HIV and Hepatitis C: Learn the Facts, Take Action

It has been five years since ACRIA last took an in-depth look at the hepatitis C virus (HCV). But it remains a serious and, unfortunately, very common comorbidity of HIV. HIV both increases the likelihood of contracting HCV and complicates its treatment. Thus education about HCV transmission, risk, and treatment are critical for people with HIV, as is support for those who are coinfected with both viruses.

In the U.S. today, 30% of persons living with HIV also have HCV, and the numbers grow each year. HIV-positive men who have sex with men have a significantly higher risk of getting HCV than someone without HIV, and pregnant women with HIV and HCV have quadruple the risk of transmitting HCV to their newborns than mothers with only HCV.

Although HCV treatment options have improved in recent years (see the fine articles by Gabriel Ionescu on treatment today and Liz Highleyman on highlights from recent conferences), the disease remains difficult to manage, with nearly one in five people stopping treatment because of side effects. Existing treatments for HCV are not only complicated by HIV, but less effective in people with HIV. Daniel Raymond reports on the ongoing effort to find treatment regimens that work well in coinfected individuals.

The intersection of both HIV and HCV with injection drug use cannot be overstated. Blood-to-blood transmission is the most efficient way to get both illnesses, especially HCV — a virulent and tiny virus. Addressing these overlapping public health concerns has to be at the forefront of our efforts to reduce HCV and HIV transmission and to ensure adequate resources for effective treatment of both illnesses.

Unlike HIV, however, or even substance use, there is little organized advocacy around HCV and no large public funding streams specifically targeted at preventing or treating the disease. HCV advocates, among others, must increase their efforts to push public policy makers to focus on HCV. We hope this issue helps to do just that. Daniel Tietz, Editor-in-Chief

Hepatitis C: A Closer Look

Many people living with HIV must deal with not only one virus, but also with a co-traveler: hepatitis C virus (HCV), which infects liver cells. For some, the liver is only slightly damaged since it is able to make new cells and bounce back. But for others, HCV infection can lead to cirrhosis (severe liver damage), and the liver can lose some or all of its function. Finally, HCV may cause cancer of the liver and liver failure in some people.

A Global Epidemic

Worldwide, 130 million people have HCV, though the vast majority don’t know it. In fact, 27% of people with cirrhosis and 25% of those with liver cancer have these serious problems due to HCV. The good news is that since 1989 we’ve seen the number of new cases of hepatitis C in the U.S. simmer down to about 20,000 new cases each year, drastically lower than the 230,000 new cases we saw each year during the 1980s. HCV still accounts for up to 13,000 deaths annually in the U.S., however, and is responsible for the majority of liver transplants. Each year, complications and deaths associated with HCV infection are higher than in the previous year, and are now a growing concern in the HIV community.

In the U.S., about 30% of people with HIV also have HCV, and in some populations up to 90% of people who got HIV from sharing needles (continued on page 3)
Crofelemer for Diarrhea
People 18 and older who have persistent diarrhea will first take crofelemer or placebo tablets for 6 weeks. Then everyone will take crofelemer for 5 months.

Avandia and Serostim
People with insulin resistance will take Avandia (rosiglitazone), or Serostim (growth hormone), or both for 6 months to see how they affect glucose, insulin levels and body shape.

KP-1461
People aged 18-60 who have taken an NRTI, NNRTI and PI, and have developed resistance or stopped the drugs for other reasons, will take KP-1461 (a new type of NRTI) with no other ARVs for four months.

SPRING: Aptivus in Diverse Populations
People 18 and older (half white and half non-white, half men and half women) who have taken an NRTI, NNRTI and PI (not Aptivus) and who have resistance to at least two PIs, will take a standard dose of Aptivus or receive therapeutic drug monitoring to find the best dose for them.

IMPACT: Reyataz Resistance
People who have developed resistance to Reyataz will come in for one day of blood tests to study the I50L mutation.

Etravirine (TMC 125) Expanded Access
People 18 and older who have limited treatment options and resistance to approved NNRTIs, and who have taken an NRTI, NNRTI and at least two PIs, may qualify for early access to this experimental NNRTI.

For more information on these trials, contact Mervin Otero at 212-924-3934 ext. 121.
also have HCV. This is due largely to the fact that HCV most easily enters the body through the bloodstream. HIV can also enter the body in this way, which is why infection with both viruses is not uncommon. Yet there do seem to be some differences between the two.

Compare and Contrast
Both HIV and HCV carry their genes in the form of RNA (the mirror image of the DNA found in most viruses). However, the similarities seem to end there. HCV is a smaller virus, but can churn out many more copies a day (up to a trillion) than can HIV. It is also much more resilient than HIV. For example, HCV can still be infectious even in a dried droplet of blood, whereas HIV is often noninfectious seconds after it reaches the air. And while both can be transmitted through blood (sharing needles, for example), HCV is about ten times more infectious than HIV. Simply put, if 100 people are actively sharing needles for one year, 64 will test positive for HCV, but only 14 will test positive for HIV. For a smaller virus, HCV certainly gets a run for its money.

Blood to Blood
By far the most efficient way for HCV to enter the body is through direct blood-to-blood contact. This is usually due to sharing injection equipment, but in some areas of the world can still happen through transfusions or organ transplants. Before 1992, people with hemophilia and individuals receiving transfusions were at great risk for hepatitis C infection – 84-100% of hemophiliacs and 10% of people receiving blood transfusions received blood products infected with HCV. Today, in the U.S. and many other countries, the risk of contracting HCV through transfusions and blood products has been virtually eliminated. The risk of HCV transmission has become less than one in a million per unit of blood transfused, thanks to the screening of blood donations for HCV that began in the summer of 1992.

Organ transplants have also been cited as carrying a high risk of HCV transmission. Studies have found that three out of every four people who received an organ (kidney, heart, or liver) from a donor who had HCV also developed HCV antibodies, and about a third developed liver disease. As a result, transplant centers have developed protocols to include screening for hepatitis C that prevent infected organs from being used.

While concern about accidental needlesticks is justified, the actual risk of getting hepatitis C this way is extremely low.

“A study of 895 monogamous, heterosexual couples who did not use condoms or have anal sex or sex during menstruation found that only 1 in 1000 couples, or 0.1%, reported having gotten the virus through sexual transmission.”

Studies have looked at the number of reported cases have found that only 1% of accidental needlesticks lead to HCV infection. Solid needles (used for tattoos and acupuncture) seem to carry a lower risk of transmission than hollow needles (used for blood samples and infusions). While both types have a low risk of infection, care should be taken to avoid needlesticks and to ensure that sterile needles and ink are used for tattooing and piercing.

Heterosexual Transmission
There’s a lot of discussion surrounding the degree to which HCV can be transmitted through sex. We know that hepatitis C can be spread sexually, but how often does it happen? In short, not that often when it comes to vaginal intercourse. One study looked at 500 heterosexual couples in which one partner was HCV positive, and who had been monogamous and sexually active without condoms. The results were quite optimistic: only five cases of transmission appeared to have occurred after 16 years. A second study of 895 monogamous, heterosexual couples who did not use condoms or have anal sex or sex during menstruation found three of the negative partners became HCV positive, but none had the same type of virus as their partner, meaning they most likely got it some other way. Overall, only 1 in 1000 couples, or 0.1%, reported having gotten the virus through sexual transmission. Therefore, while vaginal sexual transmission is possible, the risk of infection appears to be quite low, particularly among monogamous couples.

