

HIV Cure Research Information Sheet

HIV CURE RESEARCH STATUS: QUICK SUMMARY

- There are currently nine examples of people considered to be cured, or very likely cured, of HIV infection (in most cases they continue to be monitored for any signs of HIV returning).
- Seven of these cases resulted from stem cell transplants that were required to treat life-threatening cancers. In most cases the stem cell transplants were sourced from donors who possessed a rare genetic mutation that makes their cells resistant to the majority of HIV variants.
- Two cases involve elite controllers, a rare group of people who can control HIV viral load to undetectable levels without ART. Evidence indicates that these two individuals have cleared all viable HIV from their bodies over time. Reports suggest there may be additional similar elite controller cases, and they are now being studied.
- These outcomes remain rare and resulted from exceptional circumstances, but they are providing important clues to researchers working to develop a broadly applicable cure.
- There are more numerous — but still relatively rare — examples of individuals who have controlled HIV viral load to low levels either naturally (elite controllers) or after an ART interruption (posttreatment controllers; most commonly after beginning treatment early), but it is not always certain how long this immune-mediated control will last and if it may come at some cost to long-term health.
- The first person to be identified as cured of HIV, Timothy Ray Brown, died in 2020 because of a recurrence of cancer but has left a profound legacy of inspiration and activism that spurs the HIV cure research effort we see today.
- While many different therapeutic approaches are being studied, so far no broadly usable interventions have produced clear evidence of cures or remissions — the best reported results involve small reductions in the amount of HIV that persists in the body despite treatment (the HIV reservoir) and some cases of extended control of HIV viral load to low levels after ART interruption.

BACKGROUND

Combination antiretroviral therapy (ART) is a highly effective treatment for HIV infection, preventing progression to AIDS (Acquired Immune Deficiency Syndrome) in most recipients who have access and can adhere consistently. When ART is started early in the course of infection, the lifespan of HIV-positive people is typically close to that of comparable HIV-negative people. But ART can have side effects, is often costly, and requires ongoing adherence to the regimen. Because of both the limitations of ART and the stigma people with HIV can still face, a cure remains a vital goal for research.

In the mid-1990s, when it was first shown that triple combinations of antiretroviral drugs could suppress HIV replication, there were hopes that long-term ART would eventually cause all HIV-infected cells in the body to die off, curing the infection. But scientists discovered that HIV persists in long-lived immune system cells known as memory CD4 T cells and these cells can become active and produce infectious viruses when ART is interrupted.

For this reason, HIV viral load almost always returns rapidly if ART is stopped. The HIV that persists despite ART is described as the HIV reservoir and is considered the major barrier to a cure.

Studies have shown that the HIV reservoir in CD4 T cells typically declines very slowly over time in people on ART. Scientists are investigating whether there's any possibility of eventual clearance of all viable virus in some people on long-term ART (20 years or more). But currently it appears that additional therapies will be needed to achieve a cure.

UNDERSTANDING HIV PERSISTENCE

HIV primarily persists in long-lived memory CD4 T cells in people on ART but can persist in several different states:

- Defective and incapable of replicating, but in some cases able to produce partial virus components (RNA and/or proteins)
- Intact, capable of replicating but asleep (latent) during ART and invisible to the immune system (latent reservoir)
- Intact, capable of replicating and persistently or intermittently generating infectious HIV that is blocked from infecting other cells by ART (active reservoir)
- Intact but trapped in the CD4 T cell and unlikely to be able to emerge and replicate

Every cell in the body, except for red blood cells, contains a copy of the entire genome (the genetic blueprint for the body). The genome consists of DNA and can be thought of as a production line capable of manufacturing all the protein components that make up your body (this is done via an intermediate step wherein the DNA makes RNA, which then makes proteins).

Different cells employ just the parts of the DNA production line they need to make the proteins that allow them to do their job, e.g., kidney cells make the proteins they use in clearing waste, and immune system cells, like CD4 T cells, make proteins needed for the work they do in responding to infections.

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HIV persists by integrating its own DNA into the DNA of the cells it infects, primarily CD4 T cells.

CD4 T cells can be in an activated state when they are responding to something — this requires the DNA production line to be busy, making the proteins the cell needs to go about its work. But CD4 T cells can also de-activate into a long-term resting “memory” state, which causes the DNA production line to largely shut down. The development of memory CD4 T cells is an important process because it underlies the immune system’s ability to respond more rapidly to an infection the second time a person is exposed.

