More Than The Virus
OIs, Metabolic Complications & AIDS-Related Cancers

Much of the focus of HIV research and treatment is on suppression of the human immunodeficiency virus itself. Unfortunately, it’s not just about the virus, damn it.

This issue of ACRIA Update explores a constellation of medical conditions that still confront people living with HIV/AIDS, some largely considered problems of the past. Thousands of individuals in the United States who are living with HIV continue to face the challenges of guarding against opportunistic infections, are in urgent need of effective treatments for HIV-related cancers, and are concerned about the metabolic complications associated with HAART therapies. We are honored to have respected, knowledgeable colleagues write about the latest research findings and advances concerning each of these HIV-associated conditions.

Once again, ACRIA Update also presents unique personal perspectives from people on the front lines of the AIDS war, people who devote themselves to helping others in the community understand treatment issues and access quality care. Three such individuals have shared their experiences attending the 10th Conference on Retroviruses and Opportunistic Infections, helping our readers understand how we can all learn about late breaking research advances for HIV and incorporate these new findings into the management of this disease.

J Daniel Stricker, Editor in Chief

Opportunistic Infections: They’re Still Here

When I first got involved in HIV/AIDS treatment education and advocacy work about ten years ago, more than 40,000 people were dying of AIDS every year in this country, compared to the 16,000 or so deaths reported annually today. In an attempt to find my own niche among the brilliant minds and eloquent voices of AIDS activism, it was the opportunistic infections – those insidious and often deadly complications of AIDS – that captured my attention. I’d watch friends and colleagues gasp for air while fighting PCP, waste away as a result of MAC or cryptosporidiosis, and literally lose their minds to toxoplasmosis, cryptococcal meningitis, AIDS-related dementia, or progressive multifocal leukoencephalopathy (PML). In turn, I heard my calling – to become an effective OI treatment activist and educator.

Soon after learning everything I could about OIs and beginning work on a number of campaigns to speed the development of life-saving treatments, OI rates plummeted, thanks to the widespread use of combination antiretroviral treatment that began in 1996. But OIs still occur, and there is growing concern that the increasing number of people failing antiretroviral therapy will soon result in a rise in the number of AIDS-related OIs and deaths. In turn, there will always be a need for effective OI treatment activism, along with comprehensive and up-to-date information that can help all HIV-positive people better understand OIs, including the ways in which they can be prevented and treated.

Because there have been significantly fewer people with HIV experiencing OIs over the past six years, the flow of OI-related treatment research has been reduced to a trickle. However, there have been a number of advances. What follows is an overview of some

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**Standard of Care Treatment vs. ZEST Once-Daily Regimen**

This trial will study whether people on their first HAART regimen who take their drugs two or more times a day can switch to a once-daily regimen. People in the trial will either remain on their current medications, or switch to Zerit XR, Epivir and Sustiva (ZEST) taken once daily. They will visit ACRIA 9 times over 11 months. All blood tests, study visits, and study medications (Zerit XR, Epivir & Sustiva), as well as medications from the Standard Of Care arm that are manufactured by the sponsor, will be provided at no charge to the participants. Prescriptions will be written for any other anti-HIV drug. You are eligible if you are HIV-positive, age 18 or over, and on an initial HAART regimen (one or more NRTIs, at least one agent must have a twice-daily dosing schedule, and no NNRTI in the past or in current regimen) with a viral load below 50. Study participants will be reimbursed $25 for each visit.

**RESIST 1: Tipranavir in Multi-Drug Resistant Patients**

This trial will study the safety and efficacy of tipranavir (a protease inhibitor) boosted with low-dose ritonavir in people who have taken multiple antiretrovirals. This study lasts about 11 months, with 10-12 visits to ACRIA. All blood tests, study visits, and study medications will be provided by the sponsor. All patients must have taken drugs from each of the three antiretroviral classes, have taken at least two protease inhibitors, have a viral load over 1,000, and must currently be taking a protease inhibitor. Patients who do not qualify will be screened for RESIST II, another trial of tipranavir. Participants will be reimbursed $25 for each visit.

For the above trials, contact Dr. Douglas Mendez at 212-924-3934 ext. 126 or Dr. Yuriy Akulov at 212-924-3934 ext. 124

**Social Networks and Barriers to Healthcare for Adults Over 50**

The purpose of the study is to understand the informal networks of people and the social services in the lives of older people with HIV/AIDS. People will fill out a survey about the types of daily assistance they need, who they ask for it, and the kinds of services and treatments used to manage their health. Participation is confidential and people receive $25 for their time.

**Using Other Treatments to Manage HIV/AIDS**

People in this study will be interviewed and complete a brief survey about treatments other than antiretrovirals, such as supplements, exercise, acupuncture, herbal remedies, etc, as well as who provides the treatments, how often they use them, and if they feel they are helpful. Participation will require approximately 90 minutes, and participants receive $25 for their time. To qualify, people must be HIV+, using HAART for at least one year and be using other treatments for HIV/AIDS.

For both studies, contact R. Andrew Shippy, MA at 212-924-3934 ext. 104, or email: ashippy@acria.org

**HIV Over 50 Database**

ACRIA is currently establishing a large cohort to conduct research on HIV+ people over 50. If you would like to be included in this database, please contact Salone Howard at 212-924-3934 x105 or email showard@acria.org.

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**Editor’s Notes**

- All material in ACRIA Update is presented for educational and informational purposes only, and is not intended as medical advice. All decisions regarding one’s personal treatment and therapy choices should be made in consultation with a physician.
- ACRIA Update refers to all drugs by both their commercial and scientific names upon their first reference in an article. Thereafter in the article, they will be identified with the name by which we feel they are most commonly known, either commercial or scientific.
Opportunistic Infections

of the more common OIs – including those that are still frequently documented in HIV-positive people – and some of the more important advances that have been made over the past ten years.

BACTERIAL INFECTIONS

Syphilis A number of bacterial infections continue to be a source of concern for many HIV-positive people, including those who are responding well to anti-retroviral drug treatment. One example is syphilis, caused by the bacterium Treponema pallidum, which has made something of a comeback in recent years. In New York City alone, syphilis rates more than doubled from 2001 to 2002, primarily among gay and bisexual men. Because syphilis is an “ulcerative” sexually transmitted disease (STD), meaning that it can cause open sores, it carries with it an increased risk of HIV transmission. Research indicates that syphilis is more likely to run a more rapid and harmful course of disease in people with HIV, including those with relatively healthy immune systems. In turn, modern-day treatment initiatives tend to be a bit more aggressive. HIV-positive patients generally receive three weekly injections of high-dose penicillin for the treatment of primary or secondary syphilis, as opposed to the one-time penicillin injection used to treat HIV-negative patients.

Tuberculosis Tuberculosis (TB) remains one of the most common causes of sickness and death among people with HIV. Even though 150,000 people in the United States have been infected with Mycobacterium tuberculosis, the bacterium that causes TB, most (between 90% and 95%) have immune systems that are healthy enough to prevent the bacteria from ever causing active TB. In people with HIV, the immune system may eventually lose control of the bacterium, causing the infection to spread and cause active disease. This process can take many months or years. In other words, Mycobacterium tuberculosis can remain alive in someone’s body for many years, but may only become active once the immune system becomes suppressed. TB has been seen in many HIV-positive patients with moderate CD4 cell suppression – meaning a CD4 cell count between 200 and 400 – and can actually result in more pronounced symptoms when the CD4 cell count is above 200.

To test for Mycobacterium tuberculosis infection, a skin test called PPD is performed. It is generally recommended that HIV-positive people receive this test on an annual basis, particularly if they are homeless, intravenous drug users, or incarcerated, due to the higher rates in these populations. PPD testing involves pieces of the bacteria that are injected directly into the skin. If someone has been exposed to the bacteria in the past, the immune system will immediately recognize the PPD, resulting in a firm, relatively large bump at the site of the injection. Depending on the size of the PPD bump – and taking into consideration other factors such as HIV status, age, immigration status, intravenous drug use history, and recent exposure to someone with active TB – treatment is sometimes deemed necessary. For HIV-positive people who were recently exposed to someone with active TB or develop PPD bumps that are 5 millimeters in diameter or larger – but do not have symptoms of TB – a nine-month course of isoniazid should be started.

...syphilis is more likely to run a more rapid and harmful course of disease in people with HIV, including those with healthy immune systems.”

For HIV-positive patients who are PPD positive and have signs and/or symptoms of disease – such as an abnormal chest x-ray and difficulty breathing – TB is suspected but must be confirmed by actually isolating the germ in the laboratory. The recommended treatment for active TB is almost the same for HIV-positive people as it is for HIV-negative people. For the first two months, a combination of four antibiotics is taken. After that, a combination of two drugs must be taken for an additional four months. However, one of the drugs typically used for the entire six months of treatment – rifampin – does not mix well with anti-HIV drugs. And because it is generally recommended that HIV-positive patients either start or continue their anti-HIV drugs while being treated for TB, researchers have determined in recent years that another, similar antibiotic – rifabutin (Mycobutin) – can be used as a substitute for rifampin. Although the dose of rifabutin may need to be reduced, depending on which anti-HIV drugs are being taken, it can be combined safely with other medications.

Bacterial Pneumonia and Bronchitis

Research has consistently found that bronchitis and bacterial pneumonia are more likely to occur in HIV-positive people, regardless of their CD4 cell counts. In other words, people with HIV – even if their CD4 cell counts are high – are more likely to experience bacterial respiratory problems than HIV-negative people. What’s more, HIV-positive people with suppressed immune systems are much more likely to see a mild case of bronchitis develop into serious pneumonia, which can be life-threatening if not diagnosed and treated promptly.

For people with HIV, respiratory problems are nothing to cough at. No matter how high the CD4 cells or low the viral load, difficulty breathing – whether or not a cough is present – should be brought to the attention of a healthcare provider.

