Key Media Coverage

20th International AIDS Conference (AIDS 2014)
Melbourne, Australia
July 20-25, 2014

towards an HIV cure
people focused science driven

International AIDS Society
Stronger Together Against HIV
## HIV Cure  AIDS 2014. Melbourne, Australia

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Celgene drug can drive HIV out of hiding: study

BY KATE KELLAND
LONDON Tue Jul 22, 2014 8:44am EDT

(Reuters) - An anti-cancer drug made by the U.S. biotech firm Celgene can re-activate hidden HIV in patients so that it can be detected, bringing researchers closer to being able to treat it, Danish scientists said on Tuesday.

In a small study presented at an international AIDS conference in Australia, the researchers said the finding was a "step in the right direction" toward finding a cure for the viral disease but that many years of research are still needed.

"There is still a long way to go and many obstacles to overcome before we can start talking about a cure against HIV," said Ole Schmeltz Sogaard, who led the research team from Aarhus University and Aarhus University Hospital in Denmark, in a statement.

The drug, known generically as romidepsin and under the brand name Istodax, is licensed to treat a type of cancer called T-cell lymphoma. In this study, however, it was investigated as a potential HIV therapy.

Human immunodeficiency virus (HIV) infection can be kept at very low levels by anti-AIDS drugs, but there is still no cure that can eradicate HIV from the body.

Some 35 million people worldwide are infected with HIV, and the global AIDS epidemic has killed 39 million since it began in the 1980s, according to the latest data from the United Nations AIDS program, UNAIDS.

Scientists working to find a cure know the virus can hide in a state of hibernation in cells called CD4 cells, which are part of the body’s immune system.

CD4 cells cannot fight the AIDS virus themselves, but killer T-cells can if they are able to tell whether or not a CD4 cell contains the hibernating HIV.

Sharon Lewin, co-chair of the AIDS2014 conference in Melbourne Australia and a professor of infectious diseases who was not directly involved in this study, said the results of the study were
significant and encouraging because they showed "we can wake up the virus reservoir and make enough of (it) to leave the cell, making it visible to an immune response".

The Danish team gave three once-weekly infusions of romidepsin to six HIV-positive adult patients who were already taking antiretroviral AIDS drugs and whose so-called "viral load" was undetectable.

They found that romidepsin increased the virus production in HIV-infected cells between 2.1 and 3.9 times above normal and that the viral load in the blood increased to measurable levels in five out of six patients.

"We have now shown that we can activate a hibernating virus with romidepsin and that the activated virus moves into the bloodstream in large amounts," Schmeltz Sogaard said in a statement about the results.

When the virus is activated and moves toward the bloodstream it leaves a trace on the outside of the infected CD4 cells, he explained. In principle, this means killer T-cells would be able to trace and destroy the HIV-infected CD4 cells.

The Danish team said the next step is a larger trial where the researchers will combine romidepsin activation of hidden HIV with an experimental vaccine called Vacc-4x being developed by the Norwegian biotech firm Bionor Pharma to strengthen the ability of T-cells to fight HIV.

(editing by Jane Baird)

http://www.reuters.com/article/2014/07/22/us-health-aids-virus-idUSKBN0FR1AC20140722
HIV Said Cleared in Two Bone Marrow Transplant Patients
By Kanoko Matsuyama and Simeon Bennett Jul 18, 2014 5:00 PM GMT+0200

Two cancer patients who were also infected with HIV went through bone marrow transplants and may no longer have the AIDS-causing virus, according to Australian doctors.

The DNA of HIV or antibodies against the virus weren’t found in the patients who suffered from non-Hodgkin’s lymphoma and leukemia and underwent the transplants in 2010 and 2011, according to a statement from the University of New South Wales. Both men continue to take antiretroviral therapy after the transplants, the university said.

The two patients remain on the therapy because of risks the virus may return, said Sam Milliken, a hematologist at St. Vincent’s Hospital in Sydney who helped lead the research. Previously, HIV returned to two transplant patients in Boston who went off the treatments and were thought to have been free from the virus.

“If we understand what the process was, why the transplant has this very strong anti-HIV effect, that would be very fascinating,” said David Cooper, an HIV specialist at the Kirby Institute of University of New South Wales and another leader of the research. “If we can understand how to harness that, then we may do better than some of the other cure strategies that are around.”

The Australian findings will be presented at a symposium which is part of the 20th International AIDS Conference in Melbourne to be held July 20-25. The researchers will study more patients to understand the mechanism that allowed the suppression of the virus to obtain insight to treat HIV better, said Milliken.

A number of centers have reported that transplants during antiretroviral therapy can lead to dramatic reduction in the amount of HIV, said Steven Deeks, a professor of medicine at University of California, San Francisco.

“Without a treatment interruption and prolonged virus-free periods (e.g., at least a month), there is not much new here,” Deeks said via e-mail.

Only one person, American Timothy Ray Brown, is known to have been cured after he had two transplants in Berlin. The second donor’s bone marrow had included both copies of genes that afford protection against HIV.

A Mississippi baby thought to have been cured of HIV was recently found to have the virus again after two years without therapy.
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AIDS Cure Quest Advances as Cancer Drug Rouses Hidden HIV
By Jason Gale and Simeon Bennett Jul 22, 2014 6:02 PM GMT+0200

AIDS researchers seeking to cure HIV with a one-two punch got an early hint the approach may work by successfully using an old cancer drug to kick the virus out of hiding in a pilot study in Denmark.

Researchers from Aarhus University gave the medicine romidepsin to six HIV-infected people in an effort to rouse the virus from the so-called reservoirs where it sleeps. It worked: Infusions of the drug woke the virus up and caused it to start reproducing, a step that may allow the immune system to clear it, according to results reported today at an International AIDS Conference in Melbourne.

Antiretroviral pills can keep the virus in check, but don’t eliminate it from hidden reservoirs deep within the body. Studies have shown that when patients stop taking their meds, the virus proliferates from the reservoirs, forcing them to resume treatment. Bionor Pharma ASA (BIONOR), based in Oslo, is studying romidepsin as part of a “kick-and-kill” approach to curing HIV in which romidepsin kicks HIV out of hiding before another drug called Vacc-4x would prompt the immune system to kill it.

“It’s still just another step toward something that may end up being a cure for HIV, so it’s a step in the right direction,” co-author Ole Schmeltz Sogaard, a senior researcher in the department of infectious diseases at Aarhus University Hospital, told reporters in Melbourne.

Well Tolerated

Romidepsin was used at a third of the normal cancer dosing, he said. Side effects were consistent with those known to occur with romidepsin and others in a class of compounds that interfere with the function of the enzyme histone deacetylase. No severe toxicity was observed and no dose reductions were necessary, Sogaard said in an e-mail.

Bionor announced successful completion of the pilot study in May, and said it would start enrolling patients during the second quarter for the second part of the study, in which HIV-infected people will receive Vacc-4x and three weeks of romidepsin, before stopping their anti-HIV treatment to see if the virus rebounds. Initial results from the second part of the trial may be ready in the first half, Bionor said today.
Bionor shares jumped 5.7 percent to 2.97 Norwegian kroner by the close in Oslo, giving the company a market value of 671 million kroner ($108 million). The stock has gained 11 percent this year.

A study in 2012 of another old cancer drug, Merck & Co.’s (MRK) Zolinza, showed that that drug too roused HIV within the reservoirs. Still, the response to romidepsin points to a more potent method of activating latent virus in a way that may allow the immune system to hunt down and kill infected cells.

The patients received three infusions of romidepsin, sold by Celgene Corp. as Istodax, over 14 days. The drug increased virus production in HIV-infected cells by 2.1 to 3.9 times more than normal, and increased the amount of virus to measurable levels in the blood of five patients, a sign that the “kick” part of the strategy is working.

Because the amount of HIV that sleeps in viral reservoirs isn’t known, the researchers have no way of knowing how much of the latent virus was flushed out by the drug, Sogaard said. Celgene, based in Summit, New Jersey, is providing a free supply of romidepsin to Bionor under an agreement between the two companies.

Worldwide, 35 million people are living with HIV, the Joint United Nations Program on HIV/AIDS estimated last week. Of those, 13.9 million are on antiretroviral therapy.

‘Serious Challenges’

Over the last few years, academic teams around the world and drugmakers including Merck, Gilead Sciences Inc. (GILD) and Johnson & Johnson (JNJ) have been looking for ways to wipe out hidden reservoirs of the virus.

“We have always thought that once the virus gets inside a cell and goes to sleep, it’s stuck there forever,” said Sharon Lewin, the AIDS conference’s co-chair, who heads a team of researchers pursuing a cure for HIV at Melbourne’s The Alfred Hospital. Bionor’s results are “the first step to get rid of these long-lived, sleeping forms of virus with a drug that’s more potent than other drugs we have used already to wake up the virus. That’s a big step.”

Still, “serious challenges” lie ahead in the pursuit of a cure, said Anthony Fauci, director of the U.S. National Institute of Allergy and Infectious Diseases.

“We do need a cure; it’s important,” Fauci said in an interview at the Melbourne AIDS meting. “But we might not get it. What we might get is a sustained virological remission of varying degrees of duration in different subsets of people.”

Cure research is still in the very early discovery phase, he said.
“There is a lot of enthusiasm,” Fauci said. “It’s certainly an aspirational goal that we should strive for. We have to be careful that we don’t make assumptions that we know the pathway to a cure and all we need to do is implement it.”

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Avance en rastreo y destrucción de células con virus del sida

Científicos lograron localizar el virus y ahora buscan una manera de matar las células refugio.

Un grupo de científicos logró desalojar el virus del sida de las células en las que se refugia cuando los pacientes son tratados con antirretrovirales, un nuevo avance en la lucha contra la enfermedad presentado este martes en la conferencia internacional de Melbourne (Australia).

Este experimento, llevado a cabo con seis voluntarios, tiene el objetivo de desalojar y eliminar (kick-and-kill) el virus, una de las estrategias probadas por los científicos para encontrar un medicamento. Tomar antirretrovirales reduce la cantidad de virus en la sangre a niveles indetectables y permite a los pacientes llevar una vida casi normal.

Sin embargo, estos medicamentos deben tomarse todos los días, son caros y tienen efectos secundarios. Si se dejan de tomar, el virus rebrota en apenas unas semanas y vuelve a infectar a las células inmunes, lo que hace al paciente vulnerable a muchos microbios, algunos mortales.

Por ello, los científicos intentan, desde hace tres años, desalojar al virus de su refugio y matar a las células en las que se esconde cuando el paciente está bajo antirretrovirales. En la Conferencia Mundial sobre el sida en Melbourne, un grupo de investigadores de la Universidad de Aarhus en Dinamarca presentó sus conclusiones.

Seis pacientes tratados con antirretrovirales tomaron también romidepsin, un anticancerígeno que hace que aumente entre 2,1 y 3,9 veces la cantidad del virus en la sangre. En cinco de los seis pacientes, el virus se volvió entonces localizable. Los investigadores tendrán ahora que determinar si todos los virus escondidos fueron "revelados" y encontrar una manera de matar las células refugio, donde el virus se multiplica apenas se deja de tomar los antirretrovirales.

"Hemos demostrado que con romidepsin podemos activar un virus que hiberna", dijo Ole Schmeltz Sogaard, jefe del equipo de investigadores. "Es un paso hacia la dirección correcta pero el camino es aún largo y los obstáculos son numerosos antes de que podamos hablar de una cura del sida", añadió.

**Destructir las células refugio**

Cuando va a la sangre, el virus "reactivado" deja una marca en el exterior de las células inmunes infectadas CD4 que puede ser observada con un microscopio. Los científicos esperan que esta
marca, similar a las huellas dactilares, pueda ser rastreada por las células T (linfocitos T), que combaten las infecciones.

El equipo de científicos desea combinar el romidepsin, que "despierta" al virus de la inmunodeficiencia humana (VIH), con una vacuna llamada vacc-4x, para incitar a las células T a identificar y luego destruir las células en las que se refugia el virus. Los seis voluntarios del experimento no sufrieron efectos secundarios importantes al tomar romidepsin y este medicamento anticancerígeno no interfirió con los efectos del tratamiento con antirretrovirales.

La Conferencia Internacional sobre el Sida, que reúne cada dos años a especialistas de todo el mundo, estuvo marcada unos días antes de su apertura por la decepción en el caso de la "niña de Mississippi".

Se trata de una niña estadounidense, conocida como "la niña de Mississippi", nacida con VIH de una madre infectada y que no había sido tratada. La pequeña había recibido al nacer fuertes dosis de medicamentos durante 18 meses, antes de que los médicos perdieran el rastro del virus.

Cinco meses más tarde, los médicos no lograron hallar rastros del virus, un descubrimiento asombroso, ya que el VIH invariablemente aumenta menos de un mes después de que se detenga el tratamiento.

Sin embargo, hace unos días se descubrió que tras vivir 27 meses sin VIH ni medicamentos, el virus había vuelto a aparecer.

APF

Two men 'cleared' of HIV after bone marrow transplants in an Australian first that has sparked hope for eradicating the deadly virus that causes AIDS

- Two HIV-positive men were treated at Sydney's St Vincent Hospital
- They had bone marrow transplants and their HIV is now undetectable
- Professor David Cooper said these are the first successful cases of HIV being cleared in Australia
- Both patients remain on antiretroviral therapy to prevent HIV coming back

By AUSTRALIAN ASSOCIATED PRESS and SARAH DEAN

Professor David Cooper carried out the ground-breaking research at Sydney's St Vincent's Hospital

Two men who were HIV-positive appear to be virus-free after bone marrow transplants, marking the first successful cases of HIV being cleared in Australia.

Both men now register undetectable levels of the virus after treatment in Sydney, according to the University of NSW's Kirby Institute director, Professor David Cooper.

In a significant breakthrough for researchers, one of the patients cleared the virus without donor marrow containing a rare gene mutation that protects against HIV.

The human immunodeficiency virus, which causes AIDS, became undetectable in both patients about three years after their transplants, Prof Cooper said.

The men, who were treated at Sydney's St Vincent's Hospital, in partnership with the Kirby Institute, remain on antiretroviral therapy.

'We're so pleased that both patients are doing reasonably well years after the treatment for their cancers and remain free of both the original cancer and the HIV virus,' he said.

The work was presented on Saturday at the Towards an HIV Cure Symposium, which is part of the 20th International AIDS Conference in Melbourne, which opens on Sunday.

The patients' success echoes that of American man Timothy Ray Brown, the famous Berlin patient, who has shown no signs of virus resurgence since he received a bone marrow transplant from a donor with a rare gene mutation conferring resistance to HIV.
This rare gene mutation, called CCR5 delta32, makes stem cells naturally resistant to the virus. It is found in less than one per cent of Caucasians, mostly northern Europeans.

In Boston, two other patients underwent similar bone marrow transplants in 2012 but the transplanted cells did not contain the rare gene mutation.

In both cases, the virus returned after antiretroviral treatment was stopped.

The first Sydney patient underwent a bone marrow transplant in 2010 for non-Hodgkin lymphoma. His donor had the mutation.

However, the second man who underwent a procedure in 2011 for acute myeloid leukaemia was matched with a donor that did not have it.

Both men no longer showed any trace of the virus after a series of tests, Prof Cooper said. 'This is another example of where the transplant can drive the amount of virus to levels that we simply cannot detect,' he said.

Timothy Ray Brown, known as the 'Berlin Patient', is currently the only person to have been cured of AIDS.
'But if we stopped the antiretroviral therapy, there would be a very strong chance that it would come back.
'We're trying to understand this strong anti-HIV effect and understand where the virus might be hiding.'

The Sydney cases could lead to new approaches to treating, and ultimately eradicating HIV, he said.