What happens when someone is living with HIV as well? How does this change the risk of transmission? In certain people with HIV, such as heterosexual couples and expectant mothers, we see higher levels of HCV transmission. In one study, about twice as many heterosexual partners became positive for hepatitis C when their partners had both HCV and HIV. This study also found that among those who became HCV-positive, having multiple sex partners, a history of sexually transmitted diseases, or not using condoms increased their risk for hepatitis C. Therefore, while sexual transmission carries an extremely low risk overall, it is higher among heterosexual couples where one person is living with both viruses.

Pregnancy
Heterosexual couples aren’t the only group whose HCV risk seems to increase with HIV infection. Pregnant women are also affected. In women with HCV only, 5% will transmit HCV to their newborns, but for women with both viruses, that number quadruples to 19%. In fact, a review of all studies on mother-to-child HCV transmission found that having HIV was the greatest factor associated with hepatitis C transmission. Other factors include a higher level of HCV at the time of delivery, especially when the hepatitis C viral load is
Hepatitis C: A Closer Look (continued from previous page)

greater than 100,000. Studies have also been done to examine whether Cesarean delivery would lower transmission, but so far this has not been shown to have an added benefit. Breastfeeding also does not appear to be a significant means of HCV transmission, but is not recommended for women living with both viruses because of the risk of HIV transmission.

Male-to-Male Transmission
Up to 4% of men who have sex with men (MSM) have hepatitis C. A few factors have been associated with higher rates of hepatitis C transmission, including unprotected anal sex, fisting, group sex, use of recreational drugs that lower inhibitions, and having a sexually transmitted disease (such as syphilis). It’s not certain that HIV increases the risk of HCV transmission in MSM, but recent reports from Europe of HIV-positive men becoming infected with HCV suggest that having HIV may be a factor. A small study in Australia looked at 26 HIV-positive and 94 HIV-negative MSM and found only one case of HCV infection among the HIV-negative men (1%), but 13 among the HIV-positive men (50%).

Several factors may be associated with having both viruses, including unprotected rough sex and having a greater number of sexual partners, along with any of the risk factors listed above. More specifically, one study that looked at 1,836 MSMs in Amsterdam found that as of the year 2000, 56% of men with both HIV and HCV who did not use injection drugs reported having unprotected rough sex. Genital ulcers may additionally increase one’s risk; one study found that 90% of men who became recently infected with HCV had ulcers.

While these risk factors are cause for concern, other studies have found that the overall number of MSM with hepatitis C is not higher than that of heterosexual men. A study of 3,455 heterosexual men and 1,699 MSM in U.S. public health clinics from 1999 to 2003 (who didn’t report injection drug use) found that the straight men were actually twice as likely to have HCV (3.6% compared to 1.5%).

But this may be changing. A study in England looked at 7,169 MSM who reported no injection drug use. HCV incidence increased from zero in 2002 to 3.6 per 1,000 patient-years in 2006. HIV-positive men were found to be about 13 times more likely to have a new HCV diagnosis compared with HIV-negative men.

“A study in England looked at 7,169 men who have sex with men who reported no injection drug use. HCV incidence increased from zero in 2002 to 3.6 per 1,000 patient-years in 2006."

Living with HCV
Once HCV has entered the bloodstream, the virus seeks out liver cells (hepatocytes), where it can set up shop. Unlike HIV, HCV does not need to enter a cell’s nucleus, where the cell’s DNA is stored. Instead, it stays in the main body of the cell – the cytoplasm. Over time, hepatocytes can become overwhelmed by the toll of the infection and can become damaged, or fibrotic. Liver tissue can scar, and over time can lead to cirrhosis, in which liver cells lose function and can die.

During this time, the liver will try to replace the damaged and dead cells, and in the process spill out enzymes called ALTs and ASTs, both of which can be measured in blood tests called liver function tests. Higher levels of ALTs and ASTs are sometimes the only sign of infection with hepatitis C, as many people can live 10 to 20 years without any other signs or symptoms. Other signs can include yellowing of the whites of the eyes or skin (jaundice), fluid in the belly (ascites), and dark urine or pale stools.

Of 100 people infected with HCV only, roughly 20 will completely clear the virus on their own. But that means that 80 of the 100 will have it for many years. Of those, about 30 will stay stable and healthy; 50 will develop fibrosis, 16 of whom will also develop cirrhosis; four will develop liver cancer; and one will die from liver disease. While only a small number of people with HCV will progress to cirrhosis, it is important for each person to be aware of the risk factors for disease, as most people living with hepatitis C will not have any symptoms but may still be progressing in their disease. Also, other cofactors, such as HIV or alcohol use, increase the risk that someone will progress to more serious liver disease.

Watching the Viruses
How should liver health be monitored in someone who has hepatitis C? Well, since HCV nestles into liver cells, monitoring ALT and AST levels in the blood can give a clue as to whether there is any liver damage at the moment. But while infection with HCV can raise these enzymes over time, other things can, too. For example, certain medications (including Tylenol and many HIV meds) are broken down in the liver and can affect overall liver health. Monitoring liver enzymes every 3 to 6 months can help keep tabs on how the liver is handling the virus and any other pressures it has on it at the time.

But monitoring liver enzymes is not enough, since these tests may be in the normal range even though hep C is doing damage. A few other tests are needed, especially if someone is also considering
starting treatment. Viral load tests are available that can tell us how much HCV virus is in a milliliter of blood. While viral loads are routinely measured in HIV disease, for hepatitis C these tests are most helpful before starting HCV treatment and for seeing how well it is working. Someone who has less than 2 million copies (or 800,000 international units) of HCV is considered to have low levels of virus. More than 2 million copies is considered a high viral load. Again, this is useful for making decisions about treatment, but repeated HCV viral loads are not routinely done, since levels of HCV often remain stable, keeping viral loads quite consistent from year to year.

If someone is considering treatment, two additional tests are useful: a genotype test and a liver biopsy. An HCV genotype test is different from an HIV genotype test (which looks for drug resistance). An HCV genotype test identifies which strain of HCV a person has. There are six types (or strains) of HCV, numbered 1 to 6. About 75% of people with HCV in the U.S. have type 1. In Europe, on the other hand, type 2 is more common. Knowing the genotype is useful, since studies have found that people with type 2 or 3 tend to do better on treatment than type 1. So knowing your genotype can help you and your doctor get a feel for how useful treatment might be. (See “Hepatitis C Treatment Today” on page 6.)

Liver biopsies are by and large the gold standard for making decisions about when to start treatment, as they give us the clearest feel for how much liver damage there is. An ultrasound of the liver is often done before a biopsy to find the best place in the liver to collect a sample. Then, using a needle that is inserted just below the right ribs, a small piece of the liver is snipped out and looked at under the microscope. This can tell whether a liver has mild, moderate, or severe damage, which is very useful as a patient and doctor decide whether treatment is needed. While any biopsy is invasive, this procedure is usually done on an outpatient basis, is not painful in most cases, and has complications only rarely. The main risk is internal bleeding, so physicians will often ask you to stay at the office for several hours to monitor for any bleeding.

In a nutshell, it is useful to monitor liver enzymes (ALT, AST) routinely every 3 to 6 months. When considering treatment, it may also be useful to have viral loads, genotype testing, and a liver biopsy, as all three provide valuable information about liver health. None of these tests can predict what’s going to happen with the liver, but they give valuable information about how the liver is responding to the virus.

