



## 12 key areas of research: non-technical summaries of 44 studies

This supplement is a non-technical review of the 17th Conference on Retroviruses and OIs. Technical reports from the conference are published in HIV Treatment Bulletin, available online and to order in print:  
<http://www.i-Base.info>

Hyperlinks in this PDF file link directly to each abstract or session.

All abstracts and webcasts can be seen online:

<http://www.retroconference.org>

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## 1. Introduction: what is CROI?

CROI stands for Conference on Retroviruses and Opportunistic Infections. It is usually just abbreviated to either 'CROI' or 'the retrovirus meeting'.

This conference is probably the most important annual scientific HIV meeting and is held in different cities in the US or Canada.

With over 1000 studies presented, we can only cover a few of the most important themes. Automatic links in the PDF version and web addresses in the print version take you to the original studies online.

Most of the presentations are available to watch free online without registration. This includes opening lectures, oral presentations and poster discussion. Summaries of all the studies, called abstracts, and PDF files of the more detailed posters are also available at the conference website:

<http://www.retroconference.org>

Although some talks are technical, other overviews are easier to follow. Watching the webcasts is a great way to learn. The questions from the audience are also included on the webcast.

While the presentations can be technical, the implications of this research can be described more easily, so we've selected a few of the most important studies for this review.

## 2. HIV treatment reduces transmission: a new theme that is here to stay

One of the most significant new themes was the prominence given to the role that HIV treatment is already playing in reducing HIV transmission.

At its most simple, this means that people on treatment are less infectious. New studies showed this, even when people are already taking care to have safer sex.

This is important for HIV-positive people, whether on treatment or not. It is also relevant both to people in Western countries and resource-limited settings, because it shows additional financial and population health benefits from wider access to treatment.

On an individual level, viral load is probably the most significant single risk factor for any type of transmission. Whether this is from sex, through shared injecting equipment, at birth, from breastfeeding or from needlestick injuries, the higher the viral load the higher the risk. Even though transmission can still take place at very low levels, this means that the lower the viral load, the lower the risk.

Four studies - two from Southern Africa and two from San Francisco - presented new information on this reduced risk. Many others supported similar conclusions, linked also to increased testing.

In a pre-conference symposium on Tuesday, Brian Williams presented a lecture based on theoretical modelling. This is a great talk and very easy to follow on the webcast. He showed that providing treatment to every HIV-positive person in South Africa with a CD4 count below 200, could stop new transmissions within 5-10 years. [1]

This would cost no more than what will currently be spent, but would save at least 3 million lives. Using higher CD4 counts to start, for example at 350, 500 or on diagnosis, also had similar costs.

Whether transmissions are already reducing was shown in a presentation from the Partners Study. [2]

This study, run in seven Southern African countries, included 3381 couples where one partner was HIV-positive and one was HIV-negative. This study was originally designed to see whether acyclovir, a treatment for herpes, could reduce HIV transmission.

Although acyclovir did not reduce HIV infections, about 350 HIV-positive people during the study needed to start HIV treatment for their own health. The rate of new HIV infections was 92% lower in the partners of people using HIV drugs compared to those who were not on treatment. Of the 103 infections between partners, only one occurred from someone who was using treatment. The single transmission also occurred very soon after ARV treatment was started, when viral load was still likely to be very high.

These clear results bring a powerful message: ARV treatment in this context had a greater protective effect than almost any other prevention method.

It is certainly higher than any expectation for a vaccine if one is ever produced.

The San Francisco studies also included a theoretical model and some real life results, this time mainly in gay men. The modelling studies calculated estimates for 'community viral load'. This is an average level of viral load based on actual results from newly diagnosed people and those already on treatment. [3]

Each year, from 2004 to 2008, the levels of community viral load fell steadily. Over the same time, HIV education and testing was expanded. Despite more testing, the numbers of new infections fell steadily from about 800 in 2004 to just over 400 in 2008. This was not related to condom use because other STIs increased - showing that many men were choosing partners with the same HIV status (serosorting) so as not to use condoms.

