Resistence to Anti-HIV Medications

Metabolic Toxicities and HIV
Of Birth and Dying
HIV Treatment Series
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World AIDS 2002: On Condoms and Needle Exchange

**Russia**

Nowhere in the world is HIV spreading faster than in the former Soviet Union, an ominous trend that has so far been driven almost exclusively by the young embracing drug experimentation. And yet Russia has devoted little if any attention to the prevention and treatment of drug abuse. Ninety percent of Russians infected by HIV are intravenous drug users. Researchers estimate that two out of every five intravenous drug users already are infected with HIV, said Andrei Kozlov, one of Russia’s leading AIDS researchers.

Other former Soviet republics faced with rapid increases in the spread of HIV have begun methadone programs to help reduce the incidence of intravenous drug use. In Russia, however, methadone is illegal. Needle exchange programs benefit about 5 percent of the country’s intravenous drug users, said one expert. But to make a significant dent in the spread of HIV among that group, at least 60 percent coverage is needed.

**India**

A condom festival was launched in the southern Indian state of Andhra Pradesh as part of an HIV/AIDS prevention program. According to a National AIDS Control Organization study, condom usage in Andhra Pradesh is the lowest in the country. One of the reasons for the low condom use is said to be the high rate of sterilization in the state. “People may think that because they have undergone sterilization, there is no need for condoms,” said Dr. Kadambari, head of Andhra Pradesh’s HIV/AIDS program. Andhra Pradesh’s Health Minister N. Janardhan Reddy added, “Because they are not following safe sex measures, AIDS is spreading. So we want to promote the use of condoms.”

**Vietnam**

Vietnam has unveiled a plan to stem escalating HIV infection rates by boosting condom usage among its sexually active younger population. The campaign aims to sell 170 million condoms each year through 2005, said Phan Song, director of Vietnam’s Family Planning Association. Many Vietnamese men do not use condoms, primarily because of ignorance about HIV/AIDS/STDs. Embarrassment about buying contraceptives in Vietnam’s deeply traditional society also remains a factor, sociologists say. “This campaign not only aims to serve family planning issues but more importantly is to protect people against HIV/AIDS, given that 58.7 percent of HIV carriers in Vietnam are aged between 13 and 29,” Song said.

**Dr. Joseph O’Neill, Director of the White House Office of National AIDS Policy at the United States Conference on AIDS, September 2002**

*On condom use and “abstinence only education”:

“This epidemic is spread by many human behaviors. That is not a moral statement. It is a statement of fact. We know there is a strong correlation between the number of sexual partners and risk for HIV. Reducing that number to one life partner is the safest of all. This is an extremely important message, especially for young people, gay or straight.”

“An approach that says the only human behavior that matters is condom use is wrong—it is medically wrong. Bringing the abstinence voice into the discussion is helpful, and the right thing to do. A clear unambiguous message to our young people that making the right choice with their bodies is a message worth giving.”

*On needle exchange:

“The administration’s position on this is clear, as was the case in the last administration, which did continued on page 45
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FTC is coming

The manufacturer of Coviracil (generic name emtricitabine, also known as FTC) has applied for a New Drug Application from the U.S. Food and Drug Administration (FDA). The new HIV drug is expected to be approved in early 2003. Coviracil is much like Epivir (lamivudine, 3TC), which is easy to remember when you think of the names, FTC and 3TC. They both fight hepatitis B virus and both select for the M184 resistance mutation of HIV. Therefore, Coviracil is not expected to be potent for people who’ve already taken Epivir—a significant number of the people who have ever been on HIV therapy. HIV specialists say the role of Coviracil will depend on only one thing: its price.

In one large study, the two drugs were highly effective in suppressing viral load and viral rebound at 48 weeks. In another large study comparing Coviracil to Zerit (d4T, stavudine), Coviracil was more effective and less toxic (as reported in the September/October issue; see “The Buzz”). Both meds were taken with Videx (ddl) and Sustiva (efavirenz). Coviracil is an experimental once-a-day nucleoside reverse transcriptase inhibitor (NRTI), also known as nucleoside analog, or “nuke” for short. This is the same class of drugs as Retrovir, Zerit, Epivir, Combivir, etc.

Coviracil’s strength and once-a-day dosing makes it an important candidate. The Coviracil/Videx-EC/Sustiva combo—taken once a day at bedtime (seven pills)—kept 85% of participants undetectable for up to two years (34/40 people). In fact, 80% of the 40 persons were below 50 copies. Moreover, eight of the nine who started out with more than 100,000 viral load were under 400 for the two years (but you would expect that with Sustiva). The median T-cell increase was 272 (half had more, half had less of an increase than this). Remember also that Epivir is now available as a once-daily drug as well. Reported side effects of Coviracil are central nervous system (CNS) symptoms, diarrhea and rash. In one small study, these side effects occurred in 73%, 37% and 10% of the participants, respectively. Elevated transaminases (an indicator of liver function) have been noted.

An expanded access program, which provides the drug free to people in great need of a new option, is now open, but only for people who are experiencing failure with Zerit or Retrovir (AZT, zidovudine), or who cannot tolerate those drugs. It is also available to people who are on Coviracil as part of a clinical trial. The program closes when the drug comes to market.

T-20 is coming too

Fuzeon (generic name enfuvirtide, also known as T-20) is also expected to come to market in early 2003. Fuzeon (pronounced fuse-ee-on) is exciting because it’s in a new class of HIV drugs, so it’s successful with people who’ve already taken many antiviral meds. As a fusion inhibitor (hence the brand name), Fuzeon blocks HIV from fusing to T-cells. However, it’s taken as a self-administered twice-daily subcutaneous injection. So far, it seems that the only side effects are mild irritations at the site of injection. Moreover, T-20 was only studied with an “optimized background” of meds, and many people with advanced disease do not have another viable drug they can add on with Fuzeon. Still, it should be a boon to those with great need (low T-cells or high viral load), while someone who can wait for new options—such as a new drug to add on with Fuzeon—may choose to do so. An expanded access program was due to open in October to offer the drug to 600 people in the U.S., out of 1,200 worldwide. Participants must have less than 100 T-cells and more than 10,000 viral load, and be unable to use an effective combination from the drugs currently on the market. Because the drug is difficult and time-consuming to make, supplies will be limited, even when it’s for sale. Physicians can visit http://www.T20EAP.com for more information on the U.S. program and to register to participate in the program. At press time,
problems with forcing prescribers to attend meetings before signing up patients were being worked out. Still, some training might be a good thing—it takes 30 minutes to mix the solution for injection, a serious hassle for patients (although they can mix both injections at the same time). According to a press release from Fuzeon’s manufacturer, “…adverse events occurring in more than 10 percent of the patients and occurring more frequently in the Fuzeon group were fatigue, insomnia, and peripheral neuropathy [but did not lead to drop outs from the study]. It was not possible to establish a causal relationship between these other adverse events and Fuzeon. In the first Phase III study, the incidence of Grade 3 and 4 laboratory abnormalities was similar between the Fuzeon and control arms. In the second, Grade 3 laboratory abnormalities were more frequent in the Fuzeon group, and Grade 4 laboratory abnormalities were more frequent in the control group.”

THE AIDS VACCINE—FOR CATS

A long-term study of children born to HIV-positive women found they have a heart abnormality, regardless of whether they are infected or not. According to a report by Dr. Brian Boyle for www.hivandhepatitis.com, “Study analysis showed that HIV-1 infected children had a statistically significant higher heart rate at all ages. In addition, all children born to HIV-1 infected women had a low left ventricular (LV) fractional shortening at birth, which improved in the uninfected children by age 8 months but not quite up to the normal level as seen in children in the control group… Based upon the results of the current cohort study, the researchers conclude: ‘Irrespective of their HIV-1 status, infants born to women infected with HIV-1 have significantly worse cardiac function than other infants, suggesting that the uterine environment has an important role in postnatal cardiovascular abnormalities.’ The researchers also suggest that appropriate treatment strategies should be considered for all children born to women infected with HIV-1 as even mild LV dysfunction has shown to effect mortality over time.”

The study by the National Institutes of Health (NIH) evaluated 556 children, of whom 93 were HIV-positive. For comparison, the NIH researchers also looked at 195 healthy children born to HIV-negative women. The findings were published in The Lancet medical journal last summer.

SUSTIVA LABEL CHANGE

In light of controversy over HIV-positive inmates on Sustiva, written about in Positively Aware, a recent change in the drug’s label now clarifies the interaction between the antiviral and marijuana tests. The label now makes it clear that other drug tests besides the ones listed may also give false positive results. At any rate, prison authorities are still required to run confirmatory tests, which they often refuse to do. See “Sustiva ‘Dirty Drops’ Put Prisoners in Solitary,” Nov./Dec. 2001.

WOMEN AND NON-NUKES

A report in the medical journal AIDS reminds us that the risk of rash from the non-nucleoside analogs (Rescriptor, Sustiva and Viramune) is greater for women. Rash represents an allergic reaction that in the most serious cases can lead to blindness, hospitalization and even death. The report notes that almost one in five people reported rash in studies that brought Sustiva and Viramune to market. A look at the chart records of 337 people on these two drugs found that women were five times as likely to experience rash (14.6% vs. 3% for the men). The Viramune was dose-escalated for all patients (to avoid rash).

PAN AFRICAN MOVEMENT

AIDS activists in South Africa have had successes in legal battles, media campaigns and attaining access to medications. In August the South Africans joined AIDS
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activists from across the continent to work together to fight the epidemic there. It is estimated that 22 of the 35 million people living with HIV worldwide are from the African continent. Zackie Achmat of South Africa’s Treatment Action Campaign told the Associated Press that, “All of us are trying to prevent a holocaust against poor people. HIV shows that the rich will live, but the poor will die.”

Lesbian HIV risk
Susan Scheer and colleagues remind us that many self-identified lesbians also have sex with men or engage in drug use that could lead to HIV infection. They note that, “While unknown, the biological risk of female-to-female sexually transmitted HIV is thought to be much lower than the risk of transmission between men and women. Yet studies have shown that some subgroups of women who have sex with women (WSW) exhibit high levels of sexual risk behaviors with men as well as unsafe injection drug use. Thus, if risk assumptions are based on self-reported or presumed sexual identity, possible risks for HIV infection may be underestimated in some subgroups of WSW.” The study was published in the American Journal of Public Health.

Nandrolone available
AIDS Treatment News reports that generic nandrolone has newly become available at some pharmacies. “The Schein brand was found in a New York pharmacy at a retail price of $13 per vial (1 ml, 200 mg/ml). This is close to the old generic price. There may be more information on the nandrolone situation at www.medibolics.com or at www.houstonbuyersclub.com,” reports the newsletter. Nandrolone, used for fighting AIDS related wasting, was also sold under the brand name Deca Durabolin, which is no longer on the market.

Flu shots
Don’t forget your flu shots. According to the Mayo Clinic, each year the flu kills as many people in the United States as does AIDS, 20,000. The U.S. Centers for Disease Control and Prevention recommends shots for infants from 6 to 23 months and their household contacts, for everyone 50 or older and for everyone older than six months with a chronic illness—such as HIV. Still, you must talk with your doctor to see if a flu shot is recommended for you.

ICAAC News

Information from the 42nd Interscience Conference on Antimicrobial Agents and Chemotherapy, held in September in San Diego.

Taz vs. Sustiva
The experimental once-a-day protease inhibitor atazanavir (Zriva) did well against the popular non-nucleoside Sustiva. These were preliminary results (six months) in a large clinical trial with people on HIV therapy for the first time. Atazanavir, or Taz for short, did not increase lipid levels as seen with many of the PIs on the market, or showed only slight increases. Sustiva and Taz were taken with Combivir (a combination of Retrovir and Epivir in one tablet).

Doctors couldn’t believe how poorly Sustiva did in this study, especially at under 50 viral load. It has performed significantly better in every other study it’s been in. All kinds of explanations were raised. The conference presenter said you can’t compare across studies, to which one doctor replied, “let’s say it’s concerning.” The researchers also tried to point the finger at different tests that were used during the study, instead of the same one for the same purpose each time. Some doctors raised the issue of reactions from different types of HIV, given that this was an international study with many participants from the developing world. All in all, as one doctor pointed out, the study was likely to be internally consistent. Therefore, if Sustiva underperformed, so did Taz.

Kaletra plus boosted Agenerase
Earlier this year there were concerns about the finding that Kaletra lowers blood levels of Agenerase. That’s not good. Here, researchers looked at saving the combination by boosting levels of Agenerase with a little bit of another protease inhibitor, Norvir—even though Kaletra already has Norvir in it. (Kaletra is a combination of the protease inhibitor lopinavir with a mini-dose of Norvir in each capsule.) The people with the additional 200 mg of Norvir had a significantly greater drop in viral load (2.5 vs. 1.4 logs) and were much more likely to be under 50 viral load. Moreover, people did well with or without extra Norvir despite having previously taken several HIV drugs and having lots of drug resistance.

Still, these are preliminary results in a small number of people (37 participants at 26 weeks). Also, the blood levels were not given. And while side effects were not detailed, adding more Norvir increases the risk of diarrhea and other gastrointestinal problems.

Sustiva vs. Viramune
Sustiva (efavirenz) and Viramune (nevirapine) are both in the same class of HIV drug, the non-nucleoside analogs (or non-nukes for short). Viramune, which came to market before Sustiva, never went head-to-head with Sustiva in a large trial. As Sustiva got ready to hit the market later, its manufacturer took on the big guns, the protease inhibitors like Crixivan and Viracept—and won every time. So the data was never clear on whether the two non-nukes are equal. Only limited data suggests that they are, and doctors have their own preferences about which drug to prescribe.