'Cure research is looking for a way to move forward and my view is that this is a very important clue, that an immune response produced by bone marrow transplantation has such a strong anti-HIV effect,' Prof Cooper said.

'We're going to use this as a model for cure research and see if we can develop some therapies that mimic what were doing with bone marrow transplantation.'

The stem cell transplant procedure, however, is not a practical strategy for the majority of HIV patients, and the risk of mortality is up to 10 per cent, Prof Cooper says.

'For someone with HIV, you certainly would not transplant them when they've got an almost normal lifespan with antiretroviral therapy.'

The men, who were treated at Sydney's St Vincent's Hospital, in partnership with the Kirby Institute, remain on antiretroviral therapy.

Between two and five HIV positive patients required bone marrow transplants for cancer each year in Australia, he said.

'It is very difficult to find a match for bone marrow donors.'

And when a donor and recipient match was found, the chances of then having the one per cent of donors who had the protective gene was going to be very small, he said.

Prof Cooper said there was a movement in the HIV cure community to try to identify these donors with the mutation and ask them to volunteer for bone marrow transplants for HIV-positive people.

Years for AIDS cure, conference told

By Australian Associated Press

Published: 10:32 GMT, 21 July 2014 | Updated: 10:32 GMT, 21 July 2014

A cure for AIDS is still many more years away, with researchers unwilling to set any sort of timetable.

"I don't know how long it will take to get a cure," said French virologist Francois Barre-Sinoussi, who is credited with discovering HIV, told Melbourne's AIDS 2014 conference.

"We cannot really answer that, I personally think we should not give any dates."

Professor Barre-Sinoussi, co-chairwoman of the 20th International AIDS Conference, said on Monday that it had been 30 years since researchers promised an AIDS vaccine was only two years away. The vaccine is yet to be delivered.

Steven Deeks, a researcher from the University of California, San Francisco, said many thought a cure would never happen, but there were areas of "progress".

"Many very smart people don't think it is possible or will ever be possible," he told reporters.

"My personal opinion is that if it is possible, and we can do it, it's going to take many, many years."

Thirty-five million people are living with HIV today, the United Nations says. In Australia it’s estimated that about 26,000 people are HIV positive.

Conference co-chairwoman and infectious diseases physician, Sharon Lewin, said the focus for researchers was on strategies to achieve long-term remission in HIV-positive people.

"We probably are looking at trying to achieve long-term remission when we talk about cure, meaning time off antiretroviral therapy, how long can we go," Professor Lewin said.

"We've realised in the last year that the virus can really hang around for quite some time and pop up at an unexpected moment in time.

"That's exactly what we found with the Mississippi baby."
A four-year-old in Mississippi, initially thought to be cured of AIDS, has again tested positive to the virus. Deborah Persaud of the Johns Hopkins Children’s Centre in Maryland, who led the team of researchers in the case of the Mississippi baby in March 2013, said it was hugely disappointing the virus had returned.

However, Dr Persaud said researchers were able to gain valuable new information about the nature of the virus in infants. "It does provide us with a strong rationale to move forward with clinical trials," she said at the conference.

Dr Deeks also noted the significance of the Mississippi baby in the search for a cure.

"So we really now know what we have to go after," he said.

http://www.dailymail.co.uk/wires/aap/article-2699922/Years-AIDS-cure-conference-told.html#ixzz3DH0kHWfv
Cancer therapy cures two Australian patients of HIV

Monday, July 21, 2014 - 20:18

Sydney: In a piece of good news after the sad announcement a few days ago about HIV virus rebounding in the "Mississippi Baby", scientists have uncovered two new cases of HIV patients in whom the virus has become undetectable.

The two Australian men became apparently HIV-free after receiving stem cells to treat cancer, the scientific journal Nature reported.

"They are still on anti-retroviral therapy (ART) 'as a precaution' but those drugs alone could not be responsible for bringing the virus to such low levels," said David Cooper, director of the Kirby Institute at University of New South Wales in Sydney.

Cooper and his team scanned the archives of St Vincent’s hospital in Sydney - one of the largest bone-marrow centres in Australia - and found these two patients.

The first patient had received a bone-marrow transplant for non-Hodgkin’s lymphoma in 2011. The other had been treated for leukemia in 2012.

Because of the risk of relapse, Cooper’s team will not claim that their patients are completely cured. According to Cooper, the results show that "there is something about bone-marrow transplantation in people with HIV that has an anti-HIV reservoir effect, such that the reservoirs go down to very low levels".

If we can understand what is that and how that happens, it will really accelerate the field of cure search, Cooper added.

At the moment, there is only one person in the world who is still considered cured of HIV - Timothy Ray Brown.

This "Berlin patient" received a bone-marrow transplant and has had no signs of the virus in his blood for six years without ART.

Stem-cell transplant in itself cannot be used as a routine HIV treatment because of the high mortality (10 percent) associated with the procedure, the Nature report added.

IANS
Sydney researchers share HIV breakthrough

Anna Vidot reported this story on Saturday, July 19, 2014 08:18:00

**ELIZABETH JACKSON:** AIDS researchers are mourning the loss of up to 100 colleagues who were travelling to Australia on the Malaysia Airlines flight to attend the conference in Melbourne this weekend.

But they remain adamant that the conference will go ahead as planned.

Many have important research results to deliver.

Perhaps the most significant, the story of two Sydney men, who were HIV positive, but now appear to be clear of the virus three years after they had bone marrow transplants to treat cancer.

Anna Vidot reports.

**ANNA VIDOT:** In 2010, a Sydney man with HIV had a bone marrow transplant at St Vincent’s Hospital to treat a rare form of lymphoma.

By sheer luck, his bone marrow donor was very rare and they were missing a particular copy of the CCR5 gene, a genetic mutation that’s known to offer some protection against HIV.

The following year, another Sydney man, also HIV-positive, had a bone marrow transplant to treat his acute leukaemia. There was nothing particularly genetically special about his donor, but the results for both men certainly were special.

**SAM MILLIKEN:** Their HIV virus has been completely suppressed, not only on the standard tests but on a number of more sophisticated molecular tests. And we’ve been unable to detect any active virus.
ANNA VIDOT: Dr Sam Milliken is the director of Haematology & Bone Marrow Transplantation at Sydney’s St Vincent Hospital.

SAM MILLIKEN: That doesn’t mean the virus has been completely eradicated, but it does indicate it’s been suppressed to levels far below what we’d normally see with standard therapies for HIV.

ANNA VIDOT: It’s not the first time this has happened.

In 2007 and 8, a man in Berlin received two transplants, the second from a donor who was missing not one but both of the crucial copies of the CCR5 gene - an extremely rare genetic mutation that makes someone resistant to HIV infection.

The Berlin patient was cured, and he’s still clear of the virus today, even though he’s no longer on antiretroviral therapy.

Last year, two bone marrow transplant patients with HIV were reported in Boston, with very similar findings to the Sydney patients. But unlike the Berlin patient, the Boston patients’ HIV returned when they stopped taking their medication.

Professor David Cooper is the director of the Kirby Institute at the University of New South Wales.

DAVID COOPER: The two Boston patients and our two patients did not have the full dose of the gene that leads to completed resistance. And I think it will become a very fruitful area for research of what bone marrow transplantation does to make the HIV go away in such a strong way.

ANNA VIDOT: Both Professor Cooper and Dr Milliken stress that the Sydney results don’t mean bone marrow transplants are a viable cure for the more than 38 million people infected with HIV worldwide.

Bone marrow transplants are costly, difficult, and Dr Milliken says, dangerous.

SAM MILLIKEN: There’s a definite mortality risk in having a bone marrow transplant, so you can only really justify it to people who’ve got a cancer, you know that if you don’t get rid of it, it’s going to take their life.

ANNA VIDOT: Dr Milliken says work will continue at St Vincent’s to build on these findings.

SAM MILLIKEN: Discoveries like this come about almost by chance, but then lead you on to coming up with new ways of dealing with the disease and hopefully eventually a cure.

ELIZABETH JACKSON: Dr Sam Milliken, the director of Haematology & Bone Marrow Transplantation at St Vincent’s Hospital in Sydney, ending that report from Anna Vidot.

http://www.abc.net.au/am/content/2014/s4049473.htm
Today’s program comes ‘live’ from the 20th International AIDS Conference, which is being held in Melbourne. One of the issues being discussed is whether condoms are outdated. There’s evidence that taking anti HIV drugs might be at least as effective in preventing infection or transmission. And where are we with a vaccine well over 30 years into the pandemic.

Melbourne prepares to host world's biggest AIDS Conference

Updated 18 July 2014, 13:12 AEST

The 20th International AIDS Conference which opens in Melbourne this weekend has been described as "a meeting of everyone affected by HIV" and more.

It brings together 14,000 delegates from nearly 200 countries, including a media contingent of 1,200 journalists, not to mention extensive coverage on Radio Australia all week.

AIDS 2014 co-chair Professor Sharon Lewin points to the 'three critical pillars' of science, leadership and community.

Professor Lewin says the current HIV situation is one of contrasts.


Presenter: Sen Lam
Speaker: Professor Dr Sharon Lewin, Head of the Department of Infectious Diseases, Alfred Hospital and Monash University. Professor Lewin is also co-head, Centre for Biomedical Research, Burnet Institute in Melbourne
LEWIN: It’s a story of tremendous gains, but still much work to be done, and I think most encouraging the numbers of new infections of substantially decreased over the last decade, numbers of AIDS-related deaths have also decreased, advances in treatment and delivery of treatment is being spectacularly successfully, but still only 40 percent of people who need treatment are on treatment globally. So there’s many challenges that lie ahead, which means getting treatment to people that need it, reducing stigma and discrimination that allows people most affected by the epidemic to access treatment and prevention messages, and still much to be done in the science to find a vaccine and a cure.

LAM: And how important are massive meetings, like AIDS 2014, how important are they in facilitating a response to HIV AIDS?

LEWIN: Well, AIDS 2014 is quite an unique meeting, it's completely multi-disciplinary. It has three major programs within it, Science, Leadership and Community and those three pillars are really essential in getting advances in the HIV response. They've been critical in the past and I think they'll be critical going forward. So this is not just a meeting of usually boring scientists and clinicians, this is a meeting of everyone affected by HIV and everyone who can make a difference. And most importantly, that's the community and our political leaders.

LAM: And, how valuable is it, to be able to meet face-to-face in person?

LEWIN: I think the history speaks for its value, this meeting has often been associated with quantum shifts in the approach to the epidemic, of course, that doesn't happen at every meeting, but there are certainly some memorable ones. The most memorable for me in recent years is the meeting in 2000, in Durban, where the world became aware of the size of the problem in Africa and the fact that drugs were not being delivered to people who needed them and that led to a revolution in how we deliver drugs to low income countries and last year, there were 13 million people on anti-retroviral therapy in low and middle income countries, in 2000, there was close to no one on treatment.

LAM: Personally, on a personal level, what does this meeting mean to you AIDS 2014, what do you hope to get out of it?

LEWIN: Well, I think it's a wonderful opportunity for Australia, first of all, to showcase what we've done so well here, which includes a response that has always been characterised by true partnerships between governments, science and the community; some very progressive public health policies that we've had around education, prevention and access to clean needles; and for some spectacular science that many of our scientists and clinicians have delivered over many years. But at that same time, there's much for us to learn and I think in going forward, we've to learn to reduce new infections, we've got much to contribute, particularly to our region. I think the conference is also a fantastic opportunity for the greater community across Melbourne and across Australia, to really understand that HIV is a big problem and we still need to do quite a bit to address it and we have an unique opportunity now to really make a difference.
LAM: And tell us about the global village at AIDS 2014. If UN AIDS in Bangkok last year is anything to go by, it's quite festive, isn't it, bringing together different layers of communities, perhaps?

LEWIN: Yes, the Global Village is actually a very unique part of the conference, first of all, it's free and open to the public, it's a space where communities can showcase what they've done and what's important in their response, be it transgender people, being it men who have sex with men, people with TB, women affected by HIV, but it's done in a very colourful, Rio carnival-like atmosphere. There are photographic exhibitions, there's music, there's dance, there's public lectures, and there's people from every part of the planet and everyone there has a pretty unique story to tell. So I really encourage members of the public to go and have a look. It's in the Exhibition Centre on Spencer Street, in Melbourne, and free and open to the public.

LAM: Are we expecting any major announcements at next week's meetings?

LEWIN: Well, we're expecting a lot of excellent science, excellent science related to new treatments for hepatitis-C and tuberculosis, which are both very common infections that occur in people living with HIV, excellent new findings in relation to the impact of prevention campaigns or prevention intervention, such as pre-exposure prophylaxis and circumcision and a lot of discussion and debate about vaccine and cure. We've got a lot of leaders from many parts of the world, including Australia, we have some very high level presentations from President Bill Clinton and Bob Geldof, ministers of health from multiple countries and maybe many of them will have something exciting to say.

http://www.radioaustralia.net.au/international/radio/program/asia-pacific/melbourne-prepares-to-host-worlds-biggest-aids-conference/1344789
Six things we learnt from Melbourne's AIDS conference
Updated 25 July 2014, 19:00 AEST

After a week marked by tragic and sudden loss of six delegates on the doomed Malaysia Airlines flight MH17, the 20th International AIDS Conference in Melbourne has wrapped up.

The conference was organised by the International AIDS Society (AIDS), the world's leading independent association of HIV professionals, with more than 16,000 members in more than 177 countries working at all levels on the global response to AIDS.

The tragic loss of [IAS president Dr Joep Lange and five other delegates on MH17](#) overshadowed the opening, but delegates rallied, galvanised by their loss.

Here are six important things the conference taught us:

1. **HIV rates are at a 20-year high**

Before the conference got underway, new data from the Kirby Institute demonstrated the need for more research on AIDS.

The figures showed that HIV rates had been steadily rising in Australia since 1999, and more than 26,000 people were now living with the virus.

External Link: [Watch the Grim Reaper ad from 1987](#)

The survey also found [HIV rates were at a 20-year-high](#), with unprotected sex between casual male partners seen as a leading cause. But there had been some progress, with only 2 per cent of all HIV cases now being attributed to unsafe drug injecting.
"Fortunately [there] was pioneering in the 1980s and [it] has really led the world in large-scale implementation of needle and syringe programs and that has led to effectively no epidemic taking off," Associate Professor David Wilson said.

2. Waking up hidden HIV

Scientists revealed a new approach to getting rid of the HIV virus, called the "kick and kill" approach, using an anti-cancer drug to kick the virus out of where it is hiding in the body.

Dr Ole Schmeltz Sogaard from Denmark's Aarhus University said he gave patients anti-cancer drugs which increased the production of HIV-infected cells more than three times above normal. The cells could then be traced and targeted with existing treatments.


"We've now shown we can activate a hibernating virus with Romidepsin and that the activating virus moves into the bloodstream in large amounts," Dr Sogaard said.

"This is a step in the right direction, but there is still a long way to go and many obstacles to overcome before we can start talking about a cure against HIV."

3. Promising bone marrow transplants

Two HIV-positive patients treated at Sydney's St Vincent's Hospital were given a bone marrow transplant, which appeared to have cleared them of the virus.

They now have undetectable levels of HIV but remain on antiretroviral therapy as a protective measure.

Although the results were significant, experts stressed that bone marrow transplants were not a cure for HIV, as it remained a costly and a potentially dangerous procedure.

4. TB breakthrough: radically reducing treatment times
The conference was told about a major breakthrough in treating tuberculosis. Tuberculosis killed one in five people with HIV and remained the largest killer in the world of people with AIDS.

Results of an international study showed a new combination of drugs meant that drug-resistant TB could be cured in as little as four months, instead of two years.

5. AIDS-free generation 'within reach': Bill Clinton

Former US president Bill Clinton declared that an AIDS-free generation was within reach, despite the fact that every year another 2 million people are infected with HIV.

