ABSTRACT

Title of Document: ESSAYS ON FEMALE EDUCATION, FERTILITY, AND HEALTH: EVIDENCE FROM TURKEY AND THE US

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Education is an important factor in reducing poverty, improving child health, and empowering women, which are key indicators of economic development and are supported by the United Nations Millennium Development Goals. While education is positively associated with these development indicators, identifying the extent that education has a causal impact in developing countries requires exogenous variation in education. Two dissertation chapters rely on a change in compulsory schooling law (CSL) in Turkey as a natural experiment to identify the causal impact of education.

In 1997, the Turkish government extended compulsory schooling from five to eight years, and in addition created new schools and classrooms, hired additional teachers, and provided transportation to rural areas. As a result, enrollment rates and education attainment significantly increased, especially for females. I use the change in the CSL as a natural experiment, using variation in the exposure to the CSL across cohorts as instruments to education.

In the second chapter, I find that mother's primary school completion improves infant health, as measured by very low birth weight, and child health, as measured by height-for-age and weight-for-age z-scores, even after controlling for many potential confounding factors. Exploring heterogeneous effects indicates that maternal education improves child health more in provinces with lower income and urbanization rates. I also demonstrate that primary school completion leads to earlier preventive care initiation and reduces smoking. Using parameters from various studies, I find that the benefits outweigh the cost of the CSL.

The third chapter demonstrates that an extra year of female schooling reduces teenage fertility by 0.03 births, which is a reduction of 33%. Exploring heterogeneous effects indicates that female education reduces teenage fertility more in provinces with higher initial fertility and lower population density. Finally, the CSL postpones childbearing by delaying marriage, thereby reducing fertility.

The fourth chapter explores the effect of teenage childbearing on health outcomes and behaviors of mothers using a nationally representative sample of twins from the Midlife Development in the US dataset. I employ within-family estimations using samples of siblings, twin pairs, and identical twin pairs, to overcome the bias generated by unobserved family background and genetic traits. The results suggest that teenage childbearing does not affect long-term health; however, it adversely affects exercise and preventive care behaviors. Further, I find that the effects of teenage childbearing may operate through education and the quality of the spouse.

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Dedication

This dissertation is dedicated to my parents, Ahmet Gunes and Yildiz Gunes, for their loving support. And to my brilliant and precious love, Dana Andersen, for his endless support, companionship, and encouragement.

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Table of Contents

Chapter 1: Introduction

Education is an important factor in reducing poverty, improving child health, and empowering women, which are key indicators of economic development and are supported by the United Nations Millennium Development Goals. While the role of female education in improving development indicators is well established, evidence on the causal impact in the context of developing countries is still not conclusive. Moreover, an understanding of the mechanisms through which education operates is crucial to designing public policies that promote these key development indicators.

In order to identify the causal effects, chapters two and three use a change in the compulsory schooling law (CSL) in Turkey in 1997, which extended compulsory schooling from five to eight years (free of charge in public schools) as a natural experiment. I therefore explore the impact of female education on child and infant health and teenage fertility in a country lacking female empowerment and facing very high teenage fertility and child mortality rates prior to the CSL. According to the World Economic Forum Global Gender Gap Report, the Middle East and North Africa (MENA) have the lowest regional ranking in comprehensive gender equality, highlighting the importance of understanding the role of education in reducing gaps in health and economic opportunities. In particular, Turkey is ranked far worse than countries previously studied and has a unique set of social and cultural institutions that have historically disadvantaged women.

Similar to many developing countries, Turkey has experienced a rapid fertility decline since the early 1960s. In 1980, Turkey implemented export-oriented policies, which increased the demand for labor in the service and industrial sectors. As a result, migration from villages to cities increased, leading to rapid urbanization and industrialization, which had a profound effect on fertility. The total fertility rate (TFR) exceeded 6 children per woman in the early-1960s, dropped to 5 in the late-1970s, and dropped further to around 3 in the late-1980s.[1] In the 1990s, however, fertility remained at around 2.6 births per

[1] TFR is defined as the average number of children that would be born to a woman by the end of her childbearing period if she were to experience the exact current age-specific fertility rates.

woman.

Proximate determinants of fertility, particularly contraceptive use and marriage, are possible factors explaining the trend in fertility. Turkey's fertility decline started with a family planning program introduced in 1965, which legalized the sale and use of contraceptives. Moreover, a new Population Planning Law in 1983 legalized induced abortions upon request for up to ten weeks gestation and allowed trained nurses and midwives to administer IUDs, which increased the prevalence of modern contraceptives and as a result subsequently decreased induced abortions. During the 1990s, Turkey experienced a leveling off in contraceptive prevalence around 60%, which coincided with a period of slowed fertility decline.[2] The traditional marriage pattern of Turkey is characterized by the universality of marriage: almost all women engage in either civil or religious marriages by the end of their reproductive ages and childbearing out of wedlock is uncommon in Turkey.[3] Hence, age at first birth depends on marriage age, which in turn affects the overall fertility. Since the time interval between marriage and first birth has been stable with an average around 1.6 years in Turkey, a delay in age at first marriage may result in an overall fertility decline by postponing first births. The singulate mean age at marriage[4] for both sexes did not change (22 for females and 25 for males) over a period of 13 years from 1985 to 1998. Thus, a delay in age at first marriage can play a crucial role in reducing fertility.

It is useful to look at changes in socioeconomic indicators in order to understand potential causes of fertility trends, especially in the later stages of the transition.[5] The Gross Domestic Product (GDP) per capita almost doubled from 1980 to 1990; however, economic recessions were frequent during the 1990s (4 major crises took place in 1991, 1994, 1999, and 2001). Following the export-oriented reforms in the 1980s, the share of the population in urban areas rose from 44% in 1980 to 59% in 1990, but did not significantly increase

[2] 2008 Turkey Demographic and Health Survey: http://www.hips.hacettepe.edu.tr.

[3] http://www.hips.hacettepe.edu.tr.

[4] It is defined as the average number of years lived as single (never-married) for females or males before they get married for the first time among those who marry before age 50.

[5] For statistics related to socioeconomic indicators, http://www.turkstat.gov.tr.

during the 1990s (reaching 65% in early 2000). The labor force in the agricultural sector decreased from 60% in the 1980s to 47% in 1990 and to 45% in 1995. From 1990 to 1995, life expectancy at birth increased from 65 to 68 and from 61 to 64 years for females and males, respectively. Despite improvements in infant and children under five mortality rates in the 1980s, both exceeded 55 per thousand live births during the first half of the 1990s, which is very high compared to developed countries. Adult literacy rates (the proportion of the adult population aged 15+ which is literate) leveled off around 70 for females and 90 for males during the 1990s prior to the CSL. Hence, the CSL took place when many development indicators either leveled off or showed very little progress and infant and under-five mortality rates were high.

Women can access preventive health care in various health facilities provided by the Ministry of Health (MOH) in Turkey. Health houses and centers are the primary public maternal health care service providers in the villages, which are staffed with at least one midwife or a nurse, whereas other health care facilities such as hospitals are mostly in urban areas. Mother and Child Health and Family Planning Centers also provide health services for pregnant women throughout the country. Overall, public services are widely used sources for preventive care compared to the private sector, which provides services mostly in large urban areas.[6]

According to the demographic health surveys in 1993 and 1998, 38% and 33% of births were to mothers who did not use formal prenatal care; 25% and 27% of births were not assisted by medical professionals; and about 40% and 30% of births were not delivered at a health facility, respectively.[7] In both years, assistance of traditional birth attendants and/or relatives at the delivery is higher in rural areas, compared to urban areas. Moreover, there has been a marked difference in health care utilization by educational levels within the country in both years.[8] Hence, the use of public preventive health care services was at

[6]Turkish Demographic Health Surveys in collaboration with MOH: http://www.hips.hacettepe.edu.tr.

[7]http://www.hips.hacettepe.edu.tr.

[8]For instance, only 40% of the mothers who did not complete 5 years of education used preventive care services, whereas almost 75% of the mothers with 5 to 8 years of education and almost 93% of the mothers

suboptimal levels during the 1990s prior to the CSL, even though public preventive health care services were available throughout the country.

Prior to the 1997 educational reform in Turkey, all citizens were required to receive five years of compulsory education, which is provided free of charge in public schools. In order to increase the education level to universal standards, in 1997 the Turkish government extended compulsory schooling from five to eight years as of the 1997/1998 Academic Year.[9] The government created new schools, added new classes to the existing schools, recruited new primary school teachers, provided transportation to children who live far from main primary schools, and provided free textbooks and uniforms to low-income students.

While the Program aimed to extend educational opportunities to a greater share of the population, the qualitative components of the education system in general and the design of the curriculum in particular stayed the same. In an in-depth case study prepared for the World Bank on the implementation of the 1997 Basic Education Law, Dulger (2004) attests that the Primary Education Program maintained the 1968 national curriculum with minor changes and that, due to time constraints in implementation, the Ministry of National Education (MONE) primarily focused on capacity issues to accommodate new students. Moreover, a 2007 OECD educational report emphasizes that the 1997 educational program in Turkey lacked implementation of a new curricula in order to improve the quality of the education system.[10]

The CSL led to a significant increase in the enrollment rates between the 1997/98 and the 2000/01 Academic Years, by around 15%.[11] Rural enrollment in grade six for females increased substantially between the 1997/98 and 1999/00 Academic Years, roughly 162%.[12] In the first year of the change in the law, the net primary enrollment rate and the sex ratio increased by 10.4% and 3.4%, respectively.[13] The net primary enrollment rate for

with more than 8 years of schooling used prenatal care services in 1993.

[9]The Basic Education Law No 4306.

[10]Reviews of National Policies for Education: Basic Education in Turkey: http://www.oecd.org.

[11]For educational statistics, see http://sgb.meb.gov.tr.

[12]http://www.unicef.org/turkey/gr/ge21ja.html.

[13]The net primary enrollment rate is calculated by dividing the number of students of a theoretical age

boys and girls has converged over time, reaching to 98.77 for males and 98.56 for females (98.67 for both sexes) in the 2011/12 Academic Year.

How did the CSL Affect Education in Turkey?

The CSL increased education by requiring households to maintain enrollment through the eighth grade, reducing the cost of education, and potentially influencing social norms and perceived returns to education.

The 8-year Basic Education Program required that all children to stay in school to at least the eighth grade. Moreover, only students completing the 8th grade became eligible for receiving the Primary School Diploma. Therefore, parents and students were compelled to complete at least eight years of education in order to comply with the law and to obtain a primary school education diploma, which confer both market and non-market benefits.

While attending public school was free both before and after the CSL, the Program did significantly reduce various costs associated with attaining education. The cost reductions included in the 8-Year Basic Education Program were aimed at providing opportunities for all children to stay in school at least to the eighth grade. Thus, low-income students were provided free textbooks, school meals, and student uniforms. Moreover, transportation expenses to children living at least 2.5 km away from nearby village schools were covered under the Bussed Primary Education Scheme launched in the 1989/1990 Academic Year in order to improve access for children in rural areas, especially for those in poor families. In addition, regional boarding primary schools (YIBO) were established to provide primary education services to settlements having no schools. Therefore, the total costs of attending school were significantly reduced by improving access to schools and providing school supplies.

The CSL might have also influenced social norms and perceived returns to education. For example, many traditional/conservative families in Turkey have an aversion to sending

group enrolled in a specific level of education by the population in that age group. Sex Ratio is calculated by dividing the female gross enrollment ratio by the male gross enrollment ratio multiplied 100.

girls to school after they reach puberty. However, if school attendance is mandatory then families may no longer be concerned about violating norms for allowing girls to attend school. Moreover, if labor markets exhibit “signaling” then the cost of not earning a primary school diploma in terms of foregone wages might be higher if most individuals have diplomas. In the same vein, the social cost of not completing primary school might be higher when a large fraction of peers complete primary school. Finally, households might change their subjective beliefs about the returns to education in response to changes in legislation. For example, households might infer that the returns to education are higher if the government strongly encourages more education.

Part A. Maternal Education and Child Health

A1. Does Education Matter?

There are several explanations for the link between maternal education and child health.[14] One explanation is that greater maternal education translates into greater health care utilization, including formal prenatal visits. Another explanation is that better educated women have higher income and may “match” with better educated, and higher income, husbands (Behrman and Rosenzweig, 2002). Educated women also have greater knowledge of modern health care services and ability to communicate with health-care providers (Caldwell, 1979; Barrera, 1990). Moreover, education may affect smoking and other health behaviors during pregnancy (Currie and Moretti, 2003). Another channel is through greater female autonomy, which in turn influences health-related decisions and the allocation of resources within the household (Caldwell, 1979; Caldwell et al., 1983). Other possible explanations include greater knowledge about diseases and increased adoption of modern medical practices (Caldwell, 1979; Barrera, 1990).

Early studies look at the relationship between maternal education and child health. However, unobserved background characteristics and omitted confounding factors, such

[14] Several studies find positive effects of maternal education on child health: among others, in Philippines Barrera, 1990; in Jamaica Handa, 1999; in Morocco Glewwe, 1999.

as ability (Griliches, 1977) and discount rates (Fuchs, 1982), could influence both education and health in the same direction, resulting in spurious relationships. Previous studies find mixed results after controlling for omitted variables or maternal childhood environment during which health related skills and habits are acquired. For example, Behrman and Wolfe (1987) show that the effects of maternal education on child height disappear once unobserved family endowments are controlled for. On the other hand, Strauss (1990) finds that maternal education increases child height and weight in Cote d'Ivoire, even after controlling for family-fixed effects. Maternal education is also found to be an important determinant of child height in Brazil after controlling for maternal height as a proxy for family background (Thomas, Strauss, and Henrique, 1990).

There is also an open question in the literature as to whether accessible health care facilities are substitutes to the knowledge of diseases and modern treatment conferred from education. For example, Rosenzweig and Schultz (1982) find support for the substitution hypothesis in urban areas in Colombia, but not in rural areas. Also, Thomas, Strauss, and Henrique (1990) find that mother's access to information explains most of the effects of education on child height in Brazil. Turkey provides an interesting case study since the educational intervention occurred at a time when health care facilities were readily accessible.

A2. Conceptual Framework

This section develops a stylized model focusing on household's allocation of resources to market and non-market goods. This stylized model draws on the insights of Becker (1965, 1981) and Grossman (2006). The primary aim of the model is to demonstrate some of the channels in which education influences child health and to illustrate the bias associated with correlations between education and child health. To address the latter, I incorporate household "ability," which has a direct effect on child health and an indirect effect through education.

Households face a single resource or budget constraint, and derive utility from conventional consumption (market goods) and the quality or healthiness of children (non-market goods). That is

$$U = u(C, Q) \tag{1}$$

where C is market goods and Q is child health. I assume all of the usual properties of the utility function (concavity, continuity, and differentiability). Child health, in turn, is a function of "purchased" health inputs (medical care, calorie intake, time spend), education, and ability. That is,

$$Q = q(H, E, A) \tag{2}$$

where H is health inputs, E is education, and A is ability. The production function Q exhibits the properties of standard neoclassical production function (increasing, concave, continuous, and differentiable). I assume that education is observed by the researcher, whereas ability is not observed. Education, however, will depend on ability as, for example, higher ability might reduce the cost of attaining education or ability and education might be complements to production of market goods and hence wages. While the determination of education is highly complex, it is sufficient for the aim of the model to express education simply as a function of ability. In other words, I express education as a reduced-form function of ability, $E = e(A)$.

Households face a budget constraint for consumption of market goods and child-health inputs, which is determined by income:

$$I = p_c C + p_h H \tag{3}$$

where p_c and p_h are the relative prices of consumption and health inputs. Without loss of generality, we can normalize the price of the consumption good to unity, and denote $p_h = p$. I assume that income is a strictly increasing, reduced-form function of education $I = i(E)$, which implies that household income is also a function of ability.

Households maximize household utility in (1) subject to (2) and (3), and incorporating the reduced-form expressions between education and ability, and income and education. Given the usual properties of the utility function, the endogenous variables C and Q can be expressed in following reduced-form (demand) relationships

$$C = c(p, E, A) \tag{4}$$

$$H = h(p, E, A) \tag{5}$$

Expression (5) is the primary interest because it relates education and ability to child health according to the following expression:

$$Q = q(h(p, E, A), E, A) \tag{6}$$

The above expression elucidates that education influences child health both indirectly, by increasing income thereby relaxing the household's budget constraint, and directly as child health directly depends on education. As long as child health is a normal good, both the indirect and direct effects increase child health. That is,

$$\frac{dQ}{dE} = \frac{\partial q}{\partial h}\frac{\partial h}{\partial E} + \frac{\partial q}{\partial E} > 0 \tag{7}$$

The first term is the indirect effect of education through income and the equality holds whenever $\partial h / \partial E > 0$. That child health is a normal good is therefore a sufficient, but not necessary condition that education improves child health. Under the assumption that child health is an inferior good then there are confounding effects between the direct and indirect effects.

To gain insight into the bias associated with ascribing the correlation between education and child to the causal impact of education on child health, I take the total derivative of Q

with respect to A and rearrange terms. That is,

$$dQ = \left(\frac{\partial q}{\partial h}\frac{\partial h}{\partial E} + \frac{\partial q}{\partial E}\right) dE + \left(\frac{\partial q}{\partial h}\frac{\partial h}{\partial A} + \frac{\partial q}{\partial A}\right) dA \tag{8}$$

where the latter term represents the direct effect of ability on child health, whereas the former term represents the indirect effect of ability on child health through education. For simplicity, I assume that education is a linear function of ability, $E = \mu A$. Substituting ability for education in the above expression implies that

$$dQ = \left(\frac{\partial q}{\partial h}\frac{\partial h}{\partial E} + \frac{\partial q}{\partial E}\right)(dE + \mu dA) \tag{9}$$

The above expression demonstrates the biased relationship found between education and child health whenever omitting the impact of ability. The precise bias is given by expression $\left(\frac{\partial q}{\partial h}\frac{\partial h}{\partial E} + \frac{\partial q}{\partial E}\right)\mu dA$. Correlations between education and child health therefore overstate the effect of education on child health whenever $\left(\frac{\partial q}{\partial h}\frac{\partial h}{\partial E} + \frac{\partial q}{\partial E}\right)\mu dA > 0$. Therefore, the estimated effect would be biased upwards whenever child health is a normal good and education is increasing in ability and education is increasing in ability.

To gain insight into the bias associated with OLS estimation, consider the following linear specification

$$Q = \alpha p + \beta_1 E + \beta_2 A + \varepsilon \tag{10}$$

where ε is random variable, satisfying the standard assumptions of the classical linear regression model, and α and β_i are the model parameters. Let's replace the non-stochastic relationship between ability and education with the stochastic relationship such that $E = \mu A + \eta$. The researcher, however, does not observe ability and therefore estimates (10) without including ability. Applying the well-known result for the omitted variable bias implies that the

$$E[b_1|p,E,A] = \beta_1 + \frac{cov(E,A)}{var(E)}\beta_2 \tag{11}$$

The bias associated with the OLS estimate is therefore

$$\frac{cov(E,A)}{var(E)}\beta_2 = \frac{cov(E,A)}{var(E)}\left[\frac{\partial q}{\partial h}\frac{\partial h}{\partial A} + \frac{\partial q}{\partial A}\right] \tag{12}$$

Using that $cov(E,A) = cov(A,E) = \mu\sigma^A$ where $\sigma^A = var(A)$ implies that the above equation can be expressed as

$$\frac{cov(E,A)}{var(E)}\beta_2 = \mu\sigma^A\left[\frac{\partial q}{\partial h}\frac{\partial h}{\partial A} + \frac{\partial q}{\partial A}\right] \tag{13}$$

The above expression is analogous to the case where education is deterministic.

A3. Effects of Maternal Education on Child Health

Chapter two explores the causal relationship between maternal primary school completion (8+ years) and infant health at birth, as measured by very low birth weight, child health, as measured by height-for-age and weight-for-age z-scores (HAZ and WAZ), and maternal health, as measured by the length of health facility stay after delivery (indicates delivery complications). Furthermore, I examine various channels through which maternal education may affect child health: health care utilization, smoking behavior, type of occupation, and spouse's education and occupation. I control for many confounding factors by including individual- and community-level characteristics, and mother's province of birth fixed effects. The empirical analysis relies on a unique dataset generated by combining the 2008 Turkish Demographic and Health Survey (TDHS-2008), the National Education Statistics books by Ministry of National Education (MONE), and detailed education data from Turkish Statistics Institute (TurkStat).

The results of the second chapter demonstrate that the CSL had a substantial effect on primary school completion, supporting the use of a change in the compulsory schooling law as a natural experiment. Using variation in the exposure to the CSL across cohorts as an instrument, I find that primary school completion improves infant, child, and maternal health, even after controlling for many potential confounding factors. The results also provide evidence of the causal effect of mother's education on maternal health behaviors–primary school completion leads to earlier preventive care initiation and reduces smoking. However, maternal education does not significantly affect formal prenatal care and delivery, mother's type of occupation, and husband's education and occupation. Hence, the results suggest that maternal education affects child health in part through changing maternal health behavior. Exploring heterogeneous effects suggests that the effects of maternal education on child health depend on province characteristics and the sex of the child. Finally, benefit-cost analysis relying on estimates from previous studies relating height to wages in other developing countries and using the estimated impact of the CSL on HAZ suggests that the benefits in terms of greater earnings outweigh the cost of the CSL.

Previous studies find different effects of maternal education on HAZ and WAZ in the context of developing countries. In Jamaica, Handa (1999) find that an additional grade completed increases child height by 0.058 standard deviations. This implies that a child of a mother with 8 years of schooling is around 0.5 standard deviations taller than a child of a mother with no schooling. In Pakistan, Aslam and Kingdon (2012) find that an additional year of maternal schooling increases HAZ and WAZ by 0.038 and 0.030 standard deviations, respectively. These effects suggest that a child of a mother with 8 years of schooling is 0.3 standard deviations taller and 0.2 standard deviations heavier than a child of a mother with no schooling. In China, Chen and Li (2009) find that an additional years of maternal education increases HAZ of adopted children by 0.064 standard deviations, while it increases HAZ of own children by 0.091 standard deviations. These results suggest that an adopted (own) child of a mother with 8 years of schooling is 0.5 (0.7) standard deviations taller than an adopted (own) child of a mother with no schooling. In Brazil, Thomas, Strauss, and Henrique (1990) find that each additional year of maternal education increases child height by about 0.50 percent in rural areas, while it increases child height by around

0.28 percent in urban areas.

In Turkey, I find that a child of a mother with at least 8 years of schooling (primary school) is around 1.0 standard deviation taller and 0.7 standard deviations heavier than a child of a mother with less than 8 years of schooling. While the effects are slightly larger than previous studies, there are many factors that should be considered. First, to compare the effect of an additional year of schooling and the completion of primary school completion, we should consider the average change in the number of years of schooling among females that complete primary school and females that do not complete primary school. In Turkey, 22 percent of the mothers who did not complete primary school has no schooling, while 57 percent of the mothers who completed primary school has at least 11 years of schooling. Moreover, the mean education is 3 years of schooling for mothers who did not complete primary school, while it is 10 for mothers who completed primary school. Therefore, on average, the effects on HAZ might be due to at least 7 years of difference in maternal education. Second, because completing primary school might confer a number of benefits beyond the completion of an additional year of schooling, there might be non-linear relationship between maternal education and child health. Therefore, studies exploiting variation in schooling primarily above or below the primary school completion cutoff will likely understate the effect of schooling in general. Finally, as discussed in chapter two, the effects for those mothers whose educational attainment has been affected due to a change in the CSL may be much higher than for the population.

Part B. Female Education and Fertility

Chapter three explores the causal relationship between female education and fertility, using a change in the CSL in Turkey in a similar fashion as the second chapter. Fertility and education are key factors in advancing development and reducing poverty. The reduced fertility typical of demographic transitions has been shown to confer a number of benefits. Examples include benefits to the mother, such as improved health, greater investment in human capital, and greater labor supply; as well as benefits to the child, such as improved nutrition, greater investment in schooling, and various improved health outcomes. As a consequence, an array of policy interventions has been adopted with the aim of accelerating the demographic transition and, thus, bringing these benefits earlier than they would otherwise occur. More generally, investment in female education has been adopted as a development tool in many developing countries. However, a thorough understanding of the causal relationship between fertility and female education has lagged behind the enthusiasm for educational interventions as a tool to reduce fertility. The case of Turkey provides a unique setting because the educational intervention occurred at a later stage in the demographic transition when the pace of fertility decline had slowed and reductions in fertility are more difficult to achieve.

Economic theory provides several mechanisms through which education may influence fertility choices. One explanation is that education increases the returns to labor market participation, thereby increasing the opportunity cost of time-intensive activities (Becker, 1981; Schultz, 1981). As a result, women might substitute time-intensive activities, such as childbearing and child rearing, in order to devote more time to the labor market participation. Therefore, education might result in fewer children for women. Also, education may affect fertility preferences–for instance, more educated women may prefer fewer but healthier (higher quality) children (Becker and Lewis, 1973). Improvements in child health resulting from female education may also reduce child mortality, thereby, lowering fertility since fewer births are required to achieve the same family size (Lam and Duryea, 1999; Schultz, 1993). Education may reduce fertility through increased knowledge about contraceptives and the effective use of contraceptive methods (Rosenzweig and Schultz, 1985, 1989). Lastly, education may increase women's autonomy and bargaining power in the household, thereby, increasing women's participation in fertility decision-making (Mason, 1986). In addition, staying in school longer might postpone childbearing if having children impedes upon attending school.

This chapter employs a similar dataset and empirical methodology as the second chapter to identify the causal effect of female education on teenage fertility. More specifically, I explore the impact of female education on the number of children before the ages of 18, 17, and 16, using the exposure to the CSL across cohorts as instruments. In addition to employing instrumental variables estimation, this chapter also employs difference-in-differences using the CSL as a treatment variable.

This chapter demonstrates that an extra year of female schooling reduces teenage fertility by around 0.03 births, which is a reduction of 33%. Further, the results are robust with respect to a rich set of controls. The effect is higher than the corresponding effect estimated by ordinary least squares, which suggests about a 17% reduction in teenage births. This difference may be a consequence of education impacting fertility greater for a subsample of women whose educational attainment has been affected by the CSL. Exploring heterogeneous effects indicates that female education reduces teenage fertility more in provinces with higher initial fertility and lower population density. Moreover, the effect of the CSL on education depends on several characteristics of birth-province: initial levels of education, income, urbanization, and population density. I also find that the effect of the educational policy operates through a delay in marriage, which in turn postpones childbearing.

Early literature documents correlations between female education and fertility in developing countries, assuming that education is exogenous. For example, Ainsworth et al. (1996) find that an additional year of education reduces the number of ever-born children by 0.06 to 0.13 births in 13 out of 14 sub-Saharan African countries. Schultz (1998) finds that a one-year increase in female schooling reduces fertility by 13%, using cross-country panel data. Recent studies exploring the causal effect of female education on fertility in the context of developing countries find mixed results. Breierova and Duflo (2004) find that average parental education is an important determinant of very early fertility (before the age of 15) in Indonesia, whereas it is unimportant for early fertility (before the age of 25). Osili and Long (2008) find that an additional year of schooling reduces early fertility (before the age of 25) by 0.26 to 0.48 births in Nigeria, which is a reduction of 11 to 19%. Compared to previous studies, the results found herein are similar to the early literature and slightly larger than the recent literature examining causal effects.

Part C. Teenage Fertility and Maternal Health in the US

Chapter four explores the effect of teenage childbearing on health outcomes and behaviors of mothers by using a nationally representative sample of twins from the Midlife Development in the United States (MIDUS) dataset. Teenage childbearing is a public policy concern, especially in countries with high teen birth rates, such as the United States. Among the general public and policy makers, it is widely believed that teenage childbearing is a primary driver of poverty, leading to numerous adverse economic, educational, and health, consequences for mothers and their children. However, it is not clear how much of these adverse outcomes can be directly attributable to teenage childbearing.

In order to overcome the bias generated by unobserved family background and genetic traits, this chapter employs within-family estimations using samples consisting of siblings, twin pairs, and identical twin pairs. More specifically, I compare the long-term health and health behaviors of teenage mothers to that of their (twin) sisters who had their first child after their teens.

The results suggest that teenage childbearing does have a causal negative effect on some, but not most, health behaviors (exercise and preventive care behaviors), whereas it does not appear to impact the long-term health of mothers. Exploring heterogeneous effects suggests that younger-generation teenage mothers engage in less vigorous exercise and are less likely to use preventive care, and teenage mothers with less parental education use less preventive care. I also find that the effects of teenage childbearing on health behaviors may operate through education and the quality of the spouse.

There are numerous studies that use the MIDUS data for various research topics, such as aging, psychological well-being, physical health, health behaviors, cognitive functioning, the effects of education on health, and the genetic influences on health behaviors.[15] I will discuss a few recent rigorous studies that attempt to identify the causal effects of education and teenage childbearing on health and health behaviors using the MIDUS survey. Lleras-Muney and Cutler (2010) explore the effects of education on several health behaviors using the MIDUS survey to account for proxies for discount values, personality, socialization, and stress. They find that these proxies account for very little of the education gradient, and that education has significant effects on health behaviors, including smoking, drinking, BMI, preventive care use, and illegal drug use, among other health behaviors. Lundborg (2013) estimates the health returns to schooling using monozygotic twins from the MIDUS survey. The results suggest that completing high school improves health; however, additional schooling does not lead to additional health returns. Contrary to the previous literature (Lleras-Muney and Cutler, 2010), Lundborg (2013) does not find that schooling affects smoking and body weight.

Fletcher (2012) uses sister pairs and finds that teenage childbearing has negligible effects on health behaviors of mothers. The empirical results regarding the effects of teenage childbearing on health behaviors are consistent with Fletcher (2012). This paper contributes to the literature by exploring the effect of teenage childbearing on health outcomes of teen mothers. Moreover, this paper contributes to the small literature on the causal effects of teenage childbearing on health behaviors of mothers in three ways. First, this paper uses a more comprehensive set of health behaviors. Second, I control for possible omitted variables by including controls for early life factors, including birth weight and age at menarche. In addition, the data allow me to explore the effects for smaller samples of twins and siblings who are arguably more alike. For example, I check the robustness of the estimates by excluding twin pairs that reported large differences in the way they dressed and had different childhood playmates. Finally, this paper explores potential mechanisms through which teenage childbearing influences health behaviors in the context of US, including ed-

[15] See http://www.midus.wisc.edu for publications that use MIDUS data.

ucation, the number of children, marriage, and the spouse's educational attainment. Moreover, I address the problem of incorrect inference due to multiple outcome variables by re-estimating the effects on overall indices.

Part D. Policy Implications

This dissertation has important implications for public policy. Chapters two and three demonstrate that the benefits of female education and maternal primary school completion are greater than typically recognized as education improves not only market outcomes, but also infant and child health and reduces teenage fertility, which are linked to adverse outcomes for both mothers and their children. It also demonstrates that educational interventions, in particular compulsory schooling laws, can accelerate the demographic transitions at later stages. Moreover, interventions aimed at specific subpopulations are important for maximizing returns to educational interventions, as well as reducing disparities in education and infant and child health. The fourth chapter demonstrates that the causal effects of teenage childbearing are overstated by studies not accounting for unobservable genetic and background factors. However, I demonstrate that there is a significant effect for certain subpopulations, particularly minorities that might lack the financial resources to cope with the opportunity costs and real costs associated with teenage childbearing. Therefore, policymakers should devote greater resources towards prevention of teenage childbearing and assistance of subpopulations that are most affected by the adverse impacts of teenage childbearing.

Chapter 2: The Role of Maternal Education in Child Health: Evidence from a Compulsory Schooling Law

1 Introduction

Significant attention has been given to the relationship between parental education, especially maternal education, and child health.[1] While the problem of endogeneity has been known for several decades, only a few recent studies have investigated the causal relationship between parental education and child health. Overcoming the problem of endogeneity is crucial as unobservable confounding factors, such as ability (Griliches, 1977) and discount rates (Fuchs, 1982), could influence both education and health in the same direction, resulting in spurious relationships. This paper uses a nationwide reform of compulsory education system in Turkey as an instrument to identify the causal effect of maternal education on infant and child health. The change in the educational policy provides an ideal natural experiment since, among women for whom this policy was binding, it encouraged more education than would have otherwise been obtained. Exploiting the policy reform, this paper shows that maternal education improves infant and child health.

This paper contributes to the literature by exploring the causal relationship between maternal education and infant and child health in a developing country. In particular, this is the first paper to investigate the relationship between maternal education and child health in a developing country. This paper also adds to the literature by exploring numerous mechanisms in which education influences child health, including health care utilization, and investigating differential impacts of maternal education.

One explanation of the correlation between parental education and child health is that greater maternal education translates into greater health care utilization, including formal prenatal visits. The relationship between maternal education and health care services uti-

[1] For a survey of the literature, see Strauss and Thomas (1995).

lization, particularly in developing countries, holds even after controlling for factors that affect both maternal schooling and health care utilization, such as childhood place of residence and ethnicity, as well as socioeconomic variables, such as current residence and husband's education.[2]

Besides health care utilization, maternal education can affect child health through several other mechanisms. For example, better educated women have higher income and may "match" with better educated, and higher income, husbands (Behrman and Rosenzweig, 2002). Educated women also have greater knowledge of modern health care services and ability to communicate with health-care providers (Caldwell, 1979; Barrera, 1990). Moreover, education may affect smoking and other health behaviors during pregnancy (Currie and Moretti, 2003). Another channel is through greater female autonomy, which in turn influences health-related decisions and the allocation of resources within the household (Caldwell, 1979; Caldwell et al., 1983). Other possible explanations include greater knowledge about diseases and increased adoption of modern medical practices (Caldwell, 1979; Barrera, 1990).[3]

In order to identify the causal effect of maternal education, this paper employs an instrumental variables estimation (IV) using variation in the exposure to the compulsory schooling law (CSL) in Turkey in 1997, which extended compulsory schooling from five to eight years (free of charge in public schools) across cohorts as an instrument. More specifically, this paper explores the causal relationship between mother's primary school completion (8+ years) and infant health at birth, as measured by very low birth weight, child health, as measured by height-for-age and weight-for-age z-scores (HAZ and WAZ), and maternal health, as measured by the length of health facility stay after delivery (indicates delivery complications). Furthermore, I examine various channels through which maternal educa-

[2]In India Navaneetham and Dharmalingam, 2002; in Bangladesh Paul and Rumsey, 2002; in Ethiopia Mekonnen and Mekonnen, 2003; in Peru Elo, 1992; in Turkey Celik and Hotchkiss, 2000; in Thailand Raghupathy, 1996; in Uganda Tann et al., 2007; in rural Guetemala Glei et al., 2003; in South America Jewell, 2009; among others.

[3]Barrera (1990) and Caldwell (1979) show that educated mothers benefited more from health care services, regardless of access to health services.

tion may affect child health: health care utilization, smoking behavior, type of occupation, and spouse's education and occupation. I control for many confounding factors by including individual- and community-level characteristics, and mother's province of birth fixed effects.[4]

In the context of a developed country, a few studies have investigated the causal relationship between parental education and infant and child health using various approaches and find mixed results.[5] The literature exploring the causal effect of maternal education on infant health in developing countries is limited. Breierova and Duflo (2004) investigate the impact of parental education on a measure of child mortality (the total number of child deaths before various ages of the mother) using a primary school construction program in Indonesia as exogenous variation in schooling. Chou et al. (2010) explore the impact of parental education on infant health, as measured by the probability of low-weight (less than 2,500 grams) birth, neonatal death, postneonatal death, and infant death, using a middle school expansion in Taiwan, as exogenous variation. The former paper finds that parental education reduces child mortality, whereas the latter paper finds that parental education reduces the probability of low-weight birth and infant mortality. To date, no studies have investigated the impact of maternal education on child health, which is the primary contribution of this paper. Moreover, this paper is the first to explore the channels in which education operates and to explore heterogeneous effects.

[4]For example, Behrman and Wolfe (1987) argue that the effect of female education on health outcomes may be overstated in studies that do not control for women's childhood environment during which health related skills and habits are acquired.

