HPV VACCINES, 7 QUESTIONS THAT YOU NEED ANSWERED

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Not long ago (in medical years), we were still trying to discover the cause of cervical cancer. Today, not only do we know that cause to be persistent human papillomavirus (HPV) infection, but we have two vaccines at our disposal to prevent the primary oncogenic strains of the virus.

We’ve come a long way.

The availability of two vaccines raises questions, however. What kind of data do we have on the bivalent (Cervarix, GlaxoSmithKline) and quadrivalent (Gardasil, Merck) vaccines so far? Is one of them clearly superior to the other? If not, what population is each vaccine best suited for—and how do we counsel patients about their options?

To address these and other questions, OBG MANAGEMENT Contributing Editor Neal M. Lonky, MD, MPH, assembled a panel of physicians who have expertise in cervical disease detection and prevention and asked them to sift the data that have accumulated thus far. In the discussion that follows, they touch on long-term efficacy, the likely impact of the vaccines on cervical cancer screening, and other aspects of disease prevention in the era of HPV vaccination.

1 How were the vaccines developed?

Neal M. Lonky, MD, MPH: What should clinicians know about the development, function, and mechanism of action of the two HPV vaccines?

Warner K. Huh, MD: The bivalent and quadrivalent vaccines are both excellent products, and their respective Phase-3 trials demonstrate that they provide impressive protection against HPV, particularly among women who test negative (by polymerase chain reaction) for the specific HPV types contained within the vaccines.1-3

Cervarix protects against HPV types 16 and 18, whereas Gardasil is effective against HPV types 6, 11, 16, and 18.

Dr. Lonky: Do the vaccines function similarly?

Diane M. Harper, MD, MS, MPH: Yes. Both stimulate an immediate antibody response in the woman who is not infected with the relevant virus and are effective in preventing cervical intraepithelial neoplasia grade 2 and...
higher (CIN 2+), as well as persistent infection, caused by vaccine-related and cross-protected HPV types. The quality of the antibody response is best for HPV 16 for both vaccines. The quality of the antibody response for HPV 6, 11, and 18 for Gardasil is much poorer than its response for HPV 16. Cervarix induces an equally high and sustained antibody response for HPV 18 as for HPV 16.

Juan C. Felix, MD: Both vaccines are based on the same virus-like particles (VLP). The functionality of the vaccines is, therefore, mainly dependent on the dosage of VLP and the adjuvant used. Gardasil uses a proprietary aluminum sulfate adjuvant, whereas Cervarix uses aluminum hydroxide and monophosphoryl lipid A.

Karen K. Smith-McCune, MD, PhD: Both adjuvants have an extensive track record of safety and efficacy in other vaccines. Because they have different structures, however, they may have varying effects on many components of the immune response elicited by the L1 antigens.

Dr. Harper: Both adjuvants contain aluminum, which has so far proved to be safe despite the newly established association between high aluminum intake and Alzheimer’s disease.

Dr. Lonky: Were there any notable challenges in developing the vaccines?

Dr. Harper: It was difficult to formulate the appropriate dosages of VLP in Gardasil. Higher dosages of HPV 11 and 16 were needed to prevent cross-inhibition by HPV 6 and 18. As a result, the antigenic protein component of Gardasil that is necessary to effect an immunologic antibody response is high, at 120 µg. In Cervarix, the antigenic VLP load is 20 µg each for HPV 16 and 18.

Dr. Lonky: What is the significance of the different VLP loads?

Dr. Harper: Side effects, such as autoimmune neurologic demyelination, albeit rare, have been associated with a higher antigenic protein load. Multiple reports of autoimmune demyelinating diseases—including paralysis, blindness, and death—have been published by neurologists in regard to Gardasil. Others have shown that young girls are more at risk than young boys for these neurologic side effects.

Dr. Felix: Some data suggest that the two vaccine formulations interact differently with the human immune system. In a head-to-head trial funded by GlaxoSmithKline, Cervarix produced higher total and neutralizing antibody titers than Gardasil did.

Although higher immunogenicity is generally thought to be beneficial, the ultimate determinant of a vaccine’s success is its efficacy—and duration of that efficacy—in clinical trials and follow-up of vaccinated populations. So far, Cervarix has demonstrated efficacy through 8.4 years in its follow-up cohort. Similarly, Gardasil has proved to be effective after 5 years of follow-up, with no
incident cases of cervical cancer reported in the vaccinated arm.9

Does either vaccine offer “extra” immunity?