Conclusions
Hepatitis C remains an important health issue for many populations, including people with HIV. Given the potential for long term liver disease, a thorough understanding of how hepatitis C is transmitted, and how it is affected by HIV, can be helpful in the long term management and care of people living with both viruses.

Donna M. Kaminski, ACRIA’s former Associate Director of Treatment Education, is currently a third-year medical student.
The decision to start treatment for hepatitis C must be shared by doctor and patient, and everyone with a chronic hep C infection should be evaluated for treatment. But since the treatment can cause difficult side effects, and is less effective in people who also have HIV, education and support are critical if it is to be worthwhile. This article will highlight the current state of treatment and the issues people coinfected with HIV should know before they begin.

The main goal of hepatitis C treatment is eradication of the virus, which currently is seen as a cure (we’ll know for certain when people have been followed for more than five years). This is called a sustained virologic response (SVR – no detectable virus in the blood six months after completing treatment). Attaining an SVR decreases liver inflammation and fibrosis (scarring), lowering the chance of cirrhosis and liver cancer. As treatment for hepatitis C virus (HCV) has improved, guidelines have been changed, so that everyone with HCV should be evaluated for treatment.

Since HIV and HCV can both be transmitted by blood, testing for HCV antibodies is recommended for everyone with HIV. People at high risk for HCV, particularly those who have shared needles, may need an HCV viral load test even if they test negative for HCV antibodies. Up to 9% of people with a history of high-risk behaviors and elevated liver enzymes have an “occult” (hidden) HCV infection. Everyone with HCV should also be tested for hepatitis A and B, and if negative should be vaccinated against both.

Current HCV treatment combines weekly injections of pegylated interferon (Pegasys or PegIntron) and twice-daily ribavirin capsules. Both drugs decrease the formation of new viruses, preventing further spread of HCV to healthy liver cells. Interferon also helps to eliminate already infected cells. Although these two interferons are different, there do not appear to be major differences in their ability to treat HCV. However, no head-to-head studies have been done. (In the U.S., only Pegasys is currently approved by the FDA for the treatment of HCV in people with HIV, but PegIntron is also used in these patients.)

A liver biopsy before treatment is useful, to check the degree of inflammation and fibrosis. A biopsy can help to predict the course of HCV disease, since most everyone with early fibrosis of the liver’s portal vein is likely to develop cirrhosis after two decades if untreated. People with less severe disease may never develop cirrhosis, so careful monitoring is an alternative to treatment. In addition, a liver biopsy may be repeated in five years to see if the disease has progressed. However, the treatment of even mild HCV before someone is 60 years old may improve survival and reduce the risk of cirrhosis, since people with less fibrosis respond better to current treatment.

Not everyone with HCV is a candidate for treatment. People who have decompensated cirrhosis (meaning the liver cannot keep up with the amount of fibrosis that has occurred) should not begin HCV treatment. Treatment is generally not recommended for people with autoimmune hepatitis, severe anemia, unstable diabetes or cardiovascular disease, or psychiatric problems that have not responded to treatment. Likewise, those who are malnourished, jaundiced, or who have swelling of the abdomen (ascites) or brain (encephalopathy) should not take current HCV treatment. In these cases, a liver transplant is often the only option, and these are now being done in coinfected people. HCV cirrhosis is the most common reason for liver transplants in the U.S. and Europe.

**Acute HCV treatment**

About 75% of people newly infected with HCV have few or no symptoms. Treatment is most effective if given early, but it’s best to wait one to three months after infection, since about 20% of people will clear the virus without treatment. Peg-interferon with or without ribavirin should be considered in those who do not clear HCV after three months, as more than 80% may respond to this treatment. People with no symptoms should be treated immediately, as they appear to have a higher risk of chronic disease than those who do have symptoms soon after infection. Six months of treatment that was started twelve weeks after acquiring HCV led to an SVR in a majority of people with HIV, regardless of their genotype.

**Treatment for HCV Monoinfection**

HCV treatment for people without HIV is usually taken for 24 weeks for those with genotype 2 or 3, or for 48 weeks for genotype 1. Studies have shown SVR rates of 80% for genotypes 2 and 3, and 42% for genotype 1. People with genotype 1 who do not show an early viral response (EVR – an undetectable HCV viral load after twelve weeks of treatment) have little chance of achieving an SVR, and treatment may be stopped. Since people with genotype 2 or 3 usually respond to treatment, checking for an EVR may not be needed.
In Europe, treatment with a shorter course of Pegasys (24 weeks) has been approved for certain monoinfected people who had a rapid viral response (RVR – an undetectable HCV viral load after one month of treatment). One study found that 16 weeks of treatment for people with genotype 2 or 3 who had an RVR was as effective as 24 weeks. A higher ribavirin dose may be the key to achieving an RVR. But shorter courses of therapy are not universally accepted and more studies are needed.

Treatment for HIV/HCV coinfection
The decision to start treatment for hepatitis C is based on a combination of blood tests and liver biopsy.

Treatment is strongly recommended for people with elevated liver enzymes (ALT and AST), CD4 counts above 350, HIV viral load below 1,000, and no alcohol intake. The degree of fibrosis should also be taken into account.

HIV treatment should be optimized before starting HCV treatment. Videx should not be taken with ribavirin because of an increased risk of lactic acidosis and pancreatitis, raising the risk of cirrhosis. Retrovir (AZT) increases the risk of anemia when taken with ribavirin, and Zerit is associated with weight loss and lipoatrophy (unwanted fat loss). Viramune and Aptivus (the latter in combination with Fuzeon) have been associated with serious liver damage in people with HIV, and Zerit and Videx have been associated with other liver problems in people who are coinfected. People with HCV genotype 3 are at greater risk for liver toxicity from HIV meds. The amount of fibrosis raised levels of Aptivus, increasing the risk for increased liver enzymes, while Reyataz levels did not increase. Also, better tolerance to HCV treatment was noted with newer NRTIs like Ziagen.

SVR rates range from up to 29% for people with genotype 1 to 73% for those with 2 or 3. Better outcomes are seen in people below age 40, in leaner patients with less fibrosis, and in those with low or undetectable HIV viral loads. A higher CD4% and HCV viral load below 400,000 IU improved the chance of SVR in people with genotype 1. However, most coinfected people have high HCV viral loads.

People with genotypes 2 and 3 have a higher chance of a viral rebound, so 48 weeks of treatment are needed. But an SVR appears to be equally durable in coinfected people. Long-term follow-up studies of coinfected people with an SVR have not seen more viral load rebounds, advanced cirrhosis, or liver cancer than that found in monoinfected patients.

Up to 17% of patients stop HCV treatment due to side effects and anemia. Drugs to raise red blood cell counts may be needed in up to 60% of people taking Retrovir (AZT) who develop anemia. Starting EPO (Procrit or Epogen) before HCV treatment may increase the chance of SVR and decrease the risk of anemia. Fortunately, HCV treatment usually does not affect HIV disease, despite the temporary decrease in CD4 counts it can cause.

The study that led to the approval of HCV therapy in people coinfected with HIV used a low dose of ribavirin (800mg daily) in an attempt to prevent anemia. Higher doses of ribavirin (1,000 - 1,200 mg) are now accepted, leading to better SVR rates. More recent findings suggest improved SVR rates with twice weekly peg-interferon or even higher doses of ribavirin.