But he said the international community had to get better at detecting the disease early.

"New data from 51 countries suggests 70 per cent of HIV-related deaths could have been prevented," he said.
"The evidence continues to build that early treatment helps prevent further transmission."

His speech was briefly interrupted by protesters calling for new taxes to support the fight against AIDS, which brings us to lesson number six.

**6. More funding needed for the home stretch**

Artist and poverty campaigner **Bob Geldof slammed the world's wealth countries over a "disgraceful" lack of HIV funding.**

He said while there had been incredible advances in the fight against HIV and AIDS in the past 30 years, the final steps were the most crucial. According to UNAIDS, 19 of the 20 most-AIDS-affected countries were in Africa and 72 per cent of all people with HIV lived in the sub-Saharan region. Sir Bob criticised the foreign aid spending of wealthy countries, especially Australia, which planned to cut its contribution by $7.6 billion over the next five years.
Waking up hidden HIV is one step closer to curing disease: study

By medical reporter Sophie Scott
Updated 23 Jul 2014, 1:04pm Wed 23 Jul 2014, 1:04pm

Results presented at the World AIDS conference in Melbourne show an anti-cancer drug can kick the virus out of where it is hiding in the body.

"It's called the kick and kill approach," said Dr Ole Schmeltz Sogaard from Aarhus University in Denmark.

He gave the anti-cancer drug Romidepsin to six patients - five men and one woman - who were HIV positive.

The patients had been on antiretroviral medications for an average of nine-and-a-half years, and were given three infusions of the Romidepsin over 14 days.

The study showed the drug increased the virus production in HIV-infected cells more than three times above normal so it could be traced.

The findings are convincing, says Professor Steven Deeks, from the AIDS Research Institute at the University of California in San Francisco.

"This is the first clear data to show we can shock the hidden virus," he said.

"It hasn't been shown like this in humans before."

The hope is that by activating the dormant cells, researchers can then eradicate them through existing treatments or vaccines which are being trialled.

That could mean the end of antiretroviral treatments, which keep patients well, but have side effects.

"We've now shown we can activate a hibernating virus with Romidepsin and that the activating virus moves into the bloodstream in large amounts," Dr Sogaard said.
"This is a step in the right direction, but there is still a long way to go and many obstacles to overcome before we can start talking about a cure against HIV."

Two cleared of HIV in Sydney after bone marrow transplants

Two men who were HIV-positive appear to be virus-free, registering undetectable levels after bone marrow transplants in Sydney.

These are the first successful cases of HIV being cleared in Australia, according to the University of NSW's Kirby Institute director, Professor David Cooper.

In a significant breakthrough for researchers, one of the patients cleared the virus without donor marrow containing a rare gene mutation that protects against HIV.

The human immunodeficiency virus, which causes AIDS, became undetectable in both patients about three years after their transplants, Prof Cooper said.

The men, who were treated at Sydney's St Vincent's Hospital, in partnership with the Kirby Institute, remain on antiretroviral therapy.

"We're so pleased that both patients are doing reasonably well years after the treatment for their cancers and remain free of both the original cancer and the HIV virus," he said.

The work was presented on Saturday at the Towards an HIV Cure Symposium, which is part of the 20th International AIDS Conference in Melbourne, which opens on Sunday.
The patients' success echoes that of American man Timothy Ray Brown, the famous Berlin patient, who has shown no signs of virus resurgence since he received a bone marrow transplant from a donor with a rare gene mutation conferring resistance to HIV.

This rare gene mutation, called CCR5 delta32, makes stem cells naturally resistant to the virus. It is found in less than one per cent of Caucasians, mostly northern Europeans.

In Boston, two other patients underwent similar bone marrow transplants in 2012 but the transplanted cells did not contain the rare gene mutation.

In both cases, the virus returned after antiretroviral treatment was stopped.

The first Sydney patient underwent a bone marrow transplant in 2010 for non-Hodgkin lymphoma. His donor had the mutation.

However, the second man who underwent a procedure in 2011 for acute myeloid leukaemia was matched with a donor that did not have it.

Both men no longer showed any trace of the virus after a series of tests, Prof Cooper said.

"This is another example of where the transplant can drive the amount of virus to levels that we simply cannot detect," he said.

"But if we stopped the antiretroviral therapy, there would be a very strong chance that it would come back.

"We're trying to understand this strong anti-HIV effect and understand where the virus might be hiding."

The Sydney cases could lead to new approaches to treating, and ultimately eradicating HIV, he said.

"Cure research is looking for a way to move forward and my view is that this is a very important clue, that an immune response produced by bone marrow transplantation has such a strong anti-HIV effect," Prof Cooper said.

"We're going to use this as a model for cure research and see if we can develop some therapies that mimic what we were doing with bone marrow transplantation."

The stem cell transplant procedure, however, is not a practical strategy for the majority of HIV patients, and the risk of mortality is up to 10 per cent, Prof Cooper says.

"For someone with HIV, you certainly would not transplant them when they've got an almost normal lifespan with antiretroviral therapy."
Between two and five HIV positive patients required bone marrow transplants for cancer each year in Australia, he said.

"It is very difficult to find a match for bone marrow donors."

And when a donor and recipient match was found, the chances of then having the one per cent of donors who had the protective gene was going to be very small, he said.

Prof Cooper said there was a movement in the HIV cure community to try to identify these donors with the mutation and ask them to volunteer for bone marrow transplants for HIV-positive people.

Anti-cancer drug wakes up HIV in hiding

DR ANDREW ROCHFORD
7News and Agencies July 22, 2014, 8:43 pm


Scientists are heralding one of the biggest steps towards and AIDS cure since the development of treatments to control it.

They have announced the breakthrough at a global conference in Melbourne.

Amid a conference marred by the deaths of six delegates aboard the doomed MH17 flight the news was well received.

In an experiment on six AIDS sufferers, Danish scientists have used a drug to flush out HIV cells hiding around the body, forcing them back into the bloodstream, where they can be targeted by the immune system.

HIV can hide in a state of hibernation in so called CD4 cells.

While HIV can be kept down by medicine there is still no cure that can eradicate the virus completely from the body.

The researchers gave the anti-cancer drug to six HIV patients and found that the drug can identify the hibernating virus and shock it out of its hiding place.
The researchers also found the drug increased the virus production in HIV-infected cells between two and almost four times above normal so it could be traced.

"We have now shown that we can activate a hibernating virus with Romidepsin and that the activated virus moves into the bloodstream in large amounts," said Dr Schmeltz Sogaard.

"This is a step in the right direction, but there is still a long way to go and many obstacles to overcome before we can start talking about a cure against HIV."

HIV researchers hope that by activating the dormant cells, they can then eradicate them through existing treatments or vaccines which are being trialled.

Sharon Lewin, an infectious disease physician and local co-chair of this year’s International AIDS Conference, said this Danish study has taken cure research one step further.

"It’s more potent than other drugs we've used already to wake up the virus and that’s a big step.”

However, even though there is no cure yet, the breakthrough is enough to give a boost to HIV patients,

Brent Allan said: “I’ve been living with HIV for a long time, my partner has been living with HIV for a long time and most of my friends have lived with HIV for a long time.

“I’ve always pinned my hopes on a cure and I’m not going to let that go.

While this is certainly good news for Australia’s 28,000 AIDS sufferers, what is not so heartening is data showing their ranks are rising faster now than at any point in the last 20 years.

The epidemic hit hard when it arrived in Australia in the early 1980s as thousands of cases were diagnosed each year.
Then, as the message got out, the tide quickly stemmed.

However, in recent years that trend has reversed with infections again on the increase.

The famous ‘Grim Reaper’ campaign, however, won’t be making a comeback, with Brett agreeing a modern approach is the way to go.

He said: “I don’t think scaring people is what works anymore. I think what we need to aim for is what I’d call informed sophistication, we want to build people to understand the complexities and give them the skills to deploy that in their lives.”

C. Online media and blogs

The Sydney Morning Herald

HIV research: Cancer drug breakthrough in "kicking" disease out of hiding place

Julia Medew
Health Editor
July 22, 2014


Danish researchers have found a way to activate and expose hidden HIV cells to the immune system - a major step towards finding a cure for the virus affecting 38 million people.

Ole Søgaard, a doctor from Aarhus University Hospital in Denmark, said a study of six HIV-positive patients showed that a cancer drug could "kick" HIV out of its hiding place and into the bloodstream, where it can theoretically be attacked by the immune system.
The finding has been hailed the single most important advance reported at the 20th International AIDS conference in Melbourne, where thousands of the world's leading HIV experts have gathered to share their research.

Dr Søgaard said his team gave the patients three doses of the cancer drug romidepsin over three weeks to see if it would move the virus out of reservoirs where it hides and cannot be detected. All patients were long-term HIV patients who remained on their antiretroviral treatment for the trial.

He said before the drug was given to the patients, they had undetectable levels of HIV in their system. But within days of receiving the cancer drug, significant amounts of the virus had moved into their blood.

"We could see that within a very short time, one to three days, we could measure a large increase in the number of viral particles," he said.

"So these cells are going from being in a resting state to exposing themselves to the immune system."

Dr Søgaard said while the patients' immune systems did not destroy the virus, a new trial involving 20 patients would combine this "kick" approach with an experimental HIV vaccine (Vacc-4x) to see if it can stimulate a strong enough immune response to kill the virus.

But he said this may not work if the virus still persists in some hidden cells.

In his study, it was unclear whether all of the hiding cells had been deployed by the romidepsin or just some of them.
"We don't know if we did it for 1 per cent of the cells or 5 per cent or 50 per cent," he said. "All we can say is we did it at least for some of the cells ... so it is still early days."

While at least one man known as the "Berlin patient" is believed to have been cured of HIV through a bone marrow transplant from a donor with a genetic mutation that protects a small number of people from HIV, doctors say this is not a practical solution for others mainly because transplants are dangerous and expensive and donors with genetic protection against HIV are rare.

Instead, most HIV researchers believe a "kick and kill" approach that "wakes up" the virus to expose it to the immune system is a promising direction for cure research.

Dr Søgaard said while antiretroviral treatments stopped HIV from spreading throughout the body, the virus also hid in "reservoir" cells that were resistant to the treatment. This was the main barrier to curing people.

"Despite taking antiretroviral treatment for years, we know that once patients stop taking treatment, in a matter of about two weeks, there will be millions and millions of viral particles throughout the body. And the reason for this is the resting cells," he said.

"In theory, if you can activate all cells and expose them to the immune system and get them killed, then you have cured a person."

Dr Søgaard said although some of the patients experienced fatigue and nausea - common side effects of the cancer drug - there was no immediate concern about the impact on their health.

Steven Deeks, a professor of medicine at the University of California who specialises in HIV research, said the Danish research was likely to have a huge impact on HIV cure research worldwide.

"Ole's data is the first clear evidence, at least to me, that we can truly identify the latent reservoir, the hidden virus, and shock it out of its hiding place," he said.

"I don't think anyone has shown that in people before to the same degree that Ole has in his study. I think actually it is the single most important advance of this meeting and it's going to have a huge impact in future."

Meanwhile, Corinne Treger, the chief executive of French pharmaceutical company Biosantech, said a phase two trial of a therapeutic vaccine to treat HIV, not prevent it, was showing some positive results.

If the experimental vaccine, known as "Tat Oyi", is proved to be safe and effective, she said people with HIV could receive several injections to control their virus permanently instead of taking antiretroviral treatment, which can have side effects and cost thousands of dollars a year.
Ms Treger said her company was collaborating with Canadian researchers testing "Immunorex", an experimental drug hoped to treat and prevent cardiovascular diseases, dementia and cancers induced and enhanced by HIV, and prevent HIV mutations that lead to drug resistances and vaccine failure.

It is hoped this drug would be jointly administered with the Tat Oyi vaccine to improve the health of people living with HIV.

Ms Treger said of about 12 studies testing a therapeutic vaccine, her company's was the most advanced. If a new trial in 80 patients in 2015 goes well, she said the vaccine could be available in 2017.

Françoise Barré-Sinoussi on the history and future of HIV research

The professor who won the Nobel prize for her part in the discovery of HIV says it is now her duty to get researchers worldwide working together to treat and prevent the disease.

Melissa Davey
theguardian.com, Friday 25 July 2014 08.24 BST

In 1981, the US Centres for Disease Control and Prevention published a report describing a seemingly rare illness that had infected five men in Los Angeles, killing two of them. It was the first official report describing a horrific illness that caused severe fever, fatigue and weight loss, seemingly only among gay men, ultimately killing many of them.

As soon as the report was released, stories flooded in from other doctors who had seen similar symptoms in their patients. The disease did not yet have an official name – that came in July 1982, when the international community adopted the name Acquired Immunodeficiency Syndrome, or Aids.

As reports of the illness continued to spread throughout the US and worldwide, Professor Françoise Barré-Sinoussi was working as a retrovirologist at the Pasteur Institute in Paris - a not-for-profit organisation with a focus on infectious diseases. And it was there she was approached by scientists in the early 80s, urging her to help them find what was causing the Aids epidemic.

That she was in such a position – working at Pasteur and ready to take on a devastating disease – was a result of her own persistence. A thirst to understand biomedical sciences at a level beyond what she felt she could by studying medicine drove her to seek out laboratory internships while she was just a student.
Laboratories such as Pasteur were the domain of fully-trained researchers, but Barré-Sinoussi convinced senior laboratory heads to let her intern there in the late 60s, in her spare time while she was still at medical school.

“Very rapidly while at university, as a young student at the time, I realised I was motivated by science, by biomedical science, without really knowing what that meant,” she told Guardian Australia.

“So after two years at university I said to myself, ‘I can’t study at university without knowing what it means to be a researcher, to understand science,’ and so I began looking for a lab that would accept me at the same time while I studied.

“It was very difficult. Research labs were not used to this kind of approach by a student.”

They were male-dominated, and not used to having young, working-class women like Barré-Sinoussi in their midst.

“I was odd,” she said.

She approached several labs, which refused her. But a Pasteur virologist, Professor Jean-Claude Chermann, accepted Barré-Sinoussi, and she went on to gain her PhD there in 1975.

Today, she is the president of the International Aids Society (IAS), the organisation that runs the Aids 2014 conference, currently taking place in Melbourne. When that conference closes on Friday evening, Barré-Sinoussi will step down as president.

Next year, Barré-Sinoussi says she will also retire and close the laboratory she still has at Pasteur, where she and her mentor Luc Montagnier discovered HIV in 1983.

That highly significant discovery revealed an urgent need for diagnostic tests to aid in controlling the spread of the disease and allowed the development of treatments that now keep people with HIV alive.
The pair were awarded the Nobel Prize for Medicine in 2008 for the breakthrough.

By the time the Aids epidemic was identified in 1981, Barré-Sinoussi had built her career on the study of retroviruses. Retroviruses have an enzyme, called reverse transcriptase (RT), that gives them a unique ability to become part of a host cell’s DNA, and then replicate. It was also in 1981 that the first retrovirus was recognised, called HTLV-1, responsible for infecting T-cells and causing types of the cancer, leukaemia.

T-cells, a type of white blood cell, are essential to protecting the immune system.

“A lot of studies at the time were trying to make a link between different families of viruses and the Aids epidemic, but none of the viruses at the time was corresponding to the right responsible agent for Aids,” Barré-Sinoussi said.

“So in 1982 a group of French clinicians [led by Willy Rozenbaum, an infectious-disease specialist] came to us at Pasteur, as we were retrovirologists, and said to us, ‘Do you think a retrovirus, maybe HTLV, could also be the cause of Aids?’

“As clinicians, we knew HTLV was a human retrovirus capable of infecting T-cells, so in these researchers minds they thought in patients with Aids – because their T-cells were rapidly disappearing – that maybe Aids was caused by HTLV-1.