Dr. Lonky: What is the potential for overlapping immunity to other high-risk viral types with these vaccines?

Dr. Harper: It is quite clear from pivotal trials of both vaccines that Gardasil produces efficacy of 46% against persistent infection caused by HPV 31. Data from the pivotal Phase-3 trial of Gardasil also show that it offers no protection against persistent infection with HPV 45, an important cause of adenocarcinoma.10 In contrast, Cervarix demonstrates substantial efficacy against both persistent infection and CIN 2+ disease caused by HPV 31, 33, and 45.3

These findings mean that Cervarix is 91% effective against HPV types that cause adenocarcinoma and 83% effective overall against squamous cell carcinoma. Compare that with Gardasil, which is 78% effective overall against HPV types that cause adenocarcinoma and 73% effective against HPV types that cause squamous cell carcinoma.

The immune titers tell a supportive story. After vaccination with Gardasil, the antibody titer immediately declines for HPV 6, 11, and 18, reaching the baseline for natural infection within 18 months.7 HPV 18 shows continued, significant loss of seropositivity over time, and antibody titers for HPV 6 and 11 also decline. In the monovalent HPV 16 pre-Gardasil experimental vaccine, 14% of women no longer had measurable titers to HPV 16 after 8.5 years.11

After vaccination with Cervarix, antibody titers for HPV 16 and 18 remain more than seven times and more than four times higher, respectively, than natural infection titers for 8.4 years, with no loss of measurable antibody titer for either type. The antibody titers for HPV 31, 33, and 45 remain substantially higher than natural infection titers for at least 6.4 years. These titers correlate with the vaccine’s very high efficacy against CIN 2+ lesions caused by HPV 16, 18, 31, 33, and 45.

In other words, Cervarix generates an immune response (and efficacy) that indicates robust protection against five of the most common oncogenic HPV types, providing maximal protection against nearly 85% of all cervical cancers. Gardasil protects against 74% of all cervical cancers overall.12 This makes Cervarix the superior cervical cancer vaccine.

Gardasil is the superior vaccine against genital warts, although the duration of its protection is uncertain.

Dr. Huh: I’d just like to point out that there are no head-to-head trials comparing the vaccines in terms of efficacy. Antibody titers are higher with Cervarix than with Gardasil, as you noted, and it may be that, over time, the higher titers are more durable with Cervarix. However, we have yet to fully correlate clinical efficacy with antibody titers. In other words, immunogenicity does not equal clinical efficacy.

Dr. Felix: The data for Gardasil are particularly interesting because there have been no incident HPV-18 lesions detected despite the absence of detectable HPV-18 antibody titers in more than 20% of vaccinated women as soon as 2 years after immunization.9 These data strongly suggest that it is not antibody titer alone that grants protection against HPV-induced lesions of the cervix.

Dr. Harper: This speaks to the difficulty of running a trial to ensure both enough participants and a sufficient attack rate of HPV 18 to cause new lesions to be detected in...
vaccinated women. In the relevant trial, there were only 112 vaccinated women—not nearly enough women to overcome the very low attack rate of HPV 18 in the trial population—and they were followed for 5 years.\(^9\) We cannot be sure that the lack of incident HPV-18 lesions in the vaccinated women is the result of efficacy.

**Dr. Felix:** As for overlapping immunity to HPV types not included in the vaccines, it has been described for both Cervarix and Gardasil. In the case of Cervarix, the manufacturer demonstrated unexpectedly high rates of protection against all CIN 2+ and CIN 3+ lesions—70% and 87%, respectively. These rates were too high to be explained by protection against types 16, 18, 31, and 45 alone. It is possible, therefore, that Cervarix may protect against other high-risk HPV types.\(^{13}\)

Gardasil has proved to be effective against HPV types 31, 33, 52, and others.\(^{10}\) When total protection against CIN 2+ and CIN 3+ lesions is examined from Phase-3 trials of the vaccine, however, the rates are only 42% and 43%, respectively. These data are difficult to interpret because HPV 16 and 18 together are thought to account for 70% of CIN 3. Some reassurance can be gained from the fact that the number of incident cases of CIN 2+ and CIN 3+ caused by HPV 16 and 18 in the vaccinated group in the Gardasil trial was identical to the number seen in the Cervarix trial.\(^3,10\) The reason for the discrepancy in total number of cases of CIN 2+ and CIN 3+ between the two trials—and, therefore, between the two vaccines—cannot be explained by cross-protection alone and is probably attributable to differences in study populations. The Gardasil trial had a higher baseline prevalence of HPV 16 and 18 (9% and 4%, respectively) than the Cervarix trial did (5% and 2%, respectively), a fact that may be explained by the different demographics of their respective populations.\(^2,14\)