HIV prefers to replicate in activated CD4 T cells because it hijacks the busy DNA production line to manufacture more viruses, which can then exit and infect other cells. But if HIV infects a CD4 T cell when the DNA production line is in the process of shutting down (or is shut down), the virus can essentially become trapped in the machinery, making it difficult to manufacture more viruses unless memory CD4 T cell becomes activated again and the DNA production line cranks back up.

In technical terms, HIV DNA becomes integrated into the genome of the resting memory CD4 T cell. The process is not always entirely successful, and researchers have discovered that, in most people, the majority of integrated HIV in people on ART is defective (missing parts of the HIV genetic code) and unable to replicate. But enough intact HIV persists to be able to restart replication if ART is stopped.

For a long time, scientists thought that while a CD4 T cell remains resting, HIV stays in an inactive or “latent” state, rendering the infected cell invisible to the immune system.

More recently, it’s been discovered that some CD4 T cells containing integrated HIV do manufacture virus proteins, either persistently or intermittently. Scientists now refer to this subset of HIV-infected cells that persist despite ART as the “active reservoir.”

While a person remains on ART, any new intact, infectious HIV that is generated by the active reservoir cannot go on to infect other cells. The hope is that this newly identified part of the HIV reservoir is more vulnerable to clearance by the immune system because the HIV proteins that are made act as a warning flag that the cell is infected. Other immune cells such as CD8 T cells and natural killer cells can potentially recognize this warning flag and destroy the infected cell.

In some rare cases, the active reservoir can generate enough HIV to cause a low-level reading on standard viral load tests. For doctors treating people with HIV, it’s important to be aware that a low-level viral load that doesn’t respond to changes in ART regimen may be caused by this phenomenon.

As new technologies allow researchers to better probe the nature of the HIV reservoir, scientists have found that there’s a subset of CD4 T cells with integrated HIV that may not present a concern for cure research. In these cells, HIV has integrated into what is essentially a dead end in the cell’s DNA. Once integrated into these areas — called gene deserts — the virus lacks access to the factors needed to reactivate and appears trapped. A cure might not need to eliminate these cells.

CD4 T CELL PROLIFERATION

An important factor that contributes to the persistence of the HIV reservoir is the ability of CD4 T cells to proliferate (copy themselves). This is a normal part of immune system function. When a CD4 T cell needs to respond to an infectious agent, it can proliferate and generate a swarm of CD4 T cells all targeting the same problem. Memory CD4 T cells can also proliferate intermittently to maintain their numbers. As an example, the memory CD4 T cells generated by a measles infection in childhood will typically be maintained for life.

In recent years researchers have shown that when a CD4 T cell with HIV integrated into its DNA proliferates, the newly generated copies of the cell also contain the integrated HIV because they are exact duplicates (referred to as “clones”). The process is now recognized as an important mechanism by which the HIV reservoir is sustained. These findings have led some scientists to investigate whether limiting the proliferation of HIV-infected CD4 T cells can represent an additional approach to reducing the HIV reservoir.

Current evidence indicates that HIV can also persist in other cell types, such as macrophages, stem cells, and certain brain cells. But there is still uncertainty regarding whether HIV can become latent and then subsequently reactivate to produce infectious virus in these cells, as it can in the case of CD4 T cells. Ongoing research is attempting to definitively ascertain if cells other than CD4 T cells contribute to the HIV reservoir.

EVIDENCE THAT AN HIV CURE IS POSSIBLE

STEM CELL TRANSPLANT HIV CURE CASES

The first clear evidence that an HIV cure is possible came from the case of a single person, Timothy Ray Brown (initially known as the Berlin patient).

Brown had been HIV-positive since 1995 and taking ART for around four years when, in 2006, he developed acute myelogenous leukemia (AML) — a potentially life-threatening cancer. The diagnosis led to the need for a stem cell transplant, a risky treatment that essentially creates a new immune system in the recipient by transferring bone marrow cells donated by another individual. Chemotherapy drugs and radiation are used to wipe out the existing immune system and make way for the donor cells. The procedure can only be used in life-threatening cancers because it carries a significant risk of death.

The doctor supervising Brown’s cancer treatment, Gero Hütter, successfully identified a bone marrow donor with a rare genetic mutation (designated CCR5Δ32) that causes immune system cells to be resistant to most strains of HIV. When it is inherited from both parents, the CCR5Δ32 mutation prevents immune system cells from being able to display a protein, named CCR5, that the most common forms of HIV use as a latch to enter target cells.