[5]Lindeboom et al. (2009) use a policy influencing time of school exit in the UK, and find little evidence of a causal relationship between parental education and child and infant health as well as parental health and smoking behavior. McCrary and Royer (2011) use age-at-school-entry policies in California and Texas, and find that education has small effects on infant health (as measured by birth weight, prematurity, and rate of infant mortality), and does not affect prenatal behaviors (as measured by smoking rates and prenatal care). Currie and Moretti (2003) use availability of colleges by county when the mother is aged seventeen as an instrument for education. They find that higher maternal education improves infant health, and assess the importance of various channels through which education may improve birth outcomes in the United States. The CSL provides a more ideal instrument compared to college openings as a source of identification. Currie and Moretti (2003) addressed concerns regarding the validity of their instrumental variables: (1) the location of college openings may not be random and (2) the endogenous mobility of women who move to attend college.

Turkey provides an interesting case study because the policy intervention occurred at a time when preventive health care services were readily accessible. Rosenzweig and Schultz (1982) argue that accessible health care facilities are substitutes to knowledge of diseases and modern treatment conferred from education.[6] However, this paper provides causal evidence that educational interventions increasing maternal education can improve child health, even in a country where health care services are readily accessible. Part of the explanation for this finding is that while health care services are accessible to women, greater education promotes earlier preventive care initiation and influences other health behaviors.

This paper demonstrates that the CSL had a substantial effect on primary school completion. Using exposure to the CSL as an instrument, I find that primary school completion improves infant, child, and maternal health, even after controlling for many potential confounding factors. The results also provide evidence of the causal effect of mother's education on maternal health behaviors–primary school completion leads to earlier preventive care initiation and reduces smoking. However, maternal education does not significantly affect formal prenatal care and delivery, mother's type of occupation, and husband's education and occupation. Hence, the results suggest that maternal education affects child health in part through changing maternal health behavior. Exploring heterogeneous effects suggests that the effects of maternal education on child health depend on province characteristics and the sex of the child. Finally, benefit-cost analysis relying on estimates from previous studies relating height to wages in other developing countries and using the estimated impact of the CSL on HAZ suggests that the benefits in terms of greater earnings outweigh the cost of the CSL.

The policy implications of this paper are straightforward. The benefits of maternal primary school completion are greater than typically recognized as completion improves not only market outcomes as often emphasized but also child health, even in a country with

[6]They find support for their substitution hypothesis in urban areas in Colombia, but not in rural areas.

accessible health care services. This result highlights the importance of raising educational completion rates for both mothers and children. The results also suggest that region and urban/rural residence are important factors of infant health and health care utilization and, additionally, pregnancy experience and ethnicity are important factors of health care utilization. Therefore interventions aimed at these subpopulations are important for reducing infant health and health care utilization disparities.

The remainder of this paper is organized as follows: Section 2 provides background on child health, health care services, and the educational policy in Turkey; Section 3 describes the data; Section 4 presents the empirical strategy; Section 5 presents the results, robustness checks, and heterogeneous effects; and Section 6 concludes.

2 Background

2.1 Child Health and Maternal Health Care in Turkey Before the CSL

Women can access preventive health care in various health facilities provided by the Ministry of Health (MOH) in Turkey. Health houses and centers are the primary public maternal health care service providers in the villages, which are staffed with at least one midwife or a nurse, whereas other health care facilities such as hospitals are mostly in urban areas. Mother and Child Health and Family Planning Centers also provide health services for pregnant women throughout the country. Overall, public services are widely used sources for preventive care compared to private sector, which provides services mostly in large urban areas.[7]

According to the demographic health surveys in 1993 and 1998, 38% and 33% of births were to mothers who did not use formal prenatal care; 25% and 27% of births were not assisted by medical professionals; and about 40% and 30% of births were not delivered

[7] Turkish Demographic Health Surveys in collaboration with MOH: http://www.hips.hacettepe.edu.tr.

at a health facility, respectively.[8] In both years, assistance of traditional birth attendants and/or relatives at the delivery is higher in rural areas, compared to urban areas. Moreover, there has been a marked difference in health care utilization by educational levels within the country in both years.[9]

Infant and children under five mortality rates were extremely high in the early 1960s (over 200 per thousand live births); however, both decreased to approximately 130 in early 1980s. Despite improvements in infant and children under five mortality rates in the 1980s, both rates exceeded 55 per thousand live births during the 1990s prior to the CSL, which is very high compared to developed countries.[10] There were large regional and residential differentials in infant and children under five mortality, despite governmental efforts to provide access to preventive health care services. In addition, there were sharp differentials by mother's educational levels. In 1993 (1998), the infant and under-five mortality rates of children of mothers with fewer than five years of education were 1.6 (1.7) times higher than the rates of mothers with at least five years of education.[11] Adult literacy rates (the proportion of the adult population aged 15+ which is literate) leveled off around 70 for females and 90 for males during the 1990s prior to the CSL. Hence, the CSL took place when infant and under-five mortality rates were high, literacy rates leveled off, and the use of public preventive health care services were at suboptimal levels, even though public preventive health care services were available throughout the country.

2.2 The 1997 Educational Reform

Prior to the 1997 educational reform in Turkey, all citizens were required to receive five years of compulsory education, which is provided free of charge in public schools. In order

[8] Ibid.

[9] For instance, only 40% of the mothers who did not complete 5 years of education used preventive care services, whereas almost 75% of the mothers with 5 to 8 years of education and almost 93% of the mothers with more than 8 years of schooling used prenatal care services in 1993.

[10] http://data.un.org/

[11] For rates in 1993 and 1998, see http://www.measuredhs.com/pubs/pdf/FR108/FR108.pdf.

to increase the education level to universal standards, in 1997 the Turkish government extended compulsory schooling from five to eight years as of the 1997/1998 Academic Year.[12] The government created new schools, added new classes to the existing schools, recruited new primary school teachers, provided transportation to children who live far from main primary schools, and provided free textbooks and uniforms to low-income students.

While the Program aimed to extend educational opportunities to a greater share of the population, the qualitative components of the education system in general and the design of the curriculum in particular stayed the same. In an in-depth case study prepared for the World Bank on the implementation of the 1997 Basic Education Law, Dulger (2004) attests that the Primary Education Program maintained the 1968 national curriculum with minor changes and that, due to time constraints in implementation, the Ministry of National Education (MONE) primarily focused on capacity issues to accommodate new students. Moreover, a 2007 OECD educational report emphasizes that the 1997 educational program in Turkey lacked implementation of a new curricula in order to improve the quality of the education system.[13]

The CSL led to a significant increase in the enrollment rates between the 1997/98 and the 2000/01 Academic Years, by around 15%.[14] Rural enrollment in grade six for females increased substantially between the 1997/98 and 1999/00 Academic Years, roughly 162%.[15] In the first year of the change in the law, the net primary enrollment rate and the sex ratio increased by 10.4% and 3.4%, respectively.[16] The net primary enrollment rate for boys and girls has converged over time, reaching to 98.77 for males and 98.56 for females (98.67 for both sexes) in the 2011/12 Academic Year.

[12] The Basic Education Law No 4306.

[13] Reviews of National Policies for Education: Basic Education in Turkey: http://www.oecd.org.

[14] For educational statistics, see http://sgb.meb.gov.tr.

[15] http://www.unicef.org/turkey/gr/ge21ja.html.

[16] The net primary enrollment rate is calculated by dividing the number of students of a theoretical age group enrolled in a specific level of education by the population in that age group. Sex Ratio is calculated by dividing the female gross enrollment ratio by the male gross enrollment ratio multiplied 100.

3 Data and Measurement of Variables

The data used in this paper come from the most recent Turkish Demographic and Health Survey (TDHS-2008), a nationally representative household survey carried out in Turkey in 2008. The TDHS covers a representative sample of 7,405 ever-married women of reproductive ages 15-49. The survey has information on socioeconomic and demographic characteristics, fertility, and family planning, as well as maternal and child health. In addition, the survey contains information on maternal health care utilization of women who had given birth during the five years preceding the survey.

The analysis is based on the latest birth of ever-married women at the ages of 18-29 during the five years prior to the interview.[17] Restricting analysis to ever-married women is justified since childbearing out of wedlock is uncommon in Turkey.[18] Since women provide more accurate information on their most recent births, the analysis considers utilization behavior associated with only one birth (latest) per woman. Thus, the final data set is restricted to 1,677 mothers.

Measurement of Child and Maternal Health, and Maternal Health Care Services

Child anthropometric measurements, birth weight, and delivery complications, are used as measures of child, infant, and maternal health. This paper uses height-for-age and weight-for-age z-scores as measures of child health.[19] Height-for-age, given gender, is a commonly used measure of child health and an indicator of long-term health status, whereas weight-for-age, given gender, is a measure of current child health status and provides information on the current malnutrition status (Thomas et al., 1991). This paper focuses on "very low birth weight" (VLBW) (infants weighing less than 1500 grams) as a measure of infant health at birth, which is linked to higher risk of death within the first year of life

[17]The reason to restrict the sample to women at the ages of 18-29 is specified under the identification section.

[18]Hacettepe University Institute of Population Studies, TDHS-2008: http://www.hips.hacettepe.edu.tr.

[19]Anthropometric z-scores are calculated using the 2006 WHO child growth standards for children 0 to 5 years of age by their sex.

and numerous adverse adulthood outcomes.[20] In order to examine the effect of mother's education on maternal health, the length of health facility stay after delivery for the mother is used as an indicator of delivery complications (the unit is "days").[21]

Four dichotomous outcomes of use of maternal health care services are analyzed: use of prenatal services from formal sources, receipt of four or more prenatal visits, delivery in a medical institution, and delivery assistance by trained medical personnel. Following World Health Organization (WHO) definitions of appropriate maternal care, both prenatal care and assistance at delivery are coded 1 if the woman obtained services from doctors, trained nurses, or trained midwives, and 0 otherwise. I also use month of prenatal care initiation as a measure of preventive care, which takes values from 1 (preventive care is initiated in the first month of pregnancy) to 10 (mother has not initiated any prenatal care).

Independent Variables

The primary variable of interest–primary schooling completion (defined as "primary")–is an indicator variable equal to one if the mother has completed at least 8 years of schooling. Because women in the sample of analysis had completed their education prior to childbearing, the educational attainment observed in the sample represents final completed education.

There are several other factors that may affect child health as well as health utilization behavior. Province of birth dummies and ethnicity are included to control for childhood environment.[22] As an indicator of ethnicity, father's mother tongue categories are used: Turkish (reference category), Kurdish, and others.[23] I also control for prior pregnancy ex-

[20]Definition of VLBW is listed in the International Statistical Classification of Diseases and Related Health Problems (ICD-10) codes (World Health Organization). For adverse outcomes, see Hack et al., 1994; Hack et al., 2002; Behrman and Rosenzweig, 2004; Black et al., 2007.

[21]Although a noisy measure, the length of health facility stay after delivery is the best available proxy for maternal health in the TDHS data.

[22]There are 80 provinces included in the estimations based on the 1995 boundaries of Turkey. In all the estimations throughout the study, women born in Düzce are assumed to be born in Bolu since Düzce broke off Bolu and became a province in November 1999.

[23]Specifications are robust to using mother's mother tongue categories.

perience, which is measured as the number of previous ever-born births.[24] The number of previous births is likely to be endogenously determined by maternal education; however, excluding previous births did not substantially change the effects of maternal schooling on child health and maternal health care utilization, except for formal prenatal care (see Appendix A). I exclude father's education from all specifications since mother's and father's educations are highly correlated (Chou et al., 2010). Also, husband's education may be a channel through which mother's education affects outcomes if there is assortative mating, which in turn, may bias the relationship. I explore the possibility of assortative mating in the following sections.

I control for community-level factors, region and urban/rural residence at the time of the survey, in order to account for differential availability and quality of health care services, unobservable social and cultural factors as well as geographic characteristics.[25]

All specifications control for the primary school aged population and enrollment rates in the province of mother's birth in 1995 (prior to the CSL) provided by the Turkish Statistics Institute (TurkStat), each interacted with mother's years of birth dummies, in order to control for any time-varying factors that may be correlated with schooling and the allocation of schooling inputs to each province (Breierova and Duflo, 2004; Chou et al., 2010).

Table 1 presents descriptive statistics for the dependent and selected independent variables. Around 16% of the sample used in the analysis was exposed to the CSL, and around 30% completed 8 years of schooling. 7% of the women gave VLBW births, the average length of hospital stay was around 1.7 days, and average HAZ and WAZ of the child were -0.6 and 0.1. 89% of the women used formal prenatal care, 67% received at least 4 prenatal visits, and around 90% used formal delivery assistance and gave birth in a health facility.

[24]There are 42 mothers who experienced at least one stillbirth (5 of them were exposed to the CSL). The results are robust to excluding them.

[25]Geographic location at the time of the survey is most likely the place of the latest birth as well.

4 Identification Strategy

In order to account for the potential endogeneity of maternal education, this paper employs variation in the exposure to the CSL across cohorts induced by the timing of the policy as an instrumental variable. The identifying assumption is that exposure to the CSL had no direct effect on outcomes of interest other than via changing education levels. The first-stage model is:

$$S_i = \alpha_0 + \sum_{k=7}^{17} (YOB_{ik})\alpha_{1k} + \mathbf{P_i}\alpha_2 + \mathbf{K_i}\alpha_3 + \mathbf{X_i}\alpha_4 + \mathbf{C_i}\alpha_5 + u_i \tag{1}$$

where S_i indicates whether a mother i completed 8 years of compulsory primary schooling; $\mathbf{P_i}$ is a vector of mother's birth-province fixed effects; $\mathbf{K_i}$ is a vector of mother's birth-province-specific variables (interactions between the number of primary school aged children in the province of mother's birth (in 1995) and mother's year of birth dummies, and interactions between the enrollment rate in the province of mother's birth (in 1995) and mother's year of birth dummies); $\mathbf{X_i}$ is a vector of mother's background characteristics; and $\mathbf{C_i}$ is a vector of community-level factors. YOB_{ik} is an indicator variable denoting whether mother i is age k in 1997 (a year-of-birth dummy). Mothers aged 18 in 1997 is the omitted control dummy.

In equation 1, the year of birth dummies are used to capture exposure to the CSL in a flexible fashion. The CSL affected children who did not complete 5 years of education in the beginning of the 1997/98 Academic Year. Children aged 7-10 in 1997 (born in or after 1987) comprise the post-reform cohort that were exposed to the CSL, while children aged 11 or older in 1997 (born before 1987) comprise the pre-reform cohort that were unlikely to have been affected by the CSL.[26] Estimates of the equation 1 with/without controls

[26] The DHS provides exact date of birth for ever-married sample, and a mother's exposure to the CSL is also defined by her year and quarter of birth. Mothers born in the last quarter of 1986 (after September) may be affected by the CSL. The results, however, did not change with the definition of exposure to the CSL by year and quarter of birth, and thus the variation in the exposure to the CSL across birth-cohorts (by age) is used.

suggest that the change in compulsory schooling had a positive effect on the education of the cohorts 10 and younger, while it did not have any effect on the education of the cohorts 11 and older.[27] Hereafter, one year-of-birth dummy (k=7&8) is used for mothers aged 7 and 8 in 1997 since the effect of the CSL was almost the same for these cohorts and statistically not different from each other.

Figure 1 plots the effect of the CSL on primary school completion for each cohort from a linear probability model (LPM) of equation 1 without controls.[28] The year of birth dummies (α_{1k} for each k) from the LPM are jointly significant for k=7 to 10 and insignificant for k=11 to 17 (p-values are 0.000 and 0.266 respectively). Imposing the restriction that mothers aged 11 to 17 in 1997 were not affected by the CSL in order to gain precision, the first-stage takes the form:

$$S_i = \alpha_0 + \sum_{k=7}^{10} (YOB_{ik})\alpha_{1k} + \mathbf{P_i}\alpha_2 + \mathbf{K_i}\alpha_3 + \mathbf{X_i}\alpha_4 + \mathbf{C_i}\alpha_5 + u_i \qquad (2)$$

where the omitted control group is now mothers aged 11 to 18 in 1997. A discrete treatment dummy indicating whether mothers aged 7-10 in 1997 (post-reform cohort) is also used as an instrumental variable (in the above equation rather than year of birth dummies).

The second stage uses the predicted primary school completion from equation 2 as follows:

$$Y_i = \gamma_0 + \hat{S}_i\gamma_1 + \mathbf{P_i}\gamma_2 + \mathbf{K_i}\gamma_3 + \mathbf{X_i}\gamma_4 + \mathbf{C_i}\gamma_5 + v_i \qquad (3)$$

where $\hat{S}_i$ is the predicted primary school completion.[29] In all specifications, the standard errors are adjusted for clustering on the province of mother's birth. Specifications are estimated for a sample of mothers ages 18-29, where mothers ages 22-29 are the unexposed

[27] Results for this unrestricted first-stage estimation are available upon request.

[28] A probit model is also used to estimate equation 1, and the marginal effects from the probit model suggests the same effect of the CSL for each cohort (they are also not statistically different from the LPM estimates).

[29] Angrist (1991) and Angrist (2001) discuss that conventional two-stage least squares (2SLS) models are appropriate when both the outcome and endogenous variable are discrete once valid instruments are found.

cohorts. Furthermore, I use a tighter age window as a robustness check (mothers ages 18-25, where mothers ages 22-25 are the unexposed cohorts). It should be noted that a few husbands (1.4% of the sample of analysis; 24 observations) were exposed to the policy; however, the results are robust to excluding these observations.

To test whether the results are driven by time trends, reduced-form estimates of a mother's exposure to the CSL on outcome variables are also performed:

$$Y_i = \alpha_0 + \sum_{k=7}^{17} (YOB_{ik})\alpha_{1k} + \mathbf{P_i}\alpha_2 + \mathbf{K_i}\alpha_3 + \mathbf{X_i}\alpha_4 + \mathbf{C_i}\alpha_5 + u_i \quad (4)$$

For all unexposed cohorts ($k \geq 11$), the set of year of birth dummies, α_{1k}, are jointly insignificant (at all conventional significance levels) for all outcomes. Figure 2 illustrates the effect of the CSL on maternal education, child health, and infant health for each cohort (α_{1k} from equations 1 and 4). The vertical solid line indicates the timing of the education policy, where cohorts to the right of the vertical line (females aged 11 in 1997) are exposed to the law. The pattern is consistent with the hypothesis that the CSL did not influence the education of unexposed cohorts and had a positive effect on the education of the cohorts 10 and younger. Figure 2 also shows that there are no pre-existing trends in maternal education, and child and infant health prior to the education policy (left side of the vertical line). Moreover, child and infant health clearly improved following the CSL (right side of the vertical line). In sum, the figure suggests that the effects are not due to pre-existing time trends. (For further verification, see section below.)

Furthermore, the following equation is estimated to test whether there are pre-existing trends in maternal education:

$$S_i = \alpha_0 + T_i\alpha_1 + (UT_i \times YOB_i)\beta + \mathbf{P_i}\alpha_2 + \mathbf{K_i}\alpha_3 + \mathbf{X_i}\alpha_4 + \mathbf{C_i}\alpha_5 + u_i \quad (5)$$

where T_i is a dummy indicating whether mother i belongs to the post-reform cohort, UT_i is a dummy indicating whether mother i belongs to the pre-reform cohort, and YOB_i is the year of birth of a mother i. If there are pre-existing trends in education, β would be significantly different than zero. The regression results of equation (5) for a sample of women ages 18-29 suggest that the CSL increased education (α_1 is 0.39, and significant at the 1% level) and rejects that pre-existing trends are present (β is estimated to be very small (0.02) and statistically indistinguishable from zero).[30] Thus, the results provide further evidence that the differences in education are not driven by pre-existing time trends.

Moreover, in order to rule out the possibility that the results are driven by the effects of the CSL on income, the following equation is estimated:

$$Income_i = \alpha_0 + \sum_{k=7}^{17} (YOB_{ik})\alpha_{1k} + \mathbf{P_i}\alpha_2 + \mathbf{K_i}\alpha_3 + \mathbf{X_i}\alpha_4 + \mathbf{C_i}\alpha_5 + u_i \quad (6)$$

where $Income_i$ is either a dummy variable indicating whether the household of mother i has a flush toilet or an aggregate wealth index based on several household characteristics.[31]

Figure 3 plots the effect of the CSL on the wealth index and the probability of having flush toilet for each cohort. The results indicate that the CSL did not have a significant effect on income, suggesting that the results are not driven by changes in household wealth.

Alternative Approaches

Difference-in-differences (DID)

This section replaces year of birth dummies in equation 4 with a dummy variable indicating treatment of the CSL:

$$Y_i = \alpha_0 + T_i\alpha_1 + \mathbf{P_i}\alpha_2 + \mathbf{K_i}\alpha_3 + \mathbf{X_i}\alpha_4 + \mathbf{C_i}\alpha_5 + u_i \quad (7)$$

[30] Estimation of equation (5) for a sample of women ages 18-25 also yields similar results: α_1 is 0.38 (significant at the 1% level), and β is 0.01 (statistically insignificant at any conventional levels of significance).

[31] The data do not include income. The presence of a flush toilet is a common measure of household wealth. The wealth index is constructed using information on whether the household has a television, refrigerator, car, flush toilet, and no earth floor. The wealth index is constructed using principle components analysis to determine the weights of the variables.

Equation 6 compares the outcomes of mothers aged 7-10 to 11-14 in 1997. As a control experiment, I compare the outcomes of mothers aged 11-14 to 15-18 in 1997 (both unexposed) in order to rule out pre-existing trends (the CSL should not have affected these women). The reduced-form results suggest that the health outcomes and prenatal care initiation are significantly different for exposed and unexposed mothers (Panel A of Appendix B1). Moreover, the coefficients of the control experiment are small and insignificant, implying that the differences between exposed and unexposed mothers are not driven by pre-existing time trends (Panel B of Appendix B1). For instance, mothers aged 7-10 in 1997 were 8 percentage points less likely to give VLBW births, compared to mothers aged 11-14 in 1997. The estimate from a comparison of the probability of giving VLBW births between mothers aged 11-14 and 15-18 in 1997 (-0.002), however, was small and statistically insignificant (the difference-in-differences (DID) estimate is -0.074).

Regression Discontinuity Design (RDD)

Regression discontinuity design (RDD) elicits causal effects even in the presence of pre-existing trends. Because I have demonstrated above that there are no pre-existing trends, I emphasize DID as a robustness check. Specifically, I use a mother's month and year of birth relative to month and year in which the CSL took place. I estimate the effects of the CSL on maternal education and outcome variables as follows:

$$S_i\ (or Y_i) = \gamma_0 + T_i\gamma_1 + f\left(R_i\right) + \mathbf{X}_i\gamma_2 + \varepsilon_i \tag{8}$$

where R_i is a mother's month-of-birth cohort relative to the cutoff (September 1986) and $\mathbf{X_i}$ is a vector of all control variables included in the paper as well as calendar month-of-birth fixed effects.[32] The function $f(\cdot)$ captures the relationship between month-of-birth cohort

[32]Mothers born in the last quarter of 1986 (after September) may be affected by the CSL, and thus the cutoff is chosen to be September 1986.

and outcomes of interest.[33] The effect of the CSL, γ_1, is estimated using a local linear regression within 51 months (4.25 years) on either side of the cutoff.[34] The reduced-form estimates suggest that the CSL led to a substantial increase in maternal education and an earlier preventive care initiation, and improved child and infant health (Appendix B2). The RDD results therefore corroborate the main results.

Intensity Measure

This section uses variation in the intensity of the CSL across provinces and cohorts as instruments to education. More than 58 thousand classrooms were constructed within the first year of the change in the law (between 1996/97 and 1997/98 Academic Years). I use the additional number of classrooms in mother's birth provinces as a measure of intensity. Specifically, I estimate the following equation, which allows for the inclusion of cohort fixed effects ($\mathbf{YOB_i}$) to account for time-varying, country-specific factors:

$$S_i = \alpha_0 + \sum_{k=7}^{17} (Intensity_j \times YOB_{ik})\alpha_{1k} + \mathbf{YOB_i}\alpha_2 + \mathbf{P_i}\alpha_3 + \mathbf{K_i}\alpha_4 + \mathbf{X_i}\alpha_5 + \mathbf{C_i}\alpha_6 + u_i \quad (9)$$

where $Intensity_j$ is the number of additional classrooms in the birth-province of mothers. Figure 4 plots the relationship between the intensity and primary school completion by mother's age in 1997 (α_{1k}).

The estimates suggest that, on average, one additional classroom per 1,000 children increases primary school completion by around 1.6 percentage points. The average number of additional classrooms per 1,000 children is 9.38, implying that the CSL increased pri-

[33] Figures A1-A3 illustrate the relationship between birth cohort and the probability of completing primary school, HAZ, and month of first prenatal care. These figures show that maternal education increased and child health improved following the CSL (right side of the vertical side). Moreover, they provide further evidence that there are no pre-existing trends in maternal education and child health prior to the CSL (left side of the vertical line).

[34] See Imbens and Lemieux (2008) and Imbens and Kalyanaraman (2012) for local linear regression and optimal bandwidth selection. Note that the appropriate bandwidth is in the range of 50-100 using different approaches, and I used 51 as the optimal bandwidth to be conservative. Standard errors are clustered at the level of the running variable (month-year-cohort).

mary school completion of mothers by around 15 percentage points (the average primary school completion of mothers in the sample is 29.9 percentage points).

The second stage uses the predicted primary school completion from equation 9 as follows:

$$Y_i = \gamma_0 + \hat{S}_i\gamma_1 + \mathbf{YOB_i}\gamma_2 + \mathbf{P_i}\gamma_3 + \mathbf{K_i}\gamma_4 + \mathbf{X_i}\gamma_5 + \mathbf{C_i}\gamma_6 + v_i \tag{10}$$

In all specifications, the standard errors are adjusted for clustering on the province of mother's birth. Specifications are estimated for a sample of mothers ages 18-29, where mothers ages 22-29 are the unexposed cohorts. Furthermore, I use a tighter age window as a robustness check (mothers ages 18-25, where mothers ages 22-25 are the unexposed cohorts). The results are consistent with the main results (Appendix C).

5 Results

Primary School Completion (First-Stage Results)

The first stage results (equation 2) using a discrete treatment dummy and mother's year of birth dummies are shown in Table 2. Columns (1) and (2) do not use "previous births" as controls, while columns (3) and (4) control for the number of previous ever-born births.[35] The estimate in column (3) suggests that mothers exposed to the CSL have a substantially higher probability of completing primary school than unexposed mothers (around 32 percentage points higher). Column (4) presents the effect for each exposed cohort: mothers aged 7&8 at the time of the policy have the highest probability of completing primary school, and the effect decreases by age.

The first-stage results indicate that the CSL led to a substantial increase in the probability of completing primary school. F-statistic provided in column (4) of the Table 2 (11.54)

[35] The effect of the CSL on maternal schooling in column (1) is not statistically different than column (3). Likewise, the effects for each cohort in column (2) are not statistically different than those in column (4).

demonstrates that the set of year of birth dummies are jointly significant at all conventional significance levels.[36]

Effects of Maternal Education on Infant and Child Health, and Health Care Utilization

Table 3 presents the effects of primary school completion on infant and child health, and health care utilization in Panel A and Panel B, respectively. OLS estimates for the sample of mothers ages 18-29 in column (1) suggest that mothers who completed primary school (8+ years of schooling) are around 4 percentage points less likely to give VLBW births, 3 percentage points more likely to have formal prenatal care, and 10 percentage points more likely to receive 4+ prenatal visits. Moreover, mothers who completed primary school have children with higher HAZ and WAZ (0.2 higher standard deviations), and have 0.7 months earlier prenatal care initiation. The estimated effects on length of health facility stay and delivery outcomes are not statistically significant at any conventional levels of significance.

Instrumental variables estimates, using either a discrete treatment dummy or mother's year of birth dummies as instruments, for the sample of mothers ages 18-29 are shown in columns (2) and (3) of Table 3, respectively. All IV estimates have the expected signs, and are statistically significant for health outcomes. IV estimates in Panel A suggest that completing primary school yields large infant health returns in terms of VLBW, child health returns in terms of HAZ and WAZ, and maternal health returns in terms of length of health facility stay after delivery: primary school completion decreases the likelihood of giving VLBW births by around 17 percentage points, increases HAZ and WAZ of the child by 1.1 and 0.7 standard deviations, and decreases the stay at the health facility after delivery for mothers by around 1.1 days (column 3).[37] Table 3 also suggests that the effect of maternal

[36]The F-statistic is greater than 10, which suggests that the instruments are not weak (Staiger and Stock, 2007).

[37]In addition to VLBW, I also explore the effect of maternal education on birth weight (in grams), log birth weight, and a discrete indicator for low birth weight (<2500 grams). The effect of maternal education on log birth weight is positive and significant, whereas the effect on birth weight is significant only for first-time mothers (Appendix D). The effect on low birth weight is statistically insignificant. These results suggest that

education on health outcomes may operate through earlier prenatal care initiation: mothers who completed primary schooling initiate preventive care around 1.6 months earlier than the ones with less than 8 years of schooling (50 percent change relative to the mean–3.2 months). Estimates on the use of formal prenatal care, receipt of 4+ visits, delivery in a health facility, and delivery by health professionals are all positive, but not statistically significant. Formal preventive care and delivery rates are high for both mothers with and without primary schooling in the sample and, therefore, it is more likely that maternal education affects infant health via other mechanisms.

The IV estimates exceed OLS estimates for all outcomes, which may be partly due to the fact that the IV estimates present the effects of maternal education for mothers whose educational attainment has been affected by the change in the compulsory schooling law (in other words, for mothers who would not have completed 8 years of schooling if there was not a change in the CSL). The effect may be much higher for those mothers than for the population, thereby, leading to larger estimates of IV than the OLS estimates.[38] The possibility of heterogeneous effects of maternal schooling on child health and health care utilization is explored in Section 5.2. Other possibilities for the downwards bias of the OLS estimates are the presence of random measurement error, and the omitted variables that positively affect the outcome variables and are negatively correlated with maternal schooling, such as family characteristics and/or peer networks.

Effects of Other Variables

I explore the other factors that determine child health and health care utilization behavior of mothers. Table 4 presents the effects of other determinants, using the specification in column (3) of Table 3 (mother's year of birth dummies as instrumental variables).

the relationship is nonlinear and VLBW is the most relevant margin in which education impacts birth weight (note that using VLBW and log birth weight results in the highest goodness-of-fit of the OLS regression of primary school completion on various birth weight outcomes).

[38]For larger IV returns to education in terms of infant health and prenatal care, see Currie and Moretti, 2003; for a review of recent studies in terms of earnings, see Card, 2001.

The effects of ethnicity, one proxy for the childhood environment, suggest that Kurdish mothers are 13 percentage points less likely to receive 4+ prenatal visits, 6 percentage points less likely to deliver at a health facility, and postpone prenatal visits by around 0.7 months, compared to Turkish mothers. Mothers with other ethnicities are 22 percentage points less likely to receive 4+ prenatal visits and postpone prenatal visits by around 1 month compared to Turkish mothers. Pregnancy experience seems to play an important role in health care utilization outcomes explored in the paper: mothers with less previous births are more likely to seek health care services. Individual-level factors, however, excluding maternal education, do not seem to explain infant health at birth.

The effect of place of residence shows that mothers in rural areas are more likely to give VLBW births (around 5 percentage points). Mothers in rural areas are also less likely to use health care services compared to mothers in urban areas. The results suggest that maternal education and place of residence have significant effects on infant health and health care utilization. There are also substantial differences in infant health and health care utilization between mothers living in Istanbul and East Anatolia, especially Southeast Anatolia in infant health and Northeast Anatolia in health care utilization. For instance, mothers living in Southeast Anatolia are 12 percentage points more likely to give VLBW births, and 15 percentage points less likely to receive 4+ visits, compared to mothers living in Istanbul. Mothers in Central Anatolia are also 11 percentage points more likely to give VLBW birth compared to mothers in Istanbul, which again highlights the importance of regional residence.

5.1 Robustness Checks

The last three columns of Table 3 present the effects of maternal education for the sample of mothers ages 18-25, where unexposed mothers are now ages 22-25. These results provide a further robustness check for ruling out that the results are driven by time-trends. The IV estimates in columns (5) and (6) are very similar to the ones in columns (2) and (3), and, additionally, the differences are not statistically significant for all outcomes, respectively. The tighter age window estimates provide further evidence that there are significant effects of maternal education of mothers who changed their educational attainment due to the change in the CSL on infant and child health, and prenatal care initiation.

Following Currie and Moretti (2003), the effect of maternal education is reestimated for a sample of first-time mothers.[39] Columns (1) and (2) of Table 5 present the results for a sample of mothers of any parity (as in Table 3, columns 1 and 2); while columns (3) and (4) present the results for a sample of first-time mothers. The significant effects of maternal education on infant and child health, length of health facility stay after delivery, and prenatal care initiation persist for the sample of first-time mothers after controlling for the endogeneity of maternal education (column 4). Thus, Table 5 presents evidence that the results are robust to the exclusion of higher parity mothers.

Sample selection is a problem if education affects the probability of being a mother.[40] I follow the two-step procedure developed by Heckman in order to account for possible sample selection bias (Heckman, 1976; Heckman, 1979). Towards this end, I estimate the probit function determining whether or not a woman becomes a mother by age 18 for the full sample using the instruments (year-of-birth dummies) as control variables, and the propensity scores calculated from these estimates are included as controls in the estimations of health and health utilization for first-time mothers.[41] Column (5) of Table 5 presents the results after correcting for sample selection, indicating the results are robust to introducing correction for sample selection.

Additional robustness checks are provided in Table 6. Column (1) repeats the estimates in column (3) of Table 3, where mother's year of birth dummies are used as instruments. Column (2) adds a control variable indicating whether mothers exercise regularly (13% of

[39] The treatment dummy, rather than mother's year of birth dummies, is used as the instrument for maternal education for the sample of mothers ages 18-29 due to small sample size. Of course, number of previous births is not used as a control in these specifications.

[40] Güneş (2013) shows that female education reduces teenage fertility and the probability of being a teenage mother.

[41] The probit estimation includes other controls used in the paper.

the sample exercises regularly), which may affect both her and her infant's health.[42] The results show no significant effect of sports on any outcome, except for receipt of 4+ visits–mothers who exercise regularly are about 8 percentage more likely to receive 4+ visits. Column (3) of Table 6 accounts for the fact that exposure to internet may be associated with the outcomes; however, there was not any evidence of such associations (around 7% of the sample uses internet regularly). Column (4) controls for the type of sanitation facility of the household (30% did not have a flush toilet) as an indicator of household wealth. Type of sanitation facility is found to be correlated with the receipt of 4+ visits–mothers living in a house with a flush toilet are 18 percentage points more likely to receive 4+ visits. The effect of maternal education is robust to the inclusion of each additional variable in columns (1)-(4). Finally, column (5) includes all additional control variables, and the IV estimates of the effect of maternal education remain robust for health outcomes and preventive care initiation.

5.2 Heterogeneous Effects

In this section, I explore heterogeneous effects. First, I explore whether the effect of the CSL on maternal schooling differs by mother's parental education. Second, I explore whether the effects of maternal schooling on child and infant health, and maternal health care utilization, differ by sex of the child and characteristics of the province of mother's birth (prior to the change in the CSL), such as income and urbanization rates.

The Effect of the CSL on Maternal Education

In order to explore heterogeneous effect of the CSL on maternal education by parental education, I interact the treatment dummy with mother's parental educational attainment dum-

[42] I find no evidence that educated mothers exercise more. Results of the effect of maternal education on exercise were insignificant (available upon request).

mies, and use the specification in column (3) of Table 2.[43] Table 7 suggests that the CSL was more successful at increasing education of women with less than nine years of parental education, while it did not significantly affect women with parental education higher than secondary school completion (columns 2 and 3). The F-statistics (reported in the Table) show that the interaction terms and the treatment dummy are jointly significant (8.37 in column 2, and 11.08 in column 3). The results, therefore, indicate that there are heterogeneous effects by mother's parental education. The effects of maternal education on infant and child health, length of health facility stay after delivery, and prenatal care initiation are robust to using the treatment dummy interacted with parental education dummies, and the treatment dummy as instruments (Appendix E).

