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## I ANTI-HIV THERAPY

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### A. Large Italian study looks at when to start HAART

#### Background & summary

Recent revisions to North American HIV treatment guidelines have taken a more cautious approach to the timing of treatment. This shift was necessary as it became clear that highly active antiretroviral therapy (HAART) has the following limitations:

- it is unable to cure HIV/AIDS
- it has complex adherence requirements
- it is associated with serious side effects
- the development of drug-resistant virus

The question of “when to start” therapy is not easily answered. To try and help find some answers to this question research teams around the globe are observing the effect of anti-HIV therapy in people at different stages of HIV disease.

One study in Italy called ICONA enrolled more than 1,400 subjects who had never been exposed to anti-HIV drugs (they were “treatment naive”). The doctors gave them HAART and have been monitoring their progress for at least two years. After analysing their data the doctors found that subjects who started therapy with fewer than 200 CD4+ cells were more likely to eventually develop detectable viral loads than subjects who started therapy with more than 200 CD4+ cells. Importantly, the researchers stated that “there was no clear immunological or virological advantage to starting HAART at a CD4+ cell count higher than 350 cells rather than when the count was between 200 to 350 cells.”

*(continued on next page)*

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## Study details

Researchers reviewed data collected from 1,421 subjects whose profile at the start of the study was as follows:

- 25% female, 75% male
- average age – 34 years
- average CD4+ count – 272 cells
- average viral load – 70,000 copies

After subjects enrolled in the study, here is a profile on their use of HAART:

- about 87% took two nucleoside analogues (nukes) and a protease inhibitor (PI)
- the most commonly used nukes were AZT and 3TC
- the most commonly used PI was indinavir (Crixivan)

## Results – CD4+ counts

The ICONA study is ongoing and the results presented here are based on about two years of data collection.

To understand the effects of therapy at different stages HIV infection researchers divided subjects into three groups based on their CD4+ cell counts. The groups were as follows:

- fewer than 200 cells
- between 200 to 350 cells
- more than 350 cells

On average, regardless of pre-study CD4+ count, CD4+ counts rose by at least 180 extra cells in all groups. This increase was maintained for up to two years after subjects began to use HAART. Despite the use of HAART, about 9% of subjects had a CD4+ count that remained below the 200 cell mark. Researchers aren't sure why this poor immunologic response occurred. They do note that 29 of the 32 subjects who had a poor immunologic response had entered the study with a CD4+ count below the 201 cell mark.

## Rising viral load

The researchers sought to find out if there was a link between rising viral load and the CD4+ count at the start of the study. They called this rising viral load “virological failure” and defined it as a viral load that was greater than 500 copies after six months of HAART on at least two occasions.

By the 2<sup>nd</sup> year of the study, the risk of virological failure — which researchers found was linked to the

CD4+ count the subjects had at the start of the study — was as follows:

- 51% – if subjects had fewer than 200 cells at the start of the study
- 44% – if subjects had between 200-350 cells at the start of the study
- 44% – if subjects had more than 350 cells at the start of the study

The pre-study viral load did not appear to have any impact on whether or not HAART regimens were able to suppress viral load during the study.

## AIDS and death

The researchers calculated the risk of subjects developing life-threatening infections (AIDS) or dying, particularly among those subjects who entered the study with fewer than 50 CD4+ cells. They found that the risk of developing AIDS or dying was high at first, but as HAART boosted the CD4+ count, the risk decreased dramatically as follows:

- fewer than 50 CD4+ cells – 19% risk
- between 50 and 199 cells – 5% risk
- more than 200 cells – 3% risk

In summarizing their results the researchers reported that there was no evidence that starting therapy when the CD4+ count was greater than 350 cells instead of at a lower level — between 201 and 350 cells — offered any increased immunological benefit two years later.

As the ICONA study continues and data collection increases, further conclusions about the benefits of early vs. late therapy will become available.

## REFERENCE

1. Cozzi Lepri A, Phillips AN, Monforte A d'A, et al. When to start highly active antiretroviral therapy (HAART) in chronically HIV-infected patients: evidence from the ICONA study. *AIDS* 2001;15(8):983-990.

