WHY US?
LEFT BEHIND AND DYING

Video Curriculum Modules

Lesson 15:
HIV and SIV

LEARNING WAS NEVER LIKE THIS

Lesson 15: HIV and SIV

Standards:

Health:

1.12.3

2.12.2

2.12.5

2.12.10

3.12.1

Science:

1.2

2.1

2.2

2.4

2.5

4.3

4.4

7.5

Skills Practiced and Gained:

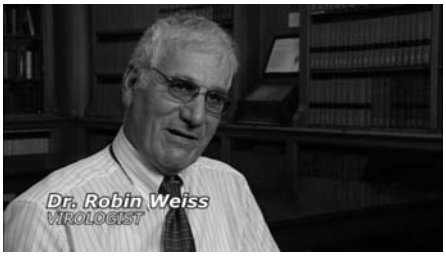
1.1—1.7

2.1—2.5

Overview

Diseases caused by viruses that infect humans have been traced back in some cases to viruses in animals, for example: Avian Flu, Severe Acute Respiratory Syndrome (SARS), and Mad Cow Disease. Similarly, HIV, human immunodeficiency virus, has been traced back to SIV, simian immunodeficiency virus. Two types of HIV have been traced back to two different hosts for SIV. HIV-1, source of the global pandemic, has been traced back to the Chimpanzee, and HIV-2 has been traced back to the Sooty Mangabey.

The transfer of a virus in an animal to humans often happens because the animal and humans are in close proximity. In the case of HIV and SIV, humans in Africa come in contact with Chimpanzees and Sooty Mangabeys because they are a source of food and are kept as pets. Unfortunately, Africans were vilified and maligned about the virus in ways that other populations—SARS and the Chinese; Mad Cow Disease and the British—never experienced. This disparagement, founded in racism, has greatly contributed to African and African American distrust of the media and medical establishment (See Module/Lesson 14).



The video module, "*HIV and SIV*," examines the ways in which HIV and SIV are connected, and the inappropriate vilification of Africans in regards to HIV/AIDS.

Key Concepts

Transfer of viruses and diseases

Proliferation of viruses and diseases

Social determinants of health

Impact of the media on the spread of HIV/AIDS in African and African American communities

Materials for Activities and Educator Background Knowledge

There are a number of resources for this lesson module. All written resources can be found at the end of this module.

Readings for Activity 15.2

Case Studies of various diseases that crossed over from animals to humans or that have similarities between animal and human strains. Full texts can be found at:

Reading 15.1
http://www.cdc.gov/flu/avian/gen-info/facts.htm
Reading 15.2
http://www.cdc.gov/ncidod/diseases/hanta/hps/noframes/outbreak.htm
Reading 15.3
http://www.sciencedaily.com/releases/2007/08/070814135646.htm
Reading 15.4
http://www.cdc.gov/ncidod/dvrd/bse/
Reading 15.5
http://www.discoveriesinmedicine.com/General-Information-and-Biographies/Jenner-Edward.html

There is a video explaining two types of viral mutations on YouTube that will be used in ***Activity 15.1***.

http://www.youtube.com/watch?v=X3dSYA64ZRc

Procedure

Part I

View *"HIV and SIV"* video module. After viewing the module use the following questions to facilitate group discussion or give the questions as prompts for journal entries.

Discussion / Journal Questions

1) What new information did you gather from the video module?

2) What are the factors that may have allowed African primates to develop SIV? And, that may have allowed SIV to enter humans? How do these factors contribute to the high rates of HIV/AIDS in African and African American communities?

3) What other questions or comments do you have?

Part II

Specific viruses usually affect particular species. However, they have also been known to mutate and affect other species. This mutation and entry into different species tends to happen when there is a close physical relationship between species.

In ***Activities 15.1 and 15.2***, you will examine viral mutations and transference.

Activity 15.1

Deliberate on the transference of SIV from African primates to humans and the mutation of SIV to HIV.

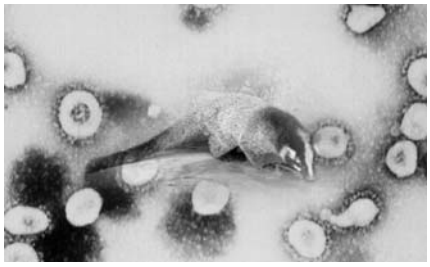
1. Watch the YouTube video on viral mutations.
2. In groups, discuss the close relationship between humans and primates in Africa. Hypothesize on the ways that SIV enters humans?

