

TREATMENT UPDATE

November - December, 2006

Welcome to the 14th Queensland Positive People (QPP) Treatment Update Newsletter!

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*The information, comments and editing in this newsletter do not necessarily represent the views of those involved in direct medical care...
...Always seek the opinion of your doctor.*



Making Sense of Antisense: HIV Gene Therapy Promising

A new type of gene therapy, based on a genetically modified version of HIV, has been successfully used in humans for the first time with "encouraging" results, according to research published this week in the online edition of the Proceedings of the National Academy of Sciences. This small proof-of-concept study suggests that gene therapy utilising genetically modified HIV as a viral vector, could be used to fight not just HIV, but other diseases as well, and "hint[s] at something more," says lead investigator, Dr Carl June, of the University of Pennsylvania School of Medicine.



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The aim of [gene therapy](#) is to modify the genetic material of living cells for therapeutic purposes. Most current research is investigating the potential for gene therapy in treating inherited genetic disorders, and researchers hope that it may be possible to correct genetic abnormalities that can cause diseases previously regarded as untreatable.

Although HIV infection is not an inherited genetic disorder, it could be viewed as an acquired genetic disorder since it inserts its own genetic material into human DNA. Consequently, gene therapy can be used to target the HIV genes in infected cells in a similar manner to the way in which they target 'faulty' human genes in inherited genetic disorders.

Researchers at the University of Pennsylvania School of Medicine used a cell culture system that allows for an individual's own CD4 T cells to be taken from their body and expanded. They then combined these CD4 cells with a modified version of HIV, called VRX496 – which contains an **antisense sequence** that prevents HIV from being able to make copies of itself by preventing translation of the full-length HIV envelope gene – and the modified CD4 cells were then reinfused back into the individuals.

Lead author, Dr Bruce Levine, describes VRX496 as "a lab-modified HIV that has been disabled to allow it to function as a Trojan horse, carrying a gene that prevents new infectious HIV from being produced," he explains. "Essentially, the vector puts a wrench in the HIV replication process."

The primary endpoints of the study were safety-focused, and looked for adverse events, and viral load and CD4 count changes. The investigators also wanted to find evidence that the genetically modified HIV was not replicating. Secondary endpoints included discovering how long the genetically modified CD4 cells persisted, as well as the individual's immune function.

This phase I study involved five highly treatment-experienced HIV-positive volunteers. All had been on failing antiretroviral therapy prior to enrolment and four decided to remain on therapy during the nine-month study period.

All participants were male – three were Caucasian, two were African-American – and they had received an average of seven previous anti-HIV drugs. All had viral loads at baseline above 20,000 and CD4 counts ranged between 220 and 316.

The first participant was infused in July 2003, and the last participant received his infusion in September 2004. Each of the five participants

received one infusion of their own gene-modified CD4 cells. The target dose was 10 billion cells, representing between 2-10% of the total number in an average person.

Viral load remained stable or decreased during the study, and one individual showed a sustained decrease in viral load. CD4 cell counts remained steady or increased in four of the five individuals.

Additionally, immune function that was specific to HIV improved in four of the five participants. The investigators also found no evidence that the genetically modified HIV was replicating, but did find that the genetically modified CD4 cells persisted for longer than a year in two of the five individuals. "That's significant," notes Dr Levine, "showing that these cells just don't die inside the patient."

The investigators also noted that the infusions were well tolerated, with "no serious adverse events that were judged as possibly, likely, or related to the VRX496 cells." All five individuals will be followed for 15 years, however, since it is possible that adverse events may take years to appear.

Overall, the study results are significant, say the researchers, because it is the first demonstration of safety in humans for a lentiviral vector (of which HIV is an example) for any disease. Additionally, they add, VRX496 produced encouraging results in some patients where other treatments have failed.

"The goal of this phase I trial was safety and feasibility and the results established that," notes Dr June. "But the results also hint at something much more. Gene therapy has long been discussed as an alternative treatment to HIV. The results from this phase I trial are encouraging – particularly since these are late-stage patients – and demonstrate that gene therapy has the potential to treat HIV and other serious human diseases."

However, cautions Dr Levine, "just because this has produced encouraging results in one or two patients doesn't mean it will work for everyone. We have much more work to do."

