COMMUNITY HIV/AIDS CHAMP MOBILIZATION PROJECT
HHS WATCH

January 2008 – **VACCINE REVIEW**
- **It's Getting To Be A Bad Habit**
- **The Vaccine We Have Already: More Trials and Tribulations**

VACCINE REVIEW

It's Getting To Be A Bad Habit

by David Gilden

Note that the STEP Study data analyzed here come from a public meeting that took place November 7, 2007 under the auspices of the HIV Vaccine Trials Network (HVTN) at the National Institutes of Health (NIH). The presentation slides are viewable at http://www.hvtn.org/science/1107.html.

The news that a medical prevention technique had failed, in this case involving an HIV vaccine, had a familiar ring to it. Worse than demonstrating that the vaccine was ineffective, researchers feared that the vaccine actually made people more likely to acquire HIV. This was the third occasion in 2007 for such a paradoxical conclusion. (See below for the Savvy microbicide and *HHSWatch* March 2007 and September 2007 on the microbicide cellulose sulfate).

The vaccine trial is the STEP Study, which was cosponsored by the HVTN and the vaccine developer, Merck & Co. Merck's vaccine (MrkAd5) is composed of a type 5 adenovirus (Ad5) genetically manipulated to produce three HIV proteins. The process also renders the virus nonreplicating.

Adenoviruses are common cold viruses ("adeno" as in "adenoid"). Adenoviruses stand out in their ability to infect a wide variety of human cells and provoke high-level persistent immune responses. Producing vast quantities in the lab is relatively easy. These qualities make adenoviruses highly advantageous as vaccine carriers, or vectors.

Adenoviruses also present a major disadvantage: A large fraction (30%-70%) of the human population has been exposed to common strains such as type 5. Previously exposed humans have attenuated or null responses to adenovirus vaccines (Bangari and Mittal. Current Gene Therapy, April 2006). Booster vaccinations containing the same strain as the initial vaccination are problematic, too. To strengthen the response, vaccines try to overwhelm prior adenovirus immunity by utilizing massive amounts of virus, 30 billion particles in the case of the Merck vaccine. (The vaccine is administered as one primary immunization and two booster shots given one and six months later.)

And then there is a worse problem. There is no roadmap to a successful HIV vaccine. HIV has developed a number of ways to evade the immune system, and the natural immune response is rarely effective. There is some hope that a vaccine-like intervention can trigger preventive immunity: Monkeys vaccinated with attenuated SIV, the simian version of HIV, can resist subsequent exposures to natural SIV. Either they do not become infected or their virus levels are very low. The monkeys do develop long-term infections with the attenuated strain, however. The attenuated strain sometimes reverts to a pathogenic one that causes the animals to develop AIDS (Stahl-Henning et al. Immunology Letters, June 1996).

Rushing to Trial Confuses the Judgment

Merck Vice President Mark Feinberg has been widely quoted as saying of the STEP Study, "I've never seen more complicated data to emerge from a study." That's more a comment on the lack of theoretical understanding than on the actual intricacy of the trial results.

In a 2004 commentary on HIV vaccine prospects, veteran researcher Ronald Desrosiers observed, "I believe that a renewed, organized and focused effort is

required to deal with [the] fundamental scientific obstacles. These problems are not likely to be overcome by repeated clinical testing of weak products that stand little chance of being effective" (Desrosiers. Nature Medicine, March 2004). Desrosiers was writing while the STEP Study was still in its planning stages. The trial's outcome has borne out his words.