Mycobacterium Avium Complex (MAC) MAC, which can affect the liver, the spleen, and the bone marrow and cause severe symptoms (fever, weight loss, and

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...diarrhea, to name a few), is typically seen in patients with fewer than 100 CD4 cells. Because antiretroviral treatment has allowed many HIV-positive people to keep their CD4 cells above and beyond this level, MAC rates are at an all-time low. However, MAC has long been a serious threat to HIV-positive people and effective preventive medications – known as prophylaxis – remain the best tools to prevent MAC from occurring in people with suppressed immune systems.

The drugs typically prescribed to prevent MAC are azithromycin (Zithromax), which is taken once a week, or clarithromycin (Biaxin), which is taken once a day. These drugs replaced rifabutin (Mycobutin) as the prophylaxis of choice back in the mid-1990s. Clinical trials have demonstrated that they are more effective than rifabutin – both clarithromycin and azithromycin, if taken correctly, can reduce the risk of MAC by 70% – and are much less likely to interact with anti-HIV medications. Clarithromycin should not be used with Sustiva (efavirenz), since Sustiva can lower blood levels and, therefore, the effectiveness of clarithromycin.

Once upon a time, it was generally recommended that, once an HIV-positive person started on MAC prophylaxis, it should be continued for life. This is no longer the case for patients who are able to increase their CD4 cell counts to levels above 100 – and keep them there for at least three months. Similarly, patients who experience MAC have long been told to continue treatment for life to prevent the disease from recurring. We now know that such maintenance therapy – which is also known as “secondary” prophylaxis – can be discontinued if CD4 cells can be pushed above 100 for at least six months.

**VIRAL INFECTIONS**

**Cytomegalovirus (CMV)** Rates of CMV, a viral infection that can affect the eyes and a number of other organs in the body, remain low in the United States. When CMV does occur, a number of treatments are available, thanks to a tremendous amount of research conducted throughout the 1990s. These include medications administered intravenously (foscarnet [Foscavir], ganciclovir [Cytovene], or cidofovir [Vistide]), by mouth (valganciclovir [Valcyte]), or directly into the eyes (ganciclovir implants [Vitraset] or fomivirsen injections [Vitravene]). The most important advance in recent years can be found in studies suggesting that CMV maintenance therapy – usually oral ganciclovir (Cytovene) – can be stopped in patients who have been treated for the disease and whose CD4 cell counts increase to levels above 100 or 150 for at least six months.

**Herpes Simplex Virus (HSV)** Herpes simplex virus 1 (HSV-1) is responsible for oral herpes (cold sores), and herpes simplex virus 2 (HSV-2) is responsible for genital herpes. While antiretroviral treatment has allowed HIV-positive people to avoid more serious herpes outbreaks, there are no guarantees that herpes sores won’t recur. Two drugs have been approved for herpes outbreaks over the past decade: valacyclovir (Valtrex), a form of acyclovir (Zovirax) that only needs to be taken once a day (Zovirax needed to be taken three to five times a day), and fomivirsen (Famvir). New treatments are also being developed for herpes, including a topical foscarnet cream; a topical gel of the anti-CMV drug cidofovir; and a topical cream with the code name SP-303T, a drug that has been shown to be active against a number of different viruses. Another topical drug being studied for oral and genital herpes is trifluridine, which is already approved as a “pre-cancer” form of disease. If cancer does occur, the four most common types are cervical cancer, anal cancer, rectal cancer, and penile cancer. If not diagnosed and treated early, these forms of cancer can be life threatening.

HIV-positive people are more likely to be infected with HPV than HIV-negative people. Because of immune-suppression, HIV-positive people are also more likely to develop genital warts, as well as cervical or anal cancer, as a result of HPV. Researchers don’t yet know if anti-HIV drugs will help reduce the number of new cases of genital warts or anal or cervical cancers. In fact, some researchers speculate that, because anti-HIV drugs have been successful in keeping people alive longer, the risk of developing genital warts or cancer might increase.

While a blood test can check for HPV infection, a positive result doesn’t really say much. Being infected with HPV does not mean that genital warts will develop, nor does it mean that dysplasia or cancer will occur. Genital warts – which can often be felt with a finger and are visible to the naked eye – should be reported to a healthcare provider. The warts should be biopsied (a sample collected in a minor surgical procedure) to determine if they might go on to cause cancer.

To check for dysplasia or cancer, a healthcare provider can perform a Pap smear, in which cells are scraped from the cervix or anus and examined under a microscope. If the cells are found to be abnormal, a second Pap smear should be conducted to confirm these results. Women should have their first cervical Pap smear by age 18 or when they become sexually active, whichever comes first. It is recommended that HIV-positive women have a cervical

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**Pacing Myself**

As a community scholarship recipient to this year’s Conference on Retroviruses and Opportunistic Infections, I looked ahead to the frigid mid-winter trip to Boston with both anticipation and a strong sense of anxiety. Anticipation at the chance to see many of my community activist friends from around the world, and anxiety at the deluge of scientific data that I would be challenged to sort through and make sense of. Despite my nerves and having to leave the relative tropical luxury of Los Angeles, I boarded my flight with my new Dell laptop (bought on credit) and headed east. This was my second Retrovirus Conference in as many years, and I was determined to learn as much as I could to share with my fellow community members back home at Being Alive Los Angeles.

Diagnosed with HIV in 1995, I didn’t have much of a science education under my belt before becoming a treatment educator and activist at an HIV women’s organization in 1998. It wasn’t until I really started to take an active stance in my own healthcare and advocate for others that I realized the importance of understanding the sophisticated realm of treatment information.

Although I had been a member of the AIDS Clinical Trials Group’s Community Constituency Group for two years, attending 2002’s Retrovirus Conference in Seattle was my first really serious science-based conference — and it left me numb for days afterward. I found myself sitting through the basic science presentations taking more notes on terms that I didn’t understand than on the actual presentation. The program said “basic” science, so I thought I would feel right at home… Ha! Some of my friends saw the glazed look in my eyes and helped guide me towards the type of sessions that would be a little more relevant and less frustrating.

Returning to LA from Seattle, I knew that, in order to become a more effective advocate for people with HIV and to keep up with other community activists and writers, I needed to know much more than I did. Rather than feel intimidated by my lack of knowledge, I began to look for ways to learn more within my own community.

As a positive step towards becoming more science-savvy, I took an advanced biology class at a local community college. I didn’t become an expert overnight, but I did manage to snag a nice fat grade of A. This basic background greatly increased my comprehension during the sessions at this year’s conference and has spurred me to continue working towards a degree. Another thing that really helped me focus and better prepare for this year’s conference was my involvement in ATAC (AIDS Treatment Activists Coalition). Individuals in this group have provided me with an overwhelming amount of encouragement and support during the past year. Knowing that many of them were going to be at the conference helped to decrease my anxiety.

Once in Boston, I was looking forward to Monday night’s opening session. Bill Clinton was to give the keynote lecture. Besides the fact that I had never seen a president in person, I was really curious about what he was going to say. Getting to the auditorium early, several fellow community members and I (very courteously) bogarted our way to the front row, so we had an excellent view of the presentations. Before Clinton took the stage, the Sinikithemba Choir from South Africa treated the audience to a lively round of African gospel. The music was nice, but knowing that all of the members of the choir are HIV-positive and only a few have access to the treatments being discussed at the conference weighed heavily on my mind. Towards the end of their program, one member walked to the podium and began to speak about her life with HIV, her struggles with opportunistic infections, and the hope that she had for the future. At one point she broke down in tears… and I lost it too. What she had to say set an important tone for the rest of the week. Her words were so eloquent and powerful that, in comparison, I found Clinton’s talk interesting, but fairly anticlimactic.

The rest of the conference was a whirlwind of activity. When I wasn’t attending plenaries and symposia, I was at poster sessions and oral abstracts. I came away feeling a lot better this year, because I learned to pace myself and stayed away from the heaviest scientific stuff. At night, I went to community meetings for ATAC and fed myself at pharma receptions. Because I was on a limited budget, these free meals really came in handy. I strategized with my fellow ATAC members over coffee during the breaks and grilled my super-smart community friends for answers to questions about stuff that I didn’t understand.

It was awesome being at the conference with so many prominent researchers and clinicians from all over the world. At least twenty different languages were being spoken in the elevator of my hotel (not unlike a New York subway car) and as many different medical and scientific disciplines among the attendees. The fact that 60% of the conference attendees were from other nations really demonstrates what a global issue AIDS is and what a truly global response is needed to find the answers we are searching for.

Attending Retrovirus gave me an eye-opening snapshot of the vast array of research being conducted around the world – from petri dish to clinical trial. I wish that there were some marvelous breakthroughs to report – something like the advent of protease inhibitors in 1995 – but the progress in general gives me a sense that better things are yet to come. The biggest challenge however, is not in finding new treatments for HIV, but ensuring that everyone has access to the ones we already have.

Cathy Olufs is a treatment activist and health educator in Los Angeles.
Metabolic Complications: A Look at the IAS Guidelines

by Heidi M. Nass

It seems like the longer HIV/AIDS is around, the more complicated it gets. Truthfully, science is still learning exactly how HIV (not to mention the immune system!) works. Then there are the treatments for HIV. They work for a lot of people, but they have to be used in just the right combinations, in just the right way… and they bring with them a whole other set of complications that have to be sorted out from the effects of HIV. On top of that, many people with HIV are living longer now – they’re getting older, and getting older has its own effects – higher rates of heart disease, diabetes and elevated cholesterol, for example – that have to be separated from those of HIV and those of the medications.

There’s also all the lifestyle and family history stuff. Do you smoke or drink? Do you exercise? What are you eating? Do your parents have heart disease? All these kinds of things can also play a role in HIV… and the treatments… and aging. It’s complicated.