A second trial involving six successive infusions in HIV-positive individuals on successful antiretroviral therapy is now recruiting. This will include a planned treatment interruption in order to test the effect of the gene therapy's ability to control HIV.

Although the specific process and targets used in this study is unique, a variety of other HIV-focused gene therapies are currently being studied, including several that are further ahead in their development. These include Johnson & Johnson's OZ1 (previously known as RRz2) – which has already advanced into a Phase II study with 74 participants* – [Cell Genesys' T-cell modifier](#), and [Enzo Biochem's HGTV43](#), which also delivers antiretroviral antisense.

* Study conducted in Australia and the US.

Reference: Levine BL et al. Gene transfer in humans using a conditionally replicating lentiviral vector. [PNAS Early Edition](#), 2006.

Source: www.aidsmap.com. *Genetically modified HIV shows early promise as a gene therapy viral vector.* Article by Edwin J. Bernard, Thursday, November 09, 2006.

Belly Fat Gains No More Common in HIV

Increases in waist size are no more common in HIV-positive men compared to HIV-negative men, according to new data from the Multicenter AIDS Cohort Study (MACS). The research, published in the November 1 issue of the *Journal of Acquired Immune Deficiency Syndromes*, affirms the results of previous studies suggesting that abdominal fat gain is not a unique complication of HIV or its treatment.

[HIV-associated lipodystrophy](#) is a term that has not been officially defined, given that researchers disagree on its signs and symptoms. Experts agree that lipoatrophy – a loss of subcutaneous fat in the face, arms, and/or legs – is clearly a unique and relatively common problem among HIV-positive people, especially those on HIV treatment. What they do not agree on is if lipohypertrophy – the accumulation of visceral fat, notably around the gut (central adiposity) – is unique or more frequently seen in HIV-positive people receiving therapy.

The most widely cited data questioning the contribution of lipohypertrophy to the definition of lipodystrophy come from the Fat Redistribution and Metabolic Change in HIV Infection (FRAM) study. It enrolled 825 HIV-positive men, 350 HIV-positive women, and 338 HIV-negative people. Results focusing on men enrolled in FRAM were published in October 2005. Results focusing on women enrolled in the study were published in August 2006 (see [aidsmap website story here: www.aidsmeds.com/news/am20060808.html](#)).

The study demonstrated that lipoatrophy was much more common in the HIV-positive volunteers than in the HIV-negative study participants. Approximately 38% of the HIV-positive men had lipoatrophy of at least one part of the body, compared to approximately 4% of the HIV-negative men. Lipoatrophy was also documented in 28% of the HIV-positive women compared to 4% of the HIV-negative women.

The study also demonstrated that HIV-positive people do experience lipohypertrophy. However, this buildup of fat – which was confirmed using MRI scans – was actually more common in the HIV-negative men than in the HIV-positive men. Approximately 40% of the HIV-positive men had



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lipohypertrophy, compared to approximately 56% of the HIV-negative men. Among women, reported lipohypertrophy was similar in both groups. Approximately 62% of the HIV-positive women had lipohypertrophy of at least one part of the body, compared to 63% of the HIV-negative women.

In short, the FRAM results suggested that lipoatrophy is a unique complication caused by HIV and/or HIV medications, whereas lipohypertrophy (belly fat gain) is not, given that fat increases were similar among the HIV-positive and HIV-negative women and even more common in the HIV-negative men.

The most recent study, conducted by Todd Brown, MD, of Johns Hopkins University and his MACS colleagues, followed 1,053 men from 1999 to 2003. Approximately 63% of those enrolled were HIV positive (the vast majority were receiving antiretroviral therapy); the remaining 37% were HIV-negative volunteers.

While most lipodystrophy studies use CT, MRI, and DEXA scanning, these tests are not routinely available to healthcare providers and tend to be expensive and cumbersome. The MACS employed a much simpler tool: tape measures to evaluate waist, hip, and limb circumferences. As primitive as this may sound, measuring waist circumference – and comparing the circumference of the waist to that of the hip (known as the waist/hip ratio) – has long been used to help diagnose obesity and to predict cardiovascular disease and diabetes in those at risk.