Part II (continued)

Activity 15.1 (continued)

3. What are possible guesses for how SIV mutated to HIV? (This question will connect to Lesson Modules 17—19.)
4. Discuss the myth of HIV having entered humans through sexual contact between humans and monkeys. How did this myth come to take hold in the minds of the public?
5. Do you think this myth would have taken hold if the myth was about another group of people (Europeans, Americans, Asians, Latinos, Native Americans, etc.)? Why or why not?

Activity 15.2



Examine how other diseases have entered human populations through close contact and whether the human populations of origin are blamed or not for these outbreaks. Break up into small groups to examine different diseases, assign each reading 15.1-15.5 to a group, and share findings in the larger group.

Reading 15.1: Smallpox and cowpox, the similarities between the diseases, the close interaction between milkmaids and cows, and the development of the first vaccine.

Reading 15.2: Hantavirus and its transfer from rodents into humans in areas of high rodent infestation in New Mexico.

Reading 15.3: Avian flu and its transfer from birds into humans through infected poultry.

Reading 15.4: West Nile virus and its transfer from birds into mosquitoes into humans in areas of high mosquito populations.

Reading 15.5: Mad Cow Disease and its transfer from cows into humans through the meat industry.

After examining these cases, ask students to discuss:

1. What are some conditions that allow for viral transference into another species?
2. Does race have anything to do with viral transference?
3. What are some conditions that exist in the United States that allow for viral transference? (factory farming, unsanitary conditions resulting from poverty, popularity of pets, etc.)

Closure

Sometimes, the conditions for viral transference results from individual actions. Other times, these conditions are created or exacerbated by societal practices.

Use the following questions to facilitate group discussion or give the questions as prompts for journal entries.

1. What are conditions in your community that may promote viral transference?
2. Who is responsible for these conditions?
3. What can you do as an informed citizen to affect these conditions?
4. What are individual actions that you can make to lessen your exposure to viruses that may transfer from another species?
5. Does understanding the viral transference change your thoughts about how you might act? Why or why not?

Reading 15.1

Key Facts About Avian Influenza (Bird Flu)
(excerpted from http://www.cdc.gov/flu/avian/gen-info/facts.htm)

This fact sheet provides general information about avian influenza (bird flu) and information about one type of bird flu, called highly pathogenic avian influenza A (H5N1), also called "HPAI H5N1," that has caused infections in birds and in humans.

Avian influenza is an infection caused by avian (bird) influenza (flu) A viruses. These influenza A viruses occur naturally among birds. Wild birds worldwide get flu A infections in their intestines, but usually do not get sick from flu infections. However, avian influenza is very contagious among birds and some of these viruses can make certain domesticated bird species, including chickens, ducks, and turkeys, very sick and kill them.

Human infection with avian influenza viruses

Usually, "avian influenza virus" refers to influenza A viruses found chiefly in birds, but infections with these viruses can occur in humans. The risk from avian influenza is generally low to most people, because the viruses do not usually infect humans. However, confirmed cases of human infection from several subtypes of avian influenza infection have been reported since 1997. Most cases of avian influenza infection in humans have resulted from contact with infected poultry (e.g., domesticated chicken, ducks, and turkeys) or surfaces contaminated with secretion/excretions from infected birds. The spread of avian influenza viruses from one ill person to another person has been reported very rarely, and has been limited, inefficient and unstained.

"Human influenza A viruses" usually refers to those influenza A subtypes that have spread widely among humans. Currently, H3N2 and H1N1 influenza A subtypes are circulating among humans and H2N2 influenza A circulated from about 1957-1968.

Some genetic parts of current human influenza A viruses had their origin in bird flu viruses originally. Influenza A viruses are constantly changing, and they might adapt over time to infect and spread among humans.

During an outbreak of avian influenza among poultry, there is a possible risk of infection for people who have contact with infected birds or surfaces that have been contaminated with secretions or excretions from infected birds.

Symptoms of avian influenza in humans have ranged from typical human influenza-like symptoms (e.g., fever, cough, sore throat, and muscle aches) to eye infections, pneumonia, severe respiratory diseases (such as acute respiratory distress), and other severe and life-threatening complications. The symptoms of avian influenza may depend on which virus caused the infection.

Reading 15.1

Highly pathogenic Avian Influenza A (H5N1)

Highly pathogenic Influenza A (H5N1) virus – also called "HPAI H5N1 virus" – is an influenza A virus that occurs mainly in birds, is highly contagious among birds, and can be deadly to them, especially domestic poultry. HPAI H5N1 virus does not usually infect people, but infections with these viruses have occurred in humans. Most of these cases have resulted from people having direct or close contact with H5N1-infected poultry or H5N1-contaminated surfaces.