Will It Work?
The most important predictor of an SVR is the HCV genotype. Treatment is less effective for people with genotype 1, which is the type most common in the U.S. Response to treatment also depends on HCV viral load, dosing, and length of treatment. The sooner HCV viral load becomes undetectable while on treatment, the greater the chance of an SVR. New research suggests that the length of HCV treatment should be changed based upon how quickly an individual achieves an undetectable HCV viral load. People with HCV only, who have genotype 1 and who do not clear HCV within three months of treatment may need to extend the length of treatment to 72 weeks. Monoinfected people with genotypes 2 or 3 who do not have an undetectable HCV viral load within a month of starting treatment had higher SVR rates if they continued treatment for 48 weeks instead of 24. But recent studies of people who are coinfected do not support extended treatment at this time.

Adherence to treatment is important for improving the chance of an SVR. Side effects leading to dose reductions or stopping treatment lower the chances. But lowering the ribavirin dose for the first four to five months may not be a problem if the dose of peg-interferon is maximized.

Even without an SVR, treatment may delay or reverse fibrosis, which reduces the risk of liver cancer and increases the liver’s ability to process HIV meds.

It’s important that people taking HCV treatment get the support they need. One approach has been directly observed therapy, which has proven effective in people enrolled in methadone maintenance programs. People with hepatitis C should avoid alcohol, as it may lead to a higher HCV viral load, increase the

(continued on page 9)
My name is Donald and I'm an addict.

I can say that today only because I'm in recovery after years of use and abuse. I managed to experience and experiment with a lot of different drugs, starting with alcohol and marijuana, on to LSD, PCP, uppers and downers, even some speed. But heroin was my drug of choice. I started injecting when I was 22 years old.

I tried many times to clean up my act. Church, retreats, detox programs (14, 21, or 28 days). Phoenix House three different times. But I always came back to the big H: Horse, P-funk, Super-D. Then came numerous arrests and jail. The want and need to have drugs brought all this about. Did I care at the time? “Hell, no!” on the outside. But I was devastated on the inside.

Around 1996, I was told that I tested positive for hepatitis C, and being uneducated about that particular virus, I paid little attention. But in 2000, I decided to get serious about myself, my addiction, and my HIV. I did two things: I became a peer educator and used the harm reduction model in my life. I enrolled in methadone maintenance and I got deeper into peer education, got more training, and eventually got a job as an educator.

Something marvelous happened to me during that time. I got good at it. So good that I was chosen to be part of a new peer education program at Woodhull Hospital, the same hospital that my doctor practiced at. Everything was coming together for me. Dealing with others helped me with my struggles.

“I was totally ignorant about the virus or that it could be treated … All I ever heard was the ‘word on the street’ type of information … I was used to being misinformed by the uninformed.”

Was this my “out” for not attempting the treatment? Well, that's where I was in my recovery process. But in 2006, my doctor and I revisited the subject. A plan was available where you came once a week, and your injections were given by an NP who monitored your weight and blood pressure and gave you semimonthly blood tests. And he told me that some people had beat the virus due to the meds. I can tell you for sure that the fear of not surviving greatly outweighed the fear of the unknown – that unknown being what the treatment entailed.
If I could stay undetectable for HIV for over four years, I knew I was about being adherent to my meds. My meds had become my life. I knew I could do it. Now I was ready! I wanted it more than anything now.

I had heard the word on the street concerning the side effects and I knew what that was like after the HIV meds. But the fact that I might be able to beat hep C just overruled whatever side effects the meds had in store for me. After each injection, I would feel dead tired by the end of the day, but that would get better after a couple of days. My normal weight was around 140 to 150 pounds, but on the interferon I got down to 131. I was very comfortable carrying less weight and looked at it as a loss of excess. But the mood changes were totally unexpected. I found myself being overly sensitive about small details of everyday living. I would cry if I didn’t get my needs met or if people said things that hurt my feelings. I didn’t know what was wrong until someone pointed out that it was probably the medication.

I knew I had to stay strong and stay with the meds no matter what. I had trouble sleeping, but I never took meds for that – I just drank tea. It was a struggle dealing with the medication. But my main focus was to beat that mean old hep C with the help of my spirituality and the support of my good friends.

After the first month of treatment, my hep C viral load went to undetectable, from a few million copies at the start. There is a God! I stayed on treatment for about 42 weeks and every blood test and doctor visit was encouraging. This is what kept me going – the knowledge that I could and would beat this virus; the same virus that I had let into my life by not caring about myself and the others in my life.

Today is different for me: happier, healthier and much more positive. Five months after stopping treatment, my hep C viral load is still undetectable and I intend to keep working for myself and others to keep it at bay. Thanks for letting me share.

Gabriel Ionescu is an attending physician in the Division of Gastroenterology at St. Luke’s-Roosevelt Hospital Center in NYC.
What’s in the Hep C Pipeline?  
by Daniel Raymond

The pressing need for new hepatitis C treatments has spawned a surge of activity in the pharmaceutical industry. Virtually all major drug companies, and a host of small biotech start-ups, have research and development programs aimed at developing new hepatitis C medications. There is potentially a multibillion dollar market for hepatitis C virus (HCV) treatment, providing a strong financial incentive for drug discovery. Yet attempts to develop new medications have been fraught with pitfalls and uncertainty, and many once-promising candidates have failed in clinical trials due to safety concerns or lack of potency.

Based on current progress, it appears that major advances in HCV treatment are unlikely to occur until 2010 at the earliest. Yet many people with HCV are unlikely to occur until 2010 at the earliest. Yet many people with HCV choose to delay treatment, often on the advice of their doctors, in the hopes that future drug regimens will be better than the current standard of care, pegylated interferon (Pegasys and PegIntron) and ribavirin.

The limitations of current treatment are well known, including a host of potential side effects and limited success in eradicating HCV (particularly for people coinfected with HIV and for African-Americans). But people considering treatment should know that most new hepatitis C drugs will be added on to the current treatment, peg-interferon and ribavirin. While it is hoped that these new classes of drugs – specifically protease and polymerase inhibitors – will increase the success rate of treatment, they will not immediately end the need for peg-interferon and ribavirin, with their well-known side effects.

At best, new drugs may shorten the length of treatment – say, from the typical 48 weeks to 24 weeks – but they will not decrease its side effects. Some companies are developing alternate versions of interferon and ribavirin which may prove more easily tolerated, but these improvements are likely to be modest at best. The greatest hope for a transformation in HCV therapy lies in the goal of developing new drugs that can be used together without peg-interferon or ribavirin (similar to the triple-drug combinations used to treat HIV), and which require only a few months of treatment. This is possible, but lies many years ahead.

In the meantime, people with HCV should weigh the risks and benefits of current treatment against the uncertain promise of future treatment options – and the potential among some (particularly those with HIV) for worsening liver disease if HCV treatment is delayed.

The role and value of new HCV drugs for people coinfected with HIV is also unknown. In theory, some of these new drugs may have negative interactions with HIV drugs, perhaps limiting their use. A 2006 hearing of the Antiviral Drugs Advisory Committee of the Food and Drug Administration (FDA) recommended that new HCV treatments be studied in people with HIV prior to FDA approval. This recommendation was echoed in the Sitges Statement, issued earlier this year at a meeting in Spain which convened advocates, community members, researchers, clinicians, and pharmaceutical companies to discuss HIV and HCV. No clear standard exists, however, for how much research would be needed to prove safety and efficacy in coinfected people prior to a new HCV drug reaching the market. Currently, no clinical trials of new HCV treatments include people with HIV.