“So immediately we told the researchers we didn’t believe in it, because HTLV was causing leukemia and the cells infected were proliferating and causing cancer.

“But we told them, ‘We can check.’”

Montagnier organised a research group and found a patient with the illness willing to take part in their study, a young gay man in the US.

A biopsy of the patient’s lymph nodes arrived with Pasteur by early January 1983, and Barré-Sinoussi and Chermann measured the RT enzyme activity in the sample every few days.

By this time, two retroviruses had been discovered in humans, but Barré-Sinoussi detected neither of them in her sample. She and her team found evidence of a new retrovirus altogether, which was later named HIV. With a virus now identified, work could begin to combat it.
Barré-Sinoussi insists that the closure of her lab at the end of 2015, following her official retirement, will not make her feel sad.

“I learned a few weeks ago that senior scientist in my lab, Dr Michaela Muller-Trutwin, has applied for a new lab at Pasteur to continue the work we have been doing together, and she has been accepted.

“I’m totally relieved as I know she will take over the lab so everything we have been working towards will continue.

“She did her PhD in my lab, she trained with me, she worked with me for 20 years. The work is in good hands. I consider her my daughter somehow. So that’s very nice. I feel it’s an achievement - knowing that all of my staff will work in other labs, I know where they’re going.

“And that is why I feel relieved.”

Perhaps because of her own experiences of fighting to intern as a young woman, Barré-Sinoussi greatly believes in the empowerment of her staff. She is fast to admit what she does not know, and says there are many senior researchers better placed to investigate HIV than she, the scientist who discovered it.

“Do you think today I am ever a principal investigator on a project or research?” she said.

“No way! I have younger, senior, principal investigators in my lab. They are capable.”

She believes in collaboration between researchers and pushes strongly for it in developing countries, where HIV still has a stronghold. And while she may be retiring from the lab and stepping down as president of the IAS, she will remain president-elect for the next two years.
It is now her duty, she says, to get researchers from around the world working together to find better ways to treat and prevent HIV. With so much progress on that front already made, an Aids-free generation is within grasp. But not without working together, she says.

“I will not name them, but in several countries the boss is the boss. If you say you want to make a project application, they need to nominate a principal investigator from their country, they immediately give you the name of the director of the national Aids program.

“And I say to them, ‘Look – I have nothing against the director of the national Aids program, but he or she is not the one to make this work.’

“We need experts – young experts. Senior people have too much other responsibility. So it’s difficult to convince them because it’s not their education, it’s not their culture, they think only one person can have this kind of responsibility – the highest level – but that’s a mistake. I think it is our responsibility and duty as senior people to prepare our staff and team to take over our positions.

“It’s part of our job.

“In resource limited settings it’s complicated because they’re used to very limited funding and have a tendency to keep any funding to themselves, they think collaborating means less money for them.

“So you have to demonstrate and show them it’s not the case, and I like that work, it’s interesting work, and it is what I will focus on now.”

Barré-Sinoussi remains humble despite her contribution to medical science. She speaks softly, but with strong conviction. And this conviction becomes especially apparent when the topic turns to access to healthcare.

She is frustrated that despite antiretroviral drugs being available which significantly improve the health of people living with HIV and greatly reduce the risk of passing the virus on, not enough people in developing countries have access to those medicines.

The big message to come out of her last conference as president is that key populations disproportionately affected by HIV - gay people, drug users, sex workers, and the poor - are still being stigmatised, she said.

She becomes fierce.

“The data presented at this conference regarding the repressive legislation in different countries, the stigmatising and discriminating of the gay population, of drug users - we know the results of such policy, of such legislation.

“We know that it will lead to an increase in HIV infections, an increase in deaths, an increase in co-infections.”
“If you are in a country where there is punitive criminalisation of populations who do not have access to health services ... it is devastating.” But she was pleased to see the strong rejection by the 12,000 people at the conference of such damaging and discriminatory policies. “I can really feel the community here mobilise in the fight for HIV, but also for global health in general.

“And we know there is no reason to give up on a cure, or at least no reason to give up on treatment that can induce permanent remission.

“We have all of this science, all of this research into HIV being presented and the key information we are getting from these models is that ‘It is possible to stop AIDS. So go ahead’.”

HIV discovery reveals virus hidden in immune system cells
Cancer-fighting drug romidepsin has been shown to expose hibernating HIV, making it susceptible to attack

Danish researchers have used an anti-cancer medicine to activate HIV hidden in the cells of patients taking anti-HIV drugs, exposing the virus to the immune system and making it susceptible to attack.

The results revealed on Tuesday constitute one of the major scientific discoveries hailed at the Aids 2014 conference in Melbourne, as much of the language shifts away from finding a cure to focusing on big steps in HIV treatment and prevention.

HIV hides in a state of hibernation in CD4 cells, an essential part of the immune system. Yet CD4 cells are unable to fight HIV themselves – that role lies with the immune system’s killer T-cells.

But because killer T-cells can’t detect the HIV hidden within CD4 cells, they are unable to attack and eliminate it from the body. While HIV patients on antiretroviral drug treatment often go on to have undetectable levels of HIV in their system, it is never eliminated.

There is always a reservoir left hiding in cells, undetectable to current screening tools and ready to take hold of the immune system again should patients stop their antiretroviral therapy.

But a research team led by Ole Søgaard at Aarhus University’s department of infectious diseases in Denmark has used the anti-cancer drug romidepsin to activate the virus and bring it out of hiding.

In principle, this means that the killer T-cells should be able to detect the virus, because it leaves a trace on the outside of CD4 cells as it is activated and moves towards the bloodstream, Søgaard said.

“Despite very effective antiretroviral treatment, there is still this reservoir left of HIV cells that are infected but not producing the virus,” he said.

“Once you activate them, these particles will go to the surface and signal to the immune system that this cell is infected and needs to be cleared from the body. So this is a two-step system where we bring the cells to the surface, and then rely on the immune system to kill them.”

In the pilot study, researchers gave six patients three doses of romidepsin over three weeks.
Before each dose, no viral particles were detectable in the patients.

“But after the dose was given we easily measured the virus being released into the plasma in five of these six patients,” Søgaard said.

“We also saw the virus go back to undetectable levels after seven days, so it came up, then hid away again, returning back to a non-active state until the next dose of cancer drug was given.”

However, the researchers found the immune system did not seem to attack the virus after detecting it. Researchers found no significant reduction in the number of infected cells each time the cancer drug brought the virus out of hiding.

“This suggests when you do this reactivation, you also need to also target and activate the immune system and teach it to recognise these cells and attack,” Søgaard said.

“That’s what we’re doing next in this study. We will teach and prime the immune system to recognise HIV before we give patients the cancer drug, and we hope there will be a better chance the immune system can clear those cells when the HIV is reactivated.”

Søgaard emphasised it was unknown how much of the HIV reservoir the immune system would be able to clear even if it could be taught to recognise the virus and attack it. Virus left in just one cell might be enough to allow HIV to thrive again.

“We’re still learning about this disease and where it hides, and it is a really, really tricky disease to cure because it hides in so many places in the body, it hides really well and can hide for an indefinite period of time,” he said.

The difficulty of an HIV cure became particularly apparent with the now famous case of the Mississippi baby, born to an HIV-positive mother.

The child was placed on a strong course of antiretroviral drugs within 30 hours of birth. She continued the treatment for 18 months and when she stopped taking those drugs, she had no detectable virus in her system. It gave hope other infants treated early might be cured.

But 27 months later, the virus was detected and she was placed on antiretroviral drugs again.

In a similar case, two Boston patients received bone marrow transplants that appeared to rid them completely of HIV. They also relapsed, and are now back on antiretroviral treatment.

These cases have been referred to frequently during the AIDS 2014 conference, sometimes brandished as setbacks. But the cases reveal how far HIV treatment has come.

Achieving more than two years without antiretroviral treatment in an infant is unprecedented, especially since the Mississippi baby had no existing immunity to HIV. It has given researchers a new
focus on where to fight HIV, as they now know it takes just a tiny amount of dormant virus for HIV to become active again.

The cases have shown that working out exactly where dormant HIV virus resides in the body, and being able to measure it, will be key to future research.

The development of HIV into lethal Aids was once considered inevitable, and less than 30 years after the epidemic began – not a long period in medical science – people living with HIV are able to lead long and healthy lives.

But that all depends on access to treatment, which has the added benefit of protecting against the transmission of HIV during unprotected sex by up to 96%.

The president of the International Aids Society, Françoise Barré-Sinoussi, won a Nobel prize for her role in discovering HIV and said she would not be drawn into talk about how far away a cure for HIV/Aids may be.

“I remember in 1984 someone said we would have a vaccine within two years, and we are now 30 years later,” she said.

“We should move on from this. We will need to collaborate and combine different approaches to HIV – work on cures as well as therapies, prevention and vaccines – and strengthen the relationship between researchers to continue to make progress in tackling HIV.”

Early HIV drugs 'may not stop virus'

By James Gallagher Health editor, BBC News website

HIV can rapidly form invulnerable strongholds in the body, dashing hopes that early treatment might cure the virus, according to new research.

A baby was thought to have been cured with treatment hours after birth, but the virus emerged years later.

Monkey research, published in the journal Nature, suggests untouchable "viral reservoirs" form even before HIV can be detected in the blood.

Experts described it as a "sobering" and "striking" finding. Reservoirs of HIV in the gut and brain tissue are the massive obstacle in the way of a cure.

Remarkable progress in developing antiretroviral drugs means HIV can be kept in check in the bloodstream and patients have a near-normal life expectancy.

But if the drugs stop, the virus will emerge from its reservoirs. International research is focused on flushing the virus out of its reservoirs, but there had been hope that early treatment could prevent them forming in the first place.

'Established'

In the study, rhesus monkeys were infected with the monkey equivalent of HIV - simian immunodeficiency virus (SIV).
The monkeys were then given antiretroviral drugs as early as three days or as late as two weeks after infection.

Treatment stopped after six months, but the virus re-emerged irrespective of how quickly antiretroviral treatment started.

It showed that viral reservoirs formed incredibly early in the course of the infection.

Dan Barouch, professor of medicine at Harvard Medical School, said: "Our data show that in this animal model, the viral reservoir was seeded substantially earlier after infection than was previously recognised.

"We found that the reservoir was established in tissues during the first few days of infection, before the virus was even detected in the blood."

WATCH VIDEO: Three quarters of new HIV cases are in just 15 countries

'Rebounding'
It had been believed a baby girl born with HIV had been cured after very early treatment.

The "Mississippi baby" was given HIV drugs for the first 18 months of life, but then they were stopped.

Initially the virus did not return and there was hope she had been effectively cured.

But last week it was announced that the girl, now four years old, was no longer in remission after nearly two years off the drugs.

"The unfortunate news of the virus rebounding in this child further emphasises the need to understand the early and refractory viral reservoir that is established very quickly following HIV infection in humans," Prof Barouch added.
Kai Deng and Robert Siliciano, of the School of Medicine at Johns Hopkins University, in Baltimore, Maryland, commented: "These data indicate that the viral reservoir could be seeded substantially earlier than previously assumed, a sobering finding that poses additional hurdles to HIV eradication efforts.

"Although early treatment may not prevent reservoir seeding, it has been consistently shown to reduce the size of the reservoir."

They highlighted significant differences between these experiments and the human HIV infection, but concluded that the findings "suggest new approaches in addition to early treatment will be necessary to eradicate HIV infection".

HIV establishes viral reservoirs with surprising speed

In a sobering discovery, researchers say that rapid treatment of HIV-like infections in monkeys failed to prevent the establishment of persistent viral reservoirs in as little as three days.

The study, published Sunday in the journal Nature, comes on the heels of news that the so-called Mississippi Baby -- a child once considered functionally cured of HIV due to antiretroviral drug treatment hours after her birth -- had in fact been infected with the virus all along.

While researchers had begun to hope that there was a window in which the virus could be prevented from establishing a permanent foothold within its host, that possibility now seems much less likely.

"We show that the viral reservoir can be seeded substantially earlier than previously recognized," wrote lead study author and Harvard Medical School virologist James Whitney, and colleagues.

HIV attacks CD4 white blood cells -- critical components of the body's immune system. The virus then uses the cells to manufacture copies of itself, destroying the blood cell in the process and steadily eroding the body's internal defenses.

However, in some cases, the virus will lay dormant within a white blood cell, only to begin reproducing itself at a later date. The virus cannot be killed in this dormant state -- either by the body's immune system or by antiretroviral drugs -- and this latent reservoir of infection has proved to be the biggest obstacle to finding a cure.
In the latest study, researchers infected 20 adult rhesus monkeys with simian immunodeficiency virus, or SIV, the simian equivalent of HIV, the disease that causes AIDS.

Some of the monkeys were treated with a cocktail of antiretroviral drugs three days after infection, yet prior to when the virus could be detected in the monkeys' bloodstream. Other monkeys received the drug treatment at seven, 10 and 14 days after infection, when evidence of the illness could be detected.

Therapy was stopped after 24 weeks. While researchers had hoped the virus would not reappear in the monkeys that were treated in three days, it in fact rebounded in all of the animals.

The researchers, however, did note that it took about three weeks for the virus to rebound in the monkeys that received drug treatment after three days, where it took only one or two weeks in the other monkeys.

In an accompanying News & Views article, Kai Deng and Dr. Robert Siliciano, both HIV researchers at Johns Hopkins University Medical School, noted that further research was needed to confirm the study's results.

"Substantial differences exist between SIV infection in rhesus macaques and HIV-1 infection in humans," the pair wrote.

Nonetheless, they called the paper's findings "striking," as they argued that still newer medical approaches are needed to eradicate HIV.

AUSTRALIAN scientists have developed a drug that not only shows promise in reducing HIV levels hidden in cells, but can lessen the impact the virus has on the immune system.

The results of a trial carried out on 21 HIV-positive people in Thailand revealed the drug BIT225 could reduce the over-activation of the immune system that can lead to their immune ageing and neurological decline.

The antiviral drug targets long-living immune cells called macrophages, which exist in the liver, brain, gut and lungs and slowly replicate HIV.

“If you take patients off anti-retroviral drugs, the virus that has been hiding in the reservoirs rebounds,” said CEO of the Sydney drug company Biotron, Dr Michelle Miller.

She said their drug targets the underlying reservoirs and had been previously shown to reduce viral levels in patients.

Results of the phase 2 trial, presented at the 20th International AIDS Conference in Melbourne on Wednesday, showed it could also reverse HIV-induced impairment of the immune system.
The company’s virologist, Dr John Wilkinson, said the aim of the drug was to reduce the virus levels, but also dampen down the associated immune activation.

If it successfully moves through clinical trials, the drug could be used in combination with other medications as part of a HIV eradication strategy.

The conference also heard how a new vaccine that could protect monkeys from an infectious immunodeficiency virus, was raising hopes the findings could be replicated in human HIV patients.

Canadian geneticist Dr Ma Luo said they allowed 12 monkeys to resist infection from the SIVmac239 virus, which is often used as the animal and disease model that best reflects HIV infection in humans.

Six monkeys did not receive the vaccination, but they contracted higher levels of the virus.

The team of researchers from Canada, the US and Spain developed the virus, which Dr Luo said they believed worked by forcing the virus to change into a less infectious form.

“We know the challenge for HIV vaccine is targeting the immune system itself. The HIV virus is diverse and develops very fast,” she said.

“With this vaccine, we think, is the mutations surrounding the protein occurring to lower the viral load and make the virus non-infectious.”

Dr Luo said further research was needed to show that the vaccine was first safe to use in humans, and that it was effective at preventing the HIV virus.

>>> AIDS-FREE GENERATION CLOSER

FORMER US President Bill Clinton says the first AIDS-free generation is within reach, but only if more exposed children born to infected mothers get immediate treatment.