Brown received two stem cell transplants from the donor as part of a difficult course of treatment for the AML. The cancer was ultimately cured, and

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even though ART had been interrupted, tests also revealed that HIV viral load had not rebounded. Hütter first reported the case at a scientific conference in 2008, at which time Brown had remained off ART with no detectable HIV in blood or tissue samples for 285 days.

Follow up continued for more than 12 years with no sign of a return of HIV. Timothy Ray Brown became an important advocate for HIV cure research, and it was a great loss when he died on September 29, 2020, due to a recurrence of cancer.

There have since been four additional cases of likely HIV cures reported in people with HIV who required stem cell transplants to treat life-threatening cancers. Following Brown's trailblazing path, most received stem cells from donors who'd inherited the CCR5 Δ 32 mutation from both parents (CCR5 Δ 32 homozygotes). Two recently reported cases have provided evidence that the mutation may not always be necessary (see below), suggesting that other aspects of the stem cell transplantation procedure can contribute to HIV clearance - follow up and investigations are ongoing.

- Adam Castillejo, initially known as the London patient, first reported in 2019 and now off ART without HIV viral load rebound for more than six years.
- Marc Franke, the Düsseldorf patient, also first reported in 2019, off ART for over five years.
- The New York City patient, a woman of mixed race first reported in early 2022 and still being followed. The first cisgender woman reported to have potentially been cured by the approach.
- Paul Edmonds, the City of Hope patient, reported in July 2022. A long-term survivor who was diagnosed with HIV in 1988, representing the oldest individual described to date at age 66.

- The Geneva Patient, who has disclosed his first name as Romuald, reported at the 2023 International AIDS Society Conference. Romauld is the first case to have received a stem cell transplant from a donor lacking the CCR5 Δ 32 mutation.
- A second Berlin patient who remains anonymous, described in a presentation at the 2024 International AIDS Conference. In this instance, the stem cell transplant donor was heterozygous for the CCR5 Δ 32 mutation meaning they had reduced CCR5 levels on their cells (as opposed to the absence of the CCR5 receptor in homozygotes).
- A second woman with comparatively short follow up off ART of one year, reported at the HIV Glasgow conference in November 2024.

The expectation is that additional cases will emerge as stem cell transplant procedures are refined and identification of CCR5 Δ 32 homozygote donors becomes more common for people with HIV in this situation. IciStem, a collaborative project based in Europe is specifically working to track and analyze potential HIV cures achieved by the approach.

The risk of illness and death associated with stem cell transplants mean they are not a practical approach to curing HIV-positive people without cancers, but these cases nevertheless represent a beacon of hope for the future.

CASES OF TEMPORARY ABSENCE OF ANY DETECTABLE HIV

There are reports describing several individuals who've experienced a transient period without any detectable HIV reservoir or viral load after interrupting ART (sometimes referred to as "HIV remission"). These cases also provide some reasons for optimism that a broadly applicable cure may eventually be possible.

The Mississippi Baby

One of the most widely publicized examples is the Mississippi baby. Born to a mother whose HIV infection was not diagnosed until in labor, the neonate was started on ART within hours of delivery. Treatment was maintained for around 18 months, at which time the mother and baby temporarily stopped attending medical follow-up visits. When they returned to care, doctors learned that ART had been interrupted in the infant but, surprisingly, HIV viral load remained undetectable.

The initial theory was that rapid initiation of ART might have prevented the formation of the HIV reservoir, leading to a cure. The Mississippi baby remained off ART for a little over two years with no measurable HIV but then experienced a rebound in viral load, requiring treatment to be restarted.

Researchers now believe that the very early start of ART greatly limited the number of CD4 T cells containing integrated HIV; however, it's likely a few were present and, eventually, one or more became activated, leading to renewed HIV production. CD4 T cells can become activated for several reasons, most commonly due to encountering an infectious agent or other substance that they recognize and respond to — part of their job as immune system cells.

The outcome in the Mississippi baby case highlighted that the HIV reservoir can persist at levels undetectable by current technologies, and that long-term monitoring is essential even if it might at first appear that an individual has been cured.

The PrEP Demonstration Project Participant

The closest adult equivalent to the Mississippi baby is an individual who was diagnosed with HIV extraordinarily early (within approximately 10 days) due to acquiring the infection during the short window of time between screening for participation in a pre-exposure prophylaxis (PrEP) demonstration project and the day they were started on the first dose of the PrEP drug Truvada.

