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# Joey's life with AIDS - Joey DiPaolo - includes related information on the spread and control of AIDS, both in the United States and around the world

[Science World](#), [Jan 12, 1998](#) by [Susan Hayes](#), [Emily Costello](#)

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Joey DiPaolo stuffs a french fry dripping with ketchup and melted mozzarella cheese into his mouth. Hanging out in his favorite diner dear his home in Staten Island, New York, Joey raps with friends about the School year, his 18th birthday, and his new girlfriend.

It seems like an average scene until the group is about to pay the check. Joey reaches into his pocket, whips out a vial of white pills, and pops two in his mouth -- the first sign all day the Joey DiPaolo has AIDS.

Doctors have pronounced many "deadlines" for Joey: a year to live, a few months, two days tops. But Joey is still going strong, in part because of improving medical treatment and a battery of potent AIDS-fighting drugs. Even newer, but much more costly drugs -- known as AIDS "cocktails" -- are now available. They may soon turn AIDS, a fatal disease, into a manageable illness. For now, however, 16 years into the epidemic, there is no cure for AIDS.

## THE FACE OF A KILLER

Joey contracted HIV (Human Immunodeficiency Virus, the virus that causes AIDS) at age four through a blood transfusion -- before the nation's blood supply became virtually 100 percent safe. HIV is spread through the exchange of blood or body fluids, mainly through sexual contact or intravenous drug use. Since contracting HIV, Joey has been in and out of hospitals, sampled one new drug after another, and had his body tested exhaustively. Perhaps the most incredible fact is that Joey is still alive at all.

It's easy to think of AIDS as a faceless killer. But 400,000 faces -- those of men and women, young and old, and babies -- have died of AIDS in America. Joey is one of an estimated 600,000 to 1 million Americans living with HIV (see chart on p. 13). The spread of HIV and AIDS appears to be leveling off among Americans in general, but among teens it is bounding upward. One in four new HIV infections occurs in people under age 22, according to the U.S. Centers for Disease Control and Prevention (CDC).

Too many people still haven't been tested for HIV, which can be detected by a blood test 6 to 12 weeks after initial infection. And because HIV can lurk in the body for years without revealing any symptoms, many people still don't know they're infected.

That's because HIV is a wily killer that can play hide-and-seek for years before it decimates the immune system, or body's defense against infection (see diagram, p. 8). In some ways, HIV is no different from other viruses -- microscopic particles that invade and often destroy animal and plant cells. Viruses boast no more than a simple core of genetic material within a protein "coat." Like any other living organism, viruses make genetic copies of themselves and evolve over time. But viruses lack critical "living" features: they don't have cells, they can't make their own proteins, and they don't use energy. So biologists consider viruses non-living.

Like other viruses, HIV invades a healthy cell and uses it as a factory to spawn copies of itself, destroying the cell in the process. But unlike most viruses, AIDS is lethal. HIV has a keen appetite for just one body cell -- the helper T cell, a white blood cell critical for the immune system to ward off disease. No one knows yet how HIV can lay dormant in T cells for years before bursting into action -- or why HIV is a master of disguise, able to mutate or create different strains of itself like a quick-change artist. But scientists do know that the virus eventually devours T cells, leaving the body open to a barrage of harmful bacteria, other diseases, and cancers. These afflictions mark the threshold of "full-blown" AIDS. Joey DiPaolo was 10 when he developed full-blown AIDS.

## CAUTION AND HOPE

Like many Americans living with AIDS, Joey's life is a mixture of caution and hope. "I have a normal life," he says. "I'm always on the go -- movies, the mall, bowling, hockey. I can do pretty much everything and anything. I never really think about AIDS," he adds, "except when I take my medicine."

Piles of bottles and jars filled with pills and capsules pack a kitchen cabinet at Joey's house. He consumes 22 pills and two doses of a liquid drug each day, including two drugs called AZT and DDI. They block HIV's ability to "upload" its genetic information to a host T cell, so the virus can't reproduce.

Every eight weeks Joey also injects himself twice a day, five days in a row, with an experimental AIDS drug, Interleukin-2. The drug can help the body manufacture more disease-fighting T cells. But all these drugs lose their power over time -- the most they can do is add a few years to Joey's life, doctors say.

Still, Joey's full-time treatment seems to work. For now, the amount of HIV in his blood has dropped to a barely detectable level. So he's decided not to take the newest and most promising AIDS drugs to date: protease inhibitors (see diagram, left).

In 80 percent of newly infected patients, protease inhibitors cause HIV levels to plummet to near-zero. But studies show the drugs aren't as effective for patients like Joey who have full-blown AIDS and have taken less-effective drugs for years, explains Margaret Fischl, director of the Comprehensive AIDS Program at the University of Miami School of Medicine. The new drugs are also so costly -- up to \$20,000 a year -- that few can afford them.

Some people experience side effects from the drugs like vomiting, diarrhea, or lack of energy. Others simply hate swallowing even more handfuls of pills every day. And the most willing patients can feel frustrated taking the drug arsenal as their doctors recommend -- some pills with a meal, others on an empty stomach. Many patients take their medication six separate times each day. "It drives patients crazy," says Fischl.

Joey agrees. "I know I'm not going to be able to comply with the regimen right now," he says, admitting he's a little lax about taking his medication. "And I'm glad I know that. Because if I were to ever go on protease inhibitors and mess it up, it would be bad for me."

Messing up carries a deadly risk. With missed doses, the drug level in the body can sink so low that even more lethal strains of HIV, called "super HIV," can flourish. Many doctors think twice before prescribing protease inhibitors to patients who may not be responsible about taking them properly.

HIV uses protease, an enzyme or protein that speeds up chemical reactions, to multiply "mature" or deadly virus particles in the blood-stream. Protease inhibitors assault virus particles before they become deadly. "The result is a non-infectious form of the virus," says Fischl. For people infected with HIV, protease inhibitors are the best hope right now.

The best hope for those not infected with HIV is sexual abstinence and avoidance of drugs. Scientists are working on a vaccine that may one day protect against exposure. But progress on an anti-HIV vaccine has crawled; partially because the virus mutates rapidly and each new strain makes old vaccines obsolete. Still, some researchers predict that they will create a successful vaccine within the next 10 years.

Joey keeps his hopes high, though his future may be grim. "If you think about living, you're going to live. If you think about dying, you're going to die," he says. "People who have the virus can live for a very long time."

## HOW HIV INFECTS A CELL

1. Knob-shaped proteins on HIV's surface bind to receptors on a healthy T cell. HIV uses the receptors to invade the cell.
2. HIV uses an enzyme called reverse transcriptase to make a "poisonous" DNA-version of HIV's RNA (DNA and RNA are genetic material that contain instructions for reproduction).
3. HIV inserts its own "poisoned" genetic code in the T cell nucleus, "tricking" the cell into becoming a virus factory.
4. HIV starts to form new protein strands, which assemble into new virus particles.
5. New HIV particles burst out, ready to invade more T cells.

## HOW PROTEASE INHIBITORS STOP HIV

Different AIDS drugs attack HIV in different ways. AZT, for example, tries to block HIV before it can reproduce in the T cell nucleus.

1. Protease inhibitors let HIV reproduce up to a point, allowing HIV to produce protein strands of immature viruses. 2. Normally, HIV then uses an enzyme called protease, which acts like a scissors. The enzyme clips long protein strands into short ones so the virus can assemble into efficient killer particles. 3. But that's when protease inhibitors go to work. They suppress protease so that the viruses remain immature and harmless.

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