Researchers are trying to sort through all of this, of course. At the top of their list is figuring out what’s going on with what they usually call metabolic abnormalities or metabolic complications. This refers to the body shape changes that happen to many people – arms and legs get skinny, middle section gets paunchy or thick, maybe a fatty lump between the shoulders or larger breasts. It also includes changes in the way the body handles sugars and fats, as well as bone disorders and disturbances that occur inside the cells and cause damage to the body.

At February’s 10th Conference on Retroviruses and Opportunistic Infections (CROI), researchers from all over the world presented results from studies that looked at the many aspects of metabolic complications, from what’s happening inside the cells of the body, to how common any one complication is, to possible treatments and interventions. In all, 51 posters and ten oral presentations addressed metabolic complications, including seven posters on the cardiovascular risks of antiretrovirals, and 12 on the pathogenesis and mechanisms of lipodystrophy.

Studies that look at metabolic complications are critical because all of these changes have been happening to people living with HIV, especially those who’ve been taking antiretrovirals since 1997 or so. They started to show up more often after people began using the combinations of medications we now refer to as HAART (Highly Active AntiRetroviral Therapy).

Since then, people have been asking the same questions about each of these complications: Am I likely to get this? Why is it happening? How is it happening? What can I do to stop it? Can I prevent it? So far, the answers to these questions have been, for the most part: “We’re not exactly sure.” In fact, some of the complications haven’t even had a precise definition or standard of measurement. For example, facial lipoatrophy can be defined as a loss of fat in the face but there is no standard way to measure it. This makes it difficult for clinicians, researchers and people with HIV to be sure that they’re all talking about the same thing when they use the term ‘facial lipoatrophy’ – let alone figure out how to treat it.

People with HIV and their physicians have been trying things like switching HIV medications to stop or maybe even reverse the body shape changes or adding medications designed to lower the levels of fats in the blood (cholesterol and triglycerides). But we’ve largely been figuring it out as we go along. Now, for the first time, we have some guidance.

A group of twelve researchers and clinicians began meeting in May 2000 to create a set of recommendations for physicians about how to best manage metabolic complications for people living with HIV, especially those who are taking potent antiretrovirals. The group was made up of specialists in fields like endocrine and metabolic disorders, antiretroviral therapy, and patient care. They were chosen by the International AIDS Society-USA, a not-for-profit physician education organization.

The panel reviewed scientific studies and data presented at research conferences over the last five years. For each metabolic complication that has been identified so far, one or more members of the group reviewed the evidence, presented it to the larger group, and then wrote a draft summary and list of recommendations. The whole panel eventually decided on the final recommendations for the management of each metabolic complication. This document, Management of Metabolic Complications Associated With Antiretroviral Therapy for HIV-1 Infection: Recommendations of an International AIDS Society–USA Panel, was published in the Journal of AIDS, Volume 31, Number 3, November 1, 2002.

The guidelines have a section on the following metabolic complications: insulin resistance and abnormal glucose homeostasis; lipid and lipoprotein metabolism abnormalities; body fat distribution abnormalities; lactic acidemia; and bone disease. Each section has three parts: background; recommendations for assessment and monitoring; and treatment.

More or less, the guidelines lay it out like this: here’s what we’ve learned; here’s what we can do; and here’s what we need to find out. It’s clear that some complications are better understood than others. The guidelines provide useful and much needed help, but...
also remind us of the work that remains to be done so that people with HIV may live long, healthy lives with the current antiretroviral medications. Here’s a summary of the guidelines’ contents.

**DIABETES, INSULIN RESISTANCE AND GLUCOSE INTOLERANCE**

**Background:** Diabetes, including the conditions that lead up to it – insulin resistance and glucose intolerance – was not particularly common in people with HIV before the advent of HAART. Now as many as 40% of people who use a protease inhibitor as part of their antiretroviral combination will develop insulin resistance and, as a result, have impaired glucose tolerance. This means that their bodies need more and more insulin, a hormone produced by the pancreas, to break down sugar, or glucose, in the blood. Eventually, if the pancreas can’t produce enough insulin or the insulin can’t break the sugar down properly, the level of glucose gets too high. This is called glucose intolerance. Other complications, including heart disease, may be influenced by glucose intolerance and insulin resistance.

It isn’t known whether insulin resistance brought on by antiretroviral medications carries the same risk of heart disease and other complications as it does in people without HIV. However, there is concern that this risk may be higher for people taking protease inhibitors who have other risk factors for diabetes, including being overweight or having a family history of diabetes.

**Recommendations:** People starting an antiretroviral regimen that includes a protease inhibitor should have their glucose levels checked before starting the medication, three to six months after starting, and at least once a year thereafter. There is some evidence to suggest that it may be best to avoid the use of a protease inhibitor in people whose glucose is too high to begin with or who have relatives with diabetes.

**Treatment:** There isn’t enough data from studies of people with HIV to know for certain what to do or which treatment is best for people with high blood sugar levels. Most of the recommendations in the guidelines are based on data from people without HIV infection and on the opinions of specialists. Losing weight if overweight, eating a balanced diet, and exercising are recommended for everyone, particularly for those with glucose intolerance.

If medications are needed to improve insulin sensitivity, the preference is for metformin (Glucophage) or insulin or others in a group of medications called thiazolidinediones, such as pioglitazone (Actos) or rosiglitazone (Avandia). However, these drugs require some caution. Metformin, for example, may cause a condition called lactic acidemia, and the thiazolidinediones can cause liver problems. Drugs are not recommended if there is evidence of insulin resistance but glucose levels are normal.

**ELEVATED LIPIDS: CHOLESTEROL AND TRIGLYCERIDES**

**Background:** Two kinds of fats, or lipids, in the blood – cholesterol and triglycerides – are the focus of the guidelines. These are the lipids that can eventually lead to heart disease if they get too high and stay high. Before HAART, people with HIV didn’t usually live long enough to worry about heart disease. Then combination therapy with protease inhibitors came along and it eventually became clear that the very drugs that were making it possible for people to imagine a future were increasing their risk for that other killer, heart disease, by increasing their lipid levels.

All protease inhibitors are not created equal when it comes to the extent to which each one affects levels of cholesterol and triglycerides in the blood, but the bottom line is the same. They increase both LDL (low density lipoprotein) cholesterol and triglycerides – the two most dangerous lipids for the arteries and, therefore, the heart.

While protease inhibitors seem to have the most profound effect on lipid levels, drugs from the other classes of antiretrovirals have also been implicated. There is also the suggestion that a person’s genetic predisposition may influence the risk of developing lipid problems.

So far, it’s been hard to assess the extent of risk caused by antiretroviral use for future heart disease. Retrospective studies, those that look back at people’s medical histories, include too many unknown variables to provide a clear picture, while prospective studies, those that look forward with the intent of finding answers to a specific question, haven’t gone on long enough yet to provide useful findings. The prospective study that may provide the most complete data so far is the D:A:D study, which was presented at CROI.

The D:A:D (Data on the Adverse Effects of Drugs) study looked at 23,468 people from around the world, comparing those on three-drug antiretroviral therapy (including a protease inhibitor and/or a non-nucleoside) to those who had not taken any antiretrovirals. The study ran from July 1999 to April 2001, with follow-up in August 2002. The researchers used a mathematical model to figure out the risk of myocardial infarction (heart attack) while on HAART for up to seven years. After adjusting for other risk factors, the researchers found that the risk and the incidence of myocardial infarction increased the longer a person was exposed to antiretrovirals. The risk did not become apparent until after a year on antiretroviral therapy.

The D:A:D study did not assess the relative risk of individual antiretrovirals. Notably, smoking and prior cardiovascular disease were associated more with myocardial infarction than was the use of antiretroviral therapy.

The authors of the D:A:D study noted that the risk of heart attack remains extremely low (126 incidents over the 21 months of observation, or less than 0.5% of all patients). They emphasized what is true for most aspects of HIV therapy – any potential risk must be balanced with the possible benefits of suppressing antiretroviral therapy.

**Recommendations:** There isn’t enough data from studies of people with HIV to know for certain how to monitor and treat lipid (continued on next page)
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abnormalities. Also, it isn’t known whether high lipid levels resulting from HIV itself or antiretroviral use carry the same risk of heart disease as in people who are HIV-negative. As with glucose levels, the guidelines suggest that people should have their lipid levels checked before beginning an antiretroviral regimen, three to six months after starting, and at least once a year after that. People who already have high lipids or risk factors for heart disease, or those with a family history should avoid protease inhibitors if possible.

When deciding whether and how to treat elevated lipids, the guidelines recommend that the decision include consideration of any other risk factors that a person might have. How old? Smoker? Diabetic? Family history of heart disease? High blood pressure? Menopausal? The decision should ultimately be made with all of these things in mind as well as how well the individual’s HIV is under control

Treatment: With no good clinical trials that establish the best treatment for elevated lipids due to antiretroviral use, the panel included in its review of evidence some data from studies of people not HIV-infected, as well as the opinion of experts. When possible, the guidelines recommend a switch from a protease inhibitor to a non-nucleoside-based (particularly Viramune [nevirapine]) or triple nucleoside antiretroviral regimen. The guidelines are clear, however, that antiretrovirals that might increase lipids should not be withheld from someone who is not able to switch from a protease inhibitor to a non-nucleoside-based or a triple nucleoside regimen.

As an example of how quickly things change, it’s likely that the suggestion to switch to a triple nucleoside regimen will be removed when the IAS guidelines are updated. In March, the AIDS Clinical Trials Group (ACTG) stopped the triple nucleoside arm of a study that was comparing three different combinations in people who had never taken antiretrovirals before. People who were taking just three nucleosides – AZT, Epivir (3TC) and Ziajen (abacavir) in the form of Trizivir – had viral loads that went above 200 sooner and more often than those who were taking Sustiva (efavirenz), a non-nucleoside, with either Trizivir or AZT and Epivir. The triple nucleoside regimen just didn’t work anywhere near as well.

