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Three Essays in Health and Education

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THREE ESSAYS IN HEALTH AND EDUCATION

by

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ABSTRACT

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Under the Supervision of Professor Scott Adams

The goal of this dissertation is to apply empirical methodologies to analyze multiple topics in economics of education and health economics which have clear policy implications.

Chapter 1 analyzes the effect of negative publicity of child abuse scandal on Catholic schools. Public notices of child abuse have surrounded Catholic Church leadership for decades, but intensified after the 2002 coverage by the Boston Globe and the ensuing accelerated media coverage. Using diocese level panel data of Catholic school enrollment, reports of abuse after 2002 appear to have a negative, long-lasting effect on both demand and supply of Catholic schools. No effect is observed from notices prior to 2002, suggesting the public awareness of the scandal from abuse reports, combined with mass media coverage, led to observable effects on Catholic School enrollment. Public notices of allegations related to the abuse scandal can explain about two-thirds of the decline in Catholic school enrollment share and the number of Catholic schools.

Chapter 2 studies the effect of various state level policies as well as receiving a physician recommendation on the decision to uptake Human Papillomavirus (HPV) vaccine. HPV is the most common sexually transmitted source of infection in the United States. Recently, two vaccines were developed to provide immunization against certain types of HPV. In addition to physician recommendations to take these vaccines, different states have adopted a wide range of policies in order to increase the vaccination rate, specifically among younger females. In this study, I use survey data to examine the effect of the two most common adopted policies, school mandates and provision of educational content for parents about the virus and its immunization, as well as the effects of physician recommendations. The results indicate that the effect of policies on encouraging the HPV vaccination has been very limited at best, but the effect of receiving a physician's advice for the HPV immunization is significant.

Chapter 3 attempts to investigate the behavioral response to HPV vaccine. Immunization can cause moral hazard by reducing the cost of risky behaviors. In this study, I examine the effect of HPV vaccination on participation in Papanicolaou test (Pap test). The Pap test is a diagnostic screening test to detect potentially precancerous and cancerous process in the transformation zone. The Pap test is strongly recommended for women between 21-65 years old even after taking the HPV vaccine. If there is a reduction in willingness to have a Pap test as a result of HPV vaccination, it should be a concern for public health policy makers. The results show no evidence of moral hazard, more specifically in the short-run. The estimates range from zero to a positive effect of HPV vaccine initiation on having a Pap test.

This dissertation is gratefully dedicated to my parents. Without your support, this would not have been possible.

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Chapter 1: Child Abuse Scandal Publicity and Catholic School Enrollment

1.1. Introduction

Catholic schools play a significant role in private and religious education in the United States. Enrollment in Catholic schools is associated with a higher probability of high school graduation and college attendance, especially for urban minorities. It is also associated with greater labor market outcomes and reductions in risky behaviors, such as teenage sexual activity and drug use (Altonji et al., 2005b; Figlio & Ludwig, 2000; Kim, 2011; Neal, 1997). On the other hand, public schools benefit from the increased competition that Catholic schools provide (Carattini et al., 2012; Hoxby, 1994).

Despite the benefits derived from Catholic schooling, the percentage of all private school students enrolled in Catholic schools decreased from 45 percent in 1995-1996 to 39 percent in 2009-2010. Much of this reduction is due to declining enrollment in parochial schools. Parochial schools are run by parishes, not by diocese or independently (National Center for Education Statistics, 2012). A total of 1,856 schools were reported closed or consolidated between 2004 and 2014. However, most inner-city and urban schools managed to remain open (National Catholic Educational Association, 2014)

There are several potential explanations that describe the aforementioned decline in the demand for Catholic schools: demographic changes, socioeconomic changes, and negative publicity that arose from the child abuse scandal crisis among leadership in the Catholic Church. The goal of this paper is to investigate the impact the Catholic child abuse scandal had on the availability of Catholic schools as well as on their share from total enrollment. The mass media coverage of the scandal accelerated profoundly in 2002,

when the Boston Globe published a series of articles on the issue. Soon after, the abuse became engrained in the national consciousness and the volume of victims that came forward and made allegations against the Catholic Church accelerated. This is certainly one of the largest institutional crisis in the history of the Catholic Church.

I find that the scandal led to a long-lasting decline in Catholic school enrollment share and the number of Catholic schools. Within 7 years after the scandal, the affected outcomes did not revert to pre-scandal levels. Public notices of allegations related to the abuse scandal account for about two-thirds of the decline in Catholic school enrollment share and the number of Catholic schools. The results also imply that there is a meaningful difference between pre- and post- 2002 in terms of the effect of abuse allegations on Catholic school enrollment share and number of schools.

I provide suggestive evidence that the aforementioned difference between these two time periods stems from a fundamental difference in media coverage of the scandal before and after 2002. News media has the power to influence the visibility of the events in public's mind by highlighting a limited number of key public issues at any given time. The more frequently and prominently the news media covers an issue, the more that issue becomes accessible in audience's mind and that issue is considered as more important (Merritt & McCombs, 2004). Allegations of child abuse in the Catholic Church received highlighted and emphatic coverage only after 2002. The significant and distinguished media coverage that the child abuse notices received after 2002 increased the public awareness of this issue and brought it to the forefront of the public's attention.

This paper contributes to a literature that studies the impact of the scandal on different outcomes. Hungerman (2013) looked at the relationship between abuse

allegations and religious participation and charitable activities with state-level data. He found that the scandal led to a substitution away from Catholicism. Dills and Hernandez-Julian (2012) examined the effect of the scandal on Catholic school availability and enrollment share with diocese-level data and found that the scandal had a small negative effect on the availability of Catholic schools and had no effect on the enrollment share. Bottan and Perez-Truglia (2011) investigated the relationship between abuse allegations and provision of social services with the zip code-level data. They found that the scandal caused a long-lasting decline in religious congregation, charitable giving, and the provision of social services, including Catholic schools. In this paper, I contribute by modeling the differing effects of abuse notices before and after 2002. I also look at the effect of negative publicity on Catholic schools over time.

The approach of this paper is primarily to identify the effect of the enhanced public awareness that occurred after 2002. Using diocese level panel data on Catholic school enrollment coupled with a rich set of control variables, I empirically examine the effect of publicity, and consequently, public awareness of child abuse scandal on Catholic school enrollment. The empirical strategy is to employ this peak in media coverage to construct an estimate of the effect of awareness of abuse on Catholic School enrollment. I contend that the timing of the peak in media coverage of the issue is exogenous and the following reports of abuse therefore have the potential to have real effects on parental behavior.

The rest of the paper is organized as follows. Section 1.2 provides background on the scope and the consequences of the Catholic child abuse scandal and reviews the relevant literature. In Section 1.3, I describe the data. Section 1.4 presents the methodology. I

discuss the results of different specifications and analyze the effect of negative publicity over time in section 1.5. Section 1.6 concludes.

1.2. Background

The Catholic child abuse scandal refers to a series of allegations of child abuse crimes committed by Catholic orders. Victims of the scandal were as young as three years old, with the majority between the ages of 10 and 14; 82% of the victims were male. The U.S. bishops have reported receiving allegations of abuse from 16,324 victims by 6,115 priests between 1950 and 2012. These numbers are believed to be underestimated (Bishopaccountability.org and United States Conference of Catholic Bishops report, 2012). More than 3,000 civil lawsuits have been filed from 1984-2009 and some estimates show that the Catholic Church has paid more than 3 billion dollars in settlements and fees to the victims (Bishopaccountability.org, 2012). Five dioceses received bankruptcy protection and eight have declared bankruptcy in response to the ever increasing claims of abuse. Many of the charges have been brought against the Church several decades after the actual abuse occurred. The scandal led to the loss of two million memberships in the Catholic Church, or 3 percent of all Catholics (Hungerman, 2013). There are also cases against Catholic hierarchy who did not report sex abuse allegations to the legal authorities. It is known that many abusive priests were moved to other parishes in order to be protected against law where abuse sometimes continued (Boston.com, 2004). In March 2010, Pope Benedict apologized for the abuse of children, saying he was “truly sorry” for their decades of suffering (Dailymail.co.uk, 2010). Pope Francis also asked the Catholic Church to “act decisively” to eradicate the sexual abuse of children (Huffingtonpost.com, 2013).

Media coverage of the scandal in the United State is mostly concentrated after 2002. In that year, the Boston Globe began publishing their Pulitzer-prize winning critical investigation. Despite the existence of previous reports on the issue, the Boston Globe's coverage resonated strongly around the country and the world. There are potentially two main reasons for the widespread dissemination. First, the Boston Globe decided to contest the confidentiality order imposed by a superior court to protect Church documents concerning one priest. In November 2001, a judge ruled that the confidentiality order in this case should be lifted, and the documents became available in January 2002 (Boston.com, 2002). This led to an even bigger release of documented information on the issue when a judge ordered the archdiocese of Boston to release all the private files on every Boston priest accused of sexual abuse. These new files provided details on the transgressions of more than 100 priests (Foxnews.com, 2003). The availability of these documents to the public shed light to the scope of the problem and raised many questions about the reliability of the institution. Additionally, internet access enabled many people all across the country to read the reports.

In order to proceed with the analysis of the effect of this negative publicity on enrollment decisions, one needs to identify the channels by which the causal relationship between public awareness through publicity of child abuse and the demand for Catholic schooling can be explained. First, public concern about the Church's ability to protect children may affect the general perception of the institution. Second, the financial burden from both lawsuits and decreased donations may prevent the Church from reinvestment in educational activities (Bottan & Perez-Truglia, 2013; Hungerman, 2013). Some of

these factors might not have an immediate impact, but play a more significant role as time passes.

Dills and Hernandez-Julian (2012) provided the first published study that specifically tests the effect of negative publicity from the abuse scandal on Catholic schools. They found that the public negativity derived from media coverage of child abuse had a very small effect on the total number of Catholic schools and almost no effect on enrollment share throughout the country. In their paper, negative publicity resulting from media coverage of the scandal can approximately explain 5 percent of the decline in the availability of Catholic schools. They suggest that changes in demographics, particularly an increasing Hispanic population, can explain a larger proportion of the decline in Catholic schooling. However, their analysis is limited to the contemporaneous effect of negative publicity on Catholic schools. Bottan and Perez-Truglia (2013) studied the effect of negative publicity on Catholic schools along with other social services provided by the Catholic Church over time. Unlike Dills and Hernandez-Julian, they provided evidence that the Catholic sex abuse scandal played an important role in the decline of the U.S. Catholic school system. They found that the scandal had a long-run effect of the number of Catholic schools. They suggest that the scandal accounts for 23% of the decline in the number of Catholic schools.

In the current study, I contend that there is a fundamental difference in media coverage of the scandal prior to 2002 and afterwards. The priorities of the mass media have a significant impact in shaping the public's priorities. There is well-established evidence that the news media has the power to set a nation's agenda by focusing on a few key public issues. People not only acquire information about public affairs but also

evaluate its relative importance based on the emphasis placed by the news media. In other words, “the news media can set the agenda for the public’s attention to that small group of issues around which public opinion forms” (Merritt & McCombs, 2004).

The agenda of a news organization can be observed in its coverage pattern of public issues in a given period of time. Over this period, a few topics receive emphatic coverage, some are covered lightly, and others are rarely mentioned. Newspapers send signals to their audiences about the salience of the topics in daily news by publishing the lead story on the front page, other front page display, and large headlines. A televised newscast’s opening story and the length of time devoted to the story has the same function for television (Merritt & McCombs, 2004). Agenda setting theory describes “the ability to influence the salience of topics on the public agenda” (Iyengar & Kinder, 1987). That is, if the news item coverage is frequent and distinguished, the audience will regard the issue as more important. Public opinion polls usually assess the variation of public agenda. This theory is concisely explained by Cohen, who noted that the press “may not be successful much of the time in telling people what to think, but it is stunningly successful in telling its readers what to think about” (Cohen, 1963).

I argue that the extent of public notices issued following abuse revelations, as measured by previous research, might not identify the potential impact of the abuse scandal if many of these notices were not sufficiently recognized. The level of importance the mass media assigned to child abuse in the Catholic Church after 2002 deeply changed the public’s attitude toward this issue and the attention to which it was paid. I view this event as providing a critical timing dimension that allows typical statistical evaluation tools to be employed. It is this approach that differs from the two

preceding studies. In order to provide qualitative evidence in support of this claim, it can be noted that even though actual abuse peaked in the 1970's, the majority of victims did not register complaints until after 2002. Moreover, for the first time, issues concerning child abuse appeared in the Gallup Public Opinion Poll as one of the most important issues in 2002 and 2003. In 2002, people responded that child abuse would be one of the most important issues facing the nation 25 years from now (The Gallup Public Opinion Poll, 2002, and 2003). As it was previously mentioned, public opinion polls usually evaluate the public agenda at any given time. It is additional evidence that despite the existence of previous reports concerning this issue, public's attention has been drawn to the child abuse notices only after 2002. In the present study, I model the differing effects of abuse notices before and after 2002.

I also explore the effect of negative publicity on Catholic school enrollment over time. It is important to know whether parental response to public notices of child abuse in the Catholic Church is immediate and whether the impact of these notices remain in effect for a long span of time. From a practical perspective, awareness and reaction of a scandal in a given year may hit after that year's enrollment decision is made. Moreover, it is likely that the public's perception of the Catholic Church depends on a lengthy history of public notices, rather than just the most recent period. Additionally, some factors such as financial burden can affect the demand for Catholic schools indirectly occur with a delay. I take seriously the pattern of reaction to abuse claims over time, both before and after the heightened media attention that began in 2002.

1.3. Construction of Panel Data on the Abuse Scandal

In order to examine the relationship between the publicity of the child abuse scandal and Catholic school enrollment, one needs to define publicity over time and space. I use the number of Church employees per 100,000 people in a population who were publicly accused for the first time of abuse in a particular diocese and period. Again, I anticipate that this measure will be more pronounced after 2002.

I use the website bishopaccounatability.org for data on abuse accusations. The website compiles information on more than 3,500 Catholic Church employees who have been involved in child abuse cases. This website is run by a non-profit organization based in Massachusetts with the goal of providing a comprehensive archive of every publicly available document and report on the crisis. As a result, the standard of inclusion of any document is broad. These documents provide data on where and when the accused served, as well as the dates that the Church and the public were informed. It also includes information about the cases that led to arrests, indictments, convictions, confessions, settlements, and lawsuits. Although the content of these documents is not verified, each reported allegation has been double-checked with a cited source document and contains citations. Wherever possible, there is a link to the main source. It should be noted that I am interested in creating a variable to measure publicity, which is not necessarily dependent on 100% accuracy of the content of the documents. An unproven allegation in the press could be detrimental to the reputation of an institution or an individual.

Figure 1.1 illustrates the total number of accusations, as well as the total number of convictions, arrests, lawsuits, settlements, or confessions that I label as a significant accusation for every two year span. The observed trend is reasonably consistent with the trend of credible allegations reported by dioceses outlined in annual reports of United

States Conference of Catholic Bishops (The Nature and Scope of Sexual Abuse of Minors by Catholic Priests and Deacons in the United States 1950-2002, Figure 5.2.1, 2004; Report on the Implementation of the Charter for the Protection of Children and Young People, Table 1, 2012) as well as Dills and Hernandez-Julian's trend (Figure 2, page 146). Public notices are not uniformly distributed among dioceses. While a few diocese, such as, Los Angeles, Boston, Chicago, and New York, have recognized many, the rest have received significantly fewer notices.

I aggregate Catholic school enrollment data to the diocese level using the Private School Universe Survey. This survey is conducted by the National Center for Education Statistics and provides biennial data on different types of private schools, including Catholic, other religions, and nondenominational institutions. Beginning in 1995, the definition of private school in this survey was expanded to include schools for which kindergarten is the highest grade. To avoid any inconsistency, enrollment data for kindergarten and pre-kindergarten were removed from the database. Figure 1.2, and figure 1.3 show the fraction of the school age population who are enrolled in Catholic schools and the number of Catholic schools in thousands at the same period of the time.

In different specifications, I also include a vector of time-diocese-variant variables that control for socioeconomic and demographic changes. It includes data on the unemployment rate, real per capita income, the percentage of the population above age 25 with a bachelor's degree or higher, the relative percentage of the Hispanic population, population density in terms of the population in thousands per square mile, and the percentage of Catholic population. I use multiple county level databases to build the control variables. I geographically match each county with its associated diocese and

aggregate the data. Annual unemployment rates are obtained from the Bureau of Labor Statistics. Educational attainment data is collected from the Census Bureau aligned with educational attainment estimates by Bode (2010). I use the decennial survey of Churches and Church Membership in the United States in 1990, 2000, and 2010 coupled with information on the website catholic-hierarchy.org to obtain the percentage of Catholic population in each diocese.[1] I use these data points to interpolate values for the intervening years. Finally, I used data from the Bureau of Economic Analysis for obtaining per capita income data.[2] Dioceses from Alaska and Hawaii are excluded from this study as a large portion of their socioeconomics data points for the time period before 2002 are missing.[3]

After combining these data, I am left with biennial panel data from 1991-2009. Table 1.1 presents the summary statistics for the variables in the analysis for dioceses that have received the greatest amount of public notices and other diocese separately.[4] It is clear that the number of public notices increased dramatically after 2002 for both groups. However, the most affected dioceses have notably larger increase. They also have higher enrollment share and number of Catholic schools than the rest of the sample, as well as a larger Catholic population. There is nothing notably different about unemployment rates between the two groups. The percentage of the population with a bachelor degree and

[1] This data are collected by the Association of American Religious Bodies (ASARB) and distributed by the Association of Religious Data Archives.
[2] Data on populations and demographics are gathered from the National Cancer Institute. This center utilizes the decennial Census to estimate annual county level population and demographic changes.
[3] The results of those including dioceses from Alaska and Hawaii are qualitatively the same and they will be available upon request.
[4] I consider any diocese that has recognized more than 50 public notices over the sample period as a heavily affected diocese. These include Los Angeles, Chicago, Louisville, Boston, Manchester, Rockville Center, Portland, Philadelphia, Baltimore, and New York

higher, real per capita income, and the Hispanic population share are higher for the more affected group, however, they have increased for both groups over time.

1.4. Methodology

1.4.1. Basic Empirical Model

I begin by using a weighted least square framework to estimate the effect of publicity of the child abuse scandal on Catholic school enrollment. The variable pu represents publicity, which is measured as the number of Catholic Church employees per 100,000 people in population who were publicly accused for the first time in each period. The basic regression is summarized by:

$$\log(y_{it}) = \alpha_i + \gamma_t + \beta_1 \log(\text{pu}_{it}) + \delta(X_{it}') + \varepsilon_{it} \qquad (1)$$

This regression is weighted by the population of school age children in each diocese to assign lower weight to smaller dioceses in which enrollment is more volatile (following a procedure by Abouk and Adams, 2013). All the variables are log transformed; therefore, the coefficients can be interpreted as elacticities in this framework. The dependent variable is either the log number enrolled in Catholic schools per 100,000 school-aged children or the log number of Catholic schools per every thousand school age child in year t and diocese i at the beginning of the school year. I include diocese fixed effects that account for time invariant characteristics of the diocese (α) and year fixed effects (γ). X_{it} is a vector of time and diocese variant control variables. Standard errors are clustered

by diocese. In some specifications I include the interaction of diocese dummies with time to control for diocese-specific time trends.

I suspect that regression equation 1 might mask the lagged effects of publicity on Catholic school enrollment. To address this possibility, I include lags of pu_{it} in different specifications. As a result, I can study whether the effect of publicity will grow or fade when time passes. I suspect the stronger effect to occur with at least a one-period lag as parents are able to react to news and change enrollment decisions. This new regression can be summarized by:

$$\log(y_{it}) = \alpha_i + \gamma_t + \sum_{\tau=0}^{3} \beta_\tau \log(\mathrm{pu}_{it-\tau}) + \delta(X_{it}') + \varepsilon_{it} \quad (2)$$

To initiate my investigation of whether there is a systematic difference in the effect of notices between before and after 2002, I run the same regression for each of these subsamples separately.

1.4.2. Difference-in-Difference Framework

Following a procedure by Card (1992), the empirical strategy to identify more formally the unique influence of the media coverage after 2002 is summarized by:

$$\log(y_{it}) = \alpha_i + \gamma_t + \beta_1(\log(\mathrm{pu}_{it}) * \mathrm{d}_t) + \delta(X_{it}') + \varepsilon_{it} \quad (3)$$

Variable d_t is a dummy variable which is equal to 1 if accusation was made after 2002 and 0 otherwise. This variable represents the time that the child abuse scandal became a widespread concern as a result of massive coverage of the issue. I argue that the timing of the peak in media coverage (d_t) is exogenous. The primary reason for the intensified coverage of the child abuse scandal in 2002 is that confidential Church documents on the issue became available to the public as a result of contest by the Boston Globe to the imposed confidentiality order. This coupled with the increasing penetration of internet access made it easier for audiences all over the nation to have access to and read the reports. There is no reason to believe that the unsealing of documents was driven by other issues in the Catholic Church that might affect enrollment in Catholic schools.