Just 30 seconds into his speech at the 20th International AIDS 2014 Conference at Melbourne Convention and Entertainment Centre, about 20 protesters rose from their seats and started chanting as they walked towards the stage.

The group was calling for a “Robin Hood” tax on financial transactions such as currency and bonds to fund the fight against AIDS and HIV. Mr Clinton gave them a minute to continue chanting, before asking them to repay the favour and give him a chance to speak, at which time they returned to their seats.

For the past 12 years, Mr Clinton’s foundation has helped reduce the cost of AIDS medications in developing countries. He said when they started work in 2002, barely 100,000 people outside Latin America were getting medication.
Despite advances in antiretroviral therapies that suppress the virus and keep people alive for longer, he said more than 2 million people were infected each year and 1.5 million people were dying from related illnesses.

“In 2002 there were people seriously arguing that medicine would be too expensive and we should simply promote prevention. Now we celebrate a very different reality,” Mr Clinton said.

“We’re on the steady march to rid the world of AIDS. We have proven that an end to AIDS is possible.”

Clinton also used his speech to pay tribute to the six AIDS researchers and lobbyists who died in the MH17 plane crash.

In a discovery that raises hope for AIDS cure, two Australian men have been found to be HIV-free after receiving stem cells to treat cancer. Reuters

In a discovery that raises hope for AIDS cure, two Australian men have been found to be HIV-free after receiving stem cells to treat cancer.

The two patients' virus levels became undetectable after bone-marrow therapy with stem cells.

They are still on antiretroviral therapy (ART) "as a precaution", but those drugs alone could not be responsible for bringing the virus to such low levels, said David Cooper, director of the Kirby Institute at the University of New South Wales in Sydney, who led the discovery.

Cooper began searching for patients who had been purged of the HIV virus after attending a presentation by a US team last year at a conference of the International AIDS Society in Kuala Lumpur.

At that meeting, researchers from Brigham and Women’s Hospital in Boston, Massachusetts, reported that two patients who had received stem-cell transplants were virus-free.

Cooper and his collaborators scanned the archives of St Vincent’s hospital in Sydney, one of the largest bone-marrow centres in Australia.

"We went back and looked whether we had transplanted [on] any HIV-positive patients, and found these two," said Cooper.
The first patient had received a bone-marrow transplant for non-Hodgkin’s lymphoma in 2011.

His replacement stem cells came from a donor who carried one copy of a gene thought to afford protection against the virus. The other had been treated for leukaemia in 2012. Because of the risk of relapse, Cooper’s team will not claim that their patients are cured, 'nature.com' reported.

However, Cooper said the results show that "there is something about bone-marrow transplantation in people with HIV that has an anti-HIV reservoir effect, such that the reservoirs go down to very low levels. And if we can understand what that is and how that happens, it will really accelerate the field of cure search."

Stem-cell transplant in itself cannot be used as a routine HIV treatment, because of the high mortality (10 per cent) associated with the procedure, researchers said.

Earlier this month, the search for AIDS cure suffered a major setback when a child in the US, who was thought to have been cured of HIV after intensive drug therapy, was found with detectable levels of the virus.

AIDS cure may come, but can we afford the cost?

ANDRÉ PICARD - PUBLIC HEALTH REPORTER
MELBOURNE, AUSTRALIA — The Globe and Mail

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Can AIDS be cured?

The answer to that question, which until recently many scientists dared not ask out loud, is creeping slowly toward the affirmative, but with some important provisos.

“Cure is an emotional word,” said Sharon Lewin, head of the Centre for Biomedical Research at the Burnet Institute in Melbourne, Australia. “Everybody wants to find a cure. It’s an aspirational goal.”

In the three decades since the AIDS-causing virus HIV was discovered, scientists have learned how to keep it from replicating – with cocktails of antiretroviral drugs – and, as a result, the death rate among people living with HIV-AIDS has dropped precipitously.

But researchers haven’t yet figured out how to eradicate the virus from the body completely, which means life-long treatment is required, and that can result in serious side effects, like heart disease, and be costly.

HIV, unlike any other pathogen, seems to have the ability to lie dormant and undetectable in reservoirs.

“Cure means no more virus, and no more treatment,” Dr. Lewin said.

Given the ability of the virus to hide, that is unlikely, she said.

But what is possible, and being seen with increasing frequency, is long-term remission, essentially keeping the virus at undetectable levels in the body so it cannot ravage the immune system. But stopping treatment remains risky.

That became clear in the case of the Mississippi baby. The baby, born HIV-positive, began treatment 31 hours after being born and continued for 18 months. Treatment was discontinued and the virus was undetectable, suggesting she was cured. But after 27 months living seemingly HIV-free, signs of the virus returned, and treatment had to be resumed.
Another intriguing example is the VISCONTI cohort, a group of 14 French patients who were infected between 1996 and 2002, all of whom began treatment within 10 weeks of infection, but subsequently stopped treatment. The virus remained undetectable in their bodies for at least four years, and some are still virus-free after more than a decade.

That’s long-term remission, but is it a cure?

Then there are the “Canadian babies,” five children who began treatment with antiretroviral drugs shortly after birth. After the case of the Mississippi baby made headlines, researchers looked back and found four of them showed no signs of HIV, between two and eight years later, though they are continuing treatment; the fifth baby stopped treatment and the virus re-appeared.

To date, only one patient out of the 78 million who have been infected HIV is believed to have been cured in the true sense of the word – permanently.

Timothy Ray Brown, better known as the Berlin patient, was diagnosed with HIV in 1995 and then with leukemia in 2006. He was treated with a bone marrow transplant from a person with something called a CCR5 receptor mutation, a rare genetic anomaly that makes people naturally resistant to HIV. Mr. Brown essentially got a new, supercharged immune system, stopped treatment for HIV and has been HIV-free for the eight years since. (He did, however, suffer a second bout of leukemia and needed another transplant.)

What is not often discussed is that the treatment that cured Mr. Brown almost killed him and left him disabled, and at least two other patients who underwent the same treatment died.

Still, the notion of rebuilding the immune system is intriguing, if not practical on a large scale. At least four other patients, two in Boston and two in Sydney, all of whom had leukemia, have been “cured” this way.

In the Boston patients, however, HIV returned to detectable levels three and seven months after the transplant. The virus remains undetectable in the Sydney cases, but they have not stopped antiretroviral treatment.

“The ethics of a possible HIV cure are complex,” said Dr. Jintanat Ananworanich, associate director of the U.S. Military HIV Research Program in Bethesda, Md.

To know whether a patient has been cured, treatment has to be discontinued, which can put their life at risk; the best candidates for research are therefore healthy, and not the most in need; and the cost of finding a cure is enormous, which may divert funds from other prevention and treatment efforts.

Still, scientists have learned a couple of important lessons from these unusual cases: Treating people as soon as possible after infection is important, and they need better tools to find the virus because “undetectable” does not mean it is gone.
What researchers are trying now is a “kick and kill approach.” They are trying to kick the virus out of its hiding place and then kill it.

One of the most discussed papers at last month’s 20th International AIDS Conference in Melbourne was by a group of Danish researchers who used a cancer drug, romidepsin, to lure HIV out of reservoirs before blasting it with high doses of drugs. Unfortunately, there was still virus left behind, meaning a cure remains elusive.

“Many very smart people don’t think a cure is possible or will ever be possible,” said Steven Deeks, a researcher with the Positive Health Program (AIDS Program) at San Francisco General Hospital.

“My personal opinion is that if it is possible, and we can do it, it’s going to take many, many years.”

And then, Dr. Deeks said, will come an even larger dilemma: Will the cure be affordable and accessible to all?

http://www.theglobeandmail.com/life/health-and-fitness/health/aids-cure-may-come-but-can-we-afford-the-cost/article20290576
HIV 'reservoirs' may form earlier than expected

"Early HIV drugs 'may not stop virus'," BBC News reports. The report is based on a study of HIV treatments in monkeys, and has been linked by the BBC to the emergence of HIV in a four-year-old girl thought to have been cured of the virus as the result of treatment from birth – the so-called "Mississippi girl".

HIV infection levels in the blood can be managed through antiretroviral therapy (ART), allowing most people to live a normal life. But if the therapy is stopped, the virus re-emerges from "viral reservoirs" in the body that are immune to ART.

HIV is a virus transmitted by bodily fluids, most commonly through unprotected sex. In 2012 it was estimated 98,400 people are living with the HIV infection in the UK, but a further 21,900 people are infected without realising. Practising safe sex is the best way to prevent infection and spreading the virus.

It was thought these reservoirs are formed during the initial infection, when the virus spreads to the bloodstream. But this study has shown the monkey version of HIV can form reservoirs within three days of infection. This occurs before the virus is detectable in the bloodstream.

It is likely such rapid development of reservoirs also occurs in humans and gives very limited chances of success for current ART to prevent their formation.

This is likely to have happened to the "Mississippi girl", who is reported to have been given ART within hours of birth and for 18 months afterwards, until she stopped attending appointments. The virus was not detectable and she was believed to have been cured, but it has now resurfaced.

Read the latest BBC report on the "Mississippi girl" for more information.

Drug development for treating the HIV virus will therefore continue to focus on new techniques to target the cells in these reservoirs.

Where did the story come from?
The study was carried out by researchers from Harvard and universities and institutes in Massachusetts, Bioqual in Maryland, Gilead Sciences in California, and the US Military HIV Research Program in Maryland.

It was funded by the National Institutes of Health, the US Army Medical Research and Material Command, the US Military HIV Research Program, and the Ragon Institute of MGH, MIT and Harvard.

The study was published in the peer-reviewed journal, Nature.

The BBC reported the story accurately and informatively.
**What kind of research was this?**

This was an animal study using rhesus monkeys to investigate simian immunodeficiency virus (SIV), a monkey virus similar to HIV. The researchers wanted to investigate the speed of infection – in particular, how quickly "viral reservoirs" are formed.

HIV infection is known to create what are known as viral reservoirs. These are pockets of infected memory CD4+ cells that are the source of reactivation of the virus when antiretroviral therapy (ART) is stopped.

It was believed these reservoirs are formed during the initial stages of infection, when the virus is present in the bloodstream (viraemia), but it was not known how quickly they form.

As ART is largely ineffective against the cells located in the reservoirs, the researchers wanted to know if there is a window of opportunity after infection to prevent the reservoirs forming in the first place.

**What did the research involve?**

Twenty rhesus monkeys were given an injection of SIV into the lining of the rectum. At this point, the virus was not detectable in the bloodstream.

Some of the monkeys then received antiretroviral therapy (ART), starting on either day 3, 7, 10 or 14 after infection and continued for 24 weeks. Control monkeys did not receive ART.

The monkeys were monitored during the six months to see if and when the virus was detectable in the bloodstream, lymph nodes and rectal lining. They were also monitored for 24 weeks after the ART was stopped to see if or how quickly the SIV came back.

**What were the basic results?**

After stopping treatment, SIV infection became detectable in the bloodstream of all of the monkeys. This took a little longer to occur in the monkeys that started treatment on day 3 (mean 21 days) compared with days 7, 10 or 14, (mean 7 days), but it still occurred.

This indicated the viral reservoirs, where cells are able to effectively hide from ART, are formed within the first three days of infection with SIV.

The virus was not detectable in the blood of monkeys given ART on day 3, either before the injections started or during the next 24 weeks. The researchers did find the virus in lymph nodes and the rectal lining, but both reduced during ART treatment.

All other monkeys had detectable, rapidly increasing levels of the virus in the blood, lymph nodes and rectal lining. The ART reduced the levels compared with the control monkeys.

The levels of the treated monkeys became undetectable within three to four weeks, and this continued for the treatment period. The control monkeys had sustained, high levels of virus in the bloodstream throughout the length of the study.
Monkeys given ART on days 10 and 14 had viral infection in the lymph nodes, which initially reduced a little but then remained constant from week 12.

**How did the researchers interpret the results?**
The researchers conclude that: "These data demonstrate that the viral reservoir is seeded rapidly after intrarectal SIV infection of rhesus monkeys, during the 'eclipse' phase, and before detectable viraemia. This strikingly early seeding of the refractory viral reservoir raises important new challenges for HIV-1 eradication strategies."

**Conclusion**
This study has shown SIV infection spreads to locations in the bodies of monkeys, forming "viral reservoirs" during the first three days of infection, before the virus is detectable in the bloodstream.

Cells in the reservoirs are resistant to treatment with ART and are the source of rebound infection when treatment is stopped. Because of the similarities between SIV and HIV, it is likely an equivalent chain of events occurs when humans are infected with the HIV virus.

This appears to have been the case for the "Mississippi girl", a four-year-old girl who was treated with ART for the first 18 months of her life and was presumed to be cured, but has now shown evidence of the infection.

This research indicates drug-resistant HIV reservoirs are likely to occur rapidly during infection in humans, and remain a challenging target for drug development.

Although it is not yet possible to eradicate HIV infection, long-term treatment with ART can help most people live full and normal lives.

**Analysis by Bazian. Edited by NHS Choices. Follow Behind the Headlines on Twitter. Join the Healthy Evidence forum.**

Two cancer patients ‘cured’ of AIDS after bone marrow transplants
July 19, 2014 12:00AM

Sean Parnell
Health Editor. Brisbane

TWO Australians with HIV and cancer have been given bone transplants that cured their cancer and all but eradicated the AIDS virus from their bodies, researchers will report today.

Ahead of the 20th International AIDS Conference in Melbourne, where attendees will mourn the loss of 108 delegates and family members in the Malaysia Airlines tragedy, the bone marrow breakthroughs give new hope of innovative HIV treatments.

Celgene drug can drive HIV out of hiding, study claims

Published July 22, 2014

An anti-cancer drug made by the U.S. biotech firm Celgene can re-activate hidden HIV in patients so that it can be detected, bringing researchers closer to being able to treat it, Danish scientists said on Tuesday.

In a small study presented at an international AIDS conference in Australia, the researchers said the finding was a "step in the right direction" toward finding a cure for the viral disease but that many years of research are still needed.

"There is still a long way to go and many obstacles to overcome before we can start talking about a cure against HIV," said Ole Schmeltz Sogaard, who led the research team from Aarhus University and Aarhus University Hospital in Denmark, in a statement.

The drug, known generically as romidepsin and under the brand name Istodax, is licensed to treat a type of cancer called T-cell lymphoma. In this study, however, it was investigated as a potential HIV therapy.

Human immunodeficiency virus (HIV) infection can be kept at very low levels by anti-AIDS drugs, but there is still no cure that can eradicate HIV from the body.

Some 35 million people worldwide are infected with HIV, and the global AIDS epidemic has killed 39 million since it began in the 1980s, according to the latest data from the United Nations AIDS program, UNAIDS.

Scientists working to find a cure know the virus can hide in a state of hibernation in cells called CD4 cells, which are part of the body's immune system.

CD4 cells cannot fight the AIDS virus themselves, but killer T-cells can if they are able to tell whether or not a CD4 cell contains the hibernating HIV.

Sharon Lewin, co-chair of the AIDS2014 conference in Melbourne Australia and a professor of infectious diseases who was not directly involved in this study, said the results of the study were
significant and encouraging because they showed "we can wake up the virus reservoir and make enough of (it) to leave the cell, making it visible to an immune response".

The Danish team gave three once-weekly infusions of romidepsin to six HIV-positive adult patients who were already taking antiretroviral AIDS drugs and whose so-called "viral load" was undetectable.

They found that romidepsin increased the virus production in HIV-infected cells between 2.1 and 3.9 times above normal and that the viral load in the blood increased to measurable levels in five out of six patients.

"We have now shown that we can activate a hibernating virus with romidepsin and that the activated virus moves into the bloodstream in large amounts," Schmeltz Sogaard said in a statement about the results.