The Effects of Maternal Education on Child Health and Health Care Utilization

To examine heterogeneity in the effect of maternal education, I split the sample into subsamples of mother's birth-province and gender of the child. I use the specification in column (2) of Table 3, which includes all controls, and use the treatment dummy as the instrument. I explore heterogeneity by income and urbanization rates by dividing the sample into provinces with average income and urbanization rates of the unexposed mothers' province of birth above (or below) the sample median.

The results are reported in Table 8. The effects of maternal education on VLBW and the length of health facility stay after delivery are greater for female children than male children (the differences are significant at the 1% and 10% levels, respectively). Specifically, for mothers of female children, primary school completion decreases the likelihood of giving VLBW births by around 35 percentage points, and decreases the stay at the health facility by around 2.2 days (columns 2-3). The effect of maternal education on HAZ is

[43] There are 4 categories of educational attainment for both the mother and father of the mother as follows: no education, primary school completion (5 years of education in this case), secondary school completion (8 years of education), and higher than secondary school completion. In the estimations, the omitted category is mothers (fathers) with no education. Estimations include dummies for parental educational attainment categories, treatment dummy, and the treatment dummy interacted with parental educational attainment dummies as well as the other controls used in the paper.

higher in provinces with income and urbanization rates below the median (columns 4-6). In provinces with income and urbanization rates below the median, primary school completion increased HAZ of the child by around 1.9 and 4.1 standard deviations, respectively (the differences between the provinces below and above the median are significant at the 10% level). These results provide evidence that there are heterogeneous effects across provinces, although standard errors are large in some cases.

5.3 Other Potential Mechanisms

In this section, other potential mechanisms through which maternal education may affect child and infant health are explored. Table 9 presents the results from instrumental variables estimation using either a discrete treatment dummy (columns 1 and 3) or mother's year of birth dummies (columns 2 and 4) as instruments for the samples of mothers ages 18-29 and ages 18-25 for various potential mechanisms.

Panel A of Table 9 explores the effect of primary school completion on husband's education and occupation. Assuming that mother's exposure to the CSL affects husband's education only through assortative mating, the first two rows of Panel A report the effect of wife's primary school completion on husband's years of education as a continuous variable and husband's primary school completion (8+ years of education).[44] The results suggest that assortative matching does not play an important role. The third row of Panel A presents the effect of maternal education on husband's occupation. Results suggest that mothers who completed primary school are more likely to marry men who work in either service or industry sectors; however, none of the results are statistically significant.

Panel B of Table 9 presents the effects of maternal education on various outcomes. One potential mechanism through which maternal education may affect child health is smoking behavior of mothers during pregnancy; however, data is available only for ever or current

[44] Few husbands were exposed to the CSL as mentioned earlier; however, results are robust to excluding them.

smoking behavior. Arguably, current smoking is a less noisy proxy for smoking during pregnancy. The results suggest that mothers with 8+ years of education are around 30 percentage points less likely to be current smokers; however, the effect on ever smoking is insignificant. The effect of maternal education on smoking behavior is consistent with the findings of Currie and Moretti (2003), which find that an additional year of schooling reduces the probability of smoking by over 30 percent in the United States. The last potential mechanism explored is mother's occupation. There is a large effect of maternal education on the probability of mother's not working or working in the agricultural sector, but the effect is only significant at the 10% significance level in columns (1) and (3).

5.4 Long-run Effects of the CSL

This section translates the gains in HAZ and birth weight associated with the CSL into long-run income gains. Moreover, it performs a benefit-cost analysis to determine whether the benefits in terms of greater earnings outweigh the cost. Since I do not observe earnings in the dataset and there have not been any studies relating height and birth weight to earnings in Turkey, I rely on estimates from previous studies in other developing countries and the US to calibrate the magnitude of the benefits and costs of the CSL. For this reason, the estimates should only be considered suggestive.

Panel A of Table 10 calculates the gains in height and wages using the estimated impact of the CSL on HAZ (column 3 of Appendix B1). Because previous studies document that variation in child height persists to adulthood, I assume that variation in standardized child height translates into variation in standardized adult height (Thomas and Strauss, 1997; Case and Paxson, 2008; among the studies below). The 0.608 standard deviation gain due to the CSL, therefore, translates into a 3.65 cm gain in adult height.[45] Rows one and two calculate the associated long-run income gains associated with the CSL using estimated

[45]Note that the calculation of gain in adult height does not vary by gender, and the height distribution of a healthy adult population suggests that 3 cm in height corresponds to 0.4-0.5 standard deviations.

marginal effects (or elasticities) of adult height on earnings from several studies conducted in developing countries. Row one follows Schultz (2005), who finds that a 1 cm increase in height is associated with 4-8% increase in wages (of men and women) in low-income countries (Brazil and Ghana), implying that wages would increase by 14.59-29.18%. Row two calculates the gain in adult height (4.757%) using the average z-score of adult males ages 25-35 (-0.68) in the study by Agüero et al. (2007). Following Thomas and Strauss (1997), who find that a 1% increase in height leads to a 2.4% increase in adult male earnings in Brazil, wages would increase by 11.56-15.98%.

Panel B of Table 10 calculates reductions in infant mortality and gains in wages from increases in birth weight (both directly and indirectly via adult height) associated with the CSL.[46] The calculations are based on US data because there are no studies in the context of developing countries. Conley et al. (2003) find that a 1 lb increase in birth weight leads to a 14% decrease in infant mortality between 28 days and 1 year, and a 11% decrease in the risk of death in the first 28 days, which in turn implies that the 0.176 kg increase in birth weight associated with the CSL reduces mortality by 5.48 and 4.30%, respectively. Behrman and Rosenzweig (2004) find that a 1 kg increase in birth weight leads to a 1.6 cm increase in height and over 18.6% increase in wages, suggesting that increases in birth weight would lead to a 0.282 cm increase in height and over 3.27% increase in wages. Black et al. (2007) find that a 10% increase in birth weight leads to a 0.57 cm increase in adult height for males. Thus, the impact of the CSL on birth weight would translate into a 0.353 cm increase in height, which in turn would lead to over 4.09% gain in wages following the study by Behrman and Rosenzweig (2004).

[46]The only available figures are from the US. The effect of the CSL on (log) birth weight is calculated by multiplying the IV effect (column 5 of Appendix D) by the average increase in education for mothers who were exposed to the CSL (column 3 of Table 2).

Benefit-Cost Analysis

This section uses the benefits in terms of wages conferred from increases in height and compares them to cost in terms of greater education. This approach estimates the benefits conservatively because education might influence wages through other channels, besides height. Moreover, I do not account for general equilibrium effects, which would have an ambiguous effect. That is, an increase in the average height might increase average wages by more or less than an increase in an individual's height, depending on whether there are positive or negative social externalities. For these reasons, and those mentioned above, I emphasize that the estimates are only suggestive.

Following Agüero et al. (2007) and Behrman et al. (2004), the present discounted value of gains in wages (in dollars) from age 25 to 65 are calculated using discount rates of 3 and 5%. Table 11 presents the discounted present values using the range of wage gains from Table 10 Panel A, under two alternative assumptions–that individuals are fully employed or are employed for 75% of the period, with random stints of unemployment. There are no rigorous studies evaluating the exact cost of the CSL in Turkey; however, the per-person cost of an additional 3 years of education is approximately $600 based on the National Education Statistics books by MONE and the detailed information provided by the Turkish Statistics Institute (TurkStat).[47] I use a more conservative cost of $900 (an additional 50%) to calculate the benefit/cost ratio. The discounted present value of gains in earnings associated with 11.56% increase in wages at a 5% discount rate is $2302, which gives a benefit/cost ratio of 2.56 if fully employed or 1.92 if employed for 75% of the period.[48] Following Behrman et al. (2004), the benefit/cost ratios are also calculated by adjusting the cost with an additional 25% increase: the ratios still exceed one.

[47] Total educational expenditures are approximately 3 billion TL for 10 million enrolled primary school students per year.

[48] To be conservative, I use the minimum wage of $335 (per month) to calculate the nominal gains–using minimum wages understates the benefits if the average earnings are greater than the minimum wage earnings.

6 Discussion and Conclusion

This paper provides evidence of the effect of maternal education on child health and explores potential mechanisms through which maternal education may affect infant and child health. I use a change in the compulsory schooling law in Turkey as a natural experiment, which extended compulsory primary schooling from five to eight years (free of charge in public schools). I find that the law increased the likelihood of mothers' completing 8+ years of schooling by around 32 percentage points on average.

Variation in the probability of completing primary school across cohorts induced by the change in the CSL is used as an instrumental variable in order to explore the causal effect of maternal education on child health and potential channels through which schooling operates. The IV estimates suggest that mother's primary school completion improves infant health, as measured by very low birth weight (reduction of approximately 17 percentage points), and child health, as measured by HAZ and WAZ (increases of 1.1 and 0.7 standard deviations, respectively), and, moreover, the results are robust to various specifications. The IV estimates also provide evidence that improvements in health outcomes are partly due to the effects of maternal education on maternal health behaviors: mother's primary school completion leads to earlier preventive care initiation and reduces smoking. Exploring other factors that are likely to influence child and infant health does not suggest significant effects of maternal education on formal prenatal care and delivery, occupation, and assortative matching. Exploring heterogeneous effects suggests that the effect of the CSL differs by mother's parental education, and the effects of maternal education on child health depend on province characteristics and the sex of the child.

The results also suggest that, even after controlling for education, region and urban/rural residence are important factors of infant health and health care utilization, and, additionally, pregnancy experience and ethnicity are important factors of health care utilization. Therefore interventions aimed at these subpopulations are important for reducing infant health and health care utilization disparities.

Future research may explore the causal effect of education along different maternal schooling margins in developing countries, which may improve child health to a greater extent. While the results are consistent with the findings of a related study for a developed country as explored by Currie and Moretti (2003), more research is needed for various settings, especially developing countries, to understand the extent that the results can be generalized.

Figure 1: Primary School Completion of Mothers

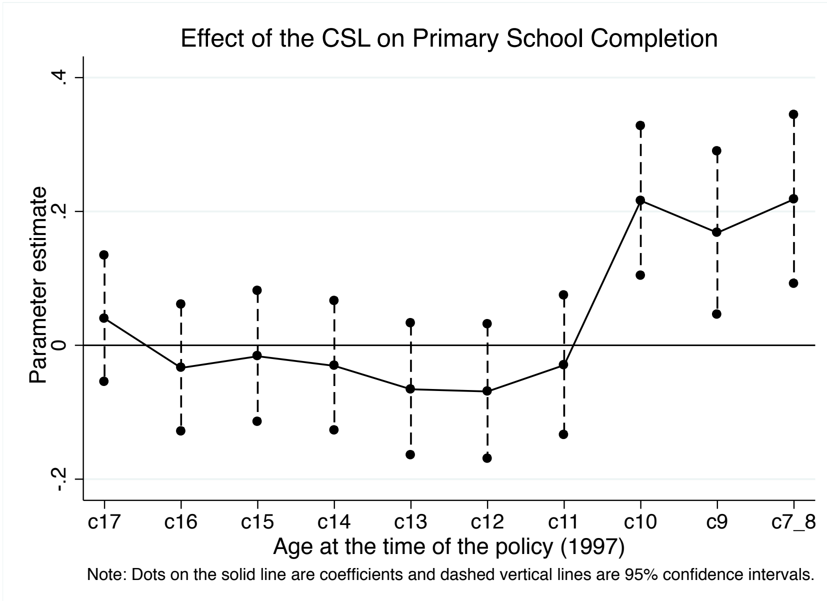
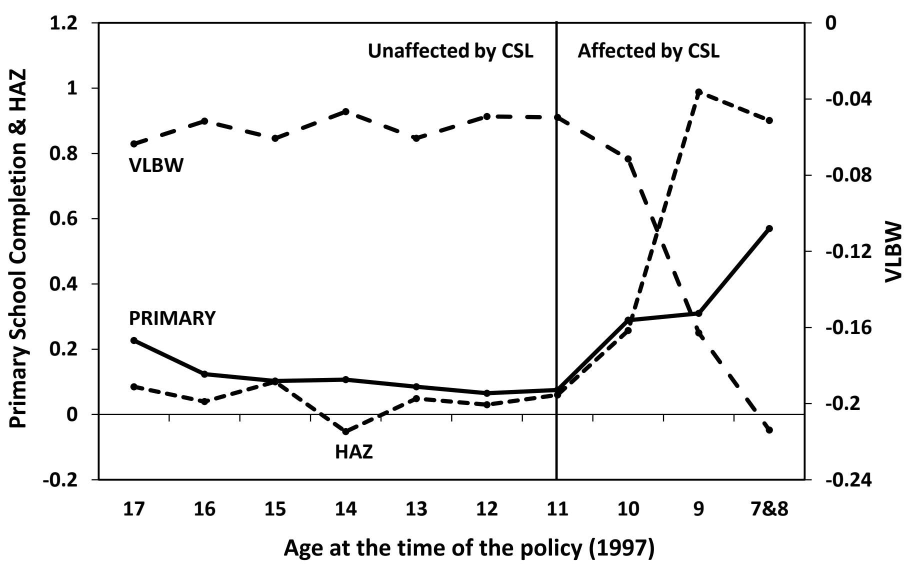


Figure 2: Effect of the CSL on Maternal Education, HAZ, and VLBW



Notes: The sample includes all mothers between the ages of 18 and 29 at the time of the survey. Females aged 11 in 1997 is the youngest unaffected cohort (solid vertical line). Each point on the solid, dotted, and dashed lines represents the effect of the CSL on the probability of completing primary school, the HAZ of the child, and the probability of giving VLBW birth for each cohort, respectively.

Figure 3: Effect of the CSL on Income

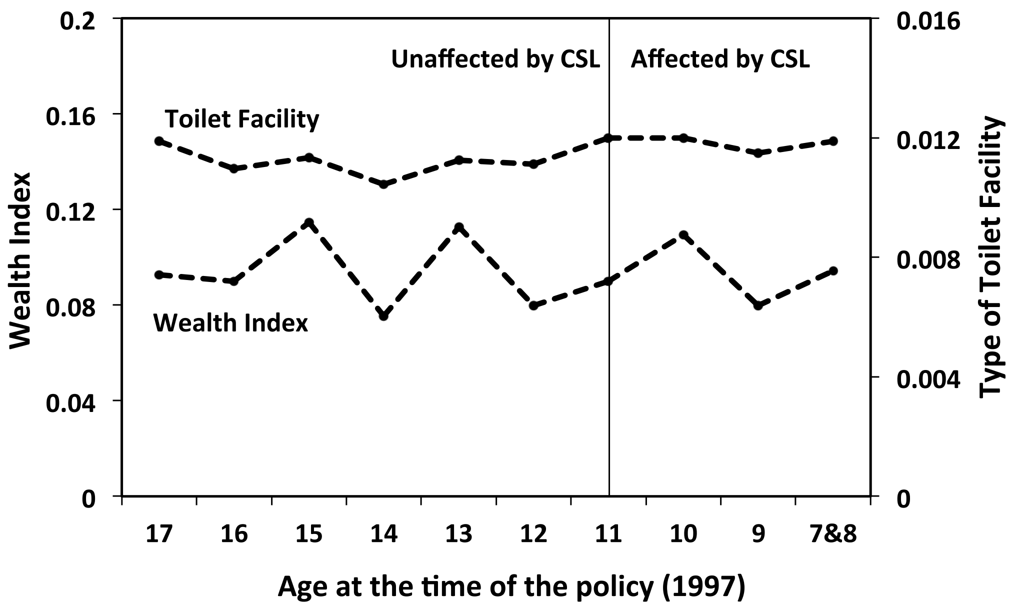


Figure 4: Coefficients of Interactions

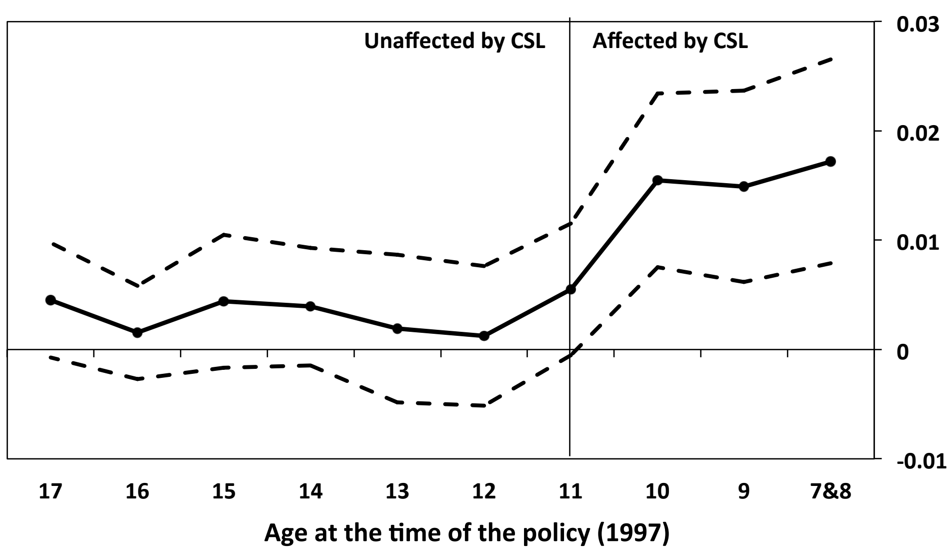


Table 1: Summary Statistics For Outcomes and Selected Explanatory Variables[a]

Variables	Mean	Std.Err.	Variables	Mean	Std.Err.
Health Outcomes			**Community-level Variables**		
VLBW (birth weight<1500 grams)	0.069	0.253	*Type of residence*		
Length of health facility stay (in days)	1.658	2.457	Rural	0.301	0.459
Height-for-age z-score (HAZ)	-0.581	1.478	Urban[b]	0.699	0.459
Weight-for-age z-score (WAZ)	0.146	1.055	*Region*		
Health Utilization Behavior of Mothers			Istanbul[b]	0.052	0.222
Prenatal care from formal sources	0.894	0.308	West Marmara	0.042	0.200
Receipt of 4+ prenatal visits	0.673	0.469	Aegean	0.061	0.239
Prenatal care initiation (in months)	3.198	2.889	East Marmara	0.064	0.246
Delivery in a health facility	0.897	0.303	West Anatolia	0.077	0.267
Delivery assistance by health professionals	0.919	0.272	Mediterranean	0.132	0.339
Individual-level Variables			Central Anatolia	0.080	0.271
Compulsory Primary School Completion (8+)	0.299	0.458	West Black Sea	0.069	0.254
Age at the time of the policy			East Black Sea	0.041	0.199
Cohort 7&8 (Age 7&8 in 1997)	0.044	0.204	Northeast Anatolia	0.095	0.294
Cohort 9 (Age 9 in 1997)	0.048	0.214	Central East Anatolia	0.114	0.319
Cohort 10 (Age 10 in 1997)	0.064	0.244	Southeast Anatolia	0.172	0.377
Pregnancy Experience					
Previous Births	0.902	1.100			
Ethnicity					
Turkish[b]	0.642	0.479			
Kurdish	0.313	0.464			
Other	0.045	0.207			

[a] N=1,677 for all; except for VLBW (N=1,616), length of health facility stay (N=1,519), HAZ (N=1,177), and WAZ (N=1,243)
[b] used as a reference category.

Table 2: Effect of the CSL on Primary School Completion (First Stage)

	(1)	(2)	(3)	(4)
Age 7-10 in 1997	0.366***		0.315***	
	(0.069)		(0.070)	
Age 7&8 in 1997		0.561***		0.520***
		(0.100)		(0.099)
Age 9 in 1997		0.294***		0.234**
		(0.096)		(0.098)
Age 10 in 1997		0.288**		0.235**
		(0.117)		(0.111)
Previous births	No	No	Yes	Yes
N	1677	1677	1677	1677
R^2	0.265	0.267	0.286	0.289
F-statistic		12.69		11.54

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. The F-statistic test the hypothesis that the coefficients of the mother's year of birth dummies are jointly zero. *Notes:* Standard errors (reported in parentheses) are adjusted for clustering on the province of mother's birth. All models include individual- and community-level variables, and interactions between the mother's year of birth dummies and the number of primary school aged children in the province of mother's birth (in 1995) as well as interactions between the mother's year of birth dummies and enrollment rates in the province of mother's birth (in 1995). Models (1) & (3) use discrete treatment dummy, and models (2) & (4) use mother's year of birth dummies.

Table 3: Effects of Maternal Education on Infant and Child Health, and Health Care Utilization

	OLS 18-29 (1)	IV (T) 18-29 (2)	IV (YOB) 18-29 (3)	OLS 18-25 (4)	IV (T) 18-25 (5)	IV (YOB) 18-25 (6)
Panel A: Health Outcomes						
Very low birth weight	-0.039***	-0.166*	-0.167*	-0.038**	-0.174*	-0.184*
	(0.009)	(0.097)	(0.098)	(0.016)	(0.107)	(0.110)
Length of health facility stay	0.011	-1.788**	-1.114*	-0.153	-2.244**	-1.454**
	(0.155)	(0.844)	(0.617)	(0.240)	(0.919)	(0.648)
Height-for-age z-score	0.226**	1.231**	1.095*	0.371**	1.489**	1.307**
	(0.104)	(0.606)	(0.598)	(0.164)	(0.660)	(0.653)
Weight-for-age z-score	0.164*	0.902*	0.717*	0.306**	1.017*	0.999*
	(0.093)	(0.479)	(0.420)	(0.122)	(0.604)	(0.563)
Panel B: Health Utilization Outcomes						
Formal prenatal care	0.032**	0.090	0.065	0.033*	0.043	0.035
	(0.014)	(0.105)	(0.086)	(0.019)	(0.110)	(0.087)
Receipt of 4+ prenatal visits	0.096***	0.092	0.061	0.073*	0.111	0.137
	(0.023)	(0.159)	(0.141)	(0.037)	(0.179)	(0.144)
Month of first prenatal care	-0.746***	-1.603*	-1.635**	-0.787***	-1.821*	-1.495*
	(0.151)	(0.850)	(0.737)	(0.191)	(0.935)	(0.810)
Delivery in a health facility	0.002	0.015	0.079	-0.008	0.033	0.085
	(0.017)	(0.091)	(0.084)	(0.025)	(0.109)	(0.092)
Delivery by health professionals	0.002	0.030	0.075	-0.006	0.037	0.076
	(0.015)	(0.082)	(0.079)	(0.022)	(0.098)	(0.090)
Observations	1,677	1,677	1,677	927	927	927

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: Standard errors (reported in parentheses) are adjusted for clustering on the province of mother's birth. All models include individual- and community-level variables, and interactions between the mother's year of birth dummies and the number of primary school aged children in the province of mother's birth (in 1995) as well as interactions between the mother's year of birth dummies and enrollment rates in the province of mother's birth (in 1995). Instrumental variables are discrete treatment dummy in models (2) and (5), and mother's year of birth dummies for those exposed to the CSL in models (3) and (6). Models (1)-(3) contain 1,616, 1,519, 1,177, and 1,243 observations, and models (4)-(6) contain 891, 836, 643, and 707 observations for VLBW, length of stay, HAZ, and WAZ outcomes, respectively. Health utilization models (1)-(3) contain 1,677, and (4)-(6) contain 927 observations.

Table 4: Effects of Other Explanatory Variables

	VLBW	Time	HAZ	WAZ	Prenatal Formal	4+ Visits	Initiation	Delivery Hospital	Delivery Formal
Individual-level variables									
Previous Births	0.018	-0.178*	-0.065	-0.056	-0.062***	-0.094***	0.505***	-0.051***	-0.044***
	(0.012)	(0.093)	(0.069)	(0.040)	(0.015)	(0.017)	(0.107)	(0.011)	(0.012)
Kurdish	0.009	-0.627***	-0.154	-0.063	-0.029	-0.130***	0.669**	-0.055*	-0.033
	(0.023)	(0.232)	(0.220)	(0.143)	(0.030)	(0.041)	(0.267)	(0.030)	(0.027)
Others	0.018	0.204	-0.312	-0.056	-0.038	-0.220***	0.994***	-0.013	-0.050
	(0.031)	(0.365)	(0.237)	(0.122)	(0.032)	(0.041)	(0.359)	(0.055)	(0.043)
Community-level variables									
Rural	0.051**	-0.339**	-0.159	-0.045	-0.054**	-0.146***	0.764***	-0.071**	-0.080**
	(0.020)	(0.154)	(0.107)	(0.096)	(0.023)	(0.030)	(0.191)	(0.033)	(0.033)
West Marmara	-0.014	1.098*	-0.090	-0.014	-0.012	-0.014	0.096	0.027	0.049
	(0.025)	(0.608)	(0.288)	(0.225)	(0.050)	(0.086)	(0.467)	(0.052)	(0.030)
Aegean	-0.028	0.390	0.009	0.017	0.072**	0.136**	-1.120***	0.039	0.068**
	(0.029)	(0.329)	(0.365)	(0.246)	(0.034)	(0.064)	(0.360)	(0.048)	(0.032)
East Marmara	0.027	-0.105	0.606**	0.084	0.023	-0.005	-0.175	0.057	0.014
	(0.029)	(0.289)	(0.300)	(0.194)	(0.043)	(0.058)	(0.323)	(0.041)	(0.032)
West Anatolia	0.050	0.283	-0.475	-0.368	-0.063	-0.094	0.992**	0.018	-0.015
	(0.031)	(0.375)	(0.358)	(0.227)	(0.054)	(0.075)	(0.456)	(0.041)	(0.033)
Mediterranean	0.077**	-0.045	-0.054	-0.259	0.054	-0.008	-0.537	-0.006	-0.019
	(0.034)	(0.370)	(0.282)	(0.167)	(0.045)	(0.061)	(0.411)	(0.068)	(0.051)
Central Anatolia	0.106*	0.206	-0.274	-0.327	0.052	-0.032	-0.588	0.034	0.011
	(0.054)	(0.423)	(0.364)	(0.249)	(0.044)	(0.073)	(0.433)	(0.037)	(0.027)
West Black Sea	0.031	1.526**	-0.163	-0.191	0.048	0.009	-0.399	0.037	0.003
	(0.028)	(0.723)	(0.387)	(0.233)	(0.049)	(0.066)	(0.429)	(0.035)	(0.029)
East Black Sea	0.025	0.742	0.336	-0.105	-0.012	0.095	-0.035	0.085*	0.058
	(0.030)	(0.543)	(0.332)	(0.219)	(0.049)	(0.086)	(0.484)	(0.044)	(0.037)
Northeast Anatolia	0.046*	0.184	-0.447	-0.401	-0.107**	-0.108*	0.977**	-0.069	-0.020
	(0.026)	(0.431)	(0.411)	(0.311)	(0.051)	(0.060)	(0.420)	(0.060)	(0.034)
Central East Anatolia	0.093*	-0.257	0.013	-0.441**	-0.048	-0.139**	0.280	-0.060	-0.105**
	(0.053)	(0.528)	(0.270)	(0.192)	(0.052)	(0.071)	(0.419)	(0.054)	(0.042)
Southeast Anatolia	0.123***	-0.041	-0.244	-0.219	-0.038	-0.149***	0.095	-0.008	-0.023
	(0.036)	(0.314)	(0.427)	(0.210)	(0.056)	(0.056)	(0.459)	(0.071)	(0.051)
N	1616	1519	1177	1243	1677	1677	1677	1677	1677

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: Standard errors (reported in parentheses) are adjusted for clustering on the province of mother's birth. Specification in column (3) of Table 3 is used.

Table 5: Robustness Checks: First Births and Sample Selection Correction

	All births		First births		
	OLS	IV (T)	OLS	IV (T)	IV (YOB)
	18-29	18-29	18-29	18-29	18-29
	(1)	(2)	(3)	(4)	(5)
Panel A: Health Outcomes					
Very low birth weight	-0.039***	-0.166*	-0.017*	-0.068*	-0.095*
	(0.009)	(0.097)	(0.010)	(0.041)	(0.055)
Length of health facility stay	0.011	-1.788**	0.083	-1.505**	-1.667**
	(0.155)	(0.844)	(0.196)	(0.585)	(0.825)
Height-for-age z-score	0.226**	1.231**	0.429***	1.665*	1.745*
	(0.104)	(0.606)	(0.152)	(0.912)	(1.058)
Weight-for-age z-score	0.164*	0.902*	0.241*	1.147*	1.320*
	(0.093)	(0.479)	(0.125)	(0.660)	(0.799)
Panel B: Health Utilization Outcomes					
Formal prenatal care	0.032**	0.090	0.036***	0.035	0.019
	(0.014)	(0.105)	(0.011)	(0.054)	(0.046)
Receipt of 4+ prenatal visits	0.096***	0.092	0.089***	0.050	0.100
	(0.023)	(0.159)	(0.028)	(0.134)	(0.202)
Month of first prenatal care	-0.746***	-1.603*	-0.697***	-1.044*	-1.436*
	(0.151)	(0.850)	(0.156)	(0.608)	(0.860)
Delivery in a health facility	0.002	0.015	0.016	0.032	0.046
	(0.017)	(0.091)	(0.010)	(0.065)	(0.053)
Delivery by health professionals	0.002	0.030	0.011	0.049	0.017
	(0.015)	(0.082)	(0.009)	(0.032)	(0.051)
Sample Selection Correction	No	No	No	No	Yes
Observations	1,677	1,677	733	733	733

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: Standard errors (reported in parentheses) are adjusted for clustering on the province of mother's birth. The coefficients in columns (1) and (2) are same with the ones in Table 3, and the same specifications (excluding the number of previous births as a control) are used for the sample of first-time mothers in columns (3) and (4). Model (5) accounts for sample selection. Models (1) and (2) contain 1,616, 1,519, 1,177, and 1,243 observations, and models (3), (4), and (5) contain 711, 714, 512, and 546 observations for VLBW, length of stay, HAZ, and WAZ outcomes, respectively.

Table 6: Additional Robustness Checks

	IV 18-29 (1)	IV 18-29 (2)	IV 18-29 (3)	IV 18-29 (4)	IV 18-29 (5)
Panel A: Health Outcomes					
Very low birth weight	-0.167*	-0.169*	-0.168*	-0.169*	-0.171*
	(0.098)	(0.099)	(0.098)	(0.098)	(0.098)
Length of health facility stay	-1.114*	-1.211*	-1.116*	-1.071*	-1.158*
	(0.617)	(0.638)	(0.615)	(0.611)	(0.626)
Height-for-age z-score	1.095*	1.090*	1.102*	1.108*	1.106*
	(0.598)	(0.597)	(0.605)	(0.600)	(0.605)
Weight-for-age z-score	0.717*	0.729*	0.714*	0.718*	0.726*
	(0.420)	(0.426)	(0.416)	(0.417)	(0.417)
Panel B: Health Utilization Outcomes					
Formal prenatal care	0.065	0.066	0.065	0.060	0.061
	(0.086)	(0.086)	(0.085)	(0.083)	(0.083)
Receipt of 4+ prenatal visits	0.061	0.052	0.057	0.037	0.029
	(0.141)	(0.143)	(0.142)	(0.138)	(0.139)
Month of first prenatal care	-1.635**	-1.653**	-1.633**	-1.505**	-1.529**
	(0.737)	(0.737)	(0.724)	(0.715)	(0.702)
Delivery in a health facility	0.079	0.074	0.078	0.078	0.072
	(0.084)	(0.085)	(0.082)	(0.083)	(0.083)
Delivery by health professionals	0.075	0.131	0.074	0.073	0.069
	(0.079)	(0.080)	(0.078)	(0.079)	(0.077)
Control Variables					
Sports	No	Yes	No	No	Yes
Internet	No	No	Yes	No	Yes
Flush toilet	No	No	No	Yes	Yes
Observations	1,677	1,677	1,677	1,677	1,677

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: Standard errors (reported in parentheses) are adjusted for clustering on the province of mother's birth. The results of specification in column (3) of Table 3 are repeated in column (1), and the same specification is used in other columns with additional control variables. Models contain 1,616, 1,519, 1,177, and 1,243 observations for VLBW, length of stay, HAZ, and WAZ outcomes, respectively.

Table 7: Heterogeneous Effects of the CSL on Education by Mother's Parental Education

	Main Effect	Mother's Education	Father's Education
	(1)	(2)	(3)
Age 7-10 in 1997 (Treatment)	0.315***	0.196***	0.193*
	(0.070)	(0.073)	(0.100)
ME (Primary)		0.130***	
		(0.027)	
ME (Secondary)		0.298***	
		(0.111)	
ME (Higher)		0.468***	
		(0.101)	
Treatment*ME (Primary)		0.141**	
		(0.054)	
Treatment*ME (Secondary)		0.276**	
		(0.123)	
Treatment*ME (Higher)		-0.021	
		(0.122)	
FE (Primary)			0.041*
			(0.024)
FE (Secondary)			0.271***
			(0.058)
FE (Higher)			0.459***
			(0.041)
Treatment*FE (Primary)			0.173**
			(0.082)
Treatment*FE (Secondary)			0.199**
			(0.100)
Treatment*FE (Higher)			0.030
			(0.109)
N	1677	1664	1598
R2	0.286	0.318	0.361
F-statistic		8.37	11.08

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: The F-statistics test the hypothesis that the coefficients of the treatment dummy and the parental education dummies interacted with the treatment dummy are jointly zero. *Notes:* Standard errors (reported in parentheses) are adjusted for clustering on the province of mother's birth. Model (1) repeats the coefficient in column (3) of Table (2). ME (FE) are mother's (father's) education categories, and omitted group is mothers (fathers) with no education.

Table 8: Heterogeneous Effects by Sex of the Child and Pre-change Province Characteristics

	Whole Sample	Sex of the child		Pre-Change Province of Birth Characteristics			
				GDP		Urbanization	
		Female	Male	< Median	>= Median	< Median	>= Median
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Panel A: Health Outcomes							
Very low birth weight	-0.166*	-0.348**	0.071	-0.177	-0.185*	-0.280	-0.181
	(0.097)	(0.148)	(0.179)	(0.141)	(0.100)	(0.200)	(0.146)
Length of health facility stay	-1.788**	-2.197**	0.386	-0.211	-2.745*	-1.716	-1.791
	(0.844)	(1.029)	(1.812)	(1.186)	(1.502)	(1.598)	(1.168)
Height-for-age z-score	1.231**	0.821	0.553	1.897*	0.691	4.058*	0.590
	(0.606)	(0.828)	(0.903)	(1.030)	(0.654)	(2.258)	(0.574)
Weight-for-age z-score	0.902*	0.961	0.458	0.203	1.054	1.150*	0.473
	(0.479)	(0.714)	(0.862)	(0.450)	(0.809)	(0.667)	(0.828)
Panel B: Health Utilization Outcomes							
Formal prenatal care	0.090	0.051	0.228	0.096	0.034	-0.133	0.204*
	(0.105)	(0.121)	(0.302)	(0.180)	(0.129)	(0.224)	(0.123)
Receipt of 4+ prenatal visits	0.092	0.189	0.209	-0.145	0.155	-0.151	0.312
	(0.159)	(0.176)	(0.392)	(0.271)	(0.190)	(0.367)	(0.221)
Month of first prenatal care	-1.603*	-1.959*	-1.749	-1.070	-1.479	-0.837	-1.814
	(0.850)	(1.060)	(2.324)	(1.427)	(1.135)	(1.626)	(1.283)
Delivery in a health facility	0.015	0.076	-0.039	-0.037	0.015	0.080	-0.042
	(0.091)	(0.078)	(0.316)	(0.170)	(0.095)	(0.226)	(0.107)
Delivery by health professionals	0.030	0.087	-0.038	0.023	0.002	0.144	-0.046
	(0.082)	(0.098)	(0.335)	(0.149)	(0.099)	(0.190)	(0.110)

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: Standard errors (reported in parentheses) are adjusted for clustering on the province of mother's birth. All models include all control variables in Table 3 and use treatment dummy as an instrument for a sample of women ages 18-29. Column (1) repeats the estimates in column (2) of Table 3.