Despite these hurdles, the field of HCV drug development has made considerable strides, with several promising candidates currently under investigation. Most likely, some of the drugs described in this article will gain FDA approval in the next few years. Improvements in treatment over the near future will likely remain incremental and uneven, with early gains in treatment success tempered by persistent problems with side effects. But, ultimately, a new generation of HCV drugs holds out the promise of highly effective, well-tolerated, short-term treatment – challenging us to begin planning for the kinds of healthcare systems and community support needed to realize these gains for everyone.

Hepatitis C Protease Inhibitors

Like HIV, HCV contains an enzyme called protease that is essential for viral replication. The success of protease inhibitors in HIV treatment generated significant interest in drugs that could target HCV’s protease. Early studies in the test tube and in people with HCV indicated that protease inhibitors could produce a rapid and substantial reduction in HCV viral load. As with HIV meds, however, HCV protease inhibitors can also lead to resistant virus. So, treatment will require using an HCV protease inhibitor in combination with other drugs (such as the current standard of interferon and ribavirin) in order to prevent resistance.

“People considering treatment should know that most new hepatitis C drugs will be added on to the current treatment, peg-interferon and ribavirin.”
Telaprevir
Two HCV protease inhibitors have advanced to phase II clinical trials. Telaprevir (formerly VX-950), developed by Vertex Pharmaceuticals, is currently in studies exploring different treatment lengths in people who have never taken HCV treatment (another study is examining the drug in people who did not respond to previous treatment).

Treatment lengths being studied include:

• 12 weeks in combination with peg-interferon and ribavirin.
• 24 weeks (12 weeks of triple therapy, followed by 12 weeks of peg-interferon and ribavirin alone).
• 48 weeks (12 weeks of triple therapy, followed by 36 weeks of peg-interferon and ribavirin alone).

Early results indicate that 12 weeks of triple therapy can clear HCV in some people, but the success rate is disappointing. Most people will likely require at least 24 weeks, and the first results for people taking the drug for that length of time will be presented at a scientific meeting in November. While telaprevir appears to be relatively well tolerated, concerns have emerged over a rash that can be severe enough to require some people to stop the drug.

Boceprevir
Schering-Plough also has an HCV protease inhibitor, boceprevir (formerly SCH 50304), in phase II trials, though no results have been made public. Like telaprevir, boceprevir needs to be taken three times a day, raising concerns about drug adherence. Dosing could be simplified by adding a small dose of the HIV med 'Norvir, which boosts the levels of these drugs in the body, but neither company is currently focusing on this line of research.

Both these drugs will likely enter into large phase III studies in 2008 – the final stage of research before submitting a new drug to the FDA for approval.

Other protease inhibitors
A third protease inhibitor under development by Intermune, ITMN 191, may allow for twice daily dosing, but this compound is further behind in development and is currently in small phase I studies. First results are expected early next year. Medivir, in collaboration with Tibotec, recently presented the first results of its protease inhibitor, TMC 435350, in people without HCV and has begun Phase I trials in people with HCV.

Polymerase Inhibitors
Polymerase inhibitors targeting HCV replication also hold out considerable promise for improving treatment. Polymerase inhibitors are similar in function to two classes of HIV treatment: nucleoside analogues (NRTIs) and non-nucleoside reverse transcriptase inhibitors (NNRTIs). Unfortunately, research on the HCV polymerase inhibitor that was furthest along in development, valopicitabine, was halted this summer due to FDA concerns that its side effects (especially nausea) outweighed its potential benefits.

However, several other investigational drugs are moving through early stages of testing, including R1626 from Roche, GS-9190 from Gilead, and R7128 from Pharmasset (in collaboration with Roche). Preliminary data suggests that these drugs may be more potent than valopicitabine, though all are several years away from reaching the clinic. As with HCV protease inhibitors, polymerase inhibitors can result in the development of drug-resistant virus, and will need to be taken with other HCV meds.

New Interferons
Some companies are developing interferons that improve on the current standard – peg-interferon, which is taken once a week. Albuferon, a new interferon developed by Human Genome Sciences, is currently in two large phase III studies that compare it to peg-interferon (both in combination with ribavirin). Albuferon may allow for dosing once every two weeks, or perhaps once every four weeks. Early research suggests that taking it once every two weeks is roughly comparable to weekly dosing with peg-interferon. The fate of albuferon depends on whether it can match or even exceed peg-interferon in treatment success, with comparable or milder side effects. Biolex Therapeutics also has a long-acting interferon called Locteron, which may allow for dosing every two weeks, in phase II studies.

Albuferon, a new interferon, is currently in two large phase III studies. It may allow for dosing once every two weeks, or perhaps once every four weeks.

Other Drugs
Several other drugs are also in development – including new classes of drugs targeting different aspects of hepatitis C – though they are generally in very early stages of research. The multiple approaches to drug development, along with the significant investment by the pharmaceutical industry, bodes well for the future of HCV treatment. However, the history of HCV drug development is littered with failures, and the future is impossible to predict with any certainty. As we keep an eye to the future, people with hepatitis C still face challenging treatment decisions in the present.

Daniel Raymond is the Policy Director at the Harm Reduction Coalition in New York City.
Allow me to introduce myself. My name is Donna Y. Kennedy. I am a 51-year-old African-American. I am a woman, wife, mother, grandmother, daughter, sister, aunt, godmother, cousin, friend. Oh, by the way, I am coinfected with HIV and hepatitis C. I was diagnosed with HIV in 1984 and with hep C in 1997. I would like to share with you my experience with hep C treatment.

I was told many things about hep C. I was told that it was a silent killer and that I would likely die from it before I would die from AIDS. I was also told that I would need to get a liver biopsy because that was the best way to tell what was actually going on with my liver. I had elevated liver enzymes a few years earlier and that is what led me to be tested for hep C.

I began my first treatment in 1998 while I was incarcerated at Beacon Correctional Facility. At that time interferon was given three days a week along with daily doses of ribavirin. I was on the treatment for over two years. I started the treatment because of the information I learned and because of my elevated liver enzymes and the results from my liver biopsy.

Initially I had my injections done by a nurse. By the time I was released from prison I was able to inject myself. I was an intravenous drug user in the past and I heard from some people that the injections would “kick up their stuff.” I must say that was not my experience. I did experience flu-like symptoms. I would get the chills and then a fever, I would have aches in all of my joints. I became anorexic and was very thin. To me, anorexic means that you do not have the desire to eat. As much as you may know that you need to eat, it is a real struggle. Just the smell or the thought of food would turn me off. My CD4 count dropped. I became anemic and also had a drop in my white blood cells.

I was given medication to help with my anemia. I learned to take Tylenol right after my injections. All in all, I felt lousy. I had to lay down within a few hours after my injections. It affected me in a way that everything that I did was a push for me. I did not lose my hair but it did thin out.

I had my sisters in the church, friends that I had made, and a very special counselor for support. My family supported me also through letters and visits. When I completed my treatment two months after my release, I had not cleared the virus but I did have some improvement.