Another related modelling study for San Francisco showed that increasing testing and earlier treatment would reduce new infections even further. [4]

The effectiveness of the current HIV testing and treatment programme was an important aspect of the San Francisco studies. Half of the gay men in the city had an HIV test every six months and 70% tested at least once a year. Only 15% of HIV-positive men were not aware of their HIV status (the rates are between 30-50% for gay men in the UK). Around 90% of HIV-positive men were on treatment, and 70% of those had viral load less than 50 copies/mL.

These are important points. In the UK we need to continue to break down stigma around talking about HIV, and testing for HIV. The virus is just a virus. No testing leads to poorer health, and more transmissions. Testing leads to the chance to live longer and healthier, and to have the knowledge to then protect your partners.

Many other studies reported on related aspects of 'test and treat' programmes in different countries and different target populations. This is a theme that will only get stronger, given the difficulties of other methods of prevention.

One important caution is that no-one is claiming that treatment in itself will stop HIV – however convincing the modelling. But the impact that treatment is already having on reducing infections is too dramatic and too important, not to be recognised as a prevention tool in itself.

Prominent experts often default to a nasty phrase saying ‘we can’t treat our way out of the epidemic’. They justify this by looking at current statistics and say ‘for every person on treatment, three new people get infected’. This combines two separate current statistics and suggests they are linked.

Instead we should hear that ‘for every person on treatment, ten people are protected’. Then, if treatment programmes expand, the ratio to new infections will reduce, and ARVs themselves will directly reduce infections.

When talking about treatment for everyone, this does not imply forcing anyone to take ARVs. The risks and benefits are different for every individual. The right to decide how and when to start treatment will always be an individual choice.

### 3. ARVs before and after exposure: PrEP, iPrEP and PEP

PEP = post-exposure prophylaxis (taking HIV drugs after exposure)

PrEP = pre-exposure prophylaxis (taking HIV drugs before exposure)

iPrEP = intermittent PrEP (taking HIV drugs as needed, rather than daily)

At least ten years ago studies showed that tenofovir, if taken beforehand (as PrEP), could protect animals from multiple exposures to SIV (the monkey form of HIV).

Eventually studies were started in humans, using a variety of doses and schedules. The main focus has been on using a daily dose of tenofovir, with or without FTC. These studies are in theory testing both PrEP and PEP together. However, unless a daily treatment is needed (because of daily exposure risk) it would be much easier, safer and less expensive if these drugs did not have to be taken every day, only when you knew you might be at risk.

A lecture at CROI covered different aspects of this research. [5]

Conclusions included that:

- tenofovir + FTC (Truvada) provided protection for monkeys after rectal exposure (as well as vaginal)
- both drugs in Truvada are playing an active role (ie both tenofovir and FTC)
- tenofovir takes about 24 hours to reach good concentrations in genital tissues, so needs to be taken a day before any potential exposure. However, once it reaches good levels, a single dose gives protection for at least three days.
- taking Truvada two hours after exposure as well as the pre-exposure dose is also essential.

These results are encouraging, but these are all animal studies. Although human studies are ongoing, they have yet to report results before we know if this provides protection in people.

### 4. Microbicides - gels to protect against infection

Several presentations looked at using HIV drugs in a gel called a microbicide to prevent infections.

Microbicides in HIV research refers to a gel or cream that could be applied to reduce the risk of HIV infection. There are many situations where one partner is unable to negotiate using condoms, and microbicides could protect someone without their partner needing to have any involvement.

In December, a large international trial reported no benefit from a gel called PRO 2000. These results were also presented at CROI, and no matter how the data was analysed no protection was seen. [6]

Two studies using currently approved HIV drugs in gels were more promising. In monkey studies a tenofovir gel had a longer period of protection [7] than one made from maraviroc [8], but both worked.

Again, this is early research but a conservative cost analysis for the maraviroc product suggested that even at the current (ie undiscounted) price of \$15, one pill could provide protect against 25 exposures.

These studies were only for vaginal application. Although the demand is just as important for protection during anal sex, the science (and mechanics) for this are significantly more difficult to overcome.

### 5. New drugs and formulations

This year there were relatively few studies on new drugs. This difference was noticeable compared to previous conferences and a few people have commented that drug development has ended. In practice though, it probably just reflects the recent wave of recently approved drugs.