Equation 3 contains the other control variables from earlier estimations. This regression is weighted by the population of school age children in each diocese as well. To investigate the possibility of lagged effects, I add a series of lag variables to equation 3 (equation 4). This regression is summarized by:

$$\log(y_{it}) = \alpha_i + \gamma_t + \sum_{\tau=0}^{3} \beta_\tau (\log(\mathrm{pu}_{it-\tau}) * \mathrm{d}_t) + \delta(X_{it}') + \varepsilon_{it} \quad (4)$$

Coefficients of β_1 through β_3 show the pattern of lagged effects of the publicity of child abuse on Catholic school enrollment and the number of Catholic schools.

1.5. Results

1.5.1. Basic Results

Table 1.2 presents the results of the basic weighted least squares regression (equation 1). The second column in each set of regressions includes the linear diocese-specific time trends, as well as the vector of control covariates. The effect of publicity on enrollment share of Catholic schools is small and statistically insignificant in both specifications. These results are consistent with Dills and Hernandez-Julian (2012).

It is noteworthy that I aggregate the number of public notices over a two year period to construct the variable of negative publicity. One should take into account that the reaction to the revelation of new information about child abuse can happen after the year's enrollment decision is made. As a result, the coefficient on the negative publicity variable might be more likely to be negative in my study compared with Dills and Hernandez-Julian's paper due to the fact that the coefficient captures this delayed effect of negative publicity at any given time, in addition to the contemporaneous effect.

Table 1.3 presents the results of the lagged effects of publicity. This again allows for the possibility that families may take some time in making their enrollment decisions. One can conclude from these estimates that the publicity of child abuse has a negative and significant effect on Catholic school enrollment share, and that this effect indeed grows over time. The estimates are qualitatively the same after inclusion of the diocese-specific time trends. These results are consistent with Bottan and Perez-Truglia (2013).

In Table 1.3, I also divide the sample between periods before and after 2002. The variable publicity has no power of prediction when I restrict the sample only to the period before 2002. The estimates are positive, small, and insignificant. Contrary to these

findings, the estimates are negative and significant for the subsample after 2002. The null hypothesis that the two subsamples have equal coefficients for the variable publicity and its lags can be rejected by conducting the Chow test (Chow, 1960).

The coefficients of interest when the number of Catholic schools is the dependent variable are also negative, but they are not significant in the overall specification. When I separate the sample before and after 2002, the same pattern of more substantial effects after 2002 holds.[5]

These results indicate that there is a meaningful difference between pre- and post-2002 in terms of the effects of accusations on school enrollment and number of schools. There was modest media coverage prior to 2002, but people did not respond to it. Unlike the current study and Bottan and Perez-Truglia's study, Dills and Hernandez-Julian did not find any lagged effects of negative publicity. It is worthwhile to point out a distinct difference between the databases used in these different studies. Dills and Hernandez-Julian used an annual panel data from 1990-2007 while Bottan and Perez-Truglia made use of a biennial data and I am using the same database. As a result, the number of panel data waves before 2002 in Dills and Hernandez-Julian's study is noticeably larger than mine. As it can be inferred from the results in Table 1.3, the non-negligible negative effect of negative publicity of child abuse scandal on Catholic school enrollment is derived from public notices that realized after 2002. Inclusion of public notices from the period before 2002 biases the coefficient toward zero, however, the negative effect may be still observed when the panel data waves are distributed evenly before and after 2002. When the number of panel data waves prior to 2002 is much greater than after 2002, the

[5] The results of those included the diocese-specific time trends are not included in Table 1.3 and are available upon request.

coefficients are likely to approach zero. Ignoring this important property of the news coverage of the scandal and utilizing a larger panel data waves before 2002 likely explains the small and insignificant findings in Dills and Hernandez-Julian (2012) compared with the substantial findings from my study and Bottan and Perez-Truglia's study. These findings motivate the application of the proposed difference-in-difference framework, in which this fundamental difference in media coverage before and after 2002 is taken into consideration more directly.

1.5.2. Difference-in-Difference Results Accounting for Change in 2002

Table 1.4 presents the results of the difference-in-difference regression that explicitly measures the effect of reported abuse cases post 2002 (equation 3). The dependent variable is either the log of Catholic school enrollment or the log number of Catholic schools per thousand of school-age children in dioceses. In each set of regressions, I include an increasingly richer set of control variables moving from the first column to the third. The first column only contains diocese and year fixed effects. The second column adds the vector X_{it} to the regression analysis, and the third column adds diocese-specific time trends.

The coefficients of interest are negative and significant when I only incorporate diocese and year fixed effects in the regressions. Inclusion of the vector X_{it} results in smaller and insignificant coefficients. The results are not robust to inclusion of diocese-specific time trends either. The outcomes of the basic difference-in-difference analysis in Table 1.4 therefore do not offer robust evidence of a contemporaneous causal link between publicity and the share and number of Catholic schools.

The lack of strong effects in Table 1.4 may stem from the fact that the impact of reports takes at least a school year to take hold, as families are unable to immediately remove their children from Catholic schools. Table 1.5 presents the results from the regression equation 4, which allows for lags. In all the specifications, I control for diocese and year fixed effects as well as the vector X_{it}. Diocese-specific time trends are also included in the second column of each set of regressions. One can conclude from the results in Table 1.5 that the publicity of the scandal is negatively associated with both the enrollment share and number of Catholic schools. The negative effects take a period to take hold, but are also sustained over time. The lagged coefficients are jointly significant at the 5% significance level for enrollment share of Catholic schools and only marginally significant for number of Catholic schools (p-value=0.11). The estimates imply that a one percent increase in publicity of the abuse after 2002 is associated with a roughly 0.25 percent decrease in overall enrollment shares of Catholic schools. Although this estimate seems to be quite small, it is worth mentioning that dioceses in the sample experienced an enormous increase in public notices. Back-of-the-envelope calculations suggest that the negative publicity derived from the increase in public notices of child abuse can approximately explain about two-thirds of the decline in Catholic school enrollment share and the number of Catholic schools.

After inclusion of the diocese-specific time trends, the coefficients become smaller and less precise. It is noteworthy that the unemployment rate and the percentage of population above age 25 with a bachelor degree or higher cannot explain Catholic school enrollment. This could be due to the considerable amount of subsidies provided by the Catholic Church to attend Catholic schools. These subsidies usually target those who

cannot otherwise afford Catholic schools. The same pattern is also observable for the number of Catholic schools. The coefficients weaken after the inclusion of diocese-specific time trends, but the point estimates remain large and fall just short of significance at conventional levels.[6] Percentage of Hispanic population and real per capita income, however, are now significant predictors of enrollment share and number of Catholic schools. The Hispanic population has grown substantially in the past decades, and the majority of Hispanics are Catholic.

The results of this section reinforce the relative importance that the notices of abuse that were made after 2002 had on enrollment. These effects still took a period to be observed in the data, likely because it took time for parents to react. Those effects still continued to grow over time. There are several additional explanations for such a pattern. In modern America, scandals have a long term impact on the shaping of public's opinion. They evolve a life and momentum of their own, which are hard to ignore (Williams, 1998). The continuous trend of reported allegations of sexual misconduct in recent years has helped create and sustain a deep level of public distrust toward the Catholic Church. The more reporting there is, the more likely it is that public distrust grows. Although the number of allegations dropped after 2005, the newest allegations will emphasize the effect of past allegations by communicating the message to the public that the problem still exists. This reinforces the public's distrust of the Catholic Church. Financial burden derived from negative publicity of the child abuse scandal is another factor that affects Catholic schooling. Catholic schools rely heavily on the financial support provided by

[6] Inclusion of linear time trends most of the time leads to smaller and less precise estimates because it captures most of the variation in data. Due to this reason and also the sample size, this change was expected.

dioceses. Financial constraints stemming from the scandal are likely to affect Catholic schools and these constraints happen with a delay and grow over time. Although I cannot distinguish between these explanations for the decline in enrollments, there does appear to be room for both possibilities since the number of schools, in addition to enrollment, also seems to decline with a noticeable lag.

1.5.3. Additional Estimates

Throughout this paper, I have argued that the decline in share of Catholic schools is exclusively derived from accusations that were realized after 2002. This is the main motivation for using the difference-in-difference estimation (equation 4). In order to provide more evidence for this argument, I estimate:

$$\log(y_{it}) = \alpha_i + \gamma_t + \sum_{\tau=0}^{3} \partial_\tau (\log(\mathrm{pu}_{it-\tau})) + \sum_{\tau=0}^{3} \beta_\tau (\log(\mathrm{pu}_{it-\tau}) * \mathrm{d}_t) + \delta(X_{it}') + \varepsilon_{it} \quad (5)$$

Both models in equations 2 and 4 are nested in this model. If ∂ s in this specification are not significantly different from zero, the model will reduce to the proposed difference-in-difference model. On the other hand, if βs are not significantly different from zero, including the accusation from the whole sample is preferred. Table 1.6 presents the results of this new regression. The results in Table 1.6 emphasize again the relative importance of accusations happened after 2002. δs are not individually and jointly significantly different from zero, while the coefficients of accusations after 2002 are jointly significant. This provides additional evidence for the difference-in-difference framework.

I use the model summarized by equation 4 for additional estimates and robustness checks. First, one might suspect that the estimates presented thus far might reflect some pre-existing trends in the data. For example, it is possible that certain diocese were experiencing a reduction in support for Catholicism before the outbreak of the scandal and these are the dioceses where people were more likely to make allegations of abuse, and this may lead to spurious findings. In order to address this concern, I control for the future accusations in the regression. If the results are driven by a pre-existing trend, the inclusion of future accusations should attenuate the coefficients of negative publicity and its lags. It is worth mentioning that due to the low number of panel data waves, specifically after treatment ones, inclusion of all the contemporaneous effect and lagged effects in the equation 4 will raise a problem of colinearity. I present two different combinations of leads and lags. I omit some lagged effects in each of these combinations based on the number of lead effects included in the regression. The results of these specifications are reported in Table 1.7. The coefficients of contemporaneous and lagged effects of abuse accusations remain the same. However, lead accusations coefficients are much smaller compared with lagged effects and they are all statistically insignificant. It indicates that the results are not driven by pre-existing trends.

I utilize a second definition of publicity, which eliminates the cases that did not end with conviction, arrest, lawsuit, settlement, or confession. Panel A in Table 1.8 presents the results of the effect of significant accusations on enrollment share and number of Catholic schools. The second column in each set of regressions includes the diocese-specific time trends. The effects of this new measure of publicity on enrollment share and number of Catholic schools follow the same patterns as before.

Unweighted least squares is an obvious alternative to the main specification, which weights estimations based on a diocese's population. Panel B in Table 1.8 presents the results of unweighted least squares and indicate that the weighted least squares results presented earlier in the paper provide more conservative estimates.

Table 1.9 presents the results of the regression analysis of equation 4 for male and female students separately. The findings do not imply that the estimates are always different for male students than female students in a uniform way. This is despite males being the victims in the vast majority of abuse cases, however, the decline in the enrollment share of female students happens with a lag relative to the enrollment share for male students. The immediate response for male students is negative and significant, which is in line with the more imminent threat perceived by the abuse scandal, and it is robust to inclusion of linear time trends. It suggests that risk realization happens for male students sooner. This difference in dynamics of response between male and female students might be due to the fact that the enrollment decision for female students is systematically affected by the comparable decision for their older brothers (Butcher and Case, 1994).[7] This difference in response dynamics between male and female students may also be responsible for insignificant coefficients presented in Table 1.5.

1.6. Conclusion

In this study, I address the role of mass media in public agenda setting. I argue that the child abuse scandal in the Catholic Church, which was widely emphasized by the majority of news media beginning in 2002, became more prominent in the public's mind

[7] The same separation between male and female students was performed on the whole sample, and the results are not conclusive.

at this time. I used the cumulative number of church employees who have been publicly accused at any period as a proxy for negative publicity. These seem to take on heightened importance among parents as measured by Catholic school enrollment after 2002. The results suggest that there is a negative relationship between publicity of the scandal and the demand and availability of Catholic schools. The effect is also sustained over time. This is likely derived from both the growing public distrust and financial burden caused by the massive media coverage of the issue.

Decline in the portion of students who enrolled in Catholic schools happens in a different pattern for male and female students. Enrollment share for male students decreases immediately in response to the public notices of child abuse and sustains over time. This decline for enrollment share of female students occurs with a delay compared with men. This implies that risk for male students is considered to be more serious.

Catholic school attendance is believed to be correlated with better academic and labor market outcomes. Catholic schools also provide competition to public schools and thereby benefit students in public schools indirectly. The decline in Catholic school enrollment could have significant welfare implications if schooling alternatives be limited to lower quality institutions. Therefore, it is critical to investigate the most common alternative of Catholic schools for parents who have decided to switch to other types of schools. This can be the subject of future research.

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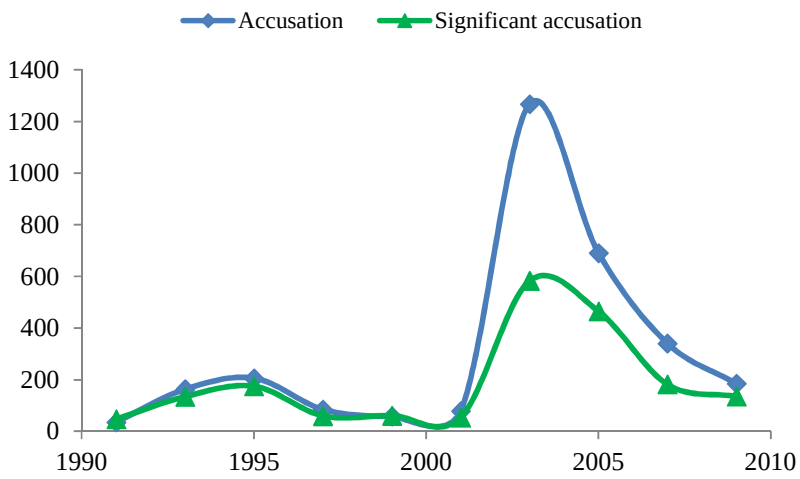

Figure 1.1. Total number of child abuse accusations and significant accusations

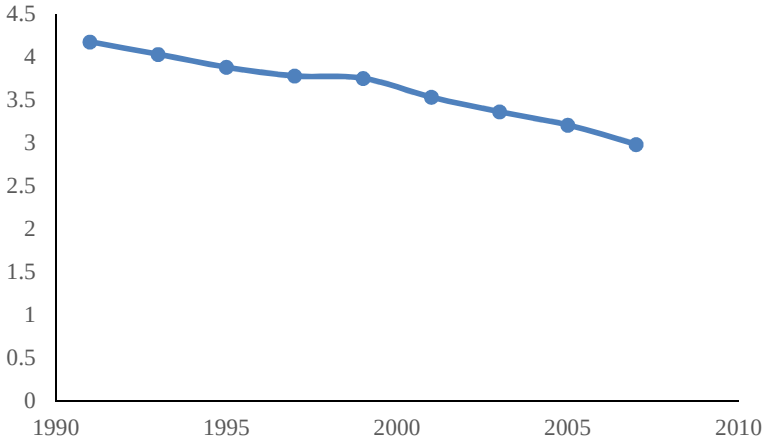

Figure 1.2. Enrollment share of Catholic schools.

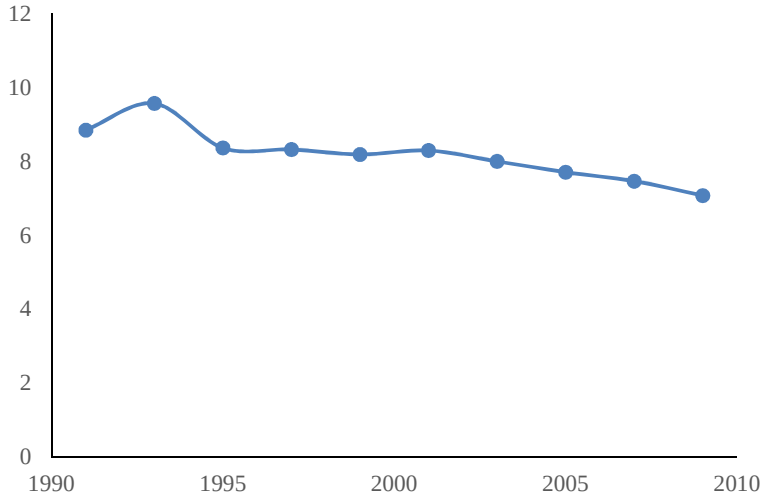

Figure 1.3. Number of Catholic schools in thousands

Table 1.1. Summary Statistics

Variables	Most affected dioceses			Other dioceses		
	Whole sample	Before 2002	After 2002	Whole sample	Before 2002	After 2002
Enrollment share of Catholic schools	6.891	7.506	5.969	3.757	3.982	3.420
Number of Catholic schools	176.714	186.622	161.853	39.611	41.558	36.690
cumulative accusations	9.58	1.933	21.05	1.269	0.571	2.316
Cumulative significant accusations	5.62	1.416	11.925	0.805	0.458	1.326
% population with a bachelor's degree and higher	29.845	27.927	32.723	22.748	21.267	24.971
unemployment rate	5.840	5.654	6.121	5.904	5.711	6.194
real per capita income in 000's	19.912	18.651	21.803	15.541	14.595	16.960
% Hispanic	12.415	11.232	14.190	10.730	9.598	12.428
population density	1.335	1.311	1.371	0.484	0.472	0.503
% catholic population	33.484	33.680	33.190	21.266	21.445	20.999

Any diocese that has recognized more than 50 public notices over the sample period is considered as a heavily affected diocese.

Table 1.2. Basic Weighted Least Square Regression

	Log (Catholic school enrollment share)		Log (Number of catholic schools per 000's students)	
VARIABLES	(1)	(2)	(3)	(4)
Log (total accusations)	-0.00281	0.0169	0.00297	0.00862
	(0.0229)	(0.0168)	(0.0249)	(0.0187)
Log (% population with a bachelor's degree and higher)	-0.00436	-0.0348	0.00474	-0.0647
	(0.0926)	(0.0556)	(0.100)	(0.0879)
Log (unemployment rate)	-0.000511	-0.0672**	0.0367	0.000429
	(0.0407)	(0.0288)	(0.0408)	(0.0350)
Log (real per capita income)	0.550***	0.0488	0.468**	0.177
	(0.161)	(0.143)	(0.180)	(0.189)
Log (% Hispanic)	0.232***	-0.0126	0.166***	-0.0799
	(0.0410)	(0.114)	(0.0390)	(0.123)
Log (population density)	0.0373	-1.061***	-0.206	-1.009***
	(0.294)	(0.249)	(0.306)	(0.326)
Log (% catholic population)	0.174**	-0.0795	0.131*	-0.0930
	(0.0748)	(0.0625)	(0.0784)	(0.0567)
Constant	-9.476***	-11.96***	-7.634***	-10.22***
	(2.235)	(1.494)	(2.437)	(2.208)
Linear diocese-specific time trend	No	Yes	No	Yes
Observations	1,718	1,718	1,718	1,718
R-squared	0.984	0.992	0.976	0.985

Note: There are 172 dioceses, regressions include year and diocese fixed effects. The numbers in parenthesis are clustered standard errors at the diocese level. *** p<0.01, ** p<0.05, * p<0.1

Table 1.3. Lagged Effects of Basic Weighted Least Square

	Log (Catholic school enrollment share)			Log (Number of catholic schools per 000's students)		
	Full sample	Before 2002	After 2002	Full sample	Before 2002	After 2002
VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)
Log (total accusations)	-0.0223	0.106	-0.0328	-0.0230	0.206	0.0170
	(0.0250)	(0.0944)	(0.0345)	(0.0255)	(0.188)	(0.0337)
1st lag of Log (total accusations)	-0.0434*	0.0173	-0.0562*	-0.0394	0.0481	-0.0197
	(0.0243)	(0.0729)	(0.0326)	(0.0328)	(0.0934)	(0.0408)
2nd lag of Log (total accusations)	-0.0576**	0.0633	-0.0831**	-0.0600	0.0148	-0.0401
	(0.0284)	(0.0505)	(0.0383)	(0.0445)	(0.0628)	(0.0491)
3d lag of Log (total accusations)	-0.0541*	0.0616	-0.0831**	-0.0648*	-0.0407	-0.0225
	(0.0298)	(0.0686)	(0.0328)	(0.0353)	(0.0905)	(0.0486)
Log (% population with a bachelor's degree and higher)	0.0278	-0.134	-0.177	0.0299	0.0235	-0.336
	(0.106)	(0.0826)	(0.135)	(0.142)	(0.101)	(0.297)
Log (unemployment rate)	0.00566	0.0125	-0.0483	0.0194	0.0859*	-0.0114
	(0.0388)	(0.0423)	(0.0486)	(0.0405)	(0.0471)	(0.0585)
Log (real per capita income)	0.474***	0.291	0.186	0.452**	0.264	0.0694
	(0.168)	(0.225)	(0.184)	(0.193)	(0.316)	(0.207)
Log (% Hispanic)	0.174***	0.196***	0.0863	0.163**	0.0916	0.0427
	(0.0579)	(0.0554)	(0.137)	(0.0673)	(0.0666)	(0.161)
Log (population density)	0.0463	0.111	0.0986	-0.146	-0.161	0.0451
	(0.396)	(0.374)	(0.271)	(0.382)	(0.478)	(0.512)
Log (% catholic population)	0.0906	-0.0106	-0.0636	0.0756	-0.0327	-0.0347
	(0.0661)	(0.121)	(0.116)	(0.0754)	(0.0772)	(0.131)
Constant	-8.884***	-6.768**	-7.063***	-7.275**	-5.316	-3.772
	(2.705)	(3.035)	(2.478)	(2.820)	(4.340)	(3.300)
Observations	1,204	516	688	1,204	516	688
R-squared	0.987	0.996	0.991	0.983	0.993	0.986

