Is an AIDS vaccine necessary?  
Is an AIDS vaccine even possible?

These questions have been constant companions in the search for a safe, effective, and affordable AIDS vaccine—a search that dates almost from the beginning of the epidemic—but they came to the foreground in September 2007, when the STEP vaccine trial was halted.

The vaccine candidate in the STEP trial, made by Merck and known as MRK-Ad5, incorporated synthetic versions of some of HIV’s own genes (gag, nef, and pol) into a delivery system, called a vector, developed from an adenovirus type 5 (Ad5) that causes the common cold. The vaccine was designed to stimulate cell-mediated immunity, in which T cells—major players in immune function—target and kill cells that are already infected with HIV.

The purpose of the trial was to find out whether the vaccine reduced the risk of HIV infection, and whether it reduced viral load set-point in people who did become infected after receiving the vaccine. Viral load set-point—the viral load level following a “spike” in viral replication just after infection—is a predictor of long-term clinical outcomes, so a lower set-point in vaccinated participants could indicate that the vaccine slows disease progression.

The initial recommendation of the trial’s independent Data and Safety Monitoring Board to halt immunizations was based on a futility finding: there were similar numbers of infections in the vaccine and placebo (or “dummy shot”) arms, and there was no possibility that MRK-Ad5 would show efficacy if the trial continued.

In addition, subsequent analyses showed that some individuals who received MRK-Ad5 and had pre-existing immunity to the Ad5 virus used as a vector were actually more susceptible to HIV infection than comparable individuals in the placebo group. (The vaccine itself did not cause HIV infection. No vaccines being tested can cause HIV infection.)
Public debate ensued as the research and advocacy field went, in a few short months, from eagerly awaiting the results of the promising MRK-Ad5 vaccine candidate to grappling with fundamental questions about what to do next in the search for an AIDS vaccine. Today, the field is moving forward with a concerted focus on answering fundamental, basic scientific questions that can help guide vaccine development, while continuing to glean invaluable data from the STEP volunteers and mapping out future vaccine “discovery” trials.

A range of vaccine candidates remains in development, largely in early-stage trials. Communities that were involved in previous vaccine trials remain committed to HIV prevention research. Advocacy has also been recalibrated over the past year, with a focus on the long haul and an even stronger belief that the discovery of an AIDS vaccine is just as urgent today as it was nearly three decades ago.

Learning in the “Post-STEP” Era

Even before the initial shock, sadness, and disappointment around the STEP trial results had fully worn off, many stakeholders in the AIDS vaccine field had already started to sketch out the questions that the trial raised—and that it might help to answer. Below are three key themes that have emerged in the first year of the post-STEP era.

Exploring Vectors, Inserts, and Pre-Existing Immunity

What happened with the Merck candidate? Why did it fail to prevent infection or reduce viral load in those participants who received it but became infected? Why was there an increased susceptibility to HIV in participants with pre-existing immunity to the Ad5 vector used in the vaccine?

The bad (but predictable) news is that there may never be answers to all of these questions. In the wake of the STEP announcement, the U.S. HIV Vaccine Trials Network (HVTN) and the National Institutes of Health (NIH) initiated a series of analyses to address these and other issues, soliciting proposals from outside researchers and establishing an independent committee to review them.

The November 29, 2008, issue of The Lancet includes the first published results from the STEP study and from the follow-up immunological studies undertaken to determine why the vaccine failed to protect against HIV or lower viral load set-point, and why it might have increased susceptibility to infection. The authors of these articles emphasize that there are no clear answers to either question—and there may never be. Juliana McElrath and her colleagues performed a range of analyses looking at immune responses in vaccine and placebo recipients who became infected and those who did not, and found no clear factor associated with infection. Likewise, it is impossible to say why, exactly, the vaccine failed to prevent infection or reduce viral load. What STEP does tell us is that these specific immune responses didn’t work; the trial sets a bar for future vaccine candidates to surpass.