The effectiveness of current meds certainly make it more difficult to find new and better ones. But the demand for HIV drugs is increasing each year. Also, the process for indentifying potential compounds is faster and more sophisticated. Computer modelling has revolutionised drug development, and makes it possible to screen hundreds of thousands of potential molecules without having to physically try them in test-tube studies.

At the conference many of the likely next compounds were presented in one session. [9]

#### QUAD – a new 4-in-1 once daily option

The QUAD pill, is a formulation of four drugs in one pill. Two of these drugs are already approved (tenofovir and FTC; Truvada). The third is a new integrase inhibitor called elvitegravir. The fourth drug is a booster for elvitegravir. The booster is cobicistat (previously called GS-9350) and is very similar to ritonavir, the only current option for boosting. Unlike ritonavir, cobicistat has no direct anti-viral activity.

Two early (Phase 2) studies on these new drugs were presented in one lecture. [10]

First, when the QUAD pill was compared to Atripla (efavirenz + tenofovir + FTC), over 80% of each group had undetectable viral load (less than 50 copies/mL) after 24 weeks. For the QUAD group, people became undetectable much quicker, as was seen

in studies another integrase inhibitor (raltegravir). For example, after 8 weeks, about 80% people were undetectable with QUAD compared to about 50% with Atripla. QUAD was better tolerated, mainly from not having efavirenz-related side effects.

While this is promising for a new one-pill combination that could be an alternative to Atripla, there were also concerns about the impact of the booster on markers of kidney function (see below).

### **Cobicistat (GS 9350) – an alternative booster to ritonavir**

The second study compared the new booster to ritonavir, each in combination with atazanavir, tenofovir and FTC.

One caution is that cobicistat had an impact on markers of renal function. Although this was apparently causing damage it would complicate interpreting results when tenofovir is being used in the same combination. Another is that cobicistat seemed to be very close to ritonavir in terms of side effects.

Two hopes for a new booster are that it might have fewer side effects and be less expensive than ritonavir.

Currently, it looks like it will mainly allow QUAD to be developed in a single pill, without having to pay royalties for using ritonavir. Until now, co-formulation of ritonavir has not occurred with any other drugs, other than Kaletra.

In summary though, results were encouraging enough for both QUAD and cobicistat to be taken into larger studies. These next trials, will include hundreds of people (compared to the 70 or so in the above studies), so will give us much more information.

### **Compounds in earlier development**

A dose-finding study of a CCR5-inhibitor called TBR-652 reduced viral load by more than 30-fold reductions (1.5 log) after 10 days monotherapy. This was in treatment-experienced people. No dose-related or serious side effects were reported, though the study was only in about 50 people. This looks like a once-daily compound with the potential for fewer drug interactions. [11]

Very little new information was available for a new integrase inhibitor called S/GSK1349572. [12]

Although given an oral presentation the results from a 10 day monotherapy trial were presented last year. The promise was shown by 300-fold reductions in viral load (2.5 log) using a 50mg dose over this short period.

Preliminary studies looked at a handful of other targets and approaches, including attempts to target sleeping cells.

Potential compounds from a new class of integrase inhibitor, called LEDGINS were identified by computer screening of over 200,000 molecules. They will not be cross-resistant to raltegravir or elvitegravir. [13]

Capsid inhibitors work in a new way to interrupt the process for assembling new HIV. These molecules were shown to have activity against HIV that is resistant to other drugs. [14]

### **Expanding goals of treatment**

Many presenters repeated the fact that eradication with current drugs is not possible. This is mainly because they only work on active cells and not on resting cells where HIV is also sleeping. Even with an undetectable viral load after many years treatment, stopping treatment causes viral load to return from sleeping cells to over 10,000 copies/mL within a week.

At least five new types of treatment are the focus of research on how to chase HIV out of target cells.

Additional new targets for treatment that were presented, included cellular restriction factors – human proteins that reduce HIV replication and that can help or block infections. These include:

- tetherin, a protein that blocks HIV release
- APOBEC3, an immunity gene that has anti-HIV activity, and
- TRIM5-alpha, a protein that in some monkeys protects against HIV infection, and that gene therapy could perhaps be modified to adapt the related human protein. [15]

Zinc finger molecules that can knock out CCR5 and block HIV entry were also discussed. Though this has been a focus for research for at least 15 years.