Combination ART was begun as soon as the HIV diagnosis was confirmed, and subsequent tests conducted while on treatment were unable to detect HIV. After 34 months, the individual agreed to interrupt ART, and HIV remained undetectable by any measure for 224 days. Viral load then rebounded, necessitating reinstatement of treatment.

The Boston Patients

The Boston patients are two men living with HIV who required stem cell transplants to treat cancers. They did not receive cells from donors with the CCR5Δ32 mutation, but nevertheless, HIV became undetectable after the procedures. This was likely due to treatments given to wipe out existing immune cells (including CD4 T cells) to allow the transplanted stem cells to flourish and generate a replacement immune system derived from the donor.

ART was maintained throughout, leading researchers to suspect that their new donor-derived immune system cells may have been protected from the virus. Both individuals also

developed a condition known as graft-versus-host disease (which involves donated cells attacking recipient tissues) after their transplants, and this was thought to have potentially contributed to the clearance of HIV-infected cells. ART was eventually interrupted, and HIV remained undetectable for 12 weeks in one case and 32 weeks in the other before viral load re-emerged and treatment was reinitiated.

The Mayo Clinic Patient

Early in 2017, researchers from the Mayo Clinic in Rochester described another HIV-positive man with similarities to the Boston patients. He also received a stem cell transplant from a donor lacking the CCR5 Δ 32 mutation as part of treatment for cancer and continued ART after the procedure, displaying declining levels of HIV reservoirs that ultimately became undetectable. A little over two years after the transplant, ART was interrupted, leading to a period of remission from detectable HIV viral load that lasted 288 days. Viral load tests then revealed that HIV replication had restarted, and the individual resumed ART.

Possible Lessons

These cases of transient absence of any detectable HIV are connected by the fact that all appeared to result from the HIV reservoir being very small at the time of ART interruption.

The size in the two Boston patients has been estimated as 290 to 2900 latently infected cells and 40 to 730 latently infected cells, respectively (an estimated reduction of more than three logs — 1,000-fold — compared to the pretransplant baseline).

This is potentially important because it suggests that one of the central goals of HIV cure research — shrinking the size of the HIV reservoir — can at least significantly delay the rebound of HIV when ART is interrupted.

Mathematical modeling studies conducted by researcher Alison Hill indicate that achieving even greater reservoir reductions — perhaps on the order of over four logs (10,000-fold or >99.99%) — could lead to a lifelong cure in most individuals. Reducing the HIV reservoir to this extent represents a significant challenge, but at least there is a sense of a target to aim at.

The other link between these cases is that the period without any detectable HIV appears to have been caused by the few HIV-infected CD4 T cells that were present remaining dormant, rather than the immune system actively controlling HIV.

No significant immune responses against HIV could be detected in any of the individuals, which was expected because of the rapidity with which ART was started in the Mississippi baby and PrEP demonstration project cases (suppressing the virus before the immune system mounted a response), and due to the fact that the Boston and Mayo Clinic patients developed new immune systems — which had not yet encountered HIV — from their HIV-negative stem cell transplant donors.

The absence of immune responses appears to make this phenomenon distinct from another form — referred to as posttreatment control — that has also received attention in mainstream media coverage of HIV cure research.

POSTTREATMENT CONTROL AND ELITE CONTROL

The best-known examples of posttreatment control are the VISCONTI cohort. Identified by researchers in France, this unusual group of HIV-positive individuals began ART early in infection, continued for several years, and then interrupted and maintained viral loads at low or undetectable levels for an extended period — in some instances for over a decade.

Several other individual case reports have broad similarities. These include a French teenager and a nine-year-old South African child who both acquired HIV at birth, received a limited period of ART, and at the time of first reporting had displayed control of HIV viral load for 12 and 8.75 years, respectively. According to an update on the South African child in 2022, viral load remains undetectable after 14 years of follow up.

More recently, in July of 2022, researchers from Spain described the case of a woman now over 70 years of age who participated in a trial involving an interruption of ART over a decade ago and has since maintained an undetectable HIV viral load for more than 15 years. Additionally, the size of the HIV reservoir has continued to decline over that time.