Note: There are 172 dioceses, regressions include year and diocese fixed effects. The numbers in parenthesis are clustered standard errors at the diocese level. *** p<0.01, ** p<0.05, * p<0.1

Table 1.4. Basic Difference-in-Difference

	Log (Catholic school enrollment share)			Log (Number of catholic schools per 000's students)		
VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)
Log (total accusations)	-0.0808**	-0.0283	0.00767	-0.0510*	-0.0272	-0.00960
	(0.0363)	(0.0267)	(0.0193)	(0.0295)	(0.0277)	(0.0200)
Log (% population with a bachelor's degree and higher)		-0.00349	-0.0346		0.00559	-0.0634
		(0.0925)	(0.0556)		(0.0999)	(0.0876)
Log (unemployment rate)		-0.000485	-0.0674**		0.0366	0.000118
		(0.0407)	(0.0288)		(0.0408)	(0.0349)
Log (real per capita income)		0.554***	0.0491		0.472***	0.179
		(0.161)	(0.143)		(0.180)	(0.189)
Log (% Hispanic)		0.231***	-0.0132		0.164***	-0.0811
		(0.0408)	(0.114)		(0.0389)	(0.122)
Log (population density)		0.0309	-1.063***		-0.213	-1.013***
		(0.294)	(0.251)		(0.307)	(0.329)
Log (% catholic population)		0.173**	-0.0800		0.129	-0.0941*
		(0.0747)	(0.0626)		(0.0786)	(0.0568)
Constant	-5.194***	-9.551***	-11.98***	-2.925***	-7.716***	-10.26***
	(0.0149)	(2.233)	(1.492)	(0.0141)	(2.442)	(2.206)
Linear diocese-specific time trend	No	No	Yes	No	No	Yes
Observations	1,718	1,718	1,718	1,718	1,718	1,718
R-squared	0.981	0.984	0.992	0.974	0.976	0.985

Note: There are 172 dioceses, regressions include year and diocese fixed effects. The numbers in parenthesis are clustered standard errors at the diocese level. *** p<0.01, ** p<0.05, * p<0.1

Table 1.5. Difference-in-Difference with Lagged Effects

	Log (Catholic school enrollment share)		Log (Number of catholic schools per 000's students)	
VARIABLES	(1)	(2)	(3)	(4)
Log (total accusations)	-0.0353	-0.0334	-0.0428*	-0.0475
	(0.0254)	(0.0259)	(0.0253)	(0.0318)
1st lag of Log (total accusations)	-0.0513*	-0.0495	-0.0579	-0.0603
	(0.0261)	(0.0335)	(0.0353)	(0.0474)
2nd lag of Log (total accusations)	-0.0821**	-0.0710*	-0.0860*	-0.0762
	(0.0326)	(0.0428)	(0.0515)	(0.0664)
3d lag of Log (total accusations)	-0.0866**	-0.0508	-0.0839**	-0.0219
	(0.0334)	(0.0524)	(0.0410)	(0.0686)
Log (% population with a bachelor's degree and higher)	0.0254	-0.0603	0.0271	-0.0879
	(0.105)	(0.0877)	(0.141)	(0.162)
Log (unemployment rate)	0.0130	-0.0497	0.0273	-0.0150
	(0.0383)	(0.0375)	(0.0398)	(0.0419)
Log (real per capita income)	0.506***	0.102	0.485**	0.0663
	(0.168)	(0.158)	(0.189)	(0.168)
Log (% Hispanic)	0.160***	0.0324	0.148**	-0.0175
	(0.0576)	(0.127)	(0.0660)	(0.154)
Log (population density)	-0.00447	-1.269***	-0.199	-1.323***
	(0.395)	(0.399)	(0.380)	(0.502)
Log (% catholic population)	0.0795	-0.0754	0.0637	-0.0691
	(0.0645)	(0.0697)	(0.0748)	(0.0727)
Constant	-9.455***	-13.35***	-7.872***	-10.66***
	(2.712)	(2.325)	(2.775)	(2.965)
Linear diocese-specific time trend	No	Yes	No	Yes
Observations	1,204	1,204	1,204	1,204
R-squared	0.987	0.994	0.983	0.990

Note: There are 172 dioceses, regressions include year and diocese fixed effects. The numbers in parenthesis are clustered standard errors at the diocese level. *** p<0.01, ** p<0.05, * p<0.1

Table 1.6. Nested Model

VARIABLES	Log (Catholic school enrollment share) (1)	Log (Number of Catholic schools per 000's students) (2)
∂_0	0.0622 (0.104)	0.212 (0.135)
∂_1	-0.0223 (0.0641)	0.0596 (0.0594)
∂_2	-0.0186 (0.0563)	-0.0175 (0.0621)
∂_3	0.00561 (0.0413)	-0.0371 (0.0425)
β_0	-0.106 (0.111)	-0.262* (0.142)
β_1	-0.0454 (0.0773)	-0.125 (0.0762)
β_2	-0.0985 (0.0692)	-0.0869 (0.0772)
β_3	-0.127** (0.0495)	-0.0682 (0.0574)
Constant	-11.65*** (2.349)	-9.277*** (2.248)
Observations	1,204	1,204
R-squared	0.976	0.972
Panel B- Hypothesis Test		
$\partial_1 = \partial_2 = \partial_3 = \partial_4 = 0$	0.28	0.91
P-Value	0.8898	0.4573
$\partial_1 + \partial_2 + \partial_3 + \partial_4 = 0$	0.02	1.33
P-Value	0.8850	0.2507
$\beta_1 = \beta_2 = \beta_3 = \beta_4 = 0$	2.24	1.30
P-Value	0.0663	0.2736
$\beta_1 + \beta_2 + \beta_3 + \beta_4 = 0$	2.83	5.02
P-Value	0.0944	0.0263

Note: There are 172 dioceses, regressions include year and diocese fixed effects. The numbers in parenthesis are clustered standard errors at the diocese level. *** p<0.01, ** p<0.05, * p<0.1

Table 1.7. Difference-in-Difference with Lead and Lagged Effects

	Log (Catholic school enrollment share)		Log (Number of catholic schools per 000's students)	
VARIABLES	(1)	(2)	(3)	(4)
Log (total accusations)	-0.0306	-0.0299	-0.0428	-0.0398
	(0.0288)	(0.0300)	(0.0286)	(0.0376)
1st lag of Log (total accusations)	-0.0540*	-0.0654**	-0.0662	-0.107**
	(0.0302)	(0.0315)	(0.0436)	(0.0420)
2nd lag of Log (total accusations)	-0.100***		-0.135**	
	(0.0350)		(0.0526)	
1st lead of Log (total accusations)	-0.00867	-0.0157	-0.00433	-0.00747
	(0.0260)	(0.0276)	(0.0261)	(0.0279)
2nd lead of Log (total accusations)		-0.00542		0.0239
		(0.0218)		(0.0251)
Constant	-10.61***	-10.92***	-8.982**	-10.44***
	(2.966)	(2.913)	(3.560)	(3.022)
Observations	1,204	1,203	1,204	1,203
R-squared	0.987	0.988	0.983	0.979

Note: There are 172 dioceses, regressions include year and diocese fixed effects. The numbers in parenthesis are clustered standard errors at the diocese level. *** p<0.01, ** p<0.05, * p<0.1

Table 1.8. Robustness Checks

Panel A: The Effect of Significant Accusations

	Log (Catholic school enrollment share)		Log (Number of catholic schools per 000's students)	
VARIABLES	(1)	(2)	(3)	(4)
Log (total significant accusations)	-0.0389	-0.0253	-0.0870***	-0.0786*
	(0.0288)	(0.0296)	(0.0281)	(0.0406)
1st lag of Log (total significant accusations)	-0.0742**	-0.0685	-0.0997**	-0.0963
	(0.0361)	(0.0475)	(0.0414)	(0.0591)
2nd lag of Log (total significant accusations)	-0.0973***	-0.0638	-0.110**	-0.0881
	(0.0356)	(0.0549)	(0.0440)	(0.0711)
3d lag of Log (total significant accusations)	-0.0647	-0.0802	-0.0807	-0.0674
	(0.0395)	(0.0629)	(0.0519)	(0.0824)
Constant	-9.098***	-13.27***	-7.666***	-10.61***
	(2.716)	(2.342)	(2.792)	(2.958)
Linear diocese-specific time trend	No	Yes	No	Yes
Observations	1,204	1,204	1,204	1,204
R-squared	0.987	0.994	0.983	0.990

Panel B: Unweighted Least Square

	Log (Catholic school enrollment share)		Log (Number of catholic schools per 000's students)	
VARIABLES	(1)	(2)	(3)	(4)
Log (total accusations)	-0.0445	-0.0448	-0.0527*	-0.0592*
	(0.0290)	(0.0324)	(0.0300)	(0.0325)
1st lag of Log (total accusations)	-0.0676**	-0.0696	-0.0681**	-0.0683
	(0.0323)	(0.0503)	(0.0333)	(0.0524)
2nd lag of Log (total accusations)	-0.117***	-0.115*	-0.106***	-0.0943
	(0.0379)	(0.0595)	(0.0353)	(0.0623)
3d lag of Log (total accusations)	-0.121***	-0.115	-0.106**	-0.0968
	(0.0350)	(0.0738)	(0.0410)	(0.0799)
Constant	-12.00***	-12.08***	-9.090***	-10.56***
	(2.202)	(3.534)	(2.131)	(3.451)
Linear diocese-specific time trend	No	Yes	No	Yes
Observations	1,204	1,204	1,204	1,204
R-squared	0.976	0.986	0.972	0.985

Note: There are 172 dioceses, regressions include year and diocese fixed effects. The numbers in parenthesis are clustered standard errors at the diocese level. *** p<0.01, ** p<0.05, * p<0.1

Table 1.9. Effect of Publicity by Gender

	Log (Catholic school male students enrollment share)		Log (Catholic school female students enrollment share)	
VARIABLES	(1)	(2)	(3)	(4)
Log (total accusations)	-0.0460*	-0.0519**	-0.0365	-0.0293
	(0.0272)	(0.0247)	(0.0267)	(0.0283)
1st lag of Log (total accusations)	-0.0508*	-0.0569	-0.0727***	-0.0566
	(0.0267)	(0.0377)	(0.0269)	(0.0375)
2nd lag of Log (total accusations)	-0.0886**	-0.0835*	-0.0850**	-0.0520
	(0.0388)	(0.0468)	(0.0342)	(0.0480)
3d lag of Log (total accusations)	-0.0871**	-0.0473	-0.114***	-0.0394
	(0.0352)	(0.0554)	(0.0331)	(0.0579)
Constant	-9.383***	-14.20***	-9.330***	-14.49***
	(2.967)	(3.038)	(2.788)	(2.223)
Linear diocese-specific time trend	No	Yes	No	Yes
Observations	1,204	1,204	1,204	1,204
R-squared	0.984	0.992	0.983	0.991

Note: There are 172 dioceses, regressions include year and diocese fixed effects. The numbers in parenthesis are clustered standard errors at the diocese level. *** p<0.01, ** p<0.05, * p<0.1

Chapter 2: School Mandates, Education for Parents, or Physician Recommendations? The Most Effective Way to Increase Human Papillomavirus Vaccine Coverage

2.1. Introduction

About 20 million people are currently infected with Human Papillomavirus (HPV) in the United States. About half of these infections are among adolescents and young adults between 15 to 24 years old. HPV is the most common sexually transmitted disease. Among the more than 40 HPV types that infect human mucosal, most infections are asymptotic and transient; however, certain oncogenic types can cause cervical cancer and a number of less common cancers, including cancers of the anus, penis, and vulva. Other non-congenic types can cause genital warts. Every year about 12,000 women are newly diagnosed with cervical cancer, and about 4,000 women die from this cancer in the United States. About 1% of sexually active males and females in the U.S. have genital warts at any given time (Center for Disease Control and Prevention, 2014).

Two vaccines have been developed recently to protect against HPV. The bivalent vaccine (Cervarix) prevents two HPV types, 16 and 18. These two types are responsible for about 70% of cervical cancers. The quadrivalent vaccine (Gardasil) protects against HPV types 16 and 18, as well as HPV types 6 and 11, which cause 90% of genital warts. The quadrivalent vaccine can also protect against cancer of the anus, vagina, and vulva. The full immunization includes three doses of vaccine in the course of six months. Research conducted on the safety of this vaccine did not show any safety concern, and both vaccines were found to be safe. Some mild side effects of the vaccine have been reported, such as pain where the shot was given, fever, dizziness, and nausea (Center for Disease Control and Prevention, 2014).

The Food and Drug Administration (FDA) approved Gardasil at 2006 and Cervarix at 2009. The Advisory Committee on Immunization Practices (ACIP) recommends either HPV vaccine for routine vaccination for all girls 11 to 12 years old and catch-up vaccination for those 13 to 26 years old who have not been vaccinated previously. Only the quadrivalent vaccine is routinely recommended for boys 11 to 12 years old, and catch-up vaccination is recommended for 13 through 21 year old males. This vaccine is also recommended for gay and bisexual men, and people with compromised immune systems. The main reason for recommending the vaccine for the age range of 11-12 years old is to increase the efficacy of the vaccination. HPV vaccination does not protect against the viruses that an individual is already exposed. As a result, vaccination at earlier ages will increase the likelihood that immunization would occur before any sexual activity. Moreover, it has been efforts to synchronize HPV vaccine delivery with other adolescent required vaccines (Daley et al., 2010).

In order to increase the immunity against this virus, many states have enacted HPV vaccine related laws. This legislation ranges widely; however, most of this legislation in general can be divided into five categories: school mandates, public awareness campaigns, education for parents, education for school children, and health insurance mandates. Figure 2.1 shows the number of states in each category. The ultimate goal of this research is to investigate whether two general classes of policies, school mandates and educational programs for parents, have promoted the vaccination or not. These two policies vary significantly in terms of incurred costs and also the extent that they interfere with freedom of choice, and as a result, it should be of interest to public health policy makers. Providing information for parents can happen at very low cost, and

if this policy is more effective in promoting the vaccine, the cost-benefit analysis would indicate that this policy is preferred. It is also preferred from a political economic perspective because it interferes less with parental choice. To compare the effectiveness of these policies to a baseline treatment, I also intend to study the effect of physician recommendation on the HPV vaccine decisions. The variable indicating receipt of advice from a physician is non-experimental and potentially endogenous, but I employ an instrumental variable approach that is specifically designed to address the endogeneity problem.

Many studies have investigated different aspects of the determinants of vaccine acceptance. Higher income levels and having health insurance are shown to be positively correlated with vaccinations (Jain et al., 2009). Greater awareness of HPV is associated with greater vaccine acceptability (Jain et al., 2009 & Black et al., 2009). However, there are some serious empirical limitations to these studies. Initiation and completion of the vaccine is found to be associated with patient's age. The receipt is lowest among the youngest and oldest eligible age groups and highest among the mid teenagers (Robin et al., 2014).

To best of my knowledge, Bugenske et al., (2012) provided the only published study that explicitly investigates the effect of middle school requirement policies, including school mandates and parental education requirements, on the vaccination rate. They found no association between parental education requirements and the coverage level for HPV. However, this study is only confined to the vaccination rate mean comparison between treatment and control states, while there might be substantive differences between state characteristics. These characteristics can affect the vaccination

rate through channels other than these policies, and these differences are not addressed in this study. As a result, this severe limitation in the applied statistical method makes it difficult to draw a comprehensive conclusion from the results. The current study, however, both controls for the differences in state characteristics, as well as empirically employs a rich individual level data to study the effect of aforementioned policies on the decision to initiate or complete the sequence of HPV vaccine.

Some studies have focused on the effect of health care provider and physician recommendation on the vaccine decision as well and found that receiving a physician recommendation is an important factor (Yilato, et al., 2013 & Rosenthal, et al., 2011). Parents frequently cited not having a physician recommendation as reason for not vaccinating their child (Holman, et al., 2014). Physician failure to start a conversation about HPV vaccine was a leading reason of delayed or missed immunization opportunities among African American adolescents even when mother expressed a strong commitment to HPV immunization (Hamlish et al., 2012). It was even more likely for parents of sons than girls to indicate not having a physician recommendation as the main reason for vaccine refusal (Laz et al., 2012). However, none of these studies took potential endogeneity of receiving a physician recommendation into account. In this study, I address the potential endogeneity by employing an instrumental variable approach.

The most important finding of this paper is that the perceived risk of infection is an important determinant factor in the ultimate success of a policy. In general, these policies were less successful in encouraging the vaccine among children younger than 13 years old. This is mainly due to the fact that the perceived risk of infection is quite low in

this age range, considering the fact that HPV is a sexually transmitted disease. The policies appear to have a limited impact at best on the vaccine outcomes among girls older than 13 years old. This implies that increasing parental awareness can lead to an increase in vaccine initiation; however, parents are willing to wait for their child to become older, and it is the time that the perceived risk of infection is greater.

Physician recommendation, however, is shown to be a strong determinant in initiating and completing the vaccination sequence, as well as initiating the vaccine before the age of 13. The strongest effect of physician recommendation can be observed on the decision to initiate the vaccine. This effect is found to be stronger than the effect of any policy and obviously much cheaper. The results support the argument that receipt of advice from a physician should be treated as an endogenous regressor. Estimates that ignore the potential endogeneity will result in underestimating the true effect of physician recommendation on vaccine initiation and completion. I also present suggestive evidence that physicians tend to recommend the vaccine to those who are less likely to initiate the vaccination by their own.

The rest of the paper is organized as follows. Section 2.2 provides a background of different policies in effect in various states. In section 2.3, I describe the data. In section 2.4, I introduce the methodologies that I employ in my study. In section 2.5, I present and discuss the results. Section 2.6 provides robustness checks. Section 2.7 concludes.

2.2. Background

Introduction of the population based Pap smear test has resulted in a sharp decline in incidence and mortality rates of cervical cancer. U.S cervical cancer incidence rates

decreased by 75%, and the mortality rates declined by 74% in the 50 years following the introduction of cervical cytology in 1949 (Saslow et al., 2008). Despite these impressive statistics, there is a significant racial and ethnic disparity in cervical cancer incidence and its related mortality rates (Reiter et al., 2009). Although these disparities have declined in recent years, the incidence rate remains higher among black women (9.6/100,000) in comparison with white women (7.9/100,000). Hispanic women also have a higher incidence rate (10.9/100,000) than white women. The mortality rate is highest among black women (National Cancer Institute, 2014). Cervical cancer mortality rates are higher in rural areas of the United States, and factors that place women at higher risk of developing cervical cancer are more prevalent in these areas (Brewer and Fazekas, 2007). Moreover, some at risk women are less likely to receive screenings. Half of all women who are diagnosed with cervical cancer have never been screened, and an additional 10% have never been screened in a period of five years before diagnosis (Saslow et al., 2008). As a result, any alternative option that can resolve this disparity could be of interest to public health policy makers.

The prevalence of vaccine types HPV declined from 11.5% in 2003-2006 to 5.1% in 2007-2010 among females aged 14 through 19 years old. This is despite the fact that only 49% of females aged 13-17 had received at least one dose of vaccine, and 32% had finished the whole sequence of three doses of vaccine at 2010. Almost all HPV vaccines administrated in the United States were the quadrivalent HPV vaccine (Markowitz et al., 2010). Figure 2.2 shows the national trend of share of girls between 13 to 17 who initiated and completed the sequence of the vaccine. The long term effect of the vaccine is unknown due to the short time span of implementation. However, some estimates

predict the possibility of 70% reduction in cervical cancer rates depending on the number of HPV types eventually included in future HPV vaccines and the vaccination participation rate (Saslow et al., 2008). Vaccine programs can potentially most benefit those lacking access to routine Pap screening programs.

There are several reasons that can be outlined for the relatively low vaccination rates in the United States. One important barrier to receipt of the vaccine is the cost of the vaccine. Depending on health insurance status, parents might have to pay part or all of the cost of the vaccine. This can include vaccine administration fees. Another potential barrier to vaccine acceptability is lack of motivation by parents to have their daughters vaccinated. The lack of interest is mostly derived from concerns about the safety of the vaccine and also the perception among parents that HPV is not an imminent risk to their daughters' health. If parents believe that their daughter is not sexually active or the child is not of the appropriate age, they might undermine the necessity of vaccination and postpone it for the future (Brewer and Fazekas, 2007; Holman, et al., 2014).