### **Vicriviroc: uncertain future**

For people who have followed new drugs studies for the last few years, the CCR5 inhibitor vicriviroc did not show any benefit compared to a placebo (inactive dummy pill) in the unfortunately named Victor-E studies. [16]

This may be a combination of effective background drugs, or that people in the vicriviroc arms had fewer active drugs. Despite early promise, it looks likely that this compound will go back to sit on the shelf.

## **6. Studies with current drugs**

As with new drugs, there were also fewer new studies at CROI looking at differences between existing drugs. A couple of these studies though were important.

### **Atazanavir vs efavirenz: ACTG 5202**

This large US study enrolled nearly 2000 patients to look at differences between four widely used combinations. The main conclusion was that efavirenz and atazanavir/r are similar enough it to be likely that atazanavir/r will be included as a first choice option in the next guidelines update. [17]

The study also compared tenofovir/FTC to abacavir/3TC. However, as the test for abacavir sensitivity was not used, the results from that part of the study are less relevant. [17]

Another study from ACTG 5202 looked in detail at metabolic changes, including lipodystrophy, in a smaller group of people from the main study. In many ways these results were more interesting, and there were small lipids differences between different components. [18]

However, the big question about whether each element has a different impact on fat accumulation and lipodystrophy was not really answered. This is because the DEXA scans only show changes in proportion of body fat, but not whether this was central (visceral) fat (inside the abdomen, around internal organs) or fat just under the skin ('love handles'). The former is more strongly linked to HIV-related lipodystrophy, and more difficult to treat. The latter is a 'return to health effect' and calls for diet and exercise...

Bone health in general is also discussed below but as DEXA scans also measure bone mineral density, these results were also shown. Tenofovir + FTC led to a larger decline in lumbar spine and hip bone mineral density compared to abacavir + 3TC. Atazanavir/r led to more loss in lumbar spine but not hip density, than efavirenz.

Although both findings were statistically important, in the short term, it is unclear how important they are in relation to risk of further bone problems.

### Once-daily darunavir/r in experienced patients

Darunavir/r is a protease inhibitor that is used as a once-daily drug for people on their first therapy, but twice-daily for people with drug resistance. The ODIN study compared once- to twice-daily to see whether once-daily darunavir/r could be used in second-line therapy. [19]

Although around 70% of people in both groups had undetectable viral load after a year, the study results do not match a common circumstance for many patients. This is because most people in the study did not have resistance to protease inhibitors and these are the people who would be most at risk from reducing the dose.

Almost half the participants had never used a PI, most had no primary PI mutations, and 85% were still sensitive to eight PIs. Around 70% also had two or more active drugs to use.

Finally, although most people had a low viral load around 2000 when they enrolled, 25% had levels over 50,000 copies/mL. This is very unusual for patients in the UK, which makes interpreting the results more complicated. Both groups showed similar activity when starting with viral load below and above 50,000 (around 77% vs 54% getting undetectable respectively) irrespective of the dosing.

If you haven't taken PIs before, this study shows once-daily darunavir is probably fine, but it did not give very much specific information for people who have a history of PI resistance. Hopefully future presentations will analyse the importance of different mutations and other differences within the study groups.

## 7. Below and above 50 copies/mL – intensification, blips and rebounds

Treatment works incredibly well for most people who get viral load to <50 copies/mL. Once viral load is this low, only 5% of people might rebound each year. But could it be better? And what is the significance of a blip?

Several groups have already reported exciting findings at low viral load levels.

Firstly, although most people are probably still detectable on a test that is sensitive to 1 copy/mL, about 50% of people probably have less than 5 copies/mL. Secondly, the virus at these low levels is exactly the same virus that a person had before treatment. Even after many years, it has not evolved or mutated. This has led researchers to conclude that it comes from sleeping cells as they wake, rather than from a compartment that the drugs do not reach. [20]

Intensifying treatment in people with <5 copies/mL has no effect. Whether this is from adding efavirenz, lopinavir/r, maraviroc, T-20 or raltegravir. Studies at CROI also seem to show this is not just in blood, but other compartments like genital fluids and in the CSF (the fluid around the brain). [21]

The exciting conclusion is that current drugs can't get any more potent. They could be made to work quicker perhaps (like integrase inhibitors), the formulations could be improved, and the side-effects reduced, but they don't need to be more potent.