At this conference, a multi-center study from Portugal and France suggests that the two are equivalent in keeping viral loads undetectable when switching people off long term use of protease inhibitors. This was after a significant amount of time, a year and a half. Of people put on Viramune, 88.5% were below 400 copies viral load, vs. 94.4% of the people switched to Sustiva. This difference was not statistically significant. T-cells went up by 124 for the Viramune group, and down 17 for the Sustiva folks, but this was also not statistically significant (due to the small numbers of people enrolled).

However, discontinuation for any reason was relatively high for both groups: 13 of the 76 people on Viramune (17.3%) vs. 13 of
the 54 people on Sustiva (24.5%), also not statistically significant.

**Kaletra/Sustiva**

French researchers reported that the nucleoside analogs are “increasingly recognized as a cause of mitochondrial toxicity, hyperlactatemia and hepatic steatosis [fatty liver]. In addition, cross-resistance within the [nuke] class is associated with difficulties for HAART sequencing [highly active antiretroviral therapy].” In a small study, they took 49 people who’ve never been on HIV therapy and five people with some treatment experience, and put them on the protease inhibitor Kaletra plus the non-nucleoside analog Sustiva, with no nucleosides.

Nearly half of the people were undetectable after a month (less than 400 viral load), but almost all were undetectable after six months. T-cells were up by 160. This was under an intent-to-treat analysis, a strict formula that takes into account all study participants, even if they drop out. (Hence, it imitates what happens in the real world.)

Serious side effects (greater than grade 2) occurred in 32 participants (59%). This included central nervous system (CNS) side effects (11 participants), rash (3), diarrhea (7), nausea (2) and vomiting (1). There were also high cholesterol levels in seven people, high triglycerides in four and liver problems in one. Seven participants discontinued the study: two had CNS side effects, one had rash, one had itchy skin and another stopped because of increased lipid levels. Two were non-adherent or lost to follow-up. The study will continue for a year. The researchers said that in these preliminary results, the combination of Kaletra and Sustiva is a strong one with acceptable tolerability.

**Triple nukes**

A triple nuke combination with Ziagen did not hold up against a protease inhibitor or non-nuke combination.

Danish researchers noted that Ziagen (abacavir, ABC) is “usually included in triple NRTI [nuke] regimens along with zidovudine [AZT, Retrovir] and lamivudine [3TC, Epivir] despite the cross-resistance observed between ABC and the other two drugs. (In addition,) the potency of the regimen has been questioned.” (Ziagen is available in combination with the two drugs in one tablet, called Trizivir, making it a convenient triple combination with only one medication.)

Here, the researchers gave Ziagen in combination with stavudine (Zerit, d4T) and ddI (Videx). The regimen was compared to the dual protease inhibitor combination of Norvir (ritonavir)/Fortovase (saquinavir), plus Retrovir and Epivir (the N/F combo). The dual PIs were given as 400 mg each twice a day. Participants in a third arm of the study used a PI/non-nuke combo consisting of Viracept (nelfinavir)/Viramune (nevirapine) plus Retrovir/Epivir (the V/V combination).

After 48 weeks, the Ziagen group had statistically significant fewer people under 20 viral load than did N/F or V/V (41%, 56%, 66%). Also, significantly more people discontinued any of their study drugs when compared to V/V (63% vs. 45%), but not when compared to N/F (56%). (Still, that’s almost half of the people on the V/V arm who changed or dropped a med.) There were about 60 people in each of the three arms, all of them on HIV therapy for the first time. All in all, the researchers said that the combination of Ziagen/Zerit/Videx had more toxicity and less potency than the other two arms, and “cannot be recommended.”

**Kaletra after 4 years**

After four years, 72 out of 100 study participants still on Kaletra/Zerit/Epivir are doing well. Sixty-five have less than 400 viral load. Another seven are below 50 despite having gone above 400 at one point during the trial. However, 20 people left before the four years were up despite having less than 400 viral load. Average T-cells went from 281 to 721. Even people with less than 50 T-cells went from an average of 23 to 446. The most common side effects were diarrhea, nausea and abdominal pain. Participants were on HIV therapy for the first time. Results use a strict intent-to-treat analysis where missing participants or information = failure.

**Agenerase pro-drug**

Preliminary results from advanced research (Phase III) were presented for the new formulation of Agenerase (amprenavir) protease inhibitor. A new formula was needed because Agenerase is taken as eight horse pills twice a day. It therefore is more popular when taken with a mini-dose of Norvir to boost its blood levels while at the same time cutting down its dosage. In this study, the pro-drug, GW433908 (908 for short), outperformed Viracept protease inhibitor at 24 weeks. That’s not surprising, but the point is that 908 works well with few side effects and at a smaller dose—two pills twice a day. That’s a welcome change that’s been waited for. Moreover, it’s expected to do well as a once-a-day drug, boosted by Norvir or Rescriptor. Of the 908 group, 73% had less than 200 viral load compared to 54% of the Viracept group. For less than 50 viral load, the numbers were 54% vs. 40% for Viracept.

A strict intent-to-treat analysis was used. There were no changes in the median levels of triglycerides or cholesterol. Everyone was taking HIV medications for the first time, and would be expected to do well. A significant number of them, 40%, had more than 100,000 viral load when they entered the study. Of these, the numbers reaching less than 200 viral load were 74% vs. 35% of the Viracept group. For under 50 viral load, the figures were 42% vs. 11%. Another significant difference in this study: 31% of the participants enrolled were women, and 75% were Latinos or of African descent. Those are numbers much higher than you normally see. The most common side effects with 908 were allergy (8%), rash (8%) and nausea (5%). Discontinuation because of adverse events was 5% for 908 and 6% for Viracept. Discontinuation for any reason was 19% for 908 and 28% for Viracept. The drugs were taken as a triple combination with Ziagen and Epivir. ☂
With that fateful smack, to which I attribute my attraction to the slightly kinky, I was born in January 1971, in Williamsburg, Virginia. I was only 11 years old when Dan Rather was talking about “Gay Cancer” and “GRID” on the evening news. It was Dan’s hair, more than this strange disease, which troubled me. A sweet little well-bred Southern boy, I graduated high school in 1989. My aspirations took me to the grand old city of Richmond, where I graduated, with honors (thank you very much), from university in 1993, the same year that I came out to my family as being a full-fledged homosexual. Two points away from receiving my Gay Card from the Association, I was well on my way to true Queer Enlightenment, a status envied by all the closeted conservatives of blue-blooded aristocracy that bog down the East Coast. I envisioned my royal birth parents reclaiming their only child, me, and returning me to the throne before the age of 30. Everything was coming up roses I believed that I was part of the “new generation” in the AIDS epidemic: the safe generation. I was of the generation that was educated on the differences between safer sex and high-risk sexual behaviors. My generation remained for the most part untouched by the epidemic, or so it seemed to me in my safe little world. Relatively speaking, it was “older men” who were infected, not younger men, not us and certainly not I. Throughout my college years, to the best of my knowledge, I never encountered anyone with HIV or who had died of AIDS. Somehow, my generation had been granted a kind of immunity. And because we were immune, we were a generation that did not need to worry about the disease ravaging the world.

Market Days, Chicago, 1997
Still in pursuit of my Gay Card, I moved to Chicago in 1995. I had grown tired of the stifling conservativeness of Virginia. I wanted to experience a big fabulous city. San Francisco, I feared, would have eaten me alive. New York was too crowded, too many actors. I picked Chicago for its cost of living, public transportation, bountiful theatre opportunities, and world-famous Boystown. And I began my “hoochie” period.

Halsted Street Market Days ’97, in Boystown, was the peak of my hoochie days. I had the best haircut I have ever had, a couple of curls lazily drooping near my eyes. Working my “daisy dukes” and tank top, cute little shoes from Payless. I was flawless.

I remember it started raining on the last hot and humid day. The rain felt so good. Tents were being taken down, tables collapsed and chairs folded. I was busy hitting the 7-11, in search of sweet cherry cigars and 40-ounces, all the while checking out the boys in the rain. Caught up in the sexiness of the atmosphere, I went home with a group of guys I didn’t even know. The rest, as they say, is history. A few weeks later I attended my brother’s wedding and then came down with what felt like a really bad flu. First, I was diagnosed with German measles. Huh? I had been vaccinated for measles, so what the fuck? Then it was mono. I’d had mono before, this was no mono. Bedridden for over a month, I started to wake up. The swollen glands. The fatigue. Then reality set in. No generation gap was going to keep me safe. No age range would keep a virus at bay. No ignorance that I hid behind would barricade my blood. I was diagnosed with HIV in February 1998. I was 27 years old.

I was in shock for months after finding out I had become infected with the virus that causes AIDS. The virus that causes AIDS. I said it over and over. The virus that causes AIDS. The virus to which I thought my generation was immune. I became paralyzed with fear, grief and confusion. I had no excuse for getting infected. I was stupid to
have gotten infected, careless to have gotten infected. I suddenly heard a clock that was ticking for me, I didn't have time for some fucking disease. One day, while standing on a street corner near Wrigley Field, I stopped and dropped what I was carrying. I began to cry. I stumbled around, lost. I finally sat on the sidewalk and just cried, uncontrollably. The dam had finally broken. That night I called a hotline and got in touch with a psychotherapist.

I hadn't just been a socially irresponsible hoochie. Before getting sick and being diagnosed HIV-positive, I had signed up to do the AIDS Ride. While I thought my generation was protected from HIV, I was conscious of a moral and social responsibility that my "protected" generation shared with others. I was in tune to the bigger picture. I wanted to do the ride to help make a difference in the lives of others. How the fuck had I become one of those lives? I continued training for the ride and I did it. I rode 500 miles. It turned out to be the only thing that carried me through those first months of total shock.

After the ride, I started to share my burden, lighten my load. I began to find my support systems. I started telling friends, one at a time. For many of my friends I was the first person that they knew of to be HIV-positive. I quit smoking, stopped drinking alcohol and coffee. I became the Crixivan and Combivir Caped Crusader, being very good and coffee. I became the Crixivan and Combivir Caped Crusader, being very god about my drug regimen, watching my diet and exercising. I became more and more open about being positive and buried myself in research about the disease and treatments. I took up Reiki, visual imagery and massage therapy. I went home to Virginia and told my family that I was living with HIV. I was making progress with my therapist. And I did the AIDS Ride again. I was regaining control of my life.

That process went on for nearly three years. Healing. Trying to make a difference, not only in my life but in the lives of others. I was rushing from massage appointments to educational expos on “Living with HIV.” Going to fundraisers. Hoping that Jerry Farwell and Reverend Felch (or is it Phelps) would get struck by lightening. I would linger over lunch telling yet another person about my life now and what my pill regimen was like. My T-cells were climbing, my viral load tittering around the undetectable zone, and my belly getting bigger.

Eventually, after disappearing into books, magazines, massage and reiki sessions, my bike and everything that kept me busy, I found myself too tired to give a shit about anything. I was keeping so busy that I never “checked in” with myself. Flip. 180. I quickly found myself depressed, canceling vacations, turning down invitations, oversleeping, over-eating, avoiding friends, drinking, smoking and letting my life just happen without me truly being present. I felt like I was running around in circles while standing still. I wasn’t getting anywhere. What the hell was going on?

The Screaming Room, a mother’s journal of her son’s struggle with AIDS, made me realize I was guilty of the same ignorance I had accused my father of. In this book, Peter Peabody fights for his life in the early 1980’s. Sadly, it seems needless to say that Peter did not survive. When my doctor first told me, “This is not a death sentence anymore, you could live a long time,” I didn’t hear him. I imagined, I assumed, that I would die, no matter what anyone told me, just as Peter did in the book. I would waste away. Why? Because that is what I heard so many times, it was the only “outcome” I knew of for people with AIDS. My dad, too, thought I would most likely endure the “uncomfortable death.” Finally it hit me. We both were ignorant. I hadn’t realized it until I read that book. We don’t know the outcome. It’s a whole new chapter. Even though I am a part of this new generation, the generation that has anti-HIV medicines, I don’t know how or when I’ll die. Times are different. There is no one certain outcome, not anymore.

In all the reading I did I was stunned that another aspect of this “new generation” was the disappearance of so many advocates, so many activists. Some had died. Some had been sick but then gotten better and needed to move on with their own lives. Some were just burnt out and needed to take a rest, regain their spirits. But I wondered, where the fuck were the replacement troops? Why so much complacency from people, well, people like me? I was of a generation that just sat there and reaped the benefits from the sweat, tears, shouts and deaths of men and women they’d never met. Christ, I was exhausted from “making the most” of every day. What a privilege. What is my generation doing now?

While I have tried my own little outreach project by telling guys who want to go down on me that I am HIV-positive, for the most part I see my generation has not changed. Just like I didn’t want to, no one wants to admit to himself or herself that they are practicing risky sexual behaviors. No one wants to hear that oral sex without a condom is unsafe. For most guys about to give me head, licking my lollipop suddenly is not so appealing, when I tell them I have HIV. Some guys surprise me and whip out a flavored condom, which, however, is the exception. Some guys say they don’t care. Sometimes it leads to a discussion or an argument. On a couple of occasions it has led to friendships.

But what is my generation doing? What am I doing? The AIDS Ride? While it’s looking more and more like a big old scandal, for me, personally, it was a very positive, life affirming event—and the awareness that it spread is extremely important—and I know dollars (no matter how few) that otherwise might not have been available were sent to HIV/AIDS organizations.