According to the report by Dr. Brown and his colleagues, both the HIV-negative and HIV-positive men experienced increases in the waist circumferences over the four-year study period. There were no statistically significant differences in waist circumferences between the two groups. What's more, both groups saw their waist circumferences increase at the same rate. These results, much like those documented in FRAM, suggest that central adiposity is no more likely to occur in HIV-positive men compared to HIV-negative men.

As for hip circumferences, these also increased in both groups. However, the hip circumference increases occurred faster in the HIV-negative men than the HIV-positive men on antiretroviral treatment. Because hip circumferences are dependent on subcutaneous fat gains – fat below the surface of the skin, as opposed to visceral fat deep within the body – these results can be taken as additional evidence that lipoatrophy (subcutaneous peripheral fat loss) is the defining characteristic of (HIV-associated) lipodystrophy.

These findings, Dr. Brown and his group writes, "underscore the fact that peripheral fat wasting is the more predominant body composition alteration among HIV-infected patients receiving [HIV treatment] and that changes in waist/hip ratio do not necessarily represent central adiposity in this population." In fact, lipodystrophy experts like Donald Kolter, MD, of St. Luke's-Roosevelt Hospital in New York have suggested that lipoatrophy can make central adiposity appear much more severe and uncharacteristic of typical fat gain.*

Treatment Officers Note: *I am aware that some people with HIV may argue against this conclusion, as it is not their individual experience. Please note these trials are about majority differences, not necessarily individual experience, despite the significance of the conclusions. If you have managed a level of restoration of body fat in relation to hip to waist ratios, consider that this may be visceral waist fat improvement, rather than peripheral lipoatrophy improvement in - more closer to the skin surface - hip fat. What's important is how you feel about your body, so continue to express this to your doctor. You are welcome at any time to discuss matters with me as well. Dieticians are great support too. Also, there are some studies in the pipeline for novel new treatments: see QPP's January – February 2006 Treatment Update Newsletter.*

Reference:

Brown T, Wang Z, Chu H, et al. Longitudinal anthropometric changes in HIV-infected and HIV-uninfected men. *J Acquir Immune Defic Syndr* 43(3):356-62, 2006.

Source: www.aidsmap.com Adapted from Article by Tim Horn, Senior Writer & Editor.

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Erectile dysfunction and heart disease

It's known that there's an association between heart disease and erectile dysfunction. The link is probably the damage to small arteries and certainly men with heart disease can have problems with sexual performance. But does it cut the other way? Could erectile dysfunction be a warning that the man is silently brewing a heart attack or stroke?

The answer is yes from a prostate cancer prevention trial which followed 19,000 men* for seven years asking them about libido and sexual function every three months. About 40 per cent of the men taking a placebo had erectile problems and their subsequent risk of a heart attack, stroke or angina was similar to having a history of high cholesterol, smoking or close family with heart disease.



Erectile dysfunction was a predictor in its own right and added to the risk from say diabetes. So it's a question that GPs should ask of their middle aged male patients and act on the answer if it's yes.

It's not clear whether reducing the heart risk factors will help the erectile dysfunction as well.

*** Treatment Officers Note:**

Not a HIV study, but it's useful to consider that HIV treatment and a number of compound lifestyle issues (smoking, bad diet) and increasing age can increase heart disease risk in PLWHA. It is also not clear if reducing heart disease risk factors will help erectile dysfunction for men with HIV, but normal sense would say that the healthier the lifestyle the more one can rule out any contribution to erectile dysfunction from unhealthy or less healthy lifestyle choices.

For reference: Thompson IM et al. Erectile dysfunction and subsequent cardiovascular disease. [Journal of the American Medical Association](#) 2005;294:2996-3002

Source: www.abc.net.au/HealthMinutes by Norman Swan. 25th September, 2006.

More on Kaletra Monotherapy: Comparable to Combination Therapy

In a study using ultra-sensitive viral load testing, most people who switched from combination therapy to *Kaletra* monotherapy were found to have extremely low HIV viral loads at 48 weeks (c. one year), comparable to those on *Kaletra* plus two nucleoside analogues.