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## II IMMUNE BOOSTERS

### A. Can ginseng help suppress HIV?

#### Background

The roots of the ginseng plant have been used for centuries by herbalists because of its anti-stress and anti-fatigue effects. Researchers in Hong Kong have been studying this herb and found that ginseng contains a protein called panaxagin which has anti-HIV activity in the test-tube. This protein

appears to work by interfering with an enzyme used by HIV called RT (reverse transcriptase). This is the same enzyme that is attacked by AZT and other nucleoside analogues, or nukes, such as 3TC (lamivudine, Epivir), d4T, ddI, ddC and ABC (abacavir, Ziagen).

### Korean red ginseng

Researchers in Seoul, South Korea, have been studying the effect of one type of ginseng — Korean red ginseng — on HIV infection in people for the past decade. In one six-month study, subjects who received 5.4 grams, or 18 capsules, of Korean red ginseng (KRG) daily were able to maintain or increase their CD4+ and CD8+ cell counts. These results prompted the researchers to conduct a more detailed investigation of the impact of KRG on HIV infection.

### Study details

Researchers enrolled 18 subjects (4 female, 14 male) who were taking AZT and monitored them for an average of six years. Note that the study was conducted between 1991 and 1997, when protease inhibitors were not widely available in South Korea. All subjects were free from symptoms of HIV/AIDS at the time they entered the study. Their average CD4+ count was 256 cells. Half the subjects were assigned to continue receiving AZT 400 mg to 600 mg/day and six capsules of KRG (each containing 300 mg) three times daily for a total of 18 capsules/day (5.4 grams). This compound is made using the roots of six-year-old KRG and is sold in South Korea by the Korea Ginseng company. The quality of this product is monitored by South Korean authorities. The remaining subjects were assigned to continue taking AZT but no KRG.

### Results – Effect on CD4+ cell count

On average, subjects who were in the group receiving both AZT and KRG did so for six years. During this time their average CD4+ cell count remained relatively unchanged, going from an average of 239 cells at the start to an average of 234 cells by the end. In the group receiving AZT alone, the average CD4+ cell count decreased from 272 cells to 146 cells over a period of four years.

### Resistance to AZT

The researchers analysed blood samples from all subjects to check for changes or mutations in HIV's genetic material that allowed the virus to resist the effect of AZT. They found that on average, subjects taking AZT and KRG had about 22% of resistance mutations while subjects taking AZT alone had about 56% of resistance mutations to AZT. These findings

suggest that KRG delays the onset of resistance to AZT.

At the time the study took place, researchers did not have access to sophisticated techniques of viral load measurement that are now in regular use in developing countries.

The research team suggests that KRG may contain compounds that have the ability to enhance the activities of immune cells.

### Caution and concerns

More research is needed on KRG to find the following:

- its impact on the immune systems of people with HIV/AIDS
- interactions between KRG and anti-HIV drugs, particularly protease inhibitors and non-nukes. This is because some herbs can interact with these drugs by either raising or lowering their levels and possibly weakening their anti-HIV effects or increasing side effects.
- its impact on blood sugar levels
- its effect on blood pressure (ginseng may raise blood pressure over the long-term)
- its effect on hormone levels, particularly in women. This is important because “menstrual abnormalities” have been reported with long-term use of ginseng in some women.

The effect of other types of ginseng preparations on the immune system may be very different from those seen in the Korean studies. KRG may be useful in resource-poor countries where it could be studied in combination with AZT and similar drugs. Further studies of KRG are underway in South Korea and Thailand (YK Cho, *written communication*).

A point not considered by the research team in Korea is possible contamination of ginseng used in supplements by heavy metals such as cadmium and lead as well as pesticides. This possible contamination is the focus of the following story.

