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# *Lowered Female Fertility Associated with Human Papilloma Virus Vaccines*

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## ABSTRACT

The falling birth rate in the United States may be owed to multiple factors, the human papilloma virus (HPV) vaccines being among them. Here I examine again the hypothesis that the likelihood of having been pregnant at least once was reduced for women aged 25 to 29 between 2007 and 2018 who received one of the HPV vaccines compared with peers who did not. Data from the National Health and Nutrition Examination Survey (NHANES) representing 7.5 million women in the United States were used. The age-range was limited in order to compare women in the optimal age-range for child-bearing who received at least one HPV shot during the study period against peers who did not. Given that the HPV vaccines are aimed at preventing cervical cancer, but not at reducing or enhancing fertility, the opportunity and choice to receive such a vaccine should be about equal across all the women in the sampled age-range and time frame. Analysis revealed that only 47% of HPV vaccine recipients had ever conceived as contrasted with 69% of comparable peers who did not receive any HPV shot. If pregnancies after receiving such a shot were unaffected by it, the women in both groups should be equally likely or unlikely to get pregnant. Nevertheless, even when covariate controls for marital status, age, education, income, race/ethnicity, obesity and smoking were used, a multivariate logistic regression showed a reduced likelihood of pregnancy in the HPV vaccinated women (OR 0.66; 95% CI 0.438, 0.998): *women who received the HPV vaccine were less likely to have been or to become pregnant during the time frame examined.* The reasonable conclusion is that receiving an HPV vaccination reduces female fertility. If the shot were aiming to be a birth-control vaccine the observed result would not be anomalous. But it is, and there is other research showing that at least two of the viruses targeted by all the HPV vaccines on the market, 16 and 18, can cause sterility in both females and males and are also associated with so-called "spontaneous" abortions and premature ovarian failure in pregnant female carriers of those pathogens.

**Keywords:** *aluminum, anti-fertility, autoimmune/inflammatory syndrome induced by adjuvants (ASIA), fertility, HPV vaccines, HPV pathogens, HPV 16 and 18, pregnancy rates, human papilloma virus vaccines, premature ovarian failure*

## 1. Introduction

Birth rates in the United States for women under the age of 30 are at a record low (Adamy, 2020) and still falling according to CDC statistics (Hamilton et al., 2020). Obviously, for a woman to give birth she must become impregnated first. What is not so obvious, as Oller pointed out (personal communication), is that impregnation requires an eye-poppingly intense sequence of billions upon billions of successful biosignalling exchanges in order for a live birth to occur downstream: first, before conception can occur an articulated sequence of exchanges must occur within both parents enabling successful gamete loading (meiosis). Then, after a sexual act (or an *in vitro* meeting of male and female gametes), the union of a male sperm with a female egg requires several billions of additional successful communications to achieve fertilization which must be followed by multiple mitosis events each involving billions of successful exchanges of information within the dividing cells before migration and implantation of the blastocyst can occur (see discussion and references in Oller, 2020). Assuming all goes

swimmingly, a multitude of additional mitosis events must occur during embryological development in order for a live birth to occur later on.

### 1.1. Interference with Biosignaling Processes

Along the way, as has been previously shown in studies of fertility and anti-fertility, either manufactured or accidental perturbation of the biosignaling events in the articulated sequence of sequences just described above can either prevent a pregnancy from developing or cause it to fail after it is underway. For instance, the disruption of the biosignaling events necessary to a successful pregnancy is deliberately interrupted by the World Health Organization (WHO) birth-control vaccines under development since the early 1970s (Talwar et al., 1976; Oller et al., 2017, 2020) and it is accidentally disrupted by the human papilloma viruses HPV 16 and 18 which are included in all the HPV shots under study in this paper (Yang et al., 2013; Depuydt et al., 2016a; Depuydt et al., 2016b; Garolla et al., 2016).

For researchers engaged in the study of the biosignaling systems associated with meiosis and mitosis — especially in connection with the articulated processes of fertilization followed by enzyme regulated growth and development — it is hardly surprising that pathogens associated with cancers, such as HPV 16 and 18 are known to be, can also interfere with fertility in both males and females. The fact is that growth processes and systems of communication commandeered by cancer producing viruses are known to be almost indistinguishable in many respects from the normal articulated growth and development sequences with respect to the gene regulating events known or believed to be involved in both normal and abnormal mitosis (Oller & Shaw, 2019).

### 1.2. The Motivation for This Study

That being noted, the HPV shots brought under close scrutiny in this paper, merit consideration in light of the falling birth rate in US women aged 25 to 29 (the peak years for child-bearing) that began to decline since 2007, coincidentally perhaps or perhaps not, after the wide-scale marketing of HPV vaccines. In the US the birth-rate index of fertility fell 20.7% from 118.1 per 1,000 women in 2007 to 93.7 in 2019. The notable decline followed an increase of 8.5% between 1995 and 2006 from 108.8 to 118.0 (Hamilton et al., 2020). The basis for the recent decline remains uncertain though among the reasonably suspected factors are the HPV shots that were introduced in 2006 when the US Food and Drug Administration licensed the first of three multivalent HPV vaccines supposedly designed to protect women against certain of the many human papillomaviruses (U.S. Food & Drug Administration, 2006).

The multivalent HPV vaccines on the market (Gardasil, Gardasil9, and Cervarix) all aim to address HPV 16 and 18 — the two strains of HPV that are believed to produce approximately 70% of cervical cancer cases according to the CDC (Centers for Disease Control and Prevention, 2020), and that, as I have noted above, also been associated in the research literature with male and female infertility. In addition, according to its manufacturer, the Gardasil9 shot is supposed to protect against genital warts by interfering with HPV 6 and 11 (Markowitz et al., 2014). Gardasil9 is also supposed to protect against HPV types 31, 33, 45, 52, and 58 (in addition to 6, 11, 16, and 18) and is approved in the United States for females and males aged 9 to 45 (U.S. Food & Drug Administration, 2020). However, in a laboratory study of Gardasil9, using highly sensitive polymerase chain reaction primers, because "no L1 [capsid protein] gene DNA of HPV 31, 33, 45, 52, and 58" — five of the nine HPVs supposedly targeted — could be found in Gardasil9, Lee (2020) concluded that "these may all be in non-B [non-biodegradable] conformations or may have been removed as contaminants by a purification protocol". He also suggested that "non-B conformations may also induce a mutagenic and genomic instability effect with far-reaching consequences (Bacolla, & Wells, 2009; Zhao, Bacolla, Wang, & Vasquez, 2010)". The clear implication, based on the relevant research, is that male and female infertility after vaccination, so-called "spontaneous abortions" in women who got the vaccination while pregnant, and premature ovarian failure (pre-menopause) may all be causally connected with HPVs and derivative components used in vaccines. At any rate, both the vaccines, the HPVs themselves, and any components of HPVs in vaccines deserve closer scrutiny with respect to their potential impact on female fertility and the birth rate.

### 1.3. Toxic Adjuvants Are Also Used

Furthermore, the HPV vaccines contain adjuvants that have been indirectly linked, or at least implicated, in reducing fertility in humans (Yan et al., 2000; Borchers et al., 2002; Colafrancesco et al., 2014; Naim et al., 2020) and have been widely studied in anti-fertility models in animals (Siel et al., 2018, 2020). All licensed HPV vaccines contain aluminum adjuvants, known to interfere with biosignaling systems in general (C. A. Shaw, Li, et al., 2014; C. A. Shaw, Seneff, et al., 2014). Because biosignaling is most intensive and vulnerable to disruption during gamete loading (meiosis), sexual exchanges required for fertilization, and in the mitosis events most intensive in early embryological growth, in roughly that order of importance, may be interfered with by the aluminum adjuvants in some vaccines, including the HPV vaccines. Those adjuvants are particularly suspect when it comes to the articulated biosignaling sequences essential for successful pregnancies (Hassold & Hunt, 2001).