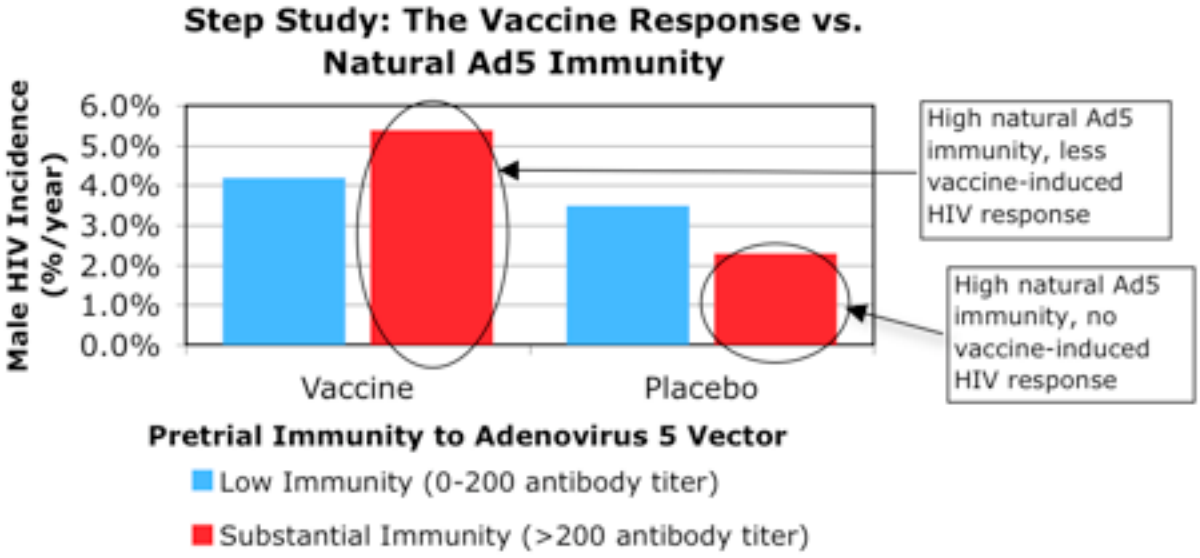
Merck's monkey experiments with adenovirus-based HIV vaccines were inconclusive. In particular, the vaccine showed no ability to prevent or control infection with SIV, which remains the best available stand-in for HIV (Casimiro et al. Journal of Virology, December 2005; McDermott et al. Journal of Virology, December 2005). The vaccine in the current human trial triggers a wider immune response than the one used for the SIV study, but there could be only small hope that this vaccine would be successful in humans.

The STEP Study is officially considered a Phase II study. Intermediate in the development process, a Phase II study should test mainly safety and immune responses to the vaccine. Phase II trials usually involve a few hundred volunteers, not enough to measure efficacy. The STEP Study is an exception: The trial enrolled 3,000 high-risk men and women in the Western Hemisphere and Australia. (The men were largely MSM, and many of the women were sex workers.) A companion 3,000-person trial began enrolling South African volunteers last January. Dubbed "test of concept" trials, these studies effectively represented a jump beyond their official status into Phase III, which provides direct data on effectiveness.

The STEP Study was supposed to last five years, but it was abruptly terminated in September, after following trial participants for six to 32 months. Investigators subsequently terminated the South African trial, too. At first, it merely looked like the Ad5 vaccine was ineffective: Among the trial population with low adenovirus 5 antibody levels, there were 24 new HIV infections in the 741 volunteers receiving the vaccine and 21 new HIV infections in the 762 volunteers receiving the placebo. There also was no evidence of lower HIV levels in the newly infected vaccine recipients. The vaccine failed on both counts.

But then it gets confusing. When the investigators looked at the male volunteers with substantial pretrial Ad5 antibodies, they found 21 new HIV infections in 392 men receiving the vaccine, whereas there were only nine new HIV infections in the 386 volunteers receiving the placebo. Ironically, the STEP Study originally did not permit persons with high-level immunity to adenovirus 5. The trial organizers decided to include them – and in the process doubled the size of the trial – when they realized from the Phase I that these people might well have some response to the vaccine (Duerr et al. Clinical Infectious Diseases, August 2006). Their experience is important to document given the great prevalence of the natural virus.

You can look at the data another way. The placebo recipients with high Ad5 immunity had a markedly lower HIV incidence than the rest of the trial participants (see figure on page 3). The placebo was just saline solution. Maybe exposure to natural adenovirus and the consequent immunity is somehow protective against HIV? Look again at the figure. The vaccine recipients with high Ad5 immunity had the highest rate of HIV. Their HIV incidence exceeded the rate in the vaccine recipients with low Ad5 immunity. But the high Ad5 immunity vaccinees were less likely to develop anti-HIV immune responses than the other vaccinees. The vaccine affected them less, and you would expect them to have a lower HIV incidence if the vaccine was somehow harmful.



There are monkey studies in which an SIV vaccine made the disease worse. The vaccine caused ineffective immune activation when the inoculated monkey was exposed to SIV. Activated immune cells are SIV's and HIV's favorite target, so the vaccine just made more food for the virus. Merck official Mark Feinberg conducted one of those studies five years ago when he was at Emory University (Strapans et al. PNAS, August 31, 2004). The idea has gained some currency that ineffective immune activation – due to the vaccine's Ad5 and/or HIV components – explains the results. Julie McElrath of the HVTN Laboratory reviewed this theory at the November 7 meeting. Immune cell activation was similar in all four subgroups of vaccine and placebo recipients. None offered substantially more "food for the virus."