McElrath and her group also suggest some areas for further exploration, noting that vaccine recipients who became infected with HIV had fewer Ad5-specific T cells in their blood—perhaps a sign that more of these cells had migrated from the blood to the vaginal or rectal mucosal tissue, where sexual transmission of HIV occurs. Migration of these activated T cells to the mucosa could mean that more target cells are available for HIV to infect, resulting in increased susceptibility to the virus. Follow-up studies of mucosal samples from STEP volunteers may explore this question, but there may never be a clear answer.

A recent article by Matthieu Perreau and colleagues, published in the Journal of Experimental Medicine, proposes another explanation for the increased susceptibility to HIV infection seen in the STEP study. It used laboratory studies to model the effects of Ad5 vector and Ad5-plus-antibody “immune complexes” on other immune cells. In this in vitro experiment, the immune complexes induced maturation of dendritic cells, which play a key role in triggering the body’s immune response and are early targets for HIV infection. The authors hypothesize that perhaps Ad5-related immune activation of this nature provided more target cells in vaccinated individuals with pre-existing antibodies. However, this proposed explanation does not account for all of the data observed in STEP, according to other researchers, since Ad5 seronegative volunteers who received the vaccine and subsequently developed antibodies to the vector were not at increased risk for HIV.

The process of developing and executing a research agenda to understand what happened in the STEP trial is also sharpening questions that apply to all vaccines aimed at cellular immunity. These include questions about the role of pre-existing immunity to vectors that may be used in future candidates; the selection of genetic inserts, or immunogens, to elicit optimal T cell defenses; and the qualities of the optimal T cell response needed for an effective vaccine.

If this all sounds like a mouthful of scientific jargon—which it is—the bottom line is this: There are many different kinds of T cells, key components of the body’s immune defense. Some T cells are effector cells, which become activated, attack invaders (such as viruses), and then die once their job is done. Others are memory cells, which remain in the body after the battle is over in order to mount a rapid response should the invader return. T cells also make a range of different substances, called chemokines and cytokines, that have important effects on the cells and tissues around them and play a major role in immune function.

Since it’s clear what kinds of T cells the Merck candidate induced, and it’s also clear what effect these had (in the majority of the vaccine recipients), then it is possible to draw some conclusions about what should
be done to improve future vaccines’ chances of inducing cell-mediated responses with beneficial effects.

**A Focus on Host Genetic Factors...and a Possible Glimmer of Hope**

Vaccines are delivered to and work in people. And people are different in many ways—including the genes that shape and define their immune responses. In the parlance of scientists, these are known as “host genetic factors.” The host is the human, and the genetic factors include the genetically determined complex of antigens (substances that provoke an immune response) occurring on the surface of almost every human cell.

The ongoing analysis of the data from STEP and Phambili (the South African trial of the same MRK-Ad5 vaccine candidate) is focusing on understanding the role of these host factors among the many variables that might explain the trial results. Other variables under investigation include behavior, geography, circumcision status, pre-existing immunity to the Ad5 vector, and infection with sexually transmitted diseases other than HIV.

Here again there are no simple answers. However, there is an intriguing, though indeterminate, finding from some of the individuals in the STEP study who received the vaccine and went on to acquire HIV. Specifically, scientists have looked closely at those vaccinated individuals who have specific genetic traits associated with HIV disease control. There is a suggestion that individuals in this group who received the Merck vaccine had lower viral loads than individuals with the same genetic traits who received the placebo. The group of individuals in question is far too small to draw any firm conclusions, but it has generated discussion about avenues for further research.

**Clinical Trials of AIDS Vaccines Can—and Must—be Done**

While STEP and Phambili halted immunizations early due to disappointing and disturbing findings, the studies themselves were clear successes; any trial that provides a quick, clear answer to its scientific questions is a success, whether those answers are positive or negative.

The STEP trial reached its recruitment goals and 94% of study participants received all three immunizations with either the vaccine or the placebo. Its data analysis plan—which lays out the schedule of independent interim data reviews, among other things—was rigorous and ensured that the findings of futility and possible enhanced susceptibility were recognized as quickly as possible. The research teams worked overtime to communicate complex findings to trial participants. Although questions and concerns remain, there is also a clear sense from community groups, trial participants, and trial staff that there was an exemplary degree of transparency and responsibility throughout the STEP and Phambili trials.