Still, I ask myself: What did I accomplish? I rode my bike for six days. I inspired some other people. I made some incredible friends who I will always treasure. I used it as an opportunity to spread my story, which had positive ramifications for others and me. I learned that one person can make a difference. All of that is great and I am thankful for it all. But I can do more. What next? What do I do now? I want to be an activist, I want to protest, I want to scream and be heard.

What’s this generation going to do now?
In an energetic, post-ICAAC presentation, Frank Palella, MD, urged people living with HIV and clinicians who work with them to put some of the adverse effects of antiretroviral treatment into a larger perspective. The point of departure for Dr. Palella’s talk was the growing concern in the HIV community about metabolic toxicities and body-shape changes (sometimes called “body habitus changes”) that, for many people, are associated with use of some antiretroviral (ARV) drugs.

Perhaps the most distressing concerns for many individuals arise from the visible, cosmetic changes that many begin to experience after starting highly active antiretroviral therapy (HAART). These changes include loss of facial fat, leading to the appearance of sunken cheeks; accumulation of fat around the waistline; buildup of fat at the back of the neck between the shoulder blades (“buffalo hump”); loss of fat in the arms and legs; and thickening of the neck. Although for many people, these changes in appearance can be quite stressful, they also represent just the tip of the iceberg of metabolic issues and other ARV treatment matters that HIV specialists are trying to address.

Today’s wide-ranging debates about metabolic toxicities and body-shape changes, explained Dr. Palella, who is assistant professor of medicine at Northwestern University School of Medicine, are taking place in the context of another critical debate—when to start HAART and what combination of drugs to start with. Earlier recommendations to start ARV therapy for almost everyone when the CD4 T-cell count reached 500 or to “hit early and hit hard” have now been replaced by more complex considerations of issues like possible long-term toxicities, quality of life, and so forth. Some HIV specialists are even recommending delaying the start of ARV therapy until the CD4 count reaches 200. Indeed, one reason for this recommendation is to avoid what some believe to be long-term toxicities of ARV treatment, such as metabolic abnormalities and body-shape changes.

Critical data from HOPS

To help explain possible causes of lipid abnormalities and body-shape changes, as well as what they may imply for an individual’s ARV therapy, Dr. Palella drew on the vast amount of data coming from the HIV Outpatient Study (HOPS). HOPS is a huge study of some 7,000 HIV patients at 10 key treatment centers across the United States. (The largest of the centers is at Chicago’s Northwestern Memorial Hospital.) The study collects and analyzes data from these patients’ specific treatment regimens, outcomes, adverse events, survival rates, and other information.*

The following are just some of the key issues that the HOPS data are helping Dr. Palella and other researchers address:

• What are all of the factors, besides possible adverse drug events, that are associated with lipid abnormalities and body-shape changes?

• Can a single definition of lipodystrophy be agreed upon?

• Is HAART associated with reduced mortality and morbidity?

Using a variety of graphs and charts, Dr. Palella stressed that use of HAART, which began in early 1996 with the introduction of protease inhibitors (PIs), has a clear association with a dramatic drop in the number of deaths from HIV-related illnesses. In fact, for people who are able to take HAART, the number of deaths plunged by 90–95 percent. He also pointed out that during that same time the rates of illness due to *pneumocystis carinii* pneumonia, *mycobacterium avium* complex, and cytomegalovirus have also greatly declined. Furthermore, the HOPS data indicate that earlier initiation of ARV therapy is highly beneficial: People who began therapy with CD4 counts between 200 and 350 cells, as opposed to delaying therapy until below 200, had a five-times lower death rate. In addition, those
who started therapy at between 350 and 500 CD4 cells had almost half the death rate as those who delayed starting therapy.

Dr. Palella urged that, for those wondering whether use of HAART is really a good idea, “we have to have a long memory, because prior to 1996 AIDS was the leading cause of death for men between the ages of 20 and 60 and the second leading cause of death for women. But today the death rate has been dramatically reduced. So, when talking about lipoatrophy and other metabolic effects—even if we believe that adverse drug effects play a role—we have to keep in mind what the alternative to not treating is.”

Among the arguments in favor of starting ARV therapy at a higher CD4 count Dr. Palella pointed to the following:

- Control of HIV replication is easier to achieve and maintain.
- Immune deficiency is delayed or prevented.
- The risk of developing drug-resistant virus is lower.
- The risk of HIV transmission may be decreased.
- Adverse events, like metabolic abnormalities, may be more likely in people with lower CD4 counts.

Whether delaying therapy helps to avoid cumulative adverse events represents the “black hole that all of the lipid abnormality issues fall into,” stated Palella. “This is because we do not have a complete understanding of the extent to which the drugs that we use in fact contribute to what are called ‘drug-related adverse events.’”

First he sketched out some of the issues being addressed in trying to define lipodystrophy. Four general sets of abnormalities, which overlap with each other to varying degrees, come into play in any working definition of lipodystrophy:

- Blood lipid abnormalities—high total cholesterol, high low-density lipoprotein (LDL or “bad” cholesterol), and high triglycerides.
- Fat accumulation—around the waist, between the shoulders (buffalo hump), and around the neck.
- Lipoatrophy—fat loss, especially in the face, arms, and legs.
- Dysregulation of glucose metabolism—meaning problems in processing sugars, possibly leading to insulin resistance or even diabetes.

Dr. Palella explained that these abnormalities can occur in different combinations in different individuals. For example, one person may experience high cholesterol and loss of facial fat, but none of the others. Another person may have insulin resistance and abdominal fat accumulation, while a third may experience some degree of all four types of abnormality.

As early as 1996, reports began to appear of diabetic problems occurring in some patients who were taking protease inhibitors, so the concern about metabolic abnormalities in HIV treatment is not new. Because virtually all HAART regimens at that time contained at least one PI, that class of drugs was thought to be the most likely cause of the abnormalities. With a variety of failed efforts to explain what the association could be, Dr. Palella said, “attention shifted to the nucleoside analog class of drugs [Zerit, Retrovir, Videx, Epivir, Comvir, etc.], and that is where it has stayed for the last couple of years, especially when talking about fat atrophy.”

Now that analysis of a variety of factors associated with different manifestations of metabolic abnormalities has become available, for example from the HOPS data, some things have become clearer. “While some drugs themselves might account for certain aspects of these problems, especially the association of blood lipid abnormalities with use of protease inhibitors, a close association of one drug and the development of one of these abnormalities does not imply that there is any association with development of any of the other abnormalities.”

Dr. Palella went on to explain that HOPS investigators looked at data from about 1,100 patients in the study to try to uncover two things:

- The prevalence of either fat gain or loss
- What factors, either drug-related or non-drug-related, might be associated with fat changes.

### MULTIPLE FACTORS LINKED TO FAT CHANGES

After this explanation of how HOPS information clarifies the relationship between use of HAART and the drop in HIV-related death and disease, Dr. Palella focused on how careful analysis of HOPS, and other, data have begun to shed light on the host of factors that seem to be associated with the development of metabolic and body-shape problems with HIV infection.

...we do not have a complete understanding of the extent to which the drugs that we use in fact contribute to what are called ‘drug-related adverse events.’”
HOPS investigators identified the following associations:

- Having an AIDS diagnosis for longer than three years.
- Being older than 40.
- Having a low CD4 T-cell count.
- Being Caucasian and male.
- Fat loss of more than one kilogram (2.2 pounds) per square meter of body area.

The two medications most associated with these abnormalities were Crixivan and Zerit. However, Dr. Palella stressed that real accountability for the occurrence of the problems could not be assigned to either drug because these were the most commonly used medications among that group, with more than 85 percent of them being treated with Zerit. He compared this situation to “walking into a room full of dark-skinned people who are all HIV-positive and saying that they’re positive because they’re dark-skinned. Of course, you can’t say that, because the real reason is that these just happen to be the people that you’re looking at now.”

Another later analysis of HOPS data found an association between an individual’s CD4 nadir (the lowest level that her or his count had reached) and the development of facial fat loss. This analysis found that the lower a person’s CD4 count got, the higher or his count had reached) and the development of facial fat loss.

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No retreat on treatment progress

To drive home his core message that patients and providers alike should keep their attention more on the larger context of HIV treatment, rather than on possible adverse events, Dr. Palella suggested an analogy: “If we were talking about a cancer, like small-cell carcinoma of the lung, and we had combination chemotherapy that could result in durable remission—with more than 90 percent reduction of death rates—but the therapy had to be continued for many years. And if we found some metabolic abnormalities that may or may not be due in part to use of the drugs, would that ever be considered justification for delaying or modifying what is known to be life-saving therapy? Absolutely not. We should never think that withholding therapy for anybody at risk for HIV disease progression is something that is admissible. Especially since the majority of metabolic changes described here, like high cholesterol, insulin resistance, and high triglycerides, are treatable. People who are taking ARV therapy are living their lives, holding down jobs, raising families, and so on. Therapy is saving lives that we could not save a decade ago, and I don’t want to go back to that time again.”

Steve McGuire is a Chicago-based writer and consultant specializing in medicine, public policy, and non-profit issues.

* Much of the credit for analyzing the HOPS data belongs to Kenneth Lichtenstein, MD, of the University of Colorado, who is one of the lead HOPS investigators.
In 1999, Congress passed and the President signed the Ticket to Work and Work Incentives Improvement Act. Through this Act, the Social Security Administration (SSA) created a new system of vocational rehabilitation services for beneficiaries of Social Security. This new system is called the Ticket to Work Program. In order to access this new system in a meaningful way, it is important for beneficiaries of Social Security to understand the purposes of this new system, how this system works, and a beneficiary’s rights under the system. This article will spell out this important information so that any Social Security beneficiary, including those living with HIV/AIDS, interested in working can make an informed choice about participating in this new program.

The Purposes Behind the Ticket to Work Program

The purpose of the Ticket to Work Program is not to force individuals receiving Social Security into the workforce. Rather, this program represents a serious attempt by the Social Security Administration to provide meaningful assistance to those beneficiaries interested in finding employment. This is one of the main reasons why individuals who identify with the disability rights community (including individuals from the HIV/AIDS service community) have supported this legislation and have been heavily involved in its implementation.

Another purpose of this legislation is to save the federal government money. According to the U.S. General Accounting Office, less than one percent of Social Security beneficiaries leave the rolls each year as a result of employment. If another one-half of one percent of those receiving Social Security were to leave the rolls, as a result of paid employment, the federal government would save $3.5 billion over the work-life of those individuals. Through the successful employment placement of beneficiaries utilizing this new Ticket Program, the federal government hopes to realize these savings.

How the Ticket to Work Program Works

Under the Ticket to Work Program, most beneficiaries of Social Security will receive a document in the mail called a “ticket.” This ticket enables the beneficiary to seek employment services from entities designated by Social Security as Employment Networks (EN). The medical conditions of all beneficiaries of Social Security are given the designation of medical improvement expected, medical improvement possible, or medical improvement not expected. Beneficiaries whose condition is designated as medical improvement expected will only receive a ticket after they have had a continuing disability review (CDR) and been determined still disabled. Most, if not all, individuals receiving Social Security due to HIV-related illness have had their conditions designated as medical improvement not expected and will receive tickets.

If an EN accepts a beneficiary’s ticket, the EN will assist that individual in finding employment. This program is entirely voluntary. A beneficiary can choose whether or not to participate in this program. This is not an attempt by the Social Security Administration to force beneficiaries to work. At the same time, an EN network can choose whether or not it wants to work with a beneficiary on finding employment. An EN cannot be forced to assist a beneficiary who requests services. (The one exception to this rule involves the state vocational rehabilitation agency. These agencies must make eligibility determinations under the rules established by the Rehabilitation Act.)

The Social Security Administration has contracted with a private company, Maximus, to administer the Ticket to Work Program. Among other things, Maximus distributes the tickets, recruits employment networks, tracks the assignment of tickets, and provides referrals to beneficiaries with questions about the program. To contact Maximus, call their toll free number at (866) 968–7842.
The Ticket to Work Program is being implemented throughout the country in three phases (contact Maximus to determine which phase for your state). Those Social Security beneficiaries living in “first phase” states should have already received their tickets in the mail. Those living in “second phase” states, distribution of tickets should begin in November of this year. In third phase states, beneficiaries should receive their tickets in 2003. Tickets are distributed over a four-month period, based upon the last digit of a beneficiary’s Social Security number. However, if tickets are being distributed in a particular beneficiary’s state, that beneficiary can request his or her ticket at any time during the distribution. Maximus has been very good at responding to the requests in phase 1 states.

**Using the Ticket to Secure Meaningful Employment**

For a beneficiary of Social Security, making the decision to become employed is a difficult one. The rules on how employment income will affect public benefits are complicated and confusing. However, no beneficiary of Social Security should accept a job without understanding exactly how the income from that job will impact his or her Social Security cash benefits, Medicare, Medicaid, any housing subsidy, and state Social Security cash benefits, Medicare, income from that job without understanding exactly how the complicated and confusing. However, no ment income will affect public benefits are a difficult one. The rules on how employ-

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ENs (except the state vocational rehabilitation agency) involved in the Ticket to Work program only get paid for working with a beneficiary if that beneficiary works at an income level that eventually disqualifies him or her from receiving cash benefits from SSA. Therefore, before deciding to use a ticket, a beneficiary must decide if he or she is willing to stop receiving cash benefits from Social Security. An EN will not be interested in assisting beneficiaries who want to continue receiving any level of cash benefits from Social Security because they won’t receive compensation for working with such individuals. If a beneficiary decides that they do not want to work at a level that disqualifies them from benefits, that beneficiary should not participate in this new program. However, beneficiaries can still participate in the state vocational rehabilitation programs, which do not require that a person work at a level that disqualifies them from receiving Social Security benefits.