Researchers have also proposed that differences between the populations with high and low Ad5 immunity explain the results. The men with high Ad5 immunity tended to be from Latin America, hence poorer and with less access to good diets and medical care. They were less often circumcised, too. You would expect these environmental differences to increase their HIV susceptibility. That does not appear to be the case given the placebo data. At any rate, about half the men in both Ad5 immunity groups had unprotected receptive anal sex, and 14% of both groups had STDs. These are the most direct indicators of HIV risk.

Throwing Dollars at the Data

The bottom line is that the vaccine did not work. The differences observed so far appear to be a statistical quirk: They do not make much sense, and they are too small to be considered statistically significant. McElrath promised a laundry list of follow-up studies and solicited ideas for more. Follow-up studies, involving smaller and smaller subgroups of trial participants, are not likely to reach meaningful conclusions. The ever-present danger is that if you keep running studies, some coincidental, misleading association will appear statistically significant. The iron laws of probability come into play here: The more coins you flip, the more the chance of an improbably long series of heads or tails.

The HVTN has now halted or delayed its trials involving adenovirus-based vaccines. First of these was the now cancelled South African MrkAd5 trial. It made little sense in the first place since the potential trial population almost entirely has strong Ad5 immunity from prior natural exposure. Also, the predominate HIV subtype in South Africa is subtype C,

whereas the vaccine is based on subtype B, the kind prevalent in the Western Hemisphere and Europe. The subtype B responses triggered by MrkAd5 are less frequently active against subtype C (Casimiro et al. 12th Retrovirus Conference, February 2005).

Haste has given way to caution. $100 million 8,500-person PAVE 100 was about to begin enrollment but has been put on hold for at least six months. PAVE 100 will test a combination pure DNA vaccine plus one booster immunization with an Ad5-based vaccine. This vaccination protocol is more promising than the one in the STEP Study. Merck has tested a similar protocol in monkeys. No monkey was protected against SIV infection, but vaccinated monkeys who became infected had viral levels 30-fold lower than non-vaccinated monkeys. These results were obtained under ideal conditions, and they don't provide much optimism for this particular vaccine strategy. Still, they do suggest that some vaccines will prove better than others. The trick will be to predict which are the promising vaccines and select only those for large-scale human testing.

Meanwhile, an actual Phase III trial, RV144, will continue as planned through next June. It has enrolled 16,000 low-risk Thai heterosexuals. The population is not optimal for testing an HIV vaccine, and the vaccine has performed poorly or not at all in previous studies. (The vaccine includes a prime immunization with canarypox expressing HIV proteins plus a booster shot composed of HIV envelope protein.) RV144's rationale is highly disputed (Burton et al. Science, January 16, 2004). At the NIH, the HVTN cancelled a similar trial in the US. The military researchers behind RV144 insisted on pushing forward, though. Eventually, the NIH accepted responsibility for it. The trial's total cost exceeds $120 million.

Vaccine testing has become a scattershot process. In his 2004 commentary, Ronald Desrosier said, "While it is true that empirical trial-and-error approaches have been sufficient for the development of other successful vaccines, it is highly unlikely, based on what we know today, that this is going to be enough to make an effective vaccine against HIV-1. The major difficulties blocking development of an effective vaccine against HIV-1 are fundamental scientific questions, not issues of manufacturing, numbers of trial sites, international site preparation or validated testing procedures."

Not so Savvy after All

A further STEP Study conundrum is that only one woman in the trial out of about 1,140 acquired HIV. Receiving the vaccine did not harm them. The women notably were more likely than the men to have high natural Ad5 immunity. On a basic level, the male and female trial populations differed in their social as well as biological makeup. The women probably started out at lower risk for HIV, and they may have been more receptive to the trial's safe sex counseling.

Lower-than-expected HIV incidence has been a major stumbling block in large vaginal microbicide trials, too. The Ghanaian and Nigerian trials of Savvy were halted in 2006 for just this reason. The Savvy microbicide is composed of an experimental surfactant, or detergent, that damages viral coatings.

A year after the closures comes news that in the Nigerian trial, there was a 2.0% HIV rate among the Savvy users compared with only 1.1% among women in the placebo arm. That trial included 2,153 HIV-negative high-risk women. The HIV rates were virtually equal in the similarly sized Ghana trial – 0.7% and 0.8% in the vaccine and placebo arms, respectively. With these low numbers, it is hard to tell whether anything substantive is going on. As with the STEP Study, one thing is clear: the product does not look promising.