Table 9: IV Estimates of Other Potential Mechanisms

Potential Mechanisms	IV (T) 18-29 (1)	IV (YOB) 18-29 (2)	IV (T) 18-25 (3)	IV (YOB) 18-25 (4)
Panel A: Husband Characteristics				
Husband Education	0.320	0.279	0.373	0.124
	(1.013)	(0.932)	(1.065)	(0.940)
Husband Completed Primary School	0.057	0.055	0.051	0.027
	(0.198)	(0.181)	(0.198)	(0.182)
Husband Occupation	-0.135	-0.054	-0.122	-0.039
(Agricultural Sector and No Work)	(0.138)	(0.138)	(0.158)	(0.154)
Panel B: Mothers Characteristics				
Ever Smoke	-0.173	-0.173	-0.136	-0.112
	(0.201)	(0.204)	(0.206)	(0.192)
Current Smoke	-0.310*	-0.298*	-0.274*	-0.217*
	(0.166)	(0.171)	(0.181)	(0.166)
Occupation	-0.300*	-0.164	-0.351**	-0.187
(Agricultural Sector and No Work)	(0.162)	(0.145)	(0.175)	(0.156)
Observations	1,677	1,677	927	927

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: Standard errors (reported in parentheses) are adjusted for clustering on the province of mother's birth. Instrumental variables are discrete treatment dummy in models (1) and (3), and mother's year of birth dummies for those exposed to the CSL in models (2) and (4).

Table 10: Long-run Effects of the CSL

Panel A. HAZ (0.608 standard deviation gain)	Gain in height	Gain in wages	
1) Schultz (2005): Low-income countries (Brazil, Ghana, Cote d'Ivoire) 1 cm increase in height: 4-8% increase in wages	0.608 sd (3.65 cm gain)	14.59 - 29.18 %	
2) Aguero et al. (2007): South Africa (males) and Thomas and Strauss (2007): Brazil (urban males) 1% increase in height: 2.43-3.36% increase in wages	4.757 % (5.32 cm gain)	11.56 - 15.98 %	
Panel B. Birth Weight (0.176 kg and 0.062 log birth weight gains)	Gain in height	Gain in wages	Reduction in Infant Mortality
1) Conley et al. (2003): US			
1 lb increase at birth: 14% decrease in mortality between 28 days and 1 year	-	-	5.48 %
1 lb increase at birth: 11% decrease in risk of death in the first 28 days	-	-	4.30 %
2) Behrman and and Rosenzweig (2004): US 1 kg increase in birth weight: 1.6 cm increase in height and over 18.6% increase in wages	0.282 cm	over 3.27 %	-
3) Black et al. (2007): US 10% increase in birth weight: 0.57 cm increase in height for males	0.353 cm	over 4.09 %	-

Notes: 0.608 standard deviation gain is the estimate in column 3 of Appendix B1. 0.176 kg and 0.062 log birth weight gains are calculated by multiplying the IV effects (column 5 of Appendix D) by the average increase in education for mothers who were exposed to the CSL (column 3 of Table 2).

Table 11: Benefit-Cost Analysis

	Discount Rate 3 %				Discount Rate 5 %			
Gain in wages	Cost	Benefit	Benefit/Cost Ratio Fully Employed	Benefit/Cost Ratio Employed 75% of the time	Cost	Benefit	Benefit/Cost Ratio Fully Employed	Benefit/Cost Ratio Employed 75% of the time
11.56%	900	5113.27	5.68	4.26	900	2302.47	2.56	1.92
14.59%	900	6453.51	7.17	5.38	900	2905.97	3.23	2.42
15.98%	900	7068.34	7.85	5.89	900	3182.82	3.54	2.65
11.56%	1125	5113.27	4.55	3.41	1125	2302.47	2.05	1.53
14.59%	1125	6453.51	5.77	4.33	1125	2905.97	2.58	1.94
15.98%	1125	7068.34	6.28	4.71	1125	3182.82	2.83	2.12

Notes: Values are in dollars (1 TL=0.7 dollar). Minimum wage per month is assumed to be 335 dollars. The estimates in the first 3 rows are based on a cost of $900 and the estimates in the last 3 rows are based on an adjusted cost of $1125 (25% increase).

Appendix A. Effects With and Without Previous Births

	OLS	IV (T)	IV (YOB)	OLS	IV (T)	IV (YOB)	OLS	IV (T)	IV (YOB)	OLS	IV (T)	IV (YOB)
	18-29	18-29	18-29	18-25	18-25	18-25	18-29	18-29	18-29	18-25	18-25	18-25
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Panel A: Health Outcomes												
Very low birth weight	-0.039***	-0.166*	-0.167*	-0.038**	-0.174*	-0.184*	-0.042***	-0.179*	-0.188**	-0.041***	-0.196*	-0.198**
	(0.009)	(0.097)	(0.098)	(0.016)	(0.107)	(0.110)	(0.009)	(0.096)	(0.090)	(0.016)	(0.106)	(0.098)
Length of health facility stay	0.011	-1.788**	-1.114*	-0.153	-2.244**	-1.454**	0.040	-1.377**	-1.010*	-0.113	-1.903**	-1.343**
	(0.155)	(0.844)	(0.617)	(0.240)	(0.919)	(0.648)	(0.147)	(0.686)	(0.584)	(0.233)	(0.799)	(0.598)
Height-for-age z-score	0.226**	1.231**	1.095*	0.371**	1.489**	1.307**	0.253**	1.284**	1.144**	0.389**	1.469**	1.278**
	(0.104)	(0.606)	(0.598)	(0.164)	(0.660)	(0.653)	(0.103)	(0.587)	(0.579)	(0.166)	(0.631)	(0.619)
Weight-for-age z-score	0.164*	0.902*	0.717*	0.306**	1.017*	0.999*	0.181*	0.932**	0.754*	0.347***	1.109*	1.089**
	(0.093)	(0.479)	(0.420)	(0.122)	(0.604)	(0.563)	(0.096)	(0.465)	(0.412)	(0.124)	(0.574)	(0.533)
Panel B: Health Utilization Outcomes												
Formal prenatal care	0.032**	0.090	0.065	0.033*	0.043	0.035	0.059***	0.203**	0.167**	0.061***	0.141	0.115
	(0.014)	(0.105)	(0.086)	(0.019)	(0.110)	(0.087)	(0.015)	(0.095)	(0.081)	(0.021)	(0.103)	(0.084)
Receipt of 4+ prenatal visits	0.096***	0.092	0.061	0.073*	0.111	0.137	0.135***	0.266*	0.182	0.114***	0.251	0.138
	(0.023)	(0.159)	(0.141)	(0.037)	(0.179)	(0.144)	(0.023)	(0.149)	(0.136)	(0.038)	(0.164)	(0.145)
Month of first prenatal care	-0.746***	-1.603*	-1.635**	-0.787***	-1.821*	-1.495*	-1.026***	-2.732***	-1.965***	-1.103***	-2.313**	-1.560*
	(0.151)	(0.850)	(0.737)	(0.191)	(0.935)	(0.810)	(0.154)	(0.840)	(0.739)	(0.219)	(0.903)	(0.802)
Delivery in a health facility	0.002	0.015	0.079	-0.008	0.033	0.085	0.026	0.122	0.101	0.018	0.122	0.135
	(0.017)	(0.091)	(0.084)	(0.025)	(0.109)	(0.092)	(0.016)	(0.078)	(0.078)	(0.024)	(0.097)	(0.088)
Delivery by health professionals	0.002	0.030	0.075	-0.006	0.037	0.076	0.023	0.121	0.117	0.019	0.122	0.134
	(0.015)	(0.082)	(0.079)	(0.022)	(0.098)	(0.090)	(0.015)	(0.075)	(0.072)	(0.022)	(0.086)	(0.082)
Previous Births	Yes	Yes	Yes	Yes	Yes	Yes	No	No	No	No	No	No
Observations	1,677	1,677	1,677	927	927	927	1,677	1,677	1,677	927	927	927

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: Standard errors (reported in parentheses) are adjusted for clustering on the province of mother's birth. All models include individual- and community-level variables, and interactions between the mother's year of birth dummies and the number of primary school aged children in the province of mother's birth (in 1995) as well as interactions between the mother's year of birth dummies and enrollment rates in the province of mother's birth (in 1995). Instrumental variables are discrete treatment dummy in models (2), (5), (8), and (11), and mother's year of birth dummies for those exposed to the CSL in models (3), (6), (9), and (12). Models (1)-(3) and (7)-(9) contain 1,616, 1,519, 1,177, and 1,243 observations, and models (4)-(6) and (10)-(12) contain 891, 836, 643, and 707 observations for VLBW, length of stay, HAZ, and WAZ outcomes, respectively. Health utilization models contain 1,677 for the sample of females ages 18-29 and 927 observations for the sample of females ages 18-25.

Appendix B1. The Effect of the CSL on Outcome Variables (DID)

	Dependent Variable				
	VLBW	Length of facility stay	HAZ	WAZ	Month of first prenatal care
	(1)	(2)	(3)	(4)	(5)
Panel A: Women Aged 7-10 to 11-14 in 1997					
Treatment dummy for women ages 7 to 10 in 1997	-0.076**	-0.777**	0.608***	0.407*	-1.316***
	(0.037)	(0.320)	(0.203)	(0.229)	(0.481)
Observations	891	836	643	707	927
Panel B: Women Aged 11-14 to 15-18 in 1997					
Treatment dummy for women ages 11 to 14 in 1997	-0.002	-0.034	-0.017	-0.015	-0.042
	(0.022)	(0.200)	(0.142)	(0.120)	(0.309)
Observations	1363	1280	995	1063	1416

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: Standard errors (reported in parentheses) are adjusted for clustering on the province of mother's birth. All models include individual- and community-level variables, and interactions between the mother's year of birth dummies and the number of primary school aged children in the province of mother's birth (in 1995) as well as interactions between the mother's year of birth dummies and enrollment rates in the province of mother's birth (in 1995). The DID estimate can be simply determined by subtracting the estimate in Panel B from the estimate in Panel A.

Appendix B2. The Effect of the CSL on Education and Outcome Variables (RDD)

	Dependent Variable					
	Primary School Completion	VLBW	Length of health facility stay	HAZ	WAZ	Month of first prenatal care
	(1)	(3)	(2)	(4)	(5)	(6)
Reduced-form Estimate (No Controls)	0.232***	-0.072**	-0.654**	0.604***	0.523***	-0.725**
	(0.052)	(0.029)	(0.277)	(0.194)	(0.151)	(0.351)
Reduced-form Estimate (Controls Included)	0.215***	-0.076***	-0.696**	0.617***	0.549**	-0.683**
	(0.058)	(0.029)	(0.324)	(0.201)	(0.215)	(0.282)
Observations	1025	986	924	736	803	1025

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: Standard errors (reported in parentheses) are adjusted for clustering on month-of-birth cohort (relative to September 1986). First Row shows reduced-form estimates from local linear regressions (which include a linear function of month of birth relative to cutoff and a dummy for being born after the cutoff) without controls, while Second Row shows results with controls included in the paper as well as calendar month-of-birth fixed effects. The bandwidth is 51 months (4.25 years) on either side of September 1986.

Appendix C. Effects of Maternal Education on Infant and Child Health, and Health Care Utilization

	OLS 18-29 (1)	IV (T) 18-29 (2)	IV (YOB) 18-29 (3)	OLS 18-25 (4)	IV (T) 18-25 (5)	IV (YOB) 18-25 (6)
Panel A: Health Outcomes						
Very low birth weight	-0.034***	-0.177*	-0.174*	-0.034**	-0.187*	-0.177*
	(0.010)	(0.106)	(0.103)	(0.015)	(0.107)	(0.104)
Length of health facility stay	0.103	-1.653*	-1.568*	-0.023	-2.262*	-2.005*
	(0.148)	(0.977)	(0.942)	(0.239)	(1.355)	(1.213)
Height-for-age z-score	0.262**	1.088**	1.068*	0.411**	1.448*	1.443*
	(0.105)	(0.629)	(0.628)	(0.165)	(0.827)	(0.826)
Weight-for-age z-score	0.156**	1.024*	1.010*	0.202*	1.171**	1.230*
	(0.072)	(0.543)	(0.531)	(0.120)	(0.575)	(0.740)
Panel B: Health Utilization Outcomes						
Formal prenatal care	0.033**	0.029	0.027	0.033*	0.024	0.020
	(0.014)	(0.137)	(0.137)	(0.019)	(0.163)	(0.165)
Receipt of 4+ prenatal visits	0.100***	0.044	0.031	0.076*	0.111	0.095
	(0.024)	(0.263)	(0.266)	(0.039)	(0.207)	(0.209)
Prenatal care initiation	-0.868***	-1.634*	-1.559*	-0.878***	-1.768*	-1.623*
	(0.144)	(0.949)	(0.935)	(0.145)	(0.961)	(0.977)
Delivery in a health facility	0.001	0.117	0.055	-0.011	0.077	0.139
	(0.017)	(0.358)	(0.312)	(0.027)	(0.298)	(0.326)
Delivery by health professionals	0.000	0.047	0.050	0.001	0.178	0.175
	(0.015)	(0.097)	(0.095)	(0.015)	(0.118)	(0.116)
Observations	1,677	1,677	1,677	927	927	927

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: Standard errors (reported in parentheses) are adjusted for clustering on the province of mother's birth. All models include individual- and community-level variables, province fixed effects, cohort of birth fixed effects, and interactions between the mother's year of birth dummies and the number of primary school aged children in the province of mother's birth (in 1995) as well as interactions between the mother's year of birth dummies and enrollment rates in the province of mother's birth (in 1995). Instrumental variables are discrete treatment dummy interacted with the intensity measure in models (2) and (5), and dummies for exposed mother's age in 1997 interacted with the intensity measure in models (3) and (6). Models (1)-(3) contain 1,616, 1,519, 1,177, and 1,243 observations, and models (4)-(6) contain 891, 836, 643, and 707 observations for VLBW, length of stay, HAZ, and WAZ outcomes, respectively. Health utilization models (1)-(3) contain 1,677, and (4)-(6) contain 927 observations.

Appendix D. Effect of Maternal Education on Different Measures of Birth Weight

	Goodness-of-Fit of the OLS Regression (R-square Statistics)			
Dependent Variable:	VLBW	Log (Birth Weight)	Birth Weight	Low Birth Weight
Primary School Completion	0.1768	0.1612	0.1425	0.1195

	Effects of Maternal Education				
	All births		First births		
	OLS 18-29 (1)	IV 18-29 (2)	OLS 18-29 (3)	IV 18-29 (4)	IV 18-29 (5)
VLBW (<1500)	-0.039***	-0.167*	-0.017*	-0.068*	-0.095*
	(0.009)	(0.098)	(0.010)	(0.041)	(0.055)
Log (Birth weight)	0.072***	0.221*	0.060***	0.147*	0.193*
	(0.015)	(0.130)	(0.015)	(0.086)	(0.105)
Birth Weight	166.635***	440.948	152.988***	391.557*	559.670*
	(37.571)	(302.017)	(38.035)	(228.527)	(287.135)
Low Birth Weight (<2500)	-0.067***	-0.106	-0.091***	-0.087	-0.119
	(0.019)	(0.155)	(0.025)	(0.133)	(0.148)
Sample Selection Correction	No	No	No	No	Yes
Observations	1616	1616	711	711	711

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: Standard errors (reported in parentheses) are adjusted for clustering on the province of mother's birth. All models include individual- and community-level variables, and interactions between the mother's year of birth dummies and the number of primary school aged children in the province of mother's birth (in 1995) as well as interactions between the mother's year of birth dummies and enrollment rates in the province of mother's birth (in 1995). Instrumental variables are mother's year of birth dummies for those exposed to the CSL.

Appendix E. Effect of Maternal Education with Heterogeneous Treatment Effects

	IV 18-29 (1)	IV 18-29 (2)	IV 18-29 (3)	IV 18-29 (4)	IV 18-29 (5)
	Instrument: Treatment	Instrument: Treatment	Instrument: Treatment	Instrument: Treatment*ME	Instrument: Treatment*FE
Panel A: Health Outcomes					
Very low birth weight	-0.166*	-0.171*	-0.171*	-0.150*	-0.154*
	(0.097)	(0.101)	(0.100)	(0.091)	(0.090)
Length of health facility stay	-1.788**	-1.955**	-2.069**	-1.632*	-2.145**
	(0.844)	(0.965)	(0.847)	(0.844)	(0.868)
Height-for-age z-score	1.231**	1.232*	1.412*	1.454**	1.215**
	(0.606)	(0.680)	(0.640)	(0.593)	(0.549)
Weight-for-age z-score	0.902*	0.961*	0.788*	1.240**	0.645*
	(0.479)	(0.530)	(0.475)	(0.546)	(0.391)
Panel B: Health Utilization Outcomes					
Formal prenatal care	0.090	0.077	0.097	0.101	0.092
	(0.105)	(0.118)	(0.101)	(0.094)	(0.093)
Receipt of 4+ prenatal visits	0.092	0.105	0.060	0.060	0.122
	(0.159)	(0.174)	(0.158)	(0.148)	(0.143)
Prenatal care initiation	-1.603*	-1.585*	-1.438*	-1.698**	-2.018**
	(0.850)	(0.905)	(0.849)	(0.849)	(0.870)
Delivery in a health facility	0.015	0.008	0.005	0.050	0.020
	(0.091)	(0.101)	(0.109)	(0.058)	(0.078)
Delivery by health professionals	0.030	0.034	0.000	0.092	0.058
	(0.082)	(0.090)	(0.082)	(0.061)	(0.072)
Controls:					
Mother's Education Dummies	No	Yes	No	Yes	No
Father's Education Dummies	No	No	Yes	No	Yes
Observations	1,677	1,664	1,598	1,664	1,598

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: Standard errors (reported in parentheses) are adjusted for clustering on the province of mother's birth. Observation numbers change due to missing values. Instruments in column (4) are treatment dummy and the treatment dummy interacted with mother's education dummies, and instruments in column (5) are treatment dummy and the treatment dummy interacted with father's education dummies. See columns 2-3 of Table 7 for the first-stage estimations.

Appendix F. The Effect of the CSL on Different Levels of Education

I estimate the equation below to check if the CSL succeeded in affecting mainly the targeted groups.

$$E_{im} = \mu + G_i\theta_t + T_i\delta_m + \varepsilon_i \tag{11}$$

where E_{im} is a schooling variable which takes value 1 if mother i completed m level of education, 0 otherwise. μ is a constant, θ_t accounts for group time fixed effects (7-10; 11-14; 15-18 in 1997), and T_i is the treatment dummy. δ_m is the estimated impact of the CSL for 4 different levels of education (m): primary (5 years of education), secondary (8 years of education), high (11 years of education), and advanced (12+ years of education). Figure below presents the estimated probabilities from LPM with corresponding 95% confidence intervals.

The effect of the CSL is the highest for the probability of completing 8 years of education, at the level of the goal of the education policy. There is a small effect of the CSL on the probability of completing high school, and a negligible effect on the probability of completing advanced education. Despite the small spillovers, the program substantially increased schooling through the level of education associated with the CSL.

Figure for Probability of Completing at Least "M" Level of Education

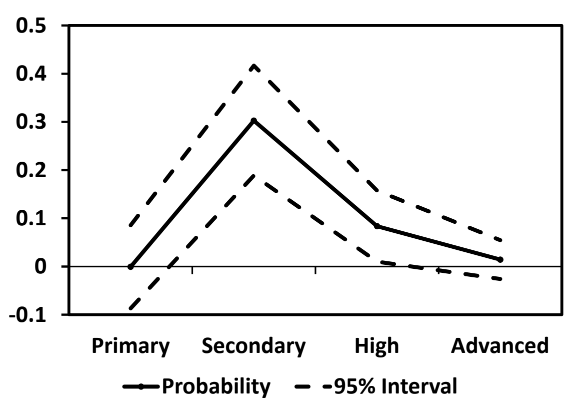


Figure A.1: Primary School Completion

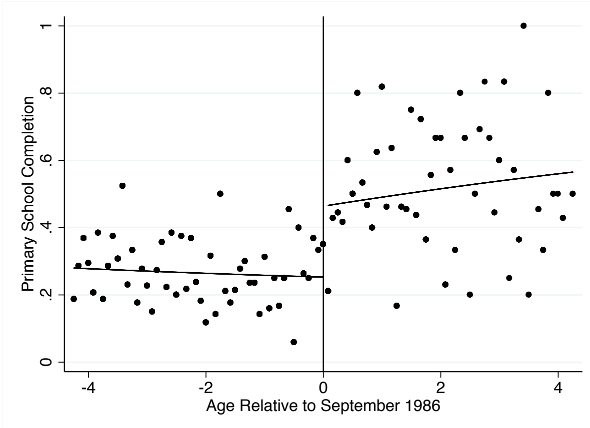


Figure A.2: Height-for-age Z-score

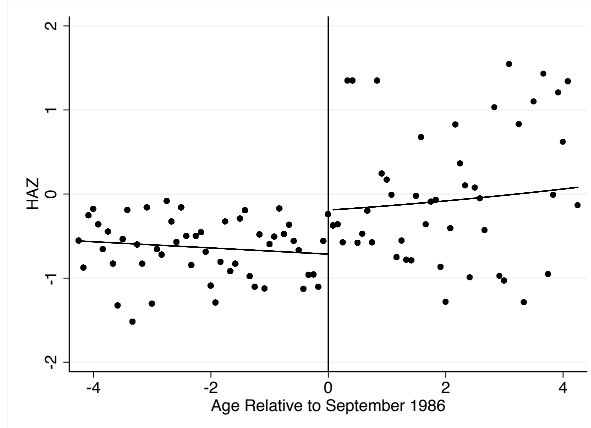
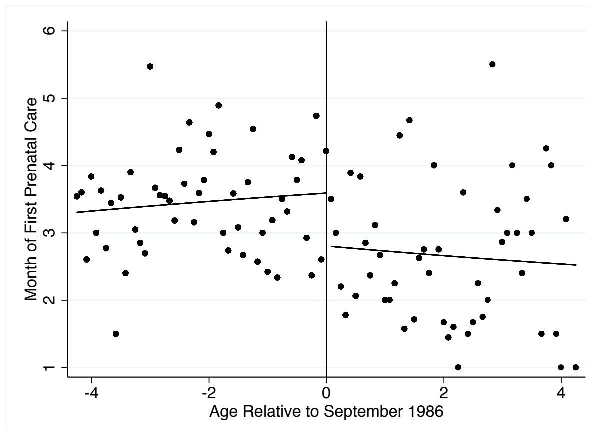


Figure A.3: Month of First Prenatal Care



Notes: Points represent the mean of primary school completion (8+), HAZ of the child, and month of first prenatal care for each month-of-birth cell within 51 months (4.25 years) on either side of September 1986 (solid vertical line). Solid line is a local linear smoother.

Chapter 3: The Impact of Female Education on Fertility: Evidence from Turkey

1 Introduction

Fertility and education are key factors in advancing development and reducing poverty. The reduced fertility typical of demographic transitions has been shown to confer a number of benefits (Schultz, 2008). Examples include benefits to the mother, such as improved health, greater investment in human capital, and greater labor supply; as well as benefits to the child, such as improved nutrition, greater investment in schooling, and various improved health outcomes. As a consequence, an array of policy interventions has been adopted with the aim of accelerating the demographic transition and, thus, bringing these benefits earlier than they would otherwise occur. More generally, development economists have emphasized the role of female education in fertility, health, and human capital formation, and investment in female education has been adopted as a development tool in many developing countries (Schultz, 1993). However, a thorough understanding of the causal relationship between fertility and female education has lagged behind the enthusiasm for educational interventions as a tool to reduce fertility.

This paper uses exposure to a nationwide reform of compulsory education system in 1997 in Turkey, which extended the basic educational requirement from five to eight years (free of charge in public schools) as an instrumental variable. The main objectives of the compulsory schooling law (CSL) were to increase the education level to universal standards and to provide assistance to ensure that all children can comply. In order to accommodate the increased number of primary school students, additional classes and schools were constructed, new teachers were recruited, and transportation was arranged for children living in rural areas, who are often far away from existing schools. The CSL led to a significant increase in the number of students in primary school between the 1996/97 and the 2000/01

Academic Years, by around 21% from 8.65 million to 10.48 million.[1] The CSL provides an ideal natural experiment, providing an instrument that varied the number of years of schooling, without significant curriculum changes, to identify the impact of schooling on fertility.[2]

This paper demonstrates that the CSL had a significant immediate effect on female education, thereby, providing a useful instrument for evaluating the impact of schooling on fertility. Using exposure to the CSL by year of birth as an instrumental variable, I show that an extra year of female schooling reduces teenage fertility by around 0.03 births, which is a reduction of 33%. Further, the results are robust with respect to a rich set of controls. The effect is higher than the corresponding effect estimated by ordinary least squares (OLS), which suggests about a 17% reduction in teenage births. This difference may be a consequence of education impacting fertility greater for a subsample of women whose educational attainment has been affected by the CSL. Exploring heterogeneous effects indicates that female education reduces teenage fertility more in provinces with higher initial fertility and lower population density. Moreover, the effect of the CSL on education depends on several characteristics of birth-province: initial levels of education, income, urbanization, and population density. I also find that the effect of the educational policy operates through a delay in marriage, which in turn postpones childbearing.

The existing literature documents strong associations between education and child health and fertility, even after controlling for family and community background variables. (For a survey of the literature, see Strauss and Thomas, 1995.) However, the observed associations do not imply causality. Omitted variables (Berger and Leigh, 1989)–in particular ability (Griliches, 1977) and discount rates (Fuchs, 1982), which are highly correlated with both education and fertility decisions–may bias the relationship between education and fertility. Thus, studies treating education levels as exogenous fall short of answering the question of whether there is a causal relationship between education and fertility.

[1]For educational statistics, see http://sgb.meb.gov.tr.

[2]I discuss the features of the CSL in Section 2.

While there is a growing literature examining the causal effect of education on non-market outcomes, there are surprisingly few studies examining the effect of education on fertility in developing countries. Part of the reason for this is difficulty in identifying natural experiments. Breierova and Duflo (2004) examine the effect of a primary school construction program in Indonesia (INPRES) on child mortality and fertility, using the number of schools constructed in the region of birth and exposure across age cohorts induced by the timing of the program as instrumental variables. Their results suggest that the average parental education is an important determinant of very early fertility (before the age of 15), whereas it is unimportant for early fertility (before the age of 25). Similarly, Osili and Long (2008) estimate the effect of female schooling on early fertility (before the age of 25) by exploiting regional and age differences in the exposure to an Universal Primary Education (UPE) program implemented for the non-Western regions of Nigeria. Using the state classroom construction funds per capita as a measure of program intensity, their results suggest that female education reduces the number of early births.

This paper follows recent studies using instrumental variables (IV) and adds to the existing literature in several respects.[3] First, previous studies have investigated the impact of female education on fertility at markedly different points in the demographic transition; however, the role of policies is highly dependent on the stage of demographic transition. The case of Turkey provides an interesting case study because the policy intervention occurred at a later transition stage when the pace of fertility decline had slowed (starting from the early 1990s), which makes further reductions in fertility more difficult to achieve. Second, previous studies use variation in only the supply of education (school and classroom construction and free public provision), whereas the CSL entailed variation in both the supply of and demand for education. Third, this study explores heterogeneous effects across

[3]Recent studies have used similar strategies to uncover the causal effect of schooling on different outcomes of nonmarket returns: for adult health, see van Kippersluis, 2011; Kenkel et al., 2006; de Walque, 2007; Webbink et al., 2010; Grimard and Parent, 2007; Lleras-Muney, 2005; Silles, 2009; Clark and Royer, 2010; Kemptner et al., 2011; Albouy and Lequien, 2009; for child health, see Currie and Moretti, 2003; Lindeboom et al., 2009; McCrary and Royer, 2011.

various dimensions, including pre-change levels of initial fertility and education, population density, urbanization, agricultural activity, and income in the province of birth as well as parental education and ethnicity. This understanding is crucial for understanding the effect of educational reforms on fertility as these dimensions vary significantly within developing countries. Finally, this study discusses several channels through which education affects fertility.

This paper focuses on early fertility for a number of reasons. First, early fertility is an important precursor in total fertility. For instance, Osili and Long (2008) show that variation in fertility before age 25 explains 58 percent of the variation in completed fertility in Nigeria. Similarly, in Turkey, I find that births before age 18 and completed fertility are highly correlated (0.62) and, importantly, 20% of first births occurred before age 18 for women who completed their fertility.[4] Thus, the impact of female education, which has been the emphasis of several recent educational reforms in developing countries, can be accessed without waiting for several decades of time to lapse. Second, education is a key determinant of early childbearing, thus exploring the impact of education on early fertility is of particular interest. Finally, teenage childbearing, in particular, adversely affects social and economic well-being for both the mother and her children. Therefore, uncovering the effects of early fertility uncovers the effects of fertility in general, and the particular effects associated with early childbearing in particular.

This paper has several important implications for development policy, especially for countries experiencing slowed pace of fertility declines. While Turkey roughly followed a similar demographic transitional history as other developing countries over the second half of the 20th century, Turkey has recently exhibited a marked divergence in two particular areas. Firstly, Turkey recently exhibits the low-fertility patterns (postponed marriage and first births and lower fertility rates) of a developed country. Second, the gap in female education levels between urban and rural areas has narrowed dramatically.[5] The CSL was success-

[4]Author's calculation, using the 2008 Turkish Demographic Health Survey.

[5]The difference in the median number of years of education is 0.3 for ever-married women ages 15-49 in

ful at increasing female enrollment rates in both rural and urban areas (rural enrollment in grade six increased significantly in the first year of the change in the law, roughly 162%[6]) and, partially as a consequence, the tempo effects (postponed marriage and delayed motherhood) accelerated the fertility transition. Thus, this paper contributes to the literature by providing causal evidence that educational interventions can accelerate the demographic transitions at later stages.

The remainder of this paper is organized as follows: Section 2 provides background on fertility, development, and the educational policy in Turkey; Section 3 describes the data and the identification strategy; Section 4 presents the empirical strategy and the results; and Section 5 concludes.

2 Background

In this section, I discuss the conceptual framework, present the trends in fertility prior to the change in the CSL, discuss the background on education system in Turkey, and describe the CSL and the trends in education. The CSL increased compulsory schooling from five to eight years (free of charge in public schools) in 1997, with the objective of increasing education level to universal standards. However, it also had an effect on early fertility.

2.1 Conceptual Framework

Economic theory provides several mechanisms through which education may influence fertility choices. One explanation is that education increases the returns to labor market participation, thereby, increasing the opportunity cost of time-intensive activities (Becker, 1981; Schultz, 1981). As a result, women might substitute time-intensive activities, such as childbearing and child rearing, in order to devote more time to the labor market participation. Therefore, education might result in fewer children for women. Also, education

2008: http://www.hips.hacettepe.edu.tr.

[6]http://www.unicef.org/turkey/gr/ge21ja.html.

may affect fertility preferences–for instance, more educated women may prefer fewer but healthier (higher quality) children (Becker and Lewis, 1973). Improvements in child health resulting from female education may also reduce child mortality, thereby, lowering fertility since fewer births are required to achieve the same family size (Lam and Duryea, 1999; Schultz, 1993). Education may reduce fertility through increased knowledge about contraceptives and the effective use of contraceptive methods (Rosenzweig and Schultz, 1985, 1989). Lastly, education may increase women's autonomy and bargaining power in the household, thereby, increasing women's participation in fertility decision-making (Mason, 1986). In addition, staying in school longer might postpone childbearing if having children impedes upon attending school.

Economic theory points to a number of mechanisms in which education influences fertility; however, according to the demography literature, the relevance of these mechanisms is highly dependent on a country's stage of demographic transition. Changes in fertility behavior, including the adoption of birth control methods and preferences for smaller family size, caused by the spread of new ideas and information through mass media, family planning programs, etc., account for changes in the decline of the fertility rate in the early phase of the transition. However, as a country approaches the later stages of the transition, fertility becomes more closely tied to the level of socioeconomic development (Bongaarts, 2002). Further fertility declines, therefore, depend on improvements in socioeconomic conditions, particularly female education and child survival (Caldwell, 1980; Sen, 1999; Bongaarts, 2001). Therefore, increases in female education in demographic transitions may be linked to fertility declines at later stages. Many developing countries have experienced rapid fertility declines since the 1960s; however, the pace of decline slowed in a number of countries, including Turkey in the 1990s (Bongaarts, 2006). Causal evidence that policies can influence fertility at later stages, however, is absent. This paper therefore contributes to the literature by exploring the role of education in fertility declines at later stages of the transition.

2.2 Trends in fertility and development in Turkey before the CSL

Similar to many developing countries, Turkey has experienced a rapid fertility decline since the early 1960s. In 1980, Turkey implemented export-oriented policies, which increased the demand for labor in the service and industrial sectors. As a result, migration from villages to cities increased, leading to rapid urbanization and industrialization, which had a profound effect on fertility. The total fertility rate (TFR) exceeded 6 children per woman in the early-1960s, dropped to 5 in the late-1970s, and dropped further to around 3 in the late-1980s.[7] In the 1990s, however, fertility remained at around 2.6 births per woman (Figure 1).

Proximate determinants of fertility, particularly contraceptive use and marriage, are possible factors explaining the trend in fertility. Turkey's fertility decline started with a family planning program introduced in 1965, which legalized the sale and use of contraceptives. Moreover, a new Population Planning Law in 1983 legalized induced abortions upon request for up to ten weeks gestation and allowed trained nurses and midwives to administer IUDs, which increased the prevalence of modern contraceptives and as a result subsequently decreased induced abortions. During the 1990s, Turkey experienced a leveling off in contraceptive prevalence around 60%, which coincided with a period of slowed fertility decline.[8] The traditional marriage pattern of Turkey is characterized by the universality of marriage: almost all women engage in either civil or religious marriages by the end of their reproductive ages and childbearing out of wedlock is uncommon in Turkey.[9] Hence, age at first birth depends on marriage age, which in turn affects the overall fertility. Since the time interval between marriage and first birth has been stable with an average around 1.6 years in Turkey, a delay in age at first marriage may result in an overall fertility decline by postponing first births. The singulate mean age at marriage[10] for both sexes did

[7] TFR is defined as the average number of children that would be born to a woman by the end of her childbearing period if she were to experience the exact current age-specific fertility rates.

[8] 2008 Turkey Demographic and Health Survey: http://www.hips.hacettepe.edu.tr.

[9] Ibid.

[10] It is defined as the average number of years lived as single (never-married) for females or males before

not change (22 for females and 25 for males) over a period of 13 years from 1985 to 1998 (Figure 1). Thus, a delay in age at first marriage can play a crucial role in reducing fertility.

Even though there is not a consensus on the effects of socioeconomic factors on fertility, it is useful to look at changes in socioeconomic indicators in order to understand fertility trends, especially in the later stages of the transition.[11] The Gross Domestic Product (GDP) per capita almost doubled from 1980 to 1990; however, economic recessions were frequent during the 1990s (4 major crises took place in 1991, 1994, 1999, and 2001). Following the export-oriented reforms in the 1980s, the share of the population in urban areas rose from 44% in 1980 to 59% in 1990, but did not significantly increase during the 1990s (reaching 65% in early 2000). The labor force in the agricultural sector decreased from 60% in the 1980s to 47% in 1990 and to 45% in 1995. From 1990 to 1995, life expectancy at birth increased from 65 to 68 and from 61 to 64 years for females and males, respectively. Despite improvements in infant and children under five mortality rates in the 1980s, both exceeded 55 per thousand live births during the first half of the 1990s, which is very high compared to developed countries. Adult literacy rates (the proportion of the adult population aged 15+ which is literate) leveled off around 70 for females and 90 for males during the 1990s prior to the CSL. Hence, the CSL took place when many development indicators either leveled off or showed very little progress.