Nayak (2002) reports the negative effects on reproduction — both male and female — of aluminum. One of the possible routes for this disruption to occur involves the association between aluminum and autoimmune disorders. Colafrancesco et al. (2013) reported antiovarian antibodies — biomarkers of an autoimmune response — in a young woman who experienced premature ovarian failure (POF), the kind of failure not expected to occur until menopause, after receiving an HPV vaccination. Gruber and Shoenfeld (2015) explored the possible link between aluminum in HPV shots and POV. Little and Ward (2012, 2014) documented case studies of young women experiencing menstrual disorders that developed into POV. In those instances, the POV occurred shortly after an HPV vaccination. Pellegrino et al. (2015) found evidence to support the existence of the ASIA syndrome among HPV vaccine recipients by investigating the Vaccine Adverse Events Reporting System (VAERS) database — a passive system where vaccine administrators or recipients can report adverse effects after being vaccinated. In another study of VAERS data, between 2006 and 2014, Geier and Geier (2017) found 461 cases of serious autoimmune adverse events associated with an HPV vaccination, including 48 cases of ovarian damage. Another suspect adjuvant in the HPV shots is polysorbate 80, which Esposito et al. (2014) reported as also associated with autoimmune/inflammatory syndrome induced by adjuvants (ASIA).

### 1.4. Grossly Incomplete Reporting of Adverse Events

Putting the foregoing findings into their proper context, the actual number of post-HPV vaccination adverse events is almost certainly much more common than could be discovered by studies based on VAERS data. We know from an empirical study by Lazarus et al. (2010) encompassing an estimated 1.4 million doses of 45 different vaccines to 356,452 individuals, there were actually "35,570 possible [adverse] reactions (2.6 percent of vaccinations)" that might be reported, but a vastly smaller number of such reactions will ever actually appear in VAERS data. Although largely ignored by the CDC, the FDA and other federal agencies, this publicly sponsored study done by the Harvard Pilgrim group concluded that "fewer than 1% of vaccine adverse events are reported" under the existing systems provided by the US government agencies. If that estimate is correct, and it is solidly grounded in empirical research, the estimated number of injuries associated with any given vaccination must be considered much greater than the very few that get into VAERS. In fact, that report leads us to suppose that the number of adverse events that come to the attention of the CDC, FDA, and other government agencies supposedly guarding the public safety should be multiplied by a factor of about 100 to put the estimated number of injuries in the ballpark vicinity of rational thought. Plainly, VAERS estimates are almost certain to be absurdly lower than they ought to be.

## 2. Methods

The study conducted here, examined the possible impact of HPV vaccinations on pregnancy rates. It began from the evidence of the still declining birth rate in the United States. The decline dramatically appeared in CDC's National Center for Health Statistics (NCHS) database beginning in 2007 as seen in Figure 1. There, the NCHS (2020) provides data on live births per 1,000 females aged 25 to 29 — the peak child-bearing years. The figure shows the national birth rate from 1995 to 2019. The line reveals a fairly steady increase continuing through 2006, followed by a sharp decline that is evident from 2007 forward: the notable down-turn seen in the NCHS data is the

focus of the analysis examined here for the second time.[1] It might be coincidental that about a year before that decline became distinctly noticeable, HPV vaccines became available and were widely administered in the US. But the introduction of HPV shots in 2006 might not be unrelated to the downturn in the US birth rate as seen in Figure 1.

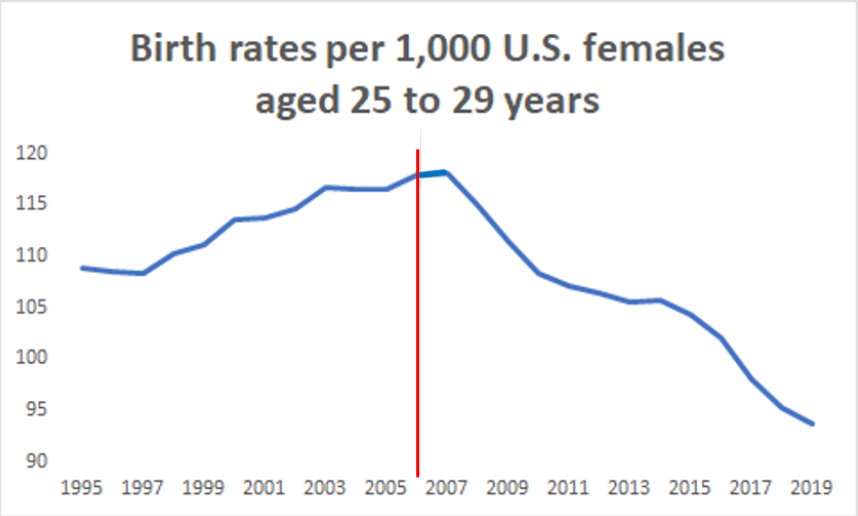


Figure 1. Birth rates per 1,000 females in the United States aged 25-29 from 1995-2019. The vertical line marks the year 2006 when the first HPV vaccine was licensed in the United States (see Meites et al., 2020).

### 2.2. Explaining the Critical Survey Questions Used Here

To test the possibility that the HPV shots could possibly be a causal factor in the observed birth rate decline, data from the National Health and Nutrition Examination Survey (NHANES) from the years 2007 to 2018 focusing only on respondents to that survey during their peak child-bearing years were assembled and critically scrutinized statistically.

The NHANES collects health and nutrition data along with demographic and socioeconomic information from its respondents. The National Center for Health Statistics (NCHS), a subagency of the Centers for Disease Control and Prevention (CDC), administers the survey and tries to obtain a representative sample of the US population based on a complex subjective, not a strict algorithmic sampling procedure. The approach is roughly described at the CDC website so that participants in the NHANES will know what to expect and how the information they provide will be used (see https://www.cdc.gov/nchs/nhanes/participant.htm). Data are reported in two-year increments.

Starting in 1999, the NHANES asked females aged 12 and up "RHQ131 [Respondent's Health Question number 131]: Has the survey participant ever been pregnant? Please include current pregnancy, live births, miscarriages, stillbirths, tubal pregnancies and abortions." Responses could be (1) yes, (2) no, (7) refused, (9) don't know, or could be skipped over and left missing. Starting in 2007, the NHANES also asked females aged 9 years and older "IMQ040 [Immunization Question number 40]: Has the survey participant ever received one or more doses of the

[1] This paper is an extensive rewrite of one that the author believes was retracted without just cause from the *Journal of Toxicology and Environmental Health, Part A* (DeLong, 2018). I objected formally in a series of emails. Here I take the matter a step further and re-issue my argument, adding more years of data as well as more explanatory variables, reviewing additional relevant research, some of which has appeared in the interim, while also addressing the expressed and implied concerns of those critics (the most important of whom remained in the shadows) who insisted, unfairly I believe, on the retraction. It is impossible for me to examine their credentials because the critics instrumental in forcing the retraction of my earlier work were unnamed by the publisher of the journal. That journal published by Taylor and Francis Group (Informa Group, 2021) is merely one of 2,700 in their list, and is focusing more and more according to the parent company, Informa Group, on " Pharma and Consumer Retail Banking" (Informa, Chairman Derek Mapp, 2021). In 2018 that company posted revenues of £385,000,000 of which the revenue contributed by the publisher Taylor & Francis with was £530,000,000 producing an adjusted operating profit that year of nearly £200,000,000. Clearly there is money to be made in academic Pharma publishing. Also see recent articles by Children's Health Defense Team (2021) and the article in this journal by Daniel Broudy (2021) as well as Christopher Shaw's book *Dispatches From the Vaccine Wars* (2021, pp. 312–319).

HPV vaccine?" Possible responses were the same as for the pregnancy question (RHQ131). In March 2020, the NCHS suspended the collecting of NHANES data due to COVID-19. Therefore, the two-year 2019-20 cycle was incomplete. The time frame for this study, because of the crucial questions (RHQ131 and IMQ040), included the years from 2007 — the first year after the introduction of an HPV vaccination for US women in 2006 — through 2018, the final year of complete two-year cycles.

### 2.2. Multiple Logistic (Logit) Regressions

To analyze the data, the SURVEY LOGISTIC procedure from SAS Version 9.4 was used. In a set up to be explained below, I performed both a global logistic regression analysis without covariates and several separate multiple logistic regressions with covariates included to test the following hypothesis:

> ***The Alternative Hypothesis*****:** *Receiving one or more HPV shots, at any time during the years 2007-2018, is associated with a reduced probability of a survey participant subsequently becoming pregnant.*

That hypothesis can be rejected with the NHANES data if the introduction of the HPV shots does not reduce the likelihood of a pregnancy in the persons who received them as contrasted with those women who did not get any HPV vaccinations.

