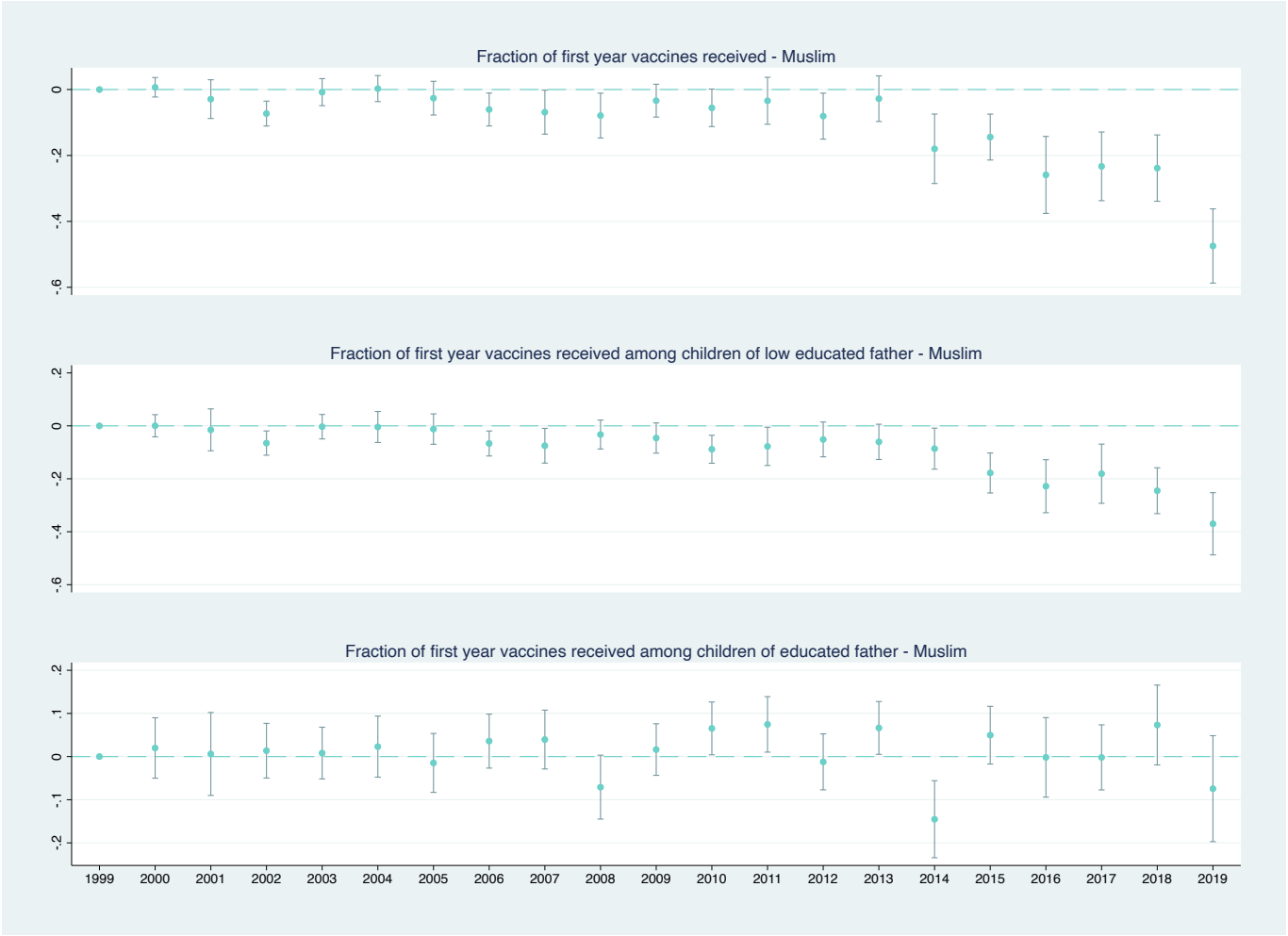
Notes: Year by year coefficients and 95% confidence interval of the event-study regressions.

Figure 3.5: Fraction of vaccinations - Mother's education



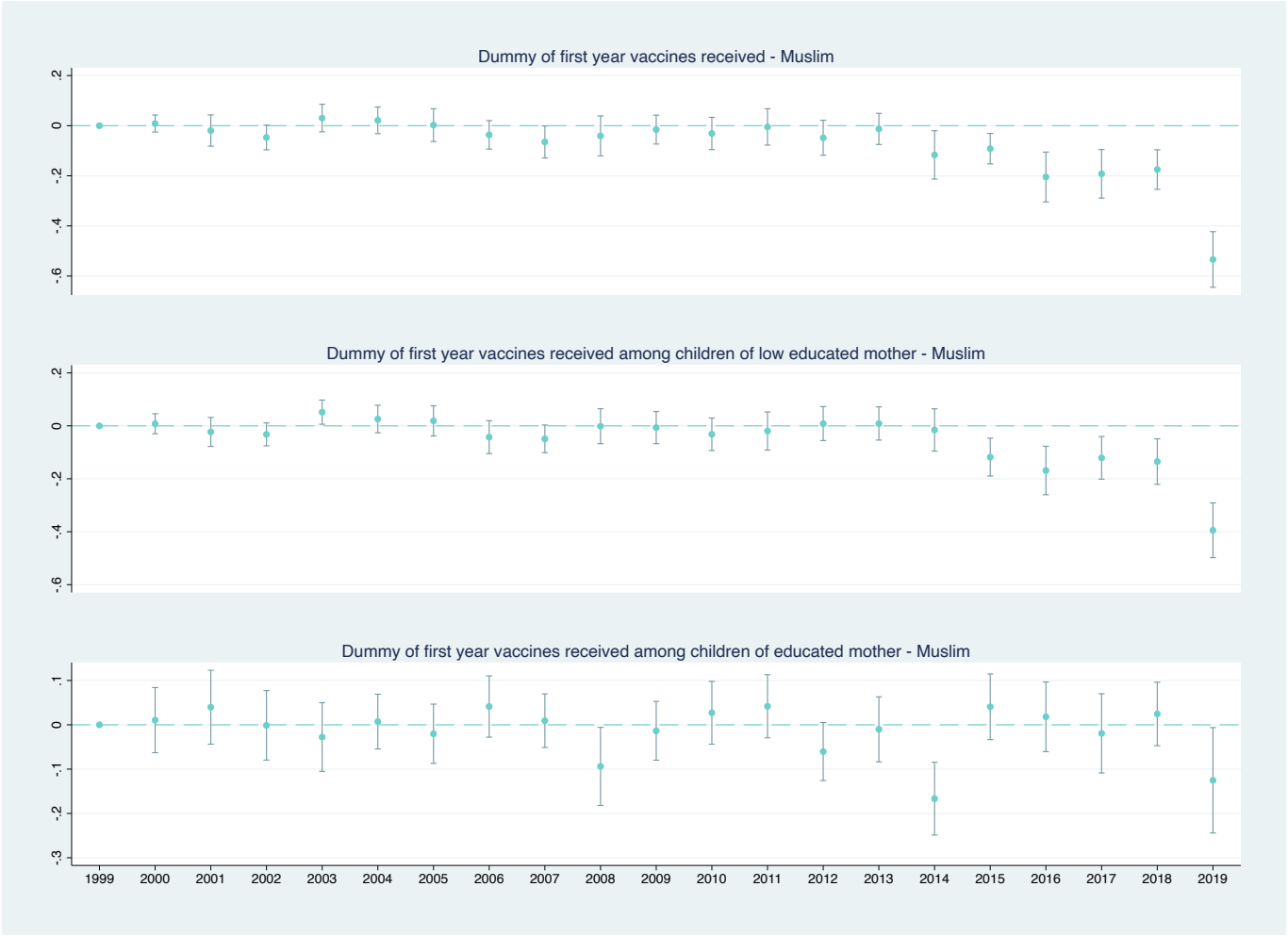
Notes: Year by year coefficients and 95% confidence interval of the event-study regressions.

Figure 3.6: Fraction of vaccinations - Father's education



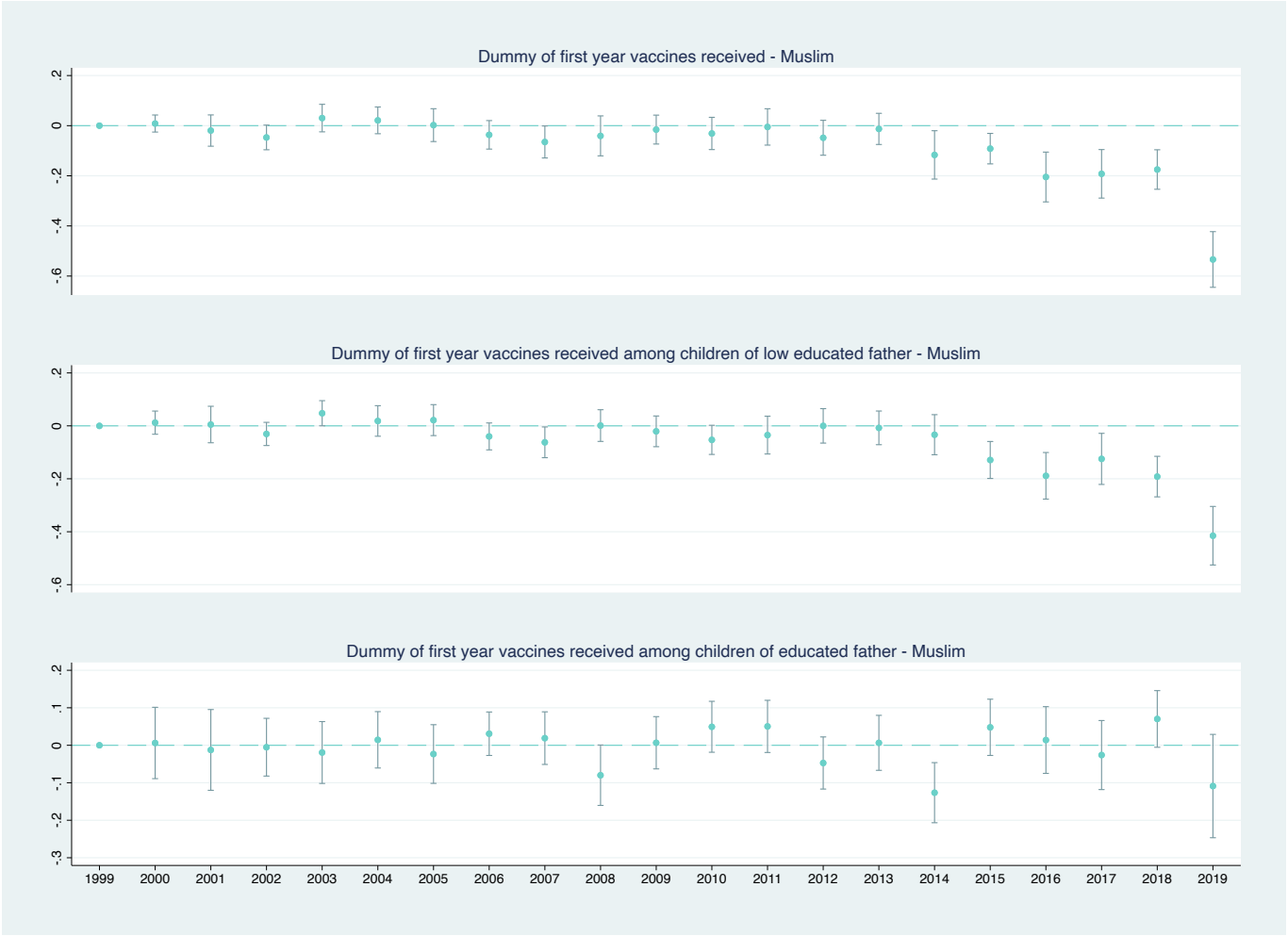
Notes: Year by year coefficients and 95% confidence interval of the event-study regressions.

Figure 3.7: Dummy for all vaccinations - Mother's education



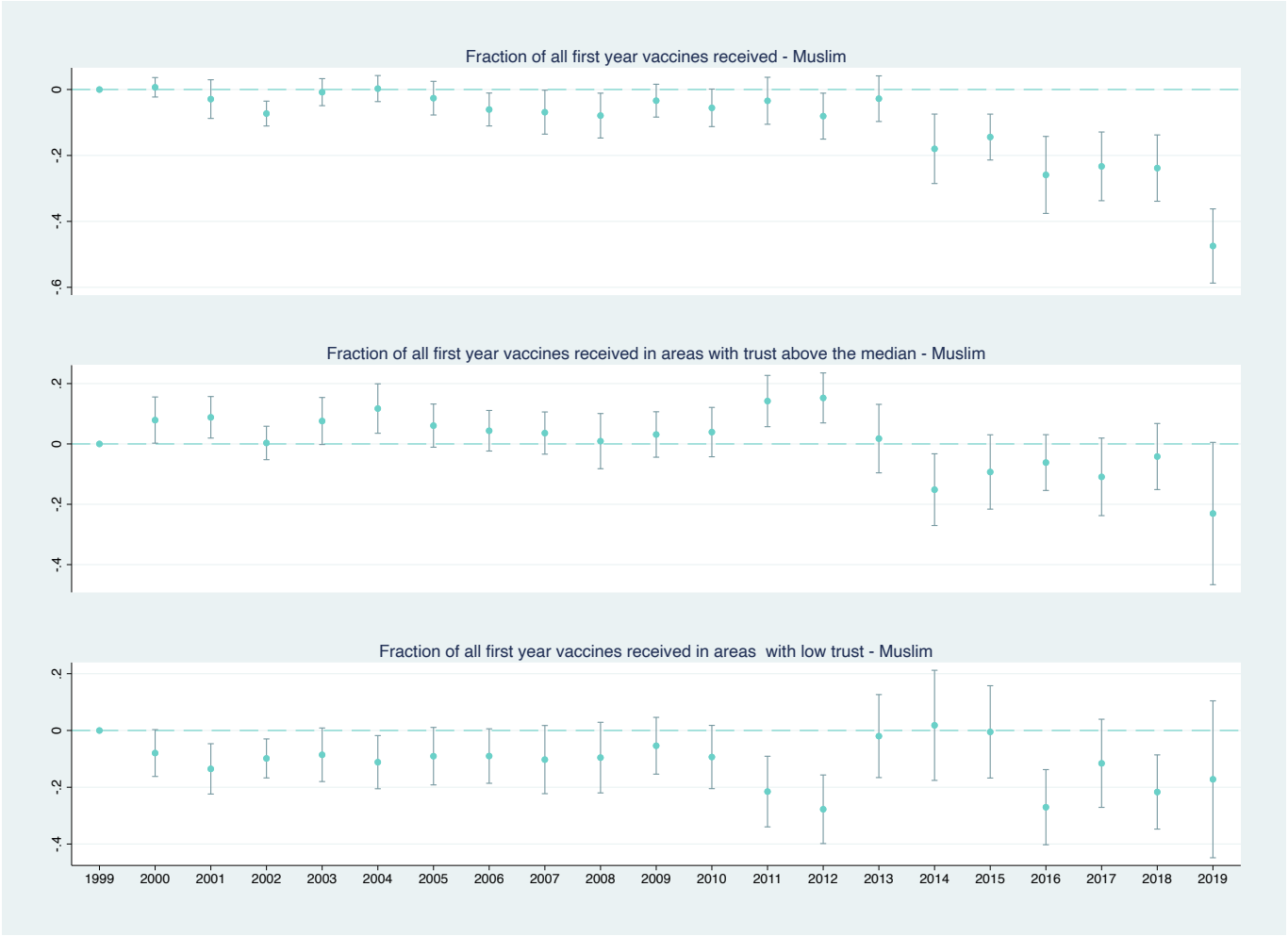
Notes: Year by year coefficients and 95% confidence interval of the event-study regressions.

Figure 3.8: Dummy for all vaccinations - Father's education



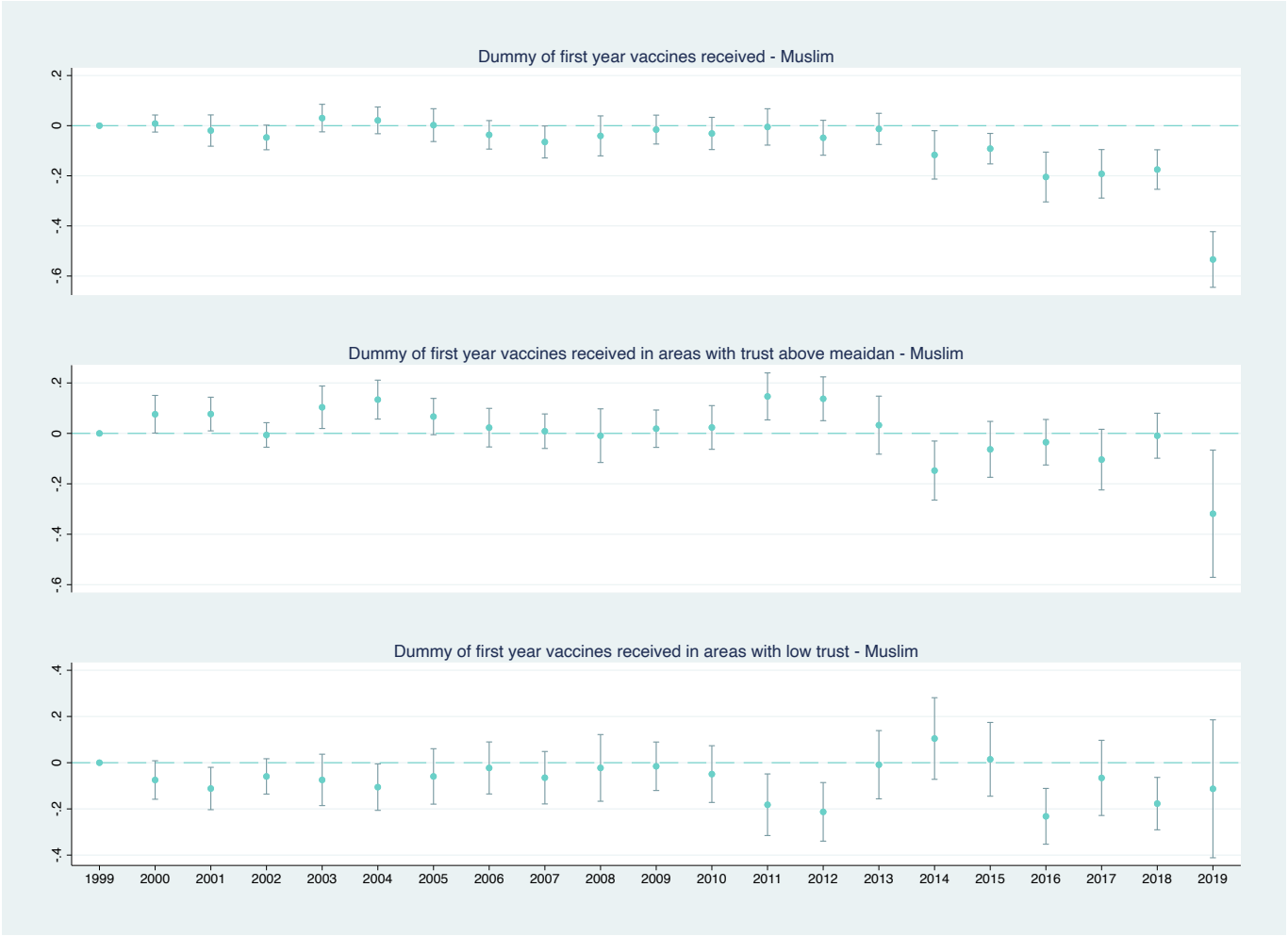
Notes: Year by year coefficients and 95% confidence interval of the event-study regressions.