About a year after I completed my treatment, I decided to start again. This time the treatment was pegylated, which meant that instead of taking the interferon three times a week I was able to take one injection a week along with daily doses of ribavirin. This worked out so much better. It meant less sore spots at the injection site and that the drug stayed in your body at a steady state for seven days.

When I first started taking the peg-interferon, I would feel lousy most of the time because just when my body would feel all right it was time for another injection. I would take the injections on Friday night so that I would be able to attend church on Sunday and work during the week. I was extremely fatigued, white blood cells were low (I was treated with Neupogen once a week), and I became anorexic again, so I took Megace, an appetite inducer.
After a few weeks of treatment, the side effects became more bearable. Towards the end of the treatment I began to have anxiety, which was helped by talk therapy with a psychiatrist. But then my glucose got out of whack. I would be hyperglycemic and hypoglycemic (high and low blood sugar). This let me know that I was predisposed to diabetes, so I had to do daily sticks to check my glucose levels, and to change my way of eating. Once the treatment was over my glucose levels were back to normal.

After four weeks of this treatment I was nondetectable, and my doctor and I were elated. But just as quickly as it went nondetectable it shot up sky-high. I completed the treatment, which lasted for one year. And even though I did not have a nondetectable hep C viral load, I did benefit. I had less fibrosis and I gave my liver the chance to get stronger so that I could continue taking my HIV meds with minimal side effects.

I would recommend the treatment to all those who are able to take it. To me it was well worth all the negative side effects that I had to suffer through. I do not use the word suffer lightly because you do suffer. Everyone cannot tolerate the treatment and either they quit or they are advised by their physician to stop treatment. I am glad that I took the treatment, but I will be much more happy when they come out with pills to treat hep C. I would consider taking maintenance treatment if it comes out in pill form.

I am a person who likes to be informed, so I do my homework. I went to many seminars, conferences and workshops that taught about hep C. I read about the disease and I asked many questions. I recommend that anyone who has a disease do their research. Knowledge is power.

I found a very good physician whom I trusted and was very comfortable with. I learned that in order for treatment to work for you, you must be comfortable with the treatment. Many times throughout the treatment I wanted to quit but I am not a quitter. I hope that I was able to encourage others to do their homework and make the right decision about hep C treatment.

News from the Front

This year’s conferences covering HIV/AIDS and liver disease featured numerous presentations concerning hepatitis B virus (HBV) and hepatitis C virus (HCV) infection in people with HIV. Below are highlights from several of the most interesting reports.

**Hepatitis C Treatment**

Numerous studies looked at hepatitis C treatment in people coinfected with HIV and HCV. HCV treatment has improved in recent years, but as many as half of all treated patients do not achieve a sustained virological response (SVR) – an undetectable HCV viral load six months after finishing treatment — which is usually considered a cure. Plus, response rates are lower for coinfected people as compared to those with HCV alone. Many presentations focused on individually tailored treatment – including higher doses of peg-interferon or ribavirin and longer treatment length – as well as looking at which factors predict response to treatment.

The Spanish PRESCO trial studied one brand of peg-interferon (Pegasys) plus ribavirin in 389 people coinfected with HCV and HIV. Those with hard-to-treat HCV genotypes (1 or 4 – about 60% of the total) received treatment for either the standard duration of 48 weeks or for 72 weeks. Those with genotypes 2 or 3 (which respond better to treatment) received either the standard 24 weeks or 48 weeks. At the 14th Conference on Retroviruses and Opportunistic Infections (CROI) in February, researchers reported that the overall SVR rates were 35% for people with genotype 1 or 4 and 72% for those with genotype 2 or 3, but extending the length of treatment did not reduce the risk of relapse for any of the genotypes.

The higher SVR rate in this study than in earlier ones (for example, the pivotal APRICOT trial reported SVR rates of 29% for genotype 1 and 62% for genotypes 2 or 3) may be due to the fact that people in PRESCO had higher CD4 counts (at least 300) and were using higher doses of ribavirin, based on body weight. In addition, they were less likely to use Videx or Retrovir (AZT), both of which can lead to worse side effects when taken with ribavirin.

The researchers found that an undetectable HCV viral load four weeks after starting treatment predicted eventual SVR. As is the case for HIV-negative people, those who did not demonstrate a four-week response were unlikely to later achieve an SVR, suggesting that the four-week mark can guide decisions about whether to stop treatment early. Interferon can cause difficult side effects, so many people prefer to stop treatment that is not likely to produce a cure. Starting treatment with a lower HCV viral load also predicted a higher chance of SVR in people with genotype 1.

(continued on next page)
At the Digestive Disease Week (DDW) meeting in May, Alex Monto reported surprisingly good outcomes in a study of 21 HIV-positive and 112 HIV-negative patients with hepatitis C. Coinfected people actually had a slightly higher SVR rate than those with HCV alone (62% vs. 47%), although the difference was not large enough to reach statistical significance. However, the coinfected patients were about twice as likely (48% vs. 23%) to need EPO injections to manage anemia due to ribavirin. Coinfected people taking “friendly” nucleosides (any other than Retrovir, Videx, or Zerit) or none at all were more likely to respond to interferon treatment.

In another study, presented at the 4th International AIDS Society Conference on HIV Pathogenesis, Treatment and Prevention (IAS) in July, Michael Polis and colleagues treated 11 coinfected patients with Pegasis either once weekly for 48 weeks or twice weekly for four weeks followed by once weekly for an additional 44 weeks. Everyone also took ribavirin. After one week, 72% in the double-dose arm achieved more than a one log reduction in HCV viral load, compared with 9% in the standard-dose arm. Side effects were similar in both groups, but people in the double-dose arm were more likely to achieve an SVR (40% vs. 18%).

Research has shown that people who achieve an SVR experience slower liver disease progression, and may even have a reduction in the degree of fibrosis or cirrhosis (scarring of the liver). In promising news, Anna De Bona reported that interferon appears to reduce the rate of cirrhosis progression even in coinfected people who do not achieve an SVR. In a look back at 25 untreated patients and 25 who were treated but did not achieve an SVR, half the untreated patients saw their cirrhosis progress, compared to just 16% of the non-responders.

Other encouraging news came from Mark Swain, who reported at the DDW meeting that people who achieve an SVR after 24 weeks of treatment have a very good chance of remaining permanently free of HCV. In a study of HIV-negative patients who achieved an SVR, 989 out of 997 – or 99% – still had undetectable HCV viral load five years later. Further research is needed to find out if this will also hold true for coinfected individuals.

Sexual Transmission of HCV
In recent years, outbreaks of HCV that appears to be sexually transmitted have been reported among gay and bisexual men in large European cities, most of whom also have HIV. To date, nearly 400 such cases have been reported in London and Brighton in the U.K., with smaller clusters in France, Germany, and the Netherlands. Similar small outbreaks have also been reported in Australia and the U.S.

At the IAS conference, Mark Danta reported on a study exploring how these outbreaks are related. The researchers constructed “family trees” of HCV taken from 107 men who have sex with men (MSM) in the U.K., 51 in the Netherlands, 24 in Germany, and eight in France. Although half the men had HCV genotype 1a, which is common in Europe, an unusually high number had genotype 4d, the predominant type in Africa and the Middle East. The analysis revealed ten clusters of related HCV strains which accounted for 88% of all infections. Seven clusters contained HCV strains from more than one country, while four included HCV from more than two countries. These findings suggest a large HCV transmission network among HIV-positive MSM in Europe, likely due to travel between cities.