For most individuals on Social Security, maintaining their health insurance (Medicare or Medicaid) is their primary or only concern. There are ways to keep this insurance and work at a level where the cash benefits stop. Therefore, a person can decide to use their ticket, disqualify themselves from cash benefits, and maintain their health insurance. Again, the benefits planners can ask the staff person specific questions about their services to see if these services match the beneficiary’s employment goal. Both the beneficiary and the EN will make a decision on whether or not placing the ticket with that agency will benefit the beneficiary and the agency. Remember, both the beneficiary and the EN have the right to choose who they will accept.

If an EN and beneficiary agree to work together, the parties must create an

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Individualized Work Plan (IWP). The IWP is then submitted to Maximus. The IWP spells out exactly what the EN will do and what the beneficiary will do to reach an employment goal. It is very important that the beneficiary participate in creating this plan. The ticket is not officially placed with the EN until this IWP is submitted to Maximus. Therefore, the beneficiary has the power to reject this IWP and cannot be forced to participate in a plan. If the parties cannot agree on a plan, the beneficiary can simply stop working with that EN and search for a new EN.

**Rights and Responsibilities of the Beneficiary Under the Ticket to Work Program**

Once a beneficiary places their ticket with an EN, he or she should begin working on his or her employment goal by following the directives of the IWP. While this is being done, SSA will measure the beneficiary’s progress towards employment. During the first twenty-four months of the plan, there is no work requirement. During months 25-36, the beneficiary should earn a gross income over the substantial gainful activity amount (SGA; $780 per month in 2002) for three months. During months 37-48, the beneficiary should earn a gross income over SGA for six out of twelve months. During months 49-60 of the plan, the beneficiary should earn a gross income that disqualifies him or her from receiving SSDI or SSI.

If a beneficiary meets these goals, he or she is considered to be making “timely progress” under the program. When a beneficiary has placed the ticket and is making timely progress, SSA cannot conduct a Continuing Disability Review (CDR) of the beneficiary’s current medical condition. A CDR occurs for most beneficiaries every three or seven years. The CDR involves a request for current documentation of a beneficiary’s medical condition. Once this documentation is collected, SSA reviews the documentation to determine if the beneficiary still qualifies for Social Security payments. If a determination is made that the beneficiary is no longer eligible for Social Security, benefits cease. One of the advantages of using a ticket for a beneficiary is that he or she will not be subjected to a CDR while the ticket is in use and timely progress is being made.

If a beneficiary fails to make “timely progress,” it does not mean that he or she will be withdrawn from the Ticket to Work Program or automatically lose benefits. This is very important to understand. The Ticket to Work Program is voluntary and there is no negative consequence to an individual’s benefits if he or she tries to participate in the program and is unsuccessful. Rather, the only consequence of failing to make progress under the Ticket Program is the beneficiary will lose the CDR protection explained above. In other words, the beneficiary would be subjected to the CDR that he or she would have had anyway if he or she did not participate in the program.

If a beneficiary is dissatisfied with the services that he or she is receiving from an EN, the beneficiary has two options. First, the beneficiary can choose to utilize the dispute resolution system created by SSA. If the EN is the state vocational rehabilitation agency, the beneficiary can also file an appeal under their pre-existing system. Second, the beneficiary can pull the ticket from the EN and try to place the ticket with another EN. If the beneficiary pulls the ticket, he or she has three months to find a new EN without losing the CDR protection described above.

Any beneficiary who needs more information about this new system or who is dissatisfied with the services provided to him or her by an EN should contact the state PABSS (Protection and Advocacy for Beneficiaries of Social Security) Project. The PABSS Advocates in Illinois can be reached at 1-800-537-2632. All PABSS Projects around the country are listed at www.ssa.gov/work/ServiceProviders/PA Directory.html or can be reached by contacting the local Social Security office.

**Conclusion**

The SSA Ticket to Work Program represents an important step toward assisting beneficiaries of Social Security in securing and maintaining meaningful employment. In its present form, this system may not work for all beneficiaries of Social Security and many beneficiaries may choose not to participate in this program. However, for HIV/AIDS impacted beneficiaries who are ready to return to the workforce and forego receiving cash benefits, the Ticket Program can be a helpful means for receiving needed support and services to get there.

John Coburn is the PABSS Project Manager and staff attorney for Equip for Equality, Inc of Chicago. This article, his second for Positively Aware, is based upon a document entitled “Checklist for Using Your Ticket to Work,” developed by Coburn, Sue Augustus and Marsie Frawley of the SSI Coalition.
Hepatitis C: Be Positively Aware of the Risk of Transmission and Treatment

By Bethsheba Johnson, APRN

Hepatitis is defined as “an inflammation of the liver.” As one of the largest and most important organs in the body, the liver takes part in almost every vital process in your body. Some important functions of your liver are the following: 1) removal of toxins in your blood, which includes alcohol and medications, 2) turning digested food into nutrients, 3) storing of carbohydrates from your meals that can be used later for energy, and 4) building proteins that your body needs to clot blood, make new cells, and create necessary chemical reactions inside cells. When the liver is inflamed or damaged by hepatitis it is unable to perform these vital functions, causing serious health problems. There are seven recognized types of viral hepatitis, which are A through G. This article will focus on hepatitis C (HCV).

Hepatitis C infection was first identified in the late 1980s and is the most serious common blood-borne disease in the United States. The actual hepatitis C virus is a small, single-stranded RNA (ribonucleic acid) virus of the family Flaviviridae. There are approximately six major genotypes (types of genetic information or genes) and more than 50 subtypes of HCV. Genotypes 1a and 1b are the most prevalent in the United States. Genotypes 2 and 3 are present in approximately 10-20% of patients. Due to rapid virus mutations or changes in its structure, this virus can evade the immune system, making it difficult to develop a 100% effective treatment or vaccine against HCV. Certain genotypes are more responsive to currently approved therapies. Hepatitis C cannot reproduce without being in liver cells. As new viral particles increase, rapidly reproduce and go into the bloodstream, they cause the body’s immune system to make antibodies against the virus; however, protective antibody is not usually developed against HCV.

It is estimated that approximately 4 million people, or 1.8 percent of the U.S. population, are infected with HCV. It is also estimated that there are 30,000 new HCV infections and 8,000 to 10,000 related deaths annually. That translates into four times the number of people infected with the virus that causes Acquired Immune Deficiency Syndrome (AIDS). Approximately 70-75% of those infected with HCV will go on to develop chronic liver disease, accounting for the top cause of liver transplants at approximately 1,000 transplants per year.

According to current research, approximately 20% of patients with chronic HCV will progress to cirrhosis over 10 to 20 years. After 20 to 40 years, a smaller percentage of patients with chronic disease develop liver cancer (hepatocellular carcinoma). Factors that can cause HCV to progress faster include the use of alcohol daily, male gender, older age at time of infection, longer disease duration, co-infection with the Human Immunodeficiency Virus (HIV) and/or hepatitis B. HCV infection occurs in as many as 33% of the patients with HIV. Of that 33%, more than 60% of HCV infections are caused by intravenous drug use and 4% from men who have sex with men.

Although routine screening of blood for transmission has improved significantly since the early 1990s, HCV is primarily spread or transmitted by contact with blood and blood products in the United States. Currently the use of shared unsterilized (cleaned at high temperatures) or inadequately sterilized needles (tattoo or drug) and drug cookers (spoons, cotton, etc.) is the most common risk factor for contracting the disease. However, many patients acquire HCV without any known exposure to blood or drug use. Other methods of transmission of HCV are sexual transmission and maternal-infant transmission, which accounts for approximately 5% of HCV infections. Surveys of monogamous sexual relationships with a partner infected with HCV have shown that less 5% became infected. Hepatitis C is not transmitted by sneezing, hugging, coughing, food, water or casual contact with another person.

Chronic HCV varies in its clinical course and outcome. Some patients will not have any signs or symptoms of liver disease and completely normal levels of alanine aminotransferase (ALT), a serum liver enzyme. ALT is a blood test that healthcare providers use to evaluate patients for liver disease. High levels of ALT suggest liver disease. The new upper limit of normal range for ALT is 30/UL for men and 19/UL for women. In the middle of the spectrum are a large number of people who have few or no symptoms, mild to moderate elevations in liver enzymes, and an uncertain prognosis. At the other end of the spectrum there are patients with severe HCV with symptoms of liver disease, elevated serum liver enzymes, and who go on to develop cirrhosis and end-stage liver disease (ESLD).

Many people with HCV have no symptoms of liver disease. The incubation period for HCV is from 15 to 160 days or an average of 7 weeks. If symptoms are present they are usually mild, non-specific, and intermittent.
Constitutional symptoms may include fatigue, mild abdominal discomfort, nausea, poor appetite, or muscle and joint pains.

Clinical signs and symptoms indicating a progression of liver disease to cirrhosis include the constitutional symptoms, muscular weakness, itching, dark urine, fluid retention, and abdominal swelling. Clinical signs of severe liver disease may include enlarged liver, enlarged spleen, jaundice, muscle wasting, fluid in the abdomen (ascites), ankle swelling (edema) and excoriations (sores from scratching).

Complications that do not involve the liver (extrahepatic—outside of the liver) can develop in approximately 1-2% of people with HCV. The most common is cryoglobulinemia, which is indicated by skin rashes, joint and muscle aches, kidney disease, neuropathy, and cryoglobulins (an abnormal protein called globulin).

The diagnosis of HCV is based on clinical signs and symptoms, blood tests and a liver biopsy. Acute HCV is diagnosed by clinical presenting symptoms such as jaundice (yellow color of the eyes and skin), nausea, and flu-like symptoms. Hepatitis C is also diagnosed by laboratory or blood tests.

The blood tests for HCV are based on AST, anti-HCV by enzyme-linked immunosorbent assay (EIA) and HCV polymerase chain reaction (PCR) assay. A 10-fold increase in serum ALT and the presence of anti-HCV is also a sign of hepatitis C. HCV is also detected by the presence of serum (blood without red and white cells) anti-HCV antibodies by a third-generation EIA test. As with all EIA there are a small percentage of false-positive results. Additional testing is useful for confirmatory diagnosis of the EIA. The gold standard to confirm the diagnosis of HCV is to test for anti-HCV is present and serum ALT levels remain high or elevated for more than 6 months.

There are several methods for measuring the amount or “titer” of HCV in the serum. These methods include a quantitative PCR and a branched DNA (bDNA) test. The viral load of HCV may not correlate with the severity of the hepatitis or with a poor prognosis (as it does with HIV); but viral load does correlate with the likelihood of a response to antiviral therapy. Low viral loads (less than 2 million) respond better to current antiviral therapy.

A genotype (the hereditary information present in the virus) is helpful in making recommendations and counseling regarding therapy. Once the genotype is tested it does not need to be tested again since it will not change. Patients with genotypes 2 and 3 are almost three times more likely to respond to therapy. Furthermore, genotype 1 may require a more lengthy treatment.

Liver biopsy is not necessary for diagnosis, but is useful for grading the severity of disease and staging the degree of fibrosis (scarring) and permanent damage to the liver such as the Histologic Activity Index (HAI). Most clinicians use a more qualitative approach to classify by stage: 0 = no fibrosis, 1 = mild fibrosis, 2 = moderate fibrosis, 3 = severe fibrosis, including bridging fibrosis, and 4 = cirrhosis.

Before treatment of hepatitis C with drug therapy can be started, the patient needs to have a thorough examination in order to identify potential co-existing health problems that may not be compatible with HCV therapy. A few of the contraindicated illnesses are major depression, heart problems, and kidney disease. Drug and alcohol use must also be controlled before therapy starts. Other factors that should be taken into consideration for treatment are viral load, age, and results of the liver biopsy. The risks and/or benefits of therapy must be assessed on an individual basis.

All patients with chronic HCV infection are considered potential candidates for antiviral therapy. However, after evaluation, treatment is clearly only recommended for a select group of patients. The current treatment of choice is a combination therapy including interferon and ribavirin. Treatment is highly recommended in
patients who are at greatest risk for cirrhosis. Patients over 60 years and children need to be evaluated and reassessed at regular periods as benefits of treatment have not been well supported in the research.

Antiviral therapy for HCV includes interferon (IFN) or pegylated interferon (PEG-IFN) or more commonly a combination therapy with IFN or PEG-IFN and ribavirin. Although interferon is produced naturally in the body to fight viruses, a synthetic or made-in-the-laboratory alpha interferon is used for medical treatments. Alpha interferon (alpha-2a, alpha-2b) is usually given at a dose of 3 million units (MU) subcutaneous (in the fat) injection three times a week or interferon alacon-1 is given 9 micrograms (mcg) 3 times a week for 24 weeks. Oral ribavirin is given twice daily in the form of a 200 mg capsule. The new pegylated interferon (PEG-IFN) was developed to avoid the peaks and troughs (low dips) of interferon levels by remaining in the blood for a longer period of time, having the advantage of lasting longer and only administered once per week. PEG-INF alpha is given based on the weight of the patient. PEG-IFN alfa combined with ribavirin is now replacing the old IFN-alpha combined with ribavirin as the standard or “gold standard” treatment for chronic HCV infection.

Treatment with interferon alone or combination therapy with interferon and ribavirin leads to rapid improvements in serum ALT levels in 50–75% of patients and the disappearance of detectable HCV RNA from the serum in 30–50%. A response is considered “sustained” if HCV RNA remains undetectable for 6 months or more after therapy stops. Combination therapy with interferon and ribavirin leads to loss of HCV RNA on treatment in 50–55% of patients and a sustained loss in 35–45%. Optimal duration of therapy varies depending on whether interferon monotherapy or combination therapy is used, as well as by HCV genotype. Patients with genotypes 2 and 3 have a high rate of response to combination treatment (60–70%), and a 24-week course of combination therapy yields results equivalent to those of a 48-week course. In contrast, patients with genotype 1 have a lower rate of response to combination therapy (25–35%), and a 48-week course yields a significantly better sustained response rate.