Human health risks from HPAI H5N1

Of the few avian influenza viruses that have crossed the species barrier to infect humans, HPAI H5N1 has caused the largest number of detected cases of severe disease and death in humans. However, it is possible that those cases in the most severely ill people are more likely to be diagnosed and reported, while milder cases are less likely to be detected and reported. For the most current information about avian influenza and cumulative case numbers, see the World Health Organization (WHO) avian influenza website.

Of the human cases associated with the ongoing HPAI H5N1 outbreaks in poultry and wild birds in Asia and parts of Europe, the Near East and Africa, about 60% of those people reported infected with the virus have died. Most cases have occurred in previously healthy children and young adults and have resulted from direct or close contact with H5N1-infected poultry or H5N1-contaminated surfaces. In general, HPAI H5N1 remains a very rare disease in people. The HPAI H5N1 virus does not infect humans easily, and if a person is infected, it is very difficult for the virus to spread to another person.

While there has been some human-to-human spread of HPAI H5N1, it has been limited, inefficient and unstained. For example, in 2004 in Thailand, probable human-to-human spread in a family resulting from prolonged and very close contact between an ill child and her mother was reported. In June 2006, WHO reported evidence of human-to-human spread in Indonesia. In this situation, eight people in one family were infected. The first family member is thought to have become ill through contact with infected poultry. This person then infected six family members. One of those six people (a child) then infected another family member (his father). No further spread outside of the exposed family was identified.

Nonetheless, because all influenza viruses have the ability to change and because the HPAI H5N1 known ability to cause human infections, scientists remain concerned that HPAI H5N1 viruses have the potential to possibly change into a form of the virus that is able to spread easily from person to person. Because these viruses do not commonly infect humans, there is little or no immune protection against them in the human population. If HPAI H5N1 virus were to gain the capacity to spread easily from person to person, an influenza pandemic (worldwide outbreak of disease) could begin. For more information about influenza pandemics, see PandemicFlu.gov.

Reading 15.2

Hantavirus Pulmonary Syndrome (HPS)

(excerpted from http://www.cdc.gov/hantavirus/hps/history.html)

In May 1993, an outbreak of an unexplained pulmonary illness occurred in the southwestern United States, in an area shared by Arizona, New Mexico, Colorado and Utah known as "The Four Corners". A young, physically fit Navajo man suffering from shortness of breath was rushed to a hospital in New Mexico and died very rapidly. The young man's fiancée had died a few days before after showing similar symptoms. An investigation combing the entire Four Corners region located five young, healthy people who had all died after acute respiratory failure. During the next few weeks, as additional cases of the disease were reported in the Four Corners area, physicians and other scientific experts worked intensively to narrow down the list of possible causes. Virologists at CDC used several tests, including new methods to pinpoint virus genes at the molecular level, and were able to link the pulmonary syndrome with a virus, in particular a previously unknown type of hantavirus.

Researchers knew that all other known hantaviruses were transmitted to people by rodents, such as mice and rats. Therefore, an important part of their mission was to trap as many different species of rodents living in the Four Corners region as possible to find the particular type of rodent that carried the virus. Among rodents trapped, the deer mouse (Peromyscus maniculatus) was found to be the main host to a previously unknown type of hantavirus. Since the deer mouse often lives near people in rural and semi-rural areas—in barns and outbuildings, woodpiles, and inside people's homes—researchers suspected that the deer mouse might be transmitting the virus to humans. About 30% of the deer mice tested showed evidence of infection with hantavirus. Tests also showed that several other types of rodents were infected, although in lesser numbers.

In November 1993, the specific hantavirus that caused the Four Corners outbreak was isolated. The Special Pathogens Branch at CDC used tissue from a deer mouse that had been trapped near the New Mexico home of a person who had gotten the disease and grew the virus from it in the laboratory. Shortly afterwards and independently, the U.S. Army Medical Research Institute of Infectious Diseases (USAMRIID) also grew the virus, from a person in New Mexico who had gotten the disease as well as from a mouse trapped in California.

The new virus was called Muerto Canyon virus — later changed to Sin Nombre virus (SNV) — and the new disease caused by the virus was named hantavirus pulmonary syndrome, or HPS.

Why Did the Outbreak Occur in the Four Corners Area?