Some factors that may come to mind are irrelevant or could only come into play in a way that would bias things against the *alternative hypothesis*. The statistical procedures in both the multiple regression procedure without covariates included and the multiple regressions with covariates in the mix, for example, are indifferent to whether an HPV shot comes early or late in the time frame referred to. The later the shot comes in the time frame, the smaller is the likelihood that the shot could prevent any impregnation of the respondent, but this factor could only bias the data against the predicted outcome (because an impact causing a reduction in pregnancies might come after the study period ended). So, that factor can be safely ignored in all the regression analyses. Also, given that the pregnancy question (RHQ131) asked about all of the respondent's life prior to answering the question, it covered any pregnancies that may have occurred before the year 2006 — pregnancies that could not possibly have been prevented by an HPV shot because no such shot was available before that year. However, any pregnancies before the interview occurred (sometime after 2007), would have had to occur prior to the respondent being exposed to any HPV shot. Therefore, those pregnancies would only be noise in the data tending to swamp and cover over any possible impact of HPV vaccines on the US pregnancy rate in the women represented in the NHANES between 2007 and 2018. Therefore, those pregnancies occurring prior to any HPV shot in women who happened to receive one or more of those vaccinations would be largely irrelevant but, again, could only bias things against the hypothesis being tested. Therefore, they too can be safely disregarded.

Assuming only that the women who got the shot were comparable in all other relevant respects to those who did not receive it, a straight-up comparison of the percentage of pregnancies, if they should turn out to be significantly reduced for shot recipients, should be interpretable as correlated with, if not directly caused by, having received one or more HPV shots.

Concerning the *alternative hypothesis* under examination (as set off from the text and italicized above), there are exactly three logically possible outcomes:

(1) There may be no contrast between the women who got the shot and those who did not which result would enable rejection of the *alternative hypothesis*.

(2) A statistically significant (non-chance) reduction in pregnancies among those who got the shot might be found and would enable rejection of the ***null counterpart hypothesis*** — *the proposition that the HPV shot has no effect on relative fertility of recipients versus non-recipients of one or more HPV vaccinations*.

(3) If the HPV shots for some outlandish (totally unexpected) reason should actually cause an increase in fertility — an outcome that is considered unlikely to a limit of absurdity, given what we know about biosignaling systems (see opening discussion above and the following immediately below) — would also enable the rejection of the *alternative hypothesis* and its *null counterpart*.

### 2.3. The Role of Biosignaling Systems in General

As one of the reviewers of this version of my paper pointed out, possible outcome (3) just above here is not a reasonable one because no injurious corruption of the human biosignaling systems should ever be expected to improve those systems. The reverse is predicted for all such experimental challenges with no long-term exceptions and only apparent short-term exceptions (Pellionisz, 2012; Oller, 2014; Davidson and Seneff, 2012; Gryder et al., 2013; Shaw, 2017). While specific immune resistance to particular pathogens may be improved for natural or induced exposures in the short-term — much in the way that strength training, military exercises, and actual combat may improve survivability in real fighting and in wars (also see Shaw, 2021) — there is no reason to suppose that real challenges to biosignaling processes can actually increase longevity any more than actual combat is likely to increase the combatant's long-term survival. Combative skills may enable survival in combat, in the short-term, but neither those skills nor the experience gained in actual combat (e.g., in real life pathogenic encounters) can be expected to lengthen a person's life if all else is held equal. Injuries are like information loss in what is called "entropy" in physics. Their negative effects accumulate and even where they are survivable and repairable, they mount up over time as what is termed "aging". Combat preparedness may help us through a skirmish or even an all out war, but on the whole, it does not contribute to the longevity of the individuals who experience the combat. On the contrary, combat, like infections with pathogens, tends to produce injuries and death.

Long-standing mathematical proofs (Peirce, 1897; Tarski, 1949,1956) have been constructed showing that adding multiple challenges (toxicants, pathogens, and other sources of injuries) on top of each other, ones that interfere with or corrupt biosignaling systems — as all injurious exposures do — are universally interactive in a negative way over the long-term. In the short-term, cutting, burning, poisoning, and infecting the body may have a curative effect, but over the long-term cumulative injuries leading to disorders and even temporary infectious diseases must cumulatively trend toward the catastrophic failure known as mortality (Davidson and Seneff, 2012; Davidson et al., 2013; Gryder et al., 2013; Kennedy et al., 2016). All of this follows from the underlying nature of what has been called biosemiotic entropy which is provably irreversible (Oller, 2010, 2014; Pellionisz, 2012). As proved more than half a century ago by Jaynes (1957a, 1957b, 1965) for all physical systems, once the corruption of entropy is introduced into a complex system, it cannot be filtered out any more than an egg can be unscrambled. Injuries may in many cases be repaired and incredible burdens may be relieved, but, sad to say, their negative effects accumulate toward the catastrophic failure that ends in death. In biological systems, what has been called "biosemiotic entropy", the kind that corrupts biosignaling systems from DNA upward, leads invariably toward mortality and is cannot be improved over the long-term, for example, by adding comorbidities on top of the burdens presented by say a multivalent HPV vaccine challenge. Even if the HPV vaccines should do exactly what they purport and claim to do, adding burdens to the challenges they present to the maintenance, repair, and defense systems of the body — say by smoking, obesity, prior illness, ongoing infections, or any other comorbidity — can only increase, not diminish, whatever risks are already present in the HPV shots.

## 3. Analysis and Results

With all the foregoing in mind, it was possible with the data from NHANES, to test certain suspected factors that probably influenced the outcome with respect to the observed contrast between women who got at least one HPV shot and those who did not (the *alternative hypothesis*). The most straightforward test without taking into account some of the detail to be examined with a logistic (logit) model using various multiple regressions, some of them employing covariates, is that only 46.7% of the women who received an HPV shot became pregnant as contrasted with 68.6% of those who did not receive the shot (as seen in Table 2a, in the first data column and on rows 12 and 13) using a simple variant of the *t*-test. The global straightforward difference between groups — despite the biases in favor of the outcome that the HPV shots would have no impact on the rate of impregnation — was an overall negative 21.9% — a result that suggests the HPV shots probably had an impact in reducing the likelihood of pregnancy in women during their peak years for child-bearing.

**Table 1.** Descriptive Statistics and Contrasts on Possibly Impactful Variables Between 25-29 Year Old Women in the National Health and Nutrition Examination Survey in the Years 2007-2018 Who Received an HPV Shot Versus Those Who Did Not:
Broken Down by Demographic, Socioeconomic, and Health Factors

| Potential Influencing Factors | Received HPV shot? | | |
|---|---|---|---|
| | Yes<br>n = 222 | No<br>n = 728 | Difference<br>(p-value)* |
| **1. Age at interview** | | | |
| Mean | 26.65 | 27.10 | -0.450 |
| standard error of mean | 0.118 | .0.065 | 0.0004* |
| **2. Married, currently or formerly**[a] | | | |
| Mean (%) | 52.17% | 68.90% | -16.73% |
| standard error of mean | 0.041 | 0.020 | 0.0004* |
| **3. Relative income** | | | |
| Mean | 3.028 | 2.540 | 0.488 |
| standard error of mean | 0.127 | 0.076 | 0.0005* |
| **4. College graduate**[a] | | | |
| Mean (%) | 50.95% | 30.16% | 20.79% |
| standard error of mean | 0.041 | 0.024 | 0.0000* |
| **5. Hispanic**[a] | | | |
| Mean (%) | 16.64% | 18.40% | -1.76% |
| standard error of mean | 0.035 | 0.017 | 0.3218 |
| **6. NH Black**[a] | | | |
| Mean (%) | 15.57% | 11.67% | 3.90% |
| standard error of mean | .024 | .013 | 0.0714 |
| **7. NH White**[a] | | | |
| Mean (%) | 57.45% | 59.92% | -2.47% |
| standard error of mean | .035 | .023 | 0.29105 |
| **8. Other**[a] | | | |
| Mean (%) | 10.24% | 10.00% | -0.24% |
| standard error of mean | 0.020 | 0.130 | 0.4666 |
| **9. Obese** | | | |
| Mean | 32.72% | 35.72% | -3.00% |
| standard error of mean | 0.041 | 0.021 | 0.2554 |
| **10. Smoker** | | | |
| Mean | 14.36% | 25.59% | -11.23% |
| standard error of mean | 0.027 | 0.017 | 0.0002* |

*This *p*-valute, significant at less than .05 if marked with an asterisk in the table, represents the likelihood of a contrast (represented as the difference between participants answering "yes" as contrasted with "no") as great as the one observed occurring by chance: calculated from the *t*-statistic = $(\text{mean}_1 - \text{mean}_2)/(\text{sqrt}(\text{standard error of mean}_1^2 + \text{standard error of mean}_2^2))$.