Further research is needed to understand what happens at levels from 5 - 50 copies/mL. Given that resistance only rarely develops at levels below 50 copies/mL, whatever is found, is unlikely to change what happens at the clinic.

However, some of the blips studies were very useful from this perspective.

One study monitored people for two years and categorised them by how long they were below 50 copies/mL, and if they blipped, how frequently and high this went. [22]

Anyone who only had an occasional blip (<25% of time, to less than 1000 copies/mL) had the same risk of serious outcomes as someone who had undetectable viral load. This means if you occasionally blip, don't worry and don't change.

The more often and higher blips/rebound groups were all linked to higher risks or events. This suggests an important role for treatment modification to reach and stay <50 copies/mL.

## 8. HIV and heart disease

With new drugs having a lower profile at the conference, long-term complications and how they are to be monitored and managed came to the fore as another major theme.

We know that treatment works short-term, but by living longer, we will have to tackle all the complications of aging, and many studies suggested that HIV may make this more complicated.

Heart disease (cardiovascular disease, CVD) has been a major concern for at least ten years. The most optimistic news is that treatment as a whole is protective of heart disease. However, as treatment also increases some CVD risk factors like cholesterol levels, how to reduce risk has focussed on the benefits of the same lifestyle changes recommended for the general population: diet, exercise, stopping smoking, and lipid lowering drugs if needed.

At CROI, the large D:A:D study reported that HIV-positive people who stop smoking have the same effect in reducing their risk of heart disease as HIV-negative people. Even more impressively, this was found to occur as quickly as 2-3 years after quitting. [23]

This was important to see in a study. Some aspects of monitoring cardiovascular health were more complicated.

Another analysis from the D:A:D database (which contains over 33,000 patient records, followed for up to seven years) reported that triglyceride levels (TG) are independently associated with heart disease. If your TG is high, most clinics will try to reduce this by changing lifestyle and/or ARVs, or by adding a new treatment to reduce TG levels. Until now it was unclear whether TG were an important independent risk factor.

D:A:D reported, for the first time, that triglycerides are such a risk, after adjusting for other cardiovascular risks, even if the risk was relatively small (only increasing by about 10% a year).

Other studies found that HIV is likely to be a separate risk factor for lung cancer [24] and that this is one of several non-HIV-related cancers (including anal, oral/pharynx and Hodgkins Disease) where HIV-positive people are at higher risk than HIV-negative people. [25]

Together they strengthen the case for stopping smoking and for funding smoking cessation programmes.

Another group of studies looked at measuring the thickness of the wall of a major artery in your neck (the intima media thickness (IMT) of the carotid artery). Many studies in the general population have found that increases in IMT increase the risk of stroke or heart disease. Although interpreting scan results are complicated, as a monitoring test IMT measuring has the advantage of being non invasive.

Several groups reported that IMT increases may be greater in HIV-positive people than the general population. [26] However, the differences between studies show that there is not yet agreement for how to use this yet in routine HIV care.

A webcast of five of these studies followed by a discussion is also online. [27]

## 9. Bone health

Every year, bone health is the focus of many studies. The interaction between HIV and its complications, bone health and HIV drugs is complicated and difficult to entangle. As bone health decreases with age, this is important now that HIV-positive people are living longer.

As background, it is well reported that HIV-positive people have much higher rates of osteopenia (asymptomatic reduced bone mineral density/BMD) and osteoporosis (even more reduced BMD with increased risk of fractures).

Firstly, although reduced BMD has been reported in many studies, this hasn't been linked to an actual increased risk of fractures. At CROI, several groups including that large HOPS database reported higher rates in HIV-positive people of fractures or breaks. [28]

The finding wasn't consistent in all studies, but risk factors included older age, lowest ever CD4 count and other health complications (including IV drug use, hepatitis C and diabetes). Fractures were four-times higher than the general population and occurring younger than in HIV-negative people.