Monotherapy (treatment with a single antiretroviral drug) is contrary to all established HIV treatment guidelines, which call for a combination of at least three antiretrovirals. However, recent studies have shown that standard-dose *Kaletra* (which contains lopinavir/ritonavir in one pill) alone, without accompanying nucleosides, keeps viral loads undetectable in a large percentage of HIV-positive people. This surprising result has led to more in-depth studies of *Kaletra* monotherapy.

The OK (Only Kaletra) study

The new analysis (described below) follows up on the earlier "OK" (Only *Kaletra*) study, a "proof-of-concept pilot trial" which enrolled 42 HIV-positive adults at hospitals in Spain. All participants were receiving standard-dose *Kaletra* (400/100mg¹ twice daily) plus two nucleoside analogues, and their viral loads were undetectable by standard tests (less than 50 copies/mL). (None had any history of

treatment failure on protease inhibitors.) Participants were randomised to either continue treatment unchanged, or to stop their "nukes" and continue on *Kaletra* alone. After 48 weeks, 95% of the triple-therapy group still had undetectable viral loads, versus 81% of the monotherapy group. Researchers noted "significantly worse adherence" in those whose viral loads rebounded on monotherapy.

Ultra-sensitive analysis

In the new findings, published in the journal *AIDS*, researchers from the University of Pittsburgh joined members of the original study team to do further analysis. An ultra-sensitive test (a modified Roche Amplicor HIV-1 RNA assay), which could measure viral levels as low as 3 copies/mL, was used on blood samples stored from the original participants. (Where values below 3 copies/mL were observed, analytical methods were used to calculate values between 0 and 3 copies/mL.)

Out of the 42 participants, 37 continued on treatment up to 48 weeks with viral loads below 50 copies/mL. The ultra-sensitive test found that, for these people, viral load levels remained essentially unchanged over the 48 weeks. Measured viral loads, in copies/mL, were as follows:

Time point	<i>Kaletra</i> only	Triple therapy
Beginning	5.1	3.0
Week 4	4.5	2.9
Week 8	3.3	2.9
Week 12	1.9	1.0
Week 24	3.7	3.6
Week 48	2.8	1.6

There was actually no statistically significant difference between these monotherapy and triple-therapy values, even when people whose treatment "failed" were included in the analysis.

The researchers concluded that "the level of persistent viremia did not change after discontinuing NRTI [nucleoside] therapy in subjects who remained suppressed < 50 copies/ml. The finding was consistent across multiple analyses". While they stopped short of recommending *Kaletra* monotherapy as a standard treatment option, they stated that "continued use of NRTI may prove to be unnecessary in 80 - 90% of patients after successful induction therapy", and that "cautious evaluation" and "careful identification of appropriate patients for this strategy" is warranted.

Treatment Officers Note:

1. 400/100mg refers to the daily dose of drugs contained in *Kaletra*, which is



400mg of lopinavir and 100mg of ritonavir (used as a 'baby-dose' to boost the levels of lopinavir up to make it work within therapeutic range for proper inhibition of HIV). Kaletra now has a **new tablet** formulation, instead of the **old capsule** form, which comprises less pills to take – i.e. **two (2) tablets twice daily** with or without food, compared to the old formulation of three (3) capsules twice daily with food.

2. Kaletra monotherapy studies are still experimental, although proving interesting for future possible new short-term reduced treatment maintenance strategy. Research interest in this method has been ongoing for some time now.

References:

McKinnon J et al. The level of persistent HIV viremia does not increase after successful simplification of maintenance therapy to lopinavir/ritonavir alone. *AIDS*. 20: 2331-2335, 2006.

Arribas J et al. *Lopinavir/ritonavir as single-drug therapy for maintenance of HIV-1 viral suppression: 48-week results of a randomized, controlled, open-label, proof-of-concept pilot trial (OK study)*. *J Acquir Immune Defic Syndr*. 40: 280-287, 2005.

Source: www.aidsmap.com. *Kaletra Monotherapy: Ultra-sensitive tests find viral loads comparable to triple combination therapy*. Article by Derek Thaczuk, Wednesday, December 06, 2006.

Micronutrient deficiencies less common in people taking antiretrovirals

A study team has found several vitamin and mineral deficiencies, noted as common before the widespread use of antiretrovirals, to be much less common in people on antiretroviral therapy. They also found these deficiencies to have relatively little effect on CD4 cell counts and HIV viral load.