To increase the vaccination rates, within a year after approval of the vaccine, a wide vaccine-related legislative activity began in different states. Legislation introduced in 41 states and the District of Columbia. It includes bills in 22 states and the District of Columbia that would mandate the HPV vaccine for sixth grade girls (Mello et al., 2012)[8]. Figure 2.3 provides an overview of legislative activity at 2006. Media reports following the burst in legislative activity made the claim that the vaccine manufacturer, Merck, was heavily involved in promoting school mandates. These reports generated a controversy

[8] It includes California, Connecticut, District of Columbia, Florida, Georgia, Illinois, Kansas, Kentucky, Maryland, Massachusetts, Michigan, Minnesota, Mississippi, Missouri, New Mexico, New York, Ohio, Oklahoma, South Carolina, Texas, Vermont, Virginia, and West Virginia.

about the degree to which industries should be involved in vaccine policy (Tomljenovic & Shaw, 2012). Since 2006, legislators in at least 45 states and territories have introduced legislation with regard to the vaccine and at least 25 states and territories have enacted legislation (National Conference of State Legislation, 2013)[9]. Table 2.1 lists each state with a vaccine-related law, the category that the law falls in, along with the year that the law became effective and whenever is needed some basic information about the legislation is provided.

The most contentious policy among all the vaccine-related policies is school mandates. School mandates make HPV vaccination compulsory for a specific age group, mostly sixth grade girls. Many states saw the introduction of at least one school mandate bill but in almost all the cases, the bills ultimately failed. On February 2, 2007, Texas became the first state that enacted the mandate by executive order; however, almost immediately, the legislators passed a bill to override the executive order and the governor withheld his veto (National Conference of State Legislation, 2014). By the end of 2011, only Washington DC and Virginia had enacted school mandates. However, both states offer liberal opt-out actions that allow parents to decline the vaccination for their daughters for almost any reason (Stewart, 2008). Multiple attempts by some legislators in Virginia to repeal the requirement that school girls be immunized against HPV failed (National Conference of State Legislation, 2014).

There are multiple impediments to the adoption of school mandates. First, school mandate bills were introduced only a few months after the vaccine became available in

[9] It includes Colorado, District of Columbia, Illinois, Indiana, Iowa, Kansas, Louisiana, Maine, Maryland, Michigan, Minnesota, Missouri, Nevada, New Mexico, New Jersey, New York, North Carolina, North Dakota, Oregon, Puerto Rico, South Dakota, Texas, Utah, Virginia, and Washington.

the market; as a result, many legislators along with public health officials did not support the law due to the fact that they believed long-term safety data are needed before mandatory vaccination can be justified. Second, the sexually transmitted nature of HPV caused some social conservatives to object to a compulsory policy because they believed it might lead to reduce the influence of their messages to promote abstinence. Third, there was an argument at the time that vaccine mandates were supposed to prevent the spread of contagious diseases and school enrollment should not be used to meet other public health goals. This argument, coupled with the fact that HPV is not contagious through casual contact, created another barrier against adoption of school mandates. The same reasoning was used in Virginia to justify the liberal opt-out provision in its legislation. Fourth, media coverage of the manufacturer's aggressive tactics to promote school mandates led to the public's perception that the proposition of these bills is merely due to the company's policy and not the product's merit, and people who were supportive otherwise pulled back. And finally, mandatory vaccination required financial resources in order to cover the vaccine's cost, especially for Medicaid and S-CHIP programs. Considering the fact that Gardasil is notably more expensive than other required vaccines, HPV vaccine mandates were believed to consume too great a share of states' Medicaid and public health budgets (Colgrove et al., 2010).

Mandate proposals for the HPV vaccine, like any other compulsory health measure, are politically and ethically sensitive because they violate freedom of choice for parents. The issue would become even more sensitive when it is realized that HPV vaccine intersects with human sexuality (Colgrove et al., 2010). To avoid such complications, some states have adopted different regulations than school mandates. One

of the most common alternatives to compulsory vaccination is the provision of educational content about HPV for parents. Among all the states that proposed school mandates, four states eventually adopted such a policy and one adopted a health insurance mandate. The remaining states have not adopted any policy. The common theme among all of these bills is that they would provide educational content to parents about HPV, its link to cervical cancer, and availability of the vaccine as well as potential side effects of vaccination. Reference groups subject to this class of policies are different in different states, but all these policies are intended to increase vaccination rate through increased parental awareness. There are also states that require health education for students to cover the information about HPV, but most of the time the requirements are not binding. School districts usually have the authority to decide whether they want to include health and sexual education in their curriculum or not.

In addition to policies that encourage immunization against HPV, coverage of the cost of the vaccine is also a crucial factor in the decision to accept the vaccine. A full course of HPV vaccination costs about $390, which is significantly more expensive than most other required vaccines. Different states offer a wide range of programs to cover the cost of the vaccination. One of the oldest programs is federal-state Vaccine for Children (VFC) program. VFC took effect in October 1994 and it covers more than 35 million children below age of 18. VFC provides recommended vaccines by ACIP, including the HPV vaccine at no cost for certain groups. Medicaid eligible children, Alaska native and American Indian, and uninsured children are eligible for VFC. The program also provides vaccines for underinsured children at Federally Qualified Health Centers (FQHC) and Rural Health Clinics. Section 317 of the public health service act is another federal

program administrated by CDC, and it provides grants to states and territories, commonwealth trusts, and several cities for vaccine purchase and surveillance programs. States could only purchase childhood immunization under section 317 prior to enactment of the Affordable Care Act (ACA); however, ACA authorizes states to purchase recommended vaccines for adults under this section (National Conference of State Legislatures, 2011).

The majority of states rely solely on federal resources to purchase vaccines, but some states supplement programs in order to cover a larger target population. State Universal Purchasing Programs supplement VFC and section 317 provisions by supplying the ACIP-recommended vaccines to privately insured children and adolescents. The number of universal states rose by 15 by end of 2000 but since then, ten states have changed their status to what is termed “universal select” meaning that they cover all but selected vaccines. The high cost of new vaccines was the main obstacle to sustain universal purchase programs and there are the vaccines most likely to be omitted in “universal select” states. At the beginning of 2008, 7 states had universal programs (Alaska, New Hampshire, New Mexico, Rhode Island, Vermont, Washington, and Wyoming). Since then, Alaska, Washington, and Wyoming have converted to universal select states (Benatar et al., 2010). None of the universal select states provides the HPV vaccine for private insurance holders and their supply is limited to VFC eligible children. Since the approval of the vaccine, many health insurance policies have stepped forward and provided coverage for the HPV vaccine. However, these policies vary significantly in terms of cost sharing. During the same period, some states have mandated health insurance policies to include the HPV vaccine in their preventive services. These policies

also vary greatly with respect to the cost sharing requirements and covered age groups. In addition to those states, according to the Affordable Care Act (ACA), new insurances and plans as of September 23, 2010 are required to provide preventive services, including ACIP-recommended vaccines without imposing out of pocket costs on the policy holder such as copayment or coinsurances.

In the current study, I investigate how two mainly competing policies, school mandates and providing educational content for parents, can affect the decision to initiate and complete the vaccination. Moreover, I also intend to further examine the effect of physician recommendation on the probability of vaccine initiation and completion and to compare its effect with the effect of policy variables. Physician recommendation is shown to be one of the most important factors in health related decisions (Kenkel & Terza, 2001; Kreuter et al., 2000). Receiving advice from a physician with regard the vaccine might largely influence the decision to vaccinate. When it comes to social welfare, physician recommendation can be very cost-effective due to the fact that it is a more targeted intervention and it does not impose much cost on tax payers.

2.3. Data

The data I use come from National Immunization Survey-Teen (NIS-Teen) from 2008-2011. This survey collects information about vaccination records of teens between 13 to 17 years in all 50 states, District of Columbia, and selected area for oversampling. The NIS is a list-assisted random-digits-dialing telephone survey followed by a mailed survey to the teen's immunization providers. This is also a period of the time during which most states enacted their HPV related regulations. The fact that the data are

collected from providers, in addition to the households, increases their reliability. This database also collects information whether an individual has ever received a recommendation from a physician concerning HPV vaccination.

I merge the NIS-Teen data to information on the enactment of HPV related regulations in different states. I aim to study the effect of school mandates and provision of educational content for parents about HPV on both extensive and intensive margins of vaccine decisions, as well as physician recommendations. The latter is also asked of all respondents. In addition to the direct effect of these policies on the vaccination decision, they can also affect the prevalence of physician recommendation and influence the vaccine outcomes indirectly through this channel. As a result, inclusion of the policy variables is critical in estimating the effect of physician recommendation. Moreover, it provides a baseline to compare the effect of these policies and the effect of physician recommendation. School mandates are very similar in wording across states. The content of educational packages distributed among parents is very similar in different states as well; however, each state has targeted a different age group. I restrict my sample to females because mostly just women are subject to these regulations.

I drop all the states that have enacted educational programs about HPV for students and those that have required health insurance plans to include the HPV vaccine in their preventive services[10]. As previously mentioned, educational programs for students, most of the time are not binding. As a result, it is almost impossible to determine whether or not an individual in the sample had ever received any educational content about HPV or not. This impairs the possibility to appropriately control for this

[10] It includes Colorado, Illinois, Iowa, Louisiana, Nevada, New Mexico and Oregon.

variable. Health insurance requirements are heterogeneous in terms of cost-sharing policies among the states that have adopted such a policy. Coverage of the vaccine may be subject to deductibles and coinsurance depending on the choice of health insurance plan in some of the states with this policy in effect. It is worth mentioning that cost-sharing policies among various health insurance plans and different states are very different which are unobservable to me. Moreover, many health insurance policies had included HPV vaccine in their preventive services shortly after approval of the vaccine regardless of legal requirements by states. Consequently, individuals subject to this policy might not be significantly different in terms of the exposed vaccination cost with residents of other states without a health insurance mandate policy. As a result, it is technically impossible to adequately control for this variable.

In addition to the potential direct effect of educational programs for student and health insurance mandates, the vaccination decision might be affected by some unobserved channels that are influenced by these policies. For example, HPV awareness of those who have not received any information about the disease can still increase through discussing it with other families and network externality. While both of these policies can affect the vaccination decision, the adoption of these policies is likely to be correlated with adoption of school mandates and education for parents programs. Moreover, both policies, and more specifically health insurance mandates, can also influence the probability of receiving a recommendation from a physician. As a result, the disability to control for these variables will lead to biased and inconsistent estimates for both policy and physician recommendation variables.

As previously mentioned, there are a set of states dubbed "universal states." These states provide ACIP-recommended vaccines for privately insured children, as well as VFC eligible ones (Alaska, New Hampshire, New Mexico, Rhode Island, Vermont, Washington, and Wyoming). It is reasonable to assume that the cost associated with the vaccination in these states is lower in comparison with my treatment group and it might affect the vaccination decision. This might undermine the credibility of these states to be included in my control group. However, most of these states did not change their status over the sample period (New Hampshire, New Mexico, Rhode Island, Vermont, and Wyoming) and state dummies can sufficiently control for the effect of universal vaccine purchase programs in these states.[11] There are also states that have public awareness campaigns. These campaigns don't necessarily target a specific age group or parents only (Colorado, North Dakota, and Utah). This policy can encourage vaccination through the enhanced public awareness and inclusion of them in the control group might be problematic. However, all of these states had this policy in effect the whole sample period and, as a result, following the same reasoning as the above, state dummies should control for the effect of this policy.[12]

My final sample therefore consists of 43 states and District of Columbia over a 4 year period for a total of 56,004 observations. I merge the state level HPV vaccination rates data and the rates of three common sexually transmitted diseases among young people 15-24 years of age, estimated by Center for Disease Control and Prevention, and

[11] Alaska changed its status in January 2009, Washington changed it in July 2009, and Wyoming changed it in July 2011 to universal select states.

[12] I initially include all the universal states and the states with a public awareness campaigns, but I eventually drop those states that have changed their status to "universal select" during the sample period (Alaska, and Washington) and report the results including the remaining states from my control group to verify the results. The results are qualitatively the same and will be available upon request.

state level cervical cancer prevalence data from National Program of Cancer Registries (NPCR) to my database.

2.4. Methodology

In order to investigate the effect of physician recommendation on vaccine decisions, as well as the effect of school mandates and educational programs for parents, I begin by estimating:

$$Y_{ijt} = f(\alpha + \beta_1 X_{ijt} + \gamma_j + \delta_t + \beta_2(\text{age}^* \text{Year}) + \beta_3(schman_{ijt}) + \beta_4(edupar_{ijt}) + \beta_5(\text{recom}_{ijt}) + \varepsilon_{ijt}) \quad (1)$$

I estimate equation 1 using a probit model. Y_{ijt} is a variable that either represents the vaccine initiation (*dose≥1*), or the completion of the vaccine (*dose≥3*). In some specifications, I use a variable that indicates whether a child has been updated for this particular vaccine (*dose≥1*) before the age of 13. This variable allows me to study the effect of desired policies on a wider range of age groups. It also provides the opportunity to examine the possibility of heterogeneous treatment effects. A decision to get vaccinated can be strongly influenced by child's age due to the sexually transmitted nature of the HPV vaccine. I initially use the reported data by providers to construct the dependent variable, however, when these data are missing, I use the household reports to fill this variable. Failure to obtain provider data is attributable to two main reasons. First, the family did not give the consent to contact the teen's vaccination provider. Second, communication with provider was not possible because either contact information for

provider was not adequate or the provider did not respond.[13] I also utilize the vaccination records data after the age of 13 to update the vaccination status before the age of 13. I will replace the update status before the age of 13 which are missing by zero, if the vaccination records after the age of 13 indicate that a child has not received any dose of vaccine.

The variable *schman* is a dummy variable indicating that a child lives in a state where school mandates for HPV vaccines are in effect and the child has been in the covered age group of the legislation at any time after the law was enacted. *edupar* is a dummy variable that indicates those individuals who are in the states in which educational content about HPV and its immunization is being distributed among parents, and they were in the targeted age group at any time after the law's enactment. *recom* is a dummy variable indicating whether parents have received a recommendation regarding HPV vaccine from a physician or not. γ_j is a vector of state dummy variables that are intended to capture time-invariant factors that cause Y to differ between states. δ_t is a vector of year dummies that captures the difference in Y in different years that are common among the states. I also include the interaction of age dummies and year dummies to allow Y to vary differently over time by age groups.

I assume that the imposition of these policies is exogenous. As I discussed earlier, most states introduced their regulations shortly after the vaccine was approved. The main reason for this outbreak in introduction of the vaccine related bills was the legislative efforts by the manufacturer (Tomljenovic & Shaw, 2012). Forty-one states and DC

[13] I also estimated regressions that treat the unavailable vaccination records from the provider as missing, or use household data and include a dummy variable that represents when data from provider is missing, and Heckman probit model for selection. In all the cases, the results are qualitatively the same.

introduced bills with regard to the HPV vaccine in 2006-2007. Twenty-two states and DC made efforts to require the HPV vaccine for school enrollment at the same period of time. Among twenty-five states and territories that currently have the vaccine related laws in effect, twenty-two started their legislative process back in 2006-2007 (National Conference of State Legislation, 2013). One might suspect that there are some state-related factors that affect legislation adoption, the category that the law falls in, as well as the vaccination such as degree of conservatism and religiosity. However, geographic dispersion of the introduction and the passage of state laws within each category of the legislation, as well as the timing of the laws, relieve this concern. It is also worth mentioning that I include state and year dummies in my specifications, and these unobservable factors are unlikely to change over a course of a four year period. Moreover, this burst in the legislative activities only a short time after the approval of the vaccine will rule out the possibility that the introduction and passage of these bills was due to the general public demand because the awareness about the vaccine in that time was limited.

X_{ijt} is a vector of control covariates. I include controls for age, mother's marital status, mother's education, mother's age categories, race, number of children below age of 18 in the household, number of people in the household, income, and teen health status, and overall health status within the household. It also contains information about health insurance status. Health insurance status is divided into three categories. The first category is private health insurance policies that might or might not cover the cost of the HPV vaccine. The second is public health insurance that covers the entire cost of the vaccination. The third are privately insured but classified as underinsured and receive the

full coverage for the vaccination costs through VFC program in this category. The final category is military insurance plans that cover the cost of vaccination partially to fully depending on the type of the program. I separated military insurance from other categories because I am concerned that serving in the military can be associated with some unobservable socioeconomic factors that might affect the vaccination decision through some other channels rather than simply cost of the vaccine. Moreover, access to healthcare is different among different health insurance policy holders. Many physicians do not treat publicly insured patients (Currie & Grubber, 1996). Physicians are less willing to accept any new Medicaid patients compared with private health insurance holders after Affordable Care Act enactment (Decker, 2012). I consider private health insurance holders as a reference group, and I only include dummy variables for holding public and military health insurance plans. I include two more variables to further control for access to healthcare. First, I include a variable that indicates whether there was any period of the time after age 11 that a teen did not have any health insurance coverage, second, I include a variable that represents whether a child has visited a doctor in the past 12 months. I also include the rates of one of the most common STDs, Chlamydia, among young people 15-24 years of age in each state and year in the regression analysis. I include a variable that represents whether a teen has had at least one shot of Tdap (tetanus, diphtheria, and pertussis) since age 10 years. Tdap vaccine is recommended for preteens at age 11 or 12 years which is the same recommended age range for the HPV vaccine. It is likely taking Tdap vaccine influences the decision to initiate the HPV vaccine. Table 2.2 presents the definition and coding of the control variables.

It is important to point out that physicians' advice might be related to some unobserved factors that can affect the vaccine related decision outcomes simultaneously. This can result in biased and inconsistent estimates. For example, parents who have higher value for health might seek to receive a recommendation for their daughters. On the other hand, physicians might recommend the vaccine more to those individuals who are more probable to get involved in risky behaviors and at the same time tend less to get vaccinated.

The first means to identify plausibly an exogenous variation in the physician advice is instrumental variable probit model. This approach requires identifying the variables that affect the probability of receiving advice from a physician but are free of correlation with factors affecting the vaccine outcomes, conditioned on other covariates. I begin with the premise that a chronic condition or any other reason can influence the probability of visiting a physician, it can also influence the probability of receiving advice for vaccination. If these factors are unrelated to vaccine decisions conditional on other observables, they only can explain the variation in the outcomes of interest through the variation in physician recommendation and they can be used as plausible instrumental variables. The first instrumental variable I employ is the history of asthma, and the second instrumental variable is an indicator for participation in 11-12 year old well child exam. Asthma is leading chronic condition among children and adolescents in the United States. The National Heart, Lung, and Blood Institute guidelines for the clinical management of asthma recommend periodic ambulatory visits for asthma monitoring (Akinbami, et al., 2011). Child well-exam is usually required for secondary school enrollment and participation in these checkups is not necessarily correlated with the HPV

vaccine decision. The high participation rates in the sample can provide an evidence for this claim. It is also possible that parents have to take their children to this routine checkups in order to update their kids for Tdap boosters. 35 states have Tdap requirements for school entry[14]. It is worth mentioning that I control for Tdap update status in my specifications The IV-Probit estimation is summarized by:

$$\begin{aligned} Y_{ijt} &= 1(\alpha + \beta_1 X_{ijt} + \gamma_j + \delta_t + \beta_2 (hpvinit_{ijt}) + \varepsilon_{ijt} \geq 0) \\ hpvinit_{ijt} &= 1(\alpha + \varphi_1 X_{ijt} + \gamma_j + \delta_t + \varphi_2 (\text{asthma}_{it}) + \varphi_3 (\text{checkup}_{it}) + u_{ijt} \geq 0) \end{aligned} \quad (2)$$

$$\begin{pmatrix} \varepsilon \\ u \end{pmatrix} | Z : \; N\left(\begin{pmatrix} 0 \\ 0 \end{pmatrix}, \begin{pmatrix} 1 & \rho \\ \rho & 1 \end{pmatrix} \right)$$

Z is the vector of instrumental variables and exogenous variables. *asthma* is the history of asthma which is equal to 1 when a child has been diagnosed with asthma at any stage in her life and it is 0 otherwise. The second instrumental variable, *checkup* is a dummy variable which is equal to 1 when a child had 11-12 child-well exam.

2.5. Results

Table 2.3 presents the results from the baseline regression (equation 1) as well as IV-probit model as well as the results from the first stage (equation 2). I also report the marginal effects of aforementioned variables in Table 2.4. The dependent variable is either indicator for vaccine initiation (*dose≥1*) or vaccine completion (*dose≥3*) or an indicator for whether a child has been updated for the HPV vaccine (*dose≥1*) before the

[14] It includes Alabama, Alaska, Arizona, Arkansas, Colorado, Florida, Illinois, Indiana, Kansas, Kentucky, Louisiana, Massachusetts, Michigan, Minnesota, Montana, Nevada, New Hampshire, New Jersey, New Mexico, New York, North Carolina, North Dakota, Ohio, Oregon, Pennsylvania, Rhode Island, South Carolina, Tennessee, Texas, Utah, Vermont, Virginia, Washington, Wisconsin, and Wyoming

age of 13. All the specifications include state and year dummies as well as the interaction of age dummies and years.