2.3 Education and Compulsory Schooling Law in Turkey

2.3.1 Turkey's Education System

After the establishment of the Republic of Turkey in 1923, radical curricular and structural educational reforms were carried out. One major reform was the Unification of Education Law, which unified schools under a common curriculum.[12] Further reforms were the centralization of the system and organization of the decision-making authority, the Ministry

they get married for the first time among those who marry before age 50.

[11] For statistics related to socioeconomic indicators, http://www.turkstat.gov.tr.

[12] the Unification of Education Law no 430 issued on 03.03.1924.

of National Education (MONE). MONE directs all educational related policy decisions, prepares the curriculum of educational institutions, and monitors implementation in cooperation with provincial offices.

Formal education in Turkey consists of pre-school, primary, secondary and higher education. Pre-primary education is offered to children up to age 6 on a voluntary basis before compulsory primary education. Prior to the law change in 1997, five years of primary education was compulsory for all citizens. In 1997, compulsory primary education was increased from five to eight years. Following primary school, students may choose to attend one of the following secondary educational programs: general, vocational or technical high schools. The basic 8-year primary education level (public and private) gross enrollment rate is 107.58% in 32,797 schools with 503,328 teachers (Ministry of National Education, 2011).[13] As of 2010, there are more than 4.5 million students in secondary schools with a 93.34% gross enrollment rate.

2.3.2 Compulsory Schooling Law and Trends in Education

In 1997, the Turkish government took a "big bang" approach to education reform, increasing compulsory schooling from five to eight years.[14] The main objective of the 8-Year Basic Education Program was to increase the education level to universal standards. In order to encourage compliance with the law, a new Primary School Diploma is awarded for only those completing the 8th grade.

Education is provided free of charge in public schools. The 8-Year Basic Education Program included construction of schools and classes and recruiting new primary school teachers in order to accommodate a greater number of students. The Program aimed at providing opportunities for all children to stay in school at least to the eighth grade. Thus, low-income students were provided free textbooks, school meals, and student uniforms.

[13] MONE calculates the gross enrollment rate by dividing the total number of students in a specific level of education by the population in the theoretical age group.

[14] Compulsory education was extended to 8 years with the Basic Education Law No 4306 dated 18.08.1997 as of the 1997/1998 Academic Year.

Moreover, transportation expenses to children living at least 2.5 km away from nearby village schools were covered under the Bussed Primary Education Scheme launched in the 1989/1990 Academic Year in order to improve access for children in rural areas, especially for those in poor families. In addition, regional boarding primary schools (YIBO) were established to provide primary education services to settlements having no schools.[15]

While the Program aimed to extend educational opportunities to a greater share of the population, the qualitative components of the education system in general and the design of the curriculum in particular stayed the same. In an in-depth case study prepared for the World Bank on the implementation of the 1997 Basic Education Law, Dulger (2004) attests that the Primary Education Program maintained the 1968 national curriculum with minor changes and that, due to time constraints in implementation, the MONE primarily focused on capacity issues to accommodate new students. Moreover, a 2007 OECD educational report emphasizes that the 1997 educational program in Turkey lacked implementation of a new curricula in order to improve the quality of the education system.[16]

The CSL had an impressive effect on enrollment rates of both sexes, especially on female enrollments in rural areas. The net primary enrollment rate increased from 84.74 in the 1997/1998 Academic Year to 93.54 in the 1999/2000 Academic Year (Ministry of National Education, 2011).[17] The increase in the net primary enrollment rate was greater for females than for males: 90.25 to 98.41 for males and 78.97 to 88.45 for females. The sex ratio in primary education rose from 85.63 to 88.54.[18] Presently, the net primary enrollment rate is 98.41 for both sexes, 98.59 for boys and 98.22 for girls, and the sex ratio is 100.42. Over ten million children in all types of education institutions, including YIBOs receive 8 years of basic education in about 33,000 schools with approximately 503,000 teachers.

[15]According to MONE, there were 687,056 children bussed to the primary schools, and 539 YIBO with 247,563 students in the 2010/2011 Academic Year.

[16]Reviews of National Policies for Education: Basic Education in Turkey: http://www.oecd.org.

[17]The net primary enrollment rate is calculated by dividing the number of students of a theoretical age group enrolled in a specific level of education by the population in that age group.

[18]Sex Ratio indicates the relative greatness of female gross enrollment ratio as compared to male gross enrollment ratio in a specific educational year and level of education. It is calculated by dividing the female gross enrollment ratio by the male gross enrollment ratio multiplied 100.

Figure 2 shows the trend in the number of students in basic education by academic year. The figure demonstrates that the CSL in 1997 had a significant effect on student primary school participation. Enrollments increased by around 15% from 9.08 million in the 1997/98 Academic Year to 10.48 million in the 2000/01 Academic Year.

3 Data and Identification Strategy

3.1 Data

The analyses are based on the 2008 Turkish Demographic and Health Survey (TDHS-2008). The TDHS survey is a demographic health survey conducted every five years by Hacettepe University Institute of Population Studies (HUIPS) since 1993. Of the 11,911 households surveyed in 2008, 10,525 were successfully interviewed, which yields an approximate response rate of 88%. Among 10,525 interviewed households, 8,003 women were ever-married of reproductive ages at 15-49. The response rate for the ever-married sample is approximately 92%.

The TDHS-2008 uses two types of questionnaires, the Household Questionnaire and the Individual Questionnaire. The latter targets ever-married women of reproductive ages (15-49), whereas the former targets all usual members of and visitors to the household. The nationally representative survey provides information on socioeconomic and demographic characteristics of a large number of women and contains a detailed fertility history of women surveyed with the Individual Questionnaire. The TDHS-2008 data is appropriate for the purpose of this paper since it contains data on the education, year and province of birth, fertility and marriage history of women. Table 1 presents summary statistics for women in the sample of analysis, which are between the ages of 18 and 30.[19] The data include 4,684 women, of which 2,185 have children.[20] The average level of completed

[19]The basis of sample selection is discussed under the identification section.

[20]The TDHS-2008 contains information on whether a pregnancy has ever ended in a miscarriage, induced abortion, or a still birth; however, it does not provide the year of pregnancy termination, except for the latest

schooling is 6.84. The average fertility (the number of ever-born children) is 0.94 for the entire sample, 0.09 for fertility before the age of 18, 0.04 for fertility before the age of 17, and 0.02 for fertility before the age of 16.

Other data sources are the National Education Statistics books by MONE, Turkey's Statistical Year Books and detailed education data by Turkish Statistics Institute (TurkStat). The sources contain detailed information on enrollment rates in formal and non-formal education for both sexes, number of teachers, number of schools and classes for different age groups in all provinces and for all academic years. I use mid-year population projections provided by TurkStat for the number of primary school age children in the province of birth in 1995 (80 provinces).[21]

3.2 Identification Strategy: Effect of the Compulsory Schooling Law on Education and Fertility

Exposure to the CSL is determined by year of birth. Children aged 12 or older in 1997, when the CSL took place, had already graduated from primary school in 1997 since most Turkish children attend primary school between the ages of 7 and 11, and therefore are "unexposed" to the policy.[22] On the other hand, children aged 11 or younger in 1997 would be affected by the CSL and therefore are "exposed" to the policy. Because I use the 2008 TDHS data set, the children exposed to the education policy were between the ages of 18 and 22 at the time of the survey, while the unexposed were aged 23 and over. The exposed cohort is a young group at the time of the 2008 TDHS survey, thereby, limiting the analysis to teenage fertility indicators rather than completed fertility. I use the number

pregnancy. Thus, I cannot determine whether or not the pregnancy was ended as a teenager. However, the results remain robust to the exclusion of such cases (653 women out of 4,684 for the sample of women ages 18-30, and 157 women out of 2,993 for the sample of women ages 18-25).

[21] Turkey is divided into 5 main regions: West, South, Central, North, and East. However, a new regional breakdown has been adopted from the European Union for statistical purposes as of 2002. Accordingly, there are 12 regions (NUTS I) with 81 provinces (Figure 3).

[22] It is possible that some of these females might have been exposed to the policy because of grade repetition. However, there was not a high prevalence of them, and, moreover, results are robust to excluding these females.

of children born before age 18 as a key dependent variable due to the fact that the fertility history of the exposed cohort is censored.

Figure 4 illustrates the effect of the CSL on female schooling for each cohort. The vertical solid line indicates the timing of the education policy, where cohorts to the right of the vertical line (females aged 12 in 1997) are exposed to the law. Each point on the solid line represents the average education for each cohort. The pattern is consistent with the hypothesis that the change in compulsory schooling had no impact on the education of unexposed cohorts and had a positive effect on the education of the cohorts 11 and younger. Thus, the figure shows that the 1997 law appears to have a substantial effect on female schooling. Moreover, there is no clear trend in female schooling prior to the CSL (left side of the vertical line).

Figure 4 also shows the percentage of mothers for each cohort, where the percentage of mothers is the percentage of women with at least 1 ever-born child before age 18 and 17 ("Before 18" and "Before 17"). The figure shows that there are no particular pre-existing trends in the percentage of teenage mothers prior to the education policy (left side of the vertical line) and that the percentage of teenage mothers declined following the CSL (right side of the vertical line).

Formal analysis of the effects of the CSL on female education and teenage fertility are presented in the following section.

4 Empirical Methodology and Results

In this section, I first explore the reduced form effect of the CSL on female education using a number of different specifications and provide evidence for the identification strategy. Second, I estimate the effect of female education on teenage fertility using both OLS and IV, as well as an alternative approach (difference-in-difference with the CSL as a treatment variable). Third, I explore whether there are heterogeneous effects, for both the effect of the CSL on female education and fertility and the impact of female education on fertility. Further, I employ robustness checks using various intensity indicators. Lastly, I discuss several channels through which education influences fertility.

4.1 Effect of the CSL on Education

4.1.1 Reduced form

Identification follows from the generalized regression framework:

$$E_{ijk} = a + \alpha_{1j} + \sum_{l=7}^{18}(d_{il})\gamma_{1l} + \sum_{l=7}^{18}(C_j \times d_{il})\delta_{1l} + \varepsilon_{ijk} \tag{1}$$

where E_{ijk} is the education of an individual i, born in province j, in year k. As in Duflo (2001), d_{il} is a dummy that indicates whether individual i is age l in 1997 (a year-of-birth dummy), a is a constant, α_{1j} is a province-of-birth fixed effect, and C_j is a vector of province-specific variables–the number of primary school aged children and the enrollment rate in the province of birth (in 1995). Females aged 19 in 1997 is the control group and hence this dummy is omitted from the regression. In all specifications, including the above, I correct the standard errors for clustering at the province level.

In these unrestricted estimates, each parameter of interest, γ_{1l}, estimates the impact of the CSL on a given cohort. Children aged 12 and older in 1997 were unlikely to have been affected by the change in compulsory schooling. Thus, I expect the coefficients of interest would be zero for females aged greater than 11 in 1997. Specifically, I can test this restriction by $\gamma_{1l} = 0$ for $l > 11$. Exposure to the CSL implies the coefficients γ_{1l} should be increasing from l=11 to 7 and be significantly positive. The year of birth coefficients (γ_{1l} for each l) from the regression results of equation (1) with no controls are jointly significant for l=7 to 11, and insignificant for l=12 to 18 (The F-statistics are 34.97 (p=0.000) and 1.01 (p=0.419) respectively).[23] These results validate the identification strategy and indicate that

[23] See the results for this unrestricted specification in Appendix B.

the CSL had an effect on female education.

Furthermore, the following equation is estimated to test whether there are pre-existing trends in education:

$$E_i = a_1 + T_i\delta_1 + (UT_i \times YOB_i)\beta + X_i\theta + \varepsilon_i \tag{2}$$

where E_i is the education of an individual i, a_1 is a constant, T_i is a dummy indicating whether individual i belongs to the "treated" age group (exposed), UT_i is a dummy indicating whether individual i belongs to the "untreated" age group (unexposed), and YOB_i is the year of birth of an individual i. X_i is a vector of province-specific control variables, including province of birth fixed effects, interactions between the number of primary school aged children in the province of birth (in 1995) and year of birth dummies, and interactions between the enrollment rate in the province of birth (in 1995) and year of birth dummies. If there are pre-existing trends in education, β would be significantly different than zero. The regression results of equation (2) for a sample of women ages 18-30 suggest that the CSL increased female education by 2.01 years and rejects that pre-existing trends are present (β is estimated to be very small (0.066) and statistically indistinguishable from zero).[24] Thus, the results provide further evidence that the differences in education are not driven by pre-existing time trends.

4.1.2 First-Stage Estimation

To gain precision, I follow Duflo, 2001 and impose the restriction that coefficients are zero for the unexposed (age greater than 11 ($\gamma_{1l} = 0$ for $l > 11$)). This is justified because, as demonstrated in the previous section, the CSL did not affect the cohorts unexposed to it. The following estimates the impact of the law as follows:

[24] Estimation of equation (2) for a sample of women ages 18-25 also yields similar results: β is 0.005 and statistically insignificant at any conventional significance level.

$$E_{ijk} = a + \alpha_{1j} + \sum_{l=7}^{11}(d_{il})\gamma_{1l} + \sum_{l=7}^{11}(C_j \times d_{il})\delta_{1l} + \varepsilon_{ijk} \tag{3}$$

The reference group is now the women aged 12 to 19 in 1997.

The results for different specifications starting with the baseline model of no controls (column (1)) are presented in Table 2. Column (2) controls for only the province of birth to account for time-invariant, unobserved differences across provinces.[25] Column (3) adds controls for the interaction of a year of birth dummy and the primary school aged population in the province of birth in 1995. It is possible that the effect of the CSL on education is larger in areas where the initial enrollment rates were lower. Thus, in order to capture any time-varying factors correlated with pre-program enrollment rates, column (4) adds enrollment rates in the province of birth in 1995 interacted with year of birth dummies as controls as well as the other controls in column (3).

As expected, the CSL had larger effects on younger cohorts in all specifications. The estimated coefficients are all positive and statistically significant (except the estimate of age 11 in 1997 in column (4)). The F-statistics for testing the joint significance of the five age cohorts are reported in Table 2. The set of year of birth estimates are jointly significant at all conventional significance levels. The F-ratios reported in Table 2 are much larger than the rule of thumb that the instruments are weak if the first stage F-ratio is less than 10.[26] The estimates of the increase in female schooling due to the CSL range from 0.44 to 2.69 in the specification with no controls (column (1) of Table 2). The results suggest that the education of the youngest cohort increased by 2.5 years due to the CSL. The estimates increase as more controls are added to the baseline.

[25] There are 80 provinces included in the estimations based on the 1995 boundaries of Turkey. In all the estimations throughout the study, women born in Düzce are assumed to be born in Bolu since Düzce broke off Bolu and became a province in November 1999.

[26] Critical F-ratio of 10 is suggested by Staiger and Stock (1997). Cameron and Trivedi (2005) use this rule of thumb, but they also propose a less strict rule of thumb of critical F-ratio of 5.

4.1.3 Alternative Approach: Difference-in-difference (DID)

To estimate the impact of the CSL on education, we can also simply compare the average education of women exposed and unexposed to the law based on year of birth in a regression framework as follows:

$$E_i = a_1 + T_i\delta_1 + X_i\theta + \varepsilon_i \quad (4)$$

where the parameters are defined similar to equation (2).

Equation 4 is used to estimate the effect of the law on education for two samples. Table 3 (columns (1)-(4)) reports the effect of the CSL on the number of years of schooling completed. Panel A compares the education levels of women aged 7 to 11 in 1997 (exposed) with the education levels of women aged 12 to 15 in 1997 (unexposed). The results for several specifications are presented. The estimates indicate that the CSL increased the education levels of exposed females by 1.75 to 1.98 years in columns (1)-(3), while it increased female schooling by 2.23 years in column (4). The estimated coefficient in column (4) is slightly larger, indicating that it does not confound the effect of the CSL. In sum, the estimates suggest that there is a significant and positive effect of the CSL on the number of years of schooling completed for females.

As further verification that there are no pre-existing trends in educational attainment across cohorts, Panel B of Table 3 compares the education levels of the cohort aged 12 to 15 in 1997 to the cohort aged 16 to 19 in 1997 (neither cohort should have been affected by the CSL). If education levels were already increasing prior to the change in compulsory schooling then there should be a positive and significant coefficient for the cohort of aged 12 to 15 in 1997 in Panel B. However, I find no evidence of pre-existing trends in schooling– the coefficients are typically small and insignificant, thereby, providing some evidence that the identification assumption is satisfied.[27] Thus, this control experiment demonstrates that

[27] I also compare the outcomes of females aged 7-11 to females aged 15-18 in 1997 in Panel A, and of females aged 15-18 to females aged 19-22 in 1997 in Panel B in order to account for cases where the CSL

the results in Panel A are reliable.[28]

4.2 Effects of Female Schooling on Fertility: IV and OLS Estimates

The following equation estimates the impact of female education on fertility

$$Y_{ijk} = a_2 + \alpha_{2j} + \pi E_{ijk} + \sum_{l=7}^{18} (C_j \times d_{il})\delta_{2l} + \nu_{ijk} \tag{5}$$

where Y_{ijk} is the fertility of a woman i, born in province j, in year k. The other parameters are defined similar to equation (1). In order to account for the fact that the fertility history of younger women is censored, I use the number of children born before age 18 as the main dependent variable.[29]

OLS estimates may be biased if schooling (E_{ijk}) is correlated with unobserved factors (ν_{ijk}), such as family background and personal traits. For instance, women with high discount rates of time or low ability levels are less likely to have higher education levels and more likely to become teenage parents. As a result of omitted individual characteristics, OLS estimates could overstate the effect of schooling. In order to identify the causal effect of female education on fertility, a two-stage least squares (2SLS) methodology is implemented. The set of year of birth dummies are valid instruments in the above equation if exposure to the CSL had no direct effect on fertility other than via changing educational attainment of women (Breierova and Duflo, 2004). I also use a single instrument–the treat-

might have affected females aged 12-14 due to grade repetition and/or enrollments at different ages. The effects were higher for this different age cohort comparison, therefore, I decided to be more conservative and use the age cohorts in Table 3.

[28] The DID estimate can be simply determined by subtracting the estimate in Panel B from the estimate in Panel A in Table 3.

[29] The youngest women in the sample are 18. The effect of female education on teenage fertility before the ages of 17 and 16 are also explored. As a robustness check, I compare the number of children born before age 20 of females that were aged 9-11 in 1997 to females aged 12-14 in 1997, as well as the number of children born before age 19 of females that were aged 8-11 in 1997 to females aged 12-15 in 1997. The OLS estimates suggest about a 15% and a 16% reduction in the number of children born before age 20 and age 19 starting at the mean (0.25 and 0.14 for the associated samples), while the IV estimates correspond to a 32% and a 22% reduction, respectively–estimations include all controls and observation numbers are 2,132 and 2,862. These tighter age window comparison results are consistent with the findings of the effect of female education on teenage fertility before the age of 18.

ment dummy for women aged 7-11 in 1997–to determine the impact of female education on fertility.

Table 4 presents both OLS and IV estimates of the effect of female schooling on teenage fertility–both with and without controls. Panels A, B, and C of Table 4 report estimates for fertility outcomes, as measured by fertility before age 18, age 17, and age 16, respectively for the sample of 18-30 year old women in columns (1)-(4) and 18-25 year old women in column (5).

The first line of Panel A displays the OLS estimates: a one-year increase in female education reduced the number of births before age 18 by 0.015 (significant at the 1% level). This result corresponds to about a 17% reduction in teenage births starting at the mean (0.09 for women ages 18-30). The second and third lines of Panel A present the 2SLS results with different instruments, which are quite similar, but slightly higher for the IV estimates using year of birth dummies ("YOB dummies").[30] The estimates are around 0.03 across different specifications; hence, a one-year increase in female education reduces teenage fertility by 0.03 births, which is a reduction of 33%. Column (5) presents the results for a tighter window, and the results remain robust for all specifications.[31] The 2SLS estimates are significantly different from the OLS estimates, suggesting that the standard OLS estimates may underestimate the effect of female education on fertility.

Similarly, the IV estimates of the effect of female schooling on fertility before age 17 and 16 (Panels B and C) are significantly different from the OLS estimates, consistent with the view that the OLS estimates can be biased. OLS estimates suggest that a one-year increase in female education reduces fertility before age 17 and 16 by around 0.008 and 0.003 births, which is close to a 20% and 15% reduction starting at the mean (0.04 and

[30] p-values of the overidentification test are in square brackets.

[31] As a robustness check, the estimations are repeated for a sample of 20-25 year old women for all teenage fertility outcomes (2,132 observations). The estimates confirm the results reported in Table 4. For this sample, I also explore the effect of female education on fertility before age 20. As mentioned in footnote 29, OLS estimate suggests that a one-year increase in female education reduced fertility before age 20 by 0.04 births, which is a 16% reduction starting at the mean (0.25 for women ages 20-25), while 2SLS estimate suggests that it reduces fertility before age 20 by 0.08 births, which is about a 32% reduction.

0.02 for women ages 18-30, respectively). The 2SLS estimates indicate that the reduction in fertility before age 17 and 16 is around 0.02 and 0.01 births, which is about a 50% reduction in teenage births. The estimates again remain robust for the subsample of 18-25 year old women.

The IV estimates exceed the OLS estimates for all teenage fertility outcomes. The OLS estimates may be biased downwards due to omitted variables that correlate with both higher levels of schooling and early child rearing, such as access to economic opportunities. Another possible explanation is that the IV estimates pertain to only a subsample of women whose educational attainment has been affected by the change in the compulsory schooling law. The effect of education on fertility may be higher for this subsample, thereby, leading to larger IV estimates compared to OLS estimates. The possibility of heterogeneous effects of education on teenage fertility is explored in Section 4.3.

4.2.1 Alternative Approach: Difference-in-Difference (DID)

Analogous to equation (4), the following specification estimates the impact of the CSL on fertility:

$$Y_i = a_2 + T_i\delta_2 + X_i\theta + \varepsilon_i \tag{6}$$

where Y_i is the fertility of a woman i, and the other parameters are defined similar to equation (4). The outcome of interest is the teenage fertility, as measured by the number of children before age 18.

Table 3 (columns (5)-(8)) reports the results for estimating equation 6 using two subsamples based on the year of birth. The results suggest that the CSL decreased teenage fertility by 0.04 births. As in the education case, I report the control experiment in Panel B of Table 3 to verify whether the results are driven by differential time trends across cohorts. It appears that time trends do not confound the impact of the CSL on teenage fertility. The coefficients of the control experiment are small and insignificant. In both comparisons of

Panel A and Panel B, the coefficients are increasing as additional controls are added to the baseline specification of no control. The same exercise for fertility before age 17 and 16 suggests that the CSL decreased fertility before age 17 and 16 by 0.04 and 0.02 births, respectively, and, moreover, the results are not driven by time trends (the coefficients of control experiment of Panel B are small and insignificant).[32]

Furthermore, Table 5 demonstrates that the effect of the CSL on fertility persists for fertility before the ages of 19, 20, 21, and 22 (much beyond the ages above which the CSL binds). For instance, the CSL decreased early fertility before age 20 of females aged 9-11 in 1997 and before age 21 of females aged 10-11 in 1997 by around 0.07 births, compared to females aged 12-15 in 1997, which is a reduction of 27% and 18% starting at the mean (0.26 and 0.40 for the associated samples), respectively.

4.3 Are there heterogeneous effects?

The effect of education on fertility is likely to depend on certain characteristics. That is, there are heterogeneous effects–possibly for both the effect of the CSL on education and fertility and the effect of education on fertility.[33] Uncovering heterogeneous effects has important policy implications both in understanding the expected outcomes and for designing optimal policy interventions. To examine whether there are heterogeneous effects, I split the sample into various subsamples of birth-province and used the specifications that include all controls in Table 3 (columns (4) and (8)), and Table 4 (column (4) and 2SLS with "YOB dummies" instrument).

[32] Results are available upon request.

[33] I also test whether the effect of the CSL on education differs by parental education and ethnicity (Appendix C). The results provide evidence that the CSL was more successful at increasing education of women with lower levels of parental education; however, most of the estimates are not significant enough to be conclusive. Moreover, the effect does not seem to differ by parental ethnicity.

A. Pre-change levels of education and fertility in the province of birth

One hypothesis is that it is easier to increase education when the baseline level is lower and, similarly, to reduce fertility when the baseline level is higher (Barham, 2011). I explore heterogeneity of both the effect of female education on fertility and the effect of the CSL on education and fertility according to the baseline (pre-change) levels of fertility and education by dividing the sample into provinces (by birth) with average fertility or education of the unexposed cohort above (or below) the sample median.

Table 6A (first row) suggests that the CSL increased education levels more in provinces where the initial levels were lower than the sample median. In provinces with levels of initial education below the median, the CSL increased female education by 3.05 years, compared to 1.87 years in provinces with initial fertility above the median (the difference is significant at the 5% level). Second, in provinces with levels of initial fertility above the median, the CSL decreased teenage fertility by 0.102 births, compared to an insignificant effect in provinces with initial fertility below the median (the difference is significant at the 1% level). Third, the impact of female education on teenage fertility (third row) is higher (lower) in provinces with initial fertility above (below) the median. Specifically, one more year of female education reduces teenage fertility by 0.04 births in provinces with initial fertility above the median, whereas it reduces teenage fertility by 0.01 births in provinces with initial fertility below the median (the difference is significant at the 1% level). The results, therefore, indicate that there are heterogeneous effects by pre-fertility and pre-education levels in birth-provinces.

B. Pre-change characteristics of the province of birth

Another possible source of heterogeneity is population density because females in sparsely populated provinces are more likely to be affected by the CSL, either as a consequence of new school constructions or more transportation services. Other possible sources of heterogeneity are income (GDP per capita), urbanization rates (the percentage of population in cities), and agricultural activity (percentage of households engaged in agricultural production).[34]

The results are reported in Table 6B. As expected, the effect of the CSL on education (first row) is higher in provinces with population densities, urbanization rates, and income levels, below the median. In provinces with population densities, incomes, and urbanization rates below the median, the CSL increased female education by around 2.70 years, compared to 1.76, 1.90, and 1.86 years in provinces above the median, respectively (the differences are significant at the 5%, 10%, and 1% levels, respectively). However, the effect of the CSL on education does not appear to depend on agricultural activity. The second row of Table 6B presents the results of the effect of the CSL on teenage fertility. The results suggest that the CSL decreased teenage fertility by 0.10 births in provinces with population density below the median, compared to 0.03 births in provinces above the median (the difference is significant at the 10% level). However, the effect does not depend on income, urbanization, and agricultural activity. The impact of female education on teenage fertility (row 3) is slightly higher in provinces with lower incomes, urbanization rates, and percentages of HH's engaged in agricultural production; however, the differences are not statistically significant. The results suggest that female education reduced teenage fertility by 0.04 births in provinces with population density below the median, compared to 0.03 births in provinces above the median (the difference is significant at the 1% level).

4.4 Robustness checks by intensity measures

Following Breierova and Duflo, 2004 and Osili and Long, 2008, I use the number of children bussed in the 2000/2001 Academic Year per 1,000 primary school aged children (in 1995) in each province of birth to examine whether the effect of the CSL depends on some

[34] I use GDP per capita, population densities, and urbanization rates in 1990 (prior to CSL). However, I use agricultural activity in 2001 (the earliest data I could get by province level is 2001) and assume that the percentage of households engaged in agricultural activity has not changed significantly over the four years since the change of the CSL in 1997.

measure of intensity.[35] This is preferable to an intensity measure calculated by the planned numbers because it provides more information on the allocation of schooling inputs to the provinces.[36] The following examines if the effect of the CSL was higher for females born in higher intensity provinces:

$$E_{ijk}(or Y_{ijk}) = a + \alpha_j + \sum_{l=7}^{11}(Intensity_j \times d_{il})\gamma_l + \sum_{l=7}^{11}(C_j \times d_{il})\delta_l + \varepsilon_{ijk} \quad (7)$$

where $Intensity_j$ is the intensity measure in the province of birth. γ_l gives the reduced form estimate of the effect of the CSL on either education or fertility of each cohort in the treated group. Y_{ijk} is the teenage fertility of a female i, born in province j, in year k. Other variables are defined as before. In equation 7, the omitted group is the females aged 12 to 19 in 1997.

The results for different specifications are presented in Table 7A. Columns (1)-(4) show the estimated effects of the CSL on years of education. The coefficients are all positive and mostly statistically significant (except the estimates of the interaction between the dummy for age 11 in 1997 and intensity measure in columns (3) & (4)). The F-statistics (reported in the table) show that the interaction terms are jointly significant. Column (4), which includes all controls, suggests that the CSL increased the education of females aged 8 in 1997 by 1.3 years given that 92.56 children were bussed per 1,000 children on average. I also find that a 100 increase in the number of students bussed increases the years of education of this cohort by 1.4, which corresponds to a 0.36 standard deviation gain in years of education

[35] I do not have data for the number of children bussed to primary schools at the province level for the 1997/1998 Academic Year when the CSL became effective–the earliest data by province level is for the 2000/2001 Academic Year; however, I have data from the 2000/2001 Academic Year on. I look at the changes in the number of children bussed between the 2000/01 and the 2001/02 Academic Year to corroborate the assumption that it has not changed much between the 1997/98 and the 2000/01 Academic Year. I also compared the results of the effects of the CSL to the ones in which I exclude the provinces where the change was greater than 25% (in absolute value) and got similar results. Thus, it is plausible to use the data for the 2000/01 Academic Year to measure the intensity of the CSL. I also use the 1990 Population Census of Turkey rather than the 1995 population projections for the intensity measure–obtained similar results.

[36] The intensity measure is also calculated in a different way to capture the variation by province for only females. That is, the number of female children bussed per 1,000 primary school aged female children in each province of birth is the intensity measure in the analysis. The findings follow the same pattern, therefore, I do not report the results.

(the standard deviation of the mean is 3.9).

Columns (5)-(8) present the results for the effect of the CSL on teenage fertility. The estimates shown in column (5) are all negative and significant (except the estimates for females age 11 in 1997). The effects are same for females aged 7-10 in 1997 (columns (7) & (8)). In column (8), a 100 increase in the number of students bussed per 1,000 decreases the number of teenage births by 0.03 for females aged 7-10 in 1997, which corresponds to a reduction of about 33% in teenage births (the average number of children born before age 18 in the sample is 0.09).

An alternative measure of the intensity of the CSL is the additional number of classrooms (between the 1997/98 and the 1996/97 Academic Year) per 1,000 children (in 1995) in each province of birth.[37] Table 7B presents the results of the effects of the CSL on both education and teenage fertility. The coefficients of the interactions, as shown in columns (1)-(4), are all positive and mostly significant and jointly significant for all specifications (F-statistics are given in the table). As expected, the effect is larger for younger cohorts– with the greatest effect on the education levels of the females aged 8 in 1997. As shown in column (4), one additional classroom per 1,000 children increases the years of schooling of this cohort by 0.195. The average number of additional classrooms per 1,000 children is 9.42, thus, the CSL is estimated to increase the education of females aged 8 in 1997 by 1.84 years (the average years of schooling is 6.8 years).

Similarly, the effects of the CSL on teenage fertility for different cohorts are presented in columns (5)-(8). The coefficients of the interactions are all negative and jointly significant for all specifications. The estimates of the effect of the CSL on teenage fertility are

[37]There are 5 provinces–Ağrı, Çanakkale, Giresun, Kilis, and Bartın–that actually decreased the number of classrooms in between these periods. This stems from closing down some village schools in these provinces due to the unification purposes. Rather, the students from the closed schools were transferred to the schools in the cities and some of the classrooms were united instead of adding classrooms to accommodate more students. As a consequence, the intensity measure is negative in these provinces. However, the results are robust to the exclusion of these provinces (214 observations in these 5 provinces) or to adding a constant to the intensity measure of all provinces in order to avoid negative measures. In any case, the estimated coefficients are a little higher in these robustness checks and do not statistically differ from the ones found in the analysis with negative intensity measures. Results are available upon request.

higher for younger cohorts with the greatest effect on females aged 8 in 1997. The estimate in column (8) for the youngest cohort suggests that an increase of 10 classrooms per 1,000 children decreases teenage fertility by 0.06 births, which corresponds to a 66% reduction in teenage births at its mean value of 0.09.

4.5 Channels through which education affects fertility

In this section I discuss possible channels through which education affects fertility. As mentioned, schooling may reduce fertility by delaying marriage, especially since childbearing out of wedlock is uncommon in Turkey. I estimate the effect of the CSL on the probability of being married and the probability of having no children before age 18.

Table 8 displays the results of whether the CSL had an effect on the probability that a woman remains single and does not have kids before age 18. Estimations of different specifications for both outcomes produce statistically significant coefficients with expected signs. The estimates from a linear probability model (LPM) suggest that the CSL decreased the probability of getting married before age 18 by 5 percentage points, while it raises the probability of having no kids by 3 percentage points. Thus, the fall in fertility before age 18 may be partly explained by postponed childbearing due to delayed marriage.

Furthermore, education may influence contraceptive knowledge and use. Family planning programs, starting in 1965, increased the prevalence of contraception; however, the trend stalled at around 60% during the 1990s. In the 2000s, the trend resumed, increasing to 70%, and in particular modern contraceptive use increased by 10%.[38] While we cannot precisely account for the effect of education on contraceptive knowledge and use, it is plausible that greater education had an important role on the increased prevalence. This represents another channel through which education may reduce fertility.

Another possible mechanism is via the effect of schooling on labor market participation. Female labor force participation for women aged 15+ in Turkey declined from around

[38] See footnote 8.

30% in the 1990s to around 25% in the 2000s.[39] In both decades, women with greater education (high school or more) had higher participation rates than women with less education. Because there are many confounding factors affecting the labor market from one year to another, it is difficult to draw conclusions on the effect of female education on labor force participation. This might be explored in future research as more years of data become available.

The tradeoff between "quality" and "quantity" of children may be another possible channel. Given that infant and children under five mortality stalled at very high rates in the 1990s (both rates exceeded 55 per thousand live births), increased female education may affect child health, thereby, reducing the number of children.[40] Parents with more education may also invest more in their children's schooling, which increases both the quality and the cost of children, thereby reducing the number of children desired. Future research might also explore this tradeoff as more data becomes available.

Finally, men's education might also influence fertility. Appendix D presents the effect of the CSL on men's education, number of children, and marriage. Because men received over 8 years of education on average prior to the CSL, I do not expect that the CSL would change education attainment for most men. The results suggest that the CSL had a very small effect (0.3 years) on men's education, compared to the increase in women's education (around 2 years). Furthermore, the CSL had no effect on men's marriage and number of children before 18. Moreover, Güneş (2013) shows that maternal education does not affect assortative matching in terms of husband's education and type of occupation.

5 Conclusion

In this paper, I explore the causal relationship between female education and fertility using a change in the compulsory schooling law in Turkey. The results suggest that the CSL

[39] Ibid.

[40] http://data.un.org.

increased female schooling by approximately 2 years for primary school age females (aged 7-11 in 1997) and reduced teenage fertility, as measured by the number of births before age 18, by 0.04 births on average, and the effects remain robust with different specification tests. Moreover, robustness checks employing various intensity measures confirm the effect of the CSL on education and teenage fertility.

Variation in female schooling generated by the CSL identifies the causal effect of female education on fertility. The reported IV estimates from a number of different specifications suggest that an extra year of female schooling reduces teenage fertility by around 0.03 births, which is a reduction of 33%. The findings also suggest that there are heterogeneous effects. First, there is a larger decline in teenage fertility in provinces with higher initial fertility and lower population density. Second, the effect of the CSL on female education and teenage fertility depends on several characteristics of birth-province: initial levels of fertility and education, income, urbanization, population density, and agricultural activity. Finally, I provide evidence that the effect of the educational policy can be partly explained by postponed childbearing due to delayed marriage. Exploring alternative channels, such as labor market participation and increased investment in children, represents an interesting area for future research.