Ultimately, it is hazardous to compare trials, particularly when they are conducted in significantly different populations. On this issue, I concur with the World Health Organization (WHO), which recommended that such comparisons be avoided in the determination of which type of HPV vaccine to recommend.\(^{15}\)

**Dr. Huh:** I agree that it would be inappropriate to make cross-trial comparisons, given differences in the way the trials were designed and conducted. To draw conclusions about clinical efficacy of these two excellent vaccines, based on a comparison of their trials, is completely unscientific. Only a true head-to-head study that has efficacy as its endpoint can tell us which vaccine is superior—and such a trial would require thousands (if not tens of thousands) of subjects and a considerable amount of time to complete. In my opinion, such a study would be counterproductive to our goal of vaccination.

**Dr. Harper:** I disagree. The whole purpose of this roundtable is to compare vaccines. It is not “unscientific” to compare the trials.

**Dr. Huh:** On the contrary—it is completely inappropriate to directly compare the Phase-3 clinical trials from Merck and GlaxoSmithKline. One can speculate about the differences between them, but any clinical trialist knows that a direct, scientific comparison cannot be made. Only a real head-to-head study powered for efficacy can do this.

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—Juan C. Felix, MD

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3 Is one vaccine more effective than the other?

**Dr. Lonky:** How do the vaccines compare in terms of efficacy?

**Dr. Smith-McCune:** In discussing efficacy, I think we should focus on CIN 3 because it is the immediate surrogate for cancer, whereas CIN 2 lesions can be transient in younger women. I think it is also important to focus on outcomes regardless of the HPV types associated with the lesions. This approach is more clinically relevant, as we don’t perform HPV typing of lesions in clinical practice. Nor do we manage lesions differently depending on the HPV type in the lesion.

That said, it is difficult to compare efficacy of the vaccines for several reasons, a few of which we have already discussed. For example, the bivalent and quadrivalent vaccines were studied in separate randomized trials. Although the study populations were similar, they were not identical. Women in
I discern 8 barriers to HPV vaccination

Despite solid evidence that the quadrivalent (Gardasil) HPV vaccine and the bivalent (Cervarix) HPV vaccine protect against cervical cancer, only about one fifth of the female population between 11 and 26 years of age has received the full series of Gardasil since it won FDA approval in 2006. Barriers to vaccination are not financial alone, as the vaccination rate is similarly low among women who have health insurance.

Why isn’t the vaccination rate higher? I see eight barriers to full implementation:

- **Economic disparities.** Each vaccine costs roughly $400 (national average) for the full series of injections. Although women who do not get Pap screening are most likely to benefit from the vaccines, they usually cannot afford them. Federal childhood immunization programs cover teens and young women until 18 years of age in most states, and until 21 years in a few. That leaves most women who seek vaccination from gynecologists without coverage.

- **Fear.** Pain at the injection site, syncope, and a slightly elevated incidence of thromboembolism are the adverse events most commonly associated with HPV vaccination in the literature. In the life cycle of a vaccine, reports of sudden death or neurologic injury (Guillain-Barré syndrome) occur in the early years, but are reported at a rate lower than 2 cases in every 10,000 women. Nevertheless, such events may create fear about undergoing immunization.

- **Long latency period.** Because the outcome of cancer prevention won’t become apparent for 20 to 40 years following vaccination, the need for immunization may seem less than urgent.

- **Cultural and religious beliefs.** Because carcinoenic HPV strains are sexually transmitted, some families may associate vaccination with the promotion of sexual activity. Even in states that mandate vaccination, the courts have upheld a parent’s right to refuse vaccination on these grounds.

- **The premarket push.** Aggressive promotion of vaccination by both manufacturers and a push by advocates for legislation to mandate the vaccine prior to completion of Phase-3 trials and gathering of robust safety data may have diminished trust in the vaccine and reduced its acceptance.