Point estimates of the effect of educational programs for parents about HPV for parents have the expected positive sign on the vaccine initiation, however, the estimates are not statistically significant. The estimated effect of this policy on the decision to complete the sequence of the vaccine is positive but it is smaller than the effect the policy has on the decision to initiate the vaccine and it is statistically insignificant. The effect is very small and highly insignificant when I restrict my dependent variable to the vaccine update status before age of 13. This implies that this policy failed to encourage the vaccination among the children younger than 13 years old. Parents might be reluctant to vaccinate very young children against HPV. This might be derived from sexually transmitted nature of HPV vaccine.

I find the anticipated positive association between the school mandates and vaccine initiation among the girls between 13 to 17 years old. The point estimates are very close to what I obtained for educational programs for parents, but the coefficients are not statistically significant. This perhaps reflects the small number of treatment units compared to the whole sample. The point estimates remain positive but statistically insignificant when the outcome of interest is decision to complete the sequence of the vaccination. The effect of school mandate becomes smaller and remains insignificant when I restrict my outcome to the update status prior to age 13, but the coefficients remain insignificant at conventional levels. This is somewhat an interesting finding considering the fact that school mandates target sixth grade students. Altogether, the

estimates in Table 2.3 do not allow me to draw a conclusion that both policies, school mandates and educational programs for parents, promote the HPV vaccination.

Physician recommendation is a strong predictor in all the specifications. Receiving advice from a physician can increase the probability of vaccine initiation, and vaccine completion. It can also increase the probability of starting the process of the vaccination before the age of 13. The strongest effect of physician recommendation is on the decision to initiate the vaccination. It is worth mentioning that the full immunization consists of three doses of the vaccine in the course of six months. A possible explanation for the weaker effect of physician recommendation on vaccine completion compared to vaccine initiation is that younger girls may not be currently updated but they are scheduled to complete the whole sequence of the vaccine in the future. Moreover, side effects from the vaccination might deter the parents from continuing the vaccination. Initiation of the vaccine also can decrease the perceived risk of infection resulting in declined desired to complete the sequence of the vaccination. On the other hand, the true cost of the vaccine for those who don't have a full coverage for this matter might be realized after implementing the first dose, and a result, discourages the completion of the vaccine sequence. The point estimates for physician recommendation is larger than any of the policy related coefficients and the difference is statistically significant. It indicates that receiving advice from a physician can be more effective than any policy in promoting the vaccination. As can be seen, estimates that ignore the endogeneity of advice can lead to underestimation of the effect of physician advice on the vaccine related outcomes. Physician recommendation is still the strongest predictor of the vaccine decision and the effect is greater than both policies. Coefficients of school mandate and

educational programs for parents follow the same pattern as before even after taking physician recommendation into account.

Note that ρ captures the potential correlation between unobservables that determine receipt of advice and vaccination outcomes. In all the models that corrected for endogeneity, ρ is negative and statistically significant. This is consistent with the theory that physicians recommend the vaccine more to those individuals who are less likely to begin the vaccination by their own. It also supports my contention that receive of advice should be considered as potentially endogenous variable.

The results in Table 2.3 indicate that holding public health insurance policies is associated with the higher probability of both vaccine initiation and completion compared with private health insurance holders. It also increases the relative probability of vaccine initiation before the age of 13. The results for military health insurance holders show the same pattern. These health insurance policies offer more complete coverage than private health insurance policies. Not having a health insurance coverage at some period of time since age 11, and not visiting a doctor in the past 12 months is negatively associated with all the dependent variables, and in all the specifications.

Families with 2 children in the household are more likely to accept the vaccine compared with single child families. The trend is similar for the families with 3 children or more; however, the coefficients are less precise. Increasing the number of children in a family can lead to an increase in the likelihood of family's awareness toward HPV and its immunization. For example, if parents were exposed to a vaccine related policy because one of their children was in the targeted population of the policy, they might change their decision with regard to the vaccination not only for that child but also for the rest of their

children as well. Hispanics are more willing to initiate or complete the sequence of the HPV vaccination. Mothers with some college experience are more likely to finish the entire sequence of the vaccination for their daughters. The estimated coefficients for income is surprising. Income is negatively correlated with the vaccine outcomes after the age of 13. Lower income can lead to eligibility for governmental subsidies and increase the vaccination rate through this channel. Income can also be associated with some other socioeconomics factors that tend to decline the vaccination rate. The estimated effect of health status has the expected sign; however, the coefficients become less precise for vaccine completion and update status before age of 13. It is noteworthy that my measure of health status consists of indicators for a wide range of chronic diseases that some of them might not necessarily interfere with the vaccination decision[15]. Household health status on the other hand, is positively associated with vaccine outcomes. It is possible that those families who have to deal with chronic health conditions might seek preventive options for their children in order to protect them. Tdap update status is a strong, positive predictor of the HPV vaccine decisions. As previously mentioned, Tdap is required in many states and is recommended over the same age range that HPV vaccine is recommended, and one might expect that the decision to take one vaccine can positively influence the decision to uptake the other.

I am returning to a closer look at physician recommendation identifying assumption. Both instrumental variables have the expected positive effect on the probability of receiving advice from a physician in the first stage estimation. It indicates

[15] I drop the coefficients for the prevalence of Chlamydia in the Tables for lack of space. The estimated effect of the prevalence of Chlamydia as one of the most common sexually transmitted disease is very small and insignificant. These estimates will be available upon request.

that those individuals who need to visit a doctor frequently for asthma conditions or visited a physician for an 11-12 child-well exam are more likely to receive a recommendation to get vaccinated. As part of a standard procedure, I also report the F test from excluded IVs and it indicates that excluded IVs are not weakly identified.

Using the IV approach necessitates some post estimation diagnostic tests. I am required to verify that the IVs are not weak and they are valid, moreover, the IVs should not be correlated with error terms. Since some of these post-estimation diagnostic tests are not available for IV-Probit model, I use 2SLS for robustness checks and validating my instruments. (Angrist, 2000). Table 2.5 presents the results for this alternative for robustness checks. The results are qualitatively the same and indicate that the physician recommendation is a strong predictor of the vaccination decision. The results of the F test from excluded IVs are also reported in Table 2.5. Excluded IVs are jointly significant when I control for other explanatory variables in the first stage. It indicates that the endogenous variable is not weakly identified. I also use the Hansen test for over-identification restriction test in my 2SLS framework, the P-Value is large enough to conclude the validity of instrumental variables.

I can generally conclude that the increased awareness of the vaccine resulted from the policies or receiving advice from a physician will eventually encourage the vaccine initiation and completion. However, parents tend to wait until their daughter gets older. It is important to point out that one needs be sexually active in order to be infected by HPV; as a result, the perceived risk of infection might not be very high for young girls. This might explain why the weakest effects of different variables can be observed among girls

younger than 13 years old. It is also critical to consider that school mandates offer a liberal opt out provision and the decision to decline the vaccine is not costly for parents.

These results provide suggestive evidence that the effect of physician recommendation is substantially larger than the effect of other policies that aim to promote the vaccination. These estimates suggest that policies to encourage physician advice about the HPV vaccine to parents are likely to yield substantially more benefits than other policies considering the fact that physician recommendation can also be really cost-effective. Despite the strong effect of physician recommendation on HPV vaccine uptake, many physicians hesitate to recommend the vaccine (Hamlish, et al., 2012). There is evidence that physicians are less likely to recommend the vaccine when they are male, and uncomfortable discussing human sexuality issues with female patients (Gamble et al., 2010). Financial concerns including reimbursement for vaccination, and vaccine purchasing costs were cited as some of the most important perceived barriers to recommend the vaccine by physicians. Parents' opposition for moral or religious reasons was also perceived as a barrier to recommend the vaccine (Daley et al., 2010). The framework used by physicians to convey the message to parents is also very influential in the final decision to uptake the vaccine. CDC research shows that the "HPV vaccine is cancer prevention" message resonates strongly among parents (Center for Disease Control and Prevention, 2013).

2.6. Additional Estimates and Robustness Checks

2.6.1. Robustness Checks for the Effect of Physician Recommendation

It is important to investigate how sensitive the results are to different combinations of instrumental variables. First, one might suspect that participation in 11-12 child-well exam is endogenous. I drop this variable from my exclusion restriction and run the regressions including asthma history as the only instrument. Panel A of Table 2.6 presents the results of these new estimates. The results are firmly consistent with the previous findings.

I also include a new instrument instead of 11-12 child-well exam. This new instrument indicates whether a child missed school more than 30 days in the last year because of illness and injury. The HPV vaccine is not recommended for those who are currently ill, and it is likely that an exogenous shock in health because of the aforementioned reasons might lead to a decline in probability of receiving a recommendation by a physician. The results from these new estimates are presented in Panel B of Table 2.6. The coefficient of the variable that represents whether a child missed more than 30 days in school is negative and less precise than the asthma indicator. It implies that these individuals are less likely to receive a recommendation with regard to the HPV vaccine compared to those who missed school less than 30 days. As previously mentioned, this variable indicates whether a child has missed the school for more than 30 days because of illness or injury. Loss of school for more than 30 days may represent a critical health condition, and a physician may avoid recommending the vaccine in these circumstances and leave the vaccination for the future. Moreover, a child might experience restriction of mobility in the case of an injury, and these restrictions might

decrease the probability of visiting a physician, and as a result, receiving an advice to vaccinate against HPV. The physician recommendation remains a strong and positive predictor over different ranges of outcome. However, these new estimates do not provide any evidence of endogeneity. Correlation coefficient, ρ is very small and statistically insignificant in all the specifications but the point estimates remain negative. On the other hand, the F-statistics from excluded IVs decrease significantly in these estimations that raise the concern of weak identifications. Overall, these new estimates also indicate that physician recommendation is the strongest predictor among all on the HPV vaccine decisions[16]. I also report the marginal effects for the policies and physician recommendation in Panel C of Table 2.6.

2.6.2. Robustness Checks for the Effect of Policies

Throughout this paper, I find that the effects of both policies on vaccine outcomes are very limited. I employ multiple empirical strategies to check the robustness of these findings. I drop physician recommendation indicator in these new estimates to avoid potential multi-colinearity problem. First, I re-estimate the equation 1 without including physician recommendation. Panel A of Table 2.7 presents the results from this new identification. The results are qualitatively the same as before and only a modest effect of policies on vaccine outcome after age of 13 is observable. Second, School entry requirements for HPV vaccine are not strict and they offer a very liberal opt out. As a result, one might expect that school mandates will ultimately affect the vaccine decision through the educational content they provide for parents and enhanced parental

[16] I also run 2SLS, and the results are consistent with the IV-Probit framework and all the post-estimation diagnostic tests indicate that instruments are valid

awareness. I use this unique feature of school entry requirement for HPV vaccine to redefine school mandates as a type of educational program for parents. This can potentially increase the statistical power of my estimates especially when it comes to the effect of increased parental awareness resulted from educational content provided to them on decision to complete the entire series of the vaccine. The results if this new regression is presented in Panel B of Table 2.7. The results consistently with the previous findings indicate at best, a limited effect of the policies on vaccine outcomes for the girls between 13-17 years old. The coefficients are very small and insignificant for vaccine initiation before the age of 13, emphasizing the age sensitivity in parental decision to initiate the vaccine.

In general difference-in-difference requires careful control group selection. Finding a sensible control group becomes even more critical when the number of observations in the treatment group is very small compared to the entire sample. This concern is more pronounced for school mandates because of the fact that this policy has been enacted in only Virginia and DC. In order to address this concern, I will follow multiple strategies to construct a more comparable control group and ensure the robustness of the results.

First, I restrict my sample to those states that have introduced school mandates at some point. These states might share some features that make them more comparable in this sense. Moreover, given the controversial nature of this law, people in these states are more likely to have been exposed to related news and analysis which can affect the parental awareness of the virus and its immunization. Table 2.8 presents the marginal effect from this new comparison group. The results follow the same pattern as before, the

only modest different is that the estimated coefficient of educational program for parents on vaccine initiation becomes larger and statistically significant. Other estimates remain statistically insignificant, and consistent with the previous findings, the smallest effect can be observed on the decision to initiate the vaccine before age of 13.

A more systematic approach toward constructing a sensible control group is synthetic control method (Abadie et al., 2010). Synthetic control method is a data-driven procedure that provides a single control unit as a weighted average of characteristics of several potential comparison units. The weights determine the relative contribution of each control unit to the counterfactual of interest. Despite the many benefits, this method is designed for aggregate level panel data while I am using an individual level repeated cross section database. However, Center for Disease Control and Prevention utilizes the same individual level database in order to estimate the HPV vaccination rate among girls between 13-17 years old. I use the data on vaccination rate along with aggregate state level data on different characteristics to build my synthetic group and obtain the weights, and then, I use these weights in the basic specification to estimate the effect of school mandates. The synthetic control is created by matching on the unemployment rate, median income, the relative percentage of Hispanic population, the relative percentage of Black population, population density, the percentage of the female population above age 25 with a bachelor degree or higher, the percentage of the under age 18 population with public health insurance plans, and the percentage of married households.

I only consider Virginia as my treatment state and drop DC from my analysis in this section for various reasons. First of all, I am allowed to specify only one treatment group in this method. One might consider weighted average characteristics of these two

states instead; however, DC is significantly different in terms of different characteristics from other states, and as a result, using a weighted average approach might be misleading in specifying the synthetic control group. Moreover, Virginia only has enacted school mandates while DC has educational program for parents in effect in addition to school mandates. Therefore, not all the change in the observed trend of vaccination rate after treatment can be attributed to the effect of school mandates. Following the same reasoning as before, I drop all the states that have educational programs for students and health insurance mandates. Table 2.9 presents the vaccination rate predictor means in Virginia and synthetic Virginia. Table 2.10 presents state weights in Synthetic Virginia. The weights indicate that vaccination rate trends in Virginia prior to enactment of school mandate is best reproduced by a combination of Alaska, Hawaii, Maryland, North Dakota, South Carolina, and Utah.

The synthetic control method relies heavily on pre-treatment observations in order to match the pre-treatment trends between groups, and the fact that I only have one pre-treatment period might cause some concerns about the validity of the results. Considering the fact that Maryland has obtained the highest weight among all the potential control units, however, can reduce this concern. Maryland is Virginia's neighbor and is expected to share many characteristics with Virginia, including that it once introduced school mandates. The marginal effects from synthetic control method for both probit and linear probability model are presented in Table 2.11. These results also indicate that school mandates did not promote the vaccination on different margins.

2.6.3. Difference-in-Difference-in-Difference

In each year, a new age group is subject to school mandates. As a result, an alternative to the basic specification to identify the effect of school mandates is Difference-in-Difference-in-Difference. In this method, I use both a different state and a control group in the treatment states that were not affected by this policy as my control group. The DDD starts with the time change in averages between the treatment group in the states with the policy in effect and then nets out the change in means for treatment group in control states and non-treatment group in the treatment stat (Imbens & Wooldridge, 2007). I use Virginia as my treatment state to construct the DDD framework. Unlike DC that has enacted both school mandate and educational program for parents, Virginia only has school mandate in effect. I drop all the states that have educational programs for parents, including DC, to obtain a clear control group for this framework. This estimation is summarized by:

$$Y_{ijt} = f(\alpha + \beta_1 X_{it} + \gamma_j + \delta_t + \beta_2(\text{schman}_i) + \beta_3(\text{treat}_j * \text{after}_t) + \beta_4(schman_i * \text{after}_t) + \beta_5(schman_i * treat_j) + \beta_6(schman_i * after_t * treat_j) + \varepsilon_{ijt}) \quad (4)$$

The variable *schman* is a dummy for treatment group which is one when an individual is in the age range group that was targeted by this policy and zero otherwise. *after* is a dummy variable that is equal to one if it is after the law's enactment and zero otherwise. *treat* indicates treatment state which is Virginia in this case. B_2 controls for the time invariant characteristics of the treatment group. β_3 controls for the change over time in treatment state. B_4 controls for the change over time in the treatment group for the entire states in the analysis. B_5 captures the time invariant characteristics of treatment group in

the treatment state. And finally, β_6 is the coefficient of interest and captures the variation in vaccine outcomes specific to the treatment groups relative to two control groups before and after the law (Gruber, 1994).

Table 2.12 presents the marginal effects of the DDD estimation. The results indicate that the treatment group is less likely to initiate or complete the vaccination compared to others when the outcome of interest is vaccination outcomes after the age of 13, it is consistent with the findings before that implied age is positively related with the vaccine decision outcomes. The point estimate of being in the treatment group on the update status before age of 13 is positive and significant. It is also consistent with the previous findings that increasing age will reduce the probability of being updated before the age of 13. Additionally, the treatment group is less likely to initiate and complete the vaccination compared to others after the law's enactment. The results derived from this regression do not allow me to draw a strong conclusion about the effect of school mandates. The point estimates are positive, but they are not statistically significant at the conventional level. It again might reflect the limited number of treatment units.

2.7. Conclusion

I provide the first national study of the effect of school mandates and provision of educational content for parents imposed by states on the HPV vaccine initiation and completion. HPV is responsible for 70% of cervical cancers and is considered a major public health issue. I focus on the effect of policies on the vaccine decision on extensive and intensive margins and over different age ranges. I also study the effect of physician recommendation with regard to the vaccine on the vaccine decisions. My results suggest

that the effect of policies on the vaccine decision is at best limited. Policies also failed to encourage the vaccination for girls younger than 13 years old indicating that the decision to initiate the vaccine is age sensitive.

Physician recommendation is found to be a strong predictor of the vaccine related decision, with the strongest effect observed on the decision to initiate the vaccine, providing parents with written and verbal reminders, and scheduling follow up visits at the time of initial vaccination could increase vaccine compliance (Neubrand et al., 2009). The same strategies can be employed to increase the HPV vaccine completion rate. The results also indicate that physician advice is substantially more successful in promoting the vaccine than both school mandates and educational programs for parents. With evidence that physician advice encourages the vaccination, a prevalence of advice becomes a matter of policy concern. Considering the fact that Tdap booster is recommended for the same age range as the HPV vaccine, and taking into account that parents are less sensitive about Tdap shots, there is an opportunity for physicians to recommend the HPV vaccine at the time of Tdap vaccine uptake.

The welfare implications and cost-benefit analysis of any of these policies need to be explored more. In addition to the direct effect of this vaccine on declining the rate of HPV infection, and potentially cervical cancer, there are also negative and positive externalities involved. If these policies can convince parents to take their children to a physician or a clinic in order to get the HPV vaccine, it also will increase the probability of receiving other vaccines, and as a result, increase the immunization rates for a range of disease. Countering the effectiveness of the vaccine, there is the potential that vaccinations may lead to increased risky behavior. Numerous academic studies on

vaccinations provide a behavioral framework in vaccination models of sexually transmitted diseases, and predict that an imperfect vaccination might result in an increase in sexual activity of high risk populations, and as a result, increase the spread of the disease (Kremer, 1996). Some newly presented results show that the HPV vaccine can increase the sexual activity in low income adolescents (Hill, 2013).

Policy makers also need to consider the parental sensitivity towards the vaccination age. Given the fact that parents are unwilling to begin the vaccination for the children aged less than 13, targeting this age group coupled with a liberal opt out provision will result in an ineffective policy. The cost of the vaccine is also an important determinant in the decision to accept the vaccine. The ACA is possibly a way to solve this issue.

The Pap smear screening test is shown to be very effective in reducing the cervical cancer incidence and mortality rates. If the vaccine declines the participation rate in this program due to the false risk perception, it can have a significant welfare implication. Therefore, it is critical to investigate the effect of the vaccine acceptance on participation in Pap smear screening programs among adults. This can be subject of future research.