In addition to showing that clinical trials of vaccines can be done, the data are also a reminder that they must be done—because there is such a clear and urgent need for additional new HIV prevention strategies. For example, HIV incidence among gay men and other men who have sex with men in the STEP study was greater than 4% in both the vaccine and the placebo arms. This incidence is not a result of vaccine-related effects on susceptibility to the virus, as it was seen in men who did not receive the vaccine. It is, instead, a stark reminder that current prevention programs and tools are not meeting the needs of these men in the U.S. and around the world.

**Pressing On**

The STEP vaccine’s failure triggered intense debate and calls for reinvigorated discussions about how the search for an AIDS vaccine should proceed. Some notable scientists have said it is time to move away from large-scale clinical trials testing experimental vaccines in humans and toward more fundamental basic research to understand better the biology of the virus and its effects on the human immune system.

Much of this debate has been invigorating. But it has also generated some false dichotomies, like the “either/or” proposition of whether to continue conducting trials in humans versus returning to basic science. In reality, the new money going into AIDS vaccine research over the past three years has been directed toward basic science and discovery-oriented projects, not human clinical trials. Yet human clinical trials—both large and small—are absolutely critical for gathering much-needed information to move the field forward. The field should proceed using the combined strengths of basic science, animal studies, and human trials to guide and shape its scientific strategy.

Some called for dismantling the vaccine field altogether and would like to see the money spent instead on treatment and care for people with HIV. To this, vaccine supporters say, again, the false dichotomy of “either/or” is dangerous. Funding for vaccine research does not necessarily supplant research for treatment or other prevention areas. And if vaccine research funding were indeed pulled, there is no assurance that it would be redirected to care and treatment. (In addition, mounting evidence suggests that because treatment decreases the amount of transmissible virus in the blood, genital fluids, and breast milk, treatment may be a form of prevention. For example, mathematical models estimate that if every HIV positive person in low- and middle-income countries were started on antiretroviral therapy, the rate of new HIV infections in these regions could drop by 70%.)

There are also those who say it’s just not possible to make an AIDS vaccine, that the virus replicates and mutates too quickly for any vaccine to prevent infection or disease progression. These critics are speaking too soon. Why should the search for an AIDS vaccine continue? Because there...
is evidence that an AIDS vaccine is possible, and because history tells us that vaccines have been among the most effective public health interventions in preventing the spread of viruses and saving lives.

An AIDS Vaccine is (Still) Possible

Evidence from diverse studies suggests that an effective AIDS vaccine is still possible, and that discovery and development of future candidates can and should draw on protective immunity research. For example, there are well-researched examples of humans infected with HIV who maintain viral loads at or below the limits of detection for many years without taking antiretrovirals. This small group of “elite controllers” suggests that it may be possible for the immune system to reach what Jonas Salk, discoverer of the polio vaccine, called a “peace treaty” with the virus.

The current hypothesis is that some combination of immune responses, genetic factors, and other variables that are not yet understood allows elite controllers to maintain a clear advantage in the battle against HIV on their own. Studies are ongoing in this group to learn more and to glean clues that can be used to guide vaccine development. There is also ongoing research on individuals, like some women in a cohort of commercial sex workers in Kenya, who remain HIV uninfected in spite of repeated HIV exposures.

Furthermore, the majority of infants born to HIV positive mothers remain uninfected, and many avoid infection during breastfeeding. Antiretroviral drugs for mother and infant are essential to reduce the risk of transmission, but the example of infants who remain uninfected in the absence of these drugs is another tantalizing suggestion that the body can defend itself against the virus.

The non-human primate model shows yet another indication of protective immunity: vaccine-mediated protection against a simian version of HIV (called SIV) in non-human primates has been accomplished. In these experiments, researchers created a vaccine using a “live-attenuated” form of SIV; the virus in the vaccine had been disabled to prevent it from causing infection or illness. (This strategy has been used extensively in designing other vaccines used in humans, but it is not being explored with HIV due to the risk associated with using even a disabled live form of the virus in a vaccine.)