- **Lack of legislation.** In states that do not consider HPV vaccination to be a necessary public health intervention, the lack of mandates and funding reduce the vaccination rate. In addition, some legislators have been more active advocates of vaccination than others.

- **Reduced involvement of the ObGyn.** ObGyns don’t routinely vaccinate patients; pediatricians do. Young women are slipping through the cracks because the conventional ObGyn practice does not have a vaccination program that ensures payment, reimbursement, and completion of the vaccine series. Many ObGyn practices are reluctant to institute such a program because the profit margin is small, there are associated risks, and the time required to counsel the patient and for follow-up is extensive.

- **Failure to complete the series.** Some women do not complete the full vaccine series, owing to cost or side effects, or both. Solid evidence that a single dose could be as protective as the full series would be compelling. A single-dose vaccine would also be less expensive.

The principal danger of a low vaccination rate is the loss of insurance coverage for immunization against HPV. On one hand, payers may begin to ask whether coverage is justified when so few girls and women are vaccinated, leaving the payer with two burdens: the expense of vaccination and the expense of conventional screening programs and treatment, although the costs of treatment would be reduced with vaccination. On the other hand, Gardasil’s protection against genital warts may provide incentive for payers to cover or discount the vaccine because of the reduction in the need to diagnose, triage, and treat condyloma.

Ultimately, HPV vaccination may become another optional intervention that is paid for by the individual, despite evidence in girls and women that cervical cancer can be prevented.

—NEAL M. LONKY, MD, MPH
3% of women were from the Asian Pacific, versus 34% in the Cervarix trials, and so on.\textsuperscript{3,16}

The trials also had different protocols for referral to colposcopy, which would affect disease detection. And the length of follow-up differs between trials.\textsuperscript{3,19}

**Dr. Lonky:** Can we draw any conclusions about efficacy?

**Dr. Smith-McCune:** Yes. The trials defined outcomes in several populations of participants. In addition to the overall population (called the “intention-to-treat population” in the Gardasil trials and the “total vaccinated cohort” in the Cervarix trials), the trials defined a subpopulation of women naïve to oncogenic HPV types to gain information about the likely impact of vaccinating girls before the onset of sexual activity. The definitions of these “naïve” populations were slightly different, mainly in the number of HPV types tested, so again, some caution needs to be exercised in making comparisons.

End-of-trial data in the naïve population show a 43% reduction in CIN 3 lesions for Gardasil and 87% for Cervarix (for CIN 3 or worse). By inference, we can tell the sexually naïve patient that vaccination with either vaccine will provide significant protection against CIN 3 lesions, likely to result in significant protection against cervical cancer over time.

We can gather some estimates of efficacy in sexually non-naïve women by looking at results from all trial participants. Gardasil reduced overall CIN 3 lesions by 16% overall; Cervarix reduced CIN 3 or worse by 33%. When counseling an individual patient, if she has had a similarly low number of lifetime sexual partners (e.g., the median number in the Gardasil trials was 2), these results provide an estimate of her likely protection against CIN 3 with vaccination.

Common excisional treatments for cervical dysplasia are known to be associated with adverse perinatal outcomes.\textsuperscript{17} The ability to reduce the need for these treatments is an important outcome of vaccination. In the HPV-naïve populations, vaccination reduced definitive cervical therapies or excisions by 42% (Gardasil) and 69% (Cervarix). These figures are useful in counseling virginal patients about the long-term benefits of vaccination.

For sexually active patients 26 years and younger, HPV vaccination significantly reduced definitive cervical therapy or excisions by 23% (Gardasil) and 25% (Cervarix). Again, these figures are most applicable for counseling patients who have had relatively few lifetime sexual partners. So the exact extent of protection is likely to vary by the patient’s total number of lifetime sexual partners.

I expect that we will see more data on the effects of vaccination stratified by the number of lifetime sexual partners, because that information would be very useful in counseling individual sexually active women.