The key answer to this question is that, during this period, there were suddenly many more mice than usual. The Four Corners area had been in a drought for several years. Then, in early 1993, heavy snows and rainfall helped drought-stricken plants and animals to revive and grow in larger-than-usual numbers. The area's deer mice had plenty to eat, and as a result they reproduced so rapidly that there were ten times more mice in May 1993 than there had been in May of 1992. With so many mice, it was more likely that mice and humans would come into contact with one another, and thus more likely that the hantavirus carried by the mice would be transmitted to humans.

Reading 15.2

Person-to-Person Spread of HPS Decided Unlikely

"Although person-to-person spread [of HPS] has not been documented with any of the other known hantaviruses, we were concerned [during this outbreak] because we were dealing with a new agent", said Charles Vitek, a CDC medical investigator.

Researchers and clinicians investigating the ongoing outbreak were not the only groups concerned about the disease. Shortly after the first few HPS patients died and it became clear that a new disease was affecting people in the area, and that no one knew how it was transmitted, the news media began extensive reporting on the outbreak. Widespread concern among the public ensued.

Unfortunately, the first victims of the outbreak were Navajo. News reports focused on this fact, and the misperception grew that the unknown disease was somehow linked to Navajos. As a consequence, Navajos found themselves at the center of intense media attention and the objects of the some people's fears.

By later in the summer of 1993, the media frenzy had quieted somewhat, and the source of the disease was pinpointed. Researchers determined that, like other hantaviruses, the virus that causes HPS is not transmitted from person to person the way other infections, such as the common cold, may be. The exception to this is an outbreak of HPS in Argentina in 1996. Evidence from this outbreak suggests that strains of hantaviruses in South America may be transmissible from person to person.

To date, no cases of HPS have been reported in the United States in which the virus was transmitted from one person to another. In fact, in a study of health care workers who were exposed to either patients or specimens infected with related types of hantaviruses (which cause a different disease in humans), none of the workers showed evidence of infection or illness.

Reading 15.3

West Nile Virus

(excerpted from http://www.sciencedaily.com/releases/2007/08/070814135646.htm)
Science Daily (Aug. 15, 2007)

A gene mutation that appears to be responsible for changing relatively mild forms of the West Nile virus into a highly virulent and deadly disease in American crows has been identified by a team of scientists led by a researcher at the University of California, Davis.

Because it is highly susceptible to West Nile virus, the American crow has served as the major sentinel species, playing an important role in alerting scientists and health professionals to the movement of the disease across North America.

"The findings from this study highlight the potential for viruses like West Nile to rapidly adapt to changing environments when introduced to new geographic regions," said Aaron C. Brault, a virologist at the Center for Vectorborne Diseases in the Department of Pathology, Microbiology and Immunology of the UC Davis School of Veterinary Medicine.

"The study also suggests that the genetic mutations that create such adaptive changes may result in viral strains that have unexpected symptoms and patterns of transmission," Brault said.

About West Nile virus

West Nile virus, which is passed back and forth between birds and mosquitoes and transmitted to humans via mosquito bites, was first identified in 1937 in Uganda. Although it was recognized as a cause of severe encephalitis and meningitis (inflammation of the brain and spinal cord, respectively) during a 1957 outbreak in Israel, it has been primarily associated with mild infections accompanied by fevers in humans in Africa and the Middle East.

In 1996, West Nile virus caused an outbreak of encephalitis in Romania, moving on to cause similar outbreaks throughout the next several years in Israel, Tunisia and Russia.

In 1999, the virus was first recognized in North America and has since been reported in humans, birds, horses and mosquitoes in Canada and in all of the contiguous U.S. states. It has become the leading cause of encephalitis from a virus transmitted by arthropods, a group of invertebrates that includes insects, spiders and ticks.

West Nile in birds

A variety of North American bird species, including ring-billed gulls, house finches, crows and black-billed magpies, are extremely susceptible to West Nile virus. In fact, a hallmark of the West Nile virus in North America has been how deadly the virus has been among wild and captive birds. Particularly vulnerable to West Nile virus is the American crow, which is common in urban and suburban areas as

Reading 15.3

well as in all natural habitats except the Southwestern deserts.

Because the American crow is so common and so highly susceptible to West Nile virus, it has served as the sentinel species in North America. Epidemiological studies have found that deaths of American crows due to West Nile virus are associated with higher rates of infection among mosquito populations and clusters of the disease in humans. Although scientists and health professionals have thoroughly described how West Nile virus spreads through both human and animal populations in North America, it has been unclear just how the virus emerged to cause such serious disease in birds, particularly the American crow.