There are no good clinical trial results that suggest whether changes in diet can affect lipid levels in people with HIV. Therefore, the guidelines’ recommendations are the same as those for all people trying to control their lipid levels – a diet that is limited in fats, losing weight if overweight, increasing physical activity, and decreasing alcohol consumption. Interestingly, the guidelines do not reference smoking, even though it is frequently cited as a risk factor for heart disease.

If switching the antiretroviral regimen is not an option and lifestyle changes don’t work, the guidelines suggest the use of medications to lower lipid levels. They lay out which particular drugs are best to use – pravastatin (Pravachol) or atorvastatin (Lipitor) to lower cholesterol and gemfibrozil (Lopid) or fenofibrate (Tricor) to lower triglycerides – the particular lipid levels at which they should be used, and potential drug interactions. The long-term effects of using lipid-lowering drugs for the treatment of lipids that are increased from antiretrovirals are not known.

REDISTRIBUTION OF BODY FAT

Background: This category of metabolic complications includes things like an accumulation of fat in the trunk of the body, a loss of fat in the arms, legs, face, and/or butt, and the lump of fat that some people get on their upper backs, called buffalo hump. Sometimes people get painful nodules of fat under the skin that look like tumors, called lipomas.

These body shape changes as a group are sometimes referred to as lipodystrophy, which just means that the body isn’t breaking down and processing fats properly. It’s been difficult to know just how many people with HIV experience changes in the way fat is distributed around their bodies – there hasn’t been a good definition, which makes it hard to diagnose with any consistency. Estimates of body fat redistribution are as high as 50% in people who are on antiretrovirals.

Several factors seem to be involved in this condition, including whether people are on potent antiretroviral therapy, how long they’ve been on it, how the medications have worked against the virus, and which specific medications they’ve been taking. There is a link between the nucleoside analogs and the loss of fat, for example, and a suggestion that protease inhibitors are linked more closely to fat accumulation. The non-nucleosides don’t appear to be involved in the development of these particular conditions.

There are likely other factors, too. A person’s age, sex, race and other individual factors may be part of what determines whether body shape changes occur. Exactly what is happening in the body to lead to these conditions and the way it happens is not known.

Recommendations: The ideal way to find out if there are changes in the distribution of body fat is to take an image of the body with a CT (computed tomography) scan or an MRI (magnetic resonance imaging). The problem is, these tests are very expensive and aren’t available to a lot of people. A cheaper and less complicated way to assess changes is to do various body measurements. However, it is very difficult to ensure that any two people measuring the same patient would get the same results or even that the same person would measure a patient exactly the same way on different occasions. It is also hard to detect subtle changes with measurements like these.

It is no surprise, then, that the guidelines say that no specific technique for routinely assessing or monitoring body shape
changes can be recommended at this time. No one test is specific enough, sensitive enough and predictable enough.

**Treatment:** In the absence of other metabolic complications, like elevated lipids, there is no agreement about whether to even try to treat body fat changes. Although many ways to address fat distribution abnormalities are being studied, none have been proven to work or approved for these conditions. Given that so many things seem to be happening at the same time, and there are potentially many different factors at play, the guidelines suggest it is unlikely that a single drug or intervention will address all the aspects of fat redistribution.

There have been studies looking at whether switching antiretroviral drugs might reverse some of the fat redistribution problems. So far, it looks like taking away the protease inhibitor – thought to be a factor in fat accumulation – does not help. However, replacing Zerit (d4T) with Retrovir (AZT) or Ziagen (abacavir) may increase fat in the extremities in people who have lost fat there, although the changes won’t necessarily look significant to the eye.

A study presented at CROI switched 61 people who were experiencing fat redistribution while taking combinations that included nucleoside analogs to a combination with no nucleoside analogs – the non-nucleoside Sustiva (efavirenz) and the protease inhibitor Crixivan (indinavir), boosted by Norvir (ritonavir). After almost one year on the new combination, the study participants experienced significant improvement in lipoatrophy but increased central fat accumulation.

The guidelines report diet and exercise as possible therapies and suggest that exercise, especially, might be useful for combating fat accumulation in the trunk. Resistance exercise, like weight lifting, has been found to help decrease fat and increase muscle mass. No special diets have been found to be helpful, but any diet that results in fast weight loss is not desirable because it can speed up the loss of muscle tissue. The guidelines suggest a healthy diet.

Some hormone interventions have been studied a little, but the guidelines do not recommend them at this time. Growth hormone (Serostim) may reduce central fat accumulation, for example, but the best doses haven’t been established and there are potential negative side effects, including glucose intolerance, insulin resistance, and fluid accumulation in the extremities.

Based on what’s known, testosterone replacement may benefit some HIV-positive men with low levels of the hormone and increased fat in the trunk. The guidelines don’t recommend this as a therapy, however, because there aren’t yet any results from studies looking at its use in men with HIV and fat distribution abnormalities.

Some drugs, like rosiglitazone (Avandia) and pioglitazone (Actos), have been studied but have not shown consistent evidence of improvement of fat redistribution. Triglyceride and cholesterol levels increased in one study of rosiglitazone. More studies are ongoing, but the guidelines do not recommend the use of these drugs based on currently available data.

Although not discussed in the IAS guidelines, many researchers are paying considerable attention to surgical and cosmetic interventions for changes in body fat distribution. The results of several such studies were presented at CROI. Two studies from France looked at the use of polylactic acid (New-Fill) for severe fat loss in the face.

The first study followed 50 patients for two years after their initial polylactic acid injections. They received injections every two weeks for six weeks, for a total of four sets of injections. The benefits of New-Fill included clear, observable aesthetic improvement and better quality of life.

In the other study, 40 patients received New-Fill injections twice, 15 days apart, and were evaluated after each injection and periodically up to six months later. In this case, patients reported no significant improvement in quality of life, although there was clear visual improvement measured photographically. Neither study reported any serious side effects.

**LACTIC ACIDEMIA**

**Background:** Lactic acidemia, sometimes called lactic acidosis, means an elevated level of lactic acid in the blood. The symptoms include fatigue, weight loss, nausea, abdominal pain, difficulty breathing and heart rhythm disturbances. There may also be abnormalities in the liver, although symptoms of liver abnormalities are much harder to identify. Lactic acidemia can come on suddenly or slowly, sometimes without any symptoms at all.

It is linked to the use of nucleoside analogs, especially with treatment lasting longer than six months. While all aspects of lactic acidemia are not understood, it is thought to be the result of damage that occurs inside the cells, involving the mitochondria, the cells’ energy source.

There are no good data regarding the incidence of lactic acidemia by gender, race, or age, although pregnant women may be at higher risk.

**Recommendations:** There is no way to predict who will get lactic acidemia, or when. Mild acidemia does not seem to predict more severe acidemia. The guidelines do not recommend routine screening in the absence of symptoms. If symptoms do occur, lactate levels should be measured and, if elevated, confirmed by a second test.

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Stay There

When I got back from the 10th Conference on Retroviruses and Opportunistic Infections in February, my friend and colleague John Bell asked me, "So how did it go?" I told him it was great, but that I had understood less than half of the information presented at the conference. He looked at me, thought for a second, and then gave me an approving nod and thumbs up. This wasn't a "Great job! The glass is half full" type of nod and thumbs up; this was, "Thank God. The glass is half empty. Now stay there."

John's nod and thumbs up is about accountability - what information do you need to learn, who is the information for, and how does it need to be presented? John and I teach a treatment education and activism class called TEACH Outside for people who have recently been released from prison. Most of the class members found out their HIV status while in prison. They were released with no support systems and the threat that they were going right back inside if they couldn't figure out how to be okay. The TEACH Outside curriculum is basic - you can live healthy with HIV, services are available to support you, activism has put all of this in place, and you can be an advocate for yourself and your communities. We don't ask people to lock in on the workings of antivirals or the ins and outs of metabolic complications. We ask them to take the first step. If you overload people, you've lost them, and we can't afford to lose anyone.

When I applied for a community scholarship to attend the conference, I didn't know what I was getting myself into. I hadn't been to a scientific meeting like this before. Most of what I know I've learned from treatment activists. So I left for Boston with high ambitions of returning to Philadelphia with enough information to update the TEACH Outside curriculum and create a new presentation on HIV/hepatitis C co-infection.

From my first hour in Boston on Monday, it was obvious that I was in over my head. People were bustling around, intensely discussing the legitimacy of so and so's research on this and that, and using more acronyms and shorthand than I had ever heard from my activists friends when referring to the alphabet soup of activist organizations. Looking through the conference schedule didn't offer much reassurance. I gave up trying to figure out the complicated scientific titles of the abstracts and looked for sessions that matched my own interests as an activist and educator.

I talked to one of my activist friends that night about how to approach the conference. She said that most of the information is either "basic science" or "clinical!" On Tuesday, I realized that "basic science" isn't basic at all; it refers to all the abstracts whose titles I couldn't understand. So after my first "it must be smart, because I can't understand what's being said" session, I headed off to the next session with a friend who helped me follow the presentations by explaining the scientific jargon.

Don't lock in. In TEACH Outside, we tell people, "Don't lock in on the information. You'll always be able to go back to it. For now, just listen, and don't worry if you're overwhelmed. It will start to make sense if you give yourself time."

Tuesday evening, I made two promises to myself. First, I was going to sit through sessions, even when I didn't understand what was being said. Second, I wasn't going to take notes; I was only going to write down terms I didn't understand and look them up later.

Make the information your own. At the start of each TEACH Outside class, most of the members aren't sure how they're going to make it through the class. They don't have a place to put the information. By the end of the class, people still haven't mastered all of the information, but they have created a place to put the information; and they have the tools and resources to make that information their own.