### REFERENCES

1. Ng TB and Wang H. Panaxagin, a new protein from Chinese ginseng possesses anti-fungal, anti-viral, translation-inhibiting and ribonuclease activities. *Life Sciences* 2001;68(7):739-749.
2. Cho YK, Sung H, Lee HJ, et al. Long-term intake of Korean red ginseng in HIV-1-infected patients: development of resistance mutations to zidovudine is delayed. *International Immunopharmacology* 2001;1(7):1295-1305.
3. Anonymous. Product review: Asian and American ginseng. [www.consumerlab.com/results/ginseng.asp](http://www.consumerlab.com/results/ginseng.asp) accessed 18 July, 2001.

## B. Pesticides found in many ginseng supplements in the U.S.

The organization ConsumerLab in the U.S. conducts independent testing of supplements sold in that country. The purpose of the testing is to find out if the ingredients listed on the label match those found in the capsules, tablets or liquids that accompany the label. The company produces reports which are available on its website at [www.consumerlab.com](http://www.consumerlab.com).

In a recent study, ConsumerLab tested 22 brands of ginseng supplements sold in the U.S. and found that only nine passed its review. Unfortunately, the company only lists the brands that pass its testing. They found that eight of 12 products that were labelled to contain “Korean ginseng” were contaminated with pesticides (hexachlorobenzene and/or quinterozone). These compounds may damage the liver and kidneys and have the potential to cause cancer in people. In some cases the level of contamination by pesticides was 20 times higher than allowed under U.S. and European guidelines.

For further details about the review of ginseng supplements, readers can visit the website at [www.consumerlab.com/results/ginseng.asp](http://www.consumerlab.com/results/ginseng.asp). General safety information about ginseng is also available from the site. These results on contamination point to the need for similar research on products available in Canada.

### REFERENCE

1. Anonymous. Pesticide contamination found in many ginseng supplements tested by [consumerlab.com](http://consumerlab.com): only 9 of 22 products pass product review published online today. Press release 11 July, 2001.

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## III SIDE EFFECTS

### A. Spanish study looks at the effect of nevirapine on the liver

#### Study details

Doctors in Spain analysed data from 610 subjects who were using nevirapine (Viramune)-containing regimens. The subject profile at the start of the study was as follows:

- 30% female, 70% male
- 60% had AIDS
- 80% were using three anti-HIV drugs
- 20% were using four or more anti-HIV drugs

- 31% used a protease inhibitor in addition to nevirapine
- 13% had never used anti-HIV drugs
- 46% had hepatitis C virus (HCV) infection
- 9% had hepatitis B virus (HBV) infection
- average CD4+ count – 279 cells
- average viral load – about 10,000 copies

Other than the non-nuke nevirapine, drugs commonly used by subjects included the following:

- d4T (Zerit)
- 3TC (lamivudine, Epivir)
- AZT
- ddI (Videx)
- nelfinavir (Viracept)

The purpose of the study was to find out about nevirapine’s impact on the liver. To do this, technicians regularly tested blood samples from study subjects checking for liver enzyme levels. This is because when the liver is damaged, levels of liver enzymes in the blood rise above their normal range. The following enzyme levels were measured:

- ALT – alanine aminotransferase
- AST – aspartate aminotransferase
- AP – alkaline phosphatase
- GGT – gammaglutamyl transferase

The researchers defined liver toxicity during the study as any increase in the liver enzymes AST or ALT that was three times greater than their level at the start of the study.

### Results — toxicity

76 subjects (about 12%) developed liver toxicity (as defined previously). The following proportion of subjects had higher-than-normal levels of the following enzymes:

- 29% – GGT
- 11% – ALT
- 7% – AST
- 1% – AP

### Deaths

Twelve subjects died from causes other than nevirapine-related toxicity. The causes of death were as follows:

- cancer – 5 subjects
  - CMV (cytomegalovirus) – 1 subject
  - TB (tuberculosis) – 1 subject
  - bacterial pneumonia – 2 subjects
-

- swollen pancreas gland (pancreatitis) – 2 subjects
- liver damage – 1 subject

### Stopping nevirapine

239 subjects (nearly 40%) stopped taking nevirapine during the study, most commonly because of rising viral load.