Similar findings were reported in the US Veterans study which is a mainly male group [29], but not in a study of younger women. [30] Given that low weight and the menopause are associated with bone problems, this is perhaps not the group where problems would be expected yet.

Another session related to bone health focussed on Vitamin D. [31]

The webcast for this session included an excellent introduction by Peter Reiss that raised more questions than answers. While HIV-positive people have low levels of vitamin D, there are discussions about what targets to aim for with supplementation. This is more difficult because studies don't always show a benefit from raising levels.

An interpretation of these studies from an advocacy perspective is that Vitamin D will be increasingly measured in HIV clinics. Supplements are likely to be used because low vitamin D is associated with reduced bone strength. As bone health gets a greater focus, more HIV clinics will also use DEXA scans as part of routine monitoring.

This may be particularly important for African patients in the UK as lower sun (sunlight makes vitamin D naturally) and very low vitamin D levels, may reduce bone density.

## 10. HIV and the brain

Because last year at CROI a whole session focussed on HIV and the brain, there was a lot of hope that this year we might get more answers. Many studies last year indicated that HIV may reduce brain function, but that this may not be at a level that interferes with daily life.

While this can be good news now, we really need to know how this will develop over many years as we age. The impact of aging is a repeated theme related to many complications. [32]

These studies looked at people at one time and compared them to expected results in the general population, but we really need results from studies that follow individuals over time to see how changes progress. Although these studies are underway, this evidence was not presented this year.

A poster discussion session included a review of seven studies. [33]

The Charter group are running a long term study looking at how well different drugs get into the brain, and generally report that reducing viral load in the brain (or specifically the fluid around the brain called the CSF) is a good thing. They also report that getting undetectable viral load in the CSF is related to taking drugs that cross the blood-brain barrier. [34]

A more cautious study indicated that drug penetration may be a delicate balance, because a rat study showed that ARVs themselves may have an impact on reducing brain cells (neurons). [35]

In the panel discussion, most doctors supported the benefit of including one or more drugs that reduce viral load in the brain. This is especially true for anyone with neurological symptoms.

## 11. Coinfections: TB or hepatitis

Perhaps most important TB study was from a trial in Botswana, where TB prevalence is high. This was a randomised double-blind placebo controlled trial involving 2,000 adults. [36]

They reported that 36 months of isoniazid prophylaxis treatment (IPT) was much more effective than 6 months. Benefits from 6-month IPT lasted for about six months. IPT for 36 months reduced new TB by 50% in people who tested positive for exposure to TB using the TB skin test (TST). TST-positive patients are broadly people with latent TB. However, there was no benefit of IPT for patients who were TST-negative. Importantly, HIV treatment for a year reduced the risk of TB activation by 50% in TST-negative people though the ARV impact was less dramatic in people who were TST-positive.

This is important for any high TB risk setting, which is usually defined by geographical region, but may relate to personal circumstances for some people in low risk countries.

New hepatitis C studies covered infection, treatment, genetics and management, many included in the oral webcast. [37]

- HCV remained infectious in different sized syringes tested at different temperatures. After one week, around 96%, 70% and 50% of syringes contained infectious HCV at 4°C, 25°C and 37°C, respectively.

In some syringes infectious HCV could be recovered after 2 months. In the smallest syringes (used for insulin), after one

day HCV could only be recovered from the samples that were stored at 4°C. [38]

- In acute HCV, if HCV viral load did not drop by 100-fold (2 logs) in the first 4 weeks after infection, it is unlikely to clear without treatment. [39]
- Genetic testing could perhaps be used to predict who will respond to treatment. Differences at one point of the IL28B gene (defined as having CC rather than TC or TT at one junction) showed different responses to treatment. With genotype-1, the rates were 50% achieving treatment response (with CC) vs 17% (with TC or TT). [40]

These genetic differences vary by race: only 25% of African-Americans have CC compared to 65% of European-Americans. This could explain why African American people have a poorer response to HCV treatment in most studies.