Wednesday night, I went to a meeting the AIDS Treatment Activists Coalition (ATAC) folks were having on T-20 – side effects, pricing, expanded access, Phase IV trials, the whole nine-yards. Up until this point, I had been struggling to make the connection between the information being presented in the conference sessions and my work. Since becoming an AIDS activist, I've focused mostly on treatment access issues – international trade regulations that stand in the way of HIV medication access throughout the world, and prison health care providers who deny necessary HIV treatment because it cuts into profit margins. I knew about emerging treatment information only in so far as it affected what we were demanding for international or prison treatment access. The ATAC meeting helped me connect the dots between emerging HIV scientific research and my activism. Thursday and Friday I went to the conference sessions with a clear focus: as we expand our understanding of HIV and explore new treatment options, treatment activists need to push the questions: (1) how effective is this? (2) what are the real side effects? (3) what is not being said? and (4) who will be able to access this? For the first time that week, I wasn't struggling to follow every detail of the presentations; I was trying to figure out what was being left out.

Take action. In TEACH Outside, we talk about how all of the HIV services and medications now available were put in place by activism — people com -
Metabolic Complications

(continued from page 9)

Treatment: There are no substantial clinical trial results that evaluate interventions for lactic acidemia in people with HIV. Other than stopping any nucleosides, no proven interventions exist for lactic acidemia. Vitamins B-1 (thiamine), B-2 (riboflavin), C, E and K, coenzyme Q10 and L-carnitine have been used with some success to treat lactic acidemia in genetic mitochondrial diseases, but there are no data supporting their role in treating this condition when it’s caused by nucleoside analogs.

Bone Disease

Background: The death of bone tissue, what’s called osteonecrosis, has been associated with HIV since at least the late 1980s. However, it appears to have become more frequent since the widespread use of HAART. Generally, the bone dies because there isn’t sufficient blood circulation. It most often affects bones in the hip area, but it may involve other areas, like the shoulder and wrist. Osteonecrosis has been linked to elevated lipids and the use of steroids, but not the use of any specific antiretroviral medications.

Osteoporosis, a decrease in bone mineral density that can cause bone fractures, was rarely seen in HIV before the use of antiretrovirals. As with osteonecrosis, the incidence of osteoporosis has also increased with the widespread use of HAART. It may be associated with the use of protease inhibitors, but the link to specific antiretrovirals isn’t well established. Exactly how and why it happens are also not understood. Thankfully, bone fractures in people with HIV are rare.

Recommendations: The guidelines do not recommend routine screening for bone disorders.

Treatment: The only effective therapy for symptomatic osteonecrosis is surgical joint replacement and resection of the involved bone. It is recommended that everyone have an adequate intake of calcium and vitamin D, ideally through diet, and do weight-bearing exercise, like walking or yoga.

In Conclusion

There are a few unfortunate themes that run through the IAS guidelines. One of them is expressed as something like “the precise mechanisms are not known” or “the pathophysiologic basis is unknown.” The other one is, basically, “Few studies have been completed to guide the optimal monitoring and treatment.” In other words, we don’t know exactly what’s going on, and we often don’t know quite what to do about it.

Maybe these still rather mysterious metabolic complications are by-products of the accelerated approval of antiretroviral drugs – getting drugs out faster to save people’s lives requires the forfeiture of knowing all the side effects that they might cause once they’ve been used for a few years. Or they may just add to an already complicated disease process further complicated by treatment.

In the end, it doesn’t much matter. “Am I likely to get this?” “Why is it happening?” “How is it happening?” “What can I do to stop it?” “Can I prevent it?” These are the questions that really matter. The numerous studies presented at CROI looking at metabolic complications attest to the serious attention that research is devoting to these questions. Time – but hopefully not too much more time – will give us some answers.

Heidi M. Nass is an HIV-positive community advocate based in Madison, Wisconsin.

by Laura McTighe

As I get involved with ATAC, I’m reminded of John’s advice: “stay there.” There are a lot of questions I still don’t have answers to... What information do I need to learn? How do I keep from getting bogged down in treatment information? Do I need to set boundaries for myself to keep from getting so engrossed in the information that I can’t pull myself out of it and teach? How do I balance learning more about treatment, the parole system, prison policies, and social services available for people with HIV who are recently incarcerated?

As John says, if you aren’t what you appear to be, this group of people will see it first. So as we prepare to start the next TEACH Outside class, these questions weigh heavily on my mind. It was the lessons of TEACH Outside that got me through the conference. Now I have to figure out how to bring the conference to the members of the next TEACH Outside class.

Laura McTighe is Prison Activities Coordinator at Philadelphia FIGHT.
AIDS-Related Cancers: An Update

by Jeffrey T. Schouten, MD

The “modern era” of antiretroviral therapy has resulted in a significant decrease in the number of people who develop AIDS-related cancers. Obviously, this is great news for people living with HIV. But it has presented a challenge to the conduct of clinical trials for the treatment of AIDS-related cancers since fewer people are available to participate in these trials.

The AIDS Malignancy Consortium (AMC) is a group of 15 major medical centers funded by the National Cancer Institute (NCI) that conducts research in the field of HIV-related cancer treatment. Along with Michael Marco and Jeff Taylor, I am one of three community representatives who serve on the AMC’s Steering Committee. The AMC research effort focuses on the two most common cancers seen in people with HIV, Kaposi’s sarcoma and lymphoma. Recently, the AMC has also begun developing trials for human papillomavirus (HPV)-associated precancerous lesions and anal cancers.

Kaposi’s Sarcoma

Kaposi’s sarcoma (KS) is an abnormal cancerous growth of blood vessels associated with a herpes virus known as KSHV (KS-herpes virus) or HHV-8 (human herpes virus type 8). KS most commonly appears as flat or raised purple spots on the skin. For unknown reasons, HIV-associated KS is primarily seen in men who have sex with men, but very rarely in people with hemophilia or injection drug users. KS can occur in women, but it is much less common than in men. It is not known how KSHV is transmitted, but it is present in saliva as well as blood and genital secretions. KSHV infection in gay men has been associated in some studies with a higher than average number of sex partners, various sexually transmitted infections, and the use of poppers (amyl nitrate). It is not known how these associations are related to the development of KS, particularly in the case of amyl nitrate. The incidence of KSHV infection is twice as high in HIV-positive gay men, compared to HIV-negative gay men.

Cases of AIDS-related KS have decreased by 90% in HIV-positive gay men since the availability of combination antiretroviral therapy in 1996. In Mediterranean countries and Africa, a non HIV-associated form of KS has been present for many years, which usually presents as KS lesions on the feet and legs in elderly men and rarely causes death. This non HIV-related form of KS is also associated with KSHV infection. In Africa, KSHV infection appears to be acquired quite early in life, and it is not known how the infection is transmitted. It does not usually appear to be transmitted at birth.

KS involving the skin usually does not cause life-threatening problems. However, KS can also involve the internal organs, particularly the intestinal tract and the lungs, which can lead to death. Additionally, KS can invade and obstruct lymph node groups, causing blockage in the flow of lymph. Very serious leg swelling can result if the lymph nodes in the groin are blocked. Also, if the lymph nodes around the intestines have extensive KS, intestinal absorption can be greatly impaired. Although the incidence of KS has declined dramatically, it still remains a problem for many people.

Besides antiretroviral therapy for HIV, the most significant advance in the treatment of KS in the last few years has been the chemotherapy drugs DaunoXome (daunorubicin citrate liposome injection) and Doxil (doxorubicin HCl liposome injection). These drugs use encapsulated liposomal formulations — small amounts of the drug are encased in fat bubbles called liposomes. The liposomal formulation increases the amount of time the drug stays in the blood and improves delivery of the drug to the tumor site. Based on the FDA approval, DaunoXome is indicated as a first line chemotherapy for advanced HIV-associated KS and is not recommended for patients with less than advanced HIV-related KS. Doxil is indicated for the treatment of AIDS-related KS in patients with disease that has progressed on prior combination chemotherapy or in patients who are intolerant to such therapy.

People with KS who begin antiretroviral therapy have up to a 50% chance that suppressing HIV alone, with the subsequent strengthened immune system, will result in good control of the KS. One recent study has shown that treating HIV with antiretroviral therapy results in significant decreases in KSHV viral load in the blood, probably due to the strengthened immune system. The response rates to Doxil or DaunoXome, which are administered intravenously once every three weeks, range from 50-75% in clinical trials. Another chemotherapy drug that can suppress KS is Taxol, which is also administered intravenously every couple of weeks. In general, DaunoXome and Doxil are used as first line treatment since they have fewer side effects than Taxol. But the dose of Taxol used to treat KS is much lower than when it is used to treat other cancers, such as breast cancer, and it is pretty well tolerated. Additionally, Taxol may have an added advantage to DaunoXome and Doxil in that tumors seem to have a better and longer response to Taxol. It is no longer necessary to treat KS with combinations of three chemotherapy drugs, as was the case only 5-10 years ago. The chemotherapy combinations resulted in no greater control rates, but much higher rates of serious side effects.

There are also topical, locally applied treatments for KS lesions. The FDA approved a topical compound, Panretin Gel (alitretinoin), in 1999 for the treatment of KS. Local treatments may be useful if there are only a few lesions needing treatment. KS also responds well to low doses of radiation therapy, which is sometimes used to control localized disease. Other local treatments include freezing the lesions or injecting chemotherapy drugs directly into the KS lesions. The only role of surgery is to perform a biopsy to establish a diagnosis. Removing lesions surgically is not usually helpful due to the number of lesions and the high likelihood of developing new ones after surgical removal. Usually, if someone has a significant amount of KS requiring treatment, chemotherapy is more effective than local, topical applications.
A major reason for ongoing interest in KS research is the association between a viral infection, KSHV, and a cancer, KS. Learning more about how a virus causes cancer could provide very important insights into the causes and treatment of other cancers. The most exciting new agents being tested for the treatment of KS and some other solid organ cancers are agents that inhibit the growth of new blood vessels, known as “angiogenesis inhibitors.” Since the actual KS tumor is compromised mostly of new blood vessels, hence the purplish color, inhibition of blood vessel growth might be a good way to reduce and control KS. The AIDS Malignancy Consortium has conducted several KS trials evaluating these agents. However, to date, the results, evidenced by reductions in KS lesions, have been rather disappointing.