### Hepatitis

Only 7 subjects (1%) had to stop taking nevirapine because they developed the following signs/symptoms of hepatitis:

- weakness
- fatigue
- nausea/vomiting
- fever
- jaundice

The researchers noted that six of the seven subjects were also co-infected with HCV; two were also chronic “alcohol abusers” and one had HBV. On average, hepatitis developed about two months after subjects began using nevirapine.

### Who is at risk for liver damage?

Researchers came to the following conclusions about which factors place people at increased risk for liver damage associated with nevirapine use:

- for every year of prior use of anti-HIV medication before subjects used nevirapine the risk of liver damage increased by 10%
- subjects who were co-infected with HCV were twice as likely to develop liver toxicity as subjects who did not have this infection
- subjects with higher-than-normal levels of the liver enzyme ALT were 1½ times more likely to develop liver damage than subjects who had normal levels of ALT

The researchers noted that the risk of nevirapine-associated liver damage increased “steadily over time.”

#### REFERENCE

1. Martínez E, Blanco JL, Arnaiz JA, et al. Hepatotoxicity in HIV-1-infected patients receiving nevirapine-containing antiretroviral therapy. *AIDS* 2001;15(10):1261-1268.

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## B. Lipodystrophy — nukes vs. protease inhibitors

### Background

In the late 1990s, people with HIV/AIDS (PHAs) using HAART began to report strange changes in body shape. There were also reports of increased levels

of sugar, insulin and fatty substances in the blood of PHAs. These changes increased the risk of non-insulin-dependent diabetes and cardiovascular disease, among other complications. Together, these changes have been called the lipodystrophy syndrome.

The precise cause(s) of these problems is not clear, but their appearance in large numbers of PHAs taking HAART is striking and relatively recent. Although some people have been quick to blame protease inhibitors (PIs) for these problems, recent research suggests that nucleoside analogues, or nukes, such as AZT and similar drugs, may also play a role in the lipodystrophy syndrome.

Researchers in the Netherlands have been conducting a study in which subjects were given one of the following regimens:

- 2 protease inhibitors (PIs) – ritonavir (Norvir) and saquinavir (Fortovase)
- 2 PIs and a nuke – ritonavir and saquinavir and d4T (Zerit)

The purpose of this study was to find out if a PI-only regimen was as effective as a more conventional combination. Because researchers collected information about changes in body shape, results from this study may help in identifying the role that different drugs play in the lipodystrophy syndrome.

### Study details

Researchers reported results on 175 subjects who were monitored for up to two years. Their profile at the start of the study was as follows:

- average age – 36 years
- 12% female, 88% male
- 25% had AIDS
- 54% had never used anti-HIV drugs
- average CD4+ count – about 250 cells

Both ritonavir and saquinavir were taken at a dose of 400 mg twice daily while d4T was taken in the standard dose. After 12 weeks subjects had the option of “intensifying” their therapy by adding two nukes or a non-nuke to their regimen.

### Results — CD4+ counts and viral load

Researchers did not detect any significant differences in CD4+ cell count or viral load between the two study groups.

## Lipodystrophy

Doctors reported lipodystrophy in 29 of 175 subjects. On average signs/symptoms of lipodystrophy took about 1½ years to appear after subjects entered the study. The occurrence of lipodystrophy in each group was as follows:

- PIs only — 8%
- PIs and nuke(s) — 25%

This difference between the two groups was statistically significant, that is, not likely due to chance alone.

When the researchers analysed data from those subjects who had never used anti-HIV drugs the results were similar to those seen in the entire group:

- PIs only — 5%
- PIs and nuke(s) — 24%

## Who was at risk for lipodystrophy?

Those subjects who were assigned to receive PIs and a nuke(s) were nearly four times more likely to develop lipodystrophy than subjects who received only PIs.

The researchers noticed that those subjects who entered the study with higher-than-normal levels of cholesterol in their blood were at increased risk for the development of lipodystrophy. As well, those subjects who during the course of the study developed large increases in their cholesterol levels were likely to develop lipodystrophy.