Figure 3.9: Fraction of vaccinations - Low trust



Notes: Year by year coefficients and 95% confidence interval of the event-study regressions.

Figure 3.10: Dummy for all vaccinations - Low trust



Notes: Year by year coefficients and 95% confidence interval of the event-study regressions.

Tables

Table 3.1: Descriptive statistics.

Variable	N	Mean	SD	Min	Max
Fraction of receiving all doses of basic vaccinations	949,313	0.388	0.461	0	1
Received all doses of Polio, Pentavalent and BCG	949,313	0.322	0.467	0	1
Received 1st dose of Polio	949,313	0.423	0.494	0	1
Received 1st dose of Pentavalent	949,313	0.409	0.492	0	1
Received BCG	949,313	0.433	0.496	0	1
Anti-Muslim hate crime L1	949,313	0.521	0.325	0.076	1.751
Muslim	889,954	0.427	0.495	0	1
Share Muslim	896,893	0.431	0.397	0	1
Month year birth	949,313	590.164	61.833	468	719
Year of birth	949,313	2008.734	5.151	1999	2019
Child's age in months	636,784	27.437	17.229	0	59
Female child	949,313	0.493	0.5	0	1
Birth order	949,313	3.106	1.552	1	5
Mother's age	949,313	28.94	6.915	15	49
Mother's age at first birth	949,313	19.284	3.774	5	48
Mother's education level	949,254	0.815	0.848	0	3
Father's education level	841,160	0.965	0.937	0	3
Wealth index	906,054	2.773	1.397	1	5
Low trust	787,578	0.471	0.499	0	1
Father education (dummy)	841,160	0.6	0.49	0	1
Mother education (dummy)	949,254	0.559	0.497	0	1
Number of cell towers per area	431,513	16.407	21.265	1	135.744
Mobile broadband per 100 inhabitants (dummy)	479,419	0.499	0.5	0	1
NGO(StC) involved	949,313	0.894	0.308	0	1

Note:

Table 3.2: Culture Clash, Mistrust and Vaccination Hesitancy?

Dependent Variable:	Fraction	Received all	Polio 1st	Penta 1st	BCG
	(1)	(2)	(3)	(4)	(5)
Panel A: No controls					
Muslim	0.0121	0.00348	0.0177*	0.0222**	0.0170**
	(0.00868)	(0.00827)	(0.00964)	(0.00898)	(0.00759)
Muslim x Hate Crimes (t-1)	-0.0596***	-0.0466***	-0.0647***	-0.0747***	-0.0675***
	(0.0142)	(0.0128)	(0.0157)	(0.0137)	(0.0154)
Observations	707,265	707,265	707,265	707,265	707,265
Panel B: Full set of controls					
Muslim	0.00930	0.000487	0.0154	0.0209**	0.0118*
	(0.00890)	(0.00821)	(0.0102)	(0.00993)	(0.00683)
Muslim x Hate Crimes (t-1)	-0.0509***	-0.0386***	-0.0570***	-0.0657***	-0.0574***
	(0.00979)	(0.0106)	(0.0109)	(0.00852)	(0.00859)
Observations	378,343	378,343	378,343	378,343	378,343
Month-Year Birth FE	✓	✓	✓	✓	✓
Cluster FE	✓	✓	✓	✓	✓
Survey & age at survey FE	✓	✓	✓	✓	✓

Notes: Standard errors clustered at the ADMI1 level. *** p<0.01, ** p<0.05, * p<0.1. Controls include: child's age, female child dummy, birth order, mother's age at interview and at first birth, mother's and father's education, and wealth index. (1SD+ in HateCrime → 4%SD - of Fraction)

Table 3.3: Culture Clash, Mistrust and Vaccination Hesitancy? - Linear trends

Dependent Variable:	Fraction	Received all	Polio 1st	Penta 1st	BCG
	(1)	(2)	(3)	(4)	(5)
Panel A: No controls					
Muslim	0.255***	0.190***	0.288***	0.278***	0.329***
	(0.0569)	(0.0428)	(0.0630)	(0.0644)	(0.0833)
Muslim x Hate Crimes (t-1)	-0.0507***	-0.0450***	-0.0517***	-0.0635***	-0.0502***
	(0.0114)	(0.0122)	(0.0118)	(0.0100)	(0.00912)
Observations	707,265	707,265	707,265	707,265	707,265
Panel B: Full set of controls					
Muslim	0.318***	0.207***	0.384***	0.372***	0.439***
	(0.0711)	(0.0521)	(0.0847)	(0.0837)	(0.101)
Muslim x Hate Crimes (t-1)	-0.0484***	-0.0442***	-0.0502***	-0.0604***	-0.0471***
	(0.00918)	(0.0112)	(0.00921)	(0.00755)	(0.00568)
Observations	378,343	378,343	378,343	378,343	378,343
Month-Year Birth FE	✓	✓	✓	✓	✓
Cluster FE	✓	✓	✓	✓	✓
Survey & age at survey FE	✓	✓	✓	✓	✓
Muslim specific trend	✓	✓	✓	✓	✓
Country specific trend	✓	✓	✓	✓	✓

Notes: Standard errors clustered at the ADMI1 level. *** p<0.01, ** p<0.05, * p<0.1. Controls include: child's age, female child dummy, birth order, mother's age at interview and at first birth, mother's and father's education, and wealth index. (1SD+ in HateCrime → 3%SD - of Fraction)

Table 3.4: The role of the Internet: mobile broadband subscriptions

Dependent Variable:	Fraction	Received all	Polio 1st	Penta 1st	BCG
	(1)	(2)	(3)	(4)	(5)
Panel A: No controls					
Muslim	-0.0500	-0.292	0.0295	0.0112	0.115
	(0.274)	(0.292)	(0.266)	(0.262)	(0.167)
Muslim x Hate Crimes (t-1)	0.245**	0.322**	0.196	0.198	0.114
	(0.124)	(0.125)	(0.127)	(0.130)	(0.112)
Muslim x Hate Crimes (t-1)	-0.550***	-0.638***	-0.501***	-0.510***	-0.436***
x Mobile Subscription D	(0.116)	(0.109)	(0.123)	(0.124)	(0.120)
Observations	368,350	368,350	368,350	368,350	368,350
Panel B: Full set of controls					
Muslim	-0.166	-0.475	-0.0742	-0.0477	0.0534
	(0.309)	(0.327)	(0.294)	(0.277)	(0.228)
Muslim x Hate Crimes (t-1)	0.292**	0.403***	0.192	0.200	0.189
	(0.133)	(0.131)	(0.136)	(0.140)	(0.124)
Muslim x Hate Crimes (t-1)	-0.587***	-0.717***	-0.493***	-0.508***	-0.487***
x Mobile Subscription D	(0.130)	(0.117)	(0.138)	(0.139)	(0.135)
Observations	197,405	197,405	197,405	197,405	197,405
Month-Year Birth FE	✓	✓	✓	✓	✓
Cluster FE	✓	✓	✓	✓	✓
Survey & age at survey FE	✓	✓	✓	✓	✓
Muslim specific trend	✓	✓	✓	✓	✓
Country specific trend	✓	✓	✓	✓	✓

Notes: Standard errors clustered at the ADMI1 level. *** p<0.01, ** p<0.05, * p<0.1.

Table 3.5: The role of the Internet: cellular tower density

Dependent Variable:	Fraction	Received all	Polio 1st	Penta 1st	BCG
	(1)	(2)	(3)	(4)	(5)
Panel A: No controls					
Muslim	0.0957	-0.0677	0.238**	0.0296	0.187**
	(0.110)	(0.0974)	(0.111)	(0.120)	(0.0833)
Muslim x Hate Crimes (t-1)	-0.0240**	-0.0188*	-0.0259**	-0.0323***	-0.0183**
	(0.00978)	(0.0101)	(0.0110)	(0.00934)	(0.00811)
Muslim x Hate Crimes (t-1)	-0.00403***	-0.00447***	-0.00360***	-0.00433***	-0.00386***
x Cell. Tower Density	(0.000822)	(0.000780)	(0.000813)	(0.000809)	(0.000801)
Observations	663,122	663,122	663,122	663,122	663,122
Panel B: Full set of controls					
Muslim	0.315**	0.0803	0.529***	0.257	0.433***
	(0.150)	(0.139)	(0.154)	(0.164)	(0.119)
Muslim x Hate Crimes (t-1)	-0.0297***	-0.0262**	-0.0315***	-0.0360***	-0.0240***
	(0.0108)	(0.0116)	(0.0114)	(0.00980)	(0.00856)
Muslim x Hate Crimes (t-1)	-0.00365***	-0.00424***	-0.00308***	-0.00413***	-0.00320***
x Cell. Tower Density	(0.000960)	(0.000912)	(0.000927)	(0.000957)	(0.000889)
Observations	371,241	371,241	371,241	371,241	371,241
Month-Year Birth FE	✓	✓	✓	✓	✓
Cluster FE	✓	✓	✓	✓	✓
Survey & age at survey FE	✓	✓	✓	✓	✓
Muslim specific trend	✓	✓	✓	✓	✓
Country specific trend	✓	✓	✓	✓	✓

Notes: Standard errors clustered at the ADMI1 level. *** p<0.01, ** p<0.05, * p<0.1.

Table 3.6: Anglophone test

Dependent Variable:	Fraction	Received all	Polio 1st	Penta 1st	BCG
	(1)	(2)	(3)	(4)	(5)
Panel A: No controls					
Muslim	0.180***	0.116***	0.219***	0.201***	0.275***
	(0.0513)	(0.0375)	(0.0565)	(0.0586)	(0.0773)
Muslim x Hate Crimes (t-1)	0.0810***	0.0857***	0.0685***	0.0803***	0.0475***
	(0.0163)	(0.0157)	(0.0169)	(0.0171)	(0.0147)
Muslim x Hate Crimes (t-1)	-0.157***	-0.155***	-0.144***	-0.168***	-0.116***
	(0.0206)	(0.0213)	(0.0217)	(0.0199)	(0.0179)
Observations	707,265	707,265	707,265	707,265	707,265
Panel B: Full set of controls					
Muslim	0.272***	0.159***	0.346***	0.325***	0.416***
	(0.0686)	(0.0485)	(0.0804)	(0.0814)	(0.0993)
Muslim x Hate Crimes (t-1)	0.0668***	0.0686***	0.0541***	0.0603***	0.0248
	(0.0196)	(0.0194)	(0.0199)	(0.0197)	(0.0157)
Muslim x Hate Crimes (t-1)	-0.130***	-0.126***	-0.116***	-0.133***	-0.0781***
x Anglophone	(0.0223)	(0.0238)	(0.0230)	(0.0207)	(0.0171)
Observations	378,343	378,343	378,343	378,343	378,343
Month-Year Birth FE	✓	✓	✓	✓	✓
Cluster FE	✓	✓	✓	✓	✓
Survey & age at survey FE	✓	✓	✓	✓	✓
Muslim specific trend	✓	✓	✓	✓	✓
Country specific trend	✓	✓	✓	✓	✓

Notes: Standard errors clustered at the ADMI1 level. *** p<0.01, ** p<0.05, * p<0.1. Controls include: child's age, female child dummy, birth order, mother's age at interview and at first birth, mother's and father's education, and wealth index. (1SD+ in HateCrime → 5%SD - of Fraction)

Table 3.7: Who provides immunisation?

Dependent Variable:	Fraction	Received all	Polio 1st	Penta 1st	BCG
	(1)	(2)	(3)	(4)	(5)
Panel A: No controls					
Muslim	0.247***	0.185***	0.279***	0.260***	0.327***
	(0.0567)	(0.0440)	(0.0615)	(0.0638)	(0.0815)
Muslim x Hate Crimes (t-1)	-0.0178	-0.0239	-0.0137	-0.0161	-0.00362
	(0.0156)	(0.0155)	(0.0163)	(0.0175)	(0.0141)
Muslim x Hate Crimes (t-1)	-0.0358*	-0.0216	-0.0415*	-0.0531**	-0.0507***
x StC involved	(0.0208)	(0.0212)	(0.0219)	(0.0213)	(0.0185)
Observations	707,265	707,265	707,265	707,265	707,265
Panel B: Full set of controls					
Muslim	0.323***	0.208***	0.388***	0.368***	0.450***
	(0.0731)	(0.0556)	(0.0856)	(0.0863)	(0.102)
Muslim x Hate Crimes (t-1)	-0.0225	-0.0182	-0.0244	-0.0243	-0.00809
	(0.0172)	(0.0191)	(0.0202)	(0.0204)	(0.0153)
Muslim x Hate Crimes (t-1)	-0.0281	-0.0261	-0.0287	-0.0410*	-0.0429**
x StC involved	(0.0205)	(0.0233)	(0.0235)	(0.0222)	(0.0172)
Observations	378,343	378,343	378,343	378,343	378,343
Month-Year Birth FE	✓	✓	✓	✓	✓
Cluster FE	✓	✓	✓	✓	✓
Survey & age at survey FE	✓	✓	✓	✓	✓
Muslim specific trend	✓	✓	✓	✓	✓
Country specific trend	✓	✓	✓	✓	✓

Notes: Standard errors clustered at the ADMI1 level. *** p<0.01, ** p<0.05, * p<0.1.

Conclusion

This thesis comprises three chapters on health development with a focus on the early stages of life and uses the data from African countries and the United States as case studies. Both the OLS and DiD models were used. Chapter 1 examined the effect of exposure to drought in early life on the sex ratio and health outcomes at birth using data from 33 African countries. Chapter 2 further investigated the effect of exposure to negative shocks while in utero using wildfires as the exogenous shock in a well-institutionalised setting, such as the United States. Chapter 3 first studied the child first-year immunisation rates across Africa among Muslim households and second explored the effect of hate crimes against Muslims recorded in the US on vaccination rates among Muslim households in African countries.

Chapter 1 contributes to the literature by further testing the Trivers–Willard's hypothesis using extensive individual-level survey data and a more sophisticated measure of drought, the SPEI, and provides additional evidence for the causal relationship between in-utero conditions and birth outcomes. This chapter studied the effect of drought exposure at different times during pregnancy on the sex ratio and child health at birth. Using data on individual health and the sex ratio from the DHS program and drought data from the SPEI website, we employed the multi-dimensional linear fixed-effects model. Our results suggest that drought exposure in the first of during pregnancy has a significant effect on the sex ratio at birth, but when we focused on a total of 9-month in utero, we found no significant impact. Moreover, our findings on child health at birth show that drought exposure while in utero is significantly associated with health outcomes at birth, that is, exposure to less drought increases the HAZ score for children, and girls and boys are affected differently with girls tending to be stronger than boys.

Chapter 2 examined the sex ratio at birth and child birth weight after wildfire exposure during pregnancy in a strongly institutionalised setting that had experienced several severe wildfires in recent decades, such as the US. Using data obtained from the Public Use Data Archive of NBER for children born between 2000 and 2004 in the state of California, USA, we showed that exposure to wildfires at different times during pregnancy had an insignificant impact on the sex ratio at birth, which may due to the fact that pregnant women in strongly institutionalized countries have constant access to a high standard of medical care during pregnancy. Moreover, our results show that the health of newborns is significantly affected by wildfire exposure while in utero. Moving to an analysis by trimester, we found that exposure to wildfire in the second trimester had a significant effect on birth weight, and

the effect tended to be stronger in male infants, suggesting that male infants are more fragile than female infants.

Chapter 3 contributes to the existing literature by extending the understanding of the determinants of vaccination hesitancy and the potential mechanism of how the intensity of culture clash between Islam and Western countries influences child immunisation rates among Muslims in Africa. Using data on anti-Muslim hate crimes recorded in US history and the data on child immunisation rates from the DHS program across 35 African countries, we adopted the OLS and DiD models. Our findings show that the first-year vaccination rate for children born to Muslim mothers is lower than for children born to mothers of other religious groups across Africa. Moreover, our results on the impact of culture clashes on child vaccination rates indicate that hate crimes against Muslims in the US have a significant negative effect on the first-year immunisation rate for children in Muslim households across Africa, suggesting that vaccination hesitancy is higher among Muslim groups.

We can now specify the policy implication and discuss potential further research to better explore the topics examined in the three chapters. In Chapter 1, our findings emphasise the need for policymakers to take into account relevant public health policies to help improve the health of mothers and unborn babies, especially in areas where there is a prevalence of drought. The relevance for academic work done on early life shocks which should take into account that in-utero shocks may already generate a selected population at birth. Further research can potentially focus on exploring the causal effects of drought exposure in utero on the sex ratio and health outcomes at birth in more institutionalised settings with a richer dataset. For individuals living in countries with a high standard of healthcare services or social welfare, exposure to drought may be associated with different effects on birth outcomes.

The critical finding of Chapter 2 is that children born to mothers who were exposed to wildfires have lower birth weights. Policymakers should pay more attention to formulating policies to protect vulnerable groups from natural disasters. Moreover, the results in Chapter 2 while showing that well-developed health systems seem to mute the effects found in the context of Africa, also point out that children born in more vulnerable families, such as low-educated or minority women, can still suffer the consequences of a shock in utero, as exposure to wildfire. Special attention should be paid to these. Future studies could potentially look at the association between exposure to wildfire while in utero and the sex ratio at birth using air pollution as an alternative measure to wildfires in strongly institutionalised settings.

Chapter 3 highlights an important implication for policymakers that the Islamophobia wave following the terrorist attacks perpetrated by extreme Muslim groups in Western countries may potentially contribute to vaccination refusal among Muslim households, while more research is needed to establish the causal link we are suggesting, the results in Chapter 3 warn about the potentially far-reaching consequences of culturally hate waves, at times purposefully orchestrated by politicians eager to bank votes. Policies aiming at eliminating Islamophobia to reduce hate crimes against Muslim households and protect Muslim communities should be considered in order to promote Western-led vaccination programmes which in turn increase the global vaccination coverage among Muslim groups. Potential future research on this topic could explore the causal effects of hate crimes against Muslims on vaccine hesitancy among Muslims using hate crime data recorded in Africa.