2.9. References

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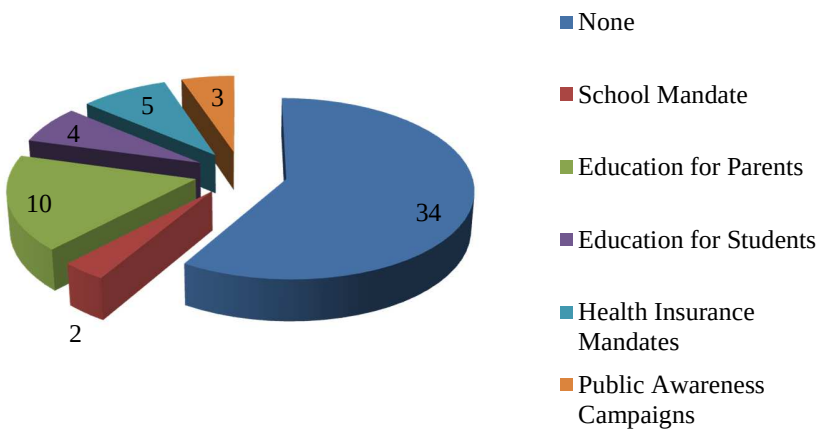


Figure 2.1. Number of states in each category of HPV vaccine related legislation

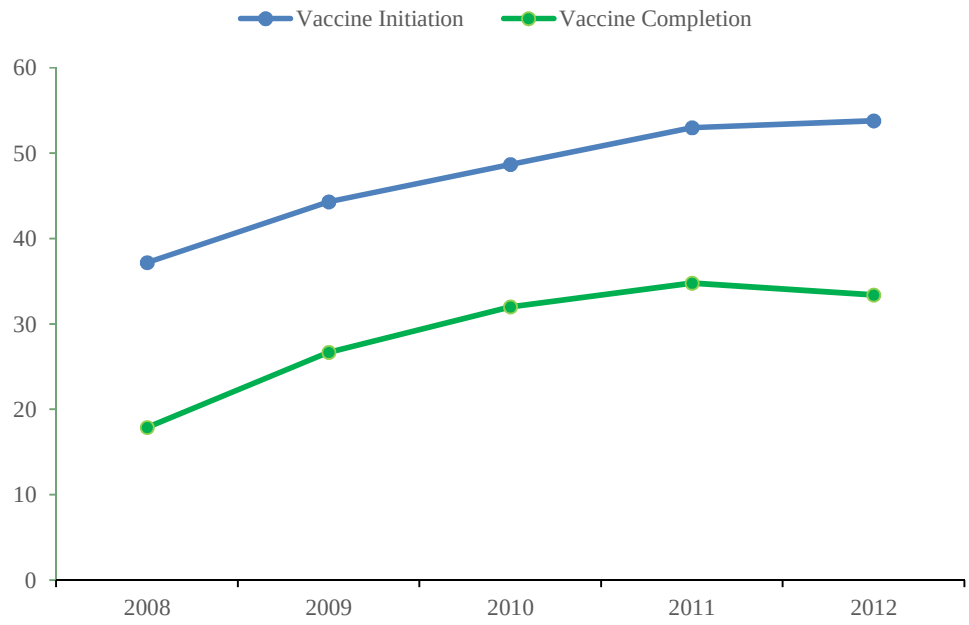
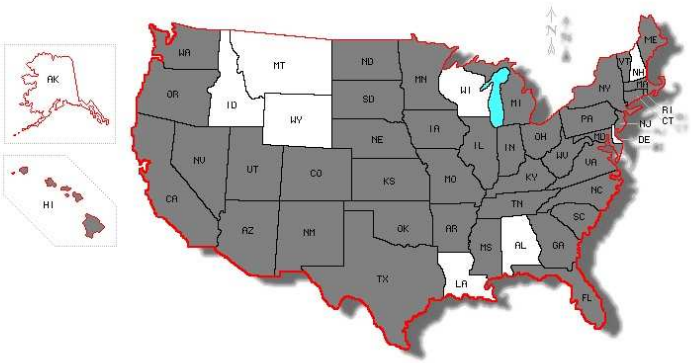


Figure 2.2. Share of 13-17 years old girls who initiated and completed the sequence of the HPV vaccine

Figure 2.3. An overview of the HPV related legislative activity at 2006

A. States that introduced HPV vaccine related legislation at 2006



B. States that introduced school mandates at 2006

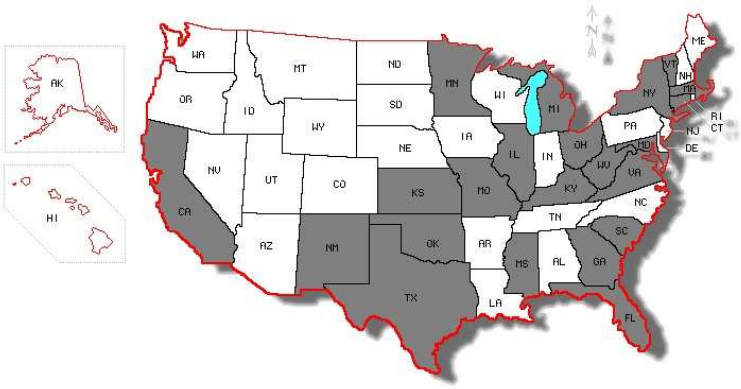


Table 2.1. HPV Related Legislation

State	Category	Covered Group	Explanation	Effective date
Colorado	Awareness Campaign	All		2008
	Health Insurance Mandate	All		2008
	Education for Students	Not Specified		2008
DC	School Mandate	11-12		2009
	Education for Parents	All		2009
Illinois	Education for Parents	11-12		2007
	Education for Students	11-12		2007
Indiana	Education for Parents	11-12		2008
Iowa	Education for Students	13-14		2007
	Health Insurance Mandate	All		2009
Kansas	Other	NA	Would urge FDA to be more cautious in approving new vaccines	2009
Louisiana	Education for Students	All		2008
Maine	Other	All	Consideration for future funding	
Maryland	Other	NA	Establish a task force to provide a recommendation for the state plan for vaccine	2007
Michigan	Education for Parents	All		2008
Minnesota	Other	NA	Study on different aspect of HPV vaccine	2007
Missouri	Education for Parents	All		2010
Nevada	Health Insurance Mandate	All		2007
New Jersey	Education for Parents	All		2007
New Mexico	Health Insurance Mandate	9-14		2007
New York	Other	All	Allocate 5,000,000 for services and expenses to promote and expand the access to cervical cancer vaccine.	Fiscal year 2007-2008
North Carolina	Education for Parents	All		2007
North Dakota	Awareness Campaign	All		2007
Oregon	Health Insurance Mandate	All		2010
South Dakota	Other	11-19	Cover the cost of the vaccine	Fiscal year 2007-2008
Texas	Education for Parents	All		2007
Utah	Awareness Campaign	All		2007
Virginia	School Mandate	11-12		2009
Washington	Education for Parents	11-12		2007

Table 2.2. Definition of Control Variables

Variable	Coding
Age	=Child's age
Number of children categories	
2 children	=1 if the number of children below age of 18 in the household is 2.
3 or more children	=1 if the number of children below age of 18 in the household is 3 or more.
Mother's age categories	
Mother below 34	=1 if mother's age is below 34
Mother below 44 and above 34	=1 if mother's age is below 44 and above 34
Race/Ethnicity	
Hispanic	=1 if the child is Hispanic
Black	=1 if the child is Black
Income	=1 if the household annual income is above 20,000$
Mother's marital status	=1 if mother is currently married
Mother's education	=1 if mother has 13 years of education or more
People in the household	=Number of people in the household
Health insurance status	
Private	=1 if person holds employer or union provided health insurance.
Public	=1 if person Medicaid, S-CHIP, or American-Indian health insurance.
Military	=1 if person holds TRICARE, CHAMPUS, or CHAMPUS-VA.
No Insurance Since 11	=1 if there is any period of the time after age 11 that teen did not have any health insurance coverage
No doctor visit last year	=1 if person has not visited a doctor in the past 12 months
Teen health status	=1 if teen has already lung condition rather than asthma, heart condition, diabetes, a kidney condition, sick cell anemia or other anemia, weakened immune system because of chronic illness or caused by medicine taken by chronic illness
Household health status	=1 if any other members of teen's household have lung condition rather than asthma, heart condition, diabetes, a kidney condition, sick cell anemia or other anemia, weakened immune system because of chronic illness or caused by medicine taken by chronic illness
Prevalence of Chlamydia	Number of diagnosed patients between 15-24 years old per every person in that age range.
Tdap update status	=1 if teen has had at least one shot of Tdap since age 10 years

Table 2.3. Effect of Physician Recommendation on Vaccine Outcomes-Probit Model

	Vaccine Initiation	Vaccine Completion	Update before Age 13
VARIABLES	(1)	(2)	(3)
Age	0.0853***	0.0850***	-0.868***
	(0.0119)	(0.0133)	(0.0757)
Public insurance	0.230***	0.148***	0.256***
	(0.0223)	(0.0230)	(0.0279)
Military insurance	0.140***	0.0213	0.0804*
	(0.0349)	(0.0364)	(0.0441)
No insurance since 11	-0.117***	-0.214***	-0.174***
	(0.0305)	(0.0331)	(0.0416)
No doctor visit last year	-0.250***	-0.145***	-0.0688**
	(0.0237)	(0.0251)	(0.0306)
2 children	0.0532***	0.0238	0.0224
	(0.0198)	(0.0204)	(0.0267)
3 or more children	0.0241	0.00530	0.00792
	(0.0414)	(0.0435)	(0.0534)
Mom's age below 34	0.0994***	-0.0567	0.102**
	(0.0346)	(0.0367)	(0.0402)
Mom below 44 and above 34	-0.0212	-0.0592***	0.0519**
	(0.0175)	(0.0181)	(0.0231)
Number of people in the household	-0.0267***	-0.0412***	-0.0291**
	(0.00910)	(0.00952)	(0.0122)
Income	-0.157***	-0.0732**	-0.0211
	(0.0298)	(0.0312)	(0.0370)
Hispanic	0.206***	0.0992***	0.189***
	(0.0288)	(0.0293)	(0.0347)
Black	0.00980	-0.163***	0.0160
	(0.0290)	(0.0312)	(0.0387)
Currently married	-0.0637***	-0.0336	-0.0299
	(0.0200)	(0.0210)	(0.0271)
College	-0.0587***	0.0257	-0.0385
	(0.0196)	(0.0204)	(0.0256)
Teen health status	-0.118***	-0.0644	-0.0318
	(0.0393)	(0.0412)	(0.0533)
Household health status	0.0641***	0.0313*	0.0641***
	(0.0163)	(0.0169)	(0.0214)
TDAP booster update	0.521***	0.408***	0.502***
	(0.0174)	(0.0183)	(0.0261)
School mandate	0.0993	0.119	0.0809
	(0.108)	(0.113)	(0.120)
Education for parents	0.0610	0.00674	-0.0607
	(0.0559)	(0.0586)	(0.0809)
Physician recommendation	0.847***	0.660***	0.572***
	(0.0162)	(0.0172)	(0.0225)
Constant	-1.842***	-2.143***	10.28***
	(0.229)	(0.250)	(1.030)
Observations	30,376	30,376	30,376

Note: Regressions include state and year fixed effects as well as the interactions of age and year. The numbers in parenthesis are robust standard errors. *** p<0.01, ** p<0.05, * p<0.1

Table 2.3. Effect of Physician Recommendation on Vaccine Outcomes-IV-Probit Model (Continued)

	Vaccine Initiation		Vaccine Completion		Update before Age 13	
	IV-Probit	Stage 1	IV-Probit	Stage 1	IV-Probit	Stage 1
VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)
Age	0.0854***	-0.0214	0.0860***	-0.0201	-0.846***	-0.0186
	(0.0118)	(0.0134)	(0.0133)	(0.0134)	(0.0748)	(0.0134)
Public insurance	0.233***	-0.0900***	0.151***	-0.0897***	0.260***	-0.0895***
	(0.0223)	(0.0234)	(0.0231)	(0.0234)	(0.0278)	(0.0234)
Military insurance	0.143***	-0.0753**	0.0241	-0.0766**	0.0850*	-0.0762**
	(0.0350)	(0.0367)	(0.0364)	(0.0367)	(0.0440)	(0.0366)
No insurance since 11	-0.113***	-0.0426	-0.210***	-0.0424	-0.165***	-0.0428
	(0.0306)	(0.0315)	(0.0332)	(0.0315)	(0.0414)	(0.0315)
No doctor visit	-0.234***	-0.257***	-0.131***	-0.259***	-0.0415	-0.256***
	(0.0241)	(0.0266)	(0.0254)	(0.0266)	(0.0308)	(0.0265)
2 children	0.0479**	0.0965***	0.0189	0.0963***	0.0127	0.0955***
	(0.0198)	(0.0207)	(0.0204)	(0.0207)	(0.0266)	(0.0207)
3 or more children	0.0223	0.0343	0.00379	0.0340	0.00460	0.0344
	(0.0413)	(0.0436)	(0.0435)	(0.0436)	(0.0531)	(0.0436)
Mom below 34	0.0970***	0.0551	-0.0594	0.0556	0.0962**	0.0518
	(0.0347)	(0.0377)	(0.0368)	(0.0377)	(0.0402)	(0.0377)
Mom below 44 and above 34	-0.0229	0.0207	-0.0606***	0.0204	0.0468**	0.0207
	(0.0175)	(0.0186)	(0.0181)	(0.0186)	(0.0230)	(0.0185)
Number of people in the household	-0.0255***	-0.0188**	-0.0400***	-0.0189**	-0.0268**	-0.0189**
	(0.00910)	(0.00953)	(0.00953)	(0.00954)	(0.0121)	(0.00953)
Income	-0.164***	0.150***	-0.0806**	0.151***	-0.0356	0.151***
	(0.0300)	(0.0307)	(0.0313)	(0.0307)	(0.0368)	(0.0307)
Hispanic	0.209***	-0.0849***	0.102***	-0.0844***	0.192***	-0.0851***
	(0.0289)	(0.0299)	(0.0294)	(0.0299)	(0.0345)	(0.0299)
Black	0.0188	-0.194***	-0.153***	-0.194***	0.0349	-0.193***
	(0.0291)	(0.0297)	(0.0313)	(0.0297)	(0.0386)	(0.0297)
Currently married	-0.0629***	-0.0112	-0.0329	-0.0118	-0.0287	-0.0101
	(0.0200)	(0.0210)	(0.0210)	(0.0210)	(0.0269)	(0.0210)
College	-0.0710***	0.223***	0.0136	0.223***	-0.0607**	0.224***
	(0.0198)	(0.0203)	(0.0207)	(0.0203)	(0.0257)	(0.0203)
Teen health status	-0.120***	0.0619	-0.0660	0.0619	-0.0308	0.0623
	(0.0392)	(0.0408)	(0.0411)	(0.0409)	(0.0527)	(0.0408)
Household health status	0.0627***	0.00333	0.0301*	0.00353	0.0604***	0.00321
	(0.0163)	(0.0174)	(0.0169)	(0.0174)	(0.0213)	(0.0174)
TDAP booster update	0.501***	0.315***	0.389***	0.317***	0.461***	0.314***
	(0.0182)	(0.0181)	(0.0191)	(0.0181)	(0.0267)	(0.0181)
School mandate	0.109	-0.158	0.130	-0.156	0.0987	-0.172
	(0.108)	(0.127)	(0.113)	(0.127)	(0.120)	(0.127)
Education for parents	0.0599	0.0743	0.00621	0.0742	-0.0596	0.0746
	(0.0558)	(0.0601)	(0.0585)	(0.0601)	(0.0803)	(0.0601)
Excluded IVs						
Asthma		0.0990***		0.0984***		0.0996***
		(0.0215)		(0.0215)		(0.0214)
Checkup 11-12		0.403***		0.404***		0.405***
		(0.0295)		(0.0296)		(0.0292)
F-Statistics		105.05***		104.75***		108.02***
ρ		-0.103***		-0.101***		-0.221***
		(0.0261)		(0.0270)		(0.0321)
Physician recommendation	0.985***		0.797***		0.832***	
	(0.0373)		(0.0394)		(0.0418)	
Observations	30,376	30,376	30,376	30,376	30,376	30,376

Note: Regressions include state and year fixed effects as well as the interactions of age and year. The numbers in parenthesis are robust standard errors. *** p<0.01, ** p<0.05, * p<0.1

Table 2.4. Marginal Effects of Physician Recommendation

	Vaccine Initiation		Vaccine Completion		Update before Age 13	
	Probit	IV-Probit	Probit	IV-Probit	Probit	IV-Probit
VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)
School mandate	0.0395	0.0436	0.0413	0.0451	0.00959	0.0124
	(0.0431)	(0.0432)	(0.0402)	(0.0405)	(0.0152)	(0.0163)
Education for Parents	0.0242	0.0238	0.00226	0.00208	-0.00656	-0.00672
	(0.0222)	(0.0222)	(0.0197)	(0.0197)	(0.00846)	(0.00877)
Physician	0.323***	0.371***	0.212***	0.253***	0.0603***	0.0904***
recommendation	(0.00576)	(0.0128)	(0.00517)	(0.0117)	(0.00377)	(0.00694)
Observations	30,376	30,376	30,376	30,376	30,376	30,376

Note: Regressions include state and year fixed effects as well as the interactions of age and year. The numbers in parenthesis are robust standard errors. *** p<0.01, ** p<0.05, * p<0.1

Table 2.5. Linear Probability Model-OLS

	Vaccine Initiation	Vaccine Completion	Update before Age 13
VARIABLES	(1)	(2)	(3)
Age	0.0273***	0.0217***	-0.0421***
	(0.00378)	(0.00331)	(0.00242)
Public insurance	0.0757***	0.0413***	0.0497***
	(0.00747)	(0.00708)	(0.00564)
Military insurance	0.0463***	0.00332	0.0167*
	(0.0118)	(0.0113)	(0.00890)
No insurance since 11	-0.0364***	-0.0594***	-0.0291***
	(0.00993)	(0.00906)	(0.00699)
No doctor visit last year	-0.0786***	-0.0370***	-0.00959*
	(0.00747)	(0.00687)	(0.00564)
2 children	0.0175***	0.00675	0.00423
	(0.00661)	(0.00636)	(0.00478)
3 children or more	0.00852	0.00314	-0.000980
	(0.0137)	(0.0129)	(0.0104)
Mom's age below 34	0.0335***	-0.0155	0.0275***
	(0.0115)	(0.0107)	(0.00982)
Mom's age below 44 and above 34	-0.00715	-0.0181***	0.00927**
	(0.00587)	(0.00555)	(0.00435)
Number of people in the household	-0.00875***	-0.0123***	-0.00500**
	(0.00303)	(0.00287)	(0.00219)
Income	-0.0527***	-0.0220**	-0.00313
	(0.00995)	(0.00956)	(0.00775)
Hispanic	0.0695***	0.0299***	0.0461***
	(0.00972)	(0.00942)	(0.00762)
Black	0.00349	-0.0458***	0.00453
	(0.00955)	(0.00884)	(0.00709)
Currently married	-0.0215***	-0.00961	-0.00850*
	(0.00666)	(0.00635)	(0.00494)
College	-0.0198***	0.00795	-0.00788*
	(0.00646)	(0.00608)	(0.00473)
Teen health status	-0.0396***	-0.0208*	-0.00836
	(0.0132)	(0.0126)	(0.00958)
Household health status	0.0211***	0.00876*	0.0125***
	(0.00547)	(0.00524)	(0.00407)
TDAP booster update	0.176***	0.119***	0.0808***
	(0.00586)	(0.00544)	(0.00383)
School mandate	0.0346	0.0349	0.0268
	(0.0376)	(0.0364)	(0.0355)
Education for parents	0.0213	-0.000574	-0.00813
	(0.0186)	(0.0174)	(0.0143)
Physician recommendation	0.298***	0.198***	0.103***
	(0.00547)	(0.00504)	(0.00391)
Observations	30,376	30,376	30,376

Note: Regressions include state and year fixed effects as well as the interactions of age and year. The numbers in parenthesis are robust standard errors. *** p<0.01, ** p<0.05, * p<0.1

Table 2.5. Linear Probability Model-2SLS-(Continued)

VARIABLES	Vaccine Initiation (1)	Vaccine Completion (2)	Update before Age 13 (3)	Stage 1 (4)
Age	0.0180***	0.0223***	-0.0316***	-0.00709
	(0.00501)	(0.00471)	(0.00310)	(0.00494)
Public insurance	0.0878***	0.0554***	0.0539***	-0.0324***
	(0.00953)	(0.00923)	(0.00661)	(0.00872)
Military insurance	0.0552***	0.00645	0.0167	-0.0279**
	(0.0148)	(0.0143)	(0.0102)	(0.0137)
No insurance since 11	-0.0325***	-0.0573***	-0.0246***	-0.0156
	(0.0126)	(0.0120)	(0.00837)	(0.0118)
No doctor visit last year	-0.0238*	0.0123	0.0311***	-0.0967***
	(0.0131)	(0.0122)	(0.00864)	(0.00996)
2 children	0.00525	-0.00708	-0.00682	0.0347***
	(0.00836)	(0.00819)	(0.00569)	(0.00750)
3 children or more	0.00447	-0.00349	-0.00574	0.0115
	(0.0169)	(0.0164)	(0.0120)	(0.0161)
Mom's age below 34	0.0125	-0.0322**	0.0107	0.0208
	(0.0148)	(0.0143)	(0.0115)	(0.0140)
Mom's age below 44 and above 34	-0.0126*	-0.0241***	0.00579	0.00744
	(0.00722)	(0.00697)	(0.00499)	(0.00673)
Number of people in the household	-0.00563	-0.00988***	-0.00207	-0.00680*
	(0.00373)	(0.00367)	(0.00255)	(0.00349)
Income	-0.0708***	-0.0384***	-0.00990	0.0569***
	(0.0130)	(0.0127)	(0.00917)	(0.0116)
Hispanic	0.0789***	0.0408***	0.0467***	-0.0305***
	(0.0120)	(0.0118)	(0.00870)	(0.0111)
Black	0.0342***	-0.0159	0.0279***	-0.0724***
	(0.0125)	(0.0121)	(0.00858)	(0.0111)
Currently married	-0.0168**	-0.00664	-0.00400	-0.00494
	(0.00811)	(0.00795)	(0.00558)	(0.00774)
College	-0.0603***	-0.0308***	-0.0300***	0.0836***
	(0.0102)	(0.00971)	(0.00678)	(0.00763)
Teen health status	-0.0578***	-0.0389**	-0.0131	0.0229
	(0.0157)	(0.0152)	(0.0107)	(0.0148)
Household health status	0.0191***	0.00653	0.00966**	0.00143
	(0.00666)	(0.00653)	(0.00463)	(0.00631)
TDAP booster update	0.114***	0.0662***	0.0300***	0.118***
	(0.0114)	(0.0107)	(0.00708)	(0.00673)
School mandate	0.0336	0.0474	-0.0150	-0.0578
	(0.0490)	(0.0485)	(0.0436)	(0.0489)
Education for parents	0.0156	-0.00490	-0.0115	0.0274
	(0.0225)	(0.0216)	(0.0168)	(0.0221)
Physician recommendation	0.736***	0.651***	0.360***	
	(0.0718)	(0.0668)	(0.0456)	
Excluded IVs				
Asthma				0.0350***
				(0.00765)
Checkup 11-12				0.149***
				(0.0111)
F-Statistics	103.23***	103.23***	103.23***	
Hansen J statistics	0.6613	0.7537	0.4840	
Observations	25,413	25,413	25,413	25,413