The researchers found that at the start of the study the following factors were **not** significantly associated with the development of lipodystrophy:

- previous exposure to anti-HIV drugs
- length of previous exposure to anti-HIV drugs
- use of a particular nuke before entering the study
- gender
- age
- CD4+ cell count
- viral load
- triglyceride levels in the blood

## Fat gain and fat loss

Researchers found that among subjects who developed lipodystrophy, changes in body shape varied. There were cases of fat gain and fat loss (wasting) and cases of both occurring in the same person. Fat gain occurred in the following proportion of subjects with lipodystrophy:

- PIs only — 9%

- PIs and nukes — 29%

Fat loss or wasting occurred in the following proportion of subjects with lipodystrophy:

- PIs only — 14%
- PIs and nukes — 32%

These differences between the two groups, although striking, were not statistically significant. The proportion of subjects with lipodystrophy experiencing both fat gain and fat loss were as follows:

- PIs only — 57%
- PIs and nukes — 59%

## Study weakness

Readers should note that some researchers might say that a weak part of the study was that lipodystrophy was assessed by the subjects' "treating physician" rather than by more "objective" measures such as the following:

- DEXA (dual energy X-ray absorptiometry )
- ultrasound
- CAT scans

Nevertheless, other studies have generally found that the results of physician-based assessment and technical measurements tend to be in agreement when it comes to detecting lipodystrophy.

The results of this study confirm those seen in several others (see *TreatmentUpdate 114*, "Nukes linked to fat wasting" and *TreatmentUpdate 115*, "Canadian researchers study 'immune healing' ") that found that nukes are likely involved in fat gain/fat loss in HAART-users.

## REFERENCE

1. van der Valk M, Gisolf EH, Reiss P, et al. Increased risk of lipodystrophy when nucleoside analogue reverse transcriptase inhibitors are included with protease inhibitors in the treatment of HIV-1 infection. *AIDS* 2001;15(10):847-855.

## C. Indinavir and bones: Does thickness = strength?

### Background & summary

In the past two years there have been reports of people who use HAART developing the following complications:

- thinner bones
- weaker, more porous bones (osteoporosis)
- weak and fragile joints

Why these problems occur is not clear. Some researchers suspect that the components of HAART — protease inhibitors, nukes and/or non-nukes — and even HIV infection may all play a role. A number of research teams are trying to understand why these bone problems occur. For background information on the possible causes of bone problems in HAART-users and possible solutions, please see *TreatmentUpdate 118*.

Researchers in Australia have been studying the impact of protease inhibitors (PIs) on the thickness of bones in PHAs. They found that over time the spines of PHAs who used the PI indinavir (Crixivan) became thicker. In contrast, in those PHAs who used the PI nelfinavir (Viracept), the density of their spine remained unchanged. At first glance, the increasing thickness of bone may seem like a good thing but the researchers caution that increased thickness does not necessarily equal increased strength. Indeed, the way that bone is formed and shaped — the architecture of bone — by the body also plays an important role in giving bones their strength. The Australian researchers note that their findings should be interpreted with the following points kept in mind:

- other research teams need to confirm that indinavir-use does indeed cause bones to grow thicker
- the thickened bones really do have a reduced risk of breaking or fracturing

### **Study details**

Researchers monitored 54 male subjects who were using indinavir- or nelfinavir-containing treatment regimens for about two years. As part of the monitoring process, certain X-ray scans called DEXA (dual energy X-ray absorptiometry) were performed. DEXA was used to measure the thickness of the bones and fat content of the subjects' bodies. It is important to note that the 54 subjects did not have conditions or habits which could decrease the thickness of their bones, including the following:

- cancer
- infections that had spread all over their body
- prolonged bed rest

Nor were subjects taking any of the following supplements or drugs which could strengthen bones and thus cause the researchers to misinterpret the effect of the protease inhibitors:

- calcium
- vitamin D
- testosterone or other hormones
- bone-building drugs
- lipid-lowering drugs (statins)

In addition to these 54 subjects, researchers also collected data from two other groups of PHAs:

- 131 subjects who were using various PI-containing regimens
- 52 subjects who had never been exposed to PIs

Researchers used subjects from the two groups above to get measurements of the thickness of their backbones so that the results could be compared to those from the first group of 54 subjects.