Appendix A: Appendix to Chapter 1

Figure A1: Histogram of the Number of Droughts per Cluster, 1950-2015: SPEI at the 1-month timescale

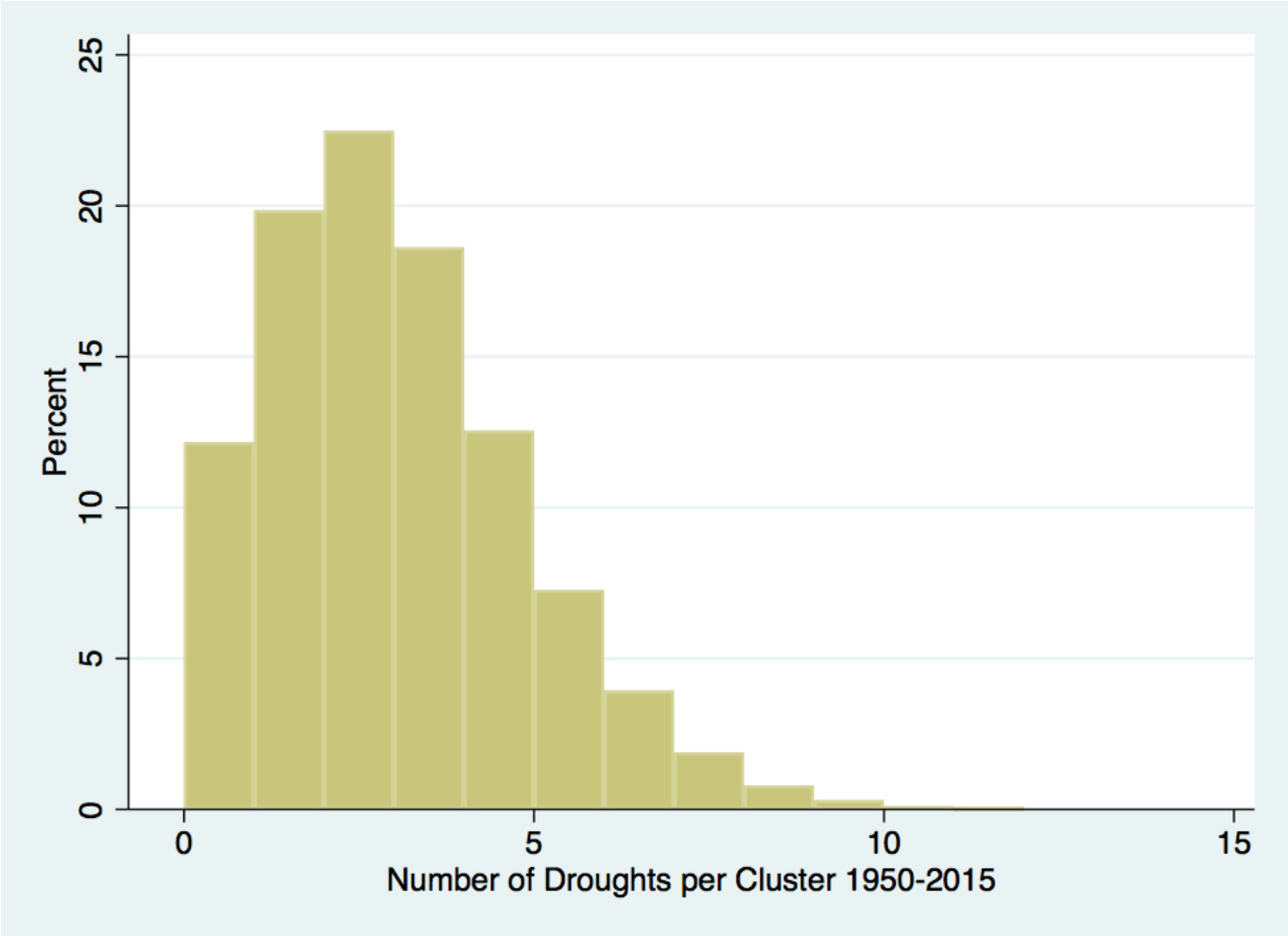
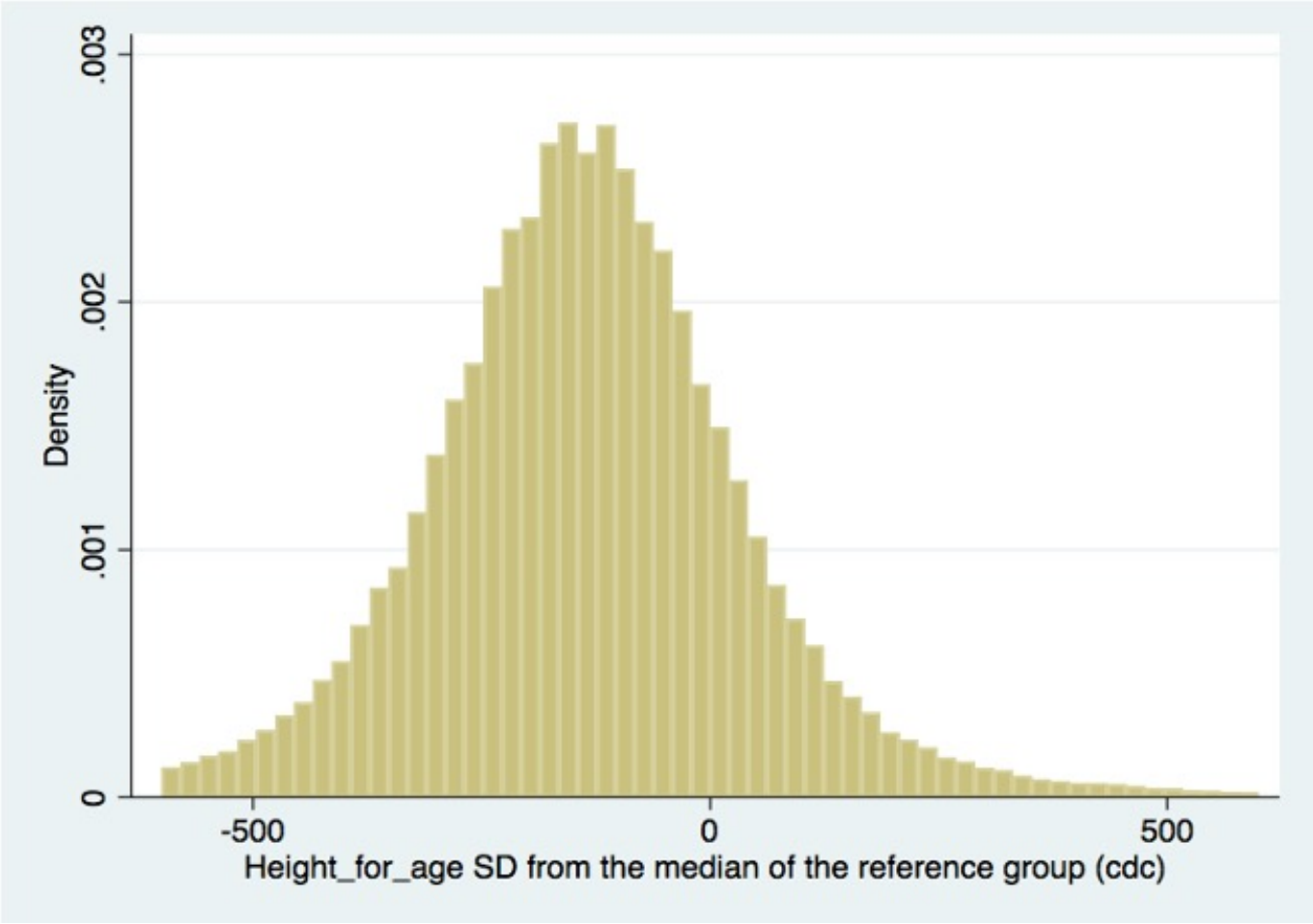


Figure A2: Height-for-age standard deviations from reference median



A1 The derivation of the SPEI

SPEI uses monthly (or weekly) differences between precipitation and potential evapotranspiration (PET). The equation is written as follows:

$$D_i = P_i - PET_i$$

Where D_i is the difference between precipitation p and PET for the month i. P_i is the monthly precipitation. And PET_i is the monthly potential evapotranspiration.

(1) The method of computing PET_i that has been used by Vicente-Serrano et al. (2010) was proposed by Thornthwaite (1948). The equation for obtaining PET is as follows:

$$PET_i = 16.9 \times (\frac{10T}{I})^m$$

Where T is the monthly-mean temperature, and I is the heat index calculated as the sum of 12 monthly . $I = \sum_{i=1}^{12}(\frac{T}{5})^2$ is a coefficient depending on I:
$m = 6.75 \times 10^-7I^3 - 7.71 \times 10^-5I^2 + 1.79 \times 10^-2I + 0.492$.Vicente-Serrano et al. (2010)

(2) Then, following the same procedures as SPI, the D_i series needed to be standardised. Because D_i may take a negative value, so SPEI index adopted a three-parameter Log-logistic distribution. The probability density function of a three-parameter Log-logistic can be as follows Vicente-Serrano et al. (2010):

$$F(x) = [1 + (\frac{\alpha}{x-\gamma})^\beta]^-1$$

where α is scale, β is shape, and γ is the origin parameter. The equations for computing these three parameters are shown below (Vicente-Serrano et al., 2010):

$$\alpha = \frac{(w_0 - 2w_1)}{\Gamma(1+\frac{1}{\beta})\Gamma(1-\frac{1}{\beta})}$$
$$\beta = \frac{2w_1 = w_0}{6w_1 - w_0 - 6w_2}$$
$$\gamma = w_0 - \alpha\Gamma(1+\frac{1}{\beta})\Gamma(1-\frac{1}{\beta})$$

Where w_0, w_1,w_2 are the probability-weighted moments (PWMs) of the D series. And can be calculated as

follow:

$$w_s = \frac{1}{N}\sum i = 1^N (1 - F_i)^s D_i$$
$$F_i = \frac{i - 0.35}{N}$$

where F_i is a frequency estimator, and N is the number of months included.

(3) Finally, with $F(x)$ the SPEI can be obtained by standardising the cumulative probability density:

$$P = 1 - F(x)$$

When: $P \leq 0.5$

$SPEI = w - \frac{C_0 + C_1 w + C_2 w^2}{1 + d_1 w + d_2 w^2 + d_3 w^3}$ where $w = \sqrt{-2ln(P)}$

When: P>0.5 then P is replaced by $1 - P$:

$SPEI = -(w - \frac{C_0 + C_1 w + C_2 w^2}{1 + d_1 w + d_2 w^2 + d_3 w^3})$ where $w = \sqrt{-2ln(P)}$

where the constants are $C0 = 2.515517$, $C1 = 0.802853$, $C2 = 0.010328$, $d1 = 1.432788$, $d2 = 0.189269$, and $d3 = 0.001308$ Vicente-Serrano et al. (2010).

A2 Category of the SPEI

The SPEI is a standardised variable with a mean of zero and a variance of one that expresses the water balance in units of standard deviation from the long-run average. It's been divided into several categories based on the level of SPEI-values. The long-run average is calculated from 1901 to 2015. An index-value of zero corresponds precisely to the long-run water average. For instance, a value of -1 indicates that the water balance is one standard deviation below the long-run average, hence dryer conditions than the long-run average. As indicated by McKee et al. (1993), the SPEI index was defined as follows:

Table A1: SPEI index

SPEI-values	Drought Category
2.00 and above	Extremely wet
1.5 to1.99	Very wet
1.00 to 1.49	Moderate wet
-0.99 to 0.99	Near normal
-1.00 to -1.49	Moderate drought
-1.5 to – 1.99	Severe drought
-2 and less	Extremely drought

Appendix B: Appendix to Chapter 2

Figure B1: Wildfires (in black) in the northern part of California and its corresponding plumes (in grey), 2000-2004

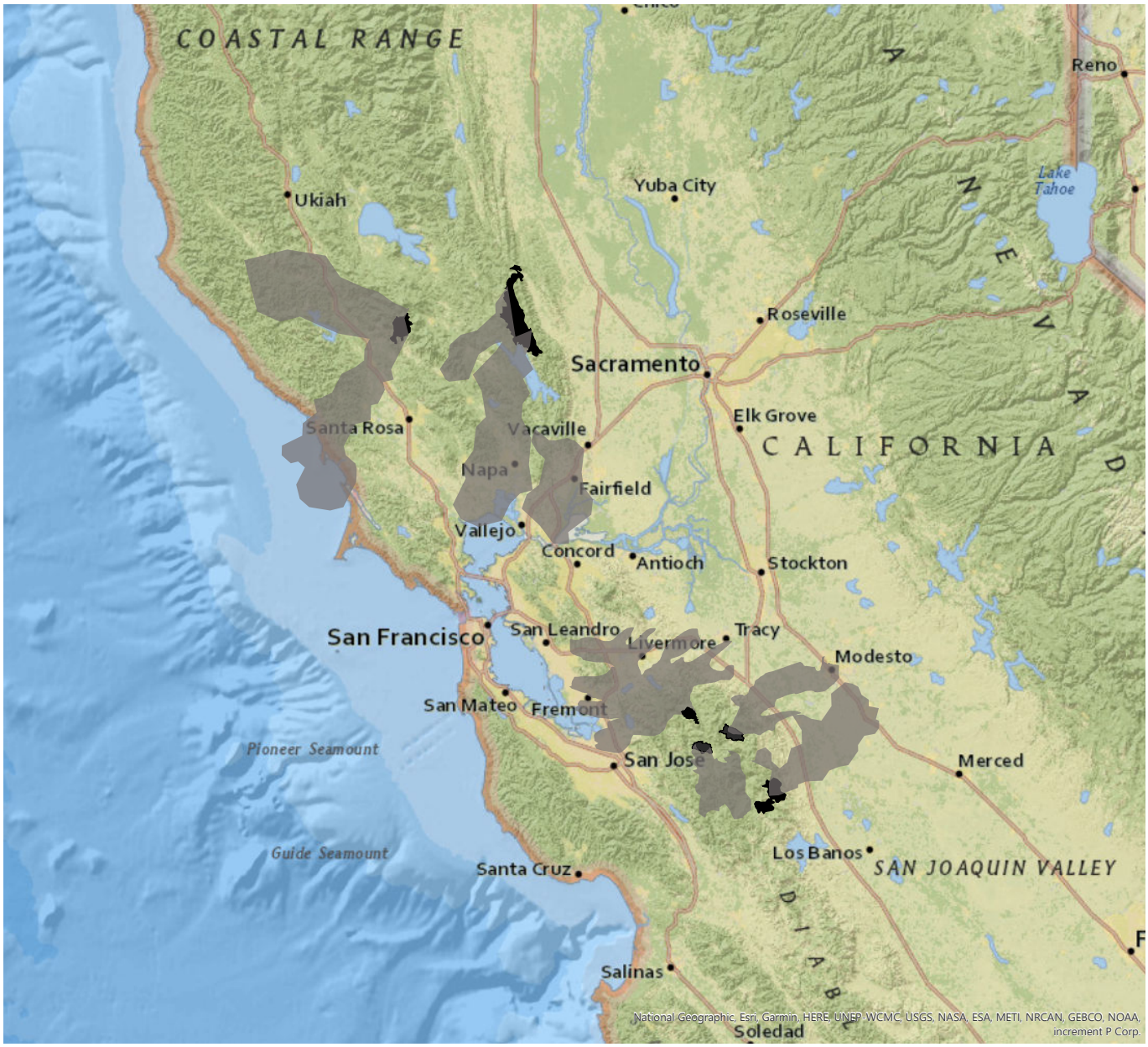


Figure B2: Wildfires (in black) in the southern part of California and its corresponding plumes (in grey), 2000-2004

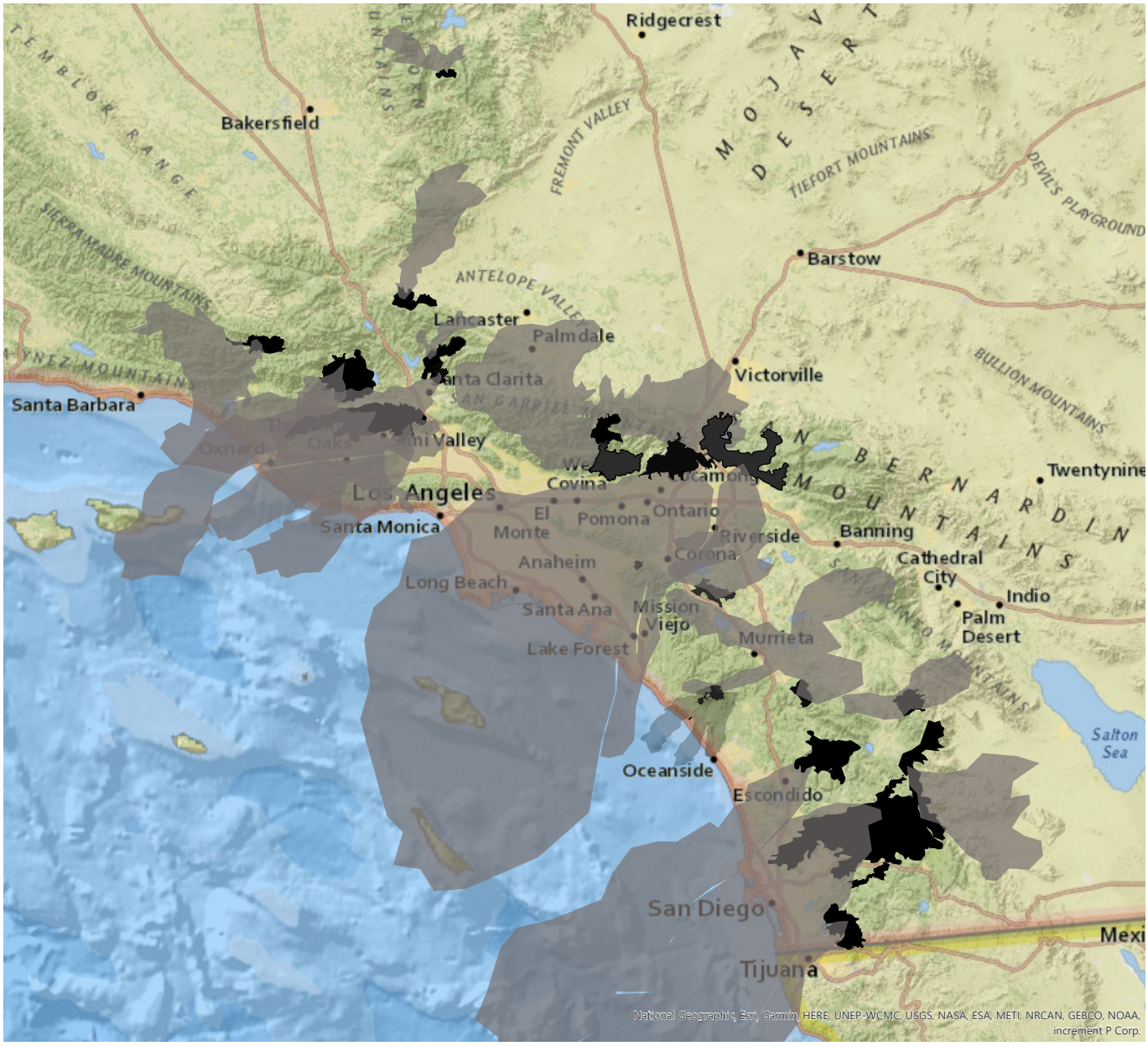
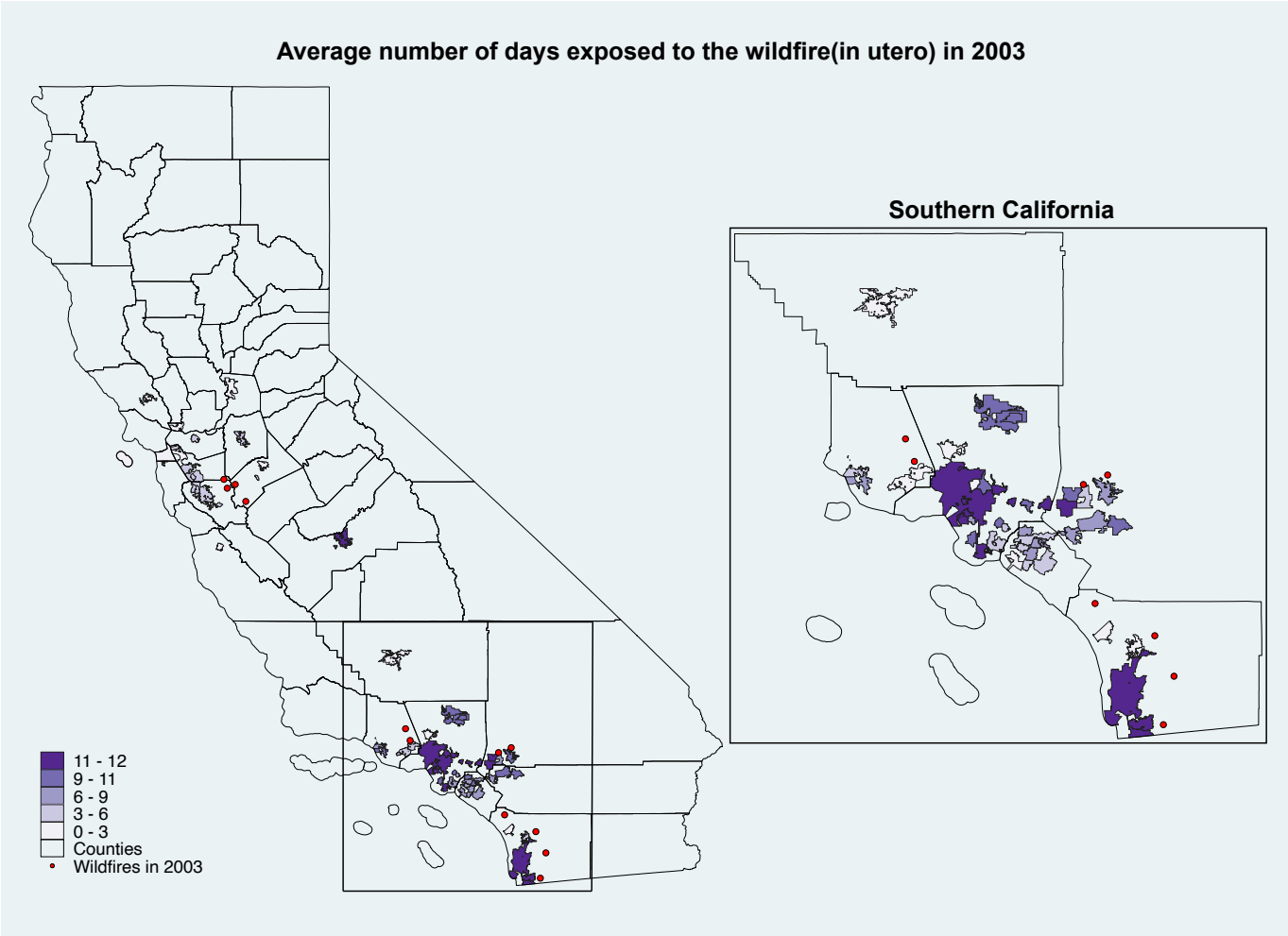


Figure B3: Distribution of wildfire exposure and location of wildfires in 2003



Notes: The dataset for shapefile boundaries for CA State, counties and places are downloaded from California open data portal (California Department of Technology, 2019).