One very significant challenge to the use of angiogenesis inhibitors is determining the best dose to use. Normally, early in drug development, the highest tolerated doses are identified in small trials. The dose is determined by the rate of side effects observed as higher doses are administered. Despite conventional testing of various doses, the best dose still has not been found. Many of the angiogenesis inhibitors can be given at very high dosages without significant side effects. One possible explanation for the low response rates seen with angiogenesis inhibitors is that a large enough dose was not given. Since they work by inhibiting blood vessel growth, we need an accurate biologic assay to determine the dose needed to inhibit blood vessel growth. This is a new concept for determining drug dosing, and there are no standardized methods to accomplish this task.

Another significant challenge today in conducting research on the treatment of KS is that the number of people requiring treatment has dropped dramatically since 1996. KS is now pretty well controlled with antiretroviral therapy and DaunoXome or Doxil. Thus, these excellent clinical improvements have made it difficult to conduct studies of the newer agents to treat KS.

**Lymphoma – Cancer of the Lymph Glands**  
Lymphoma is a cancerous growth of lymphocytes, a type of white blood cell that is part of the immune system. Lymphoma usually starts in the lymph glands. The major collections of lymph glands in the body are in the neck, under the arms, in the groin, and inside the belly. The most common symptom of lymphoma is rapid growth in one or more lymph gland. Because they filter out foreign substances from the body, normal temporary swelling of the lymph glands is not uncommon. An example of this is swollen lymph glands in the neck when you have a sore throat or a cold. However, when

there has been significant swelling in a localized lymph gland of greater than one inch, for more than 4-6 weeks, with no obvious cause such as a localized skin infection, consideration should be given to having a biopsy performed to see if the swelling could be due to lymphoma. Generalized symptoms such as fever, night sweats, and weight loss can also be caused by lymphoma. Because many of the lymph glands are just beneath the skin, a lymph gland biopsy may be a relatively minor surgical procedure.

With current antiretroviral therapy, the overall incidence of lymphoma in HIV-positive people is declining, but not as much as the decline seen in KS. Lymphoma occurs more often in Caucasian men and people with blood clotting disorders, such as hemophilia. People born in the Caribbean and Africa have much lower rates of lymphoma. It is not known why this is. The most common type of lymphoma seen in people with HIV is non-Hodgkin’s lymphoma (NHL). Hodgkin’s lymphoma (HL), which generally has a higher cure rate than NHL, was not seen frequently in HIV-positive people in the past. However, there appears to be an increase in the percent of lymphoma cases of the Hodgkin’s type recently in HIV-positive people, even though the total number of people with lymphoma appears to be declining.

Fortunately, the use of antiretroviral therapy has resulted in a dramatic decline in cases of one of the most aggressive and deadly lymphomas, that of the brain – central nervous system (CNS) lymphoma. In fact, the AMC has been unable to enroll enough people in its most recent CNS lymphoma trial and has closed the trial due to the rapidly declining incidence of CNS lymphoma.

There has also been a decline, though not as significant, in some of the other more aggressive cell types of lymphomas. However, there still remains an ongoing need for clinical research into the treatment of HIV-associated lymphomas.

Prior to 1996, people with HIV-associated lymphoma had an average survival of only six months due to much weakened immune systems. In a trial conducted before the use of combination antiretroviral therapy, the AIDS Clinical Trials Group (ACTG) compared the use of standard-dose to low-dose m-BACOD chemotherapy in people with AIDS-associated NHL. m-BACOD is a combination of six chemotherapy agents: methotrexate, bleomycin, doxorubicin, cyclophosphamide, vincristine, and dexamethasone. The study found that the standard dose of m-BACOD had a greater rate of serious side effects but no better outcome measured by either response rates or survival. While low-dose m-BACOD is a treatment option for lymphoma, other chemotherapy drugs that have less toxicity are more commonly used now.

With the use of antiretroviral therapy, both treatment and recovery from lymphoma seem to have improved. Over 50% of people treated with chemotherapy have

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a good chance of being cured. Generally, if there is a recurrence after treatment for lymphoma, it occurs within the first couple of years. So the longer someone remains free of disease after treatment, the less likely the chance of recurrence. Antiretroviral therapy has allowed standard doses of chemotherapy to be used with fewer complications, and strengthened immune systems have resulted in much higher remission rates.

The current standard chemotherapy regimen for non-Hodgkin’s lymphoma is called CHOP, which consists of cyclophosphamide, doxorubicin, vincristine, and prednisone. The first three drugs are given intravenously on the first day of treatment, and the prednisone is taken orally for five days. The “cycle” is repeated every three weeks, four to six times. The AIDS Malignancy Consortium has just completed a large non-Hodgkin’s lymphoma trial, number 010, which enrolled over 150 people. The trial data are being analyzed, and results should be presented late this spring. The 010 trial compared CHOP to CHOP plus rituximab, a monoclonal antibody that attacks lymphoma cells. (Monoclonal antibodies are drugs, administered intravenously, which contain one specific antibody that attacks a specific target.) Rituximab attacks a site on the surface of the lymphocyte known as CD20. Most HIV-associated lymphomas have CD20 present on the lymphoma cell surfaces. Rituximab plus CHOP has already been shown to be better than CHOP alone in people with lymphoma not associated with HIV infection.

The next AMC non-Hodgkin’s lymphoma trial to begin, number 034, will study a chemotherapy regimen that is probably more potent than CHOP. The regimen is called EPOCH. It requires a four-day continuous intravenous infusion of etoposide, vincristine, and doxorubicin, with cyclophosphamide added on day five, and prednisone given orally on days one through five. The “cycle” is repeated every three weeks, for two to six cycles, depending on response. The 034 study will determine if treatment results are better if rituximab is given with the EPOCH or afterwards. A total of 70 people will be enrolled in this trial. Trial participants and their healthcare providers will be able to decide whether or not to continue antiretroviral therapy during the chemotherapy treatment period.

A trial conducted by the National Cancer Institute (NCI) using a similar four-day infusion regimen, CDE (cyclophosphamide, doxorubicin, and etoposide), showed very high response rates, with few relapses in HIV-associated NHL. In the NCI study, all anti-HIV treatment was stopped during the chemotherapy treatment period because of concerns over possible drug interactions. For people who have recurrent lymphoma, or fail to get a good response to the initial treatment regimen, there are some trials investigating bone marrow transplantation and other chemotherapy agents.

**Human Papillomavirus and Anal Cancer**

Human papillomavirus (HPV) is associated with cancer of the cervix in women and anal cancer in men and women. Precancerous changes caused by HPV are called “intraepithelial neoplasia” (IN) or dysplasia – CIN for the cervix and AIN for the anal region. There is a great deal of controversy around the topic of screening for AIN with anal Pap smears in men and women, with no uniform standard of care.

There are several issues about routine screening for precancerous anal changes. First, the technique requires specific training in the performance of anal colposcopy. This is an examination of the anal canal with a magnifying anoscope. Second, there is no proven treatment for precancerous changes. Unlike in the cervix, abnormal tissue cannot be readily removed from the anal canal. Approaches have included surgical excision or removal using electricity or laser. Lastly, and perhaps most importantly, there are no data to show that any currently available treatment will lower the long term chances of developing anal cancer. Men who have sex with men are at increased risk for anal cancer, as is anyone who has anal intercourse. What is surprising, and reassuring, is that the “precancerous” changes in the anal canal observed in many HIV-positive gay men have not resulted in a much higher rate of cancer compared to HIV-negative gay men – at least not yet. [See Tim Horn’s discussion of HPV on page 4.]

Dr. Joel Palefsky of the University of California in San Francisco is evaluating a type of treatment, an instrument called an infrared coagulator, to destroy precancerous anal lesions. There are a couple of AMC trials studying AIN and anal cancer and one in development to minimize damage to the rectal lining, the mucosa, during radiation treatment for anal cancer.

**Challenges to Continued Research**

The increasing focus on the international AIDS epidemic and, hopefully, more widespread affordable treatment should present new research opportunities to study KS in Africa where both KSHV and HIV infection rates are very high. Because of the high rate of acquisition of KSHV early in life in Africa, understanding how KS is transmitted may be best learned there.

The greatest challenge currently facing AIDS-related cancer research is securing adequate funding to maintain a multi-center clinical trials network. This is particularly critical to the research effort. With fewer people to enroll into trials, the importance of a network of sites can not be overstated. Although the National Cancer Institute now funds the AIDS Malignancy Consortium, the current grant expires next year. The NCI has indicated that it may not refund the AMC after next year, has already begun to close some trials, and is not allowing any new trials to be developed. This field cannot move forward without an adequately funded multi-center trials network. If you think that funding to support AIDS-related cancer treatment is important, share your concerns with the NCI. As with so many advances in AIDS-related research, community pressure can have an enormous impact.

Jeffrey T. Schouten, MD is a Community Representative to the AIDS Malignancy Consortium.
Pap smear every six months. Men and women who practice anal sex should also have regular anal Pap smears.

An abnormal Pap smear result calls for closer examination. At this point, an anoscope or colposcope – two magnifying devices – are used to look for cancerous or pre-cancerous patches, or lesions, inside the anus or cervix. These lesions are often referred to as either anal or cervical intraepithelial neoplasia (AIN or CIN). If lesions are found, a biopsy can be performed to learn more about the abnormal cells.

Depending on the results of the biopsy, AIN and CIN are given a stage number: I, II, or III. The stage of dysplasia depends on the thickness of abnormal cells within the cervical or anal wall. AIN or CIN I is considered to be a mild form of dysplasia and generally does not require therapy (but must be monitored closely), whereas AIN or CIN II or III are considered to be more advanced forms of dysplasia and are more likely to develop into cancer. Advanced forms of AIN or CIN often require therapy to prevent them from developing into cancer.