These findings are of interest to policy makers since it has been widely pointed out that early childbearing adversely affects mother and child morbidity and mortality, labor market participation, and educational opportunities. The presence of heterogeneous effects, as demonstrated in this paper, has important policy implications for designing optimal policy interventions, especially for developing countries that share similar features with rural areas in Turkey, such as high fertility and/or low population density. The results therefore demonstrate that educational interventions in developing countries–especially ones with high teenage fertility rates–might be an effective policy tool for addressing fertility-related concerns and can accelerate the demographic transitions at later stages.

Figure 1: Total Fertility Rate (TFR) and Mean Age at First Marriage and Birth (MAFM and MAFB); Sources: TurkStat, Hacettepe University Institute of Population Studies (HUIPS)

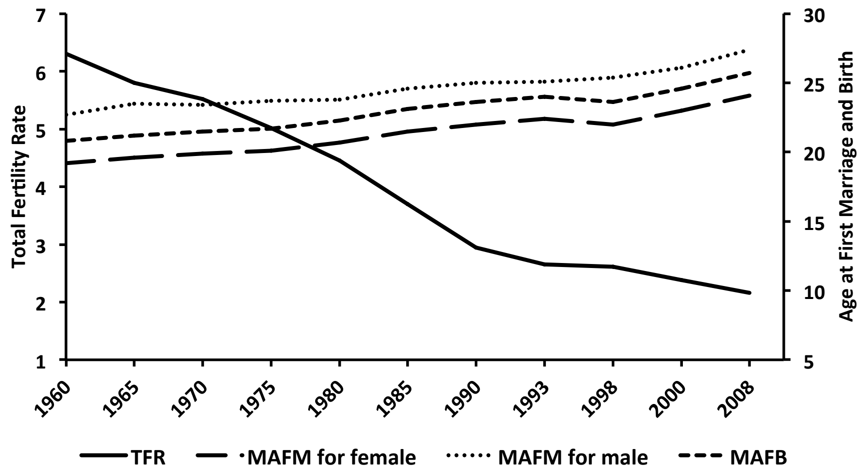


Figure 2: Number of Students in 8-year Primary Education by Academic Year; Enrollments in the 1992/93-1996/97 Academic Years prior to the change in the CSL are the sum of the number of students in the 5-year compulsory primary education and 3-year junior high school

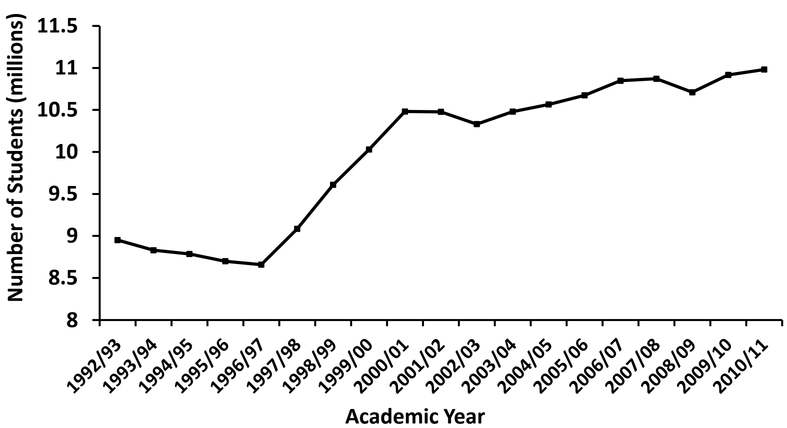
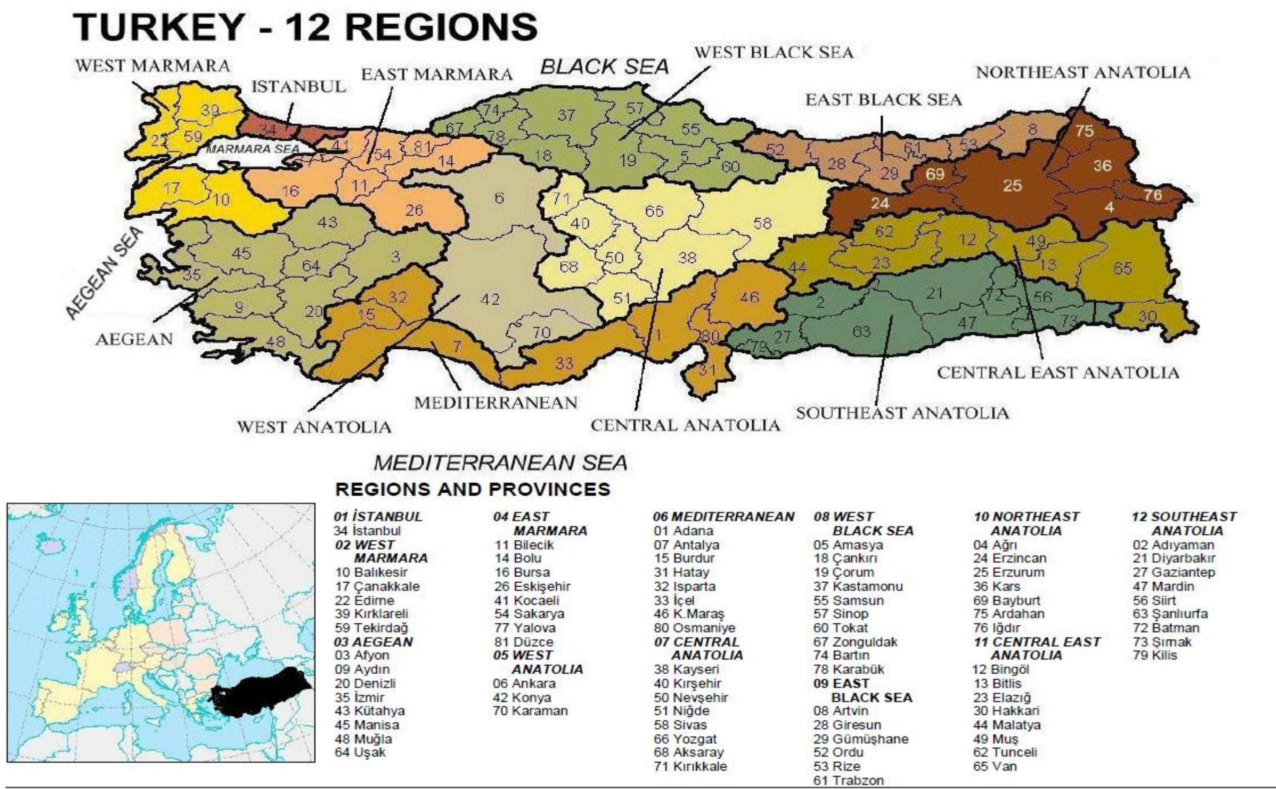
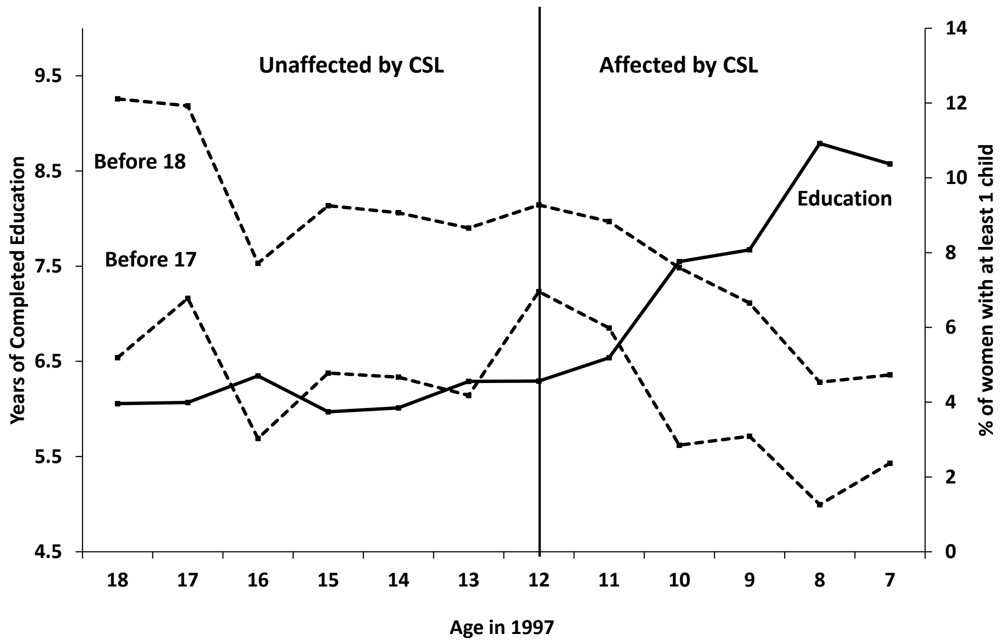


Figure 3: Administrative Regions and Provinces of Turkey



Source: Turkey Demographic and Health Survey 2008 Main Report, Hacettepe University Institute of Population Studies, http://www.hips.hacettepe.edu.tr/eng/index.html

Figure 4: Percentage of Mothers Before Age 18 and 17, and Female Education



Notes: The sample includes all females between the ages of 18 and 29 at the time of the survey. Females aged 12 in 1997 is the youngest unaffected cohort (solid vertical line). Each point on the solid line represents the average education for each cohort. Percentage of mothers for each cohort is the percentage of women with at least 1 ever-born child before age 18 and 17.

Table 1: Descriptive Statistics

	Mean
Female Age	23.76
Female Age in 1997 Year-of-Birth Dummy Fractions	
Age 7 in 1997	0.09
Age 8 in 1997	0.08
Age 9 in 1997	0.09
Age 10 in 1997	0.07
Age 11 in 1997	0.08
Female Years of Completed Education	6.84
Completed five or more years of education	0.82
Completed eight or more years of education	0.46
Fraction of married women	0.56
Fraction of married women before age 18	0.17
Fraction of women having no kids at the time of the survey	0.53
Fraction of women having no kids before age 18	0.93
Number of children ever born	0.94
Number of children before age 18	0.09
Number of children before age 17	0.04
Number of children before age 16	0.02
Number of Observations	4684
Number of Female With Children	2185

Notes: The sample includes all females between the ages of 18 and 30 at the time of the survey.

Table 2: First Stage Coefficients: Effects of the CSL on Completed Years of Schooling

	Dependent Variable: Years of Education			
Age in 1997 Dummies	(1)	(2)	(3)	(4)
Cohort 7 (age = 7 in 1997)	2.475***	2.602***	2.898***	3.449***
	(0.249)	(0.230)	(0.268)	(0.370)
Cohort 8 (age = 8 in 1997)	2.691***	2.669***	2.963***	3.522***
	(0.263)	(0.207)	(0.230)	(0.328)
Cohort 9 (age = 9 in 1997)	1.573***	1.860***	2.047***	2.307***
	(0.280)	(0.217)	(0.265)	(0.407)
Cohort 10 (age = 10 in 1997)	1.448***	1.451***	1.542***	0.883**
	(0.248)	(0.223)	(0.248)	(0.379)
Cohort 11 (age = 11 in 1997)	0.439**	0.805***	0.752***	0.439
	(0.215)	(0.214)	(0.262)	(0.386)
Control Variables:				
Province of birth dummies	No	Yes	Yes	Yes
Year of birth*Number of children in 1995	No	No	Yes	Yes
Year of birth*Enrollment rate in 1995	No	No	No	Yes
F-statistics	28.22	43.10	42.00	30.07
Adjusted R-square	0.065	0.310	0.312	0.316
Observations	4,684	4,684	4,684	4,684

*Significant at 0.1 level. ** Significant at 0.05 level. ***Significant at 0.01 level.

The F-statistics test the hypothesis that the coefficients of the cohort dummies are jointly zero.

Notes: Standard errors are in parentheses. Standard errors are adjusted for clustering on the province of birth. The number of children in 1995 is the number of primary school age children in the province of birth in 1995 (80 provinces). Enrollment rate in 1995 is the number of children enrolled in primary school in 1995 (obtained from TurkStat) divided by the number of primary school age children in the province of birth in 1995.

Table 3: Effect of the CSL on Education and Fertility Before Age 18

	Dependent Variable								
		Years of Education				Fertility Before Age 18			
	Obs	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel A: Women Aged 7-11 to 12-15 in 1997									
Women ages 7 to 11 in 1997 dummy	3,327	1.747***	1.839***	1.976***	2.231***	-0.039***	-0.043***	-0.051***	-0.039*
		(0.174)	(0.155)	(0.183)	(0.283)	(0.011)	(0.012)	(0.015)	(0.023)
Panel B: Women Aged 12-15 to 16-19 in 1997									
Women ages 12 to 15 in 1997 dummy	2,735	0.090	0.205	0.349**	0.267	-0.024	-0.030	-0.033	-0.051
		(0.153)	(0.125)	(0.148)	(0.287)	(0.016)	(0.017)	(0.022)	(0.034)
Control Variables:									
Province of birth dummies		No	Yes	Yes	Yes	No	Yes	Yes	Yes
Year of birth*Number of children in 1995		No	No	Yes	Yes	No	No	Yes	Yes
Year of birth*Enrollment rate in 1995		No	No	No	Yes	No	No	No	Yes

*Significant at 0.1 level. ** Significant at 0.05 level. ***Significant at 0.01 level.

Notes: Standard errors are in parentheses. Standard errors are adjusted for clustering on the province of birth. The number of children in 1995 is the number of primary school age children in the province of birth in 1995 (80 provinces). Enrollment rate in 1995 is the number of children enrolled in primary school in 1995 (obtained from TurkStat) divided by the number of primary school age children in the province of birth in 1995.

Table 4: Effects of Female Schooling on Fertility Outcomes: OLS and 2SLS Estimates

Method (Instrument)	(1) 18-30	(2) 18-30	(3) 18-30	(4) 18-30	(5) 18-25
Panel A: Dependent Variable:					
Fertility Before Age 18					
OLS	-0.018***	-0.016***	-0.016***	-0.015***	-0.013***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
2SLS (Treatment Dummy)	-0.028***	-0.029***	-0.031***	-0.027**	-0.023*
	(0.007)	(0.007)	(0.008)	(0.011)	(0.013)
2SLS (YOB Dummies)	-0.029***	-0.029***	-0.032***	-0.029***	-0.028***
	(0.006)	(0.006)	(0.007)	(0.008)	(0.009)
	[0.424]	[0.468]	[0.372]	[0.334]	[0.121]
Panel B: Dependent Variable:					
Fertility Before Age 17					
OLS	-0.010***	-0.009***	-0.008***	-0.008***	-0.007***
	(0.002)	(0.001)	(0.001)	(0.001)	(0.001)
2SLS (Treatment Dummy)	-0.016***	-0.016***	-0.018***	-0.018**	-0.018**
	(0.005)	(0.005)	(0.005)	(0.007)	(0.007)
2SLS (YOB Dummies)	-0.018***	-0.018***	-0.020***	-0.020***	-0.021***
	(0.004)	(0.004)	(0.005)	(0.005)	(0.006)
	[0.526]	[0.549]	[0.571]	[0.428]	[0.303]
Panel C: Dependent Variable:					
Fertility Before Age 16					
OLS	-0.005***	-0.004***	-0.004***	-0.003***	-0.003***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
2SLS (Treatment Dummy)	-0.009***	-0.009***	-0.010***	-0.010***	-0.010*
	(0.003)	(0.002)	(0.003)	(0.004)	(0.006)
2SLS (YOB Dummies)	-0.009***	-0.009***	-0.010***	-0.010***	-0.009***
	(0.002)	(0.002)	(0.003)	(0.003)	(0.003)
	[0.216]	[0.285]	[0.320]	[0.141]	[0.101]
Control Variables:					
Province of birth dummies	No	Yes	Yes	Yes	Yes
YOB*Number of children in 1995	No	No	Yes	Yes	Yes
YOB*Enrollment rate in 1995	No	No	No	Yes	Yes
Observations	4,684	4,684	4,684	4,684	2,993

*Significant at 0.1 level. ** Significant at 0.05 level. ***Significant at 0.01 level.

Treatment: Age 7-11 in 1997; Control: Age 12-19 in 1997 in columns (1)-(4). Treatment: Age 7-11 in 1997; Control: Age 12-14 in 1997 in column (5). *Notes:* Standard errors are in parentheses. Standard errors are adjusted for clustering on the province of birth. P-values of the overidentification test are in square brackets. "YOB" refers to year of birth. The number of children in 1995 is the number of primary school age children in the province of birth in 1995 (80 provinces). Enrollment rate in 1995 is the number of children enrolled in primary school in 1995 (obtained from TurkStat) divided by the number of primary school age children in the province of birth in 1995.

Table 5: Effect of the CSL on Fertility

	Dependent Variable			
	Before 19	Before 20	Before 21	Before 22
	(1)	(2)	(3)	(4)
Comparison Group	8-11 vs 12-15	9-11 vs 12-15	10-11 vs 12-15	11 vs 12-15
Treatment	-0.054***	-0.068***	-0.074**	-0.095**
	(0.017)	(0.024)	(0.028)	(0.042)
Observations	2,862	2,466	2,045	1,729

Notes: All comparison groups are defined by age in 1997. For instance, 8-11 vs 12-15 is comparing teenage fertility for females aged 8-11 in 1997 to 12-15 in 1997. Standard errors (reported in parantheses) are adjusted for clustering at the province level. Regressions do not have controls.

Table 6A: Heterogeneity of the impact and the CSL effect by pre-change levels of fertility and education

		Pre-Change Province of Birth Characteristics			
	Whole	Pre-change education		Pre-change fertility	
	Sample	<Median	>=Median	<Median	>=Median
	(1)	(2)	(3)	(4)	(5)
1) Effect of the CSL on Education					
Treatment	2.231***	3.047***	1.870***	1.837***	2.788***
(7-11 vs 12-15)	(0.283)	(0.482)	(0.274)	(0.394)	(0.369)
2) Effect of the CSL on Fertility					
Treatment	-0.039*	-0.107*	0.002	0.007	-0.102**
(7-11 vs 12-15)	(0.023)	(0.054)	(0.020)	(0.017)	(0.043)
3) Impact of Female Education on Fertility					
Years of Education	-0.025***	-0.034***	-0.017*	-0.012	-0.038***
(Treatment: 7-11 vs 12-15)	(0.008)	(0.011)	(0.009)	(0.008)	(0.010)

*Significant at 0.1 level. ** Significant at 0.05 level. ***Significant at 0.01 level.
Notes: Median pre-program education is 6.428; median pre-program fertility is 0.091.
Standard errors are in parentheses. Standard errors are adjusted for clustering on the province of birth.

Table 6B: Heterogeneity of the impact and the CSL effect by pre-change province characteristics

		Pre-Change Province of Birth Characteristics							
	Whole	Density		GDP		Urbanization		%HH in Agriculture	
	Sample	<Median	>=Median	<Median	>=Median	<Median	>=Median	<Median	>=Median
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
1) Effect of the CSL on Education									
Treatment	2.304***	2.607***	1.762***	2.693***	1.900***	2.724***	1.858***	2.290***	2.212***
7-11 vs 12-19	(0.236)	(0.267)	(0.318)	(0.301)	(0.334)	(0.375)	(0.331)	(0.307)	(0.379)
2) Effect of the CSL on Fertility									
Treatment	-0.061*	-0.100***	-0.028	-0.102**	-0.033	-0.074*	-0.052	-0.068*	-0.063*
7-11 vs 12-19	(0.026)	(0.035)	(0.040)	(0.047)	(0.025)	(0.043)	(0.036)	(0.039)	(0.035)
3) Impact of Female Education on Fertility									
Years of Education	-0.029***	-0.040***	-0.025**	-0.039***	-0.024***	-0.034***	-0.027**	-0.033***	-0.025**
(Treatment: 7-11 vs 12-19)	(0.008)	(0.010)	(0.012)	(0.010)	(0.009)	(0.010)	(0.012)	(0.010)	(0.011)

*Significant at 0.1 level. ** Significant at 0.05 level. ***Significant at 0.01 level.

Notes: Median population density is 63.14 (per square kilometers); median GDP is $1908; median urbanization rate is 48.168%; median % HH engaged in agricultural activity is 73.744 %. Standard errors are in parentheses. Standard errors are adjusted for clustering on the province of birth.

Table 7A: Effects of the CSL by Year of Birth and Intensity Measure (Number of Students Bussed)

	Dependent Variable: Years of Education				Dependent Variable: Number of kids before age 18			
Coefficients of the interactions	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Age 7 in 1997 * Students bussed per	0.020***	0.018***	0.016***	0.012***	-0.0004***	-0.0003***	-0.0003***	-0.0003***
1000 children in province of birth	(0.003)	(0.002)	(0.002)	(0.002)	(0.0001)	(0.0001)	(0.0001)	(0.0001)
Age 8 in 1997 * Students bussed per	0.020***	0.018***	0.016***	0.014***	-0.0005***	-0.0004***	-0.0003***	-0.0003***
1000 children in province of birth	(0.002)	(0.002)	(0.002)	(0.002)	(0.0001)	(0.0001)	(0.0001)	(0.0001)
Age 9 in 1997 * Students bussed per	0.017***	0.015***	0.014***	0.012***	-0.0004***	-0.0003***	-0.0003***	-0.0003***
1000 children in province of birth	(0.002)	(0.002)	(0.002)	(0.002)	(0.0001)	(0.0001)	(0.0001)	(0.0001)
Age 10 in 1997 * Students bussed per	0.012***	0.010***	0.009***	0.004**	-0.0004***	-0.0003***	-0.0003***	-0.0003***
1000 children in province of birth	(0.002)	(0.002)	(0.002)	(0.002)	(0.0001)	(0.0001)	(0.0001)	(0.0001)
Age 11 in 1997 * Students bussed per	0.005*	0.004**	0.003	0.001	-0.0001	0.0000	0.0001	0.0001
1000 children in province of birth	(0.003)	(0.002)	(0.002)	(0.002)	(0.0002)	(0.0001)	(0.0002)	(0.0002)
Control Variables:								
Province of birth dummies	No	Yes	Yes	Yes	No	Yes	Yes	Yes
YOB*Number of children in 1995	No	No	Yes	Yes	No	No	Yes	Yes
YOB*Enrollment rate in 1995	No	No	No	Yes	No	No	No	Yes
F-statistics	20.87	25.42	21.60	16.12	7.61	6.46	5.07	2.43
R-square	0.074	0.303	0.310	0.319	0.006	0.051	0.053	0.057
Observations	4,684	4,684	4,684	4,684	4,684	4,684	4,684	4,684

*Significant at 0.1 level. ** Significant at 0.05 level. ***Significant at 0.01 level.

The F-statistics test the hypothesis that the coefficients of interactions are jointly zero.

Notes: Standard errors are in parentheses. Standard errors are adjusted for clustering on the province of birth. "YOB" refers to year of birth. The number of children in 1995 is the number of primary school age children in the province of birth in 1995 (80 provinces). Enrollment rate in 1995 is the number of children enrolled in primary school in 1995 (obtained from TurkStat) divided by the number of primary school age children in the province of birth in 1995.

Table 7B: Effects of the CSL by Year of Birth and Intensity Measure (Number of Additional Classrooms)

	Dependent Variable: Years of Education				Dependent Variable: Number of kids before age 18			
Coefficients of the interactions	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Age 7 in 1997 * Additional Classrooms	0.153***	0.194***	0.187***	0.134***	-0.004***	-0.005***	-0.005***	-0.006***
per 1000 children in province of birth	(0.047)	(0.031)	(0.040)	(0.041)	(0.001)	(0.001)	(0.002)	(0.002)
Age 8 in 1997 * Additional Classrooms	0.181***	0.220***	0.222***	0.195***	-0.005***	-0.006***	-0.006***	-0.007***
per 1000 children in province of birth	(0.043)	(0.024)	(0.029)	(0.029)	(0.001)	(0.001)	(0.002)	(0.002)
Age 9 in 1997 * Additional Classrooms	0.089**	0.146***	0.141***	0.103***	-0.003*	-0.004***	-0.005***	-0.005***
per 1000 children in province of birth	(0.039)	(0.022)	(0.028)	(0.029)	(0.001)	(0.001)	(0.002)	(0.002)
Age 10 in 1997 * Additional Classrooms	0.069**	0.106***	0.093***	0.010	-0.002	-0.003*	-0.003	-0.003
per 1000 children in province of birth	(0.028)	(0.017)	(0.023)	(0.030)	(0.002)	(0.002)	(0.002)	(0.002)
Age 11 in 1997 * Additional Classrooms	0.008	0.081***	0.078***	0.060**	-0.000	-0.001	-0.001	-0.001
per 1000 children in province of birth	(0.027)	(0.016)	(0.019)	(0.025)	(0.002)	(0.002)	(0.002)	(0.002)
Control Variables:								
Province of birth dummies	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Year of birth*Number of children in 1995	No	No	Yes	Yes	No	No	Yes	Yes
Year of birth*Enrollment rate in 1995	No	No	No	Yes	No	No	No	Yes
F-statistics	6.44	26.33	16.44	10.52	6.43	6.07	4.28	2.70
R-square	0.035	0.304	0.306	0.313	0.004	0.053	0.054	0.057
Observations	4,684	4,684	4,684	4,684	4,684	4,684	4,684	4,684

*Significant at 0.1 level. ** Significant at 0.05 level. ***Significant at 0.01 level.

The F-statistics test the hypothesis that the coefficients of interactions are jointly zero.

Notes: Standard errors are in parentheses. Standard errors are adjusted for clustering on the province of birth. The number of children in 1995 is the number of primary school age children in the province of birth in 1995 (80 provinces). Enrollment rate in 1995 is the number of children enrolled in primary school in 1995 (obtained from TurkStat) divided by the number of primary school age children in the province of birth in 1995.

Table 8: Effect of the CSL on Fertility Before Age 18, Marital Status, and No kids

Dependent Variable	(1)	(2)	(3)	(4)
Fertility Before Age 18	-0.039***	-0.043***	-0.051***	-0.039*
	(0.011)	(0.012)	(0.015)	(0.023)
(R-squared)	0.004	0.047	0.049	0.050
Probability of Having No kids				
Before Age 18	0.026***	0.030***	0.034***	0.027*
	(0.009)	(0.009)	(0.011)	(0.016)
(R-squared)	0.003	0.043	0.044	0.046
Probability of Being Married				
Before Age 18	-0.043***	-0.047***	-0.046***	-0.045*
	(0.012)	(0.012)	(0.015)	(0.023)
(R-squared)	0.003	0.047	0.050	0.052
Control Variables:				
Province of birth dummies	No	Yes	Yes	Yes
Year of birth*Number of children in 1995	No	No	Yes	Yes
Year of birth*Enrollment rate in 1995	No	No	No	Yes

*Significant at 0.1 level. ** Significant at 0.05 level. ***Significant at 0.01 level.

All specifications have 3,327 observations. Treatment: Age 7-11 in 1997; Control: Age 12-15 in 1997.

Notes: Standard errors are in parentheses. Standard errors are adjusted for clustering on the province of birth. The number of children in 1995 is the number of primary school age children in the province of birth in 1995 (80 provinces). Enrollment rate in 1995 is the number of children enrolled in primary school in 1995 (obtained from TurkStat) divided by the number of primary school age children in the province of birth in 1995.

Appendix A. The CSL on different levels of education

Following Duflo (2001), I estimate the equation below to check if the CSL succeeded in affecting mainly the targeted groups since the impact of the CSL on fertility depends on it.

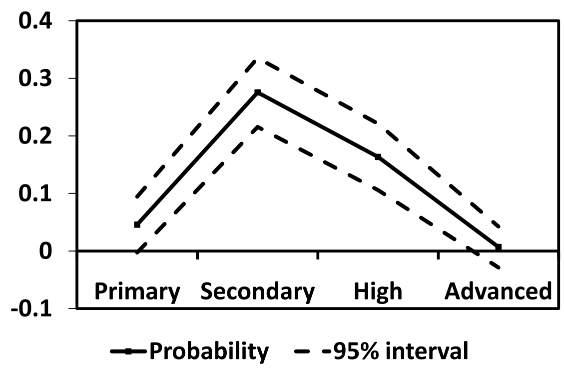
$$E_{im} = d + G_i\beta_t + T_i\hbar_m + \varepsilon_i \tag{8}$$

where E_{im} is a schooling variable which takes value 1 if individual i completed m level of education, 0 otherwise. d is a constant, β_t accounts for group time fixed effects (7-11; 12-15; 16-19 in 1997), and T_i is the treatment dummy. $\hbar_m$ is the estimated impact of the CSL for 4 different levels of education (m): primary (5 years of education), secondary (8 years of education), high (11 years of education), and advanced (11+ years of education). Figure

A.1 presents the estimated probabilities from LPM with corresponding 95% confidence intervals.

The effect of the CSL is the highest for the probability of completing 8 years of education, at the level of the goal of the education policy. There is evidence that the CSL increased the likelihood of completing high school, and a negligible effect on the probability of completing advanced education. Despite the small spillovers, the program substantially increased schooling through the level of education associated with the CSL.

Figure A.1: Probability of Completing at Least "M" Level of Education



Appendix B. First Stage Coefficients: Effects of the CSL on Completed Years of Schooling (Unrestricted Specification)

Age in 1997 Dummies	Dependent Variable: Years of Education
Cohort 7 (age = 7 in 1997)	2.854***
	(0.322)
Cohort 8 (age = 8 in 1997)	3.070***
	(0.311)
Cohort 9 (age = 9 in 1997)	1.952***
	(0.368)
Cohort 10 (age = 10 in 1997)	1.827***
	(0.329)
Cohort 11 (age = 11 in 1997)	0.818***
	(0.249)
Cohort 12 (age = 12 in 1997)	0.573
	(0.349)
Cohort 13 (age = 13 in 1997)	0.569
	(0.346)
Cohort 14 (age = 14 in 1997)	0.291
	(0.269)
Cohort 15 (age = 15 in 1997)	0.268
	(0.305)
Cohort 16 (age = 16 in 1997)	0.627*
	(0.342)
Cohort 17 (age = 17 in 1997)	0.348
	(0.266)
Cohort 18 (age = 18 in 1997)	0.335
	(0.251)
F-statistics for age 7-11 in 1997	34.97
	(p=0.000)
F-statistics for age 12-18 in 1997	1.01
	(p=0.419)
Adjusted R-square	0.316
Observations	4,684

*Significant at 0.1 level. ** Significant at 0.05 level. ***Significant at 0.01 level.

Notes: The F-statistics test the hypothesis that the coefficients are jointly zero. Standard errors (reported in parentheses) are adjusted for clustering on the province of birth.

Appendix C. Heterogeneity of the CSL effect on education by mother's education and ethnicity

	Part A: Mother's Education		Part B: Mother's Ethnicity	
	Dependent Variable: Years of Education		Dependent Variable: Years of Education	
Variables	(1)	(2)	Variables	(3)
T	1.599***	1.591***	T	2.456***
	(0.293)	(0.292)		(0.226)
ME (primary)	1.664***	1.664***	Mother's ethnicity (Kurdish)	-3.057***
	(0.163)	(0.162)		(0.263)
ME (secondary)	3.198***	3.199***	Mother's ethnicity (Other)	-2.293***
	(0.398)	(0.396)		(0.676)
ME (higher)	5.023***	5.010***		
	(0.627)	(0.625)		
T*ME (primary)	0.559**			
	(0.267)			
T*ME (secondary)	0.002			
	(0.548)			
T*ME (higher)	-0.825			
	(0.669)			
T*ME (primary)*Cohort 7		1.153***	T*Other*Cohort 7	-1.053
		(0.331)		(1.657)
T*ME (secondary)*Cohort 7		0.229	T*Kurdish*Cohort 7	-0.388
		(0.727)		(0.450)
T*ME (higher)*Cohort 7		-1.061		
		(0.646)		
T*ME (primary)*Cohort 8		0.970***	T*Other*Cohort 8	0.820
		(0.342)		(1.302)
T*ME (secondary)*Cohort 8		-0.428	T*Kurdish*Cohort 8	-0.016
		(0.768)		(0.445)
T*ME (higher)*Cohort 8		-0.876		
		(0.687)		
T*ME (primary)*Cohort 9		1.038***	T*Other*Cohort 9	-0.388
		(0.364)		(0.928)
T*ME (secondary)*Cohort 9		0.490	T*Kurdish*Cohort 9	-1.123**
		(0.641)		(0.429)
T*ME (higher)*Cohort 9		-0.325		
		(0.881)		
T*ME (primary)*Cohort 10		0.070	T*Other*Cohort 10	-0.703
		(0.403)		(0.523)
T*ME (secondary)*Cohort 10		-0.070	T*Kurdish*Cohort 10	-1.154**
		(0.860)		(0.571)
T*ME (higher)*Cohort 10		-0.905		
		(0.963)		
T*ME (primary)*Cohort 11		-0.667	T*Other*Cohort 11	0.173
		(0.500)		(1.531)
T*ME (secondary)*Cohort 11		-0.122	T*Kurdish*Cohort 11	-0.747
		(1.828)		(0.477)
T*ME (higher)*Cohort 11		-0.046		
		(0.748)		
Adj. R-squared	0.356	0.358	*Adj. R-squared*	0.364
Observations	4,186	4,186	*Observations*	4,537

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. All controls are included. Standard errors are in parentheses. Standard errors are adjusted for clustering on the province of birth. T is the treatment dummy and ME is mother's education categories. Omitted groups are mothers with no education and Turkish mothers.

Appendix D. The Effect of the CSL on Men's Education, Number of Kids, and Marriage

	Dependent Variable			
	(1)	(2)	(3)	(4)
	18-25	18-25	18-25	18-25
Panel A: Reduced Form Estimation				
	Years of Education			
Men ages 7 to 11 in 1997 dummy	0.286**	0.360**	0.495***	0.418*
	(0.138)	(0.139)	(0.170)	(0.235)
Women ages 7 to 11 in 1997 dummy	1.697***	1.745***	1.966***	2.162***
	(0.176)	(0.167)	(0.188)	(0.297)
	Number of Kids Before 18			
Men ages 7 to 11 in 1997 dummy	-0.002	-0.002	-0.003	-0.003
	(0.003)	(0.002)	(0.003)	(0.003)
	Probability of Being Married Before 18			
Men ages 7 to 11 in 1997 dummy	-0.001	-0.001	-0.001	-0.001
	(0.002)	(0.003)	(0.004)	(0.004)
Panel B: 2SLS Estimation				
(Instrument: Treatment Dummy)				
	Number of Kids Before 18			
Years of Education	-0.003	-0.003	-0.004	-0.004
	(0.006)	(0.005)	(0.005)	(0.005)
	Probability of Being Married Before 18			
Years of Education	-0.000	-0.003	-0.001	-0.001
	(0.015)	(0.013)	(0.010)	(0.010)
Control Variables:				
Province of birth dummies	No	Yes	Yes	Yes
Year of birth*Number of children in 1995	No	No	Yes	Yes
Year of birth*Enrollment rate in 1995	No	No	No	Yes

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Treatment: Age 7-11 in 1997 (65%); Control: Age 12-14 in 1997 (35%).

Notes: Standard errors (reported in parentheses) are adjusted for clustering on the province of birth. Regressions include 3,262 observations. Mean education, number of kids before 18, and probability of being married before 18 are 9.601 (9.330 for unexposed cohorts), 0.006 (0.006 for unexposed cohorts), and 0.005 (0.005 for unexposed cohorts), respectively.

Chapter 4: The Effects of Teenage Childbearing on Long-Term Health in the US: A Twin-Fixed-Effect Approach

1 Introduction

Teenage childbearing is a public policy concern, especially in countries with high teen birth rates, such as the United States. In the US, the teen birth rate rose from 50-55 births per 1,000 women between the ages of 15 and 19 in the late 1970s to around 60 in the early 1990s. Despite a decline in teen birth rates since 1990s (around 39 in 2009), the rates in the US are still high compared to other developed countries (Kearney and Levine, 2012). Among the general public and policy makers, it is widely believed that teenage childbearing is a primary driver of poverty, leading to numerous adverse economic, educational, and health, consequences for mothers and their children. However, it is not clear how much of these adverse outcomes can be directly attributable to teenage childbearing.