**Dr. Harper:** Both vaccines reduce the rate of abnormal Pap tests by 10% regardless of HPV type in that population of women.\textsuperscript{3,18}

### Are the two vaccines safe?

**Dr. Lonky:** What about safety of the vaccines? What do we know?

**Dr. Felix:** The safety profiles seen in clinical trials of both vaccines are very similar and consist almost entirely of nonserious adverse events.\textsuperscript{2,9} In the United States, a greater number of Gardasil doses has been administered, owing to its earlier development. As of January 1, 2010, more than 28 million doses had been distributed, and numerous major events had been recorded in the Vaccine Adverse Event Reporting System (VAERS). Of 15,829 adverse events reported, only 8% were considered serious by the CDC. CDC investigation, by expert panels, of all serious adverse events found no evidence linking Gardasil to any of them, including Guillain-Barré syndrome, blood clots, and death.\textsuperscript{19}

**Dr. Huh:** A few other points to consider:
- The reporting rate for Gardasil is triple that for all other vaccines combined
- Because VAERS is a passive reporting system, under-reporting is distinctly possible
- Post-licensure safety surveillance is still underway
- Both products have pregnancy registries.

**Dr. Harper:** The current postmarketing
commitment between Merck and the FDA is to recognize a rate of serious adverse events that exceeds 2 cases in every 10,000 women in a cohort of 44,000 women who have received all three doses of Gardasil. Although autoimmune neurologic sequelae have occurred after Gardasil administration, regulatory authorities are not required to evaluate these reactions, such as Guillain-Barré syndrome, because the frequency is lower than the agreed-upon threshold. Nevertheless, adverse events could be life threatening to some girls.

Any risk of death—even if it is lower than the agreed-upon threshold—should be presented to women as a possible risk of vaccination with Gardasil. In the United States, the same women could choose a lifetime of Pap screening and be afforded the same protection against cervical cancer as they would get from vaccination.

Is quadrivalent better than bivalent?

Dr. Lonky: Why would a clinician choose a bivalent vaccine when the quadrivalent vaccine protects not only against carcinogenic types 16 and 18, but also against HPV-associated genital warts?

Dr. Harper: A smart clinician would ask the patient what she values. The physician is obligated to present the evidence and let her choose!

Dr. Lonky: What does the evidence suggest?

Dr. Felix: A clear recommendation between Cervarix and Gardasil is very difficult to make at this time, for the reasons already stated. Both vaccines provide 98% protection against HPV 16 and 18 for the prevention of CIN 2+ lesions. As we have discussed, both vaccines also provide protection against high-risk strains of HPV other than types 16 and 18.

In the Cervarix trial, there was an overall reduction of all CIN 2+ lesions that was higher as a percentage of total lesions than the reduction seen in the Gardasil trial. However, unlike Cervarix, Gardasil significantly reduced the rate of vaginal intraepithelial neoplasia (VaIN) and vulvar intraepithelial neoplasia (VIN).

Dr. Harper: GlaxoSmithKline is analyzing its data on vulvar and vaginal protection, and it is likely that Cervarix will demonstrate some efficacy in this regard, too. But the economic burden of noncervical cancers is estimated to be only 8% of the economic burden of all HPV-related diseases. The prevention of cervical cancer is the dominant clinical and economic force for vaccination.

Dr. Felix: Clearly, the protection against genital warts demonstrated in the Gardasil trial will not be realized with Cervarix, as it does not offer immunization or cross-protection against HPV 6 or 11. Gardasil’s protection against HPV 6 and 11 prompted FDA approval of the vaccine for boys and men.

According to the WHO, when counseling girls and women about the HPV vaccine, the clinician should weigh the possible value of a deep reduction in total CIN 2+ lesions provided by Cervarix against the reduction in VaIN, VIN, and genital warts provided by Gardasil. Boys and men will see clinically proven benefits only from Gardasil for the prevention of external genital warts. Other benefits are strictly theoretical.

Dr. Harper: Vaccine protection must last at least 15 years to reduce the rate of cervical cancer. Otherwise, the development of cervical cancer will only be postponed, if boosters are not implemented.

It is now widely recognized that Cervarix induces high antibody titers, offering 100% efficacy even after 8.4 years, making it very likely that the protection it provides will continue for at least 15 years. It is also widely acknowledged by immunologists that Gardasil-induced titers for HPV 6, 11, and 18 are much shorter-lived, so protection is likely to wane 5 to 10 years after vaccination.

That means that Gardasil provides excellent protection against one cancer-causing type of HPV. In addition, it protects against genital warts caused by HPV types 6 and 11 for at least 5 years. In comparison, Cervarix protects against five cancer-causing types of HPV, thereby preventing about 90% of cervical cancers, and is likely to remain effective for at least 15 years.

There are 10 times as many women...
who have an abnormal Pap test as there are women who have genital warts, so one would think that Cervarix would be the vaccine of choice in preventing the life-threatening disease of cervical cancer.