Note: Regressions include state and year fixed effects as well as the interactions of age and year. The numbers in parenthesis are robust standard errors. *** p<0.01, ** p<0.05, * p<0.1

Table 2.6. Robustness Checks for Instrumental Variables

	Vaccine Initiation		Vaccine Completion		Update before Age 13	
	IV-Probit	Stage 1	IV-Probit	Stage1	IV-Probit	Stage 1
VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)
Panel A- Using History of Asthma as Only Instrument						
School mandate	0.137	-0.156	0.141	-0.156	0.104	-0.156
	(0.106)	(0.106)	(0.113)	(0.106)	(0.120)	(0.106)
Education for parents	0.0532	0.0123	0.00430	0.0136	-0.0616	0.0158
	(0.0541)	(0.0535)	(0.0578)	(0.0537)	(0.0797)	(0.0538)
Physician	1.525***		1.032***		0.968***	
recommendation	(0.158)		(0.179)		(0.194)	
Excluded IV						
Asthma		0.116***		0.112***		0.112***
		(0.0191)		(0.0200)		(0.0202)
ρ		-0.489***		-0.241*		-0.256*
		(0.148)		(0.124)		(0.135)
F-Statistics		36.59***		31.19***		30.59***
Constant	-2.004***	-0.327		-0.340	9.918***	-0.341
	(0.222)	(0.217)		(0.218)	(1.013)	(0.218)
Observations	30,376	30,376	30,376	30,376	30,376	30,376
Panel B- Using History of Asthma and Missing School as Instrumental Variables						
School mandate	0.0993	-0.152	0.126	-0.153	0.0871	-0.152
	(0.109)	(0.106)	(0.113)	(0.106)	(0.121)	(0.106)
Education for parents	0.0610	0.0155	0.00604	0.0149	-0.0610	0.0155
	(0.0559)	(0.0539)	(0.0585)	(0.0539)	(0.0808)	(0.0539)
Physician	0.847***		0.769***		0.674***	
recommendation	(0.225)		(0.159)		(0.153)	
Excluded IVs						
Asthma		0.110***		0.112***		0.111***
		(0.0217)		(0.0205)		(0.0204)
Missing school more		-0.113*		-0.117*		-0.115*
than 30 days		(0.0652)		(0.0656)		(0.0652)
ρ		-1.47e-05		-0.0683		-0.0634
		(0.138)		(0.0997)		(0.0954)
F-Statistics		13.76***		15.82***		15.90***
Constant	-1.842***	-0.361*	-2.182***	-0.357	10.22***	-0.358
	(0.244)	(0.219)	(0.255)	(0.219)	(1.030)	(0.219)
Observations	30,376	30,376	30,376	30,376	30,376	30,376
Panel C- Marginal Effects						
	Asthma	Missing School	Asthma	Missing School	Asthma	Missing School
School Mandate	0.0546	0.0395	0.0492	0.0436	0.0132	0.0104
	(0.0422)	(0.0435)	(0.0409)	(0.0405)	(0.0165)	(0.0155)
Education for Parents	0.0212	0.0242	0.00145	0.00203	-0.00699	-0.00661
	(0.0215)	(0.0223)	(0.0195)	(0.0197)	(0.00876)	(0.00847)
Physician	0.542***	0.323***	0.322***	0.245***	0.106***	0.0709***
Recommendation	(0.0457)	(0.0799)	(0.0521)	(0.0474)	(0.0259)	(0.0165)

Note: Regressions include state and year fixed effects as well as the interactions of age and year. The numbers in parenthesis are robust standard errors. *** p<0.01, ** p<0.05, * p<0.1

Table 2.7. Robustness Checks for Multi-colinearity and Statistical Power

	Vaccine Initiation	Vaccine Completion	Update Before Age 13
VARIABLES	(1)	(2)	(3)
Panel A-Robustness Check for Multi-colinearity			
School mandates	0.0186	0.0272	0.00959
	(0.0418)	(0.0390)	(0.0152)
Education for parents	0.0240	0.00468	-0.00656
	(0.0217)	(0.0198)	(0.00846)
Observations	30,557	30,557	30,376
Panel B- Consider School Mandates as Educational Program			
Education for Parents	0.0232	0.00791	-0.00439
	(0.0207)	(0.0190)	(0.00867)
Observations	39,557	30,557	30,557

Note: Regressions include state and year fixed effects as well as the interactions of age and year. The numbers in parenthesis are robust standard errors. *** p<0.01, ** p<0.05, * p<0.1

Table 2.8. Regression with Control Group of States that Introduced School Mandates

	Vaccine Initiation	Vaccine Completion	Update before Age 13
VARIABLES	(1)	(2)	(3)
School mandate	0.0598	0.0412	0.00712
	(0.0439)	(0.0412)	(0.0176)
Education for parents	0.0873***	0.0376	-0.00736
	(0.0322)	(0.0303)	(0.0143)
Observations	15,685	15,685	15,685

Note: Regressions include state and year fixed effects as well as the interactions of age and year. The numbers in parenthesis are robust standard errors. *** p<0.01, ** p<0.05, * p<0.1

Table 2.9. Vaccination Rate Predictor Means

Variable	Virginia	Synthetic Virginia
Median income	66101.65	66177.69
Unemployment rate	4	4.2619
Percentage of urban population	0.714353	0.8202806
Population density	183.1326	289.125
Percentage of Hispanic	0.0734881	0.0820556
Percentage of Blacks	0.197647	0.1945446
Prevalence of Chlamydia	2078.9	2004.011
Percentage of female population above age 25 with a bachelor's degree or higher	0.329	0.31486
Percentage of population under age 18 with public health insurance plans	0.1928294	0.2089898
Percentage of married households	0.512861	0.5132828

Table 2.10. State Weights in Synthetic Virginia

State	Weight	State	Weight
Alabama	0	Montana	0
Alaska	0.07	Nebraska	0
Arizona	0	New Hampshire	0
Arkansas	0	New Jersey	0
California	0	New York	0
Connecticut	0	North Carolina	0
Delaware	0	North Dakota	0.048
Florida	0	Ohio	0
Georgia	0	Oklahoma	0
Hawaii	0.015	Pennsylvania	0
Idaho	0	Rhode Island	0
Indiana	0	South Carolina	0.035
Kansas	0	South Dakota	0
Kentucky	0	Tennessee	0
Maine	0	Texas	0
Maryland	0.599	Utah	0.234
Massachusetts	0	Vermont	0
Michigan	0	Washington	0
Minnesota	0	West Virginia	0
Mississippi	0	Wisconsin	0
Missouri	0	Wyoming	0

Table 2.11. Synthetic Control Method

Panel A- Probit			
	Vaccine Initiation	Vaccine Completion	Update Before Age 13
VARIABLES	(1)	(2)	(3)
School mandate	0.0207	0.0202	-0.0117
	(0.0667)	(0.0609)	(0.0134)
Observations	4,092	4,092	3,919
Panel B- Linear Probability Model			
School mandate	0.0218	0.0230	-0.0265
	(0.0741)	(0.0683)	(0.0508)
Constant	0.807	0.494	1.156**
	(0.739)	(0.681)	(0.507)
Observations	1,256	1,256	1,256
R-squared	0.088	0.069	0.128

Note: Regressions include state and year fixed effects as well as the interactions of age and year. The numbers in parenthesis are standard errors. *** p<0.01, ** p<0.05, * p<0.1

Table 2.12. Difference-in-Difference-in-Difference

	Vaccine Initiation	Vaccine Completion	Update Before Age 13
VARIABLES	(1)	(2)	(3)
Schman*treat*after	0.0939	0.0514	0.0138
	(0.0694)	(0.0671)	(0.0392)
Treatment*after	-0.0543	-0.0678	-0.0366
	(0.0513)	(0.0423)	(0.0276)
Schman*after	-0.0715***	-0.0654***	-0.0801***
	(0.0130)	(0.0112)	(0.00417)
Schman*treatment	-0.0973**	-0.0166	-0.0362
	(0.0478)	(0.0443)	(0.0223)
Schman	-0.0976***	-0.0850***	0.231***
	(0.00903)	(0.00818)	(0.00690)
Observations	23,219	23,219	23,219

Note: Regressions include state and year fixed effects. The numbers in parenthesis are robust standard errors. *** p<0.01, ** p<0.05, * p<0.1

Chapter 3: Immunization and Moral Hazard: The Effect of Human Papillomavirus Vaccination on Participation in Routine Pap Test

3.1. Introduction

Cervical cancer is the second most common cancer related cause of death among women globally. Virtually all cervical cancers are causally related to infection by Human Papillomavirus (HPV). Approximately 70% of cervical cancers are caused by HPV types 16 and 18 (Saslow et al., 2007). Cervical cancer is the easiest gynecological cancer to prevent, and it only requires regular screening tests and follow-ups. There are two tests for diagnosing cervical cancer, the Pap test (or Pap smear), which looks for cell change in the cervix that might ultimately become cervical cancer if it is not treated appropriately, and the HPV test, which looks for the virus (HPV) that can cause these cell changes. The Pap test is recommended for all women between ages 21 and 65 years old, and the HPV test is recommended for women older than 30 years old along with the Pap test (Center for Disease Control and Prevention, 2014).

The Pap test is one of the most effective and successful cancer screenings in history. Most women diagnosed with cervical cancer have either never had a Pap test, or have not had it in the past 5 years (Center for Disease Control and Prevention, 2012). Participation in regular Pap tests has decreased the incidence and mortality rates of cervical cancer in the past 40 years causing cervical cancer to not be the leading cause of cancer death for women in the United States anymore (Center for Disease Control and Prevention, 2013). U.S. cervical cancer incidence rates decreased by 75% and mortality by 74% in the 50 years following the introduction of cervical cytology in 1949 (Howe et al., 2007). The most successful strategy in cervical cancer prevention is population-based

Pap-smear screening programs. The introduction of screening programs in unscreened populations can result in 60-90 % reduction in cervical cancer rates within 3 years after implementation (Saslow et al., 2002).

Recently, some progress has been made in preventive strategies for cervical cancers. Two vaccines were developed that can provide immunization against certain types of HPV. The bivalent vaccine (Cervarix) and quadrivalent vaccine (Gardasil) can protect against HPV types 16 and 18. Gardasil also protects against HPV types 6 and 11, which cause 90% of genital warts. The Food and Drug Administration (FDA) approved Gardasil in 2006 and Cervarix in 2009. The Advisory Committee on Immunization Practices (ACIP) recommends either HPV vaccine for routine immunization for girls 11 to 12 years old and catch-up vaccination for adolescents and adults 13 to 26 years old who have not been vaccinated previously. ACIP also recommends the quadrivalent vaccine for 11 to 12 years old boys (Center for Disease Control and Prevention, 2010). ACIP recommended the vaccine in 2006, but the recommendation was published in March 2007 (Robin et al., 2014). Despite impressive efficiency records of the vaccines, regular Pap tests are recommended for women who have been sexually active even after the HPV vaccine. First of all, vaccination will not protect against all HPV types not included in the first generation of the vaccines. About 30% of cervical cancers will not be prevented by the HPV vaccines. Moreover, women who got the vaccine after becoming sexually active might not get the full benefit of the vaccine if they already had been exposed to HPV. These factors, along with the fact that long term effects of the vaccine are unknown at the time being, will promote using both prevention strategies as complements (Franco et al., 2006).

In this study, I look at the behavioral response to the HPV vaccine. Medical innovations, can result in moral hazard by reducing the cost of unhealthy behaviors. For example, obesity has increased as a result of great improvement in heart disease treatments (Peltzman 2011). Low cost medical treatment for diabetes can increase the body mass index (Klick & Stratman, 2007). HPV vaccination can also cause moral hazard in low income adolescents as they are more likely to get involved in risky sexual behaviors in response to taking the HPV vaccine compared to those who have not initiated the vaccine (Hall, 2014).

I specifically look into the effect of vaccination on the decision to participate in Pap tests. The empirical evidence presented by Ferris et al. (2012) shows that women are more receptive to getting the HPV vaccine in exchange for longer Pap test intervals, and Pap test non-compliers are more likely to get the HPV vaccine if Pap test was required less frequently. If vaccination results in reduction in participation in Pap tests, this could potentially increase the prevalence of cervical cancer. This should be of interest to public health policy makers. The decision to initiate the vaccine and the decision to participate in Pap tests are being determined simultaneously. There might be some unobserved factors that derive both decisions which raise endogeneity issues and will likely lead to biased estimates. To deal with this issue, I use the fact that the HPV vaccination is recommended for women younger than age 26 years old, and the probability of vaccination should change significantly at this age. I use this cut-off point at recommended age to construct a fuzzy regression discontinuity to identify more clearly the effect of the HPV vaccine on testing.

The results indicate that vaccine initiation is positively associated with short- and long- term probability of participation in Pap tests even after controlling for potential endogeneity. This might be the result of increased awareness that people acquire at the time of vaccination. The results support the argument that the vaccine initiation indicator should be treated as an endogenous regressor. The estimates that ignore the potential endogeneity will result in under-estimating the true effect of the vaccine initiation on the decision to participate in Pap tests.

The rest of the paper is organized as follows: in the second section, I describe the data and methodology, the third section presents the results, fourth section analyses the sensitivity of the results to different specifications, and the fifth section concludes.

3.2. Data and Methodology

I use the National Health Interview Survey (NHIS) for this study. The NHIS is a cross-sectional household interview designed to collect information for monitoring the health of the United States population. The core of the database contains four major components: Household, Family, Sample Adult, and Sample Child. The Household component collects limited demographic information on all the individuals living in a particular house. The Family component verifies and collects additional demographic information on each member from each family in the household and also collects information on health status, illness and injuries, and access to healthcare and utilization. From each family in the NHIS, one sample adult and one sample child are randomly selected and detailed health related information on each is collected. The Sample Adult component of the NHIS contains information on Pap test screening history and HPV vaccination. I use the Sample Adult database and use other components to match

socioeconomic information for each individual in the sample to construct my database. The final sample consists of information from years 2008-2012, except for the year 2009, in which information about vaccination and Pap tests is missing.

To estimate the effect of HPV vaccination on the decision to participate in Pap tests, I begin by estimating:

$$Y_{ijt} = f(\alpha + \beta_1 X_{ijt} + \gamma_j + \delta_t + \beta_2(hpvinit_{ijt}) + \varepsilon_{ijt}) \qquad (1)$$

I estimate this equation using a probit model. Y_{ijt} either represents whether a person had a Pap test in the past 12 months (hereafter referred to as a short-run Pap test) or whether a person has ever had a Pap test (hereafter referred to as long-run Pap test). The variable hpvinit is a dummy variable which is equal to one when a respondent has initiated the sequence of HPV vaccination (dose≥1) in the past and zero otherwise. γ_j is a vector of region dummy variable that captures time-invariant factors that cause the outcomes to be different between regions[17]. δ_t is a vector of year dummies that captures the variation in outcomes in different years that are common among regions. X_{ijt} is a vector of control covariates. It includes control for age, race, marital status, employment status, health insurance coverage status, citizenship status, whether a person has visited a gynecologist in the past year, and whether a person has ever taken an HIV test. Table 3.1 presents the definition and coding of each variable.

[17] It includes dummies for West, Midwest, Northeast, and South census regions.

It is important to point out that the decision to initiate the vaccine might be correlated with some unobserved factors that can influence the decision to participate in Pap tests simultaneously. Failing to control for the interdependence between these two decisions will lead to biased and inconsistent estimates. The direction of the bias is unclear. For example, people placing a higher value on health might seek both preventive options and treat them as complements. On the other hand, it is possible that people with limited access to regular healthcare might find the vaccination a more convenient method of protection that involves less attention and follow ups.

A plausible strategy to identify exogenous variation in vaccine initiation is to exploit the knowledge of the rules determining the treatment (HPV vaccine initiation in this case). There is an opportunity to construct a regression discontinuity design when there is a known cut-off point in treatment assignment or the probability of treatment receipt as a function of one or more continuous assignment variables. In principle, regression discontinuity compares the average outcome for units just left and right of the discontinuity point within a very small interval around the cut-off point. Increasing the interval around the cut-off point might result in biased estimates of the treatment, specifically when the assignment variable is related to the outcome conditional on treatment assignment (van der Klaauw, 2008). CDC recommends the vaccine for women younger than 26 years old (Center for Disease control and Prevention, 2014). Therefore, one might expect the probability of vaccination to be discontinuous at this cut-off point. I use this cut-off point to construct a fuzzy regression discontinuity (RD) around this point. In fuzzy design, treatment assignment depends on x in a stochastic manner, but one in which the propensity of treatment ($Pr(T=1|x)$) is again known to have a discontinuity at

$\overline{x}$ (van der Klaauw, 2008). I use the discontinuity as an instrumental variable for treatment status. This new estimate can be summarized by:

$$\begin{aligned} Y_{ijt} &= 1(\alpha + \beta_1 X_{ijt} + \gamma_j + \delta_t + \beta_2 (hpvinit_{ijt}) + \varepsilon_{ijt} \geq 0) \\ hpvinit_{ijt} &= 1(\alpha + \varphi_1 X_{ijt} + \gamma_j + \delta_t + \varphi_2 (\mathrm{T}_{it}) + u_{ijt} \geq 0) \end{aligned} \qquad (2)$$

$$\begin{pmatrix} \varepsilon \\ u \end{pmatrix} | \, Z \sim N\left(\begin{pmatrix} 0 \\ 0 \end{pmatrix}, \begin{pmatrix} 1 & \rho \\ \rho & 1 \end{pmatrix} \right)$$

T is a dummy variable which is equal to one when the individual is in the recommended age range at any given year. I adjust the cut-off points in each year by considering the fact that people might not be in the recommended age group in that year, but they have been within that age range sometime in the past. For example, a 27 year old woman is not in the recommended age group in 2008, however, she was 26 years old in 2007, and therefore, she was in the recommended age group in that year. As a result, I consider the age of 27 as a cut-off point in 2008, and I adjust the cut-off points in the years after accordingly. Figure 3.1 shows the discontinuity in treatment at different cut-off points. Z is the vector of instrumental and exogenous variable. ρ captures the correlation between disturbances in these two equations, and it indicates endogeneity when it is different from zero. I restrict the sample to an interval of 4 years before and after the cut-off point in order to have a small interval around the cut-off point with a sufficiently large number of observations. I will eventually narrow down the interval around the cut-off point for robustness checks.

3.3. Results

Table 3.2 presents the results from the baseline regression (equation 1) as well as RD design. The first column in each set of regressions presents the results from the probit model without taking potential endogeneity into account. The next two columns present the results from regression discontinuity framework along with the results from the first stage. Table 3.3 presents the marginal effects of the HPV vaccine initiation on the Pap test participation decision in different specifications.

Most variables have the expected signs and the trends are very similar for both dependent variables. However, estimated coefficients are more precisely estimated for the short-run Pap test. It is worth mentioning that information about the long-run Pap test is not available after 2010. Fewer numbers of observations can obviously cause a loss in efficiency. This issue becomes even more pronounced for estimating the first stage in RD design when the outcome of interest is the long-run Pap test due to the fact that there is not much variation in HPV vaccine initiation over this time period perhaps reflecting the short span of time after approval of the vaccine.

The HPV vaccine initiation increases the probability of having a Pap test in the short- and long- run. I find a negative bias in those estimates that ignore the endogeneity. Initiating the vaccine can increase the probability of having a Pap test by 18% in the short-run and by 5% in the long-run. This positive association might be derived from an increased awareness of the existence of the test happening during the vaccination time. Note that ρ captures the potential correlation between unobservables that determines vaccine initiation and having a Pap test simultaneously. ρ is negative and significant in all the specifications that account for endogeneity. This is consistent with finding negative

bias in those specifications that ignore endogeneity. It also supports my contention that vaccine initiation should be considered as a potentially endogenous regressor.