Table B1: The effect of pm2.5 exposure on sex ratio during pregnancy.

	PM2.5>12.1(Yellow) Sex ratio (1)	PM2.5>12.1(Yellow) Sex ratio (2)	PM2.5>35.5(Orange) Sex ratio (3)	PM2.5>35.5(Orange) Sex ratio (4)	PM2.5>55.5(Red) Sex ratio (5)	PM2.5>55.5(Red) Sex ratio (6)
Utero	.0002	.00009	.00063	.0005	.00101	.00086
	(.0003)	(.0003)	(.00089)	(.00085)	(.00171)	(.00168)
Observations	2464	2464	2464	2464	2464	2464
All Controls		✓		✓		✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. the column headings represent the dependent variable. All controls include the Marital status of the mother, Child sex, Maternal ethnicity, Maternal education, Maternal age, Birth order and Gestational weeks. Fixed Effects include month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table B2: The effect of pm2.5 exposure on sex ratio by trimester.

	PM2.5>12.1(Yellow) Sex ratio (1)	PM2.5>12.1(Yellow) Sex ratio (2)	PM2.5>35.5(Orange) Sex ratio (3)	PM2.5>35.5(Orange) Sex ratio (4)	PM2.5>55.5(Red) Sex ratio (5)	PM2.5>55.5(Red) Sex ratio (6)
T1	.00009	-.00001	.00084	.00084	.00174	.00179
	(.00046)	(.00047)	(.00146)	(.00151)	(.00274)	(.00274)
T2	-.00027	-.00039	-.00097	-.00114	-.00247	-.00267
	(.0005)	(.0005)	(.00155)	(.00161)	(.00256)	(.00264)
T3	.00076	.00066	.00208	.00188	.00369	.00341
	(.00051)	(.0005)	(.00156)	(.00152)	(.00249)	(.0025)
Observations	2464	2464	2464	2464	2464	2464
All Controls		✓		✓		✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. the column headings represent the dependent variable. All controls include the Marital status of the mother, Child sex, Maternal ethnicity, Maternal education, Maternal age, Birth order and Gestational weeks. Fixed Effects include month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table B3: The interaction estimations with mother's education during pregnancy.

	PM2.5>12.1(Yellow) Sex ratio (1)	PM2.5>12.1(Yellow) Sex ratio (2)	PM2.5>35.5(Orange) Sex ratio (3)	PM2.5>35.5(Orange) Sex ratio (4)	PM2.5>55.5(Red) Sex ratio (5)	PM2.5>55.5(Red) Sex ratio (6)
Utero	-.00062	-.00092	-.00186	-.00219	-.00453	-.00471
	(.00099)	(.00098)	(.00271)	(.0027)	(.00585)	(.00581)
<12yrs edu	-.0271	-.02957	-.02764	-.02975	-.02832	-.0304
	(.03026)	(.03131)	(.03008)	(.0316)	(.0298)	(.03129)
Interactions:						
Reference: >12yrs edu						
Utero x <12yrs edu	.00159	.00206	.00468	.0052	.01	.01016
	(.00201)	(.00198)	(.0047)	(.00472)	(.0099)	(.00995)
Observations	2464	2464	2464	2464	2464	2464
All Controls		✓		✓		✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. the column headings represent the dependent variable. All controls include the Marital status of the mother, Child sex, Maternal ethnicity, Maternal education, Maternal age, Birth order and Gestational weeks. Fixed Effects include month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table B4: The interaction estimations with mother's education by trimester.

	PM2.5>12.1(Yellow) Sex ratio (1)	PM2.5>12.1(Yellow) Sex ratio (2)	PM2.5>35.5(Orange) Sex ratio (3)	PM2.5>35.5(Orange) Sex ratio (4)	PM2.5>55.5(Red) Sex ratio (5)	PM2.5>55.5(Red) Sex ratio (6)
T1	-.00151	-.00201	-.00227	-.00295	-.00708	-.00758
	(.00166)	(.00157)	(.0047)	(.00461)	(.00967)	(.00943)
T2	-.00291	-.00317*	-.01064**	-.0111**	-.01562*	-.01596*
	(.00182)	(.00185)	(.00429)	(.00448)	(.00784)	(.00814)
T3	.00303**	.00287**	.00709*	.00716*	.00927	.00957
	(.00134)	(.00139)	(.00399)	(.00393)	(.00754)	(.00736)
<12yrs edu	-.02734	-.02941	-.02763	-.02967	-.02829	-.03107
	(.03025)	(.03108)	(.03014)	(.03138)	(.0297)	(.03096)
Interactions						
Reference: >12yrs edu						
T1 x <12yrs edu	.0033	.00422	.00616	.00773	.0165	.01774
	(.00337)	(.00314)	(.00862)	(.00833)	(.01536)	(.01493)
T2 x <12yrs edu	.00532	.00569	.01825**	.01884**	.02387*	.02417*
	(.00385)	(.0039)	(.00765)	(.00798)	(.01364)	(.01411)
T3 x <12yrs edu	-.0045*	-.00434	-.00947	-.00983	-.01033	-.01133
Observations	2464	2464	2464	2464	2464	2464
All Controls		✓		✓		✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. the column headings represent the dependent variable. All controls include the Marital status of the mother, Child sex, Maternal ethnicity, Maternal education, Maternal age, Birth order and Gestational weeks. Fixed Effects include month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table B5: The interaction estimations with mother's race during pregnancy.

	PM2.5>12.1(Yellow) Sex ratio (1)	PM2.5>12.1(Yellow) Sex ratio (2)	PM2.5>35.5(Orange) Sex ratio (3)	PM2.5>35.5(Orange) Sex ratio (4)	PM2.5>55.5(Red) Sex ratio (5)	PM2.5>55.5(Red) Sex ratio (6)
Utero	-.00103	-.00128	-.00177	-.00219	-.00555	-.00588
	(.00098)	(.001)	(.00319)	(.00316)	(.00633)	(.00631)
ethnic Others	-.02698	-.01969	-.0253	-.01798	-.02662	-.019
	(.02286)	(.03003)	(.0227)	(.02982)	(.02277)	(.02989)
Interactions						
Reference: ethnic Hispanic						
utero x ethnic others	.00259	.00289	.00463	.0052	.01262	.01291
	(.00189)	(.00194)	(.00531)	(.00532)	(.01054)	(.01059)
Observations	2464	2464	2464	2464	2464	2464
All Controls		✓		✓		✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. the column headings represent the dependent variable. All controls include the Marital status of the mother, Child sex, Maternal ethnicity, Maternal education, Maternal age, Birth order and Gestational weeks. Fixed Effects include month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table B6: The interaction estimations with mother's race by trimester.

	PM2.5>12.1(Yellow) Sex ratio (1)	PM2.5>12.1(Yellow) Sex ratio (2)	PM2.5>35.5(orange) Sex ratio (3)	PM2.5>35.5(orange) Sex ratio (4)	PM2.5>55.5(Red) Sex ratio (5)	PM2.5>55.5(Red) Sex ratio (6)
T1	-.00051	-.00105	-.00029	-.00141	-.00608	-.0074
	(.0021)	(.00211)	(.00642)	(.00645)	(.01155)	(.01156)
T2	-.00384**	-.00411**	-.00958**	-.01008**	-.01472*	-.01512*
	(.00162)	(.00165)	(.00397)	(.00403)	(.00748)	(.00754)
T3	.00156	.00161	.00465	.00499	.0035	.00427
	(.00161)	(.0016)	(.00464)	(.00441)	(.00746)	(.0072)
ethnic others	-.02628	-.018	-.02417	-.01606	-.0255	-.01721
	(.02297)	(.0303)	(.02278)	(.03005)	(.02295)	(.03016)
Interactions						
Reference: ethnic Hispanic						
T1 x ethnic Others	.00133	.00224	.00241	.00453	.01515	.01783
	(.00442)	(.00443)	(.01171)	(.01171)	(.01944)	(.01945)
T2 x ethnic Others	.00753**	.00781**	.01619**	.01674**	.02338*	.02353*
	(.00307)	(.00313)	(.00726)	(.00745)	(.01321)	(.01344)
T3 x ethnic Others	-.00162	-.00194	-.00466	-.00559	.00037	-.00162
	(.00327)	(.00331)	(.00734)	(.00706)	(.01216)	(.01176)
Observations	2464	2464	2464	2464	2464	2464
All Controls		✓		✓		✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. the column headings represent the dependent variable. All controls include the Marital status of the mother, Child sex, Maternal ethnicity, Maternal education, Maternal age, Birth order and Gestational weeks. Fixed Effects include month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table B7: The effect of pm2.5 exposure on birth weight by trimesters and in a total of 9 months in utero.

	Birth weight All (1)	Birth weight Boys (2)	Birth weight Girls (3)	Birth weight All (4)	Birth weight Boys (5)	Birth weight Girls (6)
Panel A: pm2.5>12.1(Yellow)						
Utero	-.39773**	-.17416	-.61719***	-.47866***	-.33892*	-.62412***
	(.16715)	(.25128)	(.18099)	(.11333)	(.1826)	(.12554)
T1	-.66172**	-.50802	-.90677***	-.75985***	-.6199*	-.90589***
	(.30037)	(.53773)	(.24192)	(.19162)	(.36887)	(.24227)
T2	-.26121	-.17218	-.39091	-.49041***	-.50806**	-.47691*
	(.23149)	(.21869)	(.40661)	(.16815)	(.23153)	(.25794)
T3	-.27857	.15711	-.56607**	-.19566	.11403	-.50386**
	(.18681)	(.34929)	(.25466)	(.2018)	(.32578)	(.24387)
Panel B: pm2.5>35.5(Orange)						
Utero	-1.39195***	-.80342	-2.00205***	-1.32292***	-.97002	-1.69703***
	(.39365)	(.63185)	(.4959)	(.4531)	(.58402)	(.50787)
T1	-2.19799***	-2.48011*	-2.13739**	-2.11723***	-2.4308***	-1.78744*
	(.69082)	(1.38892)	(.89083)	(.52163)	(.89283)	(1.00604)
T2	-1.19528*	-.30484	-2.19643**	-1.56059***	-1.042	-2.13108***
	(.61267)	(.63801)	(.97306)	(.5419)	(.902)	(.53224)
T3	-.82585	.30278	-1.68242**	-.33162	.50755	-1.181
	(.59777)	(.87934)	(.74159)	(.6814)	(.94447)	(.71708)
Panel C: pm2.5>55.5(Red)						
Utero	-2.31329**	-1.1163	-3.65436***	-2.0925*	-1.32686	-2.91637**
	(.87513)	(1.20373)	(1.24525)	(1.07152)	(1.31393)	(1.26143)
T1	-2.54767	-2.67451	-2.86306	-2.36435	-2.95125	-1.77834
	(1.60093)	(2.02407)	(2.40977)	(1.80968)	(2.02613)	(2.51442)
T2	-2.20256	.47044	-5.13342***	-2.61866**	-.81093	-4.53569***
	(1.4392)	(1.85062)	(1.74286)	(1.15078)	(1.83427)	(1.24625)
T3	-2.19864*	-1.13332	-3.00883	-1.35254	-.29376	-2.46097
	(1.20597)	(1.75979)	(1.81822)	(1.3672)	(1.80036)	(1.67264)
Observations	927231	472192	455039	927231	472192	455039
All Controls				✓	✓	✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. the column headings represent the dependent variable. All controls include the Marital status of the mother, Child sex, Maternal ethnicity, Maternal education, Maternal age, Birth order and Gestational weeks. Fixed Effects include month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table B8: The interaction estimations between pm2.5(yellow) and mother's education during pregnancy

	Birth weight All (1)	Birth weight Boys (2)	Birth weight Girls (3)	Birth weight All (4)	Birth weight Boys (5)	Birth weight Girls (6)
Utero yellow	-.42208*	-.08658	-.77796***	-.46763*	-.2723	-.6761**
	(.22982)	(.28735)	(.21474)	(.23828)	(.2838)	(.25405)
1bn.edu>12 yrs						
2.edu<12 yrs	-12.07146**	-16.23941***	-7.30307	-20.10002***	-24.20125***	-15.85582***
	(5.58833)	(5.77028)	(5.43244)	(2.38305)	(2.41599)	(2.5802)
Interactions						
1bn.edu>12 yrs x utero yellow						
2.edu<12 yrs x utero yellow	.02713	-.21578	.31962	-.02306	-.14031	.10792
	(.63259)	(.67146)	(.62096)	(.53228)	(.56753)	(.53651)
Observations	927231	472192	455039	927231	472192	455039
All Controls				✓	✓	✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. the column headings represent the dependent variable. All controls include the Marital status of the mother, Child sex, Maternal ethnicity, Maternal education, Maternal age, Birth order and Gestational weeks. Fixed Effects include month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table B9: The interaction estimations between pm2.5(yellow) and mother's race during pregnancy

	Birth weight All (1)	Birth weight Boys (2)	Birth weight Girls (3)	Birth weight All (4)	Birth weight Boys (5)	Birth weight Girls (6)
Utero yellow	-.80469*	-.56922	-.98902***	-.78138**	-.62902	-.93412***
	(.42484)	(.57773)	(.36915)	(.3206)	(.39781)	(.32248)
1bn.ethinic hispanic						
ethinic others	-39.94761***	-35.82626***	-44.973***	-50.35267***	-48.18994***	-52.67966***
	(6.68973)	(6.62153)	(6.99459)	(7.32203)	(7.12197)	(7.78914)
Interactions						
1bn. hispanic x utero yellow						
2. others x utero yellow	.78515	.75369	.72815	.57298	.54295	.59367
	(.62665)	(.78171)	(.52198)	(.50928)	(.60356)	(.47859)
Observations	927231	472192	455039	927231	472192	455039
All Controls				✓	✓	✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. the column headings represent the dependent variable. All controls include the Marital status of the mother, Child sex, Maternal ethnicity, Maternal education, Maternal age, Birth order and Gestational weeks. Fixed Effects include month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table B10: The interaction estimations between pm2.5(orange) and mother's education during pregnancy

	Birth weight All (1)	Birth weight Boys (2)	Birth weight Girls (3)	Birth weight All (4)	Birth weight Boys (5)	Birth weight Girls (6)
Utero orange	-1.61186*	-.73258	-2.62433***	-1.437	-.93386	-1.97989*
	(.81169)	(.95758)	(.79938)	(1.00556)	(1.10883)	(1.04683)
1bn.edu>12 yrs						
2.edu<12 yrs	-12.24984**	-16.47586***	-7.46371	-20.25185***	-24.37665***	-15.97892***
	(5.60403)	(5.79925)	(5.42865)	(2.39126)	(2.43009)	(2.58301)
Interactions						
1bn.edu>12 yrs x utero orange						
2.edu<12 yrs x utero orange	.36362	-.17748	1.10046	.20657	-.06587	.509
	(1.51842)	(1.54706)	(1.58967)	(1.34576)	(1.37725)	(1.426)
Observations	927231	472192	455039	927231	472192	455039
All Controls				✓	✓	✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. the column headings represent the dependent variable. All controls include the Marital status of the mother, Child sex, Maternal ethnicity, Maternal education, Maternal age, Birth order and Gestational weeks. Fixed Effects include month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table B11: The interaction estimations between pm2.5(orange) and mother's race during pregnancy

	Birth weight All (1)	Birth weight Boys (2)	Birth weight Girls (3)	Birth weight All (4)	Birth weight Boys (5)	Birth weight Girls (6)
Utero orange	-2.00542**	-1.48416	-2.4429***	-1.49265*	-1.11922	-1.8756**
	(.8186)	(1.0969)	(.84794)	(.81031)	(.854)	(.93037)
1bn.ethinic hispanic						
ethinic others	-39.56159***	-35.57409***	-44.44771***	-49.65935***	-47.51873***	-51.96311***
	(6.74523)	(6.65701)	(7.01758)	(7.33444)	(7.15712)	(7.74325)
Interactions						
1bn. hispanic x utero orange						
2. others x utero orange	1.43718	1.56724	1.06767	.38142	.33156	.40604
	(1.44775)	(1.70158)	(1.30831)	(1.30815)	(1.46481)	(1.31437)
Observations	927231	472192	455039	927231	472192	455039
All Controls				✓	✓	✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. the column headings represent the dependent variable. All controls include the Marital status of the mother, Child sex, Maternal ethnicity, Maternal education, Maternal age, Birth order and Gestational weeks. Fixed Effects include month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table B12: The interaction estimations between pm2.5(red) and mother's education during pregnancy

	Birth weight All (1)	Birth weight Boys (2)	Birth weight Girls (3)	Birth weight All (4)	Birth weight Boys (5)	Birth weight Girls (6)
Utero red	-2.08081	-.6486	-3.98923*	-1.82588	-1.1437	-2.58533
	(1.9919)	(2.2169)	(2.18229)	(2.22745)	(2.4924)	(2.34063)
1bn.edu>12 yrs						
2.edu<12 yrs	-11.92668**	-16.39964***	-6.89179	-20.01633***	-24.33881***	-15.53228***
	(5.33618)	(5.48119)	(5.23381)	(2.28061)	(2.32601)	(2.44975)
Interactions						
1bn.edu>12 yrs x utero red						
2.edu<12 yrs x utero red	-.36943	-.76413	.54697	-.43066	-.29725	-.53204
	(2.75517)	(2.89935)	(2.96421)	(2.40501)	(2.61607)	(2.54309)
Observations	927231	472192	455039	927231	472192	455039
All Controls				✓	✓	✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. the column headings represent the dependent variable. All controls include the Marital status of the mother, Child sex, Maternal ethnicity, Maternal education, Maternal age, Birth order and Gestational weeks. Fixed Effects include month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table B13: The interaction estimations between pm2.5(red) and mother's race during pregnancy

	Birth weight All (1)	Birth weight Boys (2)	Birth weight Girls (3)	Birth weight All (4)	Birth weight Boys (5)	Birth weight Girls (6)
Utero red	-.22947	-.1494	-.30732	-.32172	-.26243	-.38135
	(.23852)	(.25536)	(.3199)	(.2338)	(.20082)	(.31842)
1bn.ethinic hispanic						
ethinic others	-39.23097***	-34.82216***	-44.64522***	-49.95078***	-47.57837***	-52.51977***
	(6.48924)	(6.42079)	(6.82079)	(7.1463)	(6.92093)	(7.64127)
Interactions						
1bn. hispanic x utero red						
2. others x utero red	.17632	-.03969	.36317	.17121	.01222	.33545
	(.33678)	(.36493)	(.34305)	(.27289)	(.28578)	(.29827)
Observations	927231	472192	455039	927231	472192	455039
All Controls				✓	✓	✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. the column headings represent the dependent variable. All controls include the Marital status of the mother, Child sex, Maternal ethnicity, Maternal education, Maternal age, Birth order and Gestational weeks. Fixed Effects include month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table B14: The interaction between pm2.5(yellow) and mother's education by trimesters.