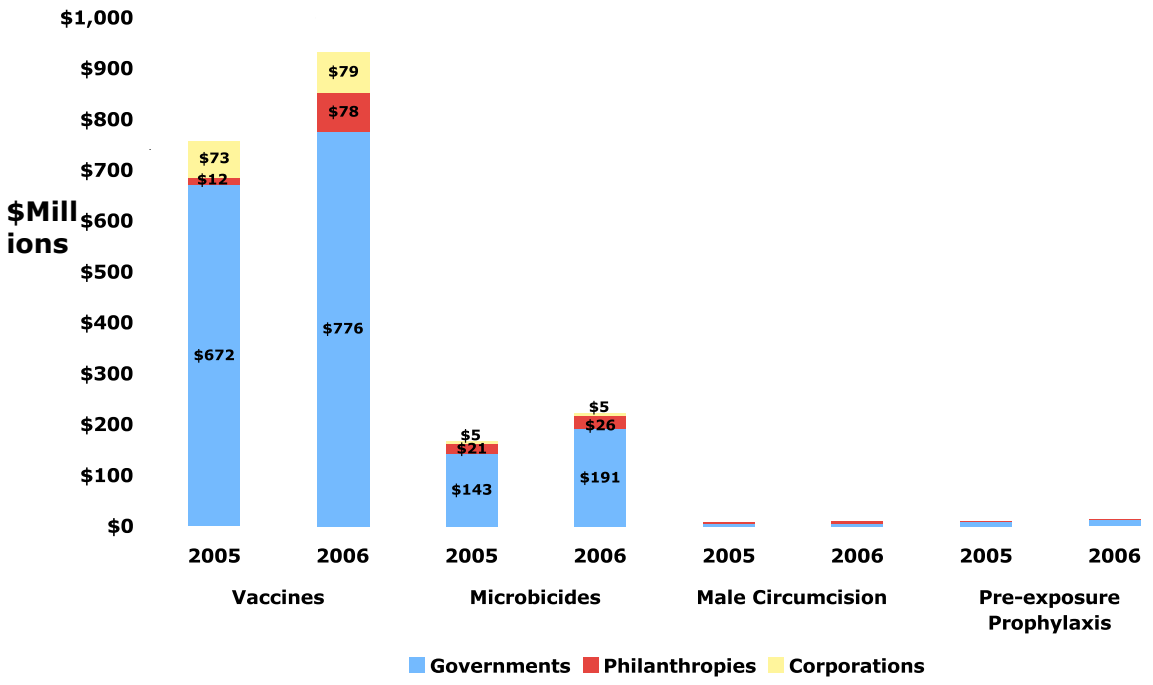
There is a follow-up investigation concerning the amount of vaginal irritation that Savvy causes. The researchers have already noted that the new HIV infections in the Savvy users were concentrated in the women who used the vaginal gel more often. A preliminary safety study in women also reported increased vaginal irritation (Mauk et al. Contraception, September 2004). A study in

mice further observed that Savvy's vaginal toxicities increase susceptibility to herpes, a forewarning that HIV could also increase (Cone et al. BMC Infectious Diseases, June 2006).

Cellulose sulfate. Savvy. MrkAd5. There's a clear signal here that trials are ramping up too quickly. It's bad enough if these products turn out to be merely ineffective. Volunteers who receive experimental vaccines are not eligible for other vaccine trials. Community goodwill is another casualty. The large financial investment is lost, too. Funders will shift their priorities to fields that appear more promising. These repercussions are greatly amplified if the agent in question proves to increase the trial participants' risk for HIV.

With the benefit of hindsight, we can clearly say that each of these three products was not ready for testing in large populations. But there were warning signs even before the trials began. There are also more advanced, arguably more hopeful products under development. HIV presents us with a health emergency. It is quicker – and cheaper – to jump over intermediate-scale testing, but only if the product does not fail.

Global HIV Medical Prevention
The US Government Pays Most of the Research Bill



Funding for HIV vaccine and microbicide research is rising rapidly. Total worldwide spending on vaccine development amounted to $933 million in 2006, a 23% increase over 2005. Total spending on microbicide development was $222 million, a 32% increase. Pharmaceutical companies, who will eventually market the fruits of this research, contributed little to the effort. For both vaccines and microbicides, the United States share was 84% of the total government investment. The part devoted to clinical trials rose even faster than total expenditures. Clinical trials received 30% of 2006 HIV vaccine funding and 43% of microbicide funding.