The instrumental variable also has the expected sign indicating that being in the recommended age group will increase the probability of vaccine initiation, however, it is not statistically significant at the conventional level when the outcome of interest is the long-run Pap test. Again, there is not much variation in HPV vaccine initiation over the time period in which the data for the long-run Pap test exists. However, the point estimate for the instrumental variable remains positive in this specification. I also report the F-statistics for the excluded IV from the first stage in order to provide a measure to test for weak identification. The estimated F-statistics cannot rule out the possibility of weak identification. Consistent with the previous findings, the F-statistics is much weaker for the long-run Pap test. Poor F-statistics from the first stage mainly resulted from two reasons. First, as previously mentioned, the potential identifying variation in the HPV vaccine initiation is limited to the first two years in the data. This affects the precision of estimated coefficients, as well as the F-statistics. Second, the RD design requires working within a small interval around the cut-off point. This obviously comes with the cost of a loss in efficiency and a decrease in the F-statistic.

The results in Table 3.2 also indicate that respondent's age is positively associated with the probability of having a Pap test and is negatively associated with the probability of initiating the HPV vaccine. The HPV vaccination is recommended only for women younger than 26 years old, whereas the Pap test is recommended until age of 65 years old, and the need for the Pap test is expected to increase with age. Visiting a gynecologist in the past 12 months is a strong predictor of having a Pap test in the short-run and long-

run. Such a strong relationship can be derived from reverse causality that people who intend to have a Pap test will visit a gynecologist. However, inclusion of this variable, along with other variables like health insurance status and income, can control for access to healthcare. An HIV test is also positively correlated with both having a Pap test and initiating the HPV vaccine in all the specifications. If an individual needs to be tested for a sexually transmitted disease, it is more likely for that person to take precautionary measures for other STDs. The estimated coefficients for pregnancy status are not statistically significant on the short-run Pap test, however, the point estimates are positive for having a Pap test and is negative for vaccine initiation. It is worth mentioning that the HPV vaccine is not recommended for pregnant women while the Pap test is recommended for them. The same pattern does not hold for the long-run Pap test. This might result from the fact that pregnant women are relatively younger and less likely to have a Pap test in the past.

A reasonable alternative for the instrumental variable probit model is 2SLS. However, I suspect that the conditional expectation function associated with the first stage is non-linear. My approach is to use the fitted value of the first stage as the instrumental variable (Wooldridge, 2011). The results are firmly consistent with the findings from the IV-Probit model. Vaccine initiation is still positively influencing the decision to have a Pap test. The F-statistics for the excluded IV is also larger than that from the IV probit model. However, this is an expected change considering the fact that the excluded IV is the fitted value from the original first stage estimation.[18]

[18] If I use the discontinuity function (t) instead of fitted value as an instrumental variable, the estimated coefficients for HPV vaccine initiation becomes negative, small, and statistically insignificant. These results will be available upon request.

Altogether, I cannot conclude from the results that moral hazard exists. Although I have a limited numbers of observations, the point estimates for the instrumental variable have the expected sign. While the share of people who have initiated the vaccine is steadily growing over time, one might expect that inclusion of observations from the most recent years, which are going to be released in the future, can improve the F-test[19].

It is important to point out that regression discontinuity results at best can be interpreted as average treatment effect for a sub-population near the cut-off point. Fuzzy regression discontinuity restricts the sub-population even further to that of compliers at this value of the covariate. Generalizing the results as population average treatment effect requires strong assumptions justifying extrapolation to other sub-populations (Imbens and Wooldridge, 2007).

3.4. Sensitivity Analysis

It is important to investigate how sensitive the parametric estimates are to alternative and more flexible specifications. First, I add different combinations of polynomial orders to the Pap test and the HPV vaccine initiation equations (van der Klaauw, 2008). Panel A of Table 3.5 presents the results of these new estimates. I restrict the outcome only to the short-run Pap test for which I have a sufficiently large number of observations to efficiently estimate the coefficients. I report the estimates of the HPV vaccine initiation from the IV-probit framework, as well as the coefficients of the instrumental variable, and the correlation parameter ρ. The results are firmly consistent

[19] Restricting the sample to only years 2011 and 2012 that relatively higher share of people initiated the vaccine in them, and increasing the interval around the cut-off point increases the F-test. These results will be available upon request.

with the previous findings indicating that the HPV vaccine initiation will increase the probability of the short-run Pap test. Additionally, neglecting the potential endogeneity will cause negative bias in the estimated effect of the HPV vaccine initiation on the short-run Pap test.

I also use the interaction term of age and discontinuity function (T) as a new instrumental variable for the treatment status (Angrist & Pischke, 2009). Moreover, I increasingly narrow the window around the cut-off point. A smaller interval around the cut-off point will decrease the risk of misspecification bias, but it obviously comes with a loss in efficiency. Panel B of Table 3.5 presents the results from these two class of robustness checks. Adding an interaction term does not change the results. Vaccine initiation remains a positive predictor of the short-run Pap test. Taking increasingly narrower windows around the cut-off point does not change the sign of the estimated coefficient of the HPV vaccine initiation. The estimated coefficients remain positive in all the specifications. However, the coefficients are not statistically significant for four and two year intervals around the cut-off point. This is perhaps reflecting fewer numbers of observations in smaller intervals. Correlation coefficients are negative in six and four year's intervals but become insignificant when the interval around the cut-off point is four years. The coefficient becomes positive and statistically insignificant when the interval is restricted to two years.[20] The results from the sensitivity analysis, consistent with the previous findings, do not provide any evidence of moral hazard. Although some estimates of the HPV vaccine initiation is not statistically significant, the point estimates remain positive even in more conservative specifications.

[20] The results are qualitatively the same after inclusion of higher order age polynomials, these results will be available upon request.

3.5. Conclusion

In this study, I look at the behavioral response to HPV vaccination. Vaccination can influence the risk perception of those who took the vaccine and cause moral hazard. I specifically look at the effect of HPV vaccination on participation in Pap test. Although HPV vaccination can protect against certain types of HPV and prevent cervical cancer, the protection is not complete and the vaccination should not be considered as a perfect substitute for Pap test.

I use the cut-off point in the recommended age for the vaccination to construct a fuzzy regression discontinuity. The results provide no evidence of moral hazard. HPV vaccination is found to be positively associated with the short-run Pap test, however, estimated coefficients are not statistically significant in some of the specifications. This is mainly derived from lack of variation in HPV vaccine initiation indicator, and as a result, lack of statistical power. The results are not conclusive about the effect of HPV vaccination on long-run Pap test. This likely is due to the fact that the data waves in which information about long-run Pap test exists is limited to two years, and share of people who initiated the vaccine over this time is very limited.

This study's limitations with regard to statistical power should be overcome with additional years of data. Regression discontinuity design requires limiting the sample to a small interval around the cut-off point that reinforces this problem. Adding more data points, potentially from future waves of NHIS can resolve this problem as well considering the fact that greater shares of the public are initiating the vaccine each year (Center for Disease Control and Prevention, 2012 & 2014).

Regression discontinuity designs also come with additional unique disadvantages. As previously mentioned, the results from regression discontinuity designs can be

interpreted as average treatment effect for a sub-population. In addition to the aforementioned theoretical shortcoming, the database I use provides little variation in the endogenous regressor. Lack of variation becomes even more pronounced considering the fact that RD designs require restricting the sample to a small interval around the cut-off point. One avenue for future research is to pursue alternative methods that do not suffer from the limitations presented by the regression discontinuity approach. One possibility is a modified control function approach, which exploits the dependence of the error on the exogenous variables (heteroscedasticity) to adjust the conventional control function approach (Klein & Vella, 2010; Farre, et al., 2008). This might be the next step in this study to estimate the effect of HPV vaccine initiation on the short-run and long-run Pap test.

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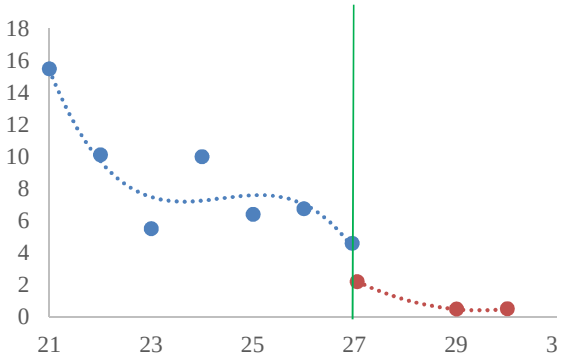
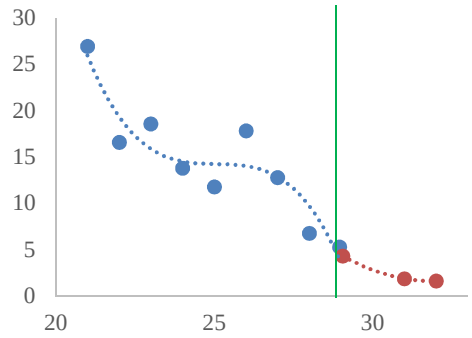
Table 3.1. Definition of Control Variables

Variable	Coding
Age	=Respondent's age
Race/Ethnicity	
Hispanic	=1 if the person is Hispanic
Black	=1 if the person is Black
Marital status	=1 if the person is currently married
Insurance	=1 if the person holds any kind of health insurance plan
Employment	=1 if the person worked for pay anytime in the last year
Citizen	=1 if the person is American citizen
Health status	=1 if the reported health status is greater than 3 in a 1-5 scale
Gynecological visit	=1 if the person visited a gynecologist in the last year
HIV test	=1 if person has ever been tested for HIV
Pregnant	=1 if the person is currently pregnant
Income	=1 if household's income is above 35,000$

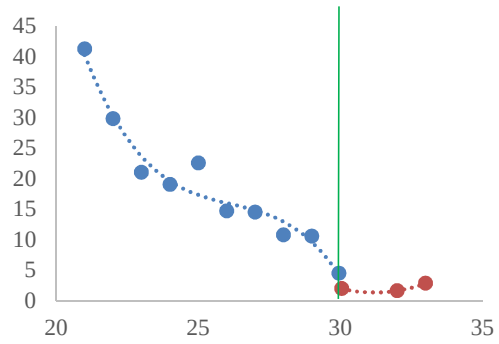
Figure 3.1. Discontinuity of HPV vaccine initiation propensity at the cut-off points



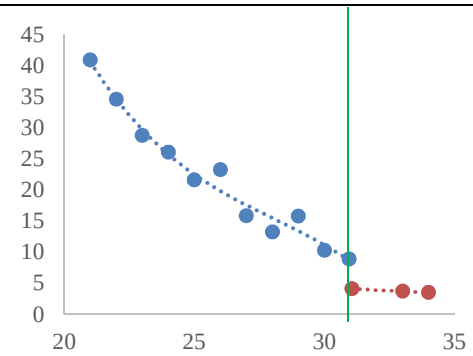
2008



2010



2011



2012

Table 3.2. Regression Discontinuity Results

	Pap test in the last year			Had Pap test in the past		
	Probit	IV-Probit	Stage 1	Probit	IV-Probit	Stage 1
VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)
Age	0.0142*	0.0267***	-0.125***	0.0705***	0.0989***	-0.167***
	(0.00841)	(0.0103)	(0.0221)	(0.0210)	(0.0206)	(0.0426)
Insurance	0.385***	0.367***	0.209***	0.0281	-0.0289	0.367***
	(0.0442)	(0.0453)	(0.0666)	(0.102)	(0.0979)	(0.128)
Hispanic	0.171***	0.181***	-0.201***	0.0755	0.109	-0.318***
	(0.0512)	(0.0511)	(0.0692)	(0.122)	(0.112)	(0.122)
Black	0.282***	0.289***	-0.135*	0.0140	0.0638	-0.252**
	(0.0572)	(0.0569)	(0.0687)	(0.128)	(0.119)	(0.122)
Currently married	-0.0796*	-0.0540	-0.349***	0.0824	0.144	-0.416***
	(0.0429)	(0.0447)	(0.0561)	(0.103)	(0.0998)	(0.103)
Employment	0.127***	0.119**	0.107*	0.0475	0.0210	0.124
	(0.0471)	(0.0468)	(0.0641)	(0.103)	(0.0976)	(0.117)
Citizen	0.157***	0.148**	0.221**	0.935***	0.857***	0.0152
	(0.0583)	(0.0579)	(0.0947)	(0.120)	(0.120)	(0.146)
Health status	0.124*	0.125*	0.0122	-0.214	-0.209	0.00199
	(0.0705)	(0.0705)	(0.0953)	(0.167)	(0.158)	(0.166)
Gynecologist visit	1.554***	1.523***	0.0970*	0.871***	0.754***	0.149
	(0.0410)	(0.0478)	(0.0525)	(0.105)	(0.111)	(0.0975)
HIV test	0.176***	0.149***	0.365***	0.722***	0.610***	0.253**
	(0.0393)	(0.0416)	(0.0550)	(0.0903)	(0.0983)	(0.104)
Pregnant	0.0600	0.0693	-0.0867	-0.367*	-0.271	-0.177
	(0.0899)	(0.0887)	(0.0991)	(0.195)	(0.187)	(0.205)
Income	0.0565	0.0423	0.168***	0.206**	0.170*	0.0981
	(0.0438)	(0.0438)	(0.0547)	(0.0929)	(0.0907)	(0.0920)
HPV vaccine initiation	0.358***	1.003***		0.420*	1.649***	
	(0.0870)	(0.310)		(0.238)	(0.288)	
Excluded IV						
t			0.233**			0.0897
			(0.0965)			(0.183)
ρ		-0.355**			-0.890***	
		(0.180)			(0.324)	
F-test		5.83**			0.24	
Constant	-1.615***	-2.024***	1.722**	-1.960***	-2.834***	2.942**
	(0.288)	(0.343)	(0.736)	(0.660)	(0.633)	(1.310)
Observations	7,056	7,586	7,586	2,546	2,557	2,557

Note: Regressions include region and year fixed effects. The numbers in parenthesis are robust standard errors. *** p<0.01, ** p<0.05, * p<0.1

Table 3.3. Marginal effects

	Pap test in the last year		Had Pap test in the past	
	Probit	IV-Probit	Probit	IV-Probit
VARIABLES	(1)	(2)	(3)	(4)
HPV vaccine initiation	0.0879***	0.189***	0.0204**	0.0500***
	(0.0181)	(0.0339)	(0.00816)	(0.0109)
Observations	7,056	7,586	2,546	2,557

Note: Regressions include region and year fixed effects. The numbers in parenthesis are robust standard errors. *** p<0.01, ** p<0.05, * p<0.1

Table 3.4. Linear Probability Model

	Pap test in the last year			Pap test in the past		
	OLS	2SLS	Stage1	OLS	2SLS	Stage 1
VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)
Age	0.00299	0.00585*	0.00202	0.00766***	0.00960***	0.00453
	(0.00196)	(0.00347)	(0.00231)	(0.00226)	(0.00372)	(0.00364)
Insurance	0.108***	0.105***	-0.00433	0.00186	-0.00101	-0.0100
	(0.0122)	(0.0125)	(0.00690)	(0.0143)	(0.0148)	(0.0102)
Hispanic	0.0427***	0.0457***	0.00198	0.0113	0.0140	0.00959
	(0.0120)	(0.0125)	(0.00776)	(0.0149)	(0.0152)	(0.0124)
Black	0.0673***	0.0698***	0.00335	0.00143	0.00445	0.00644
	(0.0120)	(0.0124)	(0.00840)	(0.0125)	(0.0134)	(0.0123)
Currently married	-0.0164*	-0.0109	0.00662	0.0148	0.0188	0.0133
	(0.00997)	(0.0115)	(0.00688)	(0.0107)	(0.0127)	(0.0113)
Employment	0.0261**	0.0248**	-0.00191	0.00158	0.000436	-0.000988
	(0.0119)	(0.0120)	(0.00655)	(0.0132)	(0.0133)	(0.00923)
Citizen	0.0440***	0.0414***	0.00333	0.156***	0.156***	0.00545
	(0.0153)	(0.0155)	(0.00786)	(0.0224)	(0.0223)	(0.0116)
Health status	0.0259	0.0264	-0.00166	-0.0239	-0.0239	-0.00368
	(0.0183)	(0.0184)	(0.0106)	(0.0162)	(0.0162)	(0.0157)
Gynecologist visit	0.447***	0.446***	-0.00319	0.0921***	0.0905***	-0.00265
	(0.0105)	(0.0105)	(0.00631)	(0.0104)	(0.0104)	(0.00974)
HIV test	0.0448***	0.0393***	-0.00924	0.0964***	0.0937***	-0.00766
	(0.00985)	(0.0113)	(0.00654)	(0.0128)	(0.0134)	(0.0104)
Pregnant	0.0162	0.0183	-0.00202	-0.0249	-0.0226	-0.000330
	(0.0148)	(0.0150)	(0.0114)	(0.0185)	(0.0190)	(0.0171)
Income	0.0168	0.0137	-0.00148	0.0236**	0.0226**	-0.00348
	(0.0103)	(0.0107)	(0.00680)	(0.0108)	(0.0109)	(0.00979)
HPV vaccine initiation	0.0751***	0.221*		0.0310**	0.134	
	(0.0164)	(0.134)		(0.0148)	(0.121)	
Excluded IV						
Fitted value			1.167***			1.316***
			(0.137)			(0.233)
F-test		72.81***			31.83***	
Constant	0.0936	-0.00272	-0.0711	0.456***	0.394***	-0.156
	(0.0674)	(0.117)	(0.0795)	(0.0757)	(0.121)	(0.120)
Observations	7,056	7,056	7,056	2,546	2,546	2,546

Note: Regressions include region and year fixed effects. The numbers in parenthesis are robust standard errors.
*** p<0.01, ** p<0.05, * p<0.1

Table 3.5. Robustness Check

Panel A- Higher Polynomial Orders

			Polynomial terms in Pap test equation		
			Linear	Quadratic	Cubic
Polynomial terms in HPV vaccine initiation equation	Linear	Vaccine initiation	1.003*** (0.310)	0.983*** (0.316)	0.985*** (0.3168)
		t	0.233** (0.0965)	0.236** (0.0964)	0.236** (0.0964)
		ρ	-0.355** (0.180)	-0.344* (0.1818)	-0.346* (0.1825)
	Quadratic	Vaccine initiation	1.049*** (0.3063)	1.009*** (0.3196)	1.011*** (0.3202)
		t	0.283** (0.103)	0.284*** (0.103)	0.284*** (0.103)
		ρ	-0.385** (0.1832)	-0.361* (0.1874)	-0.362* (0.1878)
	Cubic	Vaccine initiation	1.047*** (0.3057)	1.007*** (0.3195)	1.010*** (0.3203)
		t	0.276*** (0.1964)	0.277*** (0.1063)	0.278*** (0.1063)
		ρ	-0.383** (0.1826)	-0.359* (0.1870)	-0.361* (0.1877)
	Observations		7586	7586	7586

Panel B- Adding Interaction Term as an IV and Smaller Intervals

	Interaction	6 years interval	4 years interval	2 years interval
Vaccine Initiation	1.011*** (0.3098)	1.069*** (0.3428)	0.694 (0.4599)	0.132 (0.5498)
t	2.323** (1.1548)	0.184 (0.1188)	0.193 (0.1588)	0.655*** (0.1753)
Age*t	-0.067* (0.0367)			
ρ	-0.361** (0.1815)	-0.409** (0.2046)	-0.220 (0.2227)	0.105 (0.2038)
F-Statistics	4.13**	2.40	1.48	14.24***
Observations	7586	5731	3923	1956

Note: Regressions include region and year fixed effects. The numbers in parenthesis are robust standard errors. *** p<0.01, ** p<0.05, * p<0.1

CURRICULUM VITAE

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Education

Ph.D., Economics, University of Wisconsin-Milwaukee, Milwaukee, WI, USA, 2014
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M.A., Economics, University of Wisconsin-Milwaukee, Milwaukee, WI, USA, 2010
MBA., Mazandaran University of Science and Technology, Babol, Iran, 2008
B.Sc., Metallurgical Engineering, Sharif University of Technology, Tehran, Iran, 2005

Interests

Primary: Applied Microeconomics, Health Economics, Economics of Education,
Secondary: Economics of Development

Working Paper

"The Negative Publicity of the Child Abuse Scandal and Catholic School Enrollment"

"School Mandates, Education for Parents, or Physician Recommendations? The Most Effective Way to Increase Human Papillomavirus Vaccine Coverage"

"Immunization and Moral Hazard: The Effect of Human Papillomavirus vaccination on Participation in Routine Pap Test" (with Scott Adams)

Presentations

Midwest Economic Association Annual Meeting, Evanston, 2014
University of Wisconsin-Milwaukee Labor Lunch, Milwaukee, 2013 and 2014
Annual Iranian Casting Society Congress, Iran, 2006

Research Experience

Research Assistant, Professor Laura Grant, School of Freshwater Science and Department of Economics, University of Wisconsin-Milwaukee, Milwaukee, WI, USA, August 2013- present

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