### 3.1. Some Details of the Analysis and the Descriptive Statistics

Missing responses — coded in the NHANES data as "refused," "don't know," or "missing" — were dropped entirely from all the analyses. This is technically called a "listwise deletion" meaning the entire survey for that individual was discarded. Also excluded from the analysis were women who could not conceive or seemed actively trying to avoid pregnancy. If a woman had a hysterectomy before ever giving birth or if she was currently using a form of birth control reported in NHANES — namely, a birth control pill, condom, or an injectable, and had never been pregnant — her record was dropped from the analysis.

The advantage to a listwise deletion is that comparisons across subgroups in the NHANES data are far more likely to be apples with apples rather than apples with no one knows exactly what.

If a respondent reported being married, widowed, divorced or separated, an entry of 1 was used meaning "married, currently or formerly" and a 0 meaning "never married". The raw values reported for the respondent's age, and income (a ratio relative to a poverty index) were used as reported by the respondent. The educational level reported was recoded in the binary format for the statistical analyses as 1 if the woman reported being a graduate of a four-year college, and 0 if not. The responses concerning race and ethnicity were recoded as 1 or 0 for each of the four racial/ethnic groups — Hispanic, non-Hispanic Black, non-Hispanic White, and other — adding four binary variables that could be used as covariates in the regression analysis. Additionally, if a woman's body mass index (BMI) was 30 or greater, she was considered obese and I assigned a '1' to her observation and a '0' to those women who BMIs were less than 30. Another dichotomous variable — "smoker" — was '1' if the woman currently smoked and '0' if she did not currently smoke.

In keeping with the CDC reporting procedures for samples used to construct population level estimates, the "masked variance pseudo-stratum" adjustment — a statistical work-around to control for errors in variance estimates in the stratified clusters in the NHANES, for example — was applied in the standard way and is reported in the descriptive statistics and estimates in Table 1 (for the CDC explanation of that weighting adjustment see their website at https://wwwn.cdc.gov/Nchs/Nhanes/2013-2014/DEMO_H.htm#SDMVSTRA).[2]

Table 1 includes a breakdown (with the CDC weighting adjustments applied). That being taken into account, because the survey data used here came from twelve years worth of data obtained in two-year increments as shown in Figure 1, the statistical "masked" (not shown) weight adjustments for each 2-year period were divided in each instance by six to get the population frequency estimates reported in Table 1. The contrasts of greatest interest are those between women who reported getting one or more HPV shots (column 2 from the left side of the table) as against those who reported not having done so (column 3 from the left).

### 3.2. Controlling Extraneous Factors

Ideally, the sample of women in the NHANES who received an HPV shot with women who did not receive the shot would be matched in terms of marital status, age at the time of the interview, ratio of family income to poverty, educational level, obesity, and smoking habits. Based on the reasoning explained in the following paragraph, it was possible to test for any contrast across groups for each of the potential interfering variables, as well as the four binary race/ethnicity variables using *t*-tests.

It was surmised that the average age of the women (row 1 in Table 1) in the vaccinated versus unvaccinated group might be a contributing factor, because older women would have had a longer time frame over which to become pregnant. Indeed, as Table 1 shows, reading across row 1, there was a significant contrast between the age of those who got an HPV shot and those who did not. Similarly, it was supposed that marital status might be a contributing factor in the observed difference showing a reduced likelihood of pregnancy in the HPV vaccinated group. If fewer of the women in the vaccinated group were married, for instance, they might be more likely to have used birth control methods, and less likely to be trying to get pregnant. The relevant descriptive statistics in Table 1 on row 2 show that indeed the shot recipients in the study were significantly less likely to be married. So that factor needed to be controlled as a covariate in the regression analysis, as indeed is noted in the formula below for the logistic regressions with covariates in Tables 2, 3, and 4 below.

On row 3 of Table 1, relative income strongly favored the HPV vaccinated group with a statistical significance at $p <$ 0.0005. That variable would be included as a covariate in the appropriate logit regressions so it could be effectively removed in those analyses as discussed below. On row 4 in Table 1, educational status —being reduced to a binary variable of having graduated from college or not — favored the HPV vaccinated group significantly at

[2] Importantly, independent researchers are not able to replicate the CDC methodology. Although, as one of my reviewers pointed out, such an approach can lead to distorted rather than improved estimates (Government of Canada, 2008), it rarely changes outcomes significantly. Regardless, it is an objectionable procedure in scientific research because it cannot be replicated without a more complete accounting than the CDC provides. Taking into account the CDC rationale to enhance the accuracy of population statistics by extrapolating from under-responding groups to guestimates with hypothetically created larger samples, the "masking" of the details of any such procedure nevertheless remains questionable to say the least.

$p < 0.00$. It is a potential contributor because college-educated women tend to start families later and may not only be more inclined to get an HPV vaccination but also to use contraceptives. If so, a reduced likelihood of pregnancy in women who got the shot could be owed partly or entirely to this factor. So it needed to be included as a covariate so its contribution could be statistically removed from the relevant multiple regressions.

The several binary variables included on rows 5 through 8 accounted for no significant difference between the two groups but were included as covariates in the regression analysis to be consistent with standard previous literature that has controlled for race and ethnicity. Also, it would come out in the multiple logistic regressions that some of the race/ethnicity binary variables were significantly involved and needed to be controlled as covariates.

Finally, to finish out the examination of the descriptive statistics in Table 1, the health variables of obesity and smoking were examined in rows 9 and 10. Obese women might have more difficulty conceiving than non-obese women. However, the difference in the percentage of obese women between the group of women who received the shot and the group that didn't was not statistically significant ( $p < 0.26$). The variable was included in the regression analysis to be consistent with the race and ethnicity variables. Smoking could also imperil the ability to conceive. Since the group of women who did not receive the shot included a higher percentage of smokers than the group that did not ($p < 0.0002$), the regression analysis included the variable showing the participant's smoking habits at the time of the interview.

In a more perfect experimental design, it would be desirable to match the HPV vaccination recipients person-for-person with unvaccinated peers on all of the foregoing variables along with others such as, for instance, being a user of prescription or street drugs, exposed to toxicants, a proponent or opponent of certain birth-control methods, religious affiliation, and so forth. However, with the NHANES data available during the time frame of the HPV shots for women of peak child-bearing age focused on in this study, either the requisite questions for a more robust comparison of HPV vaccinated women versus non-vaccinated women were not included in the survey or could not be anticipated to have a straightforward relation to the likelihood of becoming pregnant. While factors such as drug use and toxicant exposure probably do negatively affect the likelihood of a pregnancy in the population at large, the expected interaction between such variables and the criterion relation between getting or not getting one or more HPV shots, and becoming pregnant or not, would require a different questionnaire/interview procedure.

In any case, the analyses applied here, based on the observed match for the average age of women in the two groups, used two methods to control for marital status and education: (1) by including these variables as covariates in a logit regression analysis, and (2) by examining the contrasts between married/never-married women separately as well as considering college graduates separately from non-graduates.