Posttreatment controllers generally display immune responses against HIV, including antibody and CD4 and CD8 T cell responses, although there is considerable individual variability. The prevailing theory is that these cases represent some sort of active containment of HIV replication by the immune system. Based on this evidence, the possibility of inducing immunological control of HIV is an avenue being explored by cure researchers.

A potential concern about posttreatment control as a model for an HIV cure relates to the

parallels with rare HIV-positive individuals known as elite controllers (the frequency is estimated to be around 1% or less). Elite controllers suppress viral replication to undetectable levels for many years without ART — a phenomenon associated with strong and effective immune responses targeting the virus, particularly CD4 and CD8 T cells. Certain genetic traits that influence the performance of CD8 T cells have been shown to increase the likelihood of becoming an elite controller.

The caveat is that long-term studies have found that elite control is not necessarily completely protective against disease progression. The efforts of the immune system to control HIV can be associated with increased levels of inflammation and a slow decline in CD4 T cell numbers, ultimately leading to AIDS (albeit at a far slower pace than is observed in individuals with higher viral loads). It has been reported that some posttreatment controllers in the VISCONTI cohort have experienced this type of slow progression since the time the cohort was originally described.

More optimistically, a subset of elite controllers exhibit extraordinarily strong control of HIV, and they may offer cure researchers a model of immune-mediated containment with less potential for detrimental effects.

In at least two elite controllers, there is evidence that all HIV capable of replicating has been cleared from their bodies, which researchers believe likely represents an immune-mediated cure. One is Loreen Willenberg, a longtime advocate for research into elite control, and the other is a woman who's chosen to remain anonymous and is known as the Esperanza patient, named after the Argentine city in which she lives (the English translation of Esperanza is "hope").

Examples of HIV remission and posttreatment control underscore the rationale for pursuing a cure for HIV but, in some cases, also illustrate a key difficulty facing HIV cure researchers: even if success appears to have been achieved, people will need to be followed for a long period to ensure there is no return of viral load or any negative health consequences if some virus remains present but controlled.

RESEARCH APPROACHES

Current strategies being pursued in HIV cure research include:

- Limiting the size of the HIV reservoir by initiating ART as soon as possible and/or reducing proliferation of CD4 T cells containing HIV
- Promoting clearance of cells containing intact HIV
- Enhancing the ability of the immune system to control any remaining HIV when ART is stopped
- Protecting vulnerable cells from HIV infection so the virus has nowhere to go when ART is stopped
- Blocking remaining intact HIV from being able to emerge from infected cells

RESERVOIR LIMITATION

Many studies have shown that starting ART as soon as possible after HIV infection occurs greatly

limits the size of the HIV reservoir formed. The longer the duration of untreated HIV infection, the larger the size of the HIV reservoir. For this reason, people who started ART early may be ideal candidates for interventions seeking to deplete the HIV reservoir, and many research studies aim to recruit this population. Early diagnosis of HIV can be challenging in adults because the acquisition of infection is not necessarily accompanied by symptoms, and even if symptoms occur, they are typically flu-like and nonspecific.

Newborns who have acquired HIV infection from their mothers represent a population in which early diagnosis is more feasible. The administration of ART shortly after birth appears to have played a key role in the Mississippi baby case. A large clinical trial, IMPAACT P1115, is evaluating whether early ART (with or without additional interventions) can lead to remissions or cures in newborns who acquired HIV because their mothers did not receive ART to prevent mother-to-child transmission (MTCT) or because MTCT was inadequate.

RESERVOIR CLEARANCE

When HIV cure research first began to expand, the scientific evidence indicated that the reservoir of virus that persists in people on ART was in a latent state. Researchers believed that strategies would be needed to essentially wake up the latent HIV and make it active so infected cells would die either because of virus replication or because they became visible to the immune system. The terms “kick & kill” or “shock & kill” were applied to this approach.

A variety of interventions that appear able to awaken latent HIV (known as latency-reversing agents or LRAs) have since been identified and tested in clinical trials, but little evidence of depletion of the HIV reservoir has been reported.

These findings, together with the discovery that the HIV reservoir can be more active than previously known, have led to some reevaluation of the kick & kill strategy.

Latency-reversing agents may still be needed, but researchers are looking to identify safer and more effective compounds than those tested initially.

A large variety of strategies that may have the capacity to promote the death of HIV-infected cells are also being evaluated. These include drugs to promote a natural death pathway called apoptosis and experimental candidates designed to promote the clearance of the HIV reservoir by the immune system.