For people with HIV, "which vitamins should I take?" is a simple question with a complicated answer. Most research to date has studied people who are not on antiretroviral treatment. Widespread micronutrient (vitamin and mineral) deficiencies (as measured by low blood serum levels) have been found in these people, and many of these have been associated with worse health outcomes.

Although supplement use is very common among HIV-positive people, studies have often been very inconsistent regarding their benefits, with some even showing worsened outcomes for certain supplements. Little research has been done on

micronutrient levels and supplementation in HIV-positive people who are on therapy.

The Nutrition for Healthy Living study

Nutrition for Healthy Living (NFHL) is an ongoing cohort study of nutrition and HIV disease status in HIV-positive adults in Boston, Massachusetts and Providence, Rhode Island. In this component of the NFHL study (published in the December 1st issue of the *Journal of Acquired Immune Deficiency Syndromes*), the researchers specifically looked at HIV-positive people on antiretroviral therapy. For each of four specific micronutrients – retinol (vitamin A), α -tocopherol (vitamin E), zinc, and selenium – they asked the following four questions:

- how many people on antiretrovirals are deficient in these micronutrients?
- how do these deficiencies affect viral load and CD4 levels?
- what are the effects of supplementation?
- how do these findings compare with other studies?

The study group was made up of 171 men and 117 women, seen between 2000 and 2003. All were on antiretroviral therapy, and the majority (62 – 69%) had undetectable viral loads. Most were in their mid-40s, and had been infected an average of ten years. The group was quite racially varied, and many were poor.

What were the results?

Except for zinc, deficiencies were much less common than those seen in people not on antiretrovirals. None of the micronutrient levels significantly affected CD4 counts. Viral loads tended to be slightly lower in people with higher zinc and selenium levels, although these were not statistically strong results. Results for retinol (vitamin A) were more complex, and are described in the 'retinol' section below. Supplement use did not have a significant effect on CD4 count or viral load for any of the micronutrients studied.

Specific results for Vitamin E

Serum levels of α -tocopherol (vitamin E) were found to be deficient (defined as less than 500 μ g/dL) in 7% of the men and essentially none of the women. Consistent with previous studies, this deficiency did not correlate with any differences in CD4 count or viral load.

For zinc

Of the four nutrients studied, zinc was the only one found to be widely deficient: 40% of the men and 36% of the women had low zinc levels. (The NFHL defined 'deficiency' as serum zinc levels less than 670 μ g/L: this is in the middle of the range of various levels used in other studies.)



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The researchers did not find that zinc deficiency affected CD4 counts or had much effect on viral load. People with higher zinc levels tended to have lower viral loads, but this effect was slight and not statistically significant. While there was no evidence that zinc intake actually increased serum zinc levels, neither was there any indication that it was harmful.

The viral load findings, slight as they were, raise a question about cause and effect: could higher zinc cause better control of HIV viral load? This study cannot answer that question: it could be true, but so could the converse – that lower viral load causes increased zinc levels. (Since HIV itself uses zinc, lower levels of HIV – due to drug treatment – could free up zinc which would otherwise be used by the virus.)

Selenium

Selenium deficiency was seen in 8% of the men and 3% of the women; much lower than some reported figures for people not on antiretrovirals. (A small study, mostly in injection drug users, reported 77% selenium deficiency rates in 1995.) The NFHL study defined selenium deficiency as serum levels less than 85 µg/L – levels which are definitely associated with increased mortality.

As with zinc, viral load levels varied slightly by selenium levels, and CD4s were not affected. Women with the lowest selenium levels tended to have the highest viral loads; there was no significant difference in men.

Retinol (Vitamin A)

Serum retinol was deficient in 5% of the men and 14% of the women. (Deficiency was defined as less than 30 µg/dL.)

Effects of low retinol were the study's oddest and most complex finding. In women, the lowest retinol levels were found in the women with the lowest viral loads; this was a statistically significant result. The same was true only for men with high CD4 counts (>350 cells/mm³). However, for men with lower CD4s (less than 350 cells/mm³), the opposite was true - higher retinol levels were found in those with lower viral loads. There was no explanation for these apparently contradictory findings, However, other studies have found that people with moderate levels of vitamin A intake are less likely to progress to AIDS than those with very low or very high intakes.