	Birth weight All (1)	Birth weight Boys (2)	Birth weight Girls (3)	Birth weight All (4)	Birth weight Boys (5)	Birth weight Girls (6)
T1 yellow	-.56518**	-.4787	-.69839**	-.76746***	-.8381***	-.69712*
	(.23174)	(.33868)	(.33905)	(.22545)	(.30078)	(.38154)
T2 yellow	-.11139	.24676	-.55056	-.21237	.08702	-.5364**
	(.27794)	(.36583)	(.33197)	(.25004)	(.37644)	(.2117)
T3 yellow	-.62017	-.05837	-1.10636***	-.45054	-.09231	-.81214*
	(.49548)	(.59978)	(.39184)	(.46442)	(.60072)	(.40748)
1bn.edu>12yrs						
2.edu<12 yrs	-12.08998**	-16.2708***	-7.30271	-20.12268***	-24.24035***	-15.85781***
	(5.59763)	(5.78548)	(5.43669)	(2.38627)	(2.42418)	(2.58141)
Interactions						
1bn.edu>12 yrs x T1 yellow						
2.edu<12 yrs x T1 yellow	-.24125	-.10591	-.46544	.0081	.44027	-.44163
	(.67275)	(.77482)	(.66747)	(.53357)	(.59245)	(.55053)
1bn.edu>12 yrs x T2 yellow						
2.edu<12 yrs x T2 yellow	-.33309	-.89398	.3073	-.56756	-1.20485**	.11261
	(.48466)	(.55892)	(.54994)	(.46348)	(.56416)	(.51894)
1bn.edu>12 yrs x T3 yellow						
2.edu<12 yrs x T3 yellow	.65215	.37772	1.06379	.49091	.3811	.60993
	(.91653)	(1.0534)	(.79848)	(.76736)	(.92049)	(.68411)
Observations	927231	472192	455039	927231	472192	455039
All Controls				✓	✓	✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. the column headings represent the dependent variable. All controls include the Marital status of the mother, Child sex, Maternal ethnicity, Maternal education, Maternal age, Birth order and Gestational weeks. Fixed Effects include month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table B15: The interaction between pm2.5(yellow) and mother's race by trimesters.

	Birth weight All (1)	Birth weight Boys (2)	Birth weight Girls (3)	Birth weight All (4)	Birth weight Boys (5)	Birth weight Girls (6)
T1 yellow	-1.31379**	-1.10487	-1.57559***	-1.203***	-1.00482*	-1.39874***
	(.52594)	(.83273)	(.43683)	(.38036)	(.54595)	(.49889)
T2 yellow	-.74187*	-.62569	-.81505	-.7735**	-.79023*	-.75404*
	(.4275)	(.428)	(.59659)	(.37442)	(.4684)	(.43956)
T3 yellow	-.38168	.00994	-.61291	-.38738	-.10234	-.67644
	(.41595)	(.70997)	(.41763)	(.32388)	(.47235)	(.42851)
1bn.ethinic hispanic						
ethinic others	-39.93446***	-35.81385***	-44.96744***	-50.35459***	-48.19523***	-52.68133***
	(6.69474)	(6.6297)	(6.99535)	(7.32701)	(7.13457)	(7.78854)
Interactions						
1bn. hispanic x T1						
2. others x T1	1.25859*	1.14255	1.30325**	.8488	.72932	.95466
	(.65184)	(.79688)	(.59911)	(.54829)	(.60856)	(.6084)
1bn. hispanic x T2						
2. others x T2	.90152	.85304	.78576	.51718	.50721	.51517
	(.60201)	(.68811)	(.60156)	(.56955)	(.66776)	(.55946)
1bn. hispanic x T3						
2. others x T3	.20034	.26269	.12511	.36431	.41141	.32867
	(.78083)	(1.1628)	(.542)	(.55136)	(.81422)	(.53158)
Observations	927231	472192	455039	927231	472192	455039
All Controls				✓	✓	✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. the column headings represent the dependent variable. All controls include the Marital status of the mother, Child sex, Maternal ethnicity, Maternal education, Maternal age, Birth order and Gestational weeks. Fixed Effects include month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table B16: The interaction between pm2.5(orange) and mother's education by trimesters.

	Birth weight All (1)	Birth weight Boys (2)	Birth weight Girls (3)	Birth weight All (4)	Birth weight Boys (5)	Birth weight Girls (6)
T1 orange	-1.95475***	-2.38072*	-1.68554	-2.20114***	-3.03187**	-1.34776
	(.68419)	(1.36722)	(1.22051)	(.80141)	(1.14649)	(1.41575)
T2 orange	-1.28669	.06407	-2.98314***	-1.26928*	-.15609	-2.48061***
	(.84898)	(.90702)	(1.10984)	(.74395)	(1.02066)	(.71722)
T3 orange	-1.64199	.05143	-3.21597**	-.87878	.33047	-2.11509
	(1.91606)	(2.04415)	(1.59301)	(1.85117)	(2.12331)	(1.70402)
1bn.edu>12yrs						
2.edu<12 yrs	-12.24667**	-16.47286***	-7.45123	-20.25366***	-24.37854***	-15.97174***
	(5.61126)	(5.80883)	(5.43207)	(2.39341)	(2.43444)	(2.5839)
Interactions						
1bn.edu>12 yrs x T1 orange						
2.edu<12 yrs x .T1 orange	-.49182	-.24366	-.83395	.1412	1.04686	-.78099
	(1.40506)	(1.51053)	(1.65123)	(1.24365)	(1.42485)	(1.40235)
1bn.edu>12 yrs x T2 orange						
2.edu<12 yrs x T2 orange	.14582	-.69417	1.37551	-.48964	-1.50884	.60523
	(1.13019)	(1.23697)	(1.21276)	(.94115)	(1.06905)	(1.06208)
1bn.edu>12 yrs x T3 orange						
2.edu<12 yrs x T3 orange	1.36699	.36294	2.62604	.9191	.26215	1.59957
	(2.70548)	(2.81457)	(2.59001)	(2.3551)	(2.52536)	(2.3099)
Observations	927231	472192	455039	927231	472192	455039
All Controls				✓	✓	✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. the column headings represent the dependent variable. All controls include the Marital status of the mother, Child sex, Maternal ethnicity, Maternal education, Maternal age, Birth order and Gestational weeks. Fixed Effects include month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table B17: The interaction between pm2.5(orange) and mother's race by trimesters.

	Birth weight All (1)	Birth weight Boys (2)	Birth weight Girls (3)	Birth weight All (4)	Birth weight Boys (5)	Birth weight Girls (6)
T1 orange	-3.42611***	-3.53705**	-3.42295**	-2.67287**	-2.79937**	-2.51635
	(1.07522)	(1.53008)	(1.40441)	(1.08485)	(1.05781)	(1.68576)
T2 orange	-1.64644*	-.63373	-2.60277**	-1.43283	-.76294	-2.13725***
	(.84939)	(.97586)	(1.06331)	(.85836)	(1.22209)	(.75215)
T3 orange	-1.04626	-.37474	-1.41343	-.44756	.13786	-1.04371
	(.82018)	(1.25782)	(1.00314)	(.7727)	(.92652)	(.99579)
1bn.ethinic hispanic						
ethinic others	-39.57285***	-35.58957***	-44.45998***	-49.67801***	-47.54261***	-51.97607***
	(6.74724)	(6.65942)	(7.01702)	(7.33416)	(7.15915)	(7.73985)
Interactions						
1bn. hispanic x T1 orange						
2. others x T1 orange	2.85545*	2.46019	2.99659*	1.30466	.86966	1.71835
	(1.5487)	(1.63727)	(1.5512)	(1.6216)	(1.69418)	(1.71288)
1bn. hispanic x T2 orange						
2. others x T2 orange	.97041	.71421	.83236	-.37031	-.71167	-.05887
	(1.33839)	(1.49221)	(1.31094)	(1.33287)	(1.50899)	(1.3124)
1bn. hispanic x T3 orange						
2. others x T3 orange	.57039	1.58835	-.48068	.28784	.91652	-.33766
	(2.06629)	(2.81278)	(1.44298)	(1.40851)	(1.89951)	(1.36013)
Observations	927231	472192	455039	927231	472192	455039
All Controls				✓	✓	✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. the column headings represent the dependent variable. All controls include the Marital status of the mother, Child sex, Maternal ethnicity, Maternal education, Maternal age, Birth order and Gestational weeks. Fixed Effects include month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

Table B18: The interaction between pm2.5(red) and mother's education by trimesters.

	Birth weight All (1)	Birth weight Boys (2)	Birth weight Girls (3)	Birth weight All (4)	Birth weight Boys (5)	Birth weight Girls (6)
T1 red	-1.40514	-2.28724	-1.00109	-2.22374	-4.15676	-.24911
	(2.00125)	(2.55535)	(3.178)	(2.57613)	(2.97464)	(3.34614)
T2 red	-2.52952	.83301	-6.73364***	-2.01693	.61965	-4.87266***
	(1.95113)	(2.34809)	(2.23608)	(1.65604)	(2.36221)	(1.74315)
T3 red	-2.33542	-.4504	-4.36639	-1.26528	.1243	-2.73264
	(3.66366)	(3.98825)	(3.56857)	(3.44052)	(3.65605)	(3.76265)
1bn.edu>12yrs						
2.edu<12 yrs	-11.92593**	-16.39546***	-6.89024	-20.01449***	-24.32761***	-15.53446***
	(5.33771)	(5.48202)	(5.23503)	(2.28133)	(2.32696)	(2.45032)
Interactions						
1bn.edu>12 yrs x T1 red						
2.edu<12 yrs x T1 red	-1.92273	-.63969	-3.13082	-.23195	2.00399	-2.54326
	(2.45272)	2.88581)	(3.03411)	(2.34358)	(2.87405)	(2.56886)
1bn.edu>12 yrs x T2 red						
2.edu<12 yrs x T2 red	.56733	-.57639	2.59426	-.96626	-2.31618	.53815
	(2.15448)	(2.43806)	(2.25497)	(1.81995)	(2.17348)	(1.87708)
1bn.edu>12 yrs x T3 red						
2.edu<12 yrs x T3 red	.21976	-1.11746	2.1531	-.1382	-.67048	.42962
	(4.7151)	(4.75773)	(5.07143)	(3.95038)	(3.91809)	(4.62943)
Observations	927231	472192	455039	927231	472192	455039
All Controls				✓	✓	✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. the column headings represent the dependent variable. All controls include the Marital status of the mother, Child sex, Maternal ethnicity, Maternal education, Maternal age, Birth order and Gestational weeks. Fixed Effects include month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

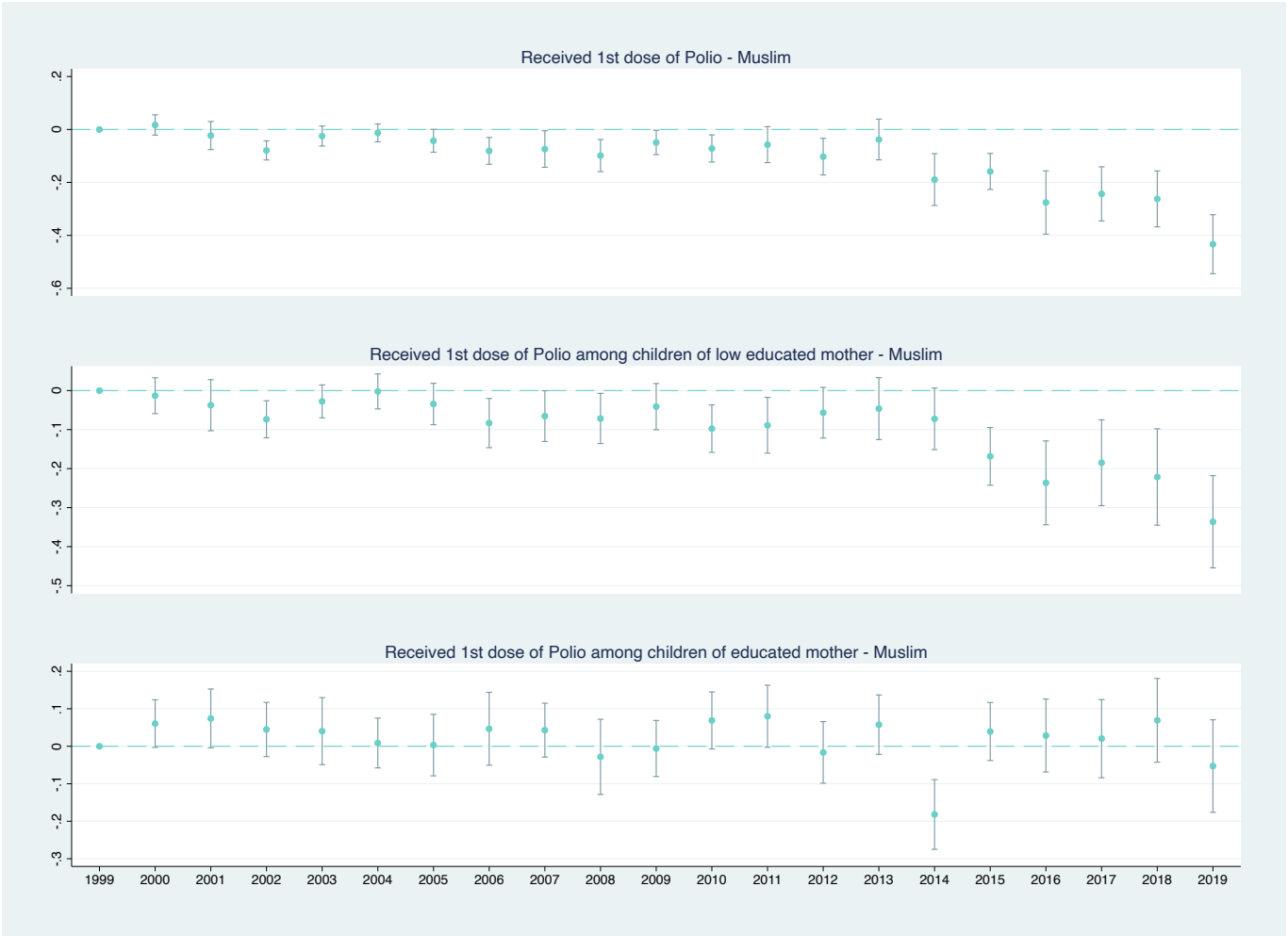
Table B19: The interaction between pm2.5(red) and mother's race by trimesters.

	Birth weight All (1)	Birth weight Boys (2)	Birth weight Girls (3)	Birth weight All (4)	Birth weight Boys (5)	Birth weight Girls (6)
T1 red	-4.47141**	-3.8635	-5.37421*	-3.3001	-3.16104	-3.42844
	(2.22965)	(2.40515)	(2.90114)	(2.38866)	(2.36278)	(3.13689)
T2 red	-3.47807**	-.87396	-6.01338***	-3.00121**	-.95258	-5.11903***
	(1.62005)	(2.21297)	(1.71637)	(1.47882)	(2.18342)	(1.45645)
T3 red	-2.99083**	-2.85841	-2.6831	-1.98762	-1.51484	-2.49236
	(1.41498)	(1.98206)	(2.06043)	(1.43055)	(1.8027)	(1.86846)
1bn.ethinic hispanic						
ethinic others	-39.4781***	-35.46949***	-44.39594***	-49.81985***	-47.64243***	-52.16113***
	(6.39609)	(6.28385)	(6.70976)	(7.14608)	(6.90051)	(7.61585)
Interactions						
1bn. hispanic x T1 red						
2. others x T1 red	4.92924*	2.99328	6.5239**	2.40012	.53488	4.26742
	(2.77333)	(3.00015)	(2.97227)	(2.50167)	(2.76992)	(2.69303)
1bn. hispanic x T2 red						
2. others x T2 red	3.2283	3.54124	1.99024	1.00211	.36785	1.54912
	(2.63692)	(3.1631)	(2.54131)	(2.6228)	(3.1655)	(2.76798)
1bn. hispanic x T3 red						
2. others x T3 red	2.15342	4.41944	-.63806	1.67794	3.17421	.08833
	(3.47946)	(4.45833)	(2.61638)	(2.50912)	(3.30801)	(2.36872)
Observations	927231	472192	455039	927231	472192	455039
All Controls				✓	✓	✓
City Fixed effects	✓	✓	✓	✓	✓	✓
Month-Year Fixed effects	✓	✓	✓	✓	✓	✓

Note: *** p<.01, ** p<.05, * p<.1. the column headings represent the dependent variable. All controls include the Marital status of the mother, Child sex, Maternal ethnicity, Maternal education, Maternal age, Birth order and Gestational weeks. Fixed Effects include month-year of birth and city fixed effects. Robust standard errors are clustered at the city level.