In several experiments, monkeys who received various types of live-attenuated SIV vaccine were protected either against infection or from disease progression. One recent study looked at monkeys who had received two doses of live-attenuated vaccine three years apart; the animals were followed for eight years, and those with immune responses triggered by the vaccine remained uninfected when dosed with infectious SIV and SHIV, another type of HIV-like virus used in non-human primate studies.

The Epidemic Demands It

More than 4 million new HIV infections occur every year. In South Africa, where three recently completed HIV prevention trials (looking at vaccines, microbicides, and the latex diaphragm) took place, there are communities where nearly one-third of women between the ages of 25 and 29 are infected with HIV. In the United States, the rates of new HIV infections in young African-American men who have sex with men are comparable to those seen in the hardest-hit low-income countries. And in 2007, for every two individuals who started on life-saving antiretroviral medications, five others were newly diagnosed.

The history of other epidemic diseases like smallpox and influenza tells us that an effective vaccine is an essential tool in halting an epidemic. The first AIDS vaccines to show benefit may not look much like the highly effective smallpox vaccines. They may be therapeutic rather than preventive, decreasing viral load and slowing HIV disease progression instead of directly preventing infection. But they are still a potentially powerful tool.

As much as the epidemic demands that the search for a vaccine continue, it also demands that we do more to implement proven prevention strategies, including improving people’s access to male and female condoms, clean needles, prevention of mother-to-child transmission (PMTCT), and risk-reduction counseling. These tools, although available today, are still inaccessible to the vast majority of those who need them.

In December 2006, we learned that male circumcision showed strong protective benefits for HIV negative heterosexual men. This strategy must be made broadly available in communities where it can have an impact. At the same time, we must also do more to bring comprehensive care, treatment, and support to people already living with HIV worldwide.

What History Tells Us about Vaccine Development

Will an AIDS vaccine be possible in the next ten, twenty, or thirty years? In the lifetime of a physician who saw the first AIDS cases on the wards in the 1980s? Maybe not. In the lifetime of an infant born today, perhaps one who is protected from HIV infection through the use of antiretrovirals for PMTCT? Hopefully.

Historically, it has taken decades—and more setbacks than advances—from the discovery of a virus or species of bacteria until an effective vaccine is licensed. Typhoid was discovered in 1884, but a typhoid vaccine was not licensed until 1989. The measles vaccine took 42 years to develop, and malaria, discovered more than a century ago, still has no vaccine. Given that HIV was discovered under 30 years ago, the search for an HIV vaccine is still comparatively young.

In the 1930s, two experimental polio vaccines failed because they were determined to be unsafe, and polio vaccine development was nearly
abandoned. At the time, we understood how to prevent infection through sanitation and by avoiding public swimming areas, just as we know how to stop HIV infection today. New tools were needed then to supplement insufficient uptake of behavioral interventions; likewise, we need new tools now to prevent HIV.

Over the last 20 years, important steps have been made toward parsing the major challenges of AIDS vaccine research into concrete projects. There have been constructive discussions of how to shore up the foundations of the AIDS vaccine search, including career paths for young investigators, adequate resources for non-human primate research, and bench-to-bedside stewardship of important research projects.

As painful and disappointing as recent failures are, donors, advocates, scientists, physicians, and clinical trial volunteers and their families must guard against “failure fatigue.” They must respond loudly and clearly to suggestions that enough money has been spent on vaccine research; that it would be easier and wiser to move on rather than press on. To do so would be to ignore the reality of the epidemic today, and to overlook the lessons from history about the long, slow process of vaccine discovery.

**How Might an AIDS Vaccine Actually Work?**

Most traditional vaccines against viral diseases—influenza and polio, for example—stimulate antibodies that control the virus and prevent disease. However, inducing such effective antibodies against HIV is an extraordinary challenge because the virus has multiple ways of evading the body’s immune defenses. First, shortly after the initial infection, HIV begins to infect and ultimately kill the very immune cells that would normally protect against the virus—and which most vaccines are designed to boost. Second, HIV mutates rapidly, making it difficult for the immune system to recognize and attack the virus. And lastly, HIV inserts itself into the DNA of human cells, where it can remain undetected by the immune system.