### 3.3. The Models to Be Tested by Logistic (Logit) Regression

Several logistic models were tested by various multiple regressions testing the validity of the predicted outcome, the *alternative hypothesis* (see page 129), that receiving an HPV shot reduces the likelihood of a pregnancy in women of peak child-bearing age. Suspected contributing factors along with race/ethnicity factors coded into the analysis as binary digitized variables (which are notably intermingled with complex socioeconomic variables) were recoded as binary digitized variables to be included as covariates in certain regressions discussed below. All of the relevant and available measures from the NHANES data are listed here in a global formula for the logistic models to be tested by the various regressions reported in subsequent tables:

Binary probability of being or having ever been pregnant$_i$ at the time of the interview = $a_i + b_1$ HPV vaccine$_i + b_2$ ever married$_i + b_3$ age$_i + b_4$ income$_i + b_5$ college$_i + b_6$ NH Black$_i + b_7$ Hispanic$_i + b_8$ other race/ethnicity$_i + b_9$ obesity$_i + b_{10}$ smoker$_i$ + e

Where becoming pregnant = 1 if the $i^{th}$ participant, respondent$_i$, reported having ever been pregnant ator prior to the time of the interview and 0 otherwise; b1 HPV vaccine$_i$ = 1 if respondent$_i$ reported having received the HPV vaccine and 0 otherwise; b2 ever married$_i$ = 1 if respondent$_i$ reported having ever been married and 0 otherwise; b3 age$_i$ = age of respondent$_i$ reported at the time of the interview; b4 income$_i$ = ratio of reported family income to poverty for respondent$_i$; b5 college$_i$ = 1 if respondent$_i$ reported having a college degree at the time of the interview and 0 otherwise, b6 NH Black$_i$ = 1 if respondent$_i$ reported being non-Hispanic Black and 0 otherwise; b7 Hispanic$_i$ = 1 if respondent$_i$ reported being Hispanic and 0 otherwise; b8 other race/ethnicity$_i$ = 1 if respondent$_i$ reported being not Black, Hispanic, or White and 0 otherwise; b9 obesity$_i$ = 1 if respondent$_i$ had

a body mass index equal to 30 or more and and 0 otherwise; b10 smoker$_i$ = 1 if respondent$_i$ reported being a smoker at the time of the interview and 0 otherwise, and e is the error term.

Table 1 above reported the relevant descriptive statistics calculated with SAS 9.4. Respondents who did not provide information on all the variables in the model were dropped (listwise deletion). This is crucial for the logistic regressions to follow because pair-wise deletion of missing cases — as contrasted with any other approach (e.g., replacing missing data by averages and such) — would introduce noise into the data and would not enable optimal comparisons across odds ratios based on subgroups. List-wise deletion of misssing data for any candidate assures that the crucial odds ratios calculated in the logistic regressions are based on actually reported data coming from the same group of NHANES interviewees.

Some variables did show contrasts across the HPV vaccinated and HPV non-vaccinated women. Among the women who received at least one HPV shot, 52% were married, while 69% of the women who did not receive the shot were married. Also, college graduation was more likely in HPV vaccination recipients: 51% of the women who received one to three shots were college graduates, whereas 30% of the women who did not receive any HPV shot had a college degree. Women who received the shot were less likely to smoke (14%) than women who did not receive the shot (26%). These differences are addressed in separate robustness checks using chi-square analysis reported below. The NHANES interviews during the study period (between 2007 and 2018) included 1,450 women of peak child-bearing age (25-29 years). Of those women, 1,055 provided responses for all the variables used in this analysis. I excluded 105 women who either had hysterectomies before ever being pregnant or were actively seeking to prevent pregnancy by using the pill, condom or injectable at the time of the interview. Recall that NHANES seeks out survey participants strategically and adjusts for under-responding groups so that the survey will hopefully represent the whole US population optimally. The sample of 950 respondents, therefore, for the time frame of the study and given the weighting adjustments used by the CDC to optimize the sampling representation, is estimated to account for approximately 7,485,281 women during their peak child-bearing years.

### 3.4. Prevalence Statistics

Table 2a and Table 2b, Parts A-D, detail the chi-square analyses of the odds ratios for the prevalence of pregnancies in women who received at least one HPV shot compared against respondents who did not receive any HPV shot. Using the SURVEYFREQ procedure in SAS 9.4, with two-by-two crosstabulations the impact of HPV vaccinations on pregnancies reported was tested for significance with the chi-square statistic. Results for the entire sample as well as the subsets of ever-married/never-married women, college graduate/not college graduate and smoker/nonsmoker were statistically significant, suggesting that the prevalence of having been pregnant was *not independent of exposure to an HPV shot*. That *null hypothesis* possibility can be definitively ruled out by the contrast as tested by the chi-square statistic in Part A of Table 2a. A contrast as great as the one found in the data could be expected to occur by chance fewer than one time in 10,000 data sets like the one at hand.

Using formulas from MedicalBiostatistics.com (2018), Table 2a, Part A shows that for the entire sample, the difference in the pregnancies for women who received one or more HPV shots (46.7%) and those who did not (68.6%) was -21.9%. Taking account of the population vaccination rate at 25.0%, as estimated from the NHANES sample at issue here, the observed contrast in the reduced pregnancy rate for women who got an HPV shot at negative 0.219 times the weighted frequency of the estimated 1,870,794 women who received the shot would come to a negative 409,924 — that many fewer women would be expected to become pregnant if the whole population had received at least one HPV vaccination. If 100% of the females in the whole population represented by this NHANES data had received such an HPV shot, in theory the number of women who would ever have been pregnant should have fallen by 1.6 million persons (= -0.219 times the weighted frequency of all 7,485,281 women theoretically represented by the NHANES data used in this study).

**Table 2a.** Prevalence Comparisons of Ever Being Pregnant for Women 25-29 Years-of-Age Who Received an HPV Shot in the Time Frame 2007-2018 Versus Women Who Did Not (Part A, Pregnancy? and Part B, Married? )

| HPV Shot Exposure | Part A (Pregnancy?) | | | Part B (Married?) | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Total sample | | | Ever-married women | | | Never-married women | | |
| | *Ever pregnant?* | | | *Ever pregnant?* | | | *Ever pregnant?* | | |
| | Yes | No | Total | Yes | No | Total | Yes | No | Total |
| Received HPV Shot | 120 | 102 | 222 | 82 | 32 | 114 | 38 | 70 | 108 |
| Estimated Population Frequency | 874,251 | 996,543 | 1,870,794 | 630,975 | 345,049 | 976,024 | 243,276 | 651,494 | 894,770 |
| Observed Percentage | 11.7 | 13.3 | 25.0 | 13.0 | 7.1 | 20.2 | 9.2 | 24.7 | 33.9 |
| No HPV Shot | 524 | 204 | 728 | 393 | 89 | 482 | 131 | 115 | 246 |
| Weighted Frequency | 3,853,969 | 1,760,517 | 5,614,486 | 3,051,009 | 815,307 | 3,866,316 | 802,959 | 945,210 | 1,748,169 |
| Weighted Percentage | 51.5 | 23.5 | 75.0 | 63.0 | 16.8 | 79.8 | 30.4 | 35.8 | 66.1 |
| **Totals** | 644 | 306 | 950 | 475 | 121 | 596 | 169 | 185 | 354 |
| Weighted Totals Frequency | 4,728,220 | 2,757,060 | 7,485,280 | 3,681,984 | 1,160,356 | 4,842,340 | 1,046,235 | 1,596,704 | 2,642,939 |
| Weighted Percentage | 63.2 | 36.8 | 100 | 76.0 | 24.0 | 100 | 39.6 | 60.4 | 100 |
| **Rao-Scott Chi-square** | **26.6777** | | | **10.4900** | | | **7.6800** | | |
| ***p* <** | 0.0001* | | | 0.0012* | | | 0.0056* | | |
| **Pregnancy with HPV Shot** | 0.4673 | | | 0.6465 | | | 0.2719 | | |
| **Pregnancy No HPV Shot** | 0.6864 | | | 0.7891 | | | 0.4593 | | |
| **Prevalence if as in Sample** | -409,924 | | | -139,231 | | | -167,705 | | |
| **Prevalence if All Got Shot** | -1,640,157 | | | -690,763 | | | -495,360 | | |

*Probabilities at less than 0.05 are marked with an asterisk and are judged to show significant contrasts on the variable of interest — whether or not the respondent received an HPV shot.