When the virus is activated and moves toward the bloodstream it leaves a trace on the outside of the infected CD4 cells, he explained. In principle, this means killer T-cells would be able to trace and destroy the HIV-infected CD4 cells.

The Danish team said the next step is a larger trial where the researchers will combine romidepsin activation of hidden HIV with an experimental vaccine called Vacc-4x being developed by the Norwegian biotech firm Bionor Pharma to strengthen the ability of T-cells to fight HIV.

Los tres días clave de los virus de la inmunodeficiencia

El tiempo que tarda en asentarse es la llave de la prevención

EMILIO DE BENITO Madrid 20 JUL 2014 - 19:06 CEST

Tres días es lo que tarda el virus de la inmunodeficiencia en simios (VIS), el microorganismo más parecido al VIH, en llegar a los reservorios (células en las que queda para siempre). La medición, que han hecho con exactitud científicos del Beth Israel Deaconess Medical Center de Boston, es clave para evitar que el macaco afectado se convierta en un portador del VIS para toda su vida. El estudio arroja luz sobre lo que sucede en los humanos con el VIH, y explica varios de los últimos logros y decepciones que ha dado la investigación sobre este agente infeccioso en los últimos años. El trabajo se publica en la última edición de Nature.

Los propios investigadores, dirigidos por Dan Baruch, advierten de que, aunque el VIS es el modelo animal más cercano al VIH, no todo lo que sucede en macacos es extrapolable a personas. Los casos de los fracasos de los modelos en vacunas contra el VIH de los últimos años así lo demuestran. Pero ese plazo también sirve para explicar el éxito de dos de las aproximaciones más exitosas de los últimos años, que van a ser unas de las estrellas de la conferencia mundial contra la enfermedad que empezó el sábado en Melbourne: las profilaxis post y preexposición. Ambas se parecen en que buscan evitar que una persona se infecte proporcionándole medicación justo antes o después de una situación de riesgo. Es precisamente ese tiempo, ese plazo de tres días, el que las hace efectivas, y el que explica que pierdan eficacia pasado ese periodo.

Precisamente la preexposición ha sido recomendada recientemente como complemento del uso de los preservativos por la Organización Mundial de la Salud y el centro de Control de Enfermedades...
para personas que, por ejemplo, tengan una pareja con VIH, pero el Centro Europeo de Control de Enfermedades, por ejemplo, no lo tiene tan claro. No porque dude de su eficacia, sino por su coste y porque parece enviar el mensaje de que es mejor usar unas pastillas que el preservativo.

La conferencia de Melbourne, que arrancó con el recuerdo a los más de 100 inscritos que fallecieron en el ataque al avión de Malaysia Airlines, también debatirá sobre los últimos dos casos australianos de dos hombres que, tras recibir un trasplante de médula, parecen haber eliminado el virus de su organismo. Este abordaje no es aplicable de manera general, pero muestra el papel de las células hematopoyéticas como reservorio del virus. En cualquier caso, en la reunión no se esperan grandes avances, aparte de seguir con lo que ya hay y se sabe que funciona. Por eso, las ONG participantes se han fijado que para 2020 todos los infectados reciban tratamiento. Ello supondría multiplicar por tres el actual alcance de la medicación.

http://sociedad.elpais.com/sociedad/2014/07/20/actualidad/1405875909_623856.html
El fracaso en la cura del VIH no hace tirar la toalla

- Tras la niña de Mississippi, el virus reaparece también en dos pacientes de Boston
- Un consorcio busca repetir el caso de Tim Brown mediante un trasplante de médula
- Hasta ahora se ha intentado repetir el procedimiento sin éxito en 20 ocasiones

AINHOA IRIBERRI Madrid
22/07/2014

En 2008, Timothy Brown se convirtió en el primer paciente seropositivo y afectado por una leucemia en el que se había conseguido la erradicación de la infección por el VIH. Al principio, todo fueron cautelas, pero seis años después, la página de Facebook de la fundación que lleva su nombre da fe de que la cura es real. El trasplante de médula ósea de un donante con la mutación CCR5 -una variante genética que aporta inmunidad al virus del sida- funcionó y sirvió para la única cura documentada de esta infección.

Desde el principio se supo que el procedimiento no iba a poder aplicarse de forma genérica. En primer lugar, sólo se podría plantear en personas que requieran del trasplante por tener un cáncer del sistema inmunológico ya que, como recuerda el especialista en enfermedades infecciosas del Hospital Gregorio Marañón Juan Carlos López Bernaldo de Quirós, se trata de un procedimiento con elevadas tasas de mortalidad.

Aún así, la comunidad científica observó con atención cada caso similar, aunque no fuera exactamente el mismo. En dos de aquellos casos saltó la alarma, para bien. Se trataba de dos pacientes del Hospital Brigham and Women of Boston (EEUU) afectados por un linfoma. Su médico optó también por el trasplante de células madre provenientes de una médula ósea de donante. En la conferencia anual de la Sociedad Internacional de Sida de 2013, se informó de que, tras el procedimiento, a ambos se les había retirado la medicación antirretroviral.

A pesar de que su donante -al contrario que el de Brown- no era positivo a la mutación CCR5, ambos seropositivos parecían haber vencido al virus, tras tres y seis meses sin tomar antirretrovirales respectivamente.

La alegría duró poco. En diciembre, los medios se hacían eco del fracaso e incluso los Institutos Nacionales de la Salud (NIH) de EEUU emitían una especie de nota de condolencia. Ahora, la revista...
Annals of Internal Medicine publica las claves del fallo, que no por esperado dejó de suponer un jarro de agua fría para la comunidad científica.

El hematólogo que curó a Brown, Gero Huetter, explica por correo electrónico a EL MUNDO que para él "no fue una sorpresa" dicho fracaso, porque el virus ha regresado tras todos los casos de trasplante de células madre si se dejaban de prescribir antirretrovirales. "La principal diferencia con los llamados pacientes de Boston fue el largo tiempo pasado desde el trasplante y los prometedores resultados en cuanto a disminución del virus, por lo que había cierta esperanza real de que se consiguiera la erradicación", subraya.

Como comenta López Bernaldo de Quirós, el fracaso en sí no causa sorpresa; la causa precisamente lo que parecía haber sucedido el pasado año. "De hecho, cuando se comunicaron los resultados negativos, sus autores estaban preparándolos como positivos, pero un nuevo análisis reveló que el virus había vuelto", explica.

Ahora, en la conclusión de la descripción científica de su fracaso, Timothy Henrich y sus colaboradores no pueden disimular su pesimismo. "El VIH puede volver incluso después de una reducción significativa del tamaño del reservorio. La cura del VIH se nos sigue escapando", escriben, justo en la misma semana en la que se celebra en Melbourne (Australia) la XX Conferencia Internacional de Sida en la que se hablará también de la eliminación del virus.

La niña de Mississippi

La publicación de Annals coincide casi en el tiempo con otro fracaso, el de la supuesta curación de un bebé estadounidense a quien el virus se le hizo indetectable tras un tratamiento antirretroviral de alta intensidad. El regreso de la infección también ha deprimido a la comunidad científica aunque, como subraya el infectólogo español, de nuevo no es una sorpresa. "Cuando se publicó el caso de la supuesta curación en The New England Journal of Medicine ya los autores describieron que había ADN del virus integrado en las células de la niña; de ahí no se puede sacar", apunta.

El editorial que acompaña la publicación del fracaso de los pacientes de Boston es también de tono pesimista, como indica el mismo título: Encontrar una cura para el VIH: mucho trabajo por delante.

Sin embargo, cabe preguntarse porqué si se consiguió la cura en Brown con un procedimiento exitoso ya descrito (el trasplante de células madre de médula ósea de un donante con la mutación CCR5) no se repite la hazaña, aunque se tenga que restringir a pacientes seropositivos y con cánceres del sistema linfático. Bernaldo López de Quirós apela a que la mutación no es algo frecuente: "Afecta como mucho al 2% de la población caucásica del norte de Europa y además tendrían que ser donantes histológicamente compatibles con el receptor".

Huetter, por su parte, comenta a este diario que se ha intentado repetir el procedimiento en 20 ocasiones desde que Brown lograra la curación. "En cuatro casos, se encontró un donante compatible, pero en dos se tuvo que suspender el trasplante por otros motivos; otro de los pacientes murió antes de que se le pudiera trasplantar y el último recibió las células madre pero falleció dos meses después del procedimiento".

El único médico al que parece no haber vencido el pesimismo subraya que, gracias al apoyo de la asociación http://www.amfar.org/ amfAR se acaba de establecer un consorcio europeo, dotado con alrededor de medio millón de euros, para buscar la repetición del caso de Brown, dado que en Europa es más fácil encontrar a individuos con esa mutación. El consorcio lo dirige el investigador de Irsi-Caixa Javier Martínez-Picado y lo componen distintos científicos europeos que afirman tener pacientes seropositivos y con cáncer en necesidad de un trasplante de médula, lo que les haría aptos para intentar buscar la doble curación. Quizás el próximo Timothy Brown sea español.

http://www.elmundo.es/salud/2014/07/22/53ccf28122601df56c8b457d.html
Latest HIV 'cure' claims prompt calls for more caution

16:32 22 July 2014 by Andy Coghlan and Jessica Hamzelou
For similar stories, visit the HIV and AIDS Topic Guide

HIV has been undetectable for more than three years in the blood of two Australian bone-marrow recipients. But recent relapses of other patients who had no detectable virus have dampened optimism about how long the effects last before the virus bounces back.

"Results of relapses in other patients should serve to warn us about the use of the phrase 'viral clearance'," says Asier Sáez-Cirión of the Pasteur Institute in Paris. "We need to be careful about assumptions made when patients are still on antiretroviral drugs," he says.

The latest results from Australia, presented on Saturday in Melbourne, Australia, at the international AIDS 2014 conference, add to evidence that bone marrow transplants seem to have a strong but as yet unexplained antiviral effect in patients with HIV. However, the impact of the transplants is difficult to interpret because the patients have also remained on antiretroviral drug therapy (ART), making it difficult to disentangle the effects of bone marrow transplant and ART on viral levels.

The results also add to a growing catalogue of cases in which the virus is driven to such low levels that the outcome almost equates to a cure, either through bone marrow transplants or through early treatment of patients with ART before the virus can establish itself and hide away in the body (see timeline).

Growing cure club

"In our two patients, we can't detect HIV," said David Cooper of the University of New South Wales's Kirby Institute in Sydney, Australia, and presenter of the results from the patients who received transplants at St Vincent's Hospital in Sydney. "Nor can we find antibodies to the virus, as they all seem to have disappeared from our patients' serum," he said.

The results mean that in total, five people worldwide have now cleared detectable virus from their blood following bone marrow transplants. As in the previous cases, the two patients in Sydney received their transplants to treat blood cancers, but they also happened to be chronically infected with HIV: one since 2003 and one since 1987.

Only one of the five people has been completely cured of HIV. Timothy Ray Brown, also known as the "Berlin patient", owes his remission to the fact that he received a bone marrow transplant from a donor who was fully resistant to HIV.
HIV resistance is provided by a variant of a gene called *CCR5*. The normal version of the gene makes a protein on the surface of white blood cells that HIV uses as a "door handle" to invade and kill the cell. Donors who are fully resistant have two "faulty" versions of the gene, which produce a misshapen protein that the virus cannot latch onto.

**HIV strikes back**

Two further patients, known as the "Boston patients", received bone marrow from normal donors, but each patient naturally had single copies of a faulty version of *CCR5*, giving them partial resistance to the virus. However, both Boston patients relapsed late last year after appearing to have been cured last July, and had to resume drug treatments.

Neither of the Sydney patients had any natural resistance. One received a graft from a resistant donor who had one faulty copy of the *CCR5* gene. But the other patient’s bone marrow came from a donor who made normal versions of the gene, and yet the transplant recipient was still apparently able to suppress the virus.

"The bone marrow in this patient didn't contain any protective genes," says Cooper. "This is a very important clue, that an immune response prompted by the bone marrow transplant has such a strong anti-HIV effect," he says.

By experimenting on samples from the patients, Cooper hopes to work out how this happens. "Maybe we can find therapies that mimic what we're doing with the bone marrow transplant, but without having to do actual transplants," he says.

Both patients have remained on ART. "We need to study more of these patients to understand what the anti-HIV effect is, where the virus is hiding and when it would be safe to stop ART," says Cooper. He suspects that if the ART was stopped, the virus would come back.

Likewise, relapses have recently been reported in high-profile patients who appeared to have the virus under control through treatment early after infection. The best-known case, the "Mississippi baby", was reported last year. The child went without ART for four years and showed no sign of HIV infection, but resumed ART last month following the discovery that the virus had rebounded.

In light of the relapses, Sharon Lewin of Monash University in Melbourne, Victoria, also sounded a note of caution about the Australian results. "Given what we know with the Boston patients, the real test of whether transplantation has made a difference to the reservoir will be a treatment interruption", she says.

"All these cases are helpful in better understanding how transplantation can impact the reservoir," says Lewin. "I know there are several other similar cases around – but they are all still on ART."
The major barrier to finding a cure is that the virus lays dormant in tissues of the gut, lymph system, bloodstream and elsewhere, but reawakens and begins invading blood cells if patients halt ART. The best hope is using drugs such as vorinostat that completely "flush out" the virus so it can be killed outright with antiretroviral drugs. Trials with these treatments are currently under way in several laboratories around the world.

A timeline of "cures"

The two Australians appear to have been cleared of the virus. But this timeline shows it may be too soon to celebrate the results as cures. HIV has a nasty habit of hiding in the body and returning just when researchers think it’s gone for good.

November 2008 – Berlin patient "cured"

A haematologist in Berlin declares that a man with HIV has been "functionally cured" after receiving a bone marrow transplant from a donor with natural immunity to the virus. The transplant was intended to treat the man's leukaemia, but his doctor chose a donor with a gene mutation that prevents HIV from entering cells. Only one per cent of Europeans have this mutation, so the treatment is unlikely to work on a large scale.

March 2013 – Mississippi baby "cured"

A baby born with HIV is said to be "cured" after an intensive treatment programme that was started within 30 hours of her birth. The infant, known as the "Mississippi baby", was treated with a combination of three antiretroviral drugs for 18 months, after which doctors lost track of her. When she was taken to a clinic ten months later, physicians found her blood remained free of the virus.

March 2013 – Visconti cohort "cured"

Within weeks of the announcement of the "cured HIV baby" comes further good news: 14 adults with HIV appear to be functionally cured after receiving therapy for three years. These individuals, known as the Visconti cohort, still have HIV in their blood, but at levels that are low enough for the immune system to control the virus without drugs. None of these adults has the genetic mutation that might make a person naturally resistant to infection – it is the drug therapy that seems to offer long-term protection.

July 2013 – Boston patients "cured"

Two men known as the Boston patients appear to have rid their bodies of HIV, although researchers stop short of declaring them cured. Both men had already been diagnosed with HIV and were taking antiretroviral drugs when they received bone marrow transplants to treat Hodgkin's lymphoma, a form of blood cancer. A few years after receiving their transplants, the men stopped taking their HIV medicine. One man was found to be virus-free 15 weeks after halting his therapy, whereas the other man showed no signs of the virus seven weeks after he stopped taking his medicine.

December 2013 – HIV detected in Boston patients

Five months after the Boston patients were thought to have rid their bodies of active HIV, the virus is found to have returned in both of them. Both men resume taking antiretroviral drugs.
July 2014 – HIV detected in Mississippi baby
More bad news: the "Mississippi baby" thought to have been cured back in 2013 is diagnosed with the virus. The virus was identified in the blood of the girl, now nearly four, during a routine medical appointment.

http://www.newscientist.com/article/dn25933-latest-hiv-cure-claims-prompt-calls-for-more-caution.html#.VA8mV0hC608
Two HIV patients are showing no signs of HIV after receiving bone marrow transplants to treat cancer, according to a case presented by David Cooper, director of the Kirby Institute at the University of New South Wales, while speaking at a press briefing at the 20th International AIDS Conference (AIDS 2014) in Melbourne, Australia.