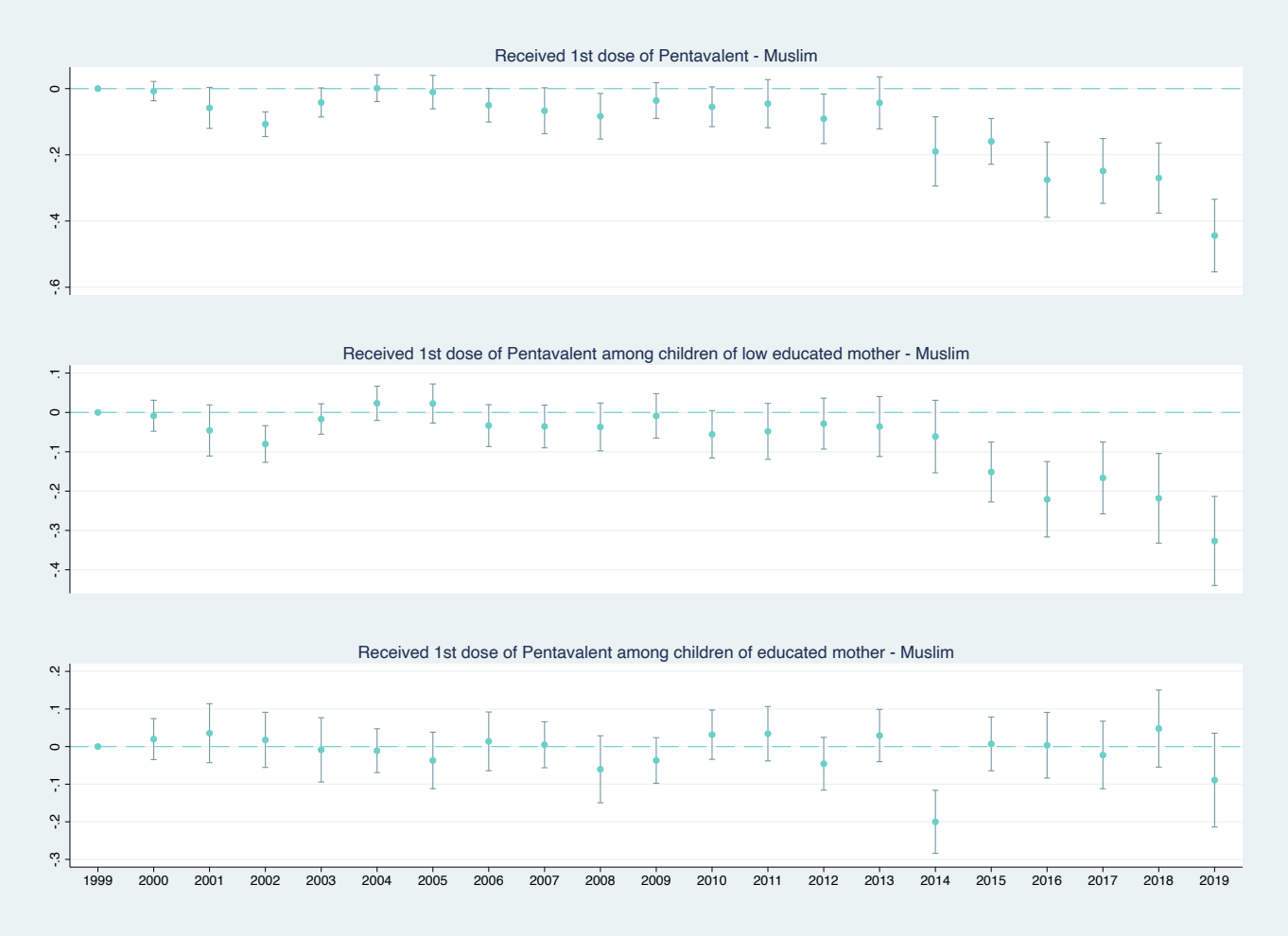
Appendix C: Appendix to Chapter 3

Figure C1: Polio - Mother's education



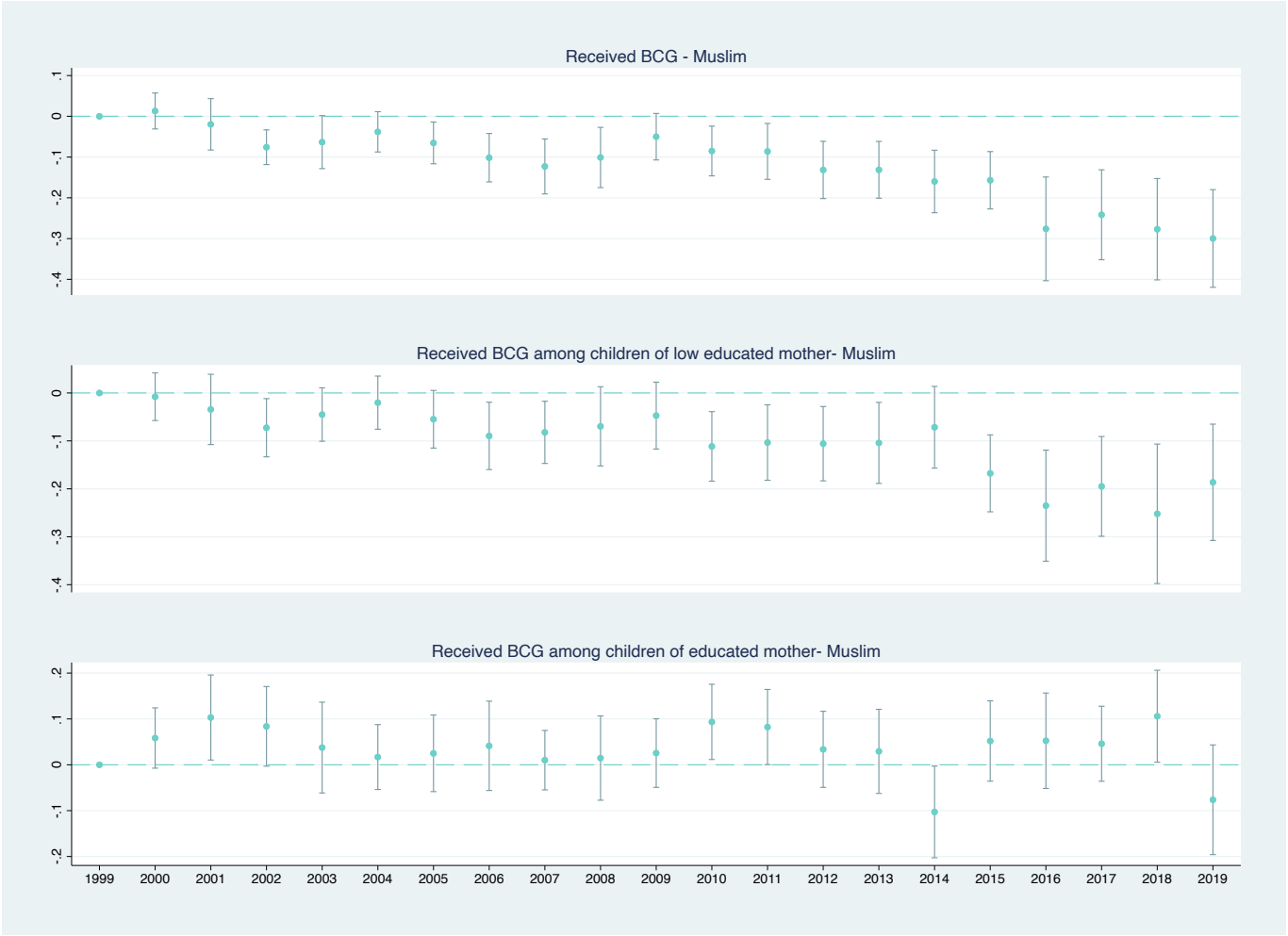
Notes: Year by year coefficients and 95% confidence interval of the event-study regressions.

Figure C2: Penta - Mother's education



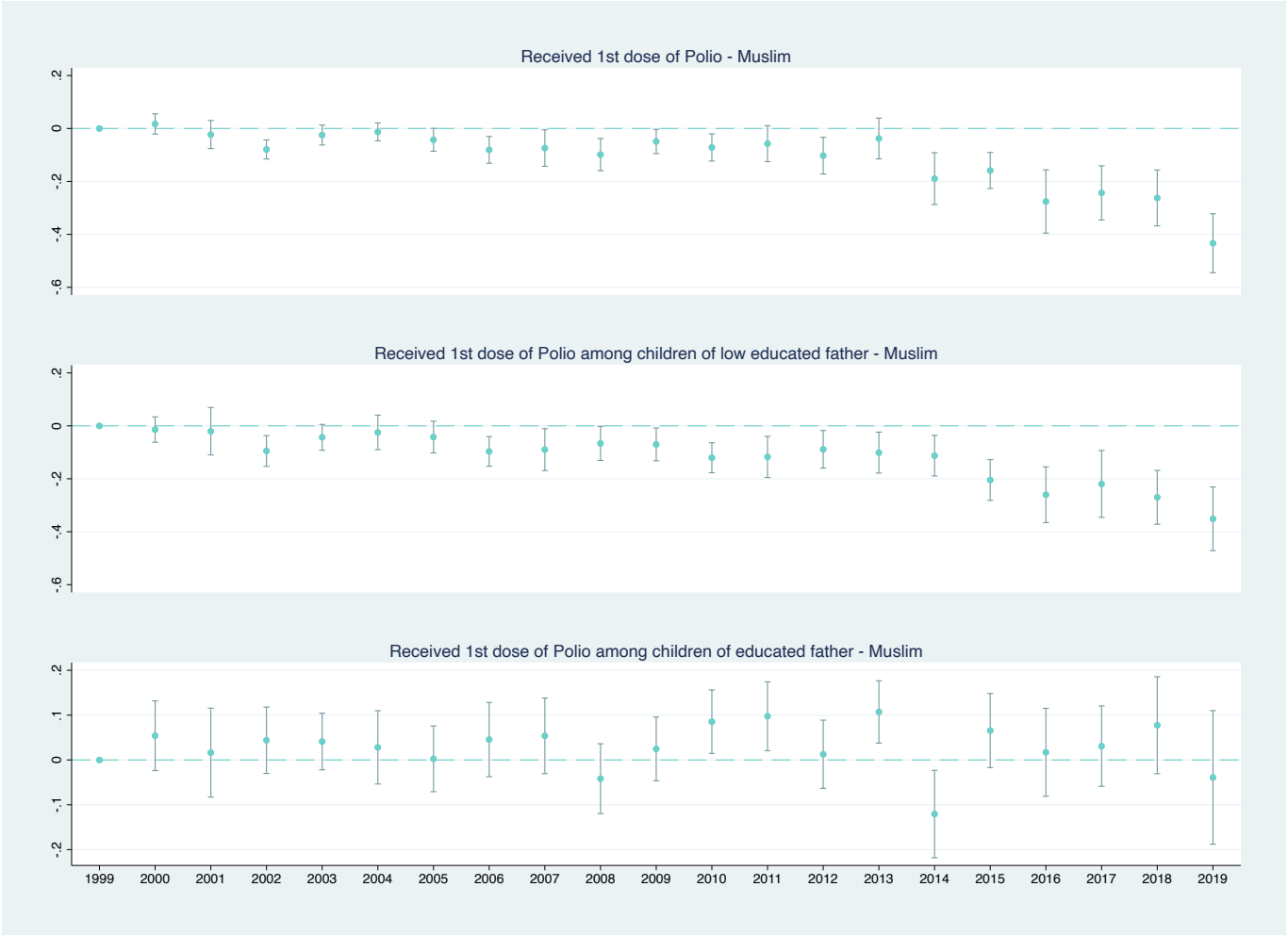
Notes: Year by year coefficients and 95% confidence interval of the event-study regressions.

Figure C3: BCG - Mother's education



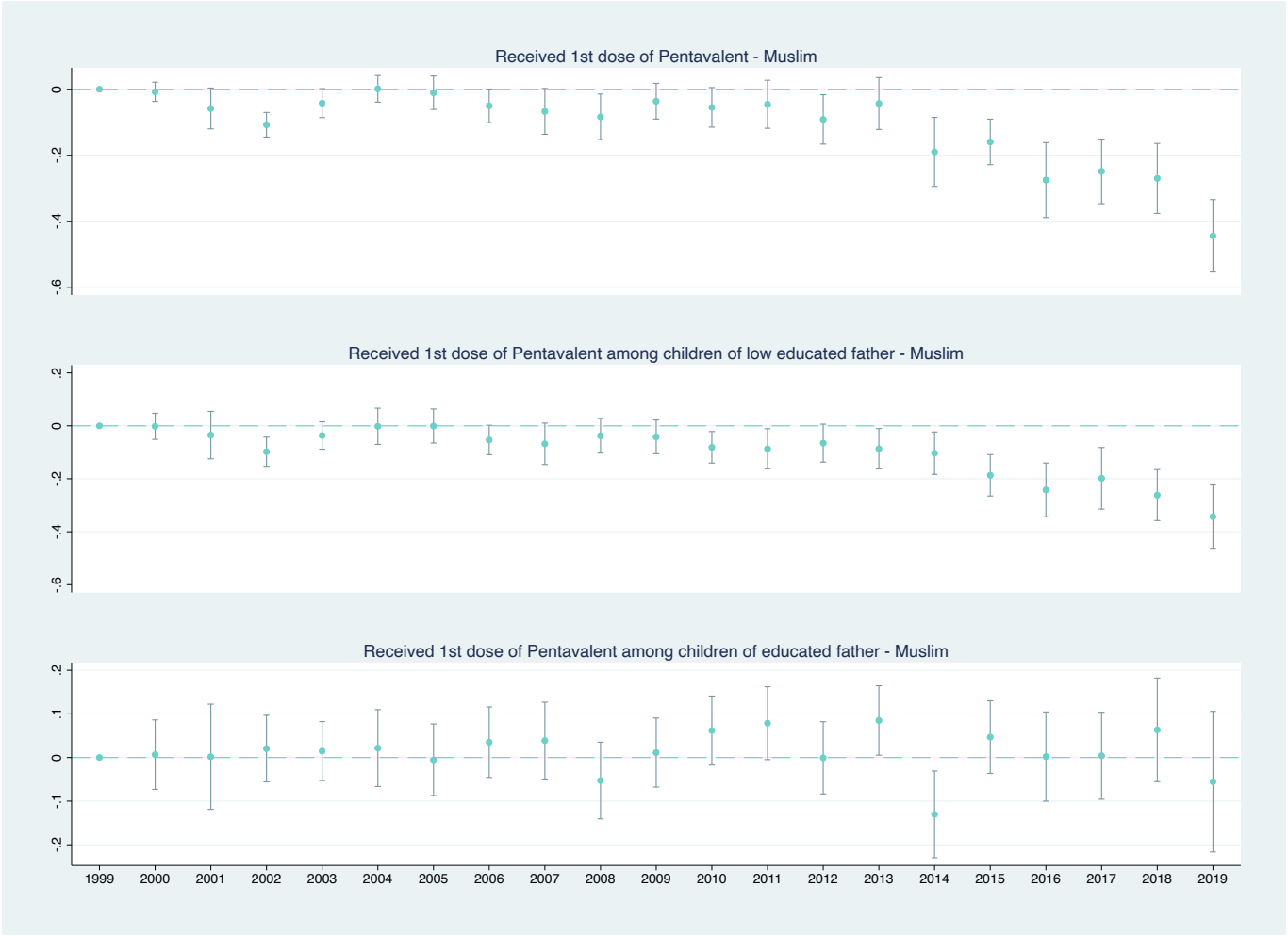
Notes: Year by year coefficients and 95% confidence interval of the event-study regressions.

Figure C4: Polio - Father's education



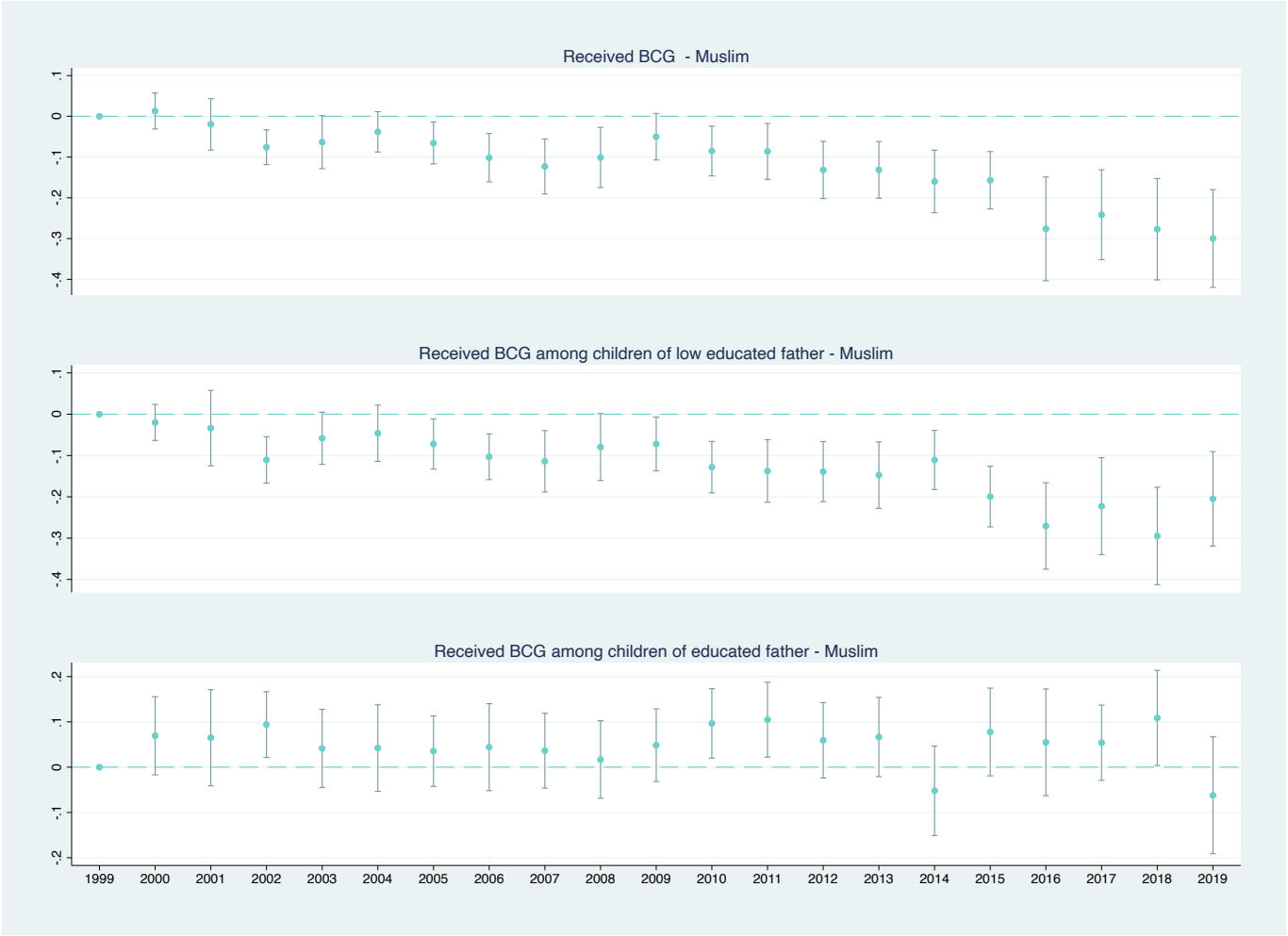
Notes: Year by year coefficients and 95% confidence interval of the event-study regressions.

Figure C5: Penta - Father's education



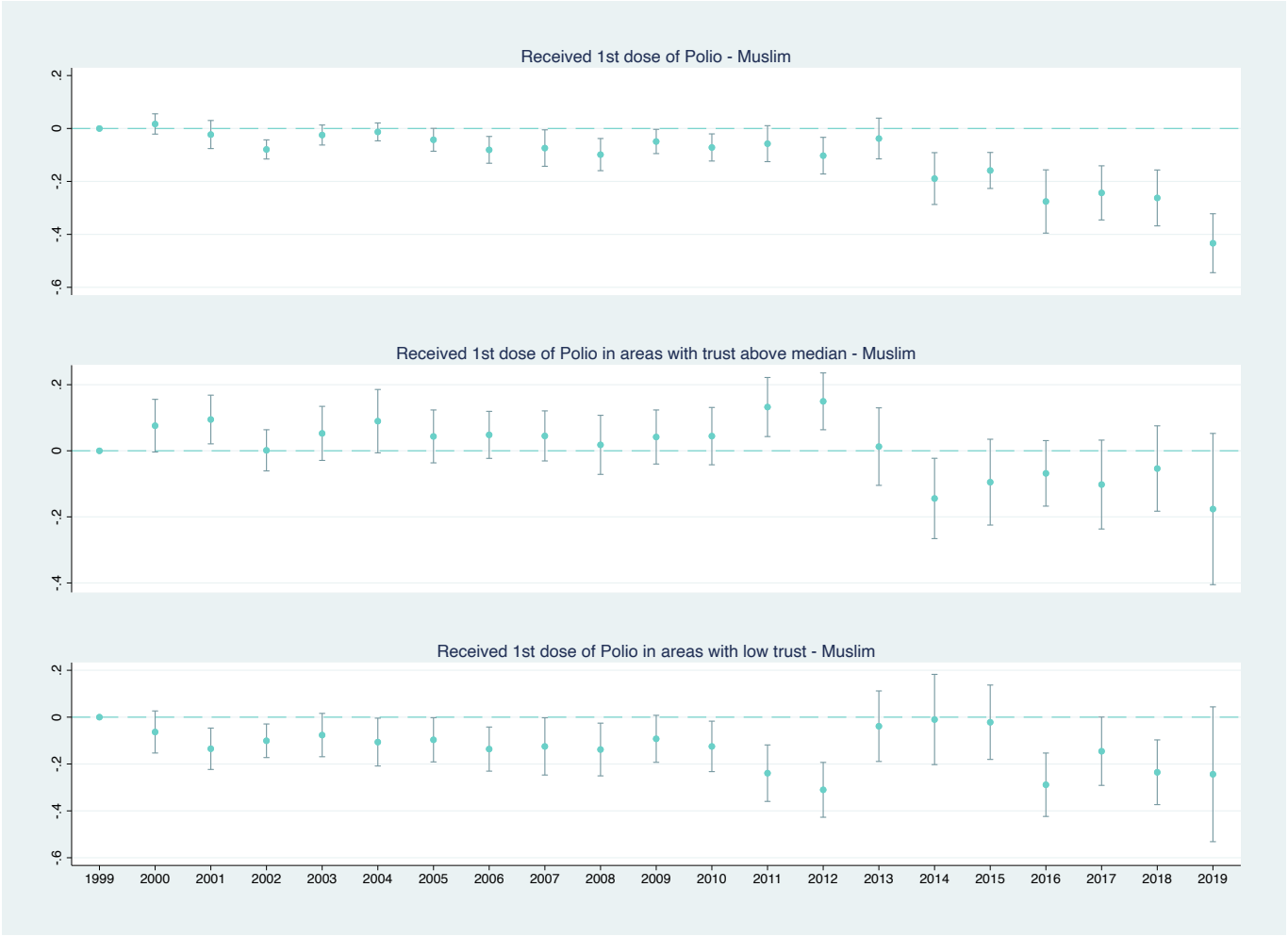
Notes: Year by year coefficients and 95% confidence interval of the event-study regressions.