**Table 2b**. Prevalence Comparisons of Ever Being Pregnant for Women 25-29 Years-of-Age Who Received an HPV Shot in the Time Frame 2007-2018 Versus Women Who Did Not (Part C, College Graduate? and Part B, Smoker? )

| HPV Shot Exposure | Part C (College Graduate?) | | | | | | Part D (Smoker?) | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | College graduate | | | Not a college graduate | | | Smoker | | | Not a smoker | | |
| | *Ever pregnant?* | | | *Ever pregnant?* | | | *Ever pregnant?* | | | *Ever pregnant?* | | |
| | Yes | No | Total | Yes | No | Total | Yes | No | Total | Yes | No | Total |
| Received HPV Shot | 26 | 69 | 95 | 94 | 33 | 127 | 27 | 4 | 31 | 93 | 98 | 191 |
| Estimated Population Frequency | 260,119 | 693,066 | 953,185 | 614,133 | 303,477 | 917,610 | 191,801 | 76,930 | 268,731 | 682,450 | 919,614 | 1,602,064 |
| Observed Percentage | 9.8 | 26.2 | 36.0 | 12.7 | 6.3 | 19.0 | 11.2 | 4.5 | 15.8 | 11.8 | 15.9 | 27.7 |
| No HPV Shot | 77 | 107 | 184 | 447 | 97 | 544 | 136 | 32 | 168 | 388 | 172 | 560 |
| Weighted Frequency | 696,300 | 997,165 | 1,693,465 | 3,157,669 | 763,352 | 3,921,021 | 1,104,657 | 332,234 | 1,436,891 | 2,749,312 | 1,428,283 | 4,177,595 |
| Weighted Percentage | 26.3 | 37.7 | 64.0 | 65.3 | 15.8 | 81.0 | 64.8 | 19.5 | 84.2 | 47.6 | 24.7 | 72.3 |
| **Totals** | 103 | 176 | 279 | 541 | 130 | 671 | 163 | 36 | 199 | 481 | 270 | 751 |
| Weighted Totals Frequency | 956,419 | 1,690,231 | 2,646,650 | 3,771,802 | 1,066,829 | 4,838,631 | 1,296,458 | 409,164 | 1,705,622 | 3,431,762 | 2,347,897 | 5,779,659 |
| Weighted Percentage | 36.1 | 63.9 | 100 | 78.0 | 22.0 | 100 | 76.0 | 24.0 | 100 | 59.4 | 40.6 | 100 |
| **Rao-Scott Chi-square** | | **8.1430** | | | **9.1481** | | | 0.2497 | | | **30.8409** | |
| ***p* <** | | 0.0064* | | | 0.0033* | | | 0.6173 | | | 0.0001* | |
| **Pregnancy with HPV Shot** | | 0.2729 | | | 0.6693 | | | 0.7137 | | | 0.4260 | |
| **Pregnancy No HPV Shot** | | 0.4112 | | | 0.8053 | | | 0.7688 | | | 0.6581 | |
| **Prevalence if as in Sample** | | -131,801 | | | -124,835 | | | -14,795 | | | -371,882 | |
| **Prevalence if All Got Shot** | | -365,964 | | | -658,264 | | | -93,902 | | | -1,341,615 | |

*Probabilities at less than 0.05 are marked with an asterisk and are judged to show significant contrasts on the variable of interest — whether or not the respondent received an HPV shot.

Further analyses confirmed the crucial result relative to the *alternative hypothesis* for the parsed-out subgroups defined by whether the women in the study reported being married or not (Part B of Table 2a), whether or not they reported having graduated from a four-year college or university (Part C of Table 2b), and whether or not they reported to be a smoker at the time of the interview. Part B reports that for married women the difference in the prevalence of pregnancy between the group exposed to at least one HPV vaccination (64.7%) and the unexposed group (78.9%) was -14.2%. Part C, then, parses the sample according to educational status. The results show that a lower percentage of women who received any HPV vaccine were ever pregnant, regardless of whether the woman was a college graduate or not. Part D shows that women who received the HPV shot were less likely to have ever been pregnant also holds for women who do not smoke; the results for smokers are not statistically significant, probably due to the small size of the sample at issue. Although the sample groups could not be matched according to marital or educational status or smoking habits, the tendency for vaccinated women to have a reduced pregnancy rate held for married and unmarried women as well as college graduates and women who did not graduate from college and women who did not smoke. In all four scenarios examined in Table 2a and 2b, the *null hypothesis*, that at least one HPV vaccination has no impact on the likelihood of a woman at peak child-bearing age subsequently becoming pregnant, can be safely rejected. That *null hypothesis* is false in every comparison made.

### 3.5. Logistic Regressions Testing Odds Ratios with and without Covariates

Results of the logistic (logit) regressions comparing NHANES respondents of peak child-bearing age who received at least one HPV vaccination are presented in Table 3 in two formats: Part A, the model without covariates included, shows that the odds ratio of women who reported having received an HPV shot would also report having been pregnant at some time prior to the NHANES interview was .401. The probability of such a ratio appearing in the data by chance is less than 1 in 10,000 — a result that is consistent with the computations reported in Table 1 for the descriptive statitistics. Then in Part B, the respective odds ratios with each of the named covariates in the mix are reported. Because the computation of such odds ratios is not always well understood by clinicians and researchers, and because the logistic (logit) regression approach is conceptually complex even without the covariates, a few words of explanation for the non-statistically minded readers of this journal are in order.

First considering the results recorded in Table 3 Part A, the main point to keep in mind is that odds ratios in general vary around the logical balance point of unity. An odds ratio of 1 (spelled out as 1/1, or 1:1) would, in the case of the study at hand, indicate no statistical relation, a zero correlation, between one or more HPV shots and becoming pregnant. Such a possibility, if it had occurred in Part A of Table 3, would require the acceptance of the *null hypothesis* outcome (2) detailed above on page 129. In effect, the actual analysis shows, however, that the likelihood of becoming pregnant after at least one HPV shot, as reported by the NHANES respondents in this study, was reduced: this, in agreement with outcome (1) detailed above on page 129. Therefore, the *null alternative* and the implausible possibility that an HPV shot could increase the likelihood of one or more pregnancies in the study poulaiton can also be ruled out. Neither of those hypotheses can stand scrutiny, so only the *alternative hypothesis* remains.

The inverse of the odds ratio showing a reduction in the likelihood of a pregnancy after even one HPV shot, would be the likelihood of a pregnancy without such a shot. That value can be computed by inverting the ratio 0.401/1 to get 1/0.401 or an odds ratio of 2.494 indicating a greater likelihood of women at peak child-bearing age reporting a pregnancy before or during the study period if they did not receive any HPV shot. Of course, it would be absurd to argue that the correlation of not getting the HPV shot with an increased likelihood of becoming pregnant, shows that not getting such a shot can cause a predictable proportion of women to become pregnant. Similarly, it would only be a little less absurd to suppose that getting such a shot could increase the likelihood of women in the sample reporting having become pregnant before the interview: the latter is a possibility ruled out completely by the results in hand in all of the analyses reported in this version of my paper (in other words, outcome 3, as detailed on page 129 above, can be ruled out).

Bearing all that in mind, one further comment is necessary about correlations in general: while it is true in general that a significant correlation between some variable of interest and any other variable or multiple of other variables does not prove a causal relation between the predictor variables and the one we are trying to explain or account for,

it is quite impossible logically for causal relationship to exist between measured variables in the complete absence of any correlation. A Venn diagram of the sort shown as Figure 2 expresses the logical relation between correlation and causation as explanatory concepts: correlation does not prove causation, but causation demands and requires (is a proof of) the existence of a correlation between the effect and its cause(s). It is possible that some variables will have only a weak causal impact on a variable of interest while others are more impactful, but it is not possible for a causal impact to exist between any pair of variables in the complete absence of any correlation between them.

Proceeding with the discussion of Table 3, the outcome without covariates is presented in Part A and shows that women who received at least one HPV shot were less likely to report having ever been pregnant before the NHANES interview took place: as noted above the odds ratio for women who received the shot to report ever having been pregnant compared with women who did not receive the shot was 0.401 (95 % CI 0.280, 0.574). Then, Part B of Table 3 reports on the logit regression model with the listed covariates in the lower portion of that table.

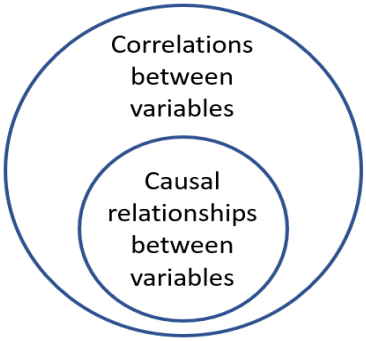


Figure 2.. While correlation cannot prove causation, the latter always is a logical proof of the former.