Limitations and Comments

The researchers concluded that "most of our participants had adequate serum levels of retinol, α-tocopherol, and selenium. Low serum zinc was common." They also noted that "although micronutrient supplement use was relatively common, it was not significantly associated with improved HIV disease status." However, 'HIV disease status' was only narrowly measured by CD4 counts and viral loads, and did not include any

quality-of-life measures or clinical outcomes. Also, this study only looked at people on antiretroviral therapy, so it adds no knowledge about those who are not on these medications.

Treatment Officers Note:

Science is a 'strange but logical creature'. It takes its analysis on the balance of proof in large randomised trials. Until such times it can neither prove nor disprove. This is a somewhat disproving summary, but there have been many small studies which prove, even in recent times in the presence, and absence, of antiretroviral therapy. Ultimately your decision to take oral supplements is a personal choice, sometimes guided by general and dietary advice along-side population-wide (non-HIV) study results, notwithstanding your own personal experiences which may, in fact, be the most valid proof. The matter remains controversial in HIV medicine. Peak complementary therapy research bodies exist in Queensland. Although they presently tend to exclude chronic conditions such as HIV; principally, in my understanding, due to lack of investment and more complex trial protocols, not lack of interest.

Reference: Jones C et al. *Micronutrient levels and HIV disease status in HIV-infected patients on highly active antiretroviral therapy in the Nutrition for Healthy Living cohort.* J Acquir Immune Defic Syndr. 43(4): 475-482, 2006.

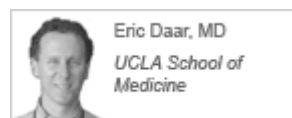
Source: www.adismap.com. Article by Derek Thaczuk, Tuesday, November 28, 2006

The continuing word about adherence to treatment...

Q undetectable and resistance

Doesn't viral replication continue at some low level even if the patient is adherent on meds and is undetectable?...and if that's true doesn't it imply that resistance to the current med the patient is on is inevitable?

A Response from Dr. Daar



Thank you for posting these great questions.

It turns out that there is some evidence that virus persists in certain parts of the body even when viral load is undetectable in plasma. However, most of the data suggests that in most of these cases virus is not evolving to become resistant to the drugs. Consequently, while I can't say for sure that resistance will never develop in these situations, I do believe that for most people with undetectable viral load who are consistently taking their



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medication resistant virus will not emerge. Obviously only time will tell, but even if I am wrong it is likely to not occur for many years, at which time there will continue to be many treatment options.
Best, Eric

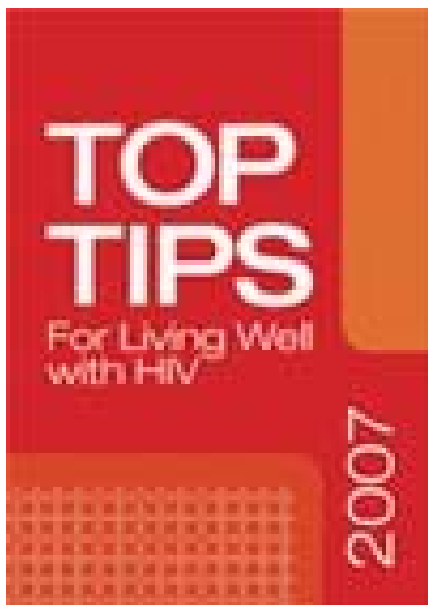
Source: "Ask the Experts" www.thebody.com/ Nov 10, 2006.

New Resource: Top Tips for Living Well With HIV

Top Tips for Living Well With HIV is a national new AFAO resource, produced in collaboration with representatives from NAPWA, PLWHA NSW and ACON.

The 25 tips cover a range of issues that have been identified as among the most important things people with HIV need to know about managing their health and well being in 2007. Tips include dealing with side effects, managing recreational drugs, health monitoring and social support.

For a copy of the resource please visit or call any QPP Statewide Office, or ask at your local HIV and Sexual Health Clinic or HIV Service Provider. Copies can also be obtained from QPP Statewide Centre by calling (07) 3013-5555 or 1800-636-241 (toll-free). Additionally, copies can also be downloaded from the AFAO website at www.afao.org.au



New Saquinavir Tablet

A new 500mg tablet formulation of *Invirase* (saquinavir) has now been approved in Australia. It was marketed in the US in 2004.