Figure C6: BCG - Father's education



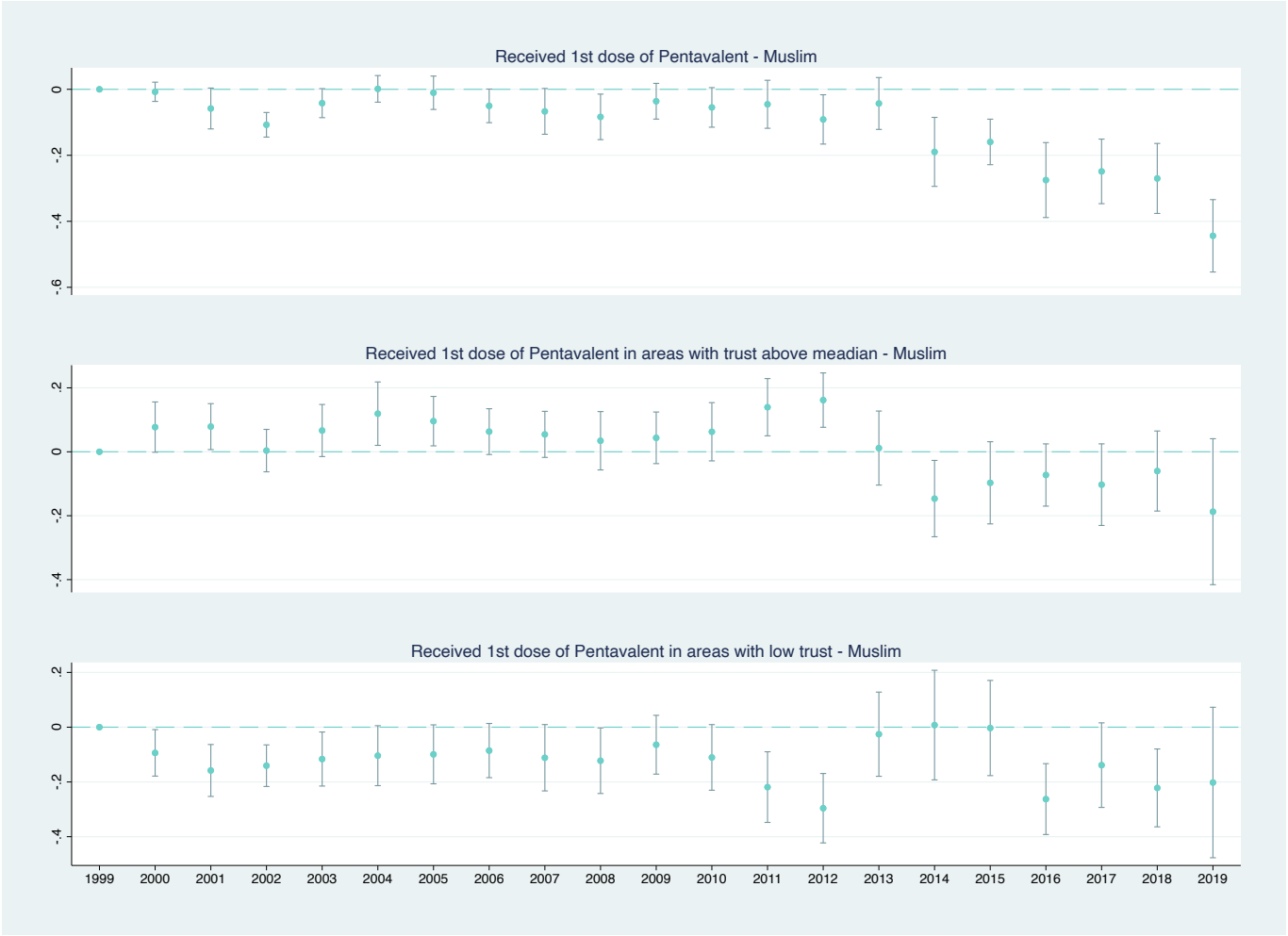
Notes: Year by year coefficients and 95% confidence interval of the event-study regressions.

Figure C7: Polio - Low trust



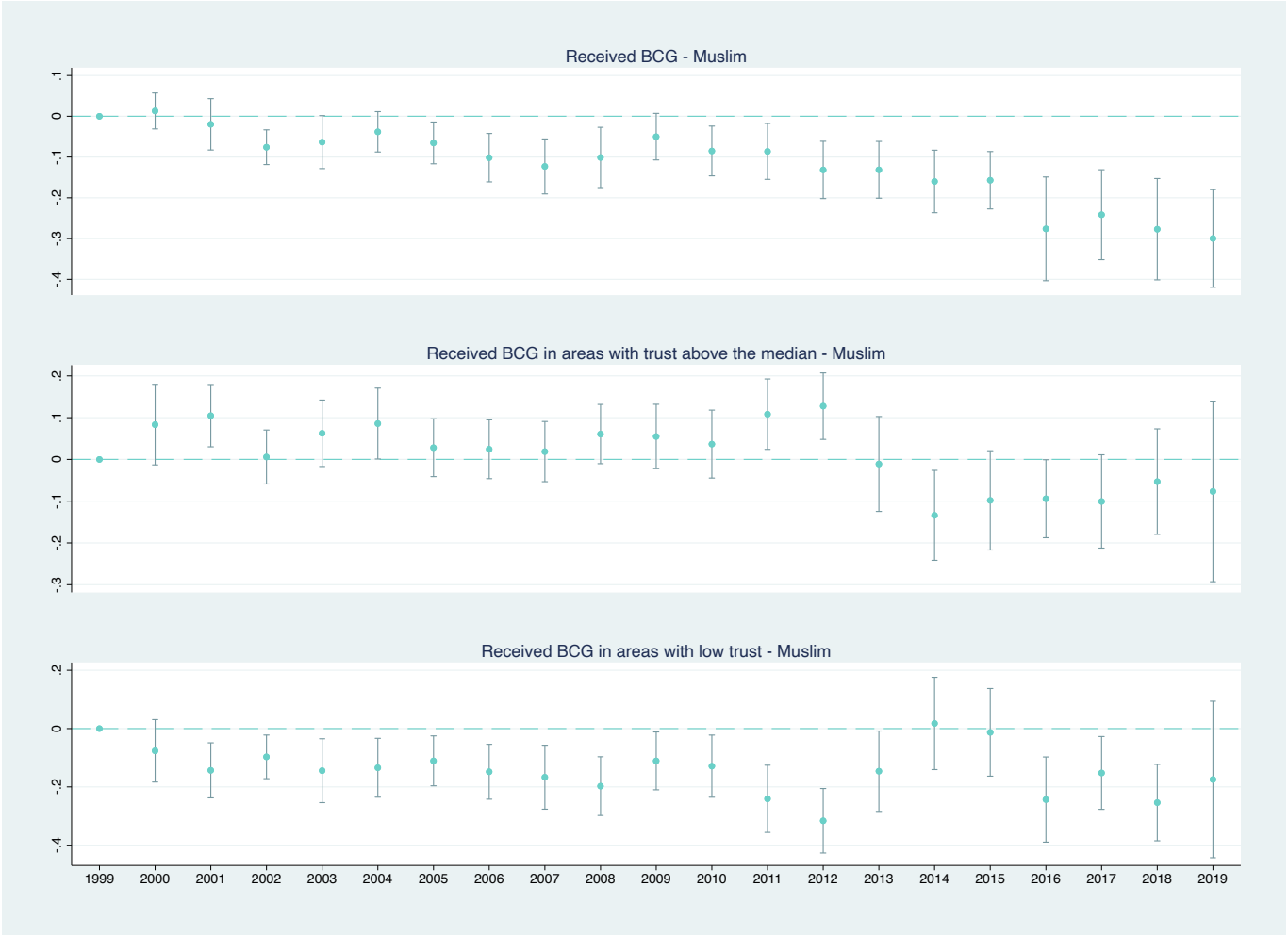
Notes: Year by year coefficients and 95% confidence interval of the event-study regressions.

Figure C8: Penta - Low trust



Notes: Year by year coefficients and 95% confidence interval of the event-study regressions.

Figure C9: BCG - Low trust



Notes: Year by year coefficients and 95% confidence interval of the event-study regressions.

Figure C10: DHS Cluster Map 1999 - 2019

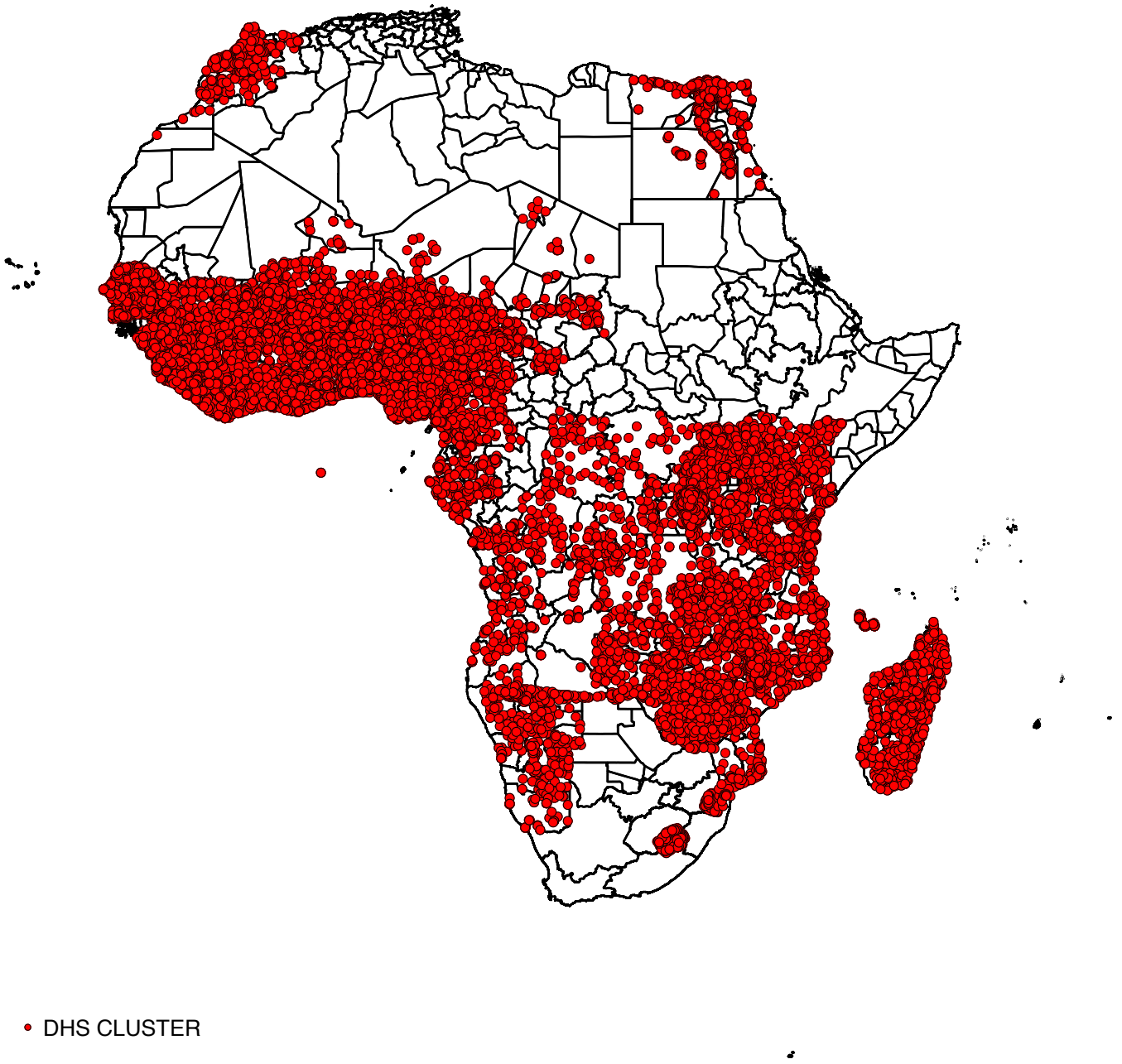
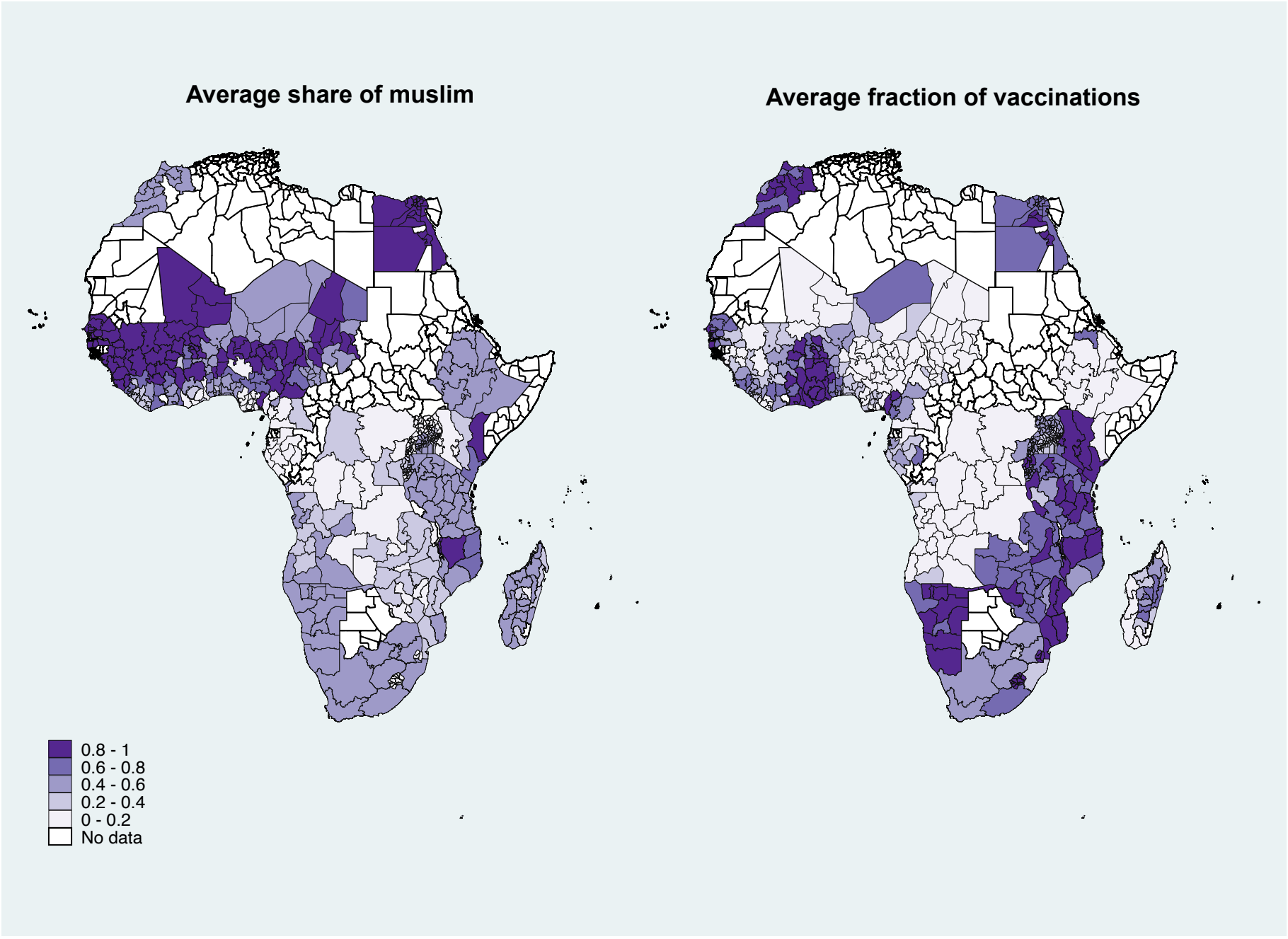


Figure C11: Average share of Muslim and average vaccination between 1999 and 2019



Notes: The dataset for shapefile boundaries for Africa is downloaded from ICPAC geoportal (ICPAC geoportal, 2019).