Including all the covariates in the logistic regression gave an estimated odds ratio at 0.661 (95 % CI 0.438, 0.998) still significant at $p < 0.0488$, and the model fit as estimated by the main concordance statitistic, 83.8, in the third row from the bottom of Table 3, Part B, is substantially greater and better than for the regression model without covariates included, 27.1 in row 2 of Part A of Table 3.

Then, accounting for the covariates and their potential impact, we must look to rows 2-10 in Part B of Table 3. Given that women who did not get any HPV shot were also more likely to report being or having been married, the odds ratio on row 2 shows that the women in the data sample who refused or did not get any HPV shot were 8.047 times more likely to report having experienced one or more pregnancies at sometime prior to the NHANES interview. Thus, with the marriage covariated in the picture, the chances of becoming pregnant for women at peak child-bearing age before or during the study period of 2007-2018 was significantly lower for the women who got an HPV shot at any time during that time frame than for women who did not get any HPV shot with $p < 0.0001$. Again, the *null hypothesis* must be ruled out and the *alternative hypothesis* (outcome 2 on page 129 above) cannot be rejected.

The statistically significant result for married women suggests that married women may be more open to becoming pregnant, or at least they seem to be avoiding it less than the unmarried women among the NHANES respondents included as subjects in this study.

Results for the other explanatory variables in the covariate analysis are as expected. The older the respondent was at the time of the interview, row 3 in Part B, the more chances she had of ever having been pregnant, so the outcome seen in the data with respect to age reported at the time of the interview is as expected. Older women in the period of peak child-bearing years have more time in which they might have been impregnated than younger women in that same age range. Age as a covariate, row 3 in Part B of Table 3, is therefore neither a factor in becoming pregnant at $p < 0.0818$, nor in getting or not getting an HPV shot.

Concerning income, row 4 in Part B of Table 3, the results obtained here are consistent with the research of Huber et al. (2010). They differentiated the reported take home income for married men and women separately. They found a positive correlation between the man's reported income and the number of children reported, but a negative correlation with the woman's reported income. The larger her reported income, the fewer were her reported offspring. Since the income variable used in this study measured relative household poverty (a measure of income where a larger value represents a larger income), the higher the ratio, the more likely the respondent was to be earning an income — odds ratio of 0.696, $p < 0.0001$ consistent with the findings of Huber et al. They also found that more highly educated women, who were more likely to be earning an income, were less likely to have children: that was the case here as well. A woman who earned a four-year college degree, row 5 in Part B of Table 3, was less likely to report having ever been pregnant by age 29 than a woman who had not completed such a degree.

**Table 3** Logistic Regression on Reported Pregnancies of Women During Peak Child-Bearing Ages (25-29 Years) in the United States Who Completed the NHANES Interview During the Years 2007 to 2018.

| **Part A. The Global Model without Covariates** | | | |
|---|---|---|---|
| **Received HPV Shot versus Did Not** | **Scatterwaite Odds Ratio of Reporting Having Been Pregnant** | ***p*-value** | **95% Confidence Interval** |
| | 0.4010 | <0.0001 | {0.280,0.574} |
| Percent concordant | 27.1 | | |
| Percent discordant | 12.4 | | |
| Percent tied | 60.5 | | |
| **Part B. Model with Multiple Covariates** | | | |
| **Received HPV Shot versus Did Not** | 0.661 | 0.0488 | {0.438,0.998} |
| Married, currently or formerly | 8.047 | <0.0001 | {4.665,13.878} |
| Age at interview (years) | 1.139 | 0.0818 | {0.983,1.319} |
| Ratio of family income to poverty | 0.696 | <0.0001 | {0.607,0.799} |
| College graduate vs Not college graduate | 0.252 | <0.0001 | {0.168,0.378} |
| Hispanic vs Non-Hispanic White | 0.866 | 0.5367 | {0.546,1.374} |
| Non-Hispanic Black versus Non-Hispanic White | 3.288 | 0.0002 | {1.774,6.092} |
| Other Race/Ethnicity versus Non-Hispanic White | 0.855 | 0.5619 | {0.500,1.461} |
| Obese | 0.944 | 0.7881 | {0.617,1.443} |
| Smoker | 1.496 | 0.1385 | {0.876,2.555} |
| Percent concordant | 83.8 | | |
| Percent discordant | 16.1 | | |
| Percent tied | 0.2 | | |
| **Reporting Having Been Pregnant** | *n* of sample = 644 | *N* of population = 4,728,220 | |
| **Reporting Never Having Been Pregnant** | *n* of sample = 306 | *N* of Population = 2,757,061 | |

The race/ethnicity variables coded as binary factors are shown on rows 6-8. Of those three binary variables, only one produced a significant odds ratio at 3.288, $p < 0.0001$, showing that Non-Hispanic Black women were 3.288 times more likely to report having been pregnant at some time in the past before the NHANES interview than their Non-Hispanic White counterparts in the data set.

Finally, obesity and smoking covariates were statistically insignificant, suggesting that in this sample they were not important in determining whether a woman was ever pregnant.

### 3.6. Concordance and Fit of Regression Models

An indication of the strength, the goodness of fit, of a logistic regression model can be assessed by the concordance statistics which are reported in Table 3. In the top section, the concordance statistics are reported on the un-numbered rows 3-5 for the global regression model without any covariates. The first step in calculating those concordance measures is to pair each "event" — reporting at least one pregnancy prior to the NHANES interview with each "non-event"reporting not having been pregnant at any time prior to the survey.

As shown at the bottom of column 1 in Table 3, the number of women who reported having been pregnant was 644, and the number of women who reported never having been pregnant was 306. Therefore, this dataset produced 197,064 (= 644 * 306) pairs. The model computes a probability for each element of every pair. If the probability of the event is higher than that of the non-event — e.g. 0.9 for the event and 0.5 for the non-event — the pair is said to be "concordant". If the probability is lower for the event than the non-event (e.g., 0.7, 0.8), the pair is "discordant". If the probabilities for each part of the pair is the same (e.g., 0.6, 0.6), the pair is tied. In rows 3-5 in the top portion of Table 3, the concordance percentages for concordant, discordant, and tied pairs are given. Those percentages, of course, are computed by dividing the number of pairs in each of the respective categories by the total number of pairs. Of the three measures, the best indicator of the strength of the model is the percentage of concordant pairs. The greater that percentage, the stronger the model.

The percentage of concordant pairs in the global model at 27.1% on row 2 in Table 3 is relatively unimpressive by comparison with the discordant and tied percentages totalling 100% minus 27.1% at 72.9%. This result suggests that the model is not a very good fit as an explanation of the potential relation between getting an HPV shot reducing the likelihood of becoming pregnant. However, when the covariates known or suspected of being correlated with the likelihood of either getting the HPV shot on the one hand or becoming pregnant on the other are included (and adjusted for), the percentage of concordant pairs for the richer explanatory model gives a concordance at 83.8% as seen on the three rows near the bottom of Table 3.

## 4. Discussion

In June 2018, an earlier version of this analysis was published in a peer-reviewed journal (DeLong, 2018). Over a year later in December 2019, the publisher issued a statement retracting the article. The publisher — supposedly having been "alerted to concerns about the scientific validity of the study", allegedly "sought advice on the methodology, analysis and interpretation from a number of experts in the field" (Taylor & Francis Online, 2019). My present publisher, however, expressed wonderment at why the editor of a prestige journal such as the *Journal of Toxicology & Environmental Health (Part A)* would admit by implication, at least, *not having sought expert peer reviews before publishing the first version*? Also, where is the evidence that the group of "experts" supposedly called upon after the paper was published were actually better informed than anyone who was responsible for acceptance of the paper in the first place? And, more importantly, if what the publisher claims is true, why are the reviewers cloaked in anonymity? The fact is that none of the reviewers were named, so it was, and remains, impossible to check their credentials. The publisher continued: "All of the post-publication reports we received described serious flaws in the statistical analysis and interpretation of the data in this paper." In fact, four reports were reported to me: three recommended retraction, but one said the issues raised in my paper should be publicly debated. The reviewers who recommended retraction did not point to any factual errors; all of the alleged "serious flaws" were grounded in opinions voiced by the un-named "expert" critics. Their complaints were subjective, debatable, and on the whole easily refuted by reference to the relevant facts of my prior published analysis although I have specifically addressed a couple of the complaints here in this version.