Early research documents that teen mothers are more likely to have less education, lower earnings, and to be welfare dependent, and are less likely to participate in the labor force. However, the more recent and rigorous research does not support that teen childbearing has a causal impact on economic and educational outcomes. (For a survey of the literature, see Kearney and Levine, 2012.) Rather than early childbearing causing adverse outcomes, it appears that the socioeconomic background and contextual factors are responsible for early childbearing. Despite the recent attention in the literature, there are few studies examining the effects of teenage childbearing on health and health behaviors of mothers. In particular, the question of whether there are effects on health has yet to be explored.[1]

There are several potential explanations for the link between teenage childbearing and

[1] Some recent studies find that teen mothers are more likely to report poor physical and mental health (Patel and Sen, 2012; Liao, 2003; Hobcraft and Kiernan, 2001), and are more likely to be current smokers (Hobcraft and Kiernan, 2001). However, these studies do not attempt to establish a causal link.

health. Teen mothers might invest less in their health due to prenatal health care and delivery costs, and subsequent costs of child rearing. Moreover, the stress associated with delivery complications, stigma of teenage childbearing, and child rearing at a young age might affect mental health.[2] Since teenage motherhood may interfere with education and employment, teen mothers may engage in risky behaviors and have adverse health. On the other hand, teenage childbearing might improve health if the mother or her family and peers are able to allocate greater resources to her health. In particular, motherhood may increase the value of future health and be associated with other changes in lifestyle that reduce risky behavior.

The observed relationships between teenage childbearing and adverse health might be the result of underlying differences between teen and non-teen mothers (Kearney and Levine, 2012). For instance, women with disadvantaged backgrounds are more likely to be teen mothers and to experience adverse outcomes even without a child as a teen. In order to address the problem of endogeneity of teenage childbearing on various outcomes, previous studies have used within-family estimations using data on pairs of sisters (Geronimus and Korenman, 1992; Holmlund, 2005), instrumental variables (IV) using twin births and miscarriage as instruments (Klepinger et al., 1999; Bronars and Grogger, 1993; Hotz et al., 2005), and propensity score matching (Chevalier and Viitanen, 2003). There is a small literature on the causal effects on health behaviors of mothers. Webbink et al. (2008) employ the within-family approach, using a sample of Australian twins and their relatives, and find that teenage childbearing leads to adverse health behaviors. However, Fletcher (2012) shows that teenage childbearing has negligible effects on health behaviors of mothers in the US using both within-family (sibling differences) and IV approaches (miscarriage as the instrument).

This paper estimates the causal effect of teenage childbearing (before the age of 20) on long-term health and health behaviors of mothers (aged 25–74 in the US in 1995/96)

[2] Younger women are at a greater risk of experiencing some pregnancy complications, such as anemia (Mirowsky and Ross, 2002).

by using a nationally representative sample of twins as well as siblings from the Midlife Development in the United States (MIDUS) dataset. I employ within-family estimations (fixed-effects approaches) using samples of siblings, twin pairs, and identical twin pairs in order to overcome the bias generated by unobserved family background and genetic traits that affect both the probability of becoming a teenage mother and health.[3] More specifically, I compare the long-term health and health behaviors of teenage mothers to that of their (twin) sisters who had their first child after their teens.

This paper contributes to the literature in several ways. This paper adds to the literature by exploring the effect of teenage childbearing on health outcomes of teen mothers. Moreover, this paper contributes to the small literature on the causal effects of teenage childbearing on health behaviors of mothers in three ways. First, this paper uses a more comprehensive set of health behaviors. Second, I control for possible omitted variables by including controls for early life factors, including birth weight and age at menarche. In addition, the data allow me to explore the effects for smaller samples of twins and siblings who are arguably more alike. For example, unlike the previous two studies, I check the robustness of the estimates by excluding twin pairs that reported large differences in the way they dressed and had different childhood playmates. Finally, this paper explores potential mechanisms through which teenage childbearing influences health behaviors in the context of US, including education, the number of children, marriage, and the spouse's educational attainment. Moreover, unlike the previous two studies, I address the problem of incorrect inference due to multiple outcome variables by re-estimating the effects on overall indices (Kling et al., 2007).

The findings indicate that not controlling for unobserved family background and genetic traits overstates the effect of teenage childbearing on the long-term health and health behaviors of mothers. The significant adverse effects of teenage childbearing on health and health behaviors from cross-section estimations disappear once I control for family fixed effects,

[3] It should be noted that heterogeneity within families might bias the effects of teenage childbearing. I address this potential bias in Section 2.

except for exercise and preventive care behaviors. More specifically, the results suggest no significant effects of teenage childbearing on self-reported and evaluated health, mental and physical health, chronic conditions, and cancer. Teenage mothers appear to be less likely to have had a blood pressure check in the past 12 months and engage in less vigorous exercise. However, the results suggest no significant effects on other health behaviors, such as smoking, exercise, marijuana use, preventive care utilization, body-mass-index (BMI), health effort, and general health behavior. These results are consistent with the findings of Fletcher (2012), but inconsistent with Webbink et al. (2008). Exploring heterogeneous effects suggests that younger-generation teenage mothers engage in less vigorous exercise and are less likely to use preventive care, and the adverse effect on preventive care depends on parental education. Further, I find that the effects on exercise and preventive care behaviors may operate through reduced educational attainment and matching with a lower "quality" spouse in terms of educational attainment. The increase in overall fertility and the decreased likelihood of marriage do not appear to account for the effects of teenage childbearing.

The remainder of this paper is organized as follows: Section 2 presents the empirical strategy, Section 3 describes the data and reports summary statistics, Section 4 presents empirical findings, Section 5 provides sensitivity analysis, Section 6 explores potential mechanisms, and Section 7 concludes.

2 Empirical Methodology

The within-family estimation uses the following econometric model:

$$y_{ij} = \alpha + \beta T_{ij} + \mathbf{X_{ij}}'\gamma + f_j + \varepsilon_{ij} \tag{1}$$

where y_{ij} is the outcome of individual i in family j, T_{ij} is a dummy variable indicating whether the individual is a teenage mother, $\mathbf{X_{ij}}$ is a vector of control variables, f_j is an unobserved family effect common to all siblings (twins) within the same family, and ε_{ij} is a random error term. The family-specific effect (f_j) is removed from equation 1 by differencing between siblings, which removes the bias due to unobservable factors common to all siblings such as family endowments (genetic traits for twins). In this within-family specification (or family fixed effects estimation), it is assumed that differences in the teenage childbearing within siblings are exogenous, conditional on the control variables.

The previous literature points out the potential bias in within-family estimations due to heterogeneity within families. Geronimus and Korenman (1992) acknowledge such heterogeneity in their comparison of sisters for estimating the effects of teenage childbearing on socioeconomics outcomes. Examples of sources of heterogeneity include variations in genetic endowments of siblings and in the way parents treat them (Rosenzweig and Wolpin, 1988). Also, Bound and Solon (1999) emphasize that the differences in individual traits between siblings and in their environments over time are potential sources of bias in estimating returns to schooling. Note that the residual of the cross-sectional estimate consists of unobservable components that are directly related to the dependent variable and fertility (endogenous component) and indirectly related to the dependent variable through fertility (exogenous component). The within-family estimator is less biased if the family fixed effect accounts for a larger fraction of the endogenous component than the exogenous component. I address the possible bias due to heterogeneity within families by using the within-family approach for samples of not only siblings but also twin pairs. While the medical literature suggests that adult health outcomes between fraternal and idential twin pairs are not significantly different (Christensen et al., 1995; Duffy, 1993), I also provide results using a sample of identical twins. I also provide several robustness checks: (1) I control for early life factors in the twins sample, more specifically birth weight and age at menarche, (2) I exclude siblings (twins) with large differences in the timing of their first birth, (3) I exclude pairs of sisters where the older sister was a teenage mother (due to possible endogeneity of the outcomes for younger sisters), (4) I exclude twins who separated before 15 years old, and (5) I exclude twins who reported large differences in the way they dressed and had different childhood playmates.

Another well-known concern is that the within-family estimator exacerbates measurement errors by differencing between siblings (twins), which may bias the estimates towards zero (Griliches, 1979). In order to deal with measurement error, previous studies on the returns to schooling use various measures of schooling, including a measure of the respondent's schooling reported by the co-twin (Ashenfelter and Krueger, 1994; Ashenfelter and Rouse, 1998). I discuss the issue of measurement error in the robustness checks section.

3 Data

The data are from the first wave of the Midlife Development in the United States (MIDUS) survey. The MIDUS is a nationally representative survey of 7,108 non-institutionalized English-speaking individuals aged 25–74 in the US in 1995/96.[4] The sample of 7,108 individuals includes subsamples of 1,914 twins and 950 siblings of the respondents. The total number of female siblings in the sample of at least two sisters is 1,354 of which 768 are female twins (384 pairs).

MIDUS is a rich data set, including respondents' socioeconomic and demographic characteristics, such as age, race, education, family background, number of children, and age at first birth. The data also includes information about health, such as self-reported and evaluated health, mental and physical health, chronic conditions, and cancer, and health behavior, such as smoking, exercise, marijuana use, preventive care utilization, BMI, health effort, and general health behavior. The sample is restricted to mothers who have a sister (twin) who is also a mother.

The primary independent variable of interest is teenage childbearing, which is a dummy

[4]See Lundborg, 2013 for an assessment of the representativeness of the MIDUS sample. Compared to the 1995 CPS data, the MIDUS sample contains more educated individuals than the general US population. Also, there are more whites in the siblings and twins samples (over 90%), compared to the CPS sample (about 85%).

variable indicating whether the women had a child before the age of 20. Other explanatory variables used in the paper are age at survey, race, age at menarche, and birth weight (available only for twins).[5]

The outcome variables are grouped into two categories: “health” and “health behaviors.” The health category includes, self-reported and evaluated health, mental and physical health, chronic conditions as well as a dummy variable indicating whether the women had at least 1 chronic condition in the last 12 months, and cancer (a dummy variable indicating whether the women ever had cancer.) The following question is used for self-reported health: “Using a scale from 0 to 10 where 0 means "the worst possible health" and 10 means "the best possible health," how would you rate your health these days?”. The question for self-evaluated health is “In general, compared to most men/women your age, would you say your health is much better (1), somewhat better (2), about the same (3), somewhat worse (4), or much worse (5)?” For number of chronic conditions, I sum the number of chronic conditions the respondents had experienced or been treated for in the past 12 months out of 29 chronic conditions (e.g., asthma, bronchitis, high blood pressure). For mental and physical health, I use dummy variables equal to 1 if the women report their physical/mental health is excellent in general and equal to 0 if they say their physical/mental health is very good, good, fair, or poor. The health behaviors category includes smoking, diet, exercise, marijuana use, preventive care, and general health behaviors. Smoking includes currently and ever smoking as well as smoking starting age. For “diet” behavior I use the following measures: the standard BMI, underweight (BMI-score of 18.5 or lower), overweight (BMI-score of 25 or more), and obese (BMI-score of 30 or more). The number of times per month engaged in vigorous (examples in the survey are running or lifting heavy objects) or moderate physical activity (examples in the survey are bowling or using a vacuum cleaner) are used as measures of exercise behavior. Marijuana use is a dummy variable indicating if the respondent used marijuana or hashish in the past 12 months. For preventive care, I use

[5] In the within-family analysis, the controls for age and race difference out of the estimations for the sample of twins; while age remains in the estimations for the sample of siblings.

information on whether or not the respondent takes vitamins or minerals at least a couple times a week, had a blood pressure test in the past 12 months, and visited a doctor in the past 12 months. I use the following question for general health behaviors: “Using a 0 to 10 scale where 0 means "no thought or effort" and 10 means "very much thought and effort," how much thought and effort do you put into your health these days?” and “How strongly you agree with the statement “I work hard at trying to stay healthy” (1 is strongly agree and 7 is disagree strongly).

Tables 1 and 2 report summary statistics (sample means, standard deviations, and proportions) for the sample of women that are mothers and have at least one sister in the sample that is also a mother. There are 968 mothers in the cross-section sample (columns 1-2), of which 223 (23.04%) had their first child before the age of 20. The within-family samples (columns 3-8) includes sisters that have differential timing of their first births (teen and non-teen), which is used to identify the effect of teenage childbearing on various outcomes. The identifying sample for siblings consists of 107 teenage mothers and 121 sisters, while the identifying sample for (identical) twins consists of (27) 67 teenage mothers and their twin sisters.[6]

The number of observations for each variable in Tables 1 and 2 may differ due to missing values, and “starting age smoking” is for the subset of “ever smoke.” Columns (1) and (2) of Tables 1 and 2 show that teen mothers are less likely to graduate high school and their parents are also less likely to graduate high school. Non-teen mothers are less likely to have ever had cancer, to be current/ever smokers, to be overweight/underweight/obese, and to use marijuana, are more likely to exercise, start smoking at a later age, have lower BMI-scores, and have better physical and mental health (columns (1) and (2)). Columns (3)-(8) show that the differences between teen mothers and non-teen mothers for almost all outcomes are much smaller than the ones in columns (1)-(2), except for “vigorous physical

[6]Note that sample of siblings (twins) where at least one is a teenage mother is used in the analysis, and only siblings (twins) in the identifying samples identify the teenage childbearing coefficient (See Tables 3 and 4 for observation numbers.)

activity”, “blood pressure test”, and “starting age smoking.”

4 Empirical Findings

Tables 3 and 4 present the estimates of the effects of teenage childbearing on health and health behaviors. Columns (1)-(3) report the results of ordinary least squares (OLS) estimation for the cross-section samples of siblings and twins, while columns (4)-(6) report the results of fixed effects (FE) estimation for the samples of siblings, twins, and identical twins (MZ twins) where at least one of the siblings (twins) is a teenage mother. Because the number of identical-twin pairs is small, I focus on the results for the samples of twins, which, consistent with the medical literature, are similar to the results for identical twins. Standard errors are adjusted for clustering within families in the OLS estimations shown in columns (1)-(3). Column (1) of Tables 3 and 4 presents the results of a baseline regression, which excludes any controls, while columns (2) and (3) control for age and race. The within-family estimations for the sample of siblings control for age in column (4), while controls of age and race difference out in columns (5) and (6). The number of observations (groups) used in the estimations for each outcome are also shown in the Tables.

4.1 Health

The estimates in columns (1)-(3) of the Table 3 suggest significant and negative associations between teenage childbearing and health for almost all health outcomes. Teenage mothers report worse health, have more chronic conditions, are less likely to report better physical and mental health, are more likely to ever had cancer, and evaluate their health worse than their sisters (twins). However, the effects of teenage childbearing on health disappear once I control for family fixed effects (columns 4-6), except the estimate of the number of chronic conditions for identical twins, which is significant but switches sign.

4.2 Health Behaviors

The OLS estimates in columns (1)-(3) of Table 4 suggest that there are significant differences in most health behaviors between teen and non-teen mothers. For instance, teen mothers are 16 percentage points more likely to be current smokers after controlling for age and race (column 2). However, the within-family estimates in columns (4)-(6) suggest no significant effects of teenage childbearing on most health behaviors. The estimates for smoking, obesity, and marijuana use are similar to the estimates obtained in Fletcher (2012), which are in contrast to the findings of Webbink et al. (2008). There is evidence that there is a causal effect of teenage childbearing on exercise and preventive care behavior. Teenage mothers engage in less vigorous exercise and are less likely to use preventive care in terms of "blood pressure test". Note that the within-family estimates for these two outcomes are larger than the cross-sectional estimates. This might be explained by the possibility of different effects of teenage childbearing by various factors, such as parental education. I explore such possibilities in the following subsection.

4.3 Inference over Multiple Outcomes

The concern of incorrect inference (increase in Type I error) due to using multiple outcome variables is addressed by re-estimating the effects on overall indices. In order to improve the statistical power, I construct overall indices of health and health behavior using all of the health outcomes for the health index and 11 of the 15 health behaviors for the health behavior index.[7] In order to create the indices, each outcome is rescaled to map higher values to worse health or health behaviors. Then, the z-score of each outcome is calculated by subtracting the mean of mothers who did not have teenage childbearing and dividing by the corresponding standard deviation. The overall indices are obtained by using the principle components analysis (PCA) to determine the weights of standardized health and health

[7] I did not use the outcomes of BMI, underweight, obese, and starting age smoking in creating the health behavior index.

behavior outcomes in the indices.[8] Following Kling et al. (2007), an equally weighted average of z-scores is also used to construct the indices; however, the results are consistent with using the PCA method.[9]

Table 3 and 4 present the effects of teenage childbearing on the overall indices of health and health behavior, respectively. The coefficient estimates in columns (1)-(3) of the Tables indicate negative and statistically significant effects of teenage childbearing on health and health behavior. For instance, teenage childbearing worsens health of the mother by 0.6 standard deviation for both samples of siblings and twins. However, the within-family estimates in columns (4)-(6) suggest no significant effects of teenage childbearing on health and health behavior.[10]

4.4 Heterogeneous Effects

Panels A, B, and C of Table 5 present the effects of teenage childbearing on health and health behaviors for the samples of siblings and twins using within-family estimations by race, parental education, and age, respectively. In Panels A and B, the dummy for teenage childbearing is interacted with a dummy indicating whether the mother is non-white and with average parental education, respectively.[11] The estimates of the teenage childbearing dummy and the interactions are reported. In Panel C, the sample is divided by the age of the mother (older than 45 or not), and the effects of teenage childbearing are reported for these subsamples. Note that only estimates for selected outcomes, which are significant, are reported in the Table 5.[12]

[8] PCA is a statistical technique of data reduction, which converts the correlated variables into an uncorrelated linear combinations of variables (principal components) that account for most of the variance.

[9] Results are available upon request.

[10] Tables A1 and A2 in the Appendix show that the cross-section and within-family estimates of the overall indices are statistically different from each other.

[11] Following Lundborg (2013), average parental education is measured as four categories from 1 to 4: less than high school, GED or high school diploma, some college (no BA degree), and college degree or more. Following the approach of Ashenfelter and Krueger (1994), which is used by Lundborg (2013), the reports of siblings (twins) for parental education are averaged before obtaining a categorical parental education variable to address measurement error. In addition, the report of the sibling (twin) is used if there is only one report.

[12] Results are available for the non-significant outcomes upon request.

The results in Panel A suggest that non-white teen mothers report significantly worse self-reported health (about 3 units), are more likely to have at least 1 chronic condition (over 30 percentage points), and are more likely to be underweight (over 20 percentage points). Panel B demonstrates that greater parental education reduces the negative impact of teenage childbearing on health behavior, although all of the interactions are insignificant except for the probability of receiving a blood pressure test. Panel C demonstrates that teenage childbearing for women that are under of 45 at the time of the survey significantly impacts health behaviors, whereas teenage childbearing has insignificant effects for women that are over 45. More specifically, for mothers under 45 in the sample, teenage mothers engage in three less vigorous physical activities per month than non-teenage mothers. Similarly, they are around 14 and 17 percentage points less likely to visit a doctor, for the samples of siblings and twins, respectively. One possible explanation is that teenage childbearing adversely impacts health while young, but there is convergence of health over time. Another explanation is that social and economic conditions have changed, making it more difficult for younger generations to cope with teenage childbearing (for example, changes in the labor market have been mostly disfavorable non-high school and non-college educated workers).

These results should not, however, be considered conclusive since in some cases the comparison groups are very small (there are only 25 (12) and 18 (9) non-white mothers (families) in the samples for siblings and twins, respectively) and many of the interactions had the expected signs, but were not statistically significant at all conventional significance levels and therefore not reported.

5 Sensitivity Analysis

In this section, I address the various concerns in within-family estimations, which were discussed earlier. First, additional controls are introduced in the estimations for the sample of twins. Second, the effects are re-estimated for smaller samples of siblings and twins excluding arguably different siblings and twins. I also address the possibility that sisters may influence each other's fertility, health, and health behaviors. This section also considers how the estimates are affected by changing the definition of teenage childbearing as well as by using a linear age at first birth variable instead of a dummy variable.

Previous studies have shown that birth weight is an important factor for various long-run outcomes, such as education and income (Behrman and Rosenzweig, 2004; Black et al., 2007). Birth weight is available for only twins in the MIDUS data, and sample means in Table 1 show that teen mothers have lower birth weight, except for twins in the within-family sample. Earlier age at menarche has been shown to be positively correlated with teenage childbearing (Klepinger et al., 1999; Chevalier and Viitanen, 2003). Sample means in Table 1 show that teen mothers experience slightly earlier menarche compared to non-teen mothers. Controlling for birth weight and age at menarche does not substantially change the main results, and moreover, the effect of teenage childbearing on vigorous physical activity becomes more significant (columns (1) and (2) of Tables 6 and 7).

Following Webbink et al. (2008), I re-estimate the effects for a sample of twins excluding the pairs who differ at least 10 years in the timing of their first birth (10 pairs, of which 4 pairs are identical twins), which is expected to reduce heterogeneity within twins. The results shown in columns (3) and (4) of Tables 6 and 7 are again similar to the main results.

It is plausible that an older sister who has a teenage birth is more likely to influence her younger sister's decisions than the other way around (Holmlund, 2005).[13] As a robustness check, I repeated the regression analysis, excluding sister pairs where the older sister is the teenage mother. Thus, the estimated effects in column (5) of Tables 6 and 7 are identified for siblings where the younger one is a teenage mother, which in turn supposedly reduces the bias due to interactions between sisters. Furthermore, sisters who differ at least 10 years in the timing of their first birth are excluded in order to reduce the het-

[13] Note that if both sisters have teenage birth, they will not be in the identifying samples.

erogeneity bias within sisters. Results for this subsample shown in column (6) of Tables 6 and 7 indicate significant negative effects of teenage childbearing on vigorous exercise behavior (-1.221). Surprisingly, these estimates suggest that teenage childbearing reduces the number of chronic conditions (columns 5-6 in the Table 6).

The identifying assumption that the mother's siblings or twin provides a counterfactual is less likely to hold in cases where siblings display marked differences prior to childbearing or are exposed to different environments, including families and peers. Therefore, I exclude twins who report separation before the age of 15 (7 pairs), report not dressing alike or having dissimilar playmates as children, or both. The results are reported in Tables 8 and 9. The results are not significantly altered by the restrictions and, in particular, the estimates of "vigorous physical activity" and "blood pressure test" are robust (-1.426 and -0.148, respectively).

Finally, I use age 19 rather than 20 as the cutoff age for teenage childbearing and use age at first birth as a continuous-type variable.[14] Tables 10 and 11 report the results, where columns (1) and (2) use the baseline age cutoff (see columns (4) and (5) of Tables 3 and 4), columns (3) and (4) use age 19 as the cutoff for teenage childbearing, and columns (5) and (6) employ age at first birth as a linear independent variable. Defining teenage childbearing as having a child before the age of 19 reduces the number of teen mothers to 59 and 35 for siblings and twins, respectively, but it does not significantly affect the results. Using age at first birth as a linear independent variable implies the coefficients have opposite signs, and I find that smoking starting age and having a blood pressure test have the expected signs and are significant.

Measurement errors in the main explanatory variable (teenage childbearing) could bias the estimates towards zero. I use the MIDUS follow-up survey in 2004 to generate an additional age of childbearing observation. The reliability ratio for the sample of twins,

[14] I did not set the threshold to age 18 because the number of teen mothers are significantly reduced (25 and 14 in the samples of siblings and twins). Note that the linear effect of age at first birth is identified by sisters (twins) who differ in the timing of their age at first births by at least one year.

defined as the correlation between the two measures of teenage childbearing is 0.928.[15] This ratio is much higher than the ratio of 0.7 obtained in Webbink et al. (2008). A back of the envelope calculation therefore suggests that the downward bias in the estimated twin FE coefficient would be 11%.[16] Thus, it cannot be completely ruled out that measurement errors might have played a role in finding insignificant effects of teenage childbearing. Following Webbink et al. (2008), I address the problem of measurement error by using instrumental variables estimation (IV), in which teenage childbearing is instrumented using the value in the follow-up survey. Consistent with the pattern of the results in the study of Webbink et al. (2008), the IV estimates are generally larger than the main estimation results (also statistical significance of the estimates are similar with the main results).[17] In addition, excluding teenage mothers with childless siblings, who may have children later, might lead to measurement error (Webbink et al., 2008). The results are robust to the inclusion of siblings and twin pairs where childless mothers become mothers in the follow-up survey.[18]

6 Discussion on Mechanisms

This section explores potential mechanisms through which teenage childbearing affects health behaviors of mothers, including education, the number of children, marriage, and the spouse's quality in terms of educational attainment (see Table 13 for the sample means).[19] Towards this end, I employ these mechanisms as additional control variables in determining exercise and preventive care measures.[20] In general, if adding the control variable reduces

[15]The two measures are different for 5 (3) observations in the sample of twins (identical twins).

[16]Note that measurement error is non-classical since teenage childbearing is a dummy variable. The estimated correlation between the measures of teenage childbearing of a twin and her co-twin is 0.334.

[17]Results are available upon request.

[18]Ibid.

[19]Note that the sample means indicate that teenage mothers are less likely to graduate from high school, have more children, are less likely to marry, and are less likely to marry men with more education than high school, compared to non-teen mothers on average.

[20]Educational attainment is measured as four categories from 1 to 4: less than high school (the omitted reference category), GED or high school diploma, some college (no BA degree), and college degree or more.

the effect of teenage childbearing then the control variable is potentially an intermediate channel. On the other hand, if the effect does not change then it is not a potential mechanism. Since I find that there are heterogeneous effects of teenage childbearing on exercise by age, I allow the mechanisms for exercise behavior to vary by age.

The results are presented in Table 12 (columns 1 and 2 present the baseline results for comparison). Columns (3) and (4) demonstrate that including education reduces the effect of teenage childbearing on various outcomes. Moreover, the effect on exercise becomes insignificant and the effect on "blood pressure test" declines in significance, indicating that these variables might be intermediate channels in which teenage childbearing operates. Columns (5)-(8) demonstrate that the effect of teenage childbearing does not change, suggesting that the number of children and marriage are not intermediate channels. On the other hand, columns (9) and (10) suggest that spouse education is a possible channel.

The sample sizes in columns (9) and (10) are reduced due to missing values. The estimates in columns (9) and (10) are smaller than the main results in columns (1) and (2) when the main results are replicated for identical samples.[21]

Hence, reduced education of the mother and the quality of the spouse in terms of educational attainment are possible mechanisms through which teenage childbearing affects health behaviors.

7 Conclusion

This paper explores the causal effects of teenage childbearing on health outcomes and behaviors of mothers in the US by using within-family estimations. While the cross-sectional estimates show significant negative associations between teenage childbearing and health, these relationships mostly disappear once I control for family fixed effects. There is, how-

For marriage, I use a dummy indicating whether the respondent is currently married.

[21] For instance, the effect of teenage childbearing on "blood pressure test" is reduced from -0.189 (significant at the 5% level) to -0.171 (significant at the 10% level) for the sample of twins.

ever, evidence that teenage childbearing adversely affects exercise and preventive care behaviors of mothers. Further, I find that adverse effects of teenage childbearing are isolated to younger generations of mothers in the sample. Similarly, the effect of teenage childbearing is greater for minorities and women with less educated parents.

I address the concern that sibling and twin fixed effects might be heterogeneous within families by controlling for pre-childbearing factors and by restricting the samples to less heterogeneous subsets. The results are mostly robust to controlling for age at menarche and birth weight and to excluding siblings and twins with the following features: large differences in the timing of their first birth, an older sister that was a teenage mother, twins that were separate before the age of 15, and twins that reported large differences in the way they dressed or had different childhood playmates. Finally, the effect on preventive care is not sensitive to using 19 as the cutoff age rather than 20 and to using age at first birth as a linear independent variable.

The main channels in which teenage childbearing affects health behaviors is via reduced educational attainment and lower spouse quality in terms of educational attainment, whereas increased overall fertility and decreased likelihood of marriage do not seem to be important.

To sum up, teenage childbearing does have a causal negative effect on some health behaviors, but not most, and does not seem to affect the health of mothers. This is in contrast to the findings of Webbink et al. (2008), which find that smoking and obesity are affected, but the differences might be due to cross-country differences between Australia and the US as pointed out by Fletcher (2012). Future research might explore more health outcomes, such as mortality and seek a better understanding of the role of social and institutional factors that interact with teenage childbearing and explain these differences. Finally, the exploration of heterogeneous effects suggests that teenage childbearing might be important for certain subpopulations, thus policymakers might target these subpopulations to optimize the allocation of public finances.

Table 1: Summary Statistics
Sample means (standard deviations) and proportions

	Cross-section		Within-family (Identifying Samples)					
			Siblings		Twins		MZ twins	
	(1) Teen Mothers	(2) Nonteen Mothers	(3) Teen Mothers	(4) Nonteen Mothers	(5) Teen Mothers	(6) Nonteen Mothers	(7) Teen Mothers	(8)Nonteen Mothers
Age	49.53	48.23	49.15	49.67	46.10	46.10	43.52	43.52
	(11.64)	(12.19)	(12.42)	(12.74)	(12.14)	(12.14)	(9.57)	(9.57)
Age at first birth	17.85	24.58	18.04	23.36	18.16	23.25	18.41	23.11
	(1.23)	(3.99)	(1.20)	(3.50)	(1.02)	(3.44)	(0.93)	(3.41)
White	0.92	0.95	0.93	0.94	0.92	0.92	0.96	0.96
	(0.27)	(0.23)	(0.25)	(0.24)	(0.27)	(0.27)	(0.19)	(0.19)
High school graduate	0.62	0.95	0.70	0.85	0.64	0.84	0.81	0.93
	(0.49)	(0.22)	(0.46)	(0.36)	(0.48)	(0.37)	(0.40)	(0.27)
High school graduate, mother	0.33	0.55	0.40	0.39	0.43	0.43	0.56	0.56
	(0.47)	(0.50)	(0.49)	(0.49)	(0.50)	(0.50)	(0.51)	(0.51)
High school graduate, father	0.29	0.49	0.32	0.32	0.35	0.35	0.44	0.44
	(0.45)	(0.50)	(0.47)	(0.47)	(0.48)	(0.48)	(0.51)	(0.51)
Age at menarche	12.59	12.82	12.38	12.79	12.25	12.95	12.27	12.60
	(1.45)	(1.58)	(1.45)	(1.66)	(1.39)	(1.98)	(1.54)	(1.78)
Birth weight	2376.63	2380.01	-	-	2462.86	2427.85	2381.36	2393.17
	(676.94)	(635.06)	-	-	(609.48)	(650.95)	(534.76)	(669.29)
Health Outcomes								
Self Reported Health	7.20	7.73	7.30	7.50	7.42	7.63	7.67	7.83
	(1.93)	(1.55)	(2.09)	(1.66)	(2.14)	(1.75)	(2.08)	(1.49)
# of Chronic Conditions	3.12	2.48	2.79	3.17	2.62	3.15	2.17	3.29
	(3.06)	(2.43)	(2.55)	(2.85)	(2.37)	(3.05)	(1.86)	(2.74)
At least 1 Chronic Condition	0.80	0.77	0.78	0.81	0.77	0.80	0.71	0.79
	(0.40)	(0.42)	(0.42)	(0.39)	(0.43)	(0.40)	(0.46)	(0.41)
Physical Health	0.10	0.20	0.11	0.14	0.14	0.17	0.22	0.26
	(0.30)	(0.40)	(0.32)	(0.35)	(0.35)	(0.38)	(0.42)	(0.45)
Mental Health	0.19	0.28	0.19	0.20	0.18	0.25	0.26	0.37
	(0.39)	(0.45)	(0.39)	(0.40)	(0.39)	(0.44)	(0.45)	(0.49)
Self Evaluated Health	2.49	2.21	2.42	2.27	2.44	2.32	2.30	2.19
	(1.00)	(0.89)	(1.02)	(0.91)	(1.04)	(0.91)	(0.91)	(0.96)
Ever Had Cancer	0.15	0.09	0.13	0.10	0.09	0.03	0.07	0.00
	(0.36)	(0.29)	(0.34)	(0.30)	(0.29)	(0.17)	(0.27)	(0.00)
Observations	223	745	107	121	67	67	27	27

Notes: The "cross-section" sample consists of mothers who have a sister in the sample who is also a mother. The "within-family" samples consist of sisters who differ in the timing of their first births (teen mother vs nonteen mother). Birth weight is available for twins.

Table 2: Summary Statistics of Health Behaviors
Sample means (standard deviations) and proportions

	Cross-section		Within-family (Identifying Samples)					
			Siblings		Twins		MZ twins	
	(1) Teen Mothers	(2) Nonteen Mothers	(3) Teen Mothers	(4) Nonteen Mothers	(5) Teen Mothers	(6) Nonteen Mothers	(7) Teen Mothers	(8)Nonteen Mothers
Smoking								
Current Smoker	0.34	0.18	0.28	0.28	0.33	0.35	0.33	0.44
	(0.48)	(0.39)	(0.45)	(0.45)	(0.48)	(0.48)	(0.48)	(0.51)
Ever Smoker	0.55	0.42	0.48	0.52	0.53	0.53	0.52	0.59
	(0.50)	(0.49)	(0.50)	(0.50)	(0.50)	(0.50)	(0.51)	(0.50)
Starting Age Smoking	18.38	19.20	17.79	19.23	17.00	18.36	17.38	18.85
	(4.79)	(4.21)	(4.06)	(4.69)	(3.92)	(4.84)	(3.07)	(5.77)
Diet								
BMI	27.41	25.71	27.44	26.35	26.39	25.96	26.90	26.53
	(6.27)	(5.16)	(6.75)	(5.61)	(6.68)	(4.84)	(4.98)	(4.57)
Underweight (bmi<=18.5)	0.04	0.02	0.04	0.04	0.05	0.04	0.09	0.05
	(0.19)	(0.15)	(0.21)	(0.19)	(0.23)	(0.19)	(0.29)	(0.21)
Overweight (bmi>=25)	0.56	0.47	0.58	0.54	0.48	0.57	0.59	0.73
	(0.50)	(0.50)	(0.50)	(0.50)	(0.50)	(0.50)	(0.50)	(0.46)
Obese (bmi>=30)	0.29	0.19	0.25	0.24	0.14	0.21	0.18	0.27
	(0.45)	(0.39)	(0.44)	(0.43)	(0.35)	(0.41)	(0.39)	(0.46)
Exercise								
Vigorous Physical Activity	4.32	5.20	4.57	5.66	4.90	6.22	5.22	7.44
	(4.85)	(5.13)	(4.93)	(5.34)	(5.10)	(5.59)	(5.15)	(5.49)
Moderate Physical Activity	8.28	9.83	8.98	9.67	9.75	9.56	9.88	11.94
	(4.93)	(4.57)	(4.92)	(4.65)	(4.74)	(4.76)	(4.66)	(3.27)
Marijuana Use								
Marijuana Use	0.05	0.02	0.03	0.01	0.03	0.02	0.00	0.00
	(0.22)	(0.15)	(0.18)	(0.10)	(0.18)	(0.13)	(0.00)	(0.00)
Preventive Care								
Vitamin Take	0.56	0.55	0.56	0.54	0.52	0.46	0.50	0.50
	(0.50)	(0.50)	(0.50)	(0.50)	(0.50)	(0.50)	(0.51)	(0.51)
Blood Pressure Test	0.81	0.84	0.78	0.87	0.74	0.86	0.67	0.85
	(0.39)	(0.37)	(0.42)	(0.34)	(0.44)	(0.35)	(0.48)	(0.36)
Doctor Visit	0.83	0.90	0.84	0.87	0.79	0.88	0.87	0.96
	(0.38)	(0.30)	(0.37)	(0.33)	(0.41)	(0.33)	(0.34)	(0.21)
Health Behavior								
Work hard to stay healthy	2.38	2.20	2.32	2.28	2.18	2.29	2.29	1.92
	(1.36)	(1.22)	(1.28)	(1.56)	(1.16)	(1.42)	(1.46)	(0.72)
Effort on health	7.46	7.49	7.54	7.74	7.59	7.88	7.08	7.88
	(2.13)	(1.90)	(2.14)	(2.01)	(2.20)	(1.86)	(2.15)	(1.51)
Observations	223	745	107	121	67	67	27	27

Notes: The "cross-section" sample consists of mothers who have a sister in the sample who is also a mother. The "within-family" samples consist of sisters who differ in the timing of their first births (teen mother vs nonteen mother).