The 2005-2006 support for vaccine and microbicide development dwarfed the research spending on male circumcision, this

period's great HIV prevention success story (*HHSWatch*, December 2006). Spending on pre-exposure prophylaxis (PrEP) was also modest despite years of promising monkey studies. Circumcision research spending totaled $10 million in 2006 (up 50% from the year before), while 2006 PrEP spending came to $15 million (a 45% rise). The US government again provided most of the money, with significant help from the Gates Foundation.

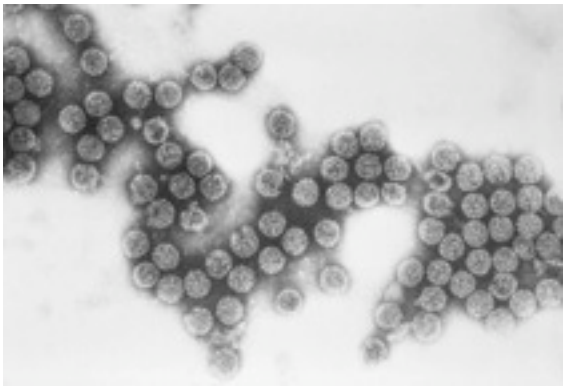
(Source: Building a comprehensive response: Funding for HIV vaccines, microbicides and other new prevention options 2000 – 2006. HIV Vaccines and Microbicides Resource Tracking Working Group. November 2007.)

The Vaccine We Have Already: More Trials and Tribulations

If researchers finally do develop an effective HIV vaccine, what then? There's a vaccine already on the market with the potential to considerably reduce HIV transmission and disease. This is the anti-HPV vaccine, the Merck-produced Gardasil (another, Cervarix, by GSK, is on the way). Gardasil's experience is a harbinger of what's in store for an HIV vaccine.

HPV, or human papillomavirus, causes anogenital warts and asymptomatic infections. Chronic infection with some of the asymptomatic strains may lead to cervical or anal cancer. These malignancies spread to surrounding organs, causing intense suffering and death. Despite highly effective surgical treatments, there are still a substantial number of deaths from anal and cervical cancer even in rich countries – about 5,000 deaths annually in the US alone.

Human Papillomavirus



Source: CDC/FA Murphy

HPV disease screening and treatment cost the US alone an estimated $6 billion per year. Kevin Ault of Emory University is one of the foremost HPV vaccine researchers. He told *HHSWatch,* "Seven percent of all cervical Pap smears are abnormal, but few turn out to be cancer. We scare everybody to death to find those few cases. The vaccine will reduce the number of these false Pap smears and the amount of inappropriate treatment." Treatment of precancerous cervical abnormalities requires carefully distinguishing high-risk from low-risk growths (American Society for Colposcopy and Cervical Pathology Consensus Guidelines, October 2007). The intervention involves excising the affected area, usually with an electric loop. These procedures compromise the cervix, doubling the risk of premature labor.

Gardasil is composed of noninfectious hollow HPV shells. Much new data on its protective ability have become available in the last few months. At the same time, the vaccine has become wracked with controversy over its safety, efficacy, high cost, muscular marketing, and efforts to make it mandatory for preteen girls.

The controversy recalls the one in the 1990s over the hepatitis B vaccine. That relatively costly vaccine protects against a virus with a number of parallels to HPV, including the potential for sexual transmission. In 1991, the CDC embarked on a policy of universal childhood hepatitis B vaccination after repeated failure of vaccination drives in high-risk groups such as hospital workers and gay men. States then adopted mandatory vaccination of schoolchildren. Although many American adults remain unvaccinated,

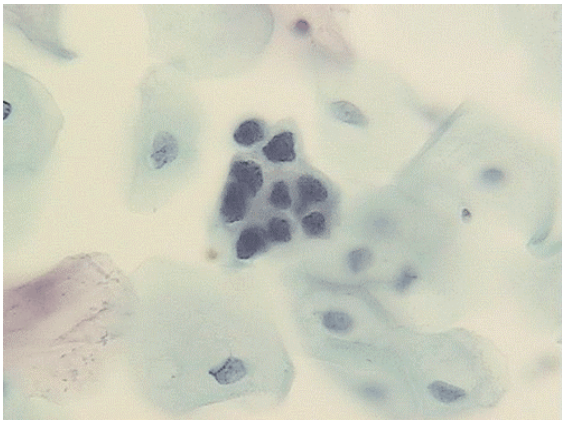
there has been an 80% decrease in new hepatitis B cases since the 1980s (CDC. Disease Burden from Hepatitis A, B, and C in the United States and MMWR, January 2 2004).