Axel Schmidt studied 22 coinfected MSM in Germany with no history of injection drug use and 44 people with HIV but no HCV. Risk factors for HCV included use of intranasal drugs like cocaine, a history of major surgery, group sex, more than five episodes of unprotected anal sex within the past year, fisting, and use of Viagra. In a multi-factor analysis, however, only intranasal drug use and anal injuries during sex remained statistically significant. Julie Fox reported on 12 MSM with recent HIV infection in London who also became infected with HCV. The median time between HIV and HCV infection was 17 months. Although there was only one new HCV diagnosis in 2003 and 2004, the number rose to six in 2005 and four in 2006. All the men reported unprotected anal sex and use of recreational drugs (including intranasal use).

Martin Fisher from Brighton reported at CROI that sexual transmission of HCV also occurs among HIV-negative MSM. After screening nearly 4,000 patients at an HIV/STD clinic since 2000, the researchers identified 25 new HCV infections: 16 in HIV-
positive men, five in HIV-negative men, and four in men of unknown HIV status. The number increased from zero cases in 2000-2002 to 13 cases in 2006. New HCV diagnosis was linked to fisting, unprotected anal sex, multiple partners, other STDs, and non-injection drug use (including intranasal use). Although HIV-positive men were about 13 times more likely to be diagnosed with hepatitis C, this study shows that HIV-negative MSM are also at risk.

Together, these studies emphasize the need for increased education and prevention efforts, and suggest that at-risk MSM – both HIV-positive and -negative – should be routinely screened for hepatitis C.

Antiretroviral Treatment Interruption

Structured HIV treatment interruption is a controversial issue, with several recent studies showing that certain types of treatment breaks encourage disease progression. The largest of these, the SMART trial, found that stopping HAART (highly active antiretroviral therapy) when the CD4 count fell below 350 and restarting when it reached 250 led not only to more HIV-related opportunistic illnesses, but also to a higher rate of cardiovascular, kidney, and liver complications compared with continuous HAART.

Two studies at the IAS conference looked at people with hepatitis B or C in SMART. Of the 5,472 people with HIV in the study, 2% had HBV, 14.6% had HCV, and 0.25% had both HBV and HCV. There were few deaths due to opportunistic illnesses in either the continuous treatment or the interruption group, and overall death rates were comparable.

But Ellen Tedaldi reported that people coinfected with HBV or HCV had nearly four times the risk of death due to non-opportunistic illnesses. They made up just 17% of all participants, but they accounted for almost 50% of non-opportunistic deaths. Although the most common non-opportunistic causes of death among the coinfected patients were related to substance abuse (overdose, for example) and non-AIDS defining cancers, coinfected people were more likely to die of liver or kidney disease than people with HIV alone, and more often died from unknown causes. The researchers concluded that "HIV treatment interruptions may be particularly harmful for people with HBV or HCV, who already have a higher underlying risk of death."

Liver Disease Progression in People with HIV

Researchers concluded that HIV treatment interruptions may be particularly harmful for people with HBV or HCV, who already have a higher underlying risk of death."

In the second SMART analysis, Greg Dore and colleagues studied whether coinfected patients were more likely than those with HIV alone to have restarted HAART because their CD4 count fell below 250. Since some HIV meds are active against both HIV and HBV, people with both viruses who are taking these drugs face an increased risk of progression of both diseases when they interrupt therapy (study investigators discouraged enrollment of people who needed antiretroviral drugs to control HBV). Indeed, the researchers found that HIV/HBV coinfected participants were significantly more likely to have restarted HAART, and did so sooner after treatment interruption, than either HIV/HCV coinfected patients or those with HIV alone. Although the number of HIV/HBV coinfected participants in SMART was small, these findings suggest they may be at risk for faster HIV disease progression after interrupting HAART.

In a study presented at the 42nd European Association for the Study of the Liver (EASL) meeting in April, Marianne Ziol compared liver fibrosis progression and immune responses in 33 HIV-positive and 33 HIV-negative people with HCV. Everyone with HIV had a CD4 count above 250 and none in either group had been treated for hepatitis C. The researchers found that both groups had similar fibrosis scores (2.69 vs. 2.72 on the Ishak scale of 0-4) and a similar fibrosis progression rate. The frequency and strength of HCV-specific immune responses were also similar. Regardless of HIV status, faster fibrosis progression was associated with reduced HCV-specific immune response.

Two studies presented at CROI also support a link between immune function and liver disease progression. In a study of 1,101 HIV/HCV coinfected Spanish patients, Jose Garcia-Garcia reported that the rate of end-stage liver disease was relatively (continued on next page)
low: about 6% developed decompensated cirrhosis and 3% died of liver-related causes. But complications like abdominal swelling (ascites), enlarged liver, and death due to liver disease were more common among people who had smaller CD4 cell gains (less than 100) after starting HAART. Looking at coinfected participants in the Swiss HIV Cohort, Andri Rauch and colleagues found that those with low CD4 counts had significantly higher HCV viral loads, though what this means in terms of disease progression is not known.

Another study presented at the same conference suggested that HIV-positive people newly infected with HCV may progress faster to advanced liver disease. Daniel Fierer and colleagues performed liver biopsies on five HIV-positive New York City men with acute HCV infection. Despite being recently diagnosed with hepatitis C, four already had moderate (stage 2) fibrosis and moderate or severe liver inflammation. Typically, it takes years or decades for fibrosis to progress to this extent. All five men had no other contributing factors (like heavy alcohol use), and all had relatively high CD4 cell counts (from about 250 to 700). All but one were on HAART. Although this study was small, it suggests that liver disease progression may be accelerated in people who already have HIV at the time of HCV infection, underlining the need for further research.

Baraclude Active Against HIV
Looking at HBV/HIV coinfection, Chloe Thio reported at CROI that, contrary to previous reports, the hepatitis B drug Baraclude (entecavir) is also active against HIV and may lead to HIV drug resistance. Several drugs (Epivir, Emtriva, and Viread) are active against both HIV and HBV, and experts advise that HIV/HBV coinfected people should include at least one of these in their antiretroviral regimens. However, Baraclude was often recommended for coinfected patients being treated for hepatitis B who did not yet require HAART, since using a single HIV drug leads to drug resistance.

Thio’s team identified three HIV/HBV coinfected patients not on HAART whose HIV viral load dropped by at least one log after starting Baraclude. They then confirmed the activity of Baraclude against HIV in laboratory tests, and found that it led to the emergence of the M184V mutation, which causes resistance to Epivir and Emtriva. This contradicts extensive prior testing by the drug’s maker, Bristol-Myers Squibb, showing that Baraclude was not active against HIV. At the 47th Interscience Conference on Antimicrobial Agents and Chemotherapy (ICAAC) in September, the same team reported that Baraclude only partially inhibits HIV, and that the M184V mutation emerges slowly, and not in all cases.

Based on these findings, Bristol-Myers Squibb and the FDA revised the Baraclude product label, and treatment guidelines were updated to recommend that the drug not be used alone by HIV/HBV coinfected patients who are not also taking HAART. This leaves Tyzeka (telbivudine) as the only approved HBV treatment (besides interferon) that appears to have no activity against HIV.