The new tablet is designed for dosing with ritonavir, and the approved dose is 1000mg (2x500mg tablets) twice daily after meals with 100mg (capsule) of ritonavir taken at the same time. This new 500mg tablet cuts the pill burden (number of pills) substantially for people taking Saquinavir. The old **hard-gel capsule** formula of *Invirase* was taken as 3x200mg **capsules** three (3) times daily = 9 capsule daily, which is quite outdated treatment methodology and excessive pill burden. This old formulation is no longer available.

Also, prior to this, the *Fortovase* **soft gel-capsule** formulation of saquinavir, was also taken off the market, because it too was an even higher pill burden and higher side effects compared to old **hard-gel capsule** *Invirase* (now also removed from market availability).

So now, people taking Saquinavir have the singular option of the new 500mg **tablet** formulation only, which is 6 pills daily (i.e. 2 x *Invirase* tablets + 1x ritonavir capsule, twice daily). *Invirase* is to be also taken as part of other combination HIV treatments for effective therapy, but many new combination treatments also have a low pill burden taken twice daily or sometimes once daily.

Ask your doctor for further information, or feel free to contact to the QPP treatments Officer on (07) 3013-5505 or 1800-636-241 (statewide toll-free).

Genital Herpes Facts

- Two viruses cause genital herpes: herpes simplex virus type 1 (HSV-1) and herpes simplex virus type 2 (HSV-2). Both cause life-long infection.
- HSV-2 affects five to 20 per cent of the population (including many individuals who are also infected with HSV-1: 80% of the population, causing recurrent cold sores on the lips). HSV2 is more often associated with genital herpes, although either virus can cause a similar disease at both anatomical sites.
- The time from exposure to the virus and when you get the symptoms is unpredictable. It can be anything from days to years, or never at all.
- You can shed herpes without apparent symptoms – this is referred to as 'asymptomatic shedding' and is thought to occur in 15 per cent of individuals. In reality, there may be small, imperceptible lesions (sores) that go unnoticed by the carrier. These asymptomatic shedding



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events probably underlie the majority of infections.

- Condoms are recommended to help prevent transmission, but they're only effective if they cover the affected area and no virus is transferred to the outside of the condom when putting it on. Having said that, it shouldn't be assumed that the penis is the source of the infection since oral sex is a very effective (and now probably the most common) means of transmission. Cold sore strains affecting the mouth can be transferred to the genitals where they cause disease akin to genital herpes.
- More than three-quarters of people with genital herpes don't receive appropriate treatment because the infection isn't properly diagnosed.
- Anti-viral medications are very effective.
- Herpes virus type 1 and type 2 can make it easier to pass on or get HIV. If you have HIV and active herpes sores, then this is a period when you are more likely to pass on Herpes or HIV. For people who do not have HIV but active herpes sores are more likely to get HIV, if HIV infected blood or semen comes into contact with those sores.

Source: www.abc.net.au/health/yourstories/herpes.htm
Extract modified from *Love Hurts: Living with Herpes*, by Kathy Graham; and *Herpes Simplex Virus Fact File*.

Websites of the Month

For update on what treatment trails are happening in your region, visit:

<http://www.ashm.org.au/research/>

and in Queensland:

<http://www.som.uq.edu.au/hivandhcvprojects/>
(click on the link "view clinical trials register")

HEPATITIS C ?!!

Where can I find out more?



Further information about Hepatitis C treatment and support can be obtained from:

- **Queensland Injectors Health Network (QuIHN)** on 3620-8171, OR
- **The Hepatitis C Council of Queensland (HCQ)** on 3238-5704, OR
- **Queensland Positive People (QPP)** on 3013-5505.

Friendly peer support and discussion groups are regularly conducted by HCQ and QuIHN. Friendly and skilled workers are available to assist in information about Hepatitis C, along with treatment information and support. For information about HCV and HIV coinfection please feel free to contact QPP also. Should you have a specific enquiry or request, among us we should be able to assist you with your needs.



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