HPV Promotes HIV Transmission and Disease

HPV is the most common STD. Most people clear the infection after six to 12 months, but they can be reinfected by another of the three dozen sexually transmitted strains. There is accumulating evidence that HPV-associated anal warts make people more susceptible to HIV. At the biannual meeting of the International Society for STD Research (ISSTDR) last July, the HIM cohort in Sydney, Australia reported its findings from annual examinations of 1,427 gay men (Feng et al. ISSTDR July 2007, abstract O-001). Nearly 20% of the men reported ever having had anal warts, with a yearly incidence of 2%. After adjustment for unprotected sex, anal warts in the past year were associated with a 3.4-fold increase in risk of acquiring HIV. This was greater than the risk from herpes in this study and comparable to the genital herpes-associated HIV risk reported elsewhere (see *HHSWatch*, September 2007).

HPV is an HIV prevention issue, and it is also an HIV *disease* prevention issue. HPV is more prevalent in populations with high HIV rates. The Women's Interagency Health Study found that women considered at high risk for HIV had anal and cervical HPV prevalences of 43% and 24%, respectively (Palefsky et al. Journal of Infectious Diseases, February 1, 2001). For HIV-positive women in the same cohort, the figures were still higher, 79% and 53%. (Note that in both cases, the anal HPV rate was even higher than the cervical one.) The San Francisco Men's Health Study detected anal HPV in 61% of HIV-negative gay men and in 93% of HIV-positive gay men (Palefsky et al. Journal of Infectious Diseases, February 1998).

Advanced Precancerous Cervical Cells (center)



Source: International Association of Chinese Pathologists

People with HIV do not clear HPV nearly as often as their HIV-negative counterparts do, and HPV-associated abnormal cell growth is two to three times more frequent (Palefsky. Topics in HIV Medicine, August-September 2007). The immune restoration accompanying antiretroviral therapy unfortunately does not decrease the frequency of HPV-related malignancy. A study published last August found that anal warts, not usually associated with cancer, frequently contain malignant cells in HIV-positive men (McCloskey et al. International Journal of STD & AIDS, August 2007). Finally, some studies indicate that the presence of HPV-triggered genital growths is associated with increased HIV in genital secretions (Spinillo et al. Obstetrics and Gynecology, February 2006). In this way, HPV could render coinfected individuals more likely to transmit HIV.

An Effective Vaccine, with Caveats

There is no doubt that the HPV vaccine is highly effective in protecting women against the associated abnormal cervical growths (Ault et al. Lancet, June 2, 2007) and warts (Ault. ISSTDR July 2007, abstract O-109). Modeling of the rate of antibody decay indicates that the protection afforded by the vaccine probably lasts for decades if not for life (Fraser et al. Vaccine, May 22, 2007).

There are four major caveats to these findings: First, they do not yet apply to men or to anal disease – those trials are still ongoing. Secondly, you have to receive all three doses of the vaccine according to schedule. Thirdly, the vaccine mainly protects against the HPV strains for which it contains representative envelope proteins. The two carcinogenic strains in the vaccine are responsible for 70% of cervical malignancy. (Gardasil, but not Cervarix, also protects against two HPV strains responsible for 90% of anogenital warts.) Finally, the vaccine is not protective against HPV strains you contracted before being immunized.

Among 20,000 women in Merck-sponsored trials, the vaccine was 99% protective against precancerous and cancerous cervical lesions for the three years of observation (Ault et al. Lancet, June 2, 2007). That's if all the above conditions were met. The vaccine provided little, if any, benefit to people who entered the trial already infected with one of the vaccine's HPV strains.

One new study reported that there is 40% cross-protection against carcinogenic strains not contained in the vaccine (Brown et al. ICAAC September 2007, abstract G-1720b). Again, you have to be vaccinated before exposure. The large Merck trials detected no protection against cervical conditions arising from nonvaccine HPV strains. That is at least partly because many trial participants had previously contracted those strains. For the general public, HPV testing and Pap smears will continue to be useful, if less critical, in reducing the overall cervical cancer rate.

These issues are magnified in poorer nations, where regular Pap smears and HPV testing are uncommon. Cervical cancer in poor countries is three to ten times more frequent than in the US. A mathematical model, taking into account both difficulties in vaccinating all women and the lack of protection against all HPV strains, forecast that providing the vaccine alone to girls aged 9 to 12 in poor countries would decrease cervical cancer by 40% (Sherris, ISSTDR July 2007, Monday morning plenary presentation – no abstract). In comparison, three lifetime cancer screenings (at ages 35, 40 and 45) would reduce cervical cancer by less than 30%. Combining the two would yield still better protection – a 60% decline in new cervical cancer.