Although the two patients, both Australian men, are seemingly HIV-free, they are both still on antiretroviral therapy (ART) as a precaution. As we've seen in two Boston patients who had similar HIV remissions after bone marrow transplants, the virus rebounded months after treatment was discontinued.

According to a report in the journal Nature, after hearing about the Boston patients last year, Cooper and his team started looking through the archives of St. Vincent's hospital in Sydney to see whether similar transplants had occurred in any of their HIV-positive patients. They found these two patients.

The first patient had received a bone marrow transplant to treat non-Hodgkin's lymphoma in 2011, while the second patient received treatment for leukemia in 2012. Interestingly, the first patient's replacement stem cells came from a donor who carried one copy of a gene that's thought to protect against HIV. Whether this was the CCR5 receptor mutation (as was the case for Timothy Brown, the first man functionally cured of HIV) was not reported.

According to the Nature report:
Because of the risk of relapse, Cooper's team will not claim that their patients are cured. But, he says, the results show that "there is something about bone-marrow transplantation in people with HIV that has an anti-HIV reservoir effect, such that the reservoirs go down to very low levels. And if we can understand what that is and how that happens, it will really accelerate the field of cure search."

Stem-cell transplant in itself cannot be used as a routine HIV treatment, because of the high mortality (10%) associated with the procedure. An important next step will be to find more such cases and compare them, says Cooper, to try to identify where the virus might be hiding. "These patients are very precious examples to help us understand how we might manipulate the immune system to drive the reservoir down to these extraordinary low levels."
Certainly, this is promising news, especially in light of the recent disappointing relapses in the Boston patients and "Mississippi Child," but it remains to be seen whether these two Australian patients are truly cured or need to remain on treatment.

http://www.thebodypro.com/content/74786/two-australian-men-aquotclearedaquot-of-hiv-after-.html
Setbacks and Progress in the Search for an HIV Cure

By Liz Highleyman  July 22, 2014

The quest for a cure for HIV has been one of the themes at the 20th International AIDS Conference (AIDS 2014) taking place this week in Melbourne, with scientists reporting both advances and setbacks.

Jintanat Ananworanich, formerly of the Thai Red Cross and now with the U.S. Military HIV Research Program, gave an overview of "Where Are We Now and Where Are We Going?" with cure research at an opening plenary on Monday.

After reviewing some special cases that offer proof-of-concept that it may be possible to control HIV at least temporarily without antiretroviral therapy (ART) -- including the Berlin Patient, the Boston Patients, and the Mississippi Baby -- she concluded that success will likely require a combination approach, for example, early ART, agents that overcome viral latency, gene therapy to protect CD4 cells from infection, and therapies that strengthen immune response.

Researchers also discussed cure-related work in oral abstract sessions at the main conference and at a two-day pre-conference "Towards an HIV Cure" symposium organized by the International AIDS Society (IAS). Several of them summarized their findings and offered their thoughts about future directions at an IAS press briefing on Monday.

Deborah Persaud from Johns Hopkins gave an update on the Mississippi Baby (now a child nearly four years old) who just before the conference was found to still have HIV after having no detectable virus while off ART for more than two years. This case suggests that HIV establishes latent reservoirs very early -- the child started treatment just 30 hours after birth -- and even a few remaining infected cells are enough to rekindle viral replication.

Though disappointing for the child, who has resumed antiretroviral treatment and is in good health, Persaud said, "we have learned a lot from this case and it provides a strong rationale for moving forward with a clinical trial" of very early combination therapy for infants.

HIV progress offers hope in tragic week

By Xinhua in Sydney, Australia (China Daily) Updated: 2014-07-22 07:35

In what has been a grim week for the international AIDS community, with the loss of former International AIDS Society president Joep Lange and Art Aids leader Jacqueline van Tongeren among the victims on Flight MH17 in Ukraine—reports have been confirmed on Monday that two HIV-positive men who were treated in Sydney now have "undetectable levels" of the virus.

The patients, who were treated at St Vincent's Hospital in partnership with the University of New South Wales' Kirby Institute, have undetectable levels of HIV more than three years after undergoing bone-marrow transplants. They were the first cases of HIV being successfully cleared in Australia.

The international AIDS community is mourning the deaths of researchers, activists, health workers and people with HIV after their plane crashed in Ukraine last week. They were traveling to Melbourne for a global AIDS conference.

David Cooper, director of the Kirby Institute at UNSW Australia, found some solace in the breakthrough, in the wake of the loss of his friend and colleague, Lange, with whom he worked for more than 30 years.

Cooper said Lange had "an absolute commitment to HIV treatment and care in Asia and Africa". "Joep was absolutely committed to the development of affordable HIV treatments, particularly combination therapies, for use in resource-poor countries," Cooper said.

The breakthrough was to be heralded at a major gathering—Towards an HIV Cure Symposium—which was scheduled as part of the 20th International AIDS Conference in Melbourne. The gathering has instead became a focal point of grief for the community.

Despite the work being overshadowed for now by the plane crash, the long-term benefits of the Kirby Institute's research will be felt for years to come, Cooper said, as it herald's a new direction in research and new hope for HIV-positive people with leukemia and lymphoma.

In the Sydney cases, one patient had a successful bone marrow transplant in 2010 for non-Hodgkin's lymphoma. His donor had one of two possible copies of a gene that affords protection against the virus.

In 2011, a second man underwent a similar procedure for acute myeloid leukaemia, although his bone marrow donation had no genetic fingerprint affording protective immunity.

Both cleared the HIV virus but remain on anti-retroviral therapy as a protective measure.
"We're so pleased that both patients are doing reasonably well years after the treatment for their cancers and remain free of both the original cancer and the HIV virus," Cooper said.

Until now, the only person thought to have cleared HIV is an American man, Timothy Ray Brown, who had two bone marrow transplants in Berlin in 2007 and 2008.

In Boston, two other patients underwent similar transplants in 2012, but the transplanted cells did not contain the CCR5 gene mutation. In both cases the virus returned after anti-retroviral treatment was stopped.

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*Flower bouquets are laid on Sunday at a sign for the AIDS Conference 2014 in Melbourne in memory of those killed in the Malaysia Airlines crash over Ukraine.

*Esther Lim / Agence France-Presse

*(China Daily 07/22/2014 page11)*

Cancer treatment clears two Australian patients of HIV

Patients’ virus levels became undetectable after bone-marrow therapy with stem cells.

Katia Moskvitch 18 July 2014

Scientists have uncovered two new cases of HIV patients in whom the virus has become undetectable.

The two patients, both Australian men, became apparently HIV-free after receiving stem cells to treat cancer. They are still on antiretroviral therapy (ART) “as a precaution”, but those drugs alone could not be responsible for bringing the virus to such low levels, says David Cooper, director of the Kirby Institute at the University of New South Wales in Sydney, who led the discovery. A year ago, a different group of researchers had reported cases with a similar outcome.

Cooper presented details of the cases today at a press briefing in Melbourne, Australia, where delegates are convening for next week’s 20th International AIDS Conference. The announcement came just a day after the news that at least six people heading to the conference died when a Malaysia Airlines flight was shot down in Ukraine.

Cooper began searching for patients who had been purged of the HIV virus after attending a presentation by a US team last year at a conference of the International AIDS Society in Kuala Lumpur. At that meeting, researchers from Brigham and Women’s Hospital in Boston, Massachusetts, reported that two patients who had received stem-cell transplants were virus-free.
Cooper and his collaborators scanned the archives of St Vincent’s hospital in Sydney, one of the largest bone-marrow centres in Australia. “We went back and looked whether we had transplanted [on] any HIV-positive patients, and found these two,” says Cooper.

The first patient had received a bone-marrow transplant for non-Hodgkin’s lymphoma in 2011. His replacement stem cells came from a donor who carried one copy of a gene thought to afford protection against the virus. The other had been treated for leukaemia in 2012.

Unfortunately, several months after the 'Boston' patients stopped taking ART, the virus returned. An infant born with HIV in Mississippi who received antiretroviral therapy soon after birth, then stopped it for more than three years, was thought to have been cured, but has had the virus rebound, too.

Natural resistance
At the moment, there is only one person in the world who is still considered cured of HIV: Timothy Ray Brown, the 'Berlin patient', who received a bone-marrow transplant and has had no signs of the virus in his blood for six years without ART. The bone marrow received by the Berlin patient came from a donor who happened to have a natural genetic resistance to his strain of HIV.

“It is very possible that the Australian men would relapse if they were to stop antiretroviral therapy,” says Timothy Henrich, an infectious-disease specialist at Brigham and Women’s Hospital who helped to treat the Boston men.

Because of the risk of relapse, Cooper’s team will not claim that their patients are cured. But, he says, the results show that “there is something about bone-marrow transplantation in people with HIV that has an anti-HIV reservoir effect, such that the reservoirs go down to very low levels. And if we can understand what that is and how that happens, it will really accelerate the field of cure search”.

Stem-cell transplant in itself cannot be used as a routine HIV treatment, because of the high mortality (10%) associated with the procedure. An important next step will be to find more such cases and compare them, says Cooper, to try to identify where the virus might be hiding. “These patients are very precious examples to help us understand how we might manipulate the immune system to drive the reservoir down to these extraordinary low levels.”

Henrich says that a larger pool of patients will provide a better understanding of how the immune system can be harnessed to fight HIV, adding that there could be many more people around the world whose virus has become undetectable. “I think the main finding is further confirmation that transplant can reduce the level of virus in peripheral blood to a level which is undetectable by research [tests],” he says.

Gero Hütter, a haematologist now at Heidelberg University in Germany who performed the transplant on the Berlin patient, says that his case “taught us that a cure could be possible. This is an important motivation to go on in this direction but it does not mean that we will reach this goal
for all patients. HIV is still an incalculable enemy, so there might be limitations that we are not aware of now."

AIDS 2014 Conference Highlight from Sharon Lewin [VIDEO]

As the 20th International AIDS Conference drew to a close in Melbourne, AIDS 2014 co-chair Sharon Lewin offered an overview of some highlights of the meeting at a Friday press briefing.


Sharon Lewinspeaks at AIDS 2014 media briefing, July 25, 2014

7/25/14

Reference


An additional 2 people with long-term HIV infection have no evidence of infectious virus or viral genetic material following bone marrow stem cell transplants to treat leukemia or lymphoma, researchers reported at the 20th International AIDS Conference last month in Melbourne. While these individuals remain on antiretroviral therapy (ART) and therefore cannot be considered functionally cured, they offer further evidence that HIV may be controlled off ART in some cases.

Several approaches are being explored in research towards a cure for HIV. The Berlin Patient, Timothy Brown, has remained free of detectable HIV for several years after receiving bone marrow transplants to treat lymphoma from a donor with an uncommon genetic mutation (CCR5-delta-32) that makes T-cells resistant to HIV infection.

Given this proof-of-concept, researchers have studied other HIV positive people undergoing bone marrow transplants to treat cancer. Bone marrow contains stem cells that give rise to all immune cells, including the CD4 T-cells targeted by HIV. It is unclear whether Brown's apparent cure was due
to the donor's genetic mutation, the process of ablating or killing off the cancerous immune cells, a
graft-vs-host reaction in which transplanted immune cells attack the recipient's body, or some other
factors.

Kersten Koelsch and David Cooper from the University of New South Wales and colleagues
presented posters at AIDS 2014 and at the preceding International AIDS Society Towards a Cure
symposium describing 2 Australian men treated at St. Vincent's Hospital in Sydney who underwent
allogeneic (self-donated) hematopoietic stem cell transplants. Unlike Brown, these patients
received reduced-intensity conditioning prior to transplantation using milder chemotherapy that
enabled them to remain on ART throughout the process.

The first patient (age 47), who received a transplant due to non-Hodgkin lymphoma in 2010, was
CCR5-delta-32 heterozygous, meaning he had 1 copy of the protective mutation. The second (age
53), who underwent transplantation due to leukemia in 2011, had normal CCR5 genes. The first
patient experienced grade 2 (moderate) graft-vs-host disease and reactivation of cytomegalovirus
(CMV) infection, while the other had only a mild skin reaction.

Following bone marrow transplantation both men have no detectable HIV RNA in their blood
plasma and no detectable HIV DNA by PCR in peripheral blood mononuclear cells (PBMCs) or CD4 T-
cells according to sensitive tests. Neither shows CD4 T-cell responses to HIV antigens. One has only
traces of HIV antibodies, while the other has absent antibodies.

"Assessment of the HIV-1 reservoir size in these 2 patients after allogeneic [bone marrow
transplantation] with reduced-intensity conditioning shows undetectable HIV-1 RNA and DNA in
peripheral blood and absent CD4+ T cell responses to HIV-1 antigen," the researchers concluded.
"We also found a profound reduction in HIV-1 antibody detectability in both patients by Western
blot."

While these results appear promising, a similar pair of bone marrow transplant patients in Boston
experienced HIV relapse several months after stopping ART. Both men also received reduced-
intensity conditioning and stayed on antiretrovirals during transplantation. After extensive testing
over 3-4 years showed continued undetectable plasma HIV RNA, no HIV DNA in PBMCs, and no
evidence of virus in lymph nodes or gut tissue, the patients underwent carefully monitored analytic
treatment interruption to see if the virus would return. As Timothy Henrich from Brigham and
Women’s Hospitalreported at this year's Retrovirus Conference and in the July 22 Annals of Internal
Medicine, both men did eventually experience viral relapse, at 4 and 8 months after stopping ART.

While the Boston results were disappointing, the men were able to remain off ART for many weeks,
indicating that some aspect of the transplant procedure helped them control HIV longer than would
be expected without treatment. These late relapses despite undetectable HIV suggest that even a
tiny amount of residual virus in reservoirs is enough to rekindle infection, making researchers more
cautious about interrupting therapy in the Australian patients.
"This is another example of where the transplant can drive the amount of virus to levels that we simply cannot detect," Cooper told the Australian Associated Press. "But if we stopped the antiretroviral therapy, there would be a very strong chance that it would come back."

Yet each new case like this adds to the body of knowledge that may one day allow people with HIV to stay off ART for extended periods without disease progression.

"Cure research is looking for a way to move forward and my view is that this is a very important clue, that an immune response produced by bone marrow transplantation has such a strong anti-HIV effect," Cooper added. "We're going to use this as a model for cure research and see if we can develop some therapies that mimic what were doing with bone marrow transplantation."

8/28/14

AIDS 2014: Researchers discuss progress towards an HIV cure
Liz Highleyman

Progress along the multi-pronged path towards a cure for HIV was one of the themes at the 20th International AIDS Conference (AIDS 2014), taking place this week in Melbourne. Researchers provided updates on the "Mississippi Baby", a novel assay for detecting low levels of hidden virus in the body, and using the anti-cancer drug romidepsin to reactivate latent virus.

Researchers discussed cure-related work in oral abstract sessions and at various symposia, including a 2-day pre-conference "Towards an HIV Cure" meeting organised by the International AIDS Society (IAS). Several researchers summarised their findings and offered their thoughts about future directions at an IAS press briefing on Monday.

Deborah Persaud from Johns Hopkins gave an update on the "Mississippi Baby" – now nearly four years old – who just before the conference was found to still have HIV after having had an undetectable viral load while off antiretroviral therapy (ART) for more than two years.

Though disappointing for the child – who has resumed ART and is in good health – "we have learned a lot from this case and it provides a strong rationale for moving forward with a clinical trial" of very early combination therapy for infants, Persaud said.