Funding for the study was provided by the Centers for Disease Control and Prevention, the National Institutes of Health and the Pacific Southwest Regional Center for Excellence.

Reading 15.4

Bovine Spongiform Encephalopathy, or Mad Cow Disease
(excerpted from http://www.cdc.gov/ncidod/dvrd/bse/)

BSE (bovine spongiform encephalopathy) is a progressive neurological disorder of cattle that results from infection by an unusual transmissible agent called a prion. The nature of the transmissible agent is not well understood. Currently, the most accepted theory is that the agent is a modified form of a normal protein known as prion protein. For reasons that are not yet understood, the normal prion protein changes into a pathogenic (harmful) form that then damages the central nervous system of cattle.

Research indicates that the first probable infections of BSE in cows occurred during the 1970's with two cases of BSE being identified in 1986. BSE possibly originated as a result of feeding cattle meat-and-bone meal that contained BSE-infected products from a spontaneously occurring case of BSE or scrapie-infected sheep products. Scrapie is a prion disease of sheep. There is strong evidence and general agreement that the outbreak was then amplified and spread throughout the United Kingdom cattle industry by feeding rendered, prion-infected, bovine meat-and-bone meal to young calves.

The BSE epizootic in the United Kingdom peaked in January 1993 at almost 1,000 new cases per week. Over the next 17 years, the annual numbers of BSE cases has dropped sharply; 14,562 cases in 1995, 1,443 in 2000, 225 in 2005 and 11 cases in 2010. Cumulatively, through the end of 2010, more than 184,500 cases of BSE had been confirmed in the United Kingdom alone in more than 35,000 herds.

There exists strong epidemiologic and laboratory evidence for a causal association between a new human prion disease called variant Creutzfeldt-Jakob disease (vCJD) that was first reported from the United Kingdom in 1996 and the BSE outbreak in cattle. The interval between the most likely period for the initial extended exposure of the population to potentially BSE-contaminated food (1984-1986) and the onset of initial variant CJD cases (1994-1996) is consistent with known incubation periods for the human forms of prion disease.

Overview of BSE in North America

Through February 2011, BSE surveillance has identified 22 cases in North America: 3 BSE cases in the U.S. and 19 in Canada. Of the 3 cases identified in the United States, one was born in Canada; of the 19 cases identified in Canada, one was imported from the United Kingdom (see figure above). Since March 2006, each of the 15 cattle reported with BSE in North America were born in Canada and identified through the Canadian BSE surveillance system.

As of March 2011, 19 BSE cases in Canadian-born cattle have been identified, 18 in Canada and 1 in the U.S. Of these 19 cases, 13 were known to have been born after the implementation of the 1997 Canadian feed ban External Web Site Policy; 12 of these 13 were born after March 1, 1999. This latter date is particularly relevant to the U.S. because since a USDA rule went into effect on November 19, 2007, Canadian cattle born on or after March 1, 1999 have been legally imported into this country for

Reading 15.4

any use. One of the 19 Canadian-born BSE cases was reported in an animal that was most likely born before or possibly very shortly after implementation of the 1997 feed ban. Based on the known or most likely year of birth, an average of 1.4 cases of BSE occurred among the group of animals born each year in Canada from 1991 through 2004. The highest reported number of cases by birth year in a single year, 3 BSE cases, occurred in 2000, 2001 and 2002. The most recently reported case extends the period of BSE transmission in Canada through at least the latter half of 2004.

As of October 26, 2009, a regulation issued by FDA in April 2009 came into effect establishing an enhanced BSE-related feed ban in the United States. This enhanced ban will further harmonize BSE feed control measures in the U.S. with those in Canada (see below). In addition, FDA continues to enforce its important 1997 mammalian-to-ruminant feed ban through its BSE inspection and BSE feed testing programs.

As of July 12, 2007, an enhanced BSE-related feed ban External Web Site Policy came into effect in Canada. CFIA External Web Site Policy established this ban to more effectively prevent and quickly eliminate BSE from Canada. The enhanced ban prohibits most proteins, including potentially BSE infectious tissues known as "specified risk materials" (SRM) from all animal feeds, pet foods, and fertilizers, not just from cattle feed as required by the ban instituted in 1997. The 1997 feed ban in Canada was similar to the feed ban instituted in the United States that same year. As recently reported by CFIA, removing SRM from the entire animal feed system addresses risks associated with the potential contamination of cattle feed during production, distribution, storage, and use. Applying the same measure to pet food and fertilizer materials addresses the possible exposure of cattle and other susceptible animals to these products. CFIA expects that with this new ban, BSE should be eliminated from the Canadian cattle herd by about the year 2017.