Safety: Everybody Has an Agenda

In 2007, with the push toward mandatory HPV vaccination of schoolchildren in 26 state legislatures, the vaccine came under considerable attack by a tacit alliance of anti-vaccine activists and right-wing groups. In October, the conservative legal gadfly Judicial Watch issued a scathing criticism of the vaccine's safety (Judicial Watch, October 4, 2007). The group examined the raw records in the CDC/FDA Vaccine Adverse Event Reporting System (VAERS).

Judicial Watch President Tom Fitton summed up his organization's conclusions by saying, "In light of this information, it is disturbing that state and local governments might mandate in any way this vaccine for young girls. These adverse reaction reports suggest the vaccine not only causes serious side effects, but might even be fatal."

HHSWatch examined the VAERS data on December 15. On that day, VAERS contained 799 serious event records for Gardasil, including 11 deaths. The problem with the VAERS records is that they are very rough and preliminary. Anybody, including the general public, can provide VAERS reports. Frequently they describe an event that one person heard about from somebody else. The only criterion for including the report is that the reporter believes there is a link to the vaccine. Judicial Watch does not seem to have read the VAERS records very closely. Its web site presents ten copies of VAERS records describing Gardasil-related deaths. Of these, two are duplicates, four are based on hearsay (one notes, "the patient may not have expired"), two involved serious viral infections, and one had evidence of a prior unrecorded heart condition. In this last case,

the patient's collapse and death occurred two weeks after vaccination. It is difficult to ascribe any of these cases to the vaccine without further investigation.

It is even hard to say how many deaths really occurred. As of last June, the CDC reported that it could confirm only four deaths occurring after vaccination (Iskander. Advisory Committee on Immunization Practices, June 28, 2007). Its investigation did not attribute any of these to Gardasil.

A more carefully recorded set of data comes from the Gardasil clinical trials, in which 10,500 people received the vaccine and a similar number received placebo. The trials noted 17 deaths, ten in the HPV vaccine recipients and seven among those receiving a placebo vaccine. No particular cause of death stood out as possibly related to Gardasil (Gardasil Prescribing Information, November 2007).

A Vaccine for the People or the Privileged?

In the end, only two states, Virginia and New Jersey, passed legislation to add the HPV vaccine to the growing list of vaccines required to attend school. The mandatory vaccination bills were either withdrawn or tabled in the other states. "We're not ready for mandatory HPV vaccination," commented Lauri Markowitz, one of the CDC's top experts on these vaccines. The issue is dead for now, but the bedrock problem of accessibility remains little examined. The nation's vaccine financing system is unraveling, and many people face unexpected hurdles when seeking HPV immunization.

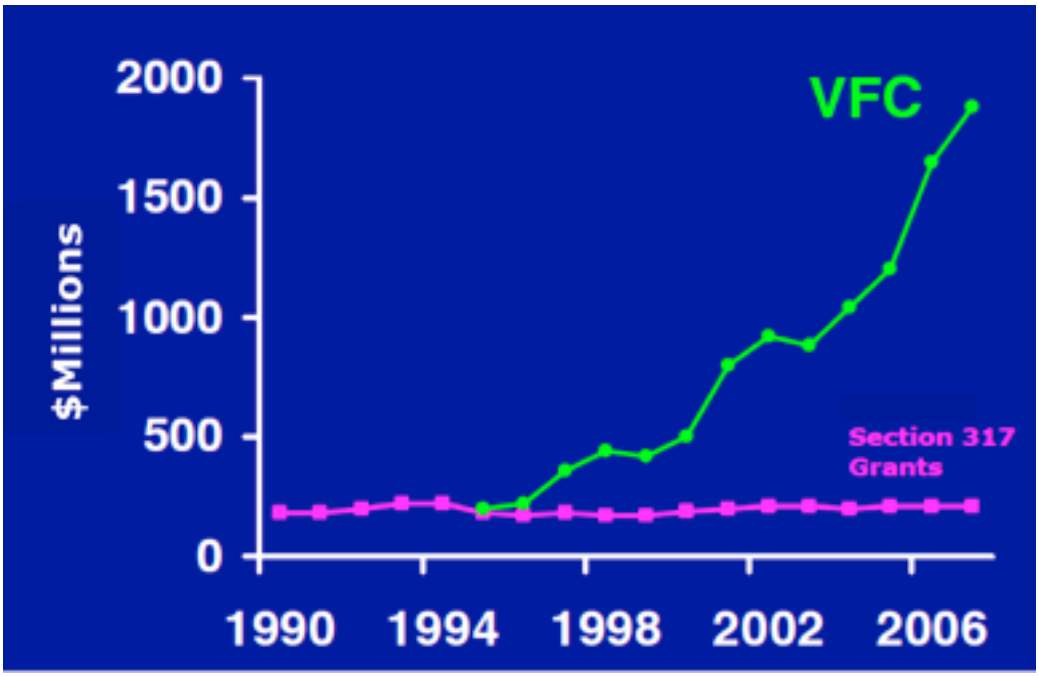
The CDC currently recommends that all women 11 to 26 receive the HPV vaccine as soon as possible. That's about 30 million women, and the three-shot vaccine costs about $400. The immediate outlay in the US alone would then be $12 billion. Every year, another 2 million American girls reaching age 11 would be vaccinated, at a cost of $800 million.