- Importance for people living with haemophilia of earlier referral for liver transplantation. [41]
- Acute (early) HCV infection is likely to produce worse FibroScan results as the inflammation makes the liver stiffer and less elastic. [42]

Unfortunately CROI had no results on new drugs for HCV in people with HIV... but these drugs are coming. Some of the most encouraging results from new classes of HCV treatment indicate much higher success rates for people with the difficult to treat genotype-1 and that future treatment may possible be much shorter.

## 12. Any news of a cure?

Finally, a good way to get your study reported by community publications is probably to show that HIV-positive people are going to live for longer. In a Dutch study, this is closing the gap even further between HIV-positive and HIV-negative people.

In this model, if you are diagnosed with HIV at 25 years old, you could expect to live an additional 52 years if you are a man and 57 years if you are a woman. This is well in to your seventies, and only a few years less than the general population. When the model looked at a 55 year old, similar close results to HIV-negative people were calculated.

Increasing research on complications of aging may unfortunately result in these very optimistic results being moderated in the future. However, even remembering that these are only average figures, this is all good news. [43]

Unfortunately, no cure was reported at the meeting – if it had been you would have heard about it by now and I'd be happy to be out of a job.

However, the lecture by Tony Fauci, who heads HIV research in the US was noted for putting 'the cure' back on the agenda as a goal that should never be forgotten. [44]

## References

Webcasts are available for all oral abstracts. Enter the abstract number into the search box and search by 'paper #'.

<http://www.retroconference.org/AbstractSearch/>

Webcasts are organised by day and time at this link:

[http://www.retroconference.org/2010/data/files/webcast\\_2010.htm](http://www.retroconference.org/2010/data/files/webcast_2010.htm)

### Treatment reduces transmission

1. Brian Williams - treatment to stop transmission.

Webcast: Guiding the global response. Tuesday 2.30pm.

2. Partner study. Deborah Donnell. Oral abstract 136.
3. San Francisco community viral load. Oral abstract 33.
4. San Francisco modelling study. Poster abstract 996.

### PrEP, PEP and iPrEP

Webcast: Antiretroviral-based prevention. Thursday 9.30am.

5. Tenofovir and FTC. Oral abstract 83.

### Microbicides – gels to prevent against infection

Webcast: Antiretroviral-based prevention. Thursday 9.30am.

6. PRO 2000. Oral abstract 87LB.
7. Tenofovir gel. Poster abstract 949.
8. Maraviroc gel. Oral abstract 84LB.

### New drugs and formulations

9. Webcast: Advances in ART. Wednesday 9.30am.

10. QUAD, elvitegravir and cobicistat. Oral abstract 58LB.
11. CCR5-inhibitor: TBR-652. Oral abstract 53.
12. New integrase inhibitor: S/GSK-572. Oral abstract 55.
13. LEDGF/p75 inhibitors. Oral abstract 49.
14. Capsid inhibitors. Oral abstract 50.
15. Targeting sleeping cells. Pre-meeting plenary.

Webcast: The HIV/AIDS Research Agenda. Tuesday 5.30pm.

16. Vicriviroc. Oral abstract 54LB.

### Studies with current drugs

Webcast: Advances with ART. Wednesday 9.30am.

17. ACTG 5202. Main study. Oral abstract 59LB.
  18. ACTG 5202. Metabolic substudy. Oral abstract 106LB.
- Webcast: Complications of HIV and ART. Thursday 9.30am.
19. Darunavir Odin study. Oral abstract 57.

### Intensification, blips and rebounds

20a/b. Low viral load background. See HTB Aug08 and Oct09.