In HIV/HCV co-infection, benefits of therapy for HCV must take into consideration the concurrent use of anti-HIV medications and medical conditions. If CD4+ counts are normal or minimally abnormal (greater than 400), responses are similar to those in patients who are not infected with HIV. Ribavirin may still have significant drug interactions with other antiretroviral drugs.

Treatment of HCV infection with the current gold standard is not always effective and treatments regimens can be difficult for a patient to tolerate. Before starting on antiviral therapy you should receive counseling on the potential side effects and how they will affect your ability to function at work and in the activities of daily living.

The major potential side effects of interferon include depression, irritability, anxiety, impaired concentration, insomnia, autoimmune disease and bone marrow compromise. Also, in addition to these symptoms, the most common side effects of interferon include flu-like symptoms: fatigue, muscle aches, headaches, nausea, vomiting, rigors (shakes), and a low-grade fever. The loss of hair (alopecia) can occur in women.

The major side effect of ribavirin is hemolytic (breakdown of red blood cells) anemia. In some cases, anemia is so severe that therapy must be discontinued. Ribavirin has also caused birth defects during animal studies. Both ribavirin and combination therapy should not be used on women who are pregnant or who may become pregnant, or on their male partners, during therapy or six months after therapy is completed. Women of childbearing potential must use two forms of effective contraception (birth control) due to the possibility of serious birth defects.

If there is no response to current therapies, some patients may be considered for controlled clinical trials. When the liver decompensates and fails, liver transplantation remains the only other option for treatment of hepatitis C at this time. However, the donor liver often becomes reinfected with the virus. Liver transplantation is still rather controversial among patients co-infected with HIV and HCV.

Since the liver is already being damaged from HCV, steps must be taken to protect it from further harm. Consultation with the healthcare provider about the use of any medication, including over-the-counter and herbal supplements, must be included. Alcoholic beverages should be stopped. Every effort should be made to stop smoking. Diet control and avoidance of obesity appear to be helpful in the care of the liver. All patients with HCV, if not previously exposed to hepatitis A and B, should be vaccinated. Ample rest and a healthy lifestyle will help strengthen the immune system. Regular medical attention is important and new problems should be identified immediately. Hepatitis C support groups are available to provide encouragement to those experiencing problems and challenges living with this disease.
How Do HIV Meds Work in the Body?

When HIV multiplies inside an infected cell, it relies on proteins called enzymes. Whenever you see a word that ends with “-ase” you’re probably looking at an enzyme. First, HIV uses reverse transcriptase to read its own genetic code and copy it. This code is a set of instructions for building a new virus. Next, HIV uses integrase to insert a copy of its code into the infected cell’s own “code book” in the cell nucleus. Now the virus can use the cell’s own machinery to make copies of itself. New viral proteins get manufactured based on the genetic code. Then HIV uses protease to assemble these proteins into a new working copy of itself.

Our current anti-HIV drugs don’t kill HIV. In fact, scientists haven’t figured out how to kill any virus yet. Instead, they design drugs that make it harder for the virus to multiply. So far, the drugs we have to fight HIV block, or inhibit, a specific enzyme. Two types of drug—the nucleoside analog reverse transcriptase inhibitors (also called nukes), and the non-nucleoside reverse transcriptase inhibitors (non-nukes or NNRTIs)—block the reverse transcriptase enzyme. A third type of drug, protease inhibitors, blocks the final step of viral assembly that depends on the protease enzyme.

The HIV genetic code is like a string of beads called nucleosides. There are just four different nucleosides, like four colors of beads, that get put together in different combinations. The viral nucleosides are adenosine (A), guanosine (G), thymidine (T) and cytidine (C). It takes three nucleosides to define a specific amino acid. Amino acids are the basic building blocks of all life. When HIV—or a human cell—multiplies, the genetic code gets “read.” Amino acids are created according to the genetic code and assembled into proteins to make a new cell, or a new virus.

Nucleoside analog reverse transcriptase inhibitors (NRTIs)

When reverse transcriptase (RT) reads HIV’s genetic code, it goes one nucleoside or “bead” at a time. It identifies which nucleoside it is (which color) and it looks around inside the cell for that nucleoside’s “mate.” Each nucleoside pairs off with just one other type of nucleoside. “A” pairs up with “T,” and “G” pairs off with “C.” When the RT enzyme reads an “A,” it looks for a “T” and vice versa. When it reads a “G,” it looks for a “C” and vice versa.

The nucleoside analog RT inhibitors are fake nucleosides (“analog” means “something similar”). The enzyme can’t tell the difference and picks up a drug molecule instead of a real nucleoside. The fake nucleosides stop the process of reverse transcription. It’s kind of like a zipper, where reverse transcriptase is the “pull” that combines the two sides. One side is HIV’s genetic code, and the other side is made up of the nucleosides that reverse transcriptase finds inside the cell. A “nuke” drug molecule is like a bent tooth on the zipper, and the process can’t continue.

Non-nucleoside reverse transcriptase inhibitors (NNRTIs)

Researchers tell us that the RT enzyme is shaped kind of like a catcher’s mitt. The strand of HIV genetic code slides between the “thumb” and the “fingers” of the enzyme, along the
palm of the hand. The NNRTI drugs block the RT enzyme by filling up the pocket in the catcher’s mitt. When there’s a baseball in the mitt, nothing can slide along the “palm.” The enzyme can’t read the genetic code.

**Protease inhibitors (Pis)**

HIV uses the protease enzyme late in its life cycle. After all the new viral proteins have been built, they get assembled into a new virus that takes shape and pushes out of the infected cell. The insides of this new virus aren’t fully formed yet, and protease plays a key role. It’s like a pair of chemical scissors that cut long strands of protein into the correct pieces needed to assemble the core of the virus. The protease inhibitor drugs block the enzyme by locking in between the two blades of the scissors so they can’t cut anything.

**What is Resistance?**

If reverse transcriptase or protease got blocked 100%, HIV couldn’t make any new copies of itself and your viral load would eventually decline to zero. Unfortunately, anti-HIV drugs aren’t 100% effective and HIV continues to multiply. Sometimes, HIV develops resistance to a drug, or in other words, it keeps on multiplying just like the drug wasn’t even there in the first place.

Resistance is a happy accident from the virus’ point of view. When new viral copies are made, they almost always contain errors: slight differences in the genetic code that result in slightly different versions of HIV. Most of these errors, or mutations, are fatal to the virus and they die without multiplying. But sometimes, mutant versions of HIV can not only multiply, but can ignore some antiviral drugs.

For the nukes, we talked about a zipper getting stuck when it hit a bent tooth. With some particular mutations, HIV acts like a self-repairing zipper. Reverse transcriptase becomes able to read “around” the bent tooth—the nucleoside analog drug molecule—and continue creating the code for a new virus.

For the non-nukes, if the reverse transcriptase enzyme changes shape just a little bit, it might “drop the ball” that we put in the catcher’s mitt. The drug molecule might not be able to stay inside the enzyme to block it, and RT can go ahead and read the viral genetic code.

For the protease inhibitors, if protease changes shape just the right way, the drug molecule might not be able to stick in between the blades of the scissors. Protease can go ahead and do its job of assembling a new virus.

Not every mutation can give HIV resistance to drugs. It’s a random process. The more new copies HIV makes, the more mutations there are, and the more likely it is that some of them will give the virus resistance to antiviral drugs.

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**Why Does Resistance Matter?**

If your virus develops resistance to a medication, it will keep multiplying even though you take the drug. Resistance can cut down your possible treatment options very quickly. For example, the latest version of the treatment guidelines lists 78 different antiviral combinations as “strongly recommended” or “recommended alternatives.” But if your virus develops resistance to AZT (Retrovir), for example, and you can’t use it any more, then there are only 39 remaining combinations you could choose from.

AZT and d4T (Zerit), both from the NRTI class, are somewhat cross-resistant. If your virus becomes highly resistant to AZT, it’s probably at least partly resistant to d4T. If you can’t use either AZT or d4T in your regimens, then there are only 13 combinations to choose from.

The current guidelines don’t include tenofovir (Viread), which was recently approved. That will add several more options for people who can’t use AZT or d4T. The important point is, however, you don’t want to run out of options and have to wait for a new medication to get approved.

**What Can I Do About Resistance?**

When HIV multiplies, it mutates. That’s just a fact of viral life. Once in a while, one of those mutations will help the virus resist medications. The more HIV multiplies, the more it mutates, and the higher the risk of new resistance mutations showing up. The best way to avoid resistance is to keep it from developing in the first place. And the best way to do that is to keep HIV under control so it has fewer chances to multiply.

To keep HIV from multiplying, you should take anti-HIV medications according to their instructions. When you do this, you should have enough of each drug in your bloodstream to keep HIV under control. The manufacturers work hard to figure out how much drug is needed to control the virus without causing too many side effects.

If you miss doses or if you don’t take them with (or without) food, according to the instructions, there might not be enough drug in your bloodstream. When drug levels drop, HIV can multiply more quickly, at least for a while. More multiplication of HIV means more mutations and a higher risk of developing resistance.

There have been some studies showing that resistance develops even at undetectable levels. Others show it can occur even with 100% adherence. Researchers are searching for the mechanisms that allow this to happen. In the meantime, what you can control is your adherence (as long as you can tolerate your regimen). Research shows that in the era of HAART (highly active antiretroviral therapy), your first
A single mutation can also give HIV resistance to all of the NNRTIs: the K103N mutation. Just like the previous example, “K” and “N” are codes for amino acids. Instead of the genetic code for “K” at position 103 of reverse transcriptase, we find the code for amino acid “N”. When the same mutation or group of mutations makes HIV resistant to more than one drug, those drugs are called “cross-resistant.” For example, the PIs Crixivan (indinavir) and Norvir (ritonavir) are cross-resistant. All of the NNRTIs are cross-resistant.

It’s not always this clear whether HIV has resistance to a certain drug or not. For most of the protease inhibitor drugs, HIV has to get several mutations, one after another, before it develops resistance.

The resistance test report for genotypic testing is a list of mutations found in the sample of HIV. Those mutations that are believed to cause resistance to specific drugs are highlighted, and the report usually indicates whether the virus is believed to be resistant or sensitive to each anti-HIV drug. Unfortunately, when the virus needs multiple mutations to develop resistance, it’s not always clear whether it’s resistant or not. Genotypic testing cannot tell you “how resistant” the virus is to any particular drug, but resistance is not an all-or-nothing thing. HIV can be sensitive to a drug (no resistance), slightly resistant (the drug still works, but not as well as against wild type virus), or highly resistant (the drug doesn’t slow HIV down at all). In some cases, mutations can make the virus hypersusceptible: a drug might work even better than against the wild type virus.

Phenotypic Testing is the second main type of resistance testing. Instead of the genetic code of the virus, it looks at how fast the virus actually multiplies when each anti-HIV drug is present. A range of doses of each drug is added to individual test tubes containing cultures of either the sample virus or of “wild type” virus. After a certain amount of time, the amount of the sample virus in each test tube is measured and compared to the amount of “wild type” virus. If there are more copies of the sample virus than the wild type virus, it has resistance to the drug.

Resistance is reported as “fold change” in a phenotypic test report. This tells you how many times more copies there are of the sample virus compared to the wild type virus. For example, if there are 500,000 copies of the wild type virus, and 2 million copies of the sample virus, then it has “4-fold” resistance to the drug being studied. Although this is easy to understand, it’s not clear what it means. For some drugs, 4-fold resistance means that the drug won’t work at all. For other drugs, 10-fold resistance means that the virus is still sensitive to the drug. There are “cut-off” levels for fold resistance used in phenotypic resistance reports. They are different for each drug, and are somewhat different for each company.

Which test is better?

Genotypic testing is indirect. It analyzes the genetic code of the virus, and reports on mutations that researchers have found to be related to resistance to particular drugs. It can be difficult to decide whether a certain collection of mutations means the virus is resistant or not. Genotypic testing has to be interpreted using a set of rules, and each company doing resistance testing might use a slightly different set of rules for its reports. Also, the rules keep changing as researchers learn more about exactly which combinations of mutations are the most relevant to resistance for each drug. Genotypic testing is faster (about one week) and less expensive than phenotypic testing.

Phenotypic testing is a direct measure of how the virus behaves in the presence of anti-HIV drugs. The report is easy to understand: it tells you how much “fold resistance” the sample virus has to each drug. However, it can be hard to know what level of fold resistance really matters. Phenotypic testing usually takes about two weeks and is more expensive than genotypic testing.
New Types of Resistance Tests

The company Tibotec-Virco provides the “Virtual Phenotype” resistance test. It’s priced between genotypic and phenotypic tests and the results come back faster than phenotypic testing. First the virtual phenotype does a genotypic test. Then, instead of using a set of rules to interpret the list of mutations found in the sample, the results are compared to a large database of paired genotypic and phenotypic test results. The test report tells you the phenotypic test results of samples in the database with similar mutation patterns.

ViroLogic provides the “PhenoSense GT” test. It’s not really a different type of test. However, for doctors who prefer to see both genotypic and phenotypic test results, it uses a single blood sample to run both tests and provides the test results in a side-by-side format.

Weaknesses of Resistance Testing

There are other problems with resistance testing besides the difficulties in interpreting results.