IMMUNE ENHANCEMENT

For many common viral infections, the immune system is highly effective at containing or clearing the virus. But in people with HIV, many of the immune system cells that target the virus have been shown to be dysfunctional, in part because the virus infects CD4 T cells that would normally coordinate the immune response.

Researchers are therefore investigating whether effective immune responses to HIV can be restored or created by new therapies.

Among the candidates are therapeutic vaccines and broadly neutralizing antibodies (bNAbs), which can inhibit the replication of many different HIV strains. In addition to inhibiting HIV, bNAbs may also have the potential to flag HIV-infected cells for destruction by the immune system.

Clinical trials of bNAbs have produced some potentially encouraging results, with a very small number of participants showing evidence of prolonged posttreatment control after ART interruption.

At least one trial has reported evidence of a slight decline in the intact HIV reservoir associated with bNAb therapy, but this needs to be confirmed in additional studies.

Chimeric Antigen Receptor (CAR) T cells represent an approach to enhancing immunity via gene therapy. T cells are genetically modified in the laboratory with the aim of making them better at recognizing and destroying HIV-infected cells. CAR T cells are approved for the treatment of certain cancers.

A wide variety of other immune-based approaches are under investigation, including immune system signaling proteins (cytokines such as IL-15 and alpha interferon); immune stimulants called toll-like receptor (TLR) agonists; and immune checkpoint inhibitors, which have shown efficacy in restoring the ability of dysfunctional immune responses to fight cancers.

Several immune checkpoint inhibitors are now approved as cancer therapies. However, initial studies in HIV have raised concerns about side effects. Research is continuing to assess whether safety can be improved.

CELL PROTECTION

The experience of the Boston and Mayo Clinic patients suggests that receipt of a stem cell transplant from a donor with the CCR5Δ32 mutation was an important contributor for Timothy Ray Brown and the similar cure cases that have been reported since. This provides a rationale for researchers attempting to mimic the effects of the CCR5Δ32 mutation using gene therapies.

These approaches focus on CD4 T cells, HIV's primary target. Researchers are testing gene-editing technologies that can prevent the CCR5 receptor from being displayed on cells. CD4 T cells, in some cases together with stem cells, are extracted from HIV-positive individuals, genetically modified, and then expanded in number and reinfused. There are also gene therapies that aim to equip cells with single or multiple proteins capable of blocking HIV replication.

A significant challenge for gene therapies is modifying enough cells to provide a benefit, and studies are investigating different methods to try to maximize the number of HIV-resistant cells created.

For people with HIV and life-threatening cancers requiring treatment with stem cell transplants, several clinical trials are employing gene therapy to modify stem cells from normal donors before transplantation to render the cells resistant to HIV in the absence of the natural CCR5Δ32 mutation.

BLOCK AND LOCK

Rather than promoting the clearance of the HIV reservoir, some researchers are testing

whether it might be possible to permanently trap the virus so it cannot exit infected cells and replicate. The idea is to lock down HIV's genetic material within the cell (termed "block and lock"). So far, it has only been explored in laboratory studies, and it's unclear if it has the potential advance into clinical trials.

RISKS, BENEFITS, AND ETHICS

Because HIV cure research is currently in an early phase, there are potentially significant risks and little or no prospect of any study participant being cured. Of particular concern is the use of analytical treatment interruptions (ATIs) in clinical trials, which require ART to be stopped and careful monitoring to try to prevent both risks to health and potential transmission when HIV viral load is detectable.

There are ongoing dialogues between scientists and the community of people with HIV and advocates about how best to define and communicate the potential risks and benefits of participation in HIV cure research. The goal is to ensure that all studies are conducted ethically, and any potential risk is minimized.

This is particularly important now that ART is so effective that most HIV-positive people can expect to live into old age.

RELATED LINKS

TAG maintains a regularly updated listing of clinical trials and observational studies related to the research effort to cure HIV infection: <http://www.treatmentactiongroup.org/cure/trials>

A related resource is tracking the demographics of participation in HIV cure-related clinical research: <https://www.treatmentactiongroup.org/cure/resource-on-the-demographics-of-participation-in-hiv-cure-related-clinical-research/>

An annual description of approaches under investigation in HIV cure research can be found in TAG's Pipeline Report: <https://www.treatmentactiongroup.org/resources/pipeline-report/>

A resource page containing articles, reports, and links to other organizations and websites relevant to cure research is available at: <http://www.treatmentactiongroup.org/cure>

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