Table 3: Effects of teenage childbearing on health

	Cross-Section			Within-family		
	(1) Siblings	(2) Siblings	(3) Twins	(4) Siblings	(5) Twins	(6) MZ Twins
Self Reported Health	-0.533***	-0.530***	-0.596***	-0.171	-0.203	-0.167
	(0.147)	(0.147)	(0.200)	(0.218)	(0.308)	(0.432)
Observations (Groups)	892	890	462	328 (152)	182 (91)	74 (37)
# of Chronic Conditions	0.642***	0.579***	0.704**	-0.468	-0.533	-1.125**
	(0.258)	(0.254)	(0.355)	(0.327)	(0.403)	(0.473)
Observations (Groups)	903	901	468	333 (154)	184 (92)	74 (37)
Chronic Condition	0.033	0.023	0.017	-0.031	-0.033	-0.083
	(0.033)	(0.032)	(0.044)	(0.045)	(0.065)	(0.082)
Observations (Groups)	903	901	468	333 (154)	184 (92)	74 (37)
Physical Health	-0.103***	-0.105***	-0.078**	-0.025	-0.030	-0.037
	(0.026)	(0.025)	(0.035)	(0.036)	(0.048)	(0.075)
Observations (Groups)	960	952	516	354 (163)	202 (101)	80 (40)
Mental Health	-0.090***	-0.093***	-0.098**	-0.044	-0.075	-0.111
	(0.032)	(0.032)	(0.042)	(0.048)	(0.060)	(0.105)
Observations (Groups)	964	956	520	356 (164)	204 (102)	80 (40)
Self Evaluated Health	0.276***	0.321***	0.225**	0.042	0.121	0.111
	(0.075)	(0.073)	(0.097)	(0.109)	(0.147)	(0.220)
Observations (Groups)	942	936	506	352 (162)	200 (100)	78 (39)
Ever Had Cancer	0.058**	0.052**	0.057	0.058	0.060	0.074
	(0.026)	(0.026)	(0.035)	(0.036)	(0.040)	(0.060)
Observations (Groups)	964	956	520	356 (164)	204 (102)	80 (40)
Overall Index	0.624***	0.613***	0.592***	0.065	0.166	0.070
	(0.137)	(0.137)	(0.162)	(0.178)	(0.221)	(0.279)
Observations (Groups)	964	956	520	356 (164)	204 (102)	80 (40)
Controls						
Age	No	Yes	Yes	Yes	-	-
Race	No	Yes	Yes	-	-	-

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are in parentheses. Standard errors are adjusted for clustering within families in the "cross-section" regressions.

Table 4: Effects of teenage childbearing on health behaviors

	Cross-Section			Within-family		
	(1) Siblings	(2) Siblings	(3) Twins	(4) Siblings	(5) Twins	(6) MZ Twins
Smoking						
Current Smoker	0.160***	0.161***	0.182***	-0.013	-0.015	-0.111
	(0.037)	(0.037)	(0.050)	(0.051)	(0.059)	(0.073)
Observations (Groups)	962	954	518	352 (163)	202 (101)	80 (40)
Ever Smoker	0.130***	0.127***	0.197***	-0.055	-0.000	-0.074
	(0.041)	(0.041)	(0.053)	(0.053)	(0.067)	(0.081)
Observations (Groups)	962	954	518	352 (163)	202 (101)	80 (40)
Starting Age Smoking	-0.821	-0.915	-0.493	-1.644**	-1.360	-1.462
	(0.581)	(0.588)	(0.734)	(0.838)	(1.084)	(1.562)
Observations (Groups)	315	312	174	138 (68)	86 (43)	38 (19)
Diet						
BMI	1.699***	1.671***	1.956***	0.439	0.424	0.370
	(0.527)	(0.526)	(0.732)	(0.609)	(0.771)	(0.671)
Observations (Groups)	846	844	426	313 (145)	172 (86)	68 (34)
Underweight (bmi<=18.5)	0.014	0.015	0.029	0.002	0.018	0.045
	(0.014)	(0.014)	(0.022)	(0.022)	(0.035)	(0.052)
Observations (Groups)	846	844	426	313 (145)	172 (86)	68 (34)
Overweight (bmi>=25)	0.095**	0.091**	0.098*	-0.002	-0.089	-0.136
	(0.044)	(0.044)	(0.053)	(0.056)	(0.072)	(0.102)
Observations (Groups)	846	844	426	313 (145)	172 (86)	68 (34)
Obese (bmi>=30)	0.101***	0.099***	0.079	-0.034	-0.071	-0.091
	(0.038)	(0.038)	(0.050)	(0.046)	(0.056)	(0.097)
Observations (Groups)	846	844	426	313 (145)	172 (86)	68 (34)
Exercise						
Vigorous Physical Activity	-0.874**	-0.760**	-0.209	-0.807	-1.319*	-2.219*
	(0.384)	(0.381)	(0.533)	(0.563)	(0.782)	(1.192)
Observations (Groups)	891	889	458	327 (151)	178 (89)	74 (37)
Moderate Physical Activity	-1.555***	-1.392***	-0.858*	-0.909	0.194	-2.063*
	(0.405)	(0.397)	(0.515)	(0.558)	(0.750)	(1.109)
Observations (Groups)	894	892	462	326 (151)	178 (89)	72 (36)
Marijuana Use						
Marijuana Use	0.027	0.030*	0.015	0.016	0.017	-
	(0.017)	(0.017)	(0.019)	(0.022)	(0.031)	-
Observations (Groups)	896	894	464	327 (151)	180 (90)	74 (37)
Preventive Care						
Vitamin Take	0.009	0.004	0.012	0.063	0.054	-0.000
	(0.040)	(0.039)	(0.051)	(0.066)	(0.100)	(0.140)
Observations (Groups)	889	887	454	323 (149)	174 (87)	74 (37)
Blood Pressure Test	-0.030	-0.028	-0.038	-0.096**	-0.123**	-0.185*
	(0.033)	(0.032)	(0.040)	(0.043)	(0.061)	(0.103)
Observations (Groups)	945	939	506	348 (161)	198 (99)	80 (40)
Doctor Visit	-0.075***	-0.073***	-0.087**	-0.035	-0.088	-0.087
	(0.028)	(0.028)	(0.040)	(0.050)	(0.074)	(0.106)
Observations (Groups)	885	883	452	322 (149)	172 (86)	70 (35)
Health Behavior						
Work hard to stay healthy	0.180	0.209*	0.083	0.074	-0.109	0.375
	(0.117)	(0.115)	(0.153)	(0.184)	(0.227)	(0.290)
Observations (Groups)	853	851	432	306 (141)	160 (80)	74 (37)
Effort on health	-0.033	-0.102	-0.186	-0.145	-0.288	-0.792
	(0.177)	(0.173)	(0.234)	(0.260)	(0.362)	(0.530)
Observations (Groups)	892	890	460	329 (152)	182 (91)	74 (37)
Overall Index	0.560***	0.559***	0.574***	0.119	0.114	0.073
	(0.119)	(0.120)	(0.168)	(0.167)	(0.224)	(0.289)
Observations (Groups)	962	954	518	352 (163)	202 (101)	80 (40)
Controls						
Age	No	Yes	Yes	Yes	-	-
Race	No	Yes	Yes	-	-	-

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are in parentheses. Standard errors are adjusted for clustering within families in the "cross-section" regressions.

Table 5: Effects of teenage childbearing by race, parental education, and age (FE)

	(1) Siblings		(2) Twins	
Panel A. By Race	Effects on		Effects on	
	Teen Mother	Nonwhite*Teen Mother	Teen Mother	Nonwhite*Teen Mother
Self Reported Health	0.010	-2.785***	0.056	-3.056***
	(0.219)	(0.863)	(0.863)	(1.063)
Observations (Groups)	328 (152)		182 (91)	
Chronic Condition	-0.052	0.325*	-0.073	0.473**
	(0.046)	(0.184)	(0.067)	(0.230)
Observations (Groups)	333 (154)		184 (92)	
Underweight (bmi<=18.5)	-0.007	0.191*	0.000	0.333**
	(0.022)	(0.100)	(0.036)	(0.154)
Observations (Groups)	313 (145)		172 (86)	
Panel B. By Parental Education	Effects on		Effects on	
	Teen Mother	Parent Educ*Teen Mother	Teen Mother	Parent Educ*Teen Mother
Blood Pressure Test	-0.317***	0.055*	-0.372**	0.057
	(0.112)	(0.029)	(0.169)	(0.041)
Observations (Groups)	320 (147)		176 (88)	
Panel C. By Age	Effect on Teen Mother		Effect on Teen Mother	
	Age<=45	Age>45	Age<=45	Age>45
Vigorous Physical Activity	-3.183***	0.510	-3.231**	0.347
	(1.066)	(0.688)	(1.229)	(1.011)
Observations (Groups)	113 (62)	214 (104)	76 (38)	102 (51)
Doctor Visit	-0.140*	0.018	-0.172*	0.000
	(0.079)	(0.065)	(0.096)	(0.111)
Observations (Groups)	115 (63)	207 (101)	78 (39)	94 (47)

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are in parentheses. Regressions for the sample of siblings control for age.

Table 6: Effects of teenage childbearing on health-Additional Controls or Sample Restrictions

	Birth weight and age at menarche		Difference in age at first birth (DAFB) <10 years		Exclude oldest sisters teen mothers	Exclude oldest sisters teen mothers and DAFB>=10 years
	(1) Twins	(2) MZ Twins	(3) Twins	(4) MZ Twins	(5) Siblings	(6) Siblings
Self Reported Health	-0.230	-0.272	-0.347	-0.350	-0.015	-0.159
	(0.329)	(0.452)	(0.337)	(0.482)	(0.243)	(0.266)
Observations (Groups)	180 (90)	74 (37)	162 (81)	66 (33)	267 (126)	234 (110)
# of Chronic Conditions	-0.543	-1.116**	-0.620	-1.250**	-0.696**	-0.918**
	(0.437)	(0.521)	(0.462)	(0.543)	(0.326)	(0.370)
Observations (Groups)	182 (91)	74 (37)	164 (82)	66 (33)	272 (128)	239 (112)
Chronic Condition	-0.043	-0.068	0.000	-0.100	-0.056	-0.044
	(0.070)	(0.089)	(0.072)	(0.095)	(0.049)	(0.054)
Observations (Groups)	182 (91)	74 (37)	164 (82)	66 (33)	272 (128)	239 (112)
Physical Health	0.007	0.014	-0.036	-0.043	-0.036	-0.050
	(0.052)	(0.084)	(0.051)	(0.070)	(0.038)	(0.041)
Observations (Groups)	188 (97)	76 (38)	182 (91)	72 (36)	291 (137)	256 (120)
Mental Health	-0.043	-0.103	-0.070	-0.087	-0.010	-0.026
	(0.066)	(0.114)	(0.068)	(0.116)	(0.050)	(0.056)
Observations (Groups)	188 (97)	76 (38)	184 (92)	72 (36)	293 (138)	258 (121)
Self Evaluated Health	0.104	0.068	0.125	0.043	0.028	0.109
	(0.169)	(0.256)	(0.162)	(0.242)	(0.120)	(0.135)
Observations (Groups)	186 (93)	74 (37)	180 (90)	70 (35)	291 (136)	254 (119)
Ever Had Cancer	0.050	0.076	0.053	0.043	0.065*	0.068
	(0.046)	(0.072)	(0.044)	(0.061)	(0.038)	(0.043)
Observations (Groups)	188 (97)	76 (38)	184 (92)	72 (36)	295 (138)	260 (121)

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are in parentheses. Columns (5) and (6) control for age.

Table 7: Effects of teenage childbearing on health behaviors-Additional Controls or Sample Restrictions

	Birth weight and age at menarche		Difference in age at first birth (DAFB) <10 years		Exclude oldest sisters teen mothers	Exclude oldest sisters teen mothers and DAFB >=10 years
	(1) Twins	(2) MZ Twins	(3) Twins	(4) MZ Twins	(5) Siblings	(6) Siblings
Smoking						
Current Smoker	0.006	-0.098	0.018	-0.087	-0.020	0.007
	(0.077)	(0.080)	(0.073)	(0.077)	(0.053)	(0.057)
Observations (Groups)	188 (97)	76 (38)	182 (91)	72 (36)	293 (137)	258 (120)
Ever Smoker	0.023	-0.024	-0.000	-0.043	-0.034	-0.010
	(0.075)	(0.091)	(0.072)	(0.086)	(0.056)	(0.062)
Observations (Groups)	188 (97)	76 (38)	182 (91)	72 (36)	293 (137)	258 (120)
Starting Age Smoking	-2.516*	-3.883*	-0.333	-0.583	-1.294	-1.241
	(1.356)	(1.812)	(1.144)	(1.487)	(0.858)	(1.007)
Observations (Groups)	80 (40)	36 (18)	78 (39)	36 (18)	118 (58)	102 (51)
Diet						
BMI	0.354	0.154	0.356	0.770	0.649	1.120
	(0.809)	(0.723)	(0.877)	(0.759)	(0.650)	(0.726)
Observations (Groups)	170 (85)	68 (34)	152 (76)	60 (30)	254 (119)	223 (104)
Underweight (bmi<=18.5)	0.028	0.074	0.022	0.056	-0.008	-0.018
	(0.038)	(0.056)	(0.042)	(0.061)	(0.027)	(0.030)
Observations (Groups)	170 (85)	68 (34)	152 (76)	60 (30)	254 (119)	223 (104)
Overweight (bmi>=25)	-0.115	-0.128	-0.065	-0.056	-0.014	0.002
	(0.077)	(0.114)	(0.078)	(0.107)	(0.059)	(0.066)
Observations (Groups)	170 (85)	68 (34)	152 (76)	60 (30)	254 (119)	223 (104)
Obese (bmi>=30)	-0.072	-0.081	-0.065	-0.056	-0.009	0.006
	(0.060)	(0.108)	(0.063)	(0.107)	(0.051)	(0.058)
Observations (Groups)	170 (85)	68 (34)	152 (76)	60 (30)	254 (119)	223 (104)
Exercise						
Vigorous Physical Activity	-1.918**	-2.833**	-1.714*	-3.113**	-1.051	-1.221*
	(0.838)	(1.288)	(0.897)	(1.259)	(0.636)	(0.728)
Observations (Groups)	176 (88)	74 (37)	158 (79)	66 (33)	266 (125)	233 (109)
Moderate Physical Activity	0.126	-2.332*	0.141	-2.475**	-0.623	-0.580
	(0.790)	(1.219)	(0.846)	(1.172)	(0.633)	(0.713)
Observations (Groups)	176 (88)	72 (36)	158 (79)	64 (32)	265 (125)	232 (109)
Marijuana Use						
Marijuana Use	0.006	-	0.021	-	0.016	0.016
	(0.034)	-	(0.036)	-	(0.024)	(0.028)
Observations (Groups)	178 (89)	74 (37)	160 (80)	66 (33)	266 (125)	233 (109)
Preventive Care						
Vitamin Take	0.091	0.082	0.043	-0.050	0.061	0.045
	(0.104)	(0.149)	(0.108)	(0.148)	(0.075)	(0.084)
Observations (Groups)	172 (86)	74 (37)	154 (77)	66 (33)	262 (123)	229 (107)
Blood Pressure Test	-0.106	-0.077	-0.109	-0.174	-0.067	-0.073
	(0.068)	(0.112)	(0.068)	(0.113)	(0.049)	(0.053)
Observations (Groups)	184 (92)	76 (38)	178 (89)	72 (36)	289 (135)	254 (118)
Doctor Visit	-0.069	-0.102	-0.064	-0.053	-0.015	-0.009
	(0.080)	(0.119)	(0.080)	(0.118)	(0.055)	(0.060)
Observations (Groups)	172 (86)	70 (35)	152 (76)	62 (31)	261 (123)	228 (107)
Health Behavior						
Work hard to stay healthy	-0.114	0.329	-0.109	0.500	-0.063	-0.035
	(0.242)	(0.316)	(0.255)	(0.332)	(0.208)	(0.220)
Observations (Groups)	158 (79)	68 (34)	142 (71)	60 (30)	246 (115)	215 (100)
Effort on health	-0.164	-0.930	-0.265	-0.650	-0.107	-0.186
	(0.381)	(0.561)	(0.398)	(0.558)	(0.299)	(0.326)
Observations (Groups)	180 (90)	74 (37)	162 (81)	66 (33)	268 (126)	235 (110)

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are in parentheses. Columns (5) and (6) control for age.

Table 8: Effects of teenage childbearing on health-Twins Sample Restrictions

	Exclude twins separated before 15 years old	Exlude twins never dress alike	Exlude twins never have same playmates	Exlude twins never dress alike or never have same playmates
	(1) Twins	(2) Twins	(3) Twins	(4) Twins
Self Reported Health	-0.304	-0.288	-0.093	-0.250
	(0.309)	(0.330)	(0.325)	(0.327)
Observations (Groups)	168 (84)	162 (81)	170 (85)	162 (81)
# of Chronic Conditions	-0.404	-0.566	-0.352	-0.385
	(0.411)	(0.425)	(0.391)	(0.403)
Observations (Groups)	170 (85)	164 (82)	170 (85)	162 (81)
Chronic Condition	-0.035	-0.075	-0.019	-0.038
	(0.068)	(0.068)	(0.070)	(0.069)
Observations (Groups)	170 (85)	164 (82)	170 (85)	162 (81)
Physical Health	-0.032	-0.018	-0.017	-0.035
	(0.050)	(0.051)	(0.050)	(0.055)
Observations (Groups)	188 (94)	178 (89)	188 (94)	178 (89)
Mental Health	-0.078	-0.017	-0.066	-0.052
	(0.064)	(0.062)	(0.064)	(0.065)
Observations (Groups)	190 (95)	180 (90)	190 (95)	180 (90)
Self Evaluated Health	0.190	0.070	0.050	0.053
	(0.145)	(0.160)	(0.152)	(0.157)
Observations (Groups)	186 (93)	176 (88)	186 (93)	176 (88)
Ever Had Cancer	0.063	0.017	0.049	0.034
	(0.042)	(0.039)	(0.041)	(0.042)
Observations (Groups)	190 (95)	180 (90)	190 (95)	180 (90)

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are in parentheses.

Table 9: Effects of teenage childbearing on health behaviors-Twins Sample Restrictions

	Exclude twins separated before 15 years old	Exlude twins never dress alike	Exlude twins never have same playmates	Exlude twins never dress alike or never have same playmates
	(1) Twins	(2) Twins	(3) Twins	(4) Twins
Smoking				
Current Smoker	0.000	-0.070	-0.033	-0.048
	(0.070)	(0.073)	(0.072)	(0.071)
Observations (Groups)	188 (94)	178 (89)	188 (94)	192 (96)
Ever Smoker	0.016	-0.053	-0.000	0.000
	(0.069)	(0.072)	(0.071)	(0.069)
Observations (Groups)	188 (94)	178 (89)	188 (94)	192 (96)
Starting Age Smoking	-1.391	-2.000*	-1.318	-1.375
	(1.188)	(1.066)	(1.205)	(1.126)
Observations (Groups)	76 (38)	74 (37)	78 (39)	82 (41)
Diet				
BMI	0.485	0.105	0.598	0.534
	(0.802)	(0.702)	(0.813)	(0.793)
Observations (Groups)	160 (80)	152 (76)	160 (80)	164 (82)
Underweight (bmi<=18.5)	0.019	0.020	0.020	0.019
	(0.031)	(0.040)	(0.039)	(0.037)
Observations (Groups)	160 (80)	152 (76)	160 (80)	164 (82)
Overweight (bmi>=25)	-0.094	-0.082	-0.078	-0.094
	(0.072)	(0.077)	(0.077)	(0.076)
Observations (Groups)	160 (80)	152 (76)	160 (80)	164 (82)
Obese (bmi>=30)	-0.075	-0.082	-0.059	-0.057
	(0.059)	(0.056)	(0.059)	(0.057)
Observations (Groups)	160 (80)	152 (76)	160 (80)	164 (82)
Exercise				
Vigorous Physical Activity	-1.357	-1.309	-1.604*	-1.426*
	(0.816)	(0.879)	(0.849)	(0.848)
Observations (Groups)	166 (83)	158 (79)	166 (83)	168 (84)
Moderate Physical Activity	-0.031	0.172	0.080	0.278
	(0.743)	(0.847)	(0.791)	(0.794)
Observations (Groups)	166 (83)	158 (79)	166 (83)	168 (84)
Marijuana Use				
Marijuana Use	0.036	0.020	0.019	0.000
	(0.030)	(0.035)	(0.026)	(0.030)
Observations (Groups)	166 (83)	160 (80)	166 (83)	170 (85)
Preventive Care				
Vitamin Take	0.057	0.059	0.058	0.074
	(0.101)	(0.103)	(0.103)	(0.101)
Observations (Groups)	160 (80)	158 (79)	164 (82)	168 (84)
Blood Pressure Test	-0.097	-0.123*	-0.153**	-0.148**
	(0.063)	(0.068)	(0.065)	(0.063)
Observations (Groups)	184 (92)	178 (89)	184 (92)	188 (94)
Doctor Visit	-0.109	-0.100	-0.078	-0.075
	(0.073)	(0.081)	(0.079)	(0.076)
Observations (Groups)	162 (81)	152 (76)	158 (79)	162 (81)
Health Behavior				
Work hard to stay healthy	-0.154	-0.163	0.020	-0.000
	(0.239)	(0.250)	(0.227)	(0.220)
Observations (Groups)	148 (74)	144 (72)	146 (73)	150 (75)
Effort on health	-0.304	-0.038	-0.315	-0.286
	(0.371)	(0.386)	(0.383)	(0.373)
Observations (Groups)	168 (84)	162 (81)	170 (85)	174 (87)

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are in parentheses.

Table 10: Effects of teenage childbearing on health-Different Age

	Within-family					
	Teen Mother<20 years		Teen Mother<19 years		Age at first birth	
	(1) Siblings	(2) Twins	(3) Siblings	(4) Twins	(5) Siblings	(6) Twins
Self Reported Health	-0.171	-0.203	-0.049	-0.091	0.024	-0.014
	(0.218)	(0.308)	(0.262)	(0.398)	(0.032)	(0.048)
# of Chronic Conditions	-0.468	-0.533	-0.441	-0.098	0.012	0.082
	(0.327)	(0.403)	(0.395)	(0.493)	(0.049)	(0.062)
Chronic Condition	-0.031	-0.033	-0.049	-0.024	0.006	0.015
	(0.045)	(0.065)	(0.054)	(0.078)	(0.007)	(0.010)
Physical Health	-0.025	-0.030	-0.026	-0.024	0.003	0.002
	(0.036)	(0.048)	(0.044)	(0.060)	(0.005)	(0.008)
Mental Health	-0.044	-0.075	-0.059	-0.023	0.005	0.009
	(0.048)	(0.060)	(0.058)	(0.076)	(0.007)	(0.010)
Self Evaluated Health	0.042	0.121	0.105	-0.023	-0.009	-0.018
	(0.109)	(0.147)	(0.133)	(0.183)	(0.016)	(0.023)
Ever Had Cancer	0.058	0.060	0.059	0.023	-0.010*	-0.008
	(0.036)	(0.040)	(0.044)	(0.050)	(0.005)	(0.006)
Controls						
Age	Yes	-	Yes	-	Yes	-
Race	-	-	-	-	-	-

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are in parentheses.

Table 11: Effects of teenage childbearing on health behaviors-Different Age

	Within-family					
	Teen Mother<20 years		Teen Mother<19 years		Age at first birth	
	(1) Siblings	(2) Twins	(3) Siblings	(4) Twins	(5) Siblings	(6) Twins
Smoking						
Current Smoker	-0.013	-0.015	-0.016	-0.047	0.007	0.008
	(0.051)	(0.059)	(0.062)	(0.087)	(0.008)	(0.011)
Ever Smoker	-0.055	-0.000	-0.038	-0.029	0.010	-0.002
	(0.053)	(0.067)	(0.074)	(0.095)	(0.008)	(0.011)
Starting Age Smoking	-1.644**	-1.360	-1.770*	-0.706	0.228**	0.324*
	(0.838)	(1.084)	(0.959)	(1.334)	(0.113)	(0.166)
Diet						
BMI	0.439	0.424	0.616	0.786	-0.060	-0.079
	(0.609)	(0.771)	(0.728)	(0.946)	(0.090)	(0.118)
Underweight (bmi<=18.5)	0.002	0.018	0.040	0.054	-0.002	-0.002
	(0.022)	(0.035)	(0.026)	(0.043)	(0.003)	(0.005)
Overweight (bmi>=25)	-0.002	-0.089	-0.020	-0.162*	-0.000	0.014
	(0.056)	(0.072)	(0.067)	(0.087)	(0.008)	(0.011)
Obese (bmi>=30)	-0.034	-0.071	0.014	0.027	0.003	0.013
	(0.046)	(0.056)	(0.055)	(0.069)	(0.007)	(0.008)
Exercise						
Vigorous Physical Activity	-0.807	-1.319*	-0.327	-1.218	0.057	0.067
	(0.563)	(0.782)	(0.685)	(0.983)	(0.084)	(0.124)
Moderate Physical Activity	-0.909	0.194	-1.031	-0.301	0.112	0.018
	(0.558)	(0.750)	(0.674)	(0.914)	(0.082)	(0.115)
Marijuana Use						
Marijuana Use	0.016	0.017	0.038	0.026	-0.005	-0.005
	(0.022)	(0.031)	(0.026)	(0.038)	(0.003)	(0.005)
Preventive Care						
Vitamin Take	0.063	0.054	0.079	0.156	-0.007	-0.012
	(0.066)	(0.100)	(0.091)	(0.128)	(0.010)	(0.015)
Blood Pressure Test	-0.096**	-0.123**	-0.143*	-0.176*	0.011*	0.019*
	(0.043)	(0.061)	(0.063)	(0.088)	(0.006)	(0.010)
Doctor Visit	-0.035	-0.088	-0.036	-0.079	0.004	0.016
	(0.050)	(0.074)	(0.061)	(0.091)	(0.007)	(0.011)
Health Behavior						
Work hard to stay healthy	0.074	-0.109	0.092	-0.161	0.007	0.017
	(0.184)	(0.227)	(0.256)	(0.311)	(0.029)	(0.037)
Effort on health	-0.145	-0.288	-0.481	-0.675	0.008	0.031
	(0.260)	(0.362)	(0.310)	(0.436)	(0.038)	(0.056)
Controls						
Age	Yes	-	Yes	-	Yes	-
Race	-	-	-	-	-	-

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are in parentheses.

Table 12: Effects on exercise and preventive care after controlling for possible mechanisms

	Main Results		Education		Number of children		Married		Spouse Education	
	(1) Siblings	(2) Twins	(3) Siblings	(4) Twins	(5) Siblings	(6) Twins	(7) Siblings	(8) Twins	(9) Siblings	(10) Twins
Vigorous Physical Activity	-0.807	-1.319*	-0.712	-1.215	-0.853	-1.398*	-0.834	-1.357	-0.726	-2.536*
	(0.563)	(0.782)	(0.590)	(0.841)	(0.576)	(0.811)	(0.569)	(0.818)	(0.900)	(1.269)
Observations (Groups)	327 (151)	178 (89)	327 (151)	178 (89)	327 (151)	178 (89)	327 (151)	178 (89)	236 (142)	133 (84)
Vigorous Physical Activity	-3.183***	-3.231**	-3.031***	-2.954**	-3.149***	-3.232**	-3.198***	-3.259***	-5.441***	-5.991***
(Age<=45)	(1.066)	(1.229)	(1.061)	(1.286)	(1.090)	(1.263)	(1.000)	(1.083)	(1.593)	(1.928)
Observations (Groups)	113 (62)	76 (38)	113 (62)	76 (38)	113 (62)	76 (38)	113 (62)	76 (38)	80 (53)	56 (37)
Blood Pressure Test	-0.096**	-0.123**	-0.091**	-0.118*	-0.087**	-0.128**	-0.092**	-0.116*	-0.150***	-0.171*
	(0.043)	(0.061)	(0.045)	(0.068)	(0.044)	(0.063)	(0.043)	(0.062)	(0.057)	(0.091)
Observations (Groups)	348 (161)	198 (99)	348 (161)	198 (99)	348 (161)	198 (99)	348 (161)	198 (99)	251 (151)	147 (93)
Controls										
Age	Yes	-	Yes	-	Yes	-	Yes	-	Yes	-
Education	No	No	Yes	Yes	No	No	No	No	No	No
Number of Children	No	No	No	No	Yes	Yes	No	No	No	No
Married	No	No	No	No	No	No	Yes	Yes	No	No
Spouse Education	No	No	No	No	No	No	No	No	Yes	Yes

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are in parentheses.

Table 13: Sample Means (standard deviations) for possible mechanisms

	Within-family (Identifying Samples)			
	Siblings		Twins	
	(1) Teen Mothers	(2) Nonteen Mothers	(3) Teen Mothers	(4) Nonteen Mothers
High school graduate	0.70	0.85	0.64	0.84
	(0.46)	(0.36)	(0.48)	(0.37)
Number of children	2.91	2.63	2.84	2.49
	(1.42)	(1.32)	(1.42)	(1.17)
Married	0.66	0.70	0.67	0.75
	(0.47)	(0.46)	(0.47)	(0.44)
Spouse more than	0.37	0.54	0.40	0.51
high school	(0.49)	(0.50)	(0.49)	(0.50)
Observations	107	121	67	67

The "within-family" samples consist of sisters who differ in the timing of their first births (teen mother vs nonteen mother).

Appendix

Table A1. Equality tests for the effects of teenage childbearing on health

	Siblings			Twins		
	Cross-Section	Within-family	Equality Test	Cross-Section	Within-family	Equality Test
Self Reported Health	-0.530***	-0.171	p= 0.0325	-0.596***	-0.203	p= 0.1140
	(0.147)	(0.218)		(0.200)	(0.308)	
Observations (Groups)	890	328 (152)		462	182 (91)	
# of Chronic Conditions	0.579***	-0.468	p= 0.0007	0.704**	-0.533	p= 0.0061
	(0.254)	(0.327)		(0.355)	(0.403)	
Observations (Groups)	901	333 (154)		468	184 (92)	
Chronic Condition	0.023	-0.031	p= 0.1740	0.017	-0.033	p= 0.3812
	(0.032)	(0.045)		(0.044)	(0.065)	
Observations (Groups)	901	333 (154)		468	184 (92)	
Physical Health	-0.105***	-0.025	p= 0.0163	-0.078**	-0.030	p= 0.2973
	(0.025)	(0.036)		(0.035)	(0.048)	
Observations (Groups)	952	354 (163)		516	202 (101)	
Mental Health	-0.093***	-0.044	p= 0.2972	-0.098**	-0.075	p= 0.6878
	(0.032)	(0.035)		(0.042)	(0.060)	
Observations (Groups)	956	356 (164)		520	204 (102)	
Self Evaluated Health	0.321***	0.042	p= 0.0023	0.225**	0.121	p= 0.3900
	(0.073)	(0.109)		(0.097)	(0.147)	
Observations (Groups)	936	352 (162)		506	200 (100)	
Ever Had Cancer	0.052**	0.058	p= 0.8959	0.057	0.060	p= 0.9470
	(0.026)	(0.036)		(0.035)	(0.040)	
Observations (Groups)	956	356 (164)		520	204 (102)	
Overall Index	0.613***	0.065	p= 0.0007	0.592***	0.166	p= 0.0520
	(0.137)	(0.178)		(0.162)	(0.221)	
Observations (Groups)	956	356 (164)		520	204 (102)	
Controls						
Age	Yes	Yes		Yes	-	
Race	Yes	-		Yes	-	

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (reported in parentheses) are adjusted for clustering within families in the "cross-section" regressions. p-values of χ^2 test are reported.

Table A2. Equality tests for the effects of teenage childbearing on health behaviors

	Siblings			Twins		
	Cross-Section	Within-family	Equality Test	Cross-Section	Within-family	Equality Test
Smoking						
Current Smoker	0.161***	-0.013	p= 0.0001	0.182***	-0.015	p= 0.0022
	(0.037)	(0.051)		(0.050)	(0.059)	
Observations (Groups)	954	352 (163)		518	202 (101)	
Ever Smoker	0.127***	-0.055	p= 0.0002	0.197***	-0.000	p= 0.0027
	(0.041)	(0.053)		(0.053)	(0.067)	
Observations (Groups)	954	352 (163)		518	202 (101)	
Starting Age Smoking	-0.915	-1.644**	p= 0.3566	-0.493	-1.360	p= 0.3342
	(0.588)	(0.838)		(0.734)	(1.084)	
Observations (Groups)	312	138 (68)		174	86 (43)	
Diet						
BMI	1.671***	0.439	p= 0.0397	1.956***	0.424	p= 0.0474
	(0.526)	(0.609)		(0.732)	(0.771)	
Observations (Groups)	844	313 (145)		426	172 (86)	
Underweight (bmi<=18.5)	0.015	0.002	p= 0.4651	0.029	0.018	p= 0.6791
	(0.014)	(0.022)		(0.022)	(0.035)	
Observations (Groups)	844	313 (145)		426	172 (86)	
Overweight (bmi>=25)	0.091**	-0.002	p= 0.0802	0.098*	-0.089	p= 0.0110
	(0.044)	(0.056)		(0.053)	(0.072)	
Observations (Groups)	844	313 (145)		426	172 (86)	
Obese (bmi>=30)	0.099***	-0.034	p= 0.0040	0.079	-0.071	p= 0.0141
	(0.038)	(0.046)		(0.050)	(0.056)	
Observations (Groups)	844	313 (145)		426	172 (86)	
Exercise						
Vigorous Physical Activity	-0.760**	-0.807	p= 0.9210	-0.209	-1.319*	p= 0.0818
	(0.381)	(0.563)		(0.533)	(0.782)	
Observations (Groups)	889	327 (151)		458	118 (59)	
Moderate Physical Activity	-1.392***	-0.909	p= 0.3266	-0.858*	0.194	p= 0.0692
	(0.397)	(0.558)		(0.515)	(0.750)	
Observations (Groups)	892	326 (151)		462	178 (89)	
Marijuana Use						
Marijuana Use	0.030*	0.016	p= 0.4552	0.015	0.017	p= 0.9103
	(0.017)	(0.022)		(0.019)	(0.031)	
Observations (Groups)	894	327 (151)		464	180 (90)	
Preventive Care						
Vitamin Take	0.004	0.063	p= 0.2508	0.012	0.054	p= 0.5893
	(0.039)	(0.066)		(0.051)	(0.100)	
Observations (Groups)	887	323 (149)		454	174 (87)	
Blood Pressure Test	-0.028	-0.096**	p= 0.0941	-0.038	-0.123**	p= 0.0993
	(0.032)	(0.043)		(0.040)	(0.061)	
Observations (Groups)	939	348 (161)		506	198 (99)	
Doctor Visit	-0.073***	-0.035	p= 0.2741	-0.087**	-0.088	p= 0.9955
	(0.028)	(0.050)		(0.040)	(0.074)	
Observations (Groups)	883	322 (149)		452	172 (86)	
Health Behavior						
Work hard to stay healthy	0.209*	0.074	p= 0.3934	0.083	-0.109	p= 0.3602
	(0.115)	(0.184)		(0.153)	(0.227)	
Observations (Groups)	851	306 (141)		432	160 (80)	
Effort on health	-0.102	-0.145	p= 0.8511	-0.186	-0.288	p= 0.7378
	(0.173)	(0.260)		(0.234)	(0.362)	
Observations (Groups)	890	329 (152)		460	182 (91)	
Overall Index	0.559***	0.119	p=0.0028	0.574***	0.114	p= 0.0247
	(0.120)	(0.167)		(0.168)	(0.224)	
Observations (Groups)	954	352 (163)		518	202 (101)	
Controls						
Age	Yes	Yes		Yes	-	
Race	Yes	-		Yes	-	

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (reported in parentheses) are adjusted for clustering within families in the "cross-section" regressions. p-values of χ^2 test are reported.

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