These challenges make it extremely difficult to design a vaccine that prevents HIV from establishing itself in the body. Much important work in developing a “neutralizing antibody-based” vaccine continues, but there are currently no such vaccines in clinical trials.

The majority of AIDS vaccine candidates in current human clinical trials attempt to induce T cell–based immunity. This arm of the immune system recognizes and eliminates already HIV-infected cells—unlike antibody vaccines, which prevent HIV from entering immune cells in the first place.

As described above, these cell-mediated immune (CMI) responses have been associated with long-term survival in elite controllers and have been observed in highly exposed, persistently HIV negative individuals. There is also evidence from the non-human primate model that a CMI response is an element of viral control in successful vaccine experiments, even if the vaccine does not completely prevent HIV infection. In animal experiments, non-human primates that received CMI-inducing vaccines had lower viral load set-point and slower disease progression compared with animals that received a placebo. There are also cases of vaccine candidates that appear to have completely prevented infection in the animal model.

A vaccine that induces cell-mediated immunity does not yet exist, but if discovered, it is expected to provide a form of partial protection. HIV negative individuals who receive the vaccine and later become infected with HIV might have lower viral loads and slower disease progression than unvaccinated people. The development of such a vaccine would include discussions about how great an impact on viral load and disease would warrant licensure and distribution. In other words, how much partial protection is enough? It’s important that these discussions start even before such a vaccine or other partially protective strategy (think male circumcision for HIV prevention) becomes available—and in many places those conversations are already underway.

It is clear, however, that this type of vaccine would need to be introduced as an additional, complementary part

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**Vaccine Development History**

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Adapted from the AIDS Vaccine Handbook, 2nd Edition.
of comprehensive HIV prevention, not as a replacement for existing options.

**The State of AIDS Vaccine Research**

There is still a great deal that we don’t know about the STEP trial results, and there is an enormous effort underway by the AIDS vaccine field at large to understand them. But these results must be seen for what they are: the failure of a vaccine candidate to show efficacy in a specific trial. Clinical testing of AIDS vaccines is a scientific process and, while the failure of this candidate is a disappointment, it is in no way the end of the search for an AIDS vaccine.

The next vaccine candidate is a combination strategy developed by the NIH’s Vaccine Research Center (VRC). The VRC strategy consists of three DNA immunizations with a single Ad5 “boost” at the end of the series. It was scheduled to enter an efficacy trial in September 2007, just as the halt of MRK-Ad5 immunizations was announced, and the trial was put on hold in order to reconsider whether to test another candidate using an Ad5-vectored vaccine—the same vector that increased HIV susceptibility in the STEP trial.

If there was a safety concern about one Ad5 AIDS vaccine, why even consider testing another? It’s a fair question—and there are several answers. In the STEP study, men who received the vaccine and were circumcised and Ad5 seronegative (meaning they had no pre-existing immunity to the Ad5 virus) were not at increased risk (compared with Ad5 seronegative, circumcised men who got the placebo). Therefore, some researchers argued that the VRC strategy could be tested in men with the same characteristics—Ad5 seronegativity and circumcision.

In addition, there are differences between MRK-Ad5 and the VRC strategy. MRK-Ad5 used three immunizations of Ad5-based vaccine; the VRC strategy only uses one. The vaccines also carry different types of genetic material from the virus. Finally, the disabled Ad5 viruses used as vectors in the two strategies are similar but not identical; different sections of the adenovirus have been deleted to make the VRC vector.

These safety issues, along with the evidence for (and against) the argument that the VRC strategy was substantially different from MRK Ad5 in terms of immunogenicity, were debated over nine-plus months following the STEP results. In July 2008, Anthony Fauci, head of the National Institute of Allergy and Infectious Diseases at NIH, announced that the study to evaluate the VRC strategy, known as the PAVE 100 trial, would not go forward. Instead, he asked researchers to design a “smaller, leaner, meaner” trial that would provide some information to help guide the next phase of AIDS vaccine development.