21. No viral load change in the CSF from raltegravir intensification. Poster abstract 286.

22. Detectable viral load blips and risk of failure. Poster 504.

### HIV and heart disease

23. D:A:D smoking. Oral abstract 124.
24. HIV linked to lung cancer. Oral abstract 30.

25. Higher risks of lung cancer. [Oral abs 27](#) and [poster 757](#).  
 26. Intima media thickness study. [Oral abstract 125](#).  
 27. Intima media thickness discussion. [Poster abstracts 702-706](#).  
[Webcast: Predictors of cardiovascular disease. Wednesday 2pm.](#)

#### Bone health

28. Increased fractures in HOPS study. [Oral abstract 128](#).  
 29. US Veterans and bone fractures. [Oral abstract 129](#).  
 30. Younger women not increasing fractures. [Oral abstract 130](#).  
 31. Vitamin D overview and posters.  
[Webcast: Vitamin D deficiency. Wednesday 1pm.](#)

#### HIV and the brain

32. See i-Base HIV Treatment Bulletin Apr 2009. HIV and the brain.  
<http://i-base.info/htb/1525>  
 33. [Webcast: NeuroAIDS treatment issues and controversies. Wednesday 1pm.](#)  
 34. CSF viral load and brain penetration. [Oral poster 172](#).  
 35. Brain penetration and toxicity. [Poster abstract 435](#).

#### Coinfections: TB and hepatitis

36. Isoniazid prophylaxis for TB. [Oral abstract 104LB](#).  
[Webcast: TB prevention and treatment. Thursday 9.30am.](#)  
 37. [Hepatitis C webcast, Transmission, Outcomes, and Treatment: Friday, 9:30 am.](#)  
 38. HepC infectivity in syringes. [Oral abstract 168](#).  
 39. HCV viral load in early infection. [Poster abstract 639](#).  
 40. HCV and genetics: IL-28B. [Oral abstract 163](#).  
 41. Earlier referral for liver transplantation. [Poster abstract 688](#).  
 42. FibroScan results in acute HCV. [Poster abstract 642](#).

#### Any news of a cure?

43. Extended life expectancy. [Poster abstract 526](#).  
 44. Tony Fauci lecture. Pre-meeting plenary.  
[Webcast: The HIV/AIDS Research Agenda. Tuesday 5.30pm.](#)

**For more information about any of these studies,  
 call the i-Base information phoneline.  
 0808 800 6013  
 Mon, Tues, Wed 12.00 noon – 4pm  
 All calls are free and in confidence.**

### HIV i-BASE

HIV i-Base is an HIV-positive led treatment information service. We produce information both for clinicians and other health workers and for people with HIV.

All i-Base publications are available online. The website is designed to be fast to access, easy to use, and simple to navigate.

<http://www.i-base.info>

i-Base produce five non-technical treatment guides:

- Introduction to combination therapy
- A guide to changing treatment
- Avoiding & managing side effects
- HIV, pregnancy & women's health
- Hepatitis C for People living with HIV

The site also includes a web-based Q&A section for people to ask questions about treatment:

<http://www.i-base.info/questions>

Recent questions include:

- Doubts when I'm told that my life expectancy is good...
- Does treatment work if you start with a low CD4 count?
- Can hepatitis B reactivate?
- Does yohimbe interact with HIV meds?
- Should I start treatment at CD4 320?
- How do I time my meds when travelling?
- Is a viral load result of 50 really a blip?
- Does skipping a dose have an immediate effect?

#### Glossary

<b>ART</b>	antiretroviral therapy
<b>ARVs</b>	antiretrovirals (HIV drugs)
<b>BMD</b>	bone mineral density
<b>CROI</b>	Conference on Retroviruses and Opportunistic Infections
<b>CCR5</b>	a type of HIV drug
<b>CSF</b>	cerebrospinal fluid
<b>CVD</b>	cardiovascular diseases
<b>D:A:D</b>	Data collection of Adverse effects of anti-HIV Drugs study
<b>DEXA</b>	non-invasive scan for distribution of fat and density of bone
<b>HOPS</b>	HIV Outpatient Study
<b>HCV</b>	hepatitis C virus

<b>HTB</b>	HIV Treatment Bulletin (i-Base publication)
<b>IMT</b>	intima media thickness
<b>IPT</b>	isoniazid prophylaxis treatment
<b>iPrEP</b>	intermittent PrEP
<b>PEP</b>	Post-Exposure Prophylaxis
<b>PrEP</b>	Pre-Exposure Prophylaxis
<b>SIV</b>	Simian Immunodeficiency Virus
<b>PI</b>	protease inhibitor
<b>TB</b>	tuberculosis
<b>TG</b>	triglyceride
<b>TST</b>	TB skin test