The only treatments available are those to remove or destroy irregular cells, such as those that make up genital warts or cervical/anal dysplasia or cancer. Treatments aimed at the underlying cause of these problems – HPV – are still being studied.

Therapy for genital warts and low-grade dysplasia is not required, but is often recommended to prevent them from progressing. Intermediate and high-grade dysplasia, as well as cervical or anal cancer, almost always require therapy to prevent them from becoming life-threatening problems.

Treating warts, dysplasia, and cancers depends on the location and severity of disease. For example, topical gels and creams – such as podofilox (Condylox), podophyllum (Podocon-25), trichloroacetic acid, and imiquimod (Aldara) – are used only for the treatment of genital warts. In general, they have shown to be 30% to 80% effective in reducing wart size, sometimes dramatically. All topical treatments, with the exception of imiquimod, can be used to treat warts inside the anus or vagina. Cervical or anal dysplasia, particularly AIN or CIN II or III, are treated using cryotherapy (liquid nitrogen to freeze warts), laser treatment (using a high-powered light beam to burn and remove abnormal anal and cervical tissues), and surgery. Cervical and anal cancer (carcinoma) are treated like other forms of cancer. Radiation and/or surgery are often necessary to either destroy or remove the cancer and the surrounding tissue. If the cancer spreads, chemotherapy is often used to kill cancer cells in other parts of the body.

Above all, it is very important that HIV-positive people engaging in vaginal or anal sex – even if condoms are being used regularly – discuss HPV and Pap smears with their healthcare providers. And if anal and/or cervical condylomas or dysplasia are suspected, follow up and treatment should be handled by an expert: an obstetrician/gynecologist or a proctologist/anal surgeon familiar with the treatment and care of HIV-positive people.

**Progressive Multifocal Leukoencephalopathy (PML)**
PML is a life-threatening infection of the brain that can occur in people living with HIV. It is caused by a virus – the JC virus. The “JC” are the initials of the first patient to be diagnosed with PML. More than 70% of all adults in the United States are infected with the JC virus, usually during early childhood. However, the virus only becomes active in people who have compromised immune systems, including those with HIV. It usually occurs in people with very low CD4 cell counts (less than 100), but has been seen in some HIV-positive people with as many as 500 CD4 cells.

PML is almost always progressive and fatal. Its symptoms include mental deterioration, vision loss, speech disturbances, ataxia (inability to coordinate movements), paralysis, and coma. Death usually occurs between one and four months after the first symptoms appear. However, there have been a number of reported cases with survival ranging from several months to years.

Unfortunately, there are still no proven treatments for PML. A number of medications have been studied, none of which...
have panned out as effective options. Hope is not lost, though – patients with PML whose CD4 cell counts improve with the use of antiretroviral therapy can sometimes halt the progression or reverse their symptoms of PML – the best PML news we’ve heard in a long time.

**FUNGAL INFECTIONS**

**Systemic Fungal Infections** Fungal infections such as aspergillosis, histoplasmosis, and coccidioidomycosis can occur in anybody, but are more likely to cause serious disease and affect a number of organs in HIV-positive people with suppressed immune systems. These three infections are generally treated using intravenous amphotericin B (Fungizone), which is often combined with oral antifungals. Intravenous amphotericin B can be a toxic drug. Fortunately, liposomal formulations of amphotericin B – which attach the drug to microscopic spheres of fat – are a possibility, as they have been shown to be just as effective and somewhat less toxic than standard amphotericin B for the treatment of several types of fungal infections. Liposomal formulations include Abelcet, Amphotec, and AmBisome.

**Cryptococcal Meningitis** Cryptococcal meningitis is a serious infection of the brain and spinal column that can occur in people living with HIV, particularly those with fewer than 50 CD4 cells. It is important to treat cryptococcal meningitis aggressively. For the first two weeks of treatment, the drug amphotericin B (Fungizone) is given every day through an IV line, along with a second drug taken by mouth: flucytosine (Ancobon). As for the possibility of using liposomal amphotericin B, recent studies conclude that it is equally toxic – and less effective – than Fungizone in patients with cryptococcal meningitis. Still, liposomal amphotericin B is sometimes prescribed for patients who become very ill while taking Fungizone or develop kidney problems, a potential side effect of Fungizone. If liposomal amphotericin B is used, experts recommend using the brand AmBisome rather than the brands Abelcet or Amphotec. After two weeks of taking amphotericin B and Ancobon, both drugs are stopped and another drug, fluconazole (Diflucan), is immediately started. This is necessary to help prevent the cryptococcal meningitis from recurring. Fluconazole is taken by mouth, every day, at a dose of 200 mg. At the present time, it is recommended that fluconazole be continued for the rest of a patient’s life. It is still not clear how high CD4 cells need to increase using anti-HIV drugs before fluconazole can be safely stopped.

**Candidiasis** In HIV-positive people, candida infection of the mouth (oral thrush) or the vagina (vaginal yeast infections) can occur at any time, regardless of their CD4 cell counts. However, oral thrush and vaginal yeast infections are more likely to occur, and recur more frequently, the more the immune system becomes damaged. HIV-positive people with damaged immune systems, usually with CD4 cell counts less than 200, are also more likely to develop candidiasis deeper in their bodies, such as in the esophagus or the lungs.

Numerous antifungals are available to treat oral thrush, including medications in the form of lozenges that are sucked or liquids that are swished around the mouth (Mycelex, Mycostatin, and Fungizone). The most common treatments for vaginal yeast infections are medicated creams or suppositories placed into the vagina (Gyne-Lotrimin, Mycelex, Mycostatin, Terazol, Vagistat, Femstat). If oral thrush and vaginal yeast infections do not go away with the use of these drugs, more potent medications such as ketoconazole (Nizoral), itraconazole (Sporanox), or fluconazole (Diflucan) can be taken. Compared to swish-and-swallow liquids and lozenges and vaginal creams, these drugs are more likely to cause side effects, including stomach upset, diarrhea, nausea, and elevated liver enzymes. There are also several novel medications being developed, including CS-758, which is similar to many of the currently available options, P-113, a peptide that may be less likely to result in drug resistance, and MycoGrab, an antibody-like product that is effective against candida strains resistant to current antifungals.

**PROTOZOAL INFECTIONS**

**Cryptosporidiosis and Microsporidiosis** “Crypto” and “micro” are infections that affect the lining of the small intestine and can cause severe diarrhea and prevent vital nutrients – and medications – from being absorbed properly (malabsorption). While anybody exposed to these two protozoa can get sick from them, diarrhea and malabsorption is usually limited to a few days in people with healthy immune systems. People with compromised immune systems – usually those with CD4 cell counts below 150 – may experience prolonged and severe bouts of diarrhea and malabsorption that can be difficult to treat.

Unfortunately, there are no universally effective treatments for crypto and micro. Many drugs have been studied in clinical trials. Some have been complete failures. Others have been shown to be effective for some people but not for others. The best treatment for these two infections appears to be antiretroviral therapy. By treating HIV effectively, it’s possible to increase CD4 cell counts to levels above 150. This has proven to work well for many HIV-positive people with crypto or micro.

**“Desensitization... has allowed many people allergic to Bactrim (TMP/SMX) to take this drug without problems.”**
**Pneumocystis Pneumonia (PCP)**

Until recently, PCP was officially called *Pneumocystis carinii pneumonia*. It is now believed that this form of pneumonia is caused by *Pneumocystis jiroveci*, not *P. carinii*. Complicating matters further are studies suggesting that *P. jiroveci* is a fungus, not a protozoan. Call it what you will, it’s still a common and life-threatening disease among HIV-positive patients who see their CD4 cells fall below 200.

Bactrim or Septra remain the most effective PCP prophylaxis drugs – these are both brand names for the combination drug trimethoprim-sulfamethoxazole (TMP/SMX) – although as many as one-third of all HIV-positive people are allergic to it. Fortunately, it’s now possible to undergo a “desensitization” protocol; under the close supervision of a doctor, patients start by taking tiny doses and gradually work their way up to the full dose within a few days or weeks. This has allowed many people allergic to TMP/SMX to take this drug without problems. And there is still the possibility of switching to aerosolized pentamidine (NebuPent), dapsone (Avlosulfon), or atovaquone (Mepron), three drugs that have been around for several years.

Similar to MAC, it was once believed that, if PCP prophylaxis is deemed necessary, it should be continued for life. This is no longer the case for patients whose CD4 cell counts increase to levels above 200 – and stay there for at least three months – with the use of antiretroviral treatment. And the same goes for patients who have been treated for PCP and have been instructed to continue taking maintenance therapy – usually Bactrim or Septra – for the rest of their lives. We now know that maintenance therapy can also be discontinued if CD4 cells are elevated above 200 for at least three months with the use of antiretroviral therapy.

**THE BOTTOM LINE**

I’ll always remember the words of a fellow advocate and educator: HIV doesn’t kill people, opportunistic infections do. These words were very much self-evident ten years ago and they are just as valid today. The good news is that the majority of HIV-positive people living in the United States today are being kept out of harm’s way thanks to the availability – and continual development – of potent anti-HIV drugs and effective anti-HIV drug combinations. The disturbing news is that OIs are still very much a reality for many HIV-positive people, and there is a growing number of people who are simply out of anti-HIV treatment options and are seeing their CD4 cell counts dwindle to levels at which OIs become possible.

A dismal picture? Perhaps. There is, however, an encouraging bottom line to consider: OI prophylaxis and treatments have advanced to the point that many OIs can either be prevented or successfully treated. Before anti-HIV drug “cocktails” came along, it was the advances in prophylaxis and OI treatment that could be credited for saving and prolonging lives. Understandably, the incredible advances in anti-HIV therapy have stolen much of the OI thunder – but it’s good to know that highly effective OI prevention and treatment options are still very much available to those who need them.