These calculations leave aside the question of whether males and older women should also be immunized. Merck is already asking the FDA for approval to administer its vaccine to women as old as 45 based on trial results presented last November at the 24th International Papillomavirus Conference (Merck Press Release. November 5, 2007). The theory is that vaccinating older women provides them with catch-up protection for the HPV strain to which they have not been exposed. Data on men, who are certainly also vulnerable to HPV's genital and anal effects, will be available in 2008. Bloomberg News (October 4, 2007) has estimated that Gardasil generated $1.4 billion in sales in 2007. According to Bloomberg, sales could reach $10 billion annually if approved for boys and men.

In the United States, the CDC's Vaccines for Children (VFC) program supplies vaccines for children enrolled in Medicaid plus those without insurance covering vaccinations (uninsured or underinsured children). VFC is an entitlement program whose budget increases automatically to meet costs. That budget has expanded four-fold since 2000, in parallel with the number and cost of vaccines. VFC now spends almost $2 billion annually to supply 43% of US children's vaccine needs. Just vaccinating 10 and 11 year-old girls for HPV will increase VFC expenditures by more than 10%.

Medicaid-enrolled and uninsured children are not the major gap in this program. They receive the VFC-paid vaccines from their regular care provider, as part of state Medicaid programs. There remain underinsured children, who make up 14% of Americans aged 0 to 17. VFC will pay for their vaccines only if they go to certain "federally qualified" or rural clinics, which exist only in a limited number of locations. Another barrier is that these clinics usually charge patients a $15 administrative fee. Worse yet, VFC does *not* cover children whose insurance will not pay for their vaccines merely because they have not met their plans' high yearly deductible.

Federal Financing for Vaccines



Source: Grace Lee, Harvard Medical School. Advisory Committee for Immunization Practices, June 27, 2007

Federal "section 317" grants and a patchwork of state funding attempt to fill these holes. The National Conference of State Legislatures lists 11 states that pay for vaccines to all their youthful residents, but restrictions are steadily increasing. Most state programs require that underinsured children go to public clinics to obtain the newer vaccines, and they may not be available even there. The CDC's National Immunization Survey estimates that in 2006, only 64% of children aged 19 to 35 months had complete vaccine coverage.

Four states – Alaska, New Hampshire, South Dakota and Washington – are now paying for universal HPV vaccination for girls. Unless they are in Medicaid, persons over the age of 18, however, have no reimburser of last resort in these states or elsewhere.

Delays in receiving the HPV vaccine may occur even for women with adequate insurance. Their doctors may be unable or unwilling to stock adequate amounts of this expensive vaccine. The traditional means of obtaining a vaccine in the United States is from your doctor, who keeps a supply on hand in the clinic, and few patients know that vaccines are also obtainable from pharmacies by prescription. For everyone, going to the drugstore to buy a vaccine is an extra, unfamiliar step.

Barriers to obtaining the HPV vaccine are critical in the adolescent years, since most women start having sex when they are 16 to 18 years old. The same can be said for young men, especially gay ones. Merck has yet to provide any data in males, and as a result, none have insurance that covers HPV immunization. Many young adults stand to miss the protection that the vaccine offers due to all these barriers. That protection marks an important advance for teens exploring their nascent sexuality. It's a human rights question as well as a health one.

This HHSWatch was written by David Gilden

***HHSWatch**, a watchdog newsletter from CHAMP, monitors and reports on activities related to HIV prevention at Health and Human Services agencies, including CDC, NIH, HRSA and SAMHSA.*

HHSWatch is a resource for community members, policy advocates, researchers and anyone interested in more fully understanding and tracking the committees, panels and administrators whose recommendations and decisions affect our work.

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