The Canadian-born cow confirmed to be infected with BSE in 2010 illustrates the difficulty in determining the effectiveness of previously instituted feed bans to prevent BSE transmissions. The initial feed bans established in both the United States and Canada were instituted in 1997. After an assessment by USDA and its Canadian counterparts, the Canadian feed ban was judged to be fully effectiveness as of March 1999. However, largely because of recognized limitations of this ban and the ban established in the United States, new, enhanced feed bans went into effect in Canada, July 12, 2007, and in the U.S., October 26, 2009. While USDA has confirmed no U.S.-born cattle as having a classic form of BSE, Canadian cattle born after March 1999 have been legally imported into the United States for any purpose since November 19, 2007.

Reading 15.5

Smallpox

(excerpted from http://www.discoveriesinmedicine.com/General-Information-and-Biographies/Jenner-Edward.html)

Edward Jenner (1749-1823) was a pioneer in the study of viruses and immunization against diseases. His work has been built upon by many successors who have discovered new vaccinations to reduce suffering and death, particularly for children.

Smallpox

Up until Jenner's time, smallpox was a common and often fatal disease worldwide. It caused high fevers and ugly pockmark scars, like those of chicken pox, only these scars could disfigure a person for life.

Many centuries before Jenner's time, the Chinese had begun the practice of blowing flakes from smallpox scabs up the nostrils of healthy persons to produce immunity to the disease. By the seventeenth century, the Turks and Greeks had discovered that, when injected into the skin of healthy individuals, the serum from the smallpox blister produced a mild case of the disease and subsequent immunity.

The practice of inoculation reached England by the eighteenth century. It was quite risky, since those who were inoculated frequently suffered a severe or fatal case of smallpox. Despite the risk, people willingly agreed to inoculation because of the widespread incidence of smallpox and the fear of suffering terrible disfigurement.

Jenner's Experiments

As a young physician, Jenner noticed that dairy workers who had been exposed to cowpox, a disease like mild smallpox, seemed immune to the more severe infection. Jenner continually put forth his theory that cowpox could be used to prevent smallpox, but his fellow physicians shunned his ideas. They maintained that they had seen smallpox victims who claimed to have had earlier cases of cowpox, so that cowpox must not give immunity. It became Jenner's task to transform a country superstition into an accepted medical practice.

After many years of observing cases of cowpox, Jenner took a step that could have branded him a criminal as easily as a hero. On May 14, 1796, he removed the fluid of a cowpox blister from dairymaid Sarah Nelmes, and inoculated James Phipps, an eight-year-old boy who soon came down with cowpox. Six weeks later, he inoculated the boy with smallpox. The boy remained healthy, and Jenner had proved his theory. Jenner called his method "vaccination," using the Latin word "vaccinia," meaning "cowpox."

The publication of Jenner's An Inquiry into the Causes and Effects of the Variolae Vaccinae set off an enthusiastic demand for vaccination throughout Europe. Within 18 months, the number of deaths from smallpox had dropped by two-thirds in England. By 1800, 100,000 people had been vaccinated worldwide.

Reading 15.5

As the demand for the vaccine rapidly increased, Jenner discovered that he could take lymph from a smallpox pustule and dry it in a glass tube for use up to three months later. The vaccine could then be transported.

Jenner became famous throughout Europe and the United States. Across the Atlantic Ocean, Thomas Jefferson received the vaccine from Jenner and proceeded to vaccinate his family and neighbors at Monticello. In his native England, however, Jenner's medical colleagues refused to allow him entry into the College of Physicians in London, insisting that he first pass a test on the theories of the ancient Greek physicians, Hippocrates (460-377 B.C) and Galen (A.D. 130-200). Jenner refused to bow to their demands, saying his accomplishments in conquering smallpox should have qualified him for election. He was never elected to the college.

Jenner continued to live in his country home and practice medicine in the years after his discovery. He also pursued his interest in birds and wrote papers on their behavior until his death in 1823. His work had far-reaching effects, inspiring Louis Pasteur (1822-1895) in his research on the causes of disease, thus leading to vaccines for other serious illnesses. And today, thanks to Jenner's curiosity and perseverance, smallpox has been largely eradicated (erased).