One prominent complaint was the absence of a covariate measure pertaining to the use of birth control. If women who received the HPV shot were more likely than non-recipients to use birth control that factor alone might account for the reduction in pregnancies reported by HPV shot recipients. One post-publication reviewer asked why the use of contraception was not included in the analysis even though the relevant data, according to that person, are obtained in the NHANES interviews. The current version of the analyses reported here excludes women who either could not conceive or appeared to actively be using birth control. The survey includes three questions on birth control, covering the use of birth control pills, condoms, and injectables as well as a question on whether a woman ever had a hysterectomy. If a woman reported she had a hysterectomy before ever conceiving or if she reported she was using birth control pills, condoms, or injectables at the time of the survey, and she had not conceived before the interview, I considered her either unable to conceive or actively avoiding pregnancy. In the data reported here, I dropped all such respondents from any analysis reported.

Other criticisms were based on results obtained in another study by Shibata and Kataoka (2019). They claimed that overall birth rates have not fallen in countries such as Australia, Italy, France and the United Kingdom, where

uptake of the HPV vaccine is at least 70% of the eligible population. As detailed in my published response to Shibata and Kataoka (DeLong, 2019), a country's overall birthrate is a clumsy measure of any possible effect of one or more HPV vaccinations: Most women over the age of 30 in the study at issue had not received the HPV vaccine, yet all women aged 15 to 44 were included in the calculations by Shibata and Kataoka. If there was a causal relationship in their relevant data it was likely to be concealed behind *a lot of irrelevant data*. A more telling measure, therefore, would be the fertility rate among the younger women. European countries that have instituted rigorous HPV vaccination programs have experienced dramatic decreases in fertility rates among such younger women. Between 2009 and 2018, fertility rates among women aged 25 to 29 fell 12.0% in the United Kingdom, 13.3% in both France and Italy, 18.7% in Italy, and 23.9% in Norway (Eurostat, 2020). Over the same time period in Romania, which suspended its school-based HPV vaccination program due to lack of interest (Sheikh et al., 2018), the fertility rate among women aged 25 to 29 increased 6.1 percent.

Shibata and Kataoka (2019) also argued that the use of the most reliable form of birth control, long-acting reversible contraceptives (LARCs), increased among women between 2006 and 2013. Perhaps birth rates were lower among HPV shot recipients because those who received the vaccine were using more efficient methods of birth control. Sundaram et al. (2017) found that overall contraceptive failure rates declined between 2002 and 2010 from 12% to 10%. However, there is no *a priori* reason to believe a decline in contraceptive failure rates would affect HPV shot recipients differently from non-recipients. Note that a falling failure rate in birth control is also consistent with females being less fertile. If a sexually active female using a particular method of birth control does not conceive, the individual might credit her birth control with preventing pregnancy when in fact she is simply less able to become pregnant. Moreover, Kavenaugh et al. (2015) found that the primary users of LARCs are women who have already given birth. Nulliparous women are significantly less likely to use LARCs than women who have children.

One post-publication reviewer suggested secular societal trends such as delayed marriage and delayed child-bearing could influence birthrates. To determine whether time trend variables could have changed the outcome in my former analysis, I added five time variables — one for each of the two-year cycles in the study less one (2007-2008) used as the baseline — to the global logistic model presented above on page 133. The *F*-test returned a value of 2.06 ($p > 0.0787$), suggesting that the contribution of the additional variables was not statistically significant.

One common whipping post for researchers who refer to correlations in any part of an analysis aiming to assess possible causal relations is to remind the castigated person of the trite but true proposition that regression analysis cannot be used as a sole test, or singular determinant, of causality. Regressions directly demonstrate statistical associations, but they are, if at all, only indirectly related to causation. Causation demands correlation between causes and effects but the reverse, as shown in Figure 2 above, is not assured. A significant correlation does not assure a causal relationship. To create an argument for causation, more than just correlation, is required.

Although the analysis presented here shows a relationship between vaccine injection and an observed lower probability of ever being pregnant for females aged 25 to 29, the conclusion that HPV vaccination may be the cause, or part of the cause, for that observed reduction in the probability of a subsequent pregnancy cannot be made on the basis of the observed correlations alone. However, as noted in the opening of this version of my paper, other arguments, theories, mathematical proofs, and empirical results can be brought to bear in ways that support the hunch that HPV viruses 16 and 18, some parts of which are included in all HPV vaccinations, do seem to be causally related not only to cancers in women of child-bearing age, but also to reduced fertility in both males and females, and to premature ovarian failure in women. Combined with the empirical findings of my formerly published work — which I believe was unfairly retracted by the publisher without sufficient justification — it certainly appears that the probability of becoming pregnant decreases for women who have received one or more HPV vaccinations. While this result does not guarantee any particular outcome for any single recipient of any HPV vaccination, it does suggest that the components and interactions of components in the HPV vaccines on the market deserve closer critical scrutiny.

Another consideration is that over the coming years, the still on-going COVID-19 pandemic could swamp the negative effects of HPV shots on fertility as suggested in the results of the present study. Both the disease itself and the shots engineered to combat it could negatively affect fertility. We do not know whether the manufactured

SARS-CoV-2 virus (Fleming, 2021; R. F. Kennedy Jr., 2021) was designed to impact fertility in a negative way, but evidence suggests it does. Dotan et al. (2021) examined the SARS-CoV-2 infection and found the spike glycoprotein shared characteristics of human proteins that could lead an infected woman to create autoantibodies that could disrupt her reproductive system. We may not know the effects of the COVID "vaccines" on fertility until years or even decades from now. However, analysis by Shimabukuro et al. (2021) revealed that of the 127 women in their study who received an mRNA shot in during their first or second trimesters 104 (82%) experienced unexplained, so-called "spontaneous" abortions. The observed rate of such unexplained losses in the Dotan et al. study was about 2.7 times greater than the rate typical for early pregnancies (first 20 weeks) estimated at 30% (Hertzpicciotto & Samuels, 1988; Alves & Rapp, 2021) with losses after week 28 estimated at about 3.3% (French & Bierman, 1962; Jarvis, 2016; Chen et al., 2020) — a rate 24.8 times greater for women getting the mRNA shot. Does such a shot interfere with the delicate communications underway during embryonic development as Fleming's (2021) documentation suggests? He argues that the spike protein in mRNA specifications is the essence of the SARS-CoV-2 bioweapon. He observed that the "best weapon doesn't kill people; it devastates and demoralizes them." He goes on to say that such a weapon "diminishes the lifestyles of the enemy, reducing the security of life as the enemy knows it…" (p. 101) What better way to disrupt enemies lives over the long-haul than to take away their ability to reproduce?

## 5. Conclusion

Birth rates in the United States have recently fallen. Analysis presented here suggests a statistical association between a woman receiving one or more HPV vaccinations and the likelihood of her experiencing a lowered probability of subsequently becoming pregnant. Several studies link the HPV shot to autoimmune disorders such as premature ovarian failure. Also, independent fertility-antifertility research shows that the viruses targeted in the HPV vaccinations are capable independently not only of causing certain cancers but also of causing infertility in both males and females. In addition, the adjuvants in the HPV shots consisting of aluminum salts and the solubilizing agent polysorbate 80, are all known to be causally associated with autoimmune disorders, are almost certainly interfering with the delicate and highly articulated processes of meiosis, fertilization, migration and implantation, along with the many mitosis events that must succeed in order for a normal pregnancy to occur. Progress from impregnation to a normal live birth requires many additional mitosis events and may, as Lee (2021) has observed, be impacted by non-biodegradable components in HPV vaccines such as Gardasil9. Further study into the targeted antigens in HPV vaccines, their adjuvants, and the interactions of them with observed cases of autoimmunity, failed pregnancies, loss of fertility, and the like, merit independent investigation by persons who do not have vested interests in the outcomes of their research. Such investigations are also needed to determine the long-term effects of COVID shots on fertility.

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