• Neither test can detect “minority” strains that make up less than about 20% of a patient’s population of viruses.

• They cannot detect resistance that might be “hiding” in resting T-cells or other viral reservoirs, and some researchers believe that this “archived” resistance can re-emerge quickly if it will help the virus survive particular drugs.

• When someone stops taking antiviral medications, the drug-resistant virus has no survival advantage, and the wild type virus is likely to re-emerge and become the most common strain. The tests may not detect any resistance if the patient has been off medications for more than a couple of weeks.

• The rules for interpreting genotypic tests, and the fold change results for phenotypic tests are all based on just one drug at a time and may not accurately reflect what’s going to happen with a combination of anti-HIV meds.

• Some doctors use both genotypic and phenotypic tests to get a more complete picture of what’s going on, but often the tests give conflicting results that can be very confusing to interpret.

Does Resistance Testing Help?

Several clinical trials studied whether doctors who had the results of resistance tests made better treatment decisions for their patients. The doctors just made their treatment decisions the normal way (without resistance test results), or they got genotypic and/or phenotypic test results. Sometimes they got expert advice on how to interpret the resistance test results.

In most of the trials, resistance test results led to viral loads about .5 log lower than those without. That’s a significant difference. Unfortunately, not every trial showed the same results, and in some cases it was difficult to tell whether it was resistance test results or expert advice that made the most difference. Although there are still some challenges in using resistance test results, most AIDS physicians believe that they help make better treatment decisions.

Who Should Get a Resistance Test?

Treatment guidelines recommend resistance testing in the following situations:

• When antiviral treatment stops working: if viral load rises rapidly or CD4 cell count drops.

• When antiviral treatment isn’t working well enough: viral load doesn’t become undetectable within a month or two.

• Pregnant HIV-positive women.

Guidelines suggest that resistance testing be “considered” for newly-infected people. Several research studies have documented an increasing rate of new infections with strains of HIV that are already resistant to one or more antiviral drugs. Resistance testing might help doctors avoid prescribing drugs that won’t control a patient’s virus. Using the wrong drugs could allow HIV to multiply and develop additional resistance.

Probably the most controversial in terms of resistance testing are people not taking antiviral medications who have been infected for several months or more. As mentioned above, if you’re not taking antiviral medications, the wild type virus will usually multiply the fastest and become the dominant strain in your body. However, some researchers believe that certain mutations can persist and be detected for several months or maybe even longer. There hasn’t been a lot of research on this question yet.

The take home message is to prevent resistance in the first place. The best way to avoid developing resistance is to take your anti-HIV medicines on schedule and as according to instructions. If you have a regimen that is working for you, then stick to it!

For additional discussion on drug resistance also see Medicine Chest: Once Again, One a Day on page 42.

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Safer sex—the use of a condom each time people engage in sexual intercourse—may be the most prudent way to protect oneself from sexually transmitted diseases. But more gay men are tossing caution to the wind in their desire to connect with their partner, skin-to-skin. That’s why the information shared and questions raised at a recent panel discussion and forum in Chicago on microbicides is of such great importance. The event was sponsored by the Hearts Foundation, AIDS Foundation of Chicago and Steamworks.

About 40 mostly gay men joined program planner and moderator Jim Pickett (Positively Aware columnist and also of The Faces of AIDS project, among other projects), along with panelists Dr. Eric Christoff (Hearts Foundation), Thomas Dunning (Howard Brown Health Center), Frank Oldham (Horizons) and Grisel Robles (AIDS Foundation Chicago).

“The idea of holding this discussion came to me as I was looking at the strides women have made in the microbicides movement,” Pickett said. “I felt it was time we as gay men became involved in the conversation and began using our own creativity and energy. A lot of gay men haven’t heard much about it but when they do, they become excited about the possibilities. Microbicides will offer another option for prevention and that’s important as so many men are forgoing the regular use of condoms. We need other tools that can be used to protect ourselves and our partners.”

A microbicide is a chemical agent that kills a microbe—any infectious agent such as gonorrhea, syphilis or HIV. And while there are some 60 compounds under investigation for use as possible microbicides, research and development efforts are at this point targeting the product for use by women, for the vagina—primarily serving as a contraceptive tool. And while that’s good news for women, discussion about and research aimed at developing an anal microbicide is much farther away.

“The push for microbicides is not a gay or heterosexual issue—it’s important for all of us,” Pickett said. “Anal sex isn’t something in which only gay men engage. But we [gay men] need to especially weigh in and say ‘we want rectal microbicides.’ This forum is the first step. But gay men need a greater consciousness because with microbicides comes the potential for us to have safer sexual lives.”

Robles likened the importance of the discussion to the steps taken when a new film is about to be released: “We have to begin now with promotional efforts so when the product is ready for public use, people will know about them and how to use them more effectively. It’s all about increasing public awareness and then motivating voters to contact elected officials demanding more dollars for research and development. We want to prepare people for the day when they’ll be able to go to the corner store for a few groceries and the microbicide of their choice.”

Robles said interest in both vaginal and rectal microbicides has increased as the rate of HIV/AIDS infection, particularly among African-American women, continues to rise.

“We have to be creative in the [microbicide] movement prevention strategies,” she
said. “Usually the leading pharmaceuticals provide funding for new drug development, but not in this case. Most of the dollars at this point are coming from smaller biotech companies and private donors. Even after first hearing about the potential of microbicides almost eight years ago, we still are only in the first phase of the three required by the federal Food and Drug Administration (FDA).”

All new drugs intended for specific therapeutic indications must first undergo three steps: pre-clinical research and development, clinical trials that study the efficacy and safety of the drug in humans and permission to market the drug—a process that in the three stages can take up to 12 years. And, even after a new drug is released on the market, the FDA continues to monitor the drug for adverse or toxic reactions which may take several years for negative effects to manifest and be discovered. Robles said that Pickett was the real push behind the forum, but now it’s time for other gay men to share their views and concerns—to become vocal and active.

“Microbicides are already being discussed to help women—now the same message regarding the potential effects and appropriate use for gay men need to take place,” Robles said. “The reality is that rectal microbicides will benefit not just the MSM population [men who have sex with men] but heterosexual couples as well. But the process is going too slow. There are only two rectal microbicides in phase one trials—none in phase two or three.”

Dr. Christoff said the failure of nonoxynol-9 (N-9) for rectal use is instructive. “N-9 is all there was and basically still is on the market,” he said. “It’s been used by women as a spermicide to prevent pregnancy for decades, and some gay men have sought out lubricants containing N-9 in the hope that it might add some protection against HIV. But it was never tested in the rectum. Now subsequent research indicates that N-9 can irritate tissue in the vagina and even increase the risk of contracting HIV. Furthermore, the vaginal and rectal walls are two very different environments—too different to assume that drugs developed for one may be appropriate for the other.”

Christoff added that due to the large number of volunteers needed when rectal microbicides reach the clinical trial stage, serious questions of efficacy will certainly arise.

“Not only will we need a huge at-risk population, but some patients will become infected with HIV and other STDs in the testing process,” he said.

Oldham expressed his anger at the delay of making microbicides available on the open market, in light of the knowledge that the discussions about these chemical agents first took place in 1994. “We are in a cultural war and it’s the oppression of gay men that has prevented the development of not only microbicides but other medications,” he said. “It is estimated that a million gay men have already died of AIDS since the first detected cases. How many of those lives could have been saved if microbicides were available as an added measure of prevention? We are all in danger because our lives are not valued. We must demand that this product be moved along in the FDA’s established process.”

Oldham added that while some view developing rectal microbicides as a stamp of gay sexuality, he said it remains our right—but one for which we must continue to fight.

“Sexuality for gay men is a culture,” he said. “Not only are we fighting to protect our very lives, but we are also fighting for our institutions, like Steamworks [a local bathhouse]. We have the right to follow any lifestyle we desire and to live our lives as we choose. Microbicides are important in that effort.”

Dunning agreed with Oldham, saying that homophobia and homo-hatred remain deeply rooted in society and that with the recent shifts in public interests, gay men must make their voices heard. “Heterosupremacy is just like white supremacy—both are ideas that say we value one portion of society over another,” he said. “But our [gay men’s] lives are just as valuable as those who are straight. Our community is not being decimated because of blow jobs—it’s because of HIV/AIDS passed on through anal sex. But people don’t want to talk about that. I guess you could say we’re lucky here in Chicago because we have access to information and products that help improve the quality of our lives, but what about gay men in rural Nebraska? We know it’s still very early in the development of rectal microbicides, but we are only a year or two away from vaginal microbicides. Gay men need to not only give women our support, but we need to demand that research and development be stepped up addressing our specific health needs.”

At the conclusion of the forum and panel discussion, sample cards were distributed addressed to [Illinois] U.S. Senators Richard Durbin and Peter Fitzgerald. Participants were urged to write them and other politicians expressing their views.

“We’re going to need to be ready for the November elections [for governor] and make sure that the candidate who supports our community’s needs is elected,” Robles said. “But in terms of rectal microbicides, this is going to be a long battle. And just like women have persevered since 94, men must get active now and be ready for the long haul.”

I’m often asked various questions from patients, nurses and physicians regarding HIV-related problems and their management. I always held the opinion that the more informed my patients are, the better acquainted they will be to assess their own needs and options. Many of the topics discussed in this month’s installment of The Buzz are day-to-day and commonplace, covering clinical problems as well as new treatments and developing options.

**What do you think has been the greatest treatment development this year?**

A new class of agents is about to land in our lap. T-20 is part of this new group of drugs that stop the virus from latching or fusing with CD4-T cells; they are about to become part of our armamentarium against the virus within a very short period of time. I believe that as T-20 is used with shrewdness and discernment by clinicians, it can reduce the amount of virus in the blood and improve immune system functioning for patients who are in the most difficult of situations. That is, those with low T-cell numbers and high viral loads. One question I am also asked is: “Will this drug be used early on?” Perhaps, but probably not in many patients; the drug will be expensive and needs to be injected (like an insulin shot) twice daily by the patient.

**What came out from The World AIDS Conference in Barcelona that will affect decision-making in your practice?**

Several studies were thought provoking, artful and provided some basis for using their design in clinical practice. One example was a study presented by Shlomo Stacznewski of Frankfurt, Germany, who successfully treated patients who either failed protease inhibitor treatment or had nucleoside-related (nuke) toxicity. Unique dual (double) protease inhibitor (PI) boosting with Kaletra plus saquinavir was used here. Other artful measures were incorporated in his work. First, for the patients who initially presented with resistance to PIs, they were placed on a treatment interruption or drug holiday until mutated virus reverted back to wild type (non-resistant or the older and native precursor virus) before starting on this new combination therapy. Second, he treated these individuals without any nuke backbone. The success of this approach, without the need for a nucleoside, is very tantalizing. I think this option should be placed on the table for patients in similar situations, but also needs much further study. (See “Kaletra/Sustiva on page 20.)

Also, information about a new and useful tool, recently presented at the drug resistance meeting in Seville, Spain just prior to Barcelona, but later discussed there again, is a lab test called “replicative capacity” (RC). This measures the ability of the virus to replicate or reproduce and is an indicator of the fitness of the virus. Decisions about “when to start” treatment may potentially be made with the help of this test. Currently I am using the test in a different situation: if the RC is low in patients with high levels of resistance with detectable viral load, it may not be necessary to change that individual’s therapy. This is because a low RC means low viral fitness and thus, immune function and T-cell count can continue to improve or be preserved. Much further work is necessary to research this new test device.

Finally, other new anti-viral drugs presented in Barcelona (discussed above and below in this article) will be useful and affect our management for many patients. They provide a great deal of hope for people because some of the newer treatments have distinct advantages over the older ones.

**What kinds of treatments will be coming in the next few years for people to look forward to?**

Several new protease inhibitors (PI) are on their way. A newly formulated version of amprenavir (Agenerase)—its “pro-drug” or active component, called 908, is in clinical trials and we at Northstar Healthcare in Chicago are also participating in this research. It is a protease inhibitor that can be taken once daily when combined with small doses of ritonavir [Norvir] and has the added advantage of a low number of pills per dose. It will be much easier and tolerable for patients than the older form of this drug. (See “Agenerase pro-drug” on page 20.)

Another PI, atazanavir, is already available through expanded access. It is administered as only two pills once daily and has
fewer side effects. It doesn’t seem to cause elevations in cholesterol or triglycerides (another type of fat found in the blood) and causes less gastrointestinal problems.

Tipranavir is a PI that has the advantage of being useful for people with resistance to other PIs. T-20, already discussed earlier in this article will be available by prescription early in 2003, as is FTC, a new nuke (see page 15, also The Buzz, September/October issue).

**My Doctor Says I Have Been Doing Well with My Protease Inhibitors But I Continue to Have Diarrhea That He Says I Should Learn to Live With; What Else Can I Do?**

First, let’s not assume that the diarrhea is due to medications; an easy test to do would be laboratory analysis to check stools for parasites or infectious bacteria. If this test demonstrates infection, then have it treated. If negative, you can try various options, but one at a time. Often taking pancreatic enzymes that improve the digestive process can reduce diarrhea quite a bit. Acidophilus supplements to replace beneficial bacteria may also help. Calcium as a binder has also been found to be effective in some patients and some people take Tums, which contains reasonable amounts of calcium. Finally, many HIV-positive patients are lactose intolerant. Thus, reducing dairy from your diet may be helpful.

**I Always Feel So Bloating Especially After Taking My Meds. Is This Normal?**

Not necessarily. If the bloating is being caused by an infection, it can easily be treated. Often bloating occurs due to dietary components such as greasy, high fatty foods, and “fast food.” Also, because of lactose intolerance, dairy products may be the cause. Avoiding these can often help. Finally, I sometimes use metronidazole for some patients to reduce gas-producing bacteria in the gut.