A protocol for this trial, known as HVTN 505, is currently being developed. If it moves forward with approval from the U.S. Food and Drug Administration, HVTN 505 will seek to enroll roughly 1,300 gay men and other men who have sex with men in the U.S. who are both circumcised and Ad5 seronegative, since there were no known safety issues with this group in the STEP study.

The trial is being called an “exploratory” study; it is not designed to determine whether the VRC strategy should be advanced, as-is, into large-scale testing that could lead to licensure. Instead, it focuses on two key questions: Is the vaccine safe—does it in any way increase susceptibility to HIV or cause any adverse events? And, does it reduce viral load set-point in people who receive the vaccine and go on to acquire HIV?

If it goes forward, HVTN 505 may be the most complicated trial the field has ever had to understand and explain. It is a study that could yield valuable information, but which involves a highly restricted set of participants, a vaccine that is not expected to provide protection against infection (as most licensed vaccines do), and a design that is not intended to lead to licensure. Explaining the study population, the expectations of the vaccine, and the trial design will require conversations at many levels, including the communities where trials may take place and the broader advocacy and activist community working on prevention, prevention research, and gay men’s health.

What else is out there? At present, more than 30 clinical trials of experimental AIDS vaccines are underway in 25 countries. The majority of these trials are small Phase I and II safety and immunogenicity studies.

One large-scale, Phase III efficacy trial in Thailand is currently testing a combination of two vaccines: ALVAC, which includes a canarypox vector candidate manufactured by SanofiPasteur, and VaxGen’s AIDSVAX, which combines genetically engineered versions of proteins from two different HIV strains. The trial has enrolled 16,000 participants, making it the largest AIDS vaccine trial ever.

Results from the trial are expected at the end of 2009. Some in the scientific community have been skeptical that this vaccine strategy will work—AIDSVAX failed to show efficacy by itself in two earlier trials—and the field must be prepared for either a positive or a negative result. Should there be a positive result in this trial, there will be questions (similar to those first raised when the trial launched in 2003) about whether the benefit

To view a list of current vaccine trials, visit [http://avac.org/trials_table.htm.](http://avac.org/trials_table.htm)
How Much Does Vaccine Research Cost?

In 2007, total global investment in vaccine research was an estimated $960 million. This total represents an impressive near-tripling of investments from 2000; but only a marginal 3% increase from 2006. Recent research results and continuing scientific challenges may further affect future funding. The challenge going forward will be to sustain the research effort and to capitalize on what we have learned thus far.

For more information about funding for HIV prevention research, visit www.hivresourcetracking.org and www.unaids.org.

Advocacy Going Forward

The past year and a half has been among the most challenging yet affirming periods of AIDS vaccine research. The failure of Merck’s Ad5 vaccine candidate to show any benefit in the STEP trial triggered an onslaught of media attention, including editorials, blog entries, mainstream reporting, and scientific commentaries—some accurate, many misinformed.

As mentioned earlier, some advocates and scientists have made these trial results the foundation of an argument that AIDS vaccine research should be halted, that the search is futile, that we are no closer to a vaccine today, and that the resources devoted to it are an exorbitant waste. More importantly, though, the vast majority of scientists and advocates involved in AIDS vaccine research and development have answered loudly and clearly that a vaccine is still possible—and necessary.

There is increasingly broad consensus that the search for an AIDS vaccine requires research into vector-based immunity, host genetics, and the factors that make proven vaccines work, as well as investment in the careers of new and young researchers and advocates, to ensure that the long-term process of vaccine development continues despite future setbacks. Benjamin Franklin said, “Perhaps the history of the errors of mankind, all things considered, is more valuable and interesting than that of their discoveries.” For the AIDS vaccine field to move forward in the struggle to end HIV and AIDS, we must mine the painful yet valuable lessons we have learned.

Emily Bass is the Program Director of the AIDS Vaccine Advocacy Coalition (AVAC). She has helped to design and coordinate AVAC’s activities around AIDS vaccines and an array of new and experimental prevention research options, including male circumcision and pre-exposure prophylaxis (PrEP).

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Mitchell Warren is the Executive Director of AVAC. He is also a member of the Global HIV Prevention Working Group and the Co-Chair of the Caucus for Evidence-Based Prevention.

Selected Sources