### **Results — changes over two years**

Among the group of 54 subjects who were using indinavir or nelfinavir, researchers found the following:

- there was a significant loss of fat in the legs of subjects who used either PI
- the thickness of the spines of nelfinavir-users remained stable
- in indinavir-users the thickness of the spine increased significantly

### **Results — Thick vs. thin**

When researchers compared results between 131 subjects who were using PIs and the 52 who had never used a PI at first they found that the non-PI-users had, on average, thicker spines. When they re-analysed their data taking into account such factors as weight and height of subjects this difference between PI- and non-PI-users disappeared. Their conclusion was that, in general, thinner subjects were more likely to develop osteoporosis than heavier subjects. This finding is similar to that from studies of HIV negative people.

### **Muscles are important**

According to the researchers, lean body mass — a term that describes mostly muscle — affects the thickness of bones. In general, the greater the amount of muscle, the thicker the bones, at least in men. In women it appears that the amount of muscle and fat play a role in maintaining the thickness of bones. So the researchers suspect that those subjects who developed thinner bones had relatively low levels of lean body mass when they entered the study.

The research team cautioned that just because the bones of indinavir-users became thicker doesn't necessarily mean that they are stronger. As a result more research is needed in the following areas:

- confirmation of the results from this Australian study
  - an understanding of how indinavir might cause bones to grow thicker
-

- if indinavir-stimulated bone growth also occurs in women who use this drug

REFERENCE

1. Nolan D, Upton R, McKinnon E, et al. Stable or increasing bone mineral density in HIV-infected patients treated with nelfinavir or indinavir. *AIDS* 2001;15(10):1275-1280.

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## **D. Fosamax for HIV-related bone problems?**

### **Background**

People with HIV/AIDS (PHAs) who use corticosteroids and/or anti-HIV therapy may be at increased risk for the development of thinner, more porous bones — a condition called osteoporosis. As bones become thinner they can easily break or fracture. The best course of action to prevent and treat osteoporosis in PHAs is not yet clear and researchers are experimenting with different strategies. In a letter published in the August 1, 2001 issue of the journal *Clinical Infectious Diseases*, researchers in Italy reported that the bone-building treatment Fosamax (alendronate) has helped one PHA recover from osteoporosis. We now report the details of their report.

### **Study details**

According to the team, the 51-year-old PHA had been on a HAART regimen consisting of indinavir (Crixivan), 3TC (lamivudine, Epivir) and d4T (Stavudine) for three years. His CD4+ count was 522 cells and his viral load was less than 50 copies. While walking one day, he hurt his back; X-rays of his spine revealed a fracture in one of his back bones. This damage grew worse after a short period of time; part of his back bone began to collapse, causing severe disability and pain. Doctors prescribed the following regimen, in addition to his existing HAART regimen, to help rebuild his bones:

- Fosamax, 10 mg/day
- calcium carbonate, 500 mg/day
- vitamin D, 450 international units (IU)/day

### **Results**

Before the PHA began this treatment, bone scans found that he had severe osteoporosis in his spine. After six months of treatment, the thickness of his bones increased by about 20% and he said that his pain was “almost completely relieved.” Also after six months the PHA developed high levels of lactic acid in his blood — a side effect of anti-HIV therapy. As a result, he had to stop taking all his medications, including nutrients.

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The Italian researchers noted that doctors in Australia have recently linked the development of high levels of lactic acid to thinning bones in PHAs. As well, the Italian researchers hope that their favourable report about Fosamax encourages other teams to study the safety and effectiveness of this drug in people using HAART.

For background information on bone health and lactic acidosis see *TreatmentUpdate 117*.

REFERENCE

1. Guaraldi G, Ventura P, Albuzza M, et al. Alendronate treatment for osteoporosis in patients infected with Human Immunodeficiency Virus. *Clinical Infectious Diseases* 2001;33:414-415.

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### Disclaimer

**Decisions about particular medical treatments should always be made in consultation with a qualified medical practitioner knowledgeable about HIV-related illness and the treatments in question.**

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