Table C1: Vaccination trends among Muslims(Figure 1)

Dependent Variable:	Fraction	Received all	Polio 1st	Penta 1st	BCG
	(1)	(2)	(3)	(4)	(5)
muslim	0.0492**	0.0209	0.0646***	0.0613***	0.0778***
	(0.0190)	(0.0233)	(0.0172)	(0.0218)	(0.0238)
2000 x muslim	0.00683	0.00847	0.0171	-0.00758	0.0131
	(0.0150)	(0.0173)	(0.0195)	(0.0148)	(0.0224)
2001 x muslim	-0.0291	-0.0195	-0.0229	-0.0582*	-0.0197
	(0.0298)	(0.0317)	(0.0270)	(0.0315)	(0.0320)
2002 x muslim	-0.0729***	-0.0470*	-0.0791***	-0.108***	-0.0759***
	(0.0192)	(0.0250)	(0.0184)	(0.0190)	(0.0216)
2003 x muslim	-0.00800	0.0302	-0.0247	-0.0420*	-0.0634*
	(0.0208)	(0.0279)	(0.0193)	(0.0224)	(0.0331)
2004 x muslim	0.00283	0.0212	-0.0129	0.00133	-0.0381
	(0.0201)	(0.0271)	(0.0172)	(0.0205)	(0.0252)
2005 x muslim	-0.0262	0.00218	-0.0427*	-0.0105	-0.0653**
	(0.0260)	(0.0333)	(0.0220)	(0.0257)	(0.0261)
2006 x muslim	-0.0606**	-0.0365	-0.0808***	-0.0502*	-0.102***
	(0.0254)	(0.0289)	(0.0258)	(0.0259)	(0.0302)
2007 x muslim	-0.0687**	-0.0650**	-0.0740**	-0.0669*	-0.123***
	(0.0339)	(0.0325)	(0.0350)	(0.0353)	(0.0343)
2008 x muslim	-0.0791**	-0.0408	-0.0986***	-0.0835**	-0.101***
	(0.0346)	(0.0406)	(0.0309)	(0.0351)	(0.0376)
2009 x muslim	-0.0338	-0.0156	-0.0491**	-0.0362	-0.0500*
	(0.0253)	(0.0293)	(0.0232)	(0.0275)	(0.0290)
2010 x muslim	-0.0556*	-0.0311	-0.0717***	-0.0549*	-0.0851***
	(0.0289)	(0.0327)	(0.0261)	(0.0305)	(0.0311)
2011 x muslim	-0.0342	-0.00505	-0.0574*	-0.0454	-0.0862**
	(0.0363)	(0.0369)	(0.0346)	(0.0370)	(0.0349)
2012 x muslim	-0.0807**	-0.0483	-0.103***	-0.0913**	-0.132***
	(0.0355)	(0.0354)	(0.0352)	(0.0380)	(0.0357)
2013 x muslim	-0.0278	-0.0129	-0.0379	-0.0430	-0.131***
	(0.0352)	(0.0316)	(0.0391)	(0.0400)	(0.0354)
2014 x muslim	-0.180***	-0.117**	-0.189***	-0.190***	-0.160***
	(0.0536)	(0.0491)	(0.0498)	(0.0532)	(0.0390)
2015 x muslim	-0.144***	-0.0917***	-0.159***	-0.160***	-0.157***
	(0.0354)	(0.0310)	(0.0346)	(0.0351)	(0.0358)
2016 x muslim	-0.259***	-0.205***	-0.276***	-0.275***	-0.276***
	(0.0594)	(0.0505)	(0.0608)	(0.0578)	(0.0647)
2017 x muslim	-0.233***	-0.192***	-0.243***	-0.249***	-0.242***
	(0.0530)	(0.0492)	(0.0520)	(0.0498)	(0.0561)
2018 x muslim	-0.238***	-0.175***	-0.262***	-0.270***	-0.277***
	(0.0512)	(0.0401)	(0.0537)	(0.0540)	(0.0632)
2019 x muslim	-0.475***	-0.534***	-0.433***	-0.444***	-0.300***
	(0.0573)	(0.0565)	(0.0566)	(0.0558)	(0.0610)
Observations	377,175	377,175	377,175	377,175	377,175
R-squared	0.426	0.378	0.412	0.406	0.414
Full set of controls	✓	✓	✓	✓	✓
Month-Year Birth FE	✓	✓	✓	✓	✓
Cluster FE	✓	✓	✓	✓	✓
Survey & age at survey FE	✓	✓	✓	✓	✓

Notes: Standard errors clustered at the ADMI1 level. *** p<0.01, ** p<0.05, * p<0.1. Controls include: child's age, female child dummy, and birth order

Table C2: Vaccination trends among Catholics (Figure 2)

Dependent Variable:	Fraction	Received all	Polio 1st	Penta 1st	BCG
	(1)	(2)	(3)	(4)	(5)
catholic	-0.0266	-0.0370	-0.0356	-0.0194	-0.0150
	(0.0336)	(0.0335)	(0.0394)	(0.0333)	(0.0413)
2000 x catholic	0.0278	0.0266	0.0187	0.0303	0.0360
	(0.0424)	(0.0400)	(0.0467)	(0.0477)	(0.0476)
2001 x catholic	-0.00520	0.0322	0.00448	-0.0242	-0.0454
	(0.0316)	(0.0298)	(0.0324)	(0.0378)	(0.0395)
2002 x catholic	0.0502	0.0753	0.0567	0.0544	0.0150
	(0.0458)	(0.0502)	(0.0545)	(0.0427)	(0.0510)
2003 x catholic	0.0430	0.0351	0.0650	0.0499	0.00648
	(0.0401)	(0.0401)	(0.0464)	(0.0362)	(0.0484)
2004 x catholic	0.0662	0.0747*	0.0815*	0.0587	0.0539
	(0.0414)	(0.0425)	(0.0473)	(0.0410)	(0.0481)
2005 x catholic	0.0721**	0.0779**	0.0902**	0.0591*	0.0514
	(0.0328)	(0.0356)	(0.0368)	(0.0355)	(0.0388)
2006 x catholic	0.0406	0.0711**	0.0363	0.0269	0.0162
	(0.0324)	(0.0301)	(0.0395)	(0.0356)	(0.0367)
2007 x catholic	0.0345	0.0377	0.0516	0.0318	0.0119
	(0.0347)	(0.0345)	(0.0406)	(0.0349)	(0.0432)
2008 x catholic	0.0500	0.0613*	0.0604	0.0428	0.0373
	(0.0385)	(0.0365)	(0.0481)	(0.0404)	(0.0493)
2009 x catholic	0.0389	0.0566	0.0386	0.0296	0.0183
	(0.0349)	(0.0350)	(0.0408)	(0.0350)	(0.0428)
2010 x catholic	0.0634*	0.0629*	0.0719*	0.0572	0.0432
	(0.0352)	(0.0351)	(0.0416)	(0.0355)	(0.0432)
2011 x catholic	0.0457	0.0520	0.0602	0.0386	0.0545
	(0.0367)	(0.0383)	(0.0424)	(0.0358)	(0.0427)
2012 x catholic	0.00844	0.0281	0.0135	-0.000812	-0.0126
	(0.0437)	(0.0398)	(0.0490)	(0.0439)	(0.0490)
2013 x catholic	0.0155	0.0328	0.0176	0.00660	0.00532
	(0.0379)	(0.0383)	(0.0433)	(0.0388)	(0.0461)
2014 x catholic	0.0602*	0.0644*	0.0675*	0.0560	0.0462
	(0.0362)	(0.0369)	(0.0396)	(0.0364)	(0.0425)
2015 x catholic	0.0521*	0.0778**	0.0513	0.0378	0.0174
	(0.0308)	(0.0315)	(0.0316)	(0.0337)	(0.0321)
2016 x catholic	0.0369	0.0473	0.0529	0.0341	0.0285
	(0.0389)	(0.0434)	(0.0439)	(0.0381)	(0.0430)
2017 x catholic	0.0393	0.0416	0.0472	0.0355	0.0293
	(0.0475)	(0.0548)	(0.0526)	(0.0422)	(0.0471)
2018 x catholic	0.0354	0.0191	0.0637	0.0518	0.0236
	(0.0451)	(0.0376)	(0.0579)	(0.0525)	(0.0603)
2019 x catholic	0.120**	0.144***	0.115*	0.104*	0.110*
	(0.0544)	(0.0556)	(0.0599)	(0.0551)	(0.0589)
Observations	180,420	180,420	180,420	180,420	180,420
R-squared	0.410	0.381	0.393	0.397	0.405
Full set of controls	✓	✓	✓	✓	✓
Month-Year Birth FE	✓	✓	✓	✓	✓
Cluster FE	✓	✓	✓	✓	✓
Survey & age at survey FE	✓	✓	✓	✓	✓

Notes: Standard errors clustered at the ADMI1 level. *** p<0.01, ** p<0.05, * p<0.1. Controls include: child's age, female child dummy, and birth order

Table C3: Vaccination trends among households of traditional religions (Figure 3)

Dependent Variable:	Fraction	Received all	Polio 1st	Penta 1st	BCG
	(1)	(2)	(3)	(4)	(5)
traditional	-0.0549	-0.0463	-0.0757*	-0.0339	-0.0526
	(0.0408)	(0.0386)	(0.0445)	(0.0439)	(0.0446)
2000 x traditional	0.0934**	0.0739*	0.141***	0.0766*	0.0955*
	(0.0451)	(0.0399)	(0.0528)	(0.0418)	(0.0518)
2001 x traditional	0.0296	0.0505	0.0522	-0.0213	0.00386
	(0.0886)	(0.0931)	(0.0874)	(0.0958)	(0.0939)
2002 x traditional	0.0233	-0.00376	0.0721	-0.0170	0.0540
	(0.0449)	(0.0549)	(0.0457)	(0.0519)	(0.0436)
2003 x traditional	4.58e-05	0.0191	0.0231	-0.0397	-0.0447
	(0.0513)	(0.0578)	(0.0495)	(0.0544)	(0.0550)
2004 x traditional	0.0282	0.0319	0.0165	-0.00661	0.0457
	(0.0558)	(0.0594)	(0.0621)	(0.0688)	(0.0540)
2005 x traditional	0.0829*	0.0891*	0.0992**	0.0752	0.0878**
	(0.0439)	(0.0468)	(0.0468)	(0.0488)	(0.0437)
2006 x traditional	0.0469	0.0476	0.0534	0.0222	0.0559
	(0.0492)	(0.0541)	(0.0506)	(0.0493)	(0.0484)
2007 x traditional	0.0370	0.00743	0.0555	0.0332	0.0408
	(0.0364)	(0.0381)	(0.0404)	(0.0425)	(0.0404)
2008 x traditional	0.0211	0.0295	0.0376	0.0114	0.00808
	(0.0420)	(0.0442)	(0.0467)	(0.0454)	(0.0473)
2009 x traditional	0.0532	0.0375	0.0799	0.0417	0.0631
	(0.0482)	(0.0475)	(0.0527)	(0.0518)	(0.0539)
2010 x traditional	0.0558	0.0426	0.0882*	0.0425	0.0840*
	(0.0403)	(0.0409)	(0.0483)	(0.0447)	(0.0453)
2011 x traditional	0.0690	0.0742	0.0843	0.0344	0.0438
	(0.0620)	(0.0492)	(0.0726)	(0.0636)	(0.0611)
2012 x traditional	0.0438	0.0350	0.0554	0.0453	0.0678
	(0.0588)	(0.0559)	(0.0640)	(0.0684)	(0.0717)
2013 x traditional	0.0644	0.0685	0.0648	0.0258	0.0132
	(0.0486)	(0.0494)	(0.0542)	(0.0542)	(0.0597)
2014 x traditional	0.00227	0.00661	0.0146	-0.0233	-0.00805
	(0.0699)	(0.0746)	(0.0645)	(0.0651)	(0.0628)
2015 x traditional	0.166***	0.174***	0.172***	0.140**	0.174***
	(0.0584)	(0.0565)	(0.0630)	(0.0621)	(0.0598)
2016 x traditional	-0.0107	0.0144	-0.0183	-0.0472	-0.0454
	(0.0758)	(0.0763)	(0.0846)	(0.0803)	(0.0775)
2017 x traditional	0.00884	0.0282	0.0106	-0.0439	0.0225
	(0.0785)	(0.0757)	(0.0829)	(0.0762)	(0.0789)
2018 x traditional	0.0186	0.0355	-0.0121	-0.0530	-0.00371
	(0.0891)	(0.0832)	(0.101)	(0.0969)	(0.0759)
2019 x traditional	-0.108	-0.101	-0.0735	-0.136*	-0.0893
	(0.0728)	(0.0745)	(0.0775)	(0.0811)	(0.0765)
Observations	180,420	180,420	180,420	180,420	180,420
R-squared	0.410	0.381	0.392	0.397	0.405
Full set of controls	✓	✓	✓	✓	✓
Month-Year Birth FE	✓	✓	✓	✓	✓
Cluster FE	✓	✓	✓	✓	✓
Survey & age at survey FE	✓	✓	✓	✓	✓

Notes: Standard errors clustered at the ADMI1 level. *** p<0.01, ** p<0.05, * p<0.1. Controls include: child's age, female child dummy, and birth order

Table C4: Vaccination trends among households with no religion (Figure 4)

Dependent Variable:	Fraction	Received all	Polio 1st	Penta 1st	BCG
	(1)	(2)	(3)	(4)	(5)
noreligion	-0.0499	-0.0249	-0.0737*	-0.0584	-0.0653
	(0.0401)	(0.0462)	(0.0419)	(0.0445)	(0.0501)
2000 x noreligion	0.0263	0.0108	0.0374	0.0237	0.0600
	(0.0489)	(0.0440)	(0.0558)	(0.0520)	(0.0579)
2001 x noreligion	0.112*	0.107*	0.146**	0.0929	0.115*
	(0.0569)	(0.0592)	(0.0626)	(0.0622)	(0.0608)
2002 x noreligion	-0.0368	-0.0842	0.00954	-0.0474	0.0292
	(0.0494)	(0.0528)	(0.0550)	(0.0572)	(0.0625)
2003 x noreligion	0.0306	0.00436	0.0407	0.0307	0.0457
	(0.0452)	(0.0568)	(0.0559)	(0.0605)	(0.0488)
2004 x noreligion	0.0519	0.0333	0.0634	0.0740	0.0242
	(0.0528)	(0.0609)	(0.0521)	(0.0569)	(0.0603)
2005 x noreligion	0.0661	0.0407	0.0777	0.0925	0.0903
	(0.0672)	(0.0743)	(0.0688)	(0.0718)	(0.0631)
2006 x noreligion	0.0578	0.0215	0.0720	0.0752	0.0591
	(0.0537)	(0.0592)	(0.0571)	(0.0578)	(0.0594)
2007 x noreligion	0.0540	0.0208	0.0791	0.0552	0.0806
	(0.0509)	(0.0564)	(0.0519)	(0.0580)	(0.0613)
2008 x noreligion	0.00628	-0.0133	0.0186	-0.00102	0.00297
	(0.0484)	(0.0574)	(0.0506)	(0.0550)	(0.0571)
2009 x noreligion	0.0393	0.000333	0.0564	0.0543	0.0402
	(0.0475)	(0.0529)	(0.0504)	(0.0528)	(0.0569)
2010 x noreligion	-0.0124	-0.0295	0.00335	-0.00743	-0.00171
	(0.0478)	(0.0500)	(0.0511)	(0.0544)	(0.0574)
2011 x noreligion	-0.0288	-0.0676	0.00501	-0.0154	0.0255
	(0.0604)	(0.0613)	(0.0619)	(0.0656)	(0.0618)
2012 x noreligion	-0.0320	-0.0536	-0.0121	-0.0283	0.0179
	(0.0527)	(0.0522)	(0.0580)	(0.0606)	(0.0601)
2013 x noreligion	0.0829	0.0326	0.126*	0.111*	0.124*
	(0.0610)	(0.0619)	(0.0656)	(0.0646)	(0.0737)
2014 x noreligion	-0.00959	-0.0311	0.0167	0.0118	0.00214
	(0.0501)	(0.0614)	(0.0499)	(0.0525)	(0.0557)
2015 x noreligion	0.0346	-0.00880	0.0499	0.0270	0.0718
	(0.0542)	(0.0574)	(0.0588)	(0.0593)	(0.0645)
2016 x noreligion	0.0670	0.0510	0.0829	0.0761	0.0887
	(0.0565)	(0.0639)	(0.0561)	(0.0573)	(0.0655)
2017 x noreligion	0.0220	-0.00323	0.0419	0.0293	0.0627
	(0.0519)	(0.0611)	(0.0540)	(0.0536)	(0.0605)
2018 x noreligion	0.0157	-0.0123	0.0499	0.0387	0.0269
	(0.0517)	(0.0573)	(0.0563)	(0.0577)	(0.0607)
2019 x noreligion	-0.162***	-0.202***	-0.132**	-0.138***	-0.155**
	(0.0486)	(0.0582)	(0.0518)	(0.0529)	(0.0601)
Observations	180,420	180,420	180,420	180,420	180,420
R-squared	0.410	0.381	0.393	0.397	0.405
Full set of controls	✓	✓	✓	✓	✓
Month-Year Birth FE	✓	✓	✓	✓	✓
Cluster FE	✓	✓	✓	✓	✓
Survey & age at survey FE	✓	✓	✓	✓	✓

Notes: Standard errors clustered at the ADMI1 level. *** p<0.01, ** p<0.05, * p<0.1. Controls include: child's age, female child dummy, and birth order

Table C5: Mother fixed effect

Dependent Variable:	Fraction (1)	Received all (2)	Polio 1st (3)	Penta 1st (4)	BCG (5)
Panel A: No controls					
Muslim x Hate Crimes (t-1)	-0.0170*	-0.0190**	-0.0133	-0.0252**	-0.0133*
	(0.00918)	(0.00955)	(0.0104)	(0.00975)	(0.00787)
Observations	561,004	561,004	561,004	561,004	561,004
Panel B: Full set of controls					
Muslim x Hate Crimes (t-1)	-0.0184*	-0.0183*	-0.0163	-0.0211*	-0.00849
	(0.0104)	(0.0106)	(0.0121)	(0.0118)	(0.00925)
Observations	282,355	282,355	282,355	282,355	282,355
Month-Year Birth FE	✓	✓	✓	✓	✓
Cluster FE	✓	✓	✓	✓	✓
Survey & age at survey FE	✓	✓	✓	✓	✓
Muslim specific trend	✓	✓	✓	✓	✓
Country specific trend	✓	✓	✓	✓	✓

Notes: Standard errors clustered at the ADMI1 level. *** p<0.01, ** p<0.05, * p<0.1. Controls include: child's age, female child dummy, and birth order

Table C6: Continuos variable - Muslim Proportion

Dependent Variable:	Fraction	Received all	Polio 1st	Penta 1st	BCG
	(1)	(2)	(3)	(4)	(5)
Panel A: No controls					
Muslim Proportion x Hate Crimes (t-1)	-0.0663***	-0.0522***	-0.0735***	-0.0825***	-0.0738***
	(0.0158)	(0.0144)	(0.0175)	(0.0153)	(0.0170)
Observations	697,820	697,820	697,820	697,820	697,820
Panel B: Full set of controls					
Muslim Proportion x Hate Crimes (t-1)	-0.0552***	-0.0430***	-0.0650***	-0.0694***	-0.0614***
	(0.0106)	(0.0116)	(0.0113)	(0.00999)	(0.00934)
Observations	377,449	377,449	377,449	377,449	377,449
Month-Year Birth FE	✓	✓	✓	✓	✓
Cluster FE	✓	✓	✓	✓	✓
Survey & age at survey FE	✓	✓	✓	✓	✓

Notes: Standard errors clustered at the ADMI1 level. *** p<0.01, ** p<0.05, * p<0.1. Controls include: child's age, female child dummy, birth order, mother's age at interview and at first birth, mother's and father's education, and wealth index.

Table C7: Excluding Nigeria

Dependent Variable:	Fraction	Received all	Polio 1st	Penta 1st	BCG
	(1)	(2)	(3)	(4)	(5)
Panel A: No controls					
Muslim	0.0605	0.0652*	0.0490	0.0473	0.0496
	(0.0388)	(0.0358)	(0.0435)	(0.0418)	(0.0437)
Muslim x Hate Crimes (t-1)	-0.0242**	-0.0247**	-0.0210**	-0.0400***	-0.0242***
	(0.0106)	(0.0108)	(0.0107)	(0.0109)	(0.00847)
Observations	607,520	607,520	607,520	607,520	607,520
Panel B: Full set of controls					
Muslim	0.0473	0.0644	-0.00109	0.0331	0.0186
	(0.0545)	(0.0566)	(0.0640)	(0.0616)	(0.0577)
Muslim x Hate Crimes (t-1)	-0.0371***	-0.0375***	-0.0358***	-0.0533***	-0.0370***
	(0.0121)	(0.0124)	(0.0123)	(0.0124)	(0.00937)
Observations	312,416	312,416	312,416	312,416	312,416
Month-Year Birth FE	✓	✓	✓	✓	✓
Cluster FE	✓	✓	✓	✓	✓
Survey & age at survey FE	✓	✓	✓	✓	✓
Muslim specific trend	✓	✓	✓	✓	✓
Country specific trend	✓	✓	✓	✓	✓

Notes: Nigeria is excluded from our sample. Standard errors clustered at the ADMI1 level. *** p<0.01, ** p<0.05, * p<0.1. Controls include: child's age, female child dummy, birth order, mother's age at interview and at first birth, mother's and father's education, and wealth index.

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