**Is There Anything New Regarding the Possible Causes of Lipodystrophy?**

A researcher from the Hospital Foch in Suresnes, France has recently discovered HIV activity in fat tissue, specifically within the fat cells of patients with lipodystrophy. If this finding correlates with fat redistribution problems, it may prove to have implications for possible treatments in the future. Research into affecting HIV replication specific to fat cells could also be investigated. Robert Gallo, the co-discoverer of HIV, stated that the finding may also explain why HIV is more difficult to eradicate. Presently, however, most experts believe that HIV and/or some of the anti-viral drugs affect the mitochondria and eventually lead to these fat-related abnormalities.

**What Can I Do About Lipodystrophy?**

Every patient with fat-redistribution problems has different manifestations. Depending on the situation, various options can be attempted. Often changing the drug combination to medication that is less likely to worsen the condition is possible. I sometimes use medications that improve insulin sensitivity to reduce visceral (body cavity) fat. Also, various anabolic agents can be helpful in combating some of the body habitus changes, while using various anti-oxidant vitamins to improve mitochondrial function. Finally, don’t underestimate the power of exercise to break down excess fat. A recent study has shown this to be quite advantageous when dealing with fat accumulation syndromes.

**What Is the Status of New-Fill? Will It Be Available Again?**

Currently the U.S. Food and Drug Administration (FDA) curtails its availability. Laws have been put in place to protect the public from many drugs with potential harmful effects. Thus all agents need to be studied adequately and the FDA has very high standards. New-Fill is an agent already proven and approved in other countries such as in Europe, Latin and South America, while
safety and effect has also been presented in at least two international medical AIDS conferences. The suffering and disfigurement of lipodystrophy could be taken into account and perhaps some loophole could be used to allow for patient access under severe facial atrophy conditions.

When will it be available again to the major HIV-impacted community? Who is to say. A pharmaceutical company needs to step up to the plate and pursue distribution rights and studies to satisfy regulations. Still, some of my patients have found a way to continue getting access while some individuals are traveling to San Diego and seeing my friend Dr. Jorge Tagle in Tijuana for its administration.

I have been getting increased numbness and shooting pains in my legs. My doctor says this is from HIV. I am currently on Epivir, Zerit and Crixivan; I am undetectable and my T cells are 390. If my numbers are so good, why am I having these problems?

The nukes and more often, Zerit, have been implicated in causing or worsening the symptoms you describe. These symptoms are often referred to as peripheral neuropathy (distal symmetric type), and denotes damage that has occurred to the nerve endings. It can often be treated or reversed with either dose reductions of the offending drug, which in this case is probably Zerit, or by substituting another drug. Talk with your doctor about your treatment options. Some doctors prefer to treat neuropathy with medications such as amitriptyline or neurontin, which suppress the symptoms but unfortunately do not reverse the problem. Often vitamins such as folic acid and vitamins B1 and B6 are also helpful in improving the nerve function and reducing symptoms. In a small study done by the NIH (National Institutes of Health), acetyl-carnitine was found to repair the damage of peripheral neuropathy in HIV disease.

I like to go to circuit parties and my friends and I do some “crystal.” What’s the harm in a little of these party drugs?

First, drugs such as “crystal-meth” or “Tina” and the other alphabet drugs are highly addictive. Once you’re hooked it may not be easy to unhook. During an evening, being off them the next day presents with difficulties and lows that are so bad that it often leads to depression, anxiety and fatigue. Being on antiviral HIV medications may also have drug interactions. Eventually a host of psychological problems, not usually obvious to the individual himself using crystal, occurs. The metabolic changes occurring while being on them increase the body’s breakdown of body mass and overworks the heart and cardiovascular system. Hence, wasting, lipodystrophy and a host of heart problems can and probably will eventually develop or worsen. Using recreational drugs also reduces the use of safe-sex practices. There is an epidemic of syphilis and hepatitis C on the rise; party drugs have added to this problem. Patients who are HIV-positive and get infected with hepatitis C then become confronted with a more complicated course that are can be overwhelming and shorten one’s life.

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Of Birth and Dying

by Laura Jones

When I don't have anything else to do, I like to help people have babies. I used to help people have babies every week, but birth assisting, like birth-giving, requires the endurance of long hours in awkward positions and the sacrifice of large amounts of sleep (and at least a few meals); you have to be pretty hardy to do it regularly. I found it impossible to handle birth assisting while holding a full-time job, so I haven't helped with babies for several years.

But this summer I found myself unemployed and I had time to help lots of people have babies. I worked with my friend Karen's labor support and postpartum service, providing care for teenage mothers and their newborns. And I got to assist my old friends Kip and Kristin with the birth of their first child… a truly nerve-wracking experience, since that was the first time I'd ever assisted at the birth of anyone I knew well.

I've never helped anyone die—but my friend G. has. Having lost many friends and lovers to AIDS in the 1990s, he's held vigil over a lot of bedside. Though he doesn't help people die with the same frequency these days, he's well aware that people are still dying. And since he believes dying people need their friends around, he tries to be present whenever possible.

We spoke at length this summer about the similarities between helping a person give birth and helping a person die. The Chux pads and vomit, and the frequent changing of sheets. The ice chips and soft lighting. The attempts to make someone in pain as comfortable as possible. The way time becomes meaningless, six hours gone in what feels like fifteen minutes. And the waiting. The work of both birth and death requires assistants to hold space for the one laboring to bring forth a new life or surrender their own—and that usually means long periods of waiting.

Despite the hand-holding and the massages and the sips of room-temperature water, you can't really do what you'd like to be able to do. You can't stop or change the process, and you can't do it for them. So you, as a chosen support person, have to put aside the feelings of helplessness and do what you can: keep them comfortable, and just wait.

Some birth or die at home; others birth or die in hospital environments (freestanding birth centers and hospices are nice in-between options when you can get them). In both cases, the work is easier when the worker is respected and allowed to do what they need to do to get the job done. G. tells me stories of deaths that were like parties, complete with music, food, and an abundance of guests. I've attended births where four generations of the family's women were all crowded together in a laboring woman's room, chanting and singing to help the woman relax and "go into the pain" instead of fighting against it. Noisy indeed, but when this is what the birthing or dying person wants, nothing could be more helpful. Living can be such a lonely business, so people should have all the company they want as they bring babies in or as they go out.

Those who find greater comfort in privacy should get to have that as well. One of my favorite births involved a very young woman who only wanted one person to sit with her and hold her hand. No guests, no music, no conversation—and, most importantly, no other people in the room unless they absolutely had to be there. So we spent fourteen hours in complete silence, breathing deeply together when she needed to breathe deeply, dozing together when she was between contractions. The birth of her tiny daughter was almost equally quiet, and I'll forever respect the medical staff who were willing to alter their normal routines and provide her with the privacy she needed in order to feel safe. Likewise, G. has stories of extremely quiet deaths, where the silence in the room afforded his loved one the serene environment he or she needed in order to most peacefully let themselves go.

One of the things that you can't have when you help someone die is the feedback. "It helped so much to have you here; it was so much easier than doing it alone." These thank you's, whether spoken or communicated non-verbally, are one of the sweetest rewards of birth assisting. While those of you who have helped people die never get to hear those words from your loved ones after the fact, you should know that your presence made a difference for them.

It takes great bravery to sit with someone at the end of life—much more so than to be present for the beginning of life, which almost always involves gain instead of loss. But at the end of the day, the skills you use in both appear to be largely the same: good hands, a good heart, and the willingness to be there for someone who must step away from the life they've known forever in order to get to their new one.

Please welcome the following babies: Raymond L., Gregor R., DaeShawn M., JaQ'uaan S., and Sophia May.
Once Again, One a Day

by Glen Pietrandoni. R.Ph.

Once-daily drugs are now more widely available to treat HIV. This has been a dream of those people forced to schedule their days around timing of the doses of drugs and food in order to maximize the benefit of the life-saving compounds. It is obvious that we all want simpler regimens with fewer side effects, but we also need to feel confident that we are making the correct clinical choices for today and the future.

Some clinicians feel that once-daily regimens will mean that patients will have better adherence to medication. After all, patients will have to remember to take drugs fewer times each day. This may also translate to fewer “reminders” that the patient has a very serious disease, and therefore be emotionally very valuable to the success of therapy.

However, it is important to note that poor adherence cannot be fixed by the availability of once-daily dosing. Adherence becomes even more critical as each dose becomes more important. A missed dose of a once-daily dose will leave a 24-hour period for HIV to replicate before the next dose is taken, and resistance may form. Missing three doses per month on a twice-daily regimen means that you had 95% compliance. This is the number that most experts agree is critical for success with treatment. If one were to miss three doses per month in a once-daily regimen, this would translate into less than optimal drug levels and possible drug failure. That is less than once each week and pretty tough to accomplish for anyone!

Failing to remember a once-daily dose will have long-term effects if resistance occurs. Probably more important is the fact that future treatments options may be limited if once-daily regimens are not adhered to. Drug resistance is the most important factor in how long drugs will work to keep viral replication suppressed. Before committing to a once-daily regimen, we have to ask ourselves which regimen will be most effective in keeping the virus undetectable for the longest period of time. The once-daily convenience would be nice, but what is more important to you? Will there really be that much of a difference in your lifestyle going from a twice-daily combo to a once-daily regimen, that may require two dosing times every day anyway?

The most interesting data about adherence is the studies that have been done in prisons with DOT (Directly Observed Therapy). DOT is when each dose of drug is given directly to a patient at the prescribed time and a clinician observes the drug being ingested. In some studies, 100% of patients using DOT for therapy are undetectable after one year. In the same study where patients are allowed to take the medication without an observer (self-reported adherence), less than 80% of these people got to undetectable in the same amount of time. Adherence is a major factor in the success of any combination therapy.

Advertising can be a tease and also a little misleading. Because prior use of HIV therapies can impact which drugs an individual can use, once-daily drugs are not for everyone, no matter how much a person would like to have them. Even patients never...
having taken drugs for HIV can have resistance to some drugs, and therefore not be able to take advantage of some of the “easier” drugs.

I also would question each person’s commitment to adherence, especially someone just starting on therapy for the first time. In my experience, the people who do better on once-daily drugs are those who were doing well on adherence to twice and three times daily regimens. If someone cannot adhere well to Trizivir twice daily, how will they do on a once-daily regimen? Remember that not all once-a-day drugs can be taken at the same time, so it is very possible that there may be two or three dosing events within the day. Timing of food is still important. For example, Videx and Videx-EC and the Sustiva 600 mg tablets are taken on an empty stomach, while Viread is taken with food. All are once-a-day medications.

We do have a few drugs that already approved for use as a single daily dose. Sustiva is somewhat forgiving about the timing of the doses because of its long half-life (the amount of time the drug stays in the blood). Other drugs like Ziagen and even Retrovir (AZT) are being studied to be used in a once-daily regimen. Until we know conclusively that these combos are workable, we have to take a wait-and-see attitude.

Once-daily regimens can offer great hope and benefit to those who are able to take advantage of them. We cannot forget that these are still potent drugs and must be taken correctly for maximum benefit. All aspects of an HIV regimen should be considered seriously, not just the number of pills or dosing times per day. Each available drug has advantages and disadvantages and the choice of which combination to choose must be evaluated on potency, durability, and resistance in addition to pill burden, side effects and the patient’s ability to tolerate the drugs.

### Once daily drugs

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage and Administration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Videx-EC</td>
<td>400 mg capsule * empty stomach</td>
</tr>
<tr>
<td>Viread</td>
<td>300 mg tablet Take with food</td>
</tr>
<tr>
<td>Sustiva</td>
<td>600 mg tablet empty stomach</td>
</tr>
<tr>
<td>Epivir</td>
<td>two 150 mg tablets Soon, one 300 mg tablet no food restrictions</td>
</tr>
<tr>
<td>Viramune</td>
<td>2-200 mg tablets (after 14 day lead-in period)</td>
</tr>
<tr>
<td>Agenerase</td>
<td>8-150 mg capsules + 2-100 mg Norvir capsules take with food</td>
</tr>
<tr>
<td>Fortovase (or Invirase)</td>
<td>8-200 mg capsules + 1-100 mg Norvir capsule take with food</td>
</tr>
</tbody>
</table>

*the dose of Videx-EC can vary based on concomitant use of Viread, on side effects and other conditions requiring lower daily doses, including weight

### Once daily drugs being studied

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage and Administration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kaletra</td>
<td>6 capsules once daily take with food</td>
</tr>
<tr>
<td>Zrivada (atazanavir)</td>
<td>2 tablets once daily (experimental drug)</td>
</tr>
<tr>
<td>Ziagen</td>
<td>2 tablets once daily no food restrictions</td>
</tr>
<tr>
<td>Retrovir</td>
<td>2 tablets once daily no food restrictions</td>
</tr>
<tr>
<td>Zerit XR</td>
<td>Soon, one 100 mg XR capsule no food restrictions</td>
</tr>
</tbody>
</table>
It Ain’t Rocket Science

by Jim Pickett

Isn’t it time we deep six that phrase? Can it be any more overused and annoying at this point? And while we’re at it, let’s deep six “deep six.” But I am especially sick of hearing the “it ain’t rocket science” refrain increasingly in reference to HIV prevention. I mean, I include myself in that whining, strident chorus as well. Ya know, “Why are we still getting infected? HIV is so easy to prevent! It ain’t rocket science, ya know. You don’t have to be a brain surgeon to figure out how to wear a condom.” Blah blah blah. Waa waa waa.