"We're looking at the moment at achieving long-term remission, and how long can we go [without antiretrovirals]," said AIDS 2014 co-chair Sharon Lewin from Monash University. "We've realised in the past year that the virus can really hang around for a very long time and pop up unexpectedly."
The Mississippi case "shows we need much better tools to detect HIV" in the body and "need to learn how to eliminate long-lived reservoirs."

Nicolas Chomont from the Vaccine and Gene Therapy Institute in Florida reported on the development of a new assay called Tilda that – unlike current tests used in cure research – uses only a small amount of blood, is less expensive, and does not require specialised equipment. Tilda "can be implemented in pretty much any lab in the world," according to Chomont.

Dan Barouch from Beth Israel Deaconess Medical Center described a study in monkeys, published this week in Nature, showing that HIV seeds itself in cell and tissue reservoirs very soon after sexual exposure – even before viral load is detectable in the blood. Very early ART reduced the size of the reservoir, but it did not prevent re-emergence of virus after treatment was stopped. "Even very early is not early enough," Barouch said.

One strategy widely used in HIV cure research is dubbed "kick and kill", the idea being to reactivate latent virus in resting cells. Once the virus is "woken up" and starts replicating, it becomes visible to the immune system and is susceptible to antiretrovirals.

Ole Schmeltz Søgaard from Aarhus University Hospital in Denmark described his team's research using the HDAC inhibitor romidepsin to kick cells containing dormant HIV out of their resting stage. In a small study of six people with long-term viral suppression on ART, romidepsin was indeed able to activate latently infected cells, but "it doesn’t look like [this led to] a significant reduction in the viral reservoir," Søgaard explained.

Results such as these indicate that neither very early antiretroviral treatment nor agents that re-activate latent virus are likely to be enough to enable a functional cure, or prolonged time off ART without disease progression.

"The Mississippi and Boston [bone marrow transplant] cases make me wonder if we will ever get rid of the entire reservoir," said Steven Deeks from the University of California at San Francisco. "We may get rid of big chunk of it...but we need a way to control what's left." Deeks predicted that the cure field will move in the direction of therapeutic vaccines or other immune-based therapies that can be used in combination approaches.

"We should not oppose vaccine and cure research, and probably we will need both," predicted IAS President Françoise Barré-Sinoussi. Added Barouch, "It’s two sides of the same coin."

A proof of concept for an HIV cure exists. Now even our setbacks are useful

The Mississippi baby, in remission for over two years, recently experienced HIV rebound. This low – if it can be called that – shouldn't dampen our hopes for a cure.

François Barré-Sinoussi and Sharon Lewin
Monday 21 July 2014 01.40 BST

An H9 T cell, blue, infected with the human immunodeficiency virus (HIV), yellow. Photograph: AP

It was a sad and disappointing turn of events: the child known as the Mississippi baby, in remission of HIV infection for more than two years after discontinuation of antiretroviral therapy (ART), now has clear evidence of HIV rebound. The announcement will undoubtedly be viewed by some as a setback in the renewed quest for an HIV cure.

It doesn't need to be. We need to take the highs and lows in our stride. We have a proof of concept that a cure for HIV is possible in the Berlin Patient, Timothy Brown, who received a bone marrow transplant and remains virus free off treatment for over six years.

We also have the case of the French Visconti cohort, now comprising 20 HIV-positive people who were treated very early with HIV drugs, but then stopped treatment. After almost 10 years off therapy, they are keeping the virus under tight control.

Only one person, who is now in remission for a non Aids-related cancer, reinitiated ART despite his HIV infection still being controlled at the time that he received anti-cancer therapy.

The lows – if the Mississippi baby case can be called a low – provide major insights into the scientific challenges that lie ahead for the researchers, scientists and others gathering at the Aids 2014 symposium in Melbourne this week.
Similar to the Mississippi baby, the Boston patients—two men who received bone marrow transplants that appeared to rid them completely of HIV—also relapsed, and are now back on antiretroviral treatment.

What has been most encouraging for all three patients was that the time the virus stayed under control off treatment, or in remission, was significantly longer than what we have ever witnessed before. The Boston patients were virus-free for 12 and 32 weeks, and the Mississippi infant virus-free for a remarkable 27 months.

However, as we have learned over the past three decades, HIV has many tricks up its sleeve.

An minute amount of virus—which none of our current tests are even close to detecting—was able to persist in these three patients and reappear at any time, with absolutely no warning.

The Mississippi baby, and the Boston patients, tell us that to achieve long term HIV remission we will likely need to tackle the problem on multiple fronts—lowering as much as possible the number of long-lived, latently infected cells present in the body, as well as bolstering the host defence. One cannot be done without the other.

We've always known that the search for an HIV cure wasn’t going to be easy. We must be optimistic, and hope that cases like the Mississippi baby will prove to be another piece in the jigsaw puzzle that is an HIV cure.

To reach this goal will require substantial academic collaboration as well as public-private partnerships that can drive innovative and well-funded strategic research over the long term.

We must continue the search for an HIV cure. We owe it to the 35m people living with HIV.

http://www.theguardian.com/commentisfree/2014/jul/21/a-proof-of-concept-for-an-hiv-cure-exists-now-even-our-setbacks-are-useful

Sharon Lewin: guiding us towards a cure for HIV

Sharon Lewin is one of those rare people who seems to be doing it all, and doing so very well. Lewin is the Co-Chair of the 20th International AIDS Conference (AIDS 2014), to be held in Melbourne, on July 20—25, 2014, with International AIDS Society (IAS) President Françoise Barré-Sinoussi. Lewin is also the Head of the Department of Infectious Diseases at the Alfred Hospital and Monash University in Melbourne. Later this year, she will become the inaugural Director of the University of Melbourne’s new Peter Doherty Institute for Infection and Immunity. “Sharon reminds me of the old legends of people in medicine who can do it all. She is the complete package, an outstanding researcher, clinician, teacher, and administrator”, says her long-term collaborator Steven Deeks at the University California, San Francisco.

Unperturbed by being local chair of the largest scientific conference in the history of Australia, Lewin is committed to the research field for which she has become best known: a cure for HIV. “Sharon is a key player in the search for a cure, both in the basic biology of HIV latency and translation into clinical trials”, says Julian Elliott, Head of Clinical Research at Monash University’s Department of Infectious Diseases.

So how did the daughter of Jewish eastern European immigrants to Melbourne after World War 2 become a top researcher on an issue that only a few years ago was not a global priority? “I originally wanted to become an engineer or an astronaut, but submitted to family expectations and did medicine at Monash University”, she explains. A year in Kenya working in a mission hospital and infectious diseases training followed. “HIV medicine was a natural fit because of the interesting clinical medicine, the social issues of caring for young but marginalised populations, and the rapidly changing science”, she says.

After completing a PhD at Melbourne’s Burnet Institute in the mid-1990s, Lewin’s supervisors Suzanne Crowe and John Mills introduced her to David Ho at the Aaron Diamond AIDS Research Center in New York. Lewin recalls how “the centre was at its peak of scientific discovery. Ho had just been lauded for his work on new potent antiretrovirals. The mathematical model he and mathematician Alan Perelson developed initially predicted that only 3 years of antiretrovirals could cure HIV. But they were proved to be wrong with the discovery of long-lived latently infected cells.” So Lewin moved to the centre, with her husband and two young sons, and began postdoctoral research on HIV latency in 1997. Her passion for this research was cemented from that moment on. “Many of the big remaining scientific questions are the same ones we debated then. How to measure very low levels of virus on therapy, whether the virus is still replicating, how long it takes to rebound after stopping therapy, and the factors that determine that.”
In 1999, Lewin returned to Melbourne to continue one of the most important working relationships in her career—a 20-year collaboration with Paul Cameron, a clinical and basic immunologist with whom Lewin co-heads the Alfred Hospital laboratory. Together they have pioneered novel laboratory models of HIV latency that mimic patients' cells, which, Lewin says, “have been critical in identifying new approaches to eliminate latently infected cells. I love talking through new ideas with Paul. His knowledge of science is encyclopaedic.” She went on to become head of the clinical department at the Alfred Hospital in 2003, which she describes as “an environment very supportive of clinician scientists”. A sabbatical in 2008 at Pitié-Salpêtrière Hospital in Paris followed, where she was encouraged to move her laboratory findings on how to activate latent HIV into clinical trials. 2 years later, Lewin was invited to give the opening plenary at the 2010 IAS meeting in Vienna. “The organisers took a risk, firstly, because I was relatively unknown, and, secondly, because I was asked to give a scientific HIV cure talk to a hugely diverse audience.” Deeks recalls how her address “galvanised the HIV and general community towards a cure”.

It was at the conference that Lewin met Barré-Sinoussi, whom Lewin describes as “passionate about a cure, global health, and women in science”. Barré-Sinoussi also appreciates Lewin’s outlook. “I admire her way of thinking, but above all as a humanist—really listening to what people living with HIV are saying and then translating this to what can be the science we develop in order to respond to their needs.” Together with Deeks, the three played a prominent part in the development of the IAS global scientific strategy for a cure for HIV launched in 2012.

This strategy will be a key focus at AIDS 2014, but as Lewin notes, “a functional cure (suppression of virus) could be in sight, particularly in young babies treated early, but complete elimination of the virus is unlikely in the near future”. Barré-Sinoussi points to some of the challenges ahead: “the virus remains in a latent form in long-lived cells present in many parts of the body, the size of the reservoir is larger than we thought, and we still do not fully understand the mechanism of latency”. To advance research Lewin believes that “large collaborative research is the way forward”. And, as Lewin notes, “one of the reasons progress in HIV has been so rapid, is because of the meaningful engagement of the affected community. Community needs to be part of all aspects of public health policy, clinical care, and research—the partnership approach so successful in the HIV response, will hopefully shape the way we tackle many other diseases in the future.”

http://www.thelancet.com/journals/lancet/article/PIIS0140-6736%2814%2961198-3/fulltext
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Towards a cure for HIV – are we making progress?

Sharon R Lewin, Steven G Deeks, Françoise Barré-Sinoussi

Until recently, no-one dared to discuss a cure when it came to HIV. There had been a general consensus among experts that the best we would ever achieve would be durable control of the infection with antiretroviral therapy (ART). And, in this regard, we have been tremendously successful. ART is often given as only one tablet a day with minimal toxicity, leads to normal life expectancy, and reduces viral transmission. But therapy clearly has its limitations. It must be administered daily for life ...

http://www.thelancet.com/journals/lancet/article/PIIS0140-6736%2814%2961181-8/fulltext
AIDS2014: Ambition and Optimism - But Also a Serious Reality Check

Last week, the 20th International AIDS Conference took place in Melbourne, Australia. The rhetoric throughout was one of ambition: the conference theme was "stepping up the pace", a Melbourne Declaration called for "no-one left behind", and there was constant talk of ending AIDS by 2030. But, while such ambition is to be applauded, it does not tally with the current situation on the ground for people who use drugs.

The conference itself brought together more than 13,000 people from the global community of HIV advocates, doctors, academics, community representatives, activists and the media. It began in a somber mood - as delegates mourned the loss of colleagues and friends on board the ill-fated Malaysian Airlines flight shot down over Ukraine a few days before - but proceeded in their honour and remembrance.
Michel Sidibe, Executive Director of UNAIDS, opened the five day event by articulating his vision for an end to AIDS - and quoted Nelson Mandela: “After climbing a great hill, we find that there are many more hills to climb”. He referred to the 2030 vision as the “final climb” and called on everyone working to end the epidemic to act “with the same sense of urgency” to reach this difficult summit. He also set a new HIV treatment target for 2020: “90% of people tested, 90% of people living with HIV on treatment, and 90% of people on treatment with suppressed viral loads”.

Cue a new slogan: “90-90-90”. In terms of the science, this is achievable. The HIV response has made incredible advances, and nearly 14 million people now have access to treatment that can render the virus undetectable. HIV is no longer the death sentence it once was.

But HIV has never been about science alone - so what about the politics? Last week’s conference highlighted many stark and sobering warnings. HIV and AIDS cannot be beaten without addressing the realities faced by the most disadvantaged and stigmatised groups. These key populations - men who have sex with men, people who inject drugs, prisoners, sex workers and transgender people - have always borne a disproportionate burden in this epidemic. It is for them that the critical nexus of economic and social marginalisation, unjust laws, HIV risk and political ignorance is most pronounced.

Accordingly, there were a number of strong calls at the conference for drug law reform and the decriminalisation of people who use drugs:

- New reports and guidance from the World Health Organisation and UNAIDS explicitly called for drug law reforms to remove barriers to the HIV response for people who use drugs.
- The former Australian high court judge Michael Kirby presented the groundbreaking report from the Global Commission on HIV and the Law, which states that “Countries must reform their approach towards drug use. Rather than punishing people who use drugs who do no harm to others, they must offer them access to effective HIV and health services, including harm reduction and voluntary, evidence-based treatment for drug dependence”.

- The Global Commission on Drug Policy held a high-level panel, which included Sir Richard Branson and Michel Kazatchkine, the UN Envoy for HIV in Eastern Europe and Central Asia. Their message was unequivocal: governments have a moral imperative to stop punishing and criminalising people for using drugs.

At the same time, however, we heard about a looming global crisis in funding for services for people who use drugs - such as needle and syringe distribution and opiate substitution treatment. While UNAIDS estimates that at least $2.3 billion is needed to provide these essential HIV prevention measures, it seems that just 7% of this amount is actually being spent, just 8% of people who inject
drugs have access to opioid substitution therapy, and just 4% received anti-retroviral treatment. The majority of these people live in middle income countries, but donors - including the Global Fund to Fight AIDS, Tuberculosis and Malaria - are slowly turning their backs on these countries. Their expectation is that middle income governments will meet the costs of their own HIV responses - but we know that many governments have historically failed to adequately invest in services for marginalised populations. Yet we also know that these same governments spend vast sums on drug law enforcement - an estimated $100 billion is squandered each year on ineffective, abusive and repressive drug policies control measures. Just a fraction of this money - 10 percent by 2020 - would cover the global funding gap for proven, effective HIV and hepatitis services.

Huge challenges remain if we are to end AIDS. Last year alone, more than 2 million people were newly infected with HIV, and there were 1.5 million AIDS-related deaths. People who inject drugs are 22 times more likely to have HIV than the general population as a result of punitive drug laws, stigma and discrimination. Crucial efforts to advocate for people who inject drugs and to protect their human rights - referred to as "critical enablers" by the UN - account for less than 1% of all HIV spending. Without addressing structural legal and policy barriers there will never be an end to AIDS and, as we embark on the next stage of this journey - towards the 90-90-90 target - people who inject drugs will be left behind.

There were, of course, plenty of positive stories showcased at the AIDS conference too: a project in Kenya where police champions are working with people who use drugs and sex workers to stop police abuse and educate their law enforcement peers about harm reduction; PKNI - the Indonesian network of people who use drugs - deservedly winning the Red Ribbon Award, and being commended by the Indonesian Health Minister for their activism. However, these important activities fall into the category of the fore-mentioned "critical enablers" and are at risk in the current funding environment.

So, for me, the take home messages from Melbourne are that the science is crucial but it will not be enough by itself, and that the ambitious new targets for the HIV movement are just empty rhetoric
unless more is done for key populations. Despite all the climbing that we have done over the last 30 years, we are still at the foot of a huge mountain - the urgent need to reform bad drug laws and policies that undermine the HIV response. Turning this around requires greater international leadership, political commitment and will, tireless health and human rights advocacy, and a scale-up (rather than down) in donor support for the countries most in need. Only then can we begin to seriously talk about a world without AIDS.

http://www.huffingtonpost.co.uk/ann-fordham/aids-2014_b_5638218.html

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