**Dr. Smith-McCune:** I would agree that the choice should be discussed with patients—and with parents. If the objective is primarily to protect against cervical cancer precursors, then the bivalent vaccine may be the better choice, with the caveat that we can’t really compare the results from the vaccine trials for reasons discussed earlier, and there are no data from a randomized head-to-head trial comparing the two vaccines. If the decision involves a desire to reduce genital warts or vulvar and vaginal dysplasia, then the quadrivalent vaccine would be the better choice.

### What impact do the vaccines have on screening?

**Dr. Lonky:** Do the vaccines have varying effects on our need to screen for, triage, and treat cervical cancer precursors?

**Dr. Harper:** The Pap smear has reduced the rate of cervical cancer in the United States by 75%—that rate is now at an all-time low of 8 cases for every 100,000 women. But the Pap smear is not perfect; there is a 30% false-negative rate among women who develop cervical cancer, and a large false-positive rate that involves referral to colposcopy for minimally abnormal cytology reports. And when CIN 2+ disease is detected, treatment is not without risk. Surgery increases the risk of reproductive morbidity in future pregnancies. Having protection against this outcome could be tremendously valuable for some women.

Compare the HPV vaccine, which has probable benefit but also the potential for serious adverse events, including demyelinating diseases that cause blindness, paralysis, and death in a small number of recipients.

If women were to choose to be vaccinated with Gardasil and forgo further Pap screening, the rate of cervical cancer in the United States would rise from 8 to 14 cases for every 100,000 women. If they were to choose Cervarix instead, with no further Pap screening, the rate would rise from 8 to 9.5 cases for every 100,000 women.

If women were to choose both HPV vaccination and continued Pap screening, the rate of cervical cancer still would not decline from its current level of 8 cases for every 100,000 women. Instead, the benefit would be that fewer women have abnormal Pap tests, and fewer women would need to be treated for CIN 2+ disease.

Women and physicians must understand these facts. A woman who chooses to be vaccinated may gain individual protection, but the overall rate of cervical cancer will not be affected.

**Dr. Huh:** Regardless of the HPV vaccine selected, we need to seriously rethink how we screen women in the United States. One could easily argue that the combination of the vaccine and continued screening is too expensive. It might be wise to consider lengthening the screening interval—and, perhaps, further delaying initial screening to 25 years of age—to make cervical cancer prevention with both modalities more cost-effective. The most important thing to recognize is that women still need to be screened, even if they have been vaccinated.

As more women are vaccinated, we expect to see a decline in the prevalence of CIN 2+ and CIN 3+ lesions, and this will ultimately weaken the positive predictive value of cytology. Perhaps it is time to consider the HPV test as a primary screen, with triage to cytology in women who test HPV-positive.

**Dr. Smith-McCune:** As we accumulate data over time about the effects of vaccination on the rates of CIN 3 and cancer, modeling will be helpful in determining the best screening algorithm for women who have been vaccinated against HPV.

It is important to remember that approximately 50% of women who are given a diagnosis of cervical cancer in the United States have never been screened. It is vital that we continue to reach out to the under-screened population and focus vaccination efforts on populations of girls who are likely to have limited access to care in the future.

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Can we vaccinate every woman?

Dr. Lonky: Is universal vaccination of women achievable for either vaccine?

Dr. Felix: Universal vaccination against HPV would be achievable only via school mandates. Without them, vaccination will not approach the 80% threshold needed to produce herd immunity.

Despite the clear benefit of such mandates to the general population—particularly the medically under-served—the issue has become a political football. As a result, school mandates will probably never be realized.

Dr. Harper: I don’t believe it is ethical to mandate vaccination of all girls and women. It is a choice that women and parents, in conversation with their physicians and daughters, must make when considering how to be protected against cervical cancer. Herd immunity is a moot point because we are only vaccinating girls (50% of the population) and can never reach the theoretical 70% threshold for herd immunity to be apparent.

Dr. Lonky: Is the availability of two vaccines a boon or a hindrance?

Dr. Smith-McCune: I think it is always a good thing to have choices in medicine.

Dr. Huh: I see the availability of two vaccines as a boon. That availability means that two companies are now putting forth consistent educational messages about the importance of vaccination and, I hope, stimulating competition that will reduce the overall cost of the vaccine series. Having two vaccines can only promote awareness, access, and greater appreciation of the considerable protection these two vaccines provide.

References