*Tim Horn is Executive Editor of The PRN Notebook, published by Physicians’ Research Network in New York. He is also the head medical writer for AIDSmeds.com.*

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**Resources**

For further info related to this issue of ACRIA Update, visit these websites:

**10th Conference on Retroviruses and Opportunistic Infections (CROI)**
www.retroconference.org/2003

Search the conference abstracts and posters for topics of particular interest to you. You can also watch Webcasts of some of the conference sessions.

**AIDS Malignancy Consortium (AMC)**
www.amc.uab.edu/

Find information about currently enrolling AMC trials, cancer-related links, and contact information for trial sites and investigators.

**AIDSinfo: The US Department of Health and Human Services (DHHS)**
www.aidsinfo.nih.gov/guidelines/

There’s lots of useful information on this site – a database of HIV-related drugs, a glossary of terms, as well as current and archived DHHS guidelines, including: Guidelines for the Use of Antiretroviral Agents in HIV-Infected Adults and Adolescents; Considerations for Antiretroviral Therapy in Women; Guidelines for the Use of Antiretroviral Agents in Pediatric HIV Infection; and 2001 USPHS/IDSA Guidelines for the Prevention of Opportunistic Infections in Persons Infected with HIV.

**International AIDS Society-USA**
www.iasusa.org/pub/index.html

Scroll down the page to the Treatment Guidelines section. The November 2002 guidelines discussed in Heidi Nass’ article are available as a PDF document. Just click on the link:

**Management of Metabolic Complications Associated With Antiretroviral Therapy for HIV-1 Infection: Recommendations of an International AIDS Society–USA Panel**

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Find information about currently enrolling AMC trials, cancer-related links, and contact information for trial sites and investigators.
Personal Perspective

Attending a Scientific Conference
When You’re Not a Scientist

by Dan Dunable

Like many treatment educators working in the HIV/AIDS community, I wasn’t trained in medicine, but learned about antiretroviral therapies, opportunistic infections, and other aspects of the disease by educating myself for my own health. I was diagnosed with HIV in 1988. In the mid-90s, I began volunteering with an AIDS service organization in Atlanta and ultimately became the manager of their treatment education program. As a writer for Survival News, AIDS Survival Project’s monthly treatment newsletter, I have had the opportunity to attend numerous conferences both nationally and internationally.

The most recent conference I had the fortune to cover was the 10th Conference on Retroviruses and Opportunistic Infections held in Boston this past February. This is one of the most important scientific and medical conferences of the year relating to HIV/AIDS and is primarily geared towards researchers, clinicians and other healthcare professionals. As such, it isn’t designed for community attendees, but for professionals with years of medical training. This is evidenced by many of the session titles: Mechanisms of HIV Neuropathogenesis, Designing Immunogens to Induce HIV Neutralizing Antibodies, DC-SIGN and Related Molecules, and Immunology: NK Cell, Cytokine, and Innate Immunology Responses, just to name a few.

When attending any conference, it’s always a challenge deciding which sessions to go to. There are usually multiple presentations occurring concurrently, and quite often I’m interested in many of them. Deciding which ones to attend and which ones to miss is often difficult. At the Retrovirus Conference, this task becomes even more difficult. Not only do I have to choose topics that are of most interest to my readers – and me – but also ones that I think I’ll actually be able to understand!

Some decisions were easy. I attended many sessions covering the metabolic complications of antiretroviral therapy such as lipodystrophy, cardiovascular disease, and insulin resistance; presentations on treatment strategies such as structured treatment interruptions; symposiums on new drugs in development; and sessions discussing new vaccine candidates. I have a basic knowledge of these topics; I could understand most of what was discussed and, hopefully, pass that information on to my readers.

I also try to expand my base of knowledge at each conference by attending a session on a topic that is completely new to me, one about which I have no clue. This year I chose a symposium entitled RNAi: A New Therapeutic Strategy. The ballroom was packed – standing room only. I felt I had chosen wisely. Obviously this is an important topic, one worth my time to learn more about. The session lasted two hours; I lasted 30 minutes. I was clueless as to what they were saying. As I left the ballroom, I noticed a few dozen other individuals leaving, all with the same glazed look in their eyes that I’m sure I had. And these were researchers and clinicians, but apparently ones who specialize in other fields. At least I wasn’t the only person who didn’t understand RNA interference pathways! But at the next conference where this is discussed, or the next article I see on this subject, I’ll learn a little more, and each time I’ll learn even more. That’s how we learn sometimes, a piece at a time.

It’s always somewhat overwhelming attending a scientific conference. In addition to deciding which sessions to attend to educate myself and which topics are important for me to attend to help educate my readers, there is also the dilemma of trying to remember everything that I heard or saw. So much of what is presented is important and I don’t want to miss any of it. (I wish that I had taken shorthand back in school now.) Fortunately, most conferences now make much of the information available online after the event ends. The official conference website makes abstracts available that were presented as well as webcasts of the symposiums. The ability to view abstracts online can be of great help, especially since it’s impossible to attend every oral session. But just getting information from the abstract can be misleading. Abstracts are very short summaries of the trial and don’t always give the true story, or even the latest information. An example that I experienced was a presentation on early microbicide research. The abstract for this particular study described very successful results in preventing SIV transmission in macaques with a vaginal microbicide being studied. However, after the abstract was submitted and accepted, the researchers, in continuing the trial, found the microbicide to be ineffective when used a second time. It worked the first time it was used but not after that. If I had only read the abstract, I would have thought the research was successful. Since I attended the oral presentation, I found out that the product did not work after all.

Lastly, just as important as gathering information is the ability to interact with other attendees. Talking directly with some of the researchers who presented the results of important clinical trials can be fascinating. And having the opportunity to sit down with community educators and advocates from other parts of the country, and even different countries, can be invaluable. Often our contemporaries are able to help decipher some of the information heard. Very often, the chance to gather in groups and discuss what we feel are some of the more important findings of the day is just as important and valuable as hearing the information presented firsthand.

Dan Dunable lives in Atlanta, GA. He is an HIV treatment educator and writer and manages the Treatment Education Department for AIDS Survival Project.
ACRIA Welcomes New Board Member

ACRIA is pleased to welcome Dolores Witherspoon-Cozier to our Board of Directors. Ms. Witherspoon-Cozier brings to our agency a unique perspective on and experience with HIV education needs for people infected and affected by this disease, and particularly youth at risk of HIV infection.

For the past 17 years, Ms. Witherspoon-Cozier has served as a Comprehensive Health Coordinator for HIV/AIDS issues at the New York City Board of Education. She has developed and led numerous staff trainings and curriculum development projects surrounding HIV infection and healthcare issues and is continually focused on updating HIV health education practices within the city to meet current needs. We expect that Ms. Witherspoon-Cozier’s active involvement in these citywide policy efforts will also substantially help ACRIA to keep our own treatment education programs relevant as the HIV epidemic changes in the years ahead.

New National Technical Assistance Program Recipient

ACRIA was able to bring our national HIV treatment education technical assistance (TA) program to Madison, Wisconsin for the first time in March. Twenty-six individuals participated in an intensive four-day training to develop and expand the skills and knowledge needed to counsel and educate people living with HIV about a wide range of HIV treatment issues. Madison is the sixth site to participate in the TA program since its inception in 1999.

Our trip to Wisconsin was particularly unique. The previous TA sites of Baltimore, Detroit, San Antonio and San Diego largely drew participants from urban settings. The Wisconsin training, however, was attended by non-medical care providers and community members from both rural and urban areas. This was ACRIA’s first experience training participants from agencies in nearly every region of a state. Our Wisconsin sessions offered an opportunity for all in attendance to learn about different needs for HIV treatment information by a wider variety of audiences than in our previous TA sites.

ACRIA was also able to return to Baltimore in March to provide a full day follow-up TA training for individuals who had participated in the initial multi-day training last October. Follow-up services are an integral feature of ACRIA’s TA initiative since they help participants to reinforce some of the more complex HIV treatment subject matter and also to build upon their skills as treatment educators within a peer group setting. The follow-up training sessions are just one example of how ACRIA’s TA program offers sustained support for establishing treatment education capacity within communities.

ACRIA Participates in the Community Planning Leadership Summit for HIV Prevention

Several ACRIA researchers recently staffed an agency-sponsored booth at the Community Planning Leadership Summit for HIV Prevention (CPLS) held in Manhattan in mid-March. We also presented an abstract on a new ACRIA research protocol at this forum.

The purpose of ACRIA’s participation in this largest ever gathering of social service personnel and health policy experts involved in HIV prevention was to introduce our increased research efforts aimed at strengthening prevention programs.

This conference gave ACRIA a special forum at which to recruit participants for our new collaborative “cyber study” with Indiana University that is assessing AIDS service organization (ASO) staff attitudes and ideas across the United States about improving prevention programs. CPLS provided an opportunity for ACRIA staff to network with over 300 ASO representatives working in every state of the country.

Centers for Disease Control and Prevention organizers of the CPLS conference stated that their goal was to at least halve new HIV infections in the United States from the current rate of 40,000 persons annually within five years. Hopefully, ACRIA’s new research will assist in achieving this goal.

is looking for new COMMUNITY ADVISORY BOARD members.

ACRIA’s Community Advisory Board (CAB) fosters partnership between the education staff and the local community impacted by HIV/AIDS. Involving community members in the development of our education programs ensures that community values and cultural differences are respected in ACRIA’s educational work.

Community Advisory Board members meet every other month, review program materials and help us identify education needs.

For more information about the CAB or if you are interested in volunteering at ACRIA, please call Mark Milano at (212) 924-3934, ext. 123.
The following persons, corporations and organizations made major donations between January 1, 2003 and March 31, 2003 to support ACRIA’s research and education efforts:

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Ortho Biotech
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