The “brain surgeon” thing too. Gotta go. And we’re right, preventing an HIV infection ain’t rocket science. Whatever that actually is. Preventing HIV is harder. Infinitely harder.

Just knowing the mechanics of safer sex, how to use lube and rubbers properly, what’s high risk, what’s low risk—easy. We all get it. We all know. I knew, c’mon, I knew. But let’s talk about the incredibly complex emotional, psychosocial stew each one of us swims in, with all our neuroses, our impulses, our needs for connection in a stressful, difficult world—this is where a brain surgeon can’t help us.

I tested positive in 1995, well into the epidemic. I fucking knew better. I knew knew knew til I was blue. I practiced safer sex, a lot of it, with zillions of men, many positive, and stayed negative. And I loved it. The sex drive has always been healthy here, and very well fed like the Wisconsin farm folk I hail from. So why the hell did I discontinue condom use with someone I became involved with?

Because I suddenly forgot that HIV existed? That I suddenly forgot what condoms were, let alone how in the hell d’ya work these things? That I did a hit of Ecstasy and consequently fried every single brain cell that had learned and absorbed the horrors of AIDS and what to do to avoid that nightmare?

No, I started “barebacking” with a boyfriend, when the term still implied horses, because…hmmmmm…denial? Was I thinking, “I made it this far, I’ll be okay?” Was I lonely? Was I afraid if I said, “We shouldn’t have done that. Condoms next time”—that there wouldn’t be a next time? Was I ever so in love?

Was I complacent?

Did I adore how it felt, skin on skin, skin in skin, how hot and slick and intensely intimate, an amazing, erotic sensation I hadn’t experienced for 10 long years? Did I quiet the voices so I could get to that place again? And again? Did I scream his name? Did I have an ongoing conversation in my head around the theme of “Maybe I’m one of the lucky ones, who for whatever reason, don’t get infected?” I have always enjoyed an enormous amount of sex, after all, and in the first couple of years after I came out, from 1984 to 1986, I didn’t use condoms that I recall, and I got fucked a lot, which I do recall, so I must have been exposed at some point. I must be special. Did I think I was special?

Was I calming my fears by saying to myself, “Well, he never comes in me, so it’s not so bad?” Did part of me think I was doomed anyway, did part of me want to get it over with so I wouldn’t have to stress one more day? Did I feel alienated and disconnected? Did I feel at odds with the community I was initially so thrilled about joining, to fling into high-fiving, twirling around the dance floor in total exuberant abandon, but left me more times than not hurt, disrespected, devalued, dehumanized and lonely?

So lonely? Was I tired? So tired?

I was on the cusp. I was barebacking before the cover stories. I was taking risks before the advent of new medications gave us new hope, before magazine advertising made being HIV positive a sexy, youthful lifestyle, before people with AIDS were turned into an attractive marketing demographic, before crystal became the scourge it has become, before Viagra kept people fucking for days, while there was still tons of social marketing around prevention, when I was still seeing my friends, tricks, and amores dying.

A rocket scientist or a brain surgeon can’t help us address these sorts of issues. Preventing HIV is much more than wearing a condom every time. It’s addressing our full range of needs as human beings in an increasingly commercial, crass, disjointed, disconnected, fearful society—human beings, who at the end of the day, just want to be close to somebody, to feel some human warmth, in some way, any way. And we will throw all the education in the world out the window, along with caution and experience, to get that. To connect. We are human beings… who still have to contend with the deadly, alienating and devaluing affects of
Not support the federal use of dollars for needle exchange. We do not oppose the use of state, local and private funds for this purpose, but we believe it's the wrong priority for federal funds. But we believe in substance abuse treatment. We know it can be treated successfully. And we think it wrongheaded, and we don't think there are enough treatment slots available in this country to divert public dollars to needle exchange.”

Be informed.

Charles E. Clifton
Executive Director / Editor
Send comments and reactions to ed@tpan.com

Russia, India and Vietnam edited excerpts taken from the CDC HIV/STD/TB Prevention News Update.

Editor’s Note cont.

continued from page 12

homophobia, both internal and external, racism and stigma.

We are human beings.

I took the risks I did in a significantly less complicated world. How will we change to appropriately, impactfully respond to the world we are in right now? With formidable barriers like dwindling funding and a hostile political climate that puts so much value on idiocies like “abstinence only?” How will we fight complacency?енного

ND 2002

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Test Positive Aware Network (TPAN) is a not-for-profit organization dedicated to providing support and information to all people impacted by HIV.

ND 2002

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Thank you for your donation. Your contribution helps to provide subscriptions to people who cannot afford them. All donations are tax-deductible to the full extent allowed by law.

*Subscriptions are mailed to those who are HIV-positive for a small donation.
# TPAN Calendar of Events

All events held at TPAN unless otherwise indicated.

For additional information on these events please contact Michael Barnett at (773) 989-9400.

## November 2002

<table>
<thead>
<tr>
<th>Date</th>
<th>Time</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tuesday, 5th</td>
<td>7:30 PM</td>
<td>Committed to Living series “HIV and Depression”</td>
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<tr>
<td></td>
<td></td>
<td>Dr. David McKirnan for the University of Illinois at Chicago; Sponsored by Agouron</td>
</tr>
<tr>
<td>Wednesday, 6th</td>
<td>7:00 PM</td>
<td>TPAN Client Advisory Board Meeting</td>
</tr>
<tr>
<td>Wednesday, 13th</td>
<td>6 to 9 PM</td>
<td>Wine Tasting Benefit at Belloc Lowndes Gallery 215 W. Huron, Chicago [See ad]</td>
</tr>
<tr>
<td>Tuesday, 19th</td>
<td>7:30 PM</td>
<td>TPAN monthly board meeting at Ann Sathers in Chicago</td>
</tr>
<tr>
<td>Thursday, 28th</td>
<td></td>
<td>TPAN Closed for Thanksgiving</td>
</tr>
<tr>
<td>Thursday, 28th</td>
<td>6-10 PM</td>
<td>Thanksgiving Party Positive at Berlin in Chicago</td>
</tr>
<tr>
<td>Friday, 29th</td>
<td></td>
<td>TPAN closed</td>
</tr>
</tbody>
</table>

## December 2002

<table>
<thead>
<tr>
<th>Date</th>
<th>Time</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sunday, 1st</td>
<td></td>
<td>World AIDS Day</td>
</tr>
<tr>
<td>Monday, 2nd</td>
<td>6-8 PM</td>
<td>TPAN’s annual Holiday Party at Sidetracks in Chicago [See ad]</td>
</tr>
<tr>
<td>Tuesday, 17th</td>
<td>7:30 PM</td>
<td>TPAN Board Meeting at Ann Sathers in Chicago</td>
</tr>
<tr>
<td>Tuesday, 24th</td>
<td></td>
<td>TPAN Closed for Christmas Eve</td>
</tr>
<tr>
<td>Wednesday, 25th</td>
<td></td>
<td>TPAN Closed for Christmas</td>
</tr>
</tbody>
</table>

## Wine Tasting

**Sampling of Holiday Wines and Foods**

A Benefit for Test Positive Aware Network

Enjoy fine foods and wines from around the world at this inaugural event.

**Wednesday, November 13, 2002, 6-9 pm**

Belloc-Lowndes Gallery

215 W. Huron, Chicago, IL

Suggested donation of $40 will be accepted in advance or at the door. Please contact Jeffrey Allen at 773.989.9400 for more information.

Presented by TPAN and Fritz & Zoe Distinctive Events

## Holiday Party

The tradition continues for the TPAN Holiday Party and Volunteer Awards Ceremony

**Monday, December 2, 2002, 6-8 pm**

3349 N. Halsted, Chicago, IL

A suggested donation of $25 and a child’s gift to be donated to Children’s Memorial Hospital will be accepted at the door.

Please contact Jeffrey Allen at 773.989.9400 for further information.
Programs and Meetings

All meetings held at TPAN unless otherwise indicated:
5537 North Broadway, Chicago.
Office hours: Monday–Thursday, 9 am–8 pm. Friday, 9 am–6 pm
phone: (773) 989-9400 • fax: (773) 989-9494
e-mail: programs@tpan.com • www.tpan.com

<table>
<thead>
<tr>
<th>MONDAY</th>
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<tbody>
<tr>
<td><strong>TPAN Daytimers</strong></td>
</tr>
<tr>
<td>A group for people with HIV who prefer to meet during the day. Mondays and Thursdays at 10:30 am.</td>
</tr>
<tr>
<td><strong>Newly Diagnosed</strong></td>
</tr>
<tr>
<td>A group for newly diagnosed individuals. 2nd and 4th Mondays at 7:30 pm. Includes HIV 101 education.</td>
</tr>
<tr>
<td><strong>Straight Talk</strong></td>
</tr>
<tr>
<td>A group for HIV-positive heterosexuals. Mondays at 7:30 pm.</td>
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<thead>
<tr>
<th>TUESDAY</th>
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<tbody>
<tr>
<td><strong>Living Positive</strong></td>
</tr>
<tr>
<td>HIV-positive gay men discuss how being positive affects relationships and deal with the impact of HIV as single men. Tuesdays at 7:30 pm.</td>
</tr>
<tr>
<td><strong>Positive Progress</strong></td>
</tr>
<tr>
<td>A group for HIV-positive people in recovery. Tuesdays from 7:00–9:00 pm.</td>
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<thead>
<tr>
<th>WEDNESDAY</th>
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<tbody>
<tr>
<td><strong>Medical Clinic</strong></td>
</tr>
<tr>
<td>Free medical care provided by a nurse practitioner. This program is in conjunction with the Needle Exchange Program and is offered by Access Community Health Network. Call for an appointment. Wednesdays 3:30 pm–6:30 pm.</td>
</tr>
<tr>
<td><strong>Needle Exchange Program</strong></td>
</tr>
<tr>
<td>Free, anonymous, legal syringe exchange and HIV/AIDS prevention. Every Wednesday 5:00 pm–7:00 pm at TPAN office. In association with Chicago Recovery Alliance.</td>
</tr>
<tr>
<td><strong>Yoga</strong></td>
</tr>
<tr>
<td>Wednesdays at 7:30 pm.</td>
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<thead>
<tr>
<th>THURSDAY</th>
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</thead>
<tbody>
<tr>
<td><strong>TPAN Daytimers</strong></td>
</tr>
<tr>
<td>A group for people with HIV who prefer to meet during the day. Mondays and Thursdays at 10:30 am.</td>
</tr>
<tr>
<td><strong>Medical Clinic</strong></td>
</tr>
<tr>
<td>See description in Friday’s listing. Thursdays 2:00 pm–5:00 pm.</td>
</tr>
<tr>
<td><strong>Brothers United in Support (BUS)</strong></td>
</tr>
<tr>
<td>A group for HIV-positive gay and bisexual men of African descent. Thursdays at 7:00 pm.</td>
</tr>
<tr>
<td><strong>Staying Strong, Healthy and Empowered (SSHE)</strong></td>
</tr>
<tr>
<td>A group for HIV-positive women. Call Kathleen at (773) 989-9400 for more information.</td>
</tr>
<tr>
<td><strong>Party Positive Social at Berlin</strong></td>
</tr>
<tr>
<td>Berlin, 954 W. Belmont, Chicago. Thursdays from 6:00–10:00 pm.</td>
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</tbody>
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<tr>
<th>FRIDAY</th>
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<tbody>
<tr>
<td><strong>Medical Clinic</strong></td>
</tr>
<tr>
<td>Free medical care provided by a nurse practitioner. This program is in conjunction with the Needle Exchange Program and is offered by Access Community Health Network. Call for an appointment. Fridays 10:00 am–12:00 pm.</td>
</tr>
<tr>
<td><strong>Safe Passage</strong></td>
</tr>
<tr>
<td>A group for young adults (ages 18–24) who are HIV-positive. Fridays at 7:00 pm.</td>
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</tbody>
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<thead>
<tr>
<th>SATURDAY</th>
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<tbody>
<tr>
<td><strong>Family AIDS Support Network (FASN)</strong></td>
</tr>
<tr>
<td>A group for family, friends, and caregivers. Call Betty Stern at (773) 989-9490.</td>
</tr>
<tr>
<td><strong>Speakers Bureau</strong></td>
</tr>
<tr>
<td>Individuals are available to community groups and organizations to educate on HIV, safer sex, harm reduction and experiences of living with HIV. Call Kathleen at (773) 989-9400.</td>
</tr>
<tr>
<td><strong>Peer Support Network</strong></td>
</tr>
<tr>
<td>Provides one-on-one support for recently diagnosed individuals. Volunteers provide support, information and referrals. Call Kathleen at (773) 989-9400 to get a buddy!</td>
</tr>
<tr>
<td><strong>Positive Buddy</strong></td>
</tr>
<tr>
<td>Volunteers provide individuals living with HIV/AIDS one-on-one emotional / physical support. Call Kathleen at (773) 989-9400 to get a buddy!</td>
</tr>
<tr>
<td><strong>Miscellaneous</strong></td>
</tr>
<tr>
<td><strong>ChicagoPos18to24 at aol.com</strong></td>
</tr>
<tr>
<td>AOL chat room for young adults (ages 18–24) who are HIV-positive. Hosted by TPAN’s Young Adult Program. Go to AOL town square. Monday through Friday 3:00 pm–5:00 pm.</td>
</tr>
</tbody>
</table>

Support groups sponsored by the Chicago Department of Public Health Peer Support and Buddy programs sponsored by the AIDS Foundation of Chicago
WORLD AIDS DAY AD HERE