Links to study abstracts are available in the online version of this article at acria.org, and at the following conference websites:

Conference on Retroviruses and Opportunistic Infections (CROI):
www.retroconference.org/2007

European Association for the Study of the Liver (EASL):
www.easl.ch/easl2007

International AIDS Society (IAS):
www.ias2007.org/pag

Interscience Conference on Antimicrobial Agents and Chemotherapy (ICAAC):
www.icaac.org

Liz Highleyman is a San Francisco-based freelance medical writer.

Links to study abstracts are available in the online version of this article at acria.org, and at the following conference websites:

Conference on Retroviruses and Opportunistic Infections (CROI):
www.retroconference.org/2007

European Association for the Study of the Liver (EASL):
www.easl.ch/easl2007

International AIDS Society (IAS):
www.ias2007.org/pag

Interscience Conference on Antimicrobial Agents and Chemotherapy (ICAAC):
www.icaac.org

Liz Highleyman is a San Francisco-based freelance medical writer.

“Despite being recently diagnosed with hepatitis C, four of the five men with HIV already had moderate (stage 2) fibrosis and moderate or severe liver inflammation.”

ACRIA
Drop-In Support Groups

The popular groups formerly offered by Body Positive have found a new home. These peer-led drop-in support groups are held every Thursday and Friday from 6:30 to 8:00 p.m. at the LGBT Community Center, 208 West 13th St., NYC.

For more information, call Gustavo Otto at 212-924-3934 x129.
We're Growing!
As we reported in the last issue of ACRIA Update, we are delighted to have been named lead agency in a $1 million New York City Council program aimed at addressing the growing issue of HIV and older adults by bringing HIV education, prevention, and services to agencies serving middle-aged and older individuals. The initiative builds on our Senior Education and Empowerment (SEE) program, which has been providing HIV education to senior centers across New York City.

All this means, of course, that we have a lot more work to do. And that means hiring some more people to do it. Please welcome these new staff members:

Vaughn Taylor is our new Older Adults Technical Assistance Manager, and he’ll be in charge of the work of the City Council initiative and everything else we will be doing in the area of HIV and aging. Vaughn has a varied and interesting background, having studied nursing and served as a battalion nurse in the U.S. Army Academy of Health Sciences and received an M.A. in Interpretive Jazz with an emphasis on African Ballet. His ten years experience in HIV/AIDS work include three years as Program Coordinator at Aid for AIDS of Nevada and two years as the Founding Executive Director of Fighting AIDS in our Community Today (FACT), both in Las Vegas. He followed that up with five years in Chicago, first as Director of Prevention Education and Programming at the Greater Chicago Committee, then as Founding Executive Director of Jelani Unified Men’s Project, and finally as Health Education Supervisor at the Haymarket Center.

Hanna Tessema joins the HIV Health Literacy Program as an HIV Educator, coming to ACRIA from The Bronx Health Link, where she has worked as Coordinator of Health Education and Outreach for the last two years. Prior to that she worked as a Health Policy Research and Program Administrator in Durban, South Africa, and before that as Minority Peer Advisor, Graduate Student Instructor, Program Coordinator/Student Activities and Leadership at the University of Michigan while pursuing her B.A. in Political Science. Hanna has also done Independent Study in Public Health at the University of KwaZulu Natal in Durban and is currently attending NYU for a Global M.P.H. in Epidemiology, which she expects to receive in 2009.

Robert Hughes, our new Bookkeeper/Junior Accountant, is new to the HIV field, and to the not-for-profit world altogether. Before joining ACRIA, he worked as Front Office Supervisor at the SoHo Grand Hotel and as the Whatever/Whenever Manager at the W New York Hotel. Before that he was first a Guest Service Attendant, then Guest Services Supervisor for the W Chicago City Center Hotel.

Mark Brennan brings impressive credentials with him. He holds a B.A. in Psychology from the University of Wisconsin—Milwaukee, an M.A. in Psychology from Fordham University, and a Ph.D. in Applied Developmental Psychology from Fordham. He has been a Fellow of the Hunter-Brookdale Center on Aging and a Brookdale Doctoral Fellow at the Brookdale Research Institute Third Age Center at Fordham. He has been named a New York State Scholar in Project 2015 by the New York State Office for the Aging and State Society on Aging in New York and has received the Pride Senior Network Recognition Award. His academic staff positions and consultancies are too numerous to mention, and he has participated as principal or co-principal investigator in ten research projects and has authored or co-authored fifty published articles.

ACRIA’s annual Cocktails at Sunset Hamptons event, held July 7 at Ross Bleckner’s home in Sagaponack, raised $150,000. A record 300 friends and supporters turned out to enjoy the music of DJ Paul Sevigny, join in a silent auction featuring works by Bleckner, Julian Schnabel, Stephen Kinsella, and others, and celebrate with event hosts Martha Nelson, Andre Balazs, Kelly Killoren Bensimon, Bob Colacello, Beth Rudin DeWoody, Alex von Furstenberg, and Gillian Hearst-Shaw. We are especially grateful to our sponsors, Banana Republic, In Style Magazine, and the Diller von Furstenberg Family Foundation.

The much anticipated 2007 Unframed art sale was held August 8 and 9 at the Barney’s Coop and raised over $200,000. Host Simon Doonan and benefit committee members Ross Bleckner, Alexis Bryan, Francisco Costa, Beth Rudin DeWoody, Amanda Hearst, Gillian Hearst-Shaw, Deborah Hughes, Adam Lippers, Martha Nelson, and Andrew Saffir welcomed several hundred guests, who lined up down the block for the chance to buy work from leading artists at benefit prices. The 16 participating artists included Vik Muniz, Mitch Epstein, Polly Apfelbaum, Eva Lundsager, Edward Burtynsky, Tobias Wong, and Ted Minow. We offer our special thanks to Barney’s New York, without whom this wonderful event would not have been possible.

ACRIA will once again be the charitable beneficiary for the domino Bazaar, hosted by domino Magazine and Conde Nast and held October 11 through 14. This year’s event will be hosted by Ross Bleckner, Beth Brenner, Dara Caponigro, Francisco Costa, and Sara Rufino Costello. The Bazaar will include unique design stations curated by domino and a special ACRIA gallery featuring new and original works by Bleckner, Jack Pierson, and Christo and Jeanne Claude, among others.

The annual Holiday Dinner will take place on December 11, once again at Donna Karan’s Stephan Weiss Studio. The 2007 dinner will be a celebration of generous and longtime supporter Bob Colacello by Karan, Ross Bleckner, Carolina and Reinaldo Herrera, and Charla Lawhon. Vanessa Carlton will perform. Save-the-dates and sponsorship and ticketing information are forthcoming.
generous contributions

The following persons, corporations and organizations made major donations between July 1 and September 30, 2007 to support ACRIA’s research and education efforts:

Shelley & Philip Aarons
Ivan Abrams
Banana Republic
Barneys New York
Jason Binn
Boehringer Ingelheim Pharmaceuticals
Lorie Broser
Russell Calabrese
Ellen Callamari
Marisa Cardinale
King Yap Chong
Andrew S. Clark
Francisco Costa and John De Stephano
Kathleen Cullen
Dr. Yael Danieli
Beth Rudin DeWoody
GlaxoSmithKline
Dave Greenblatt
Alex Gruen
Couri Hay
Pat Healing
Andreas Hildebrand
Stephen Holden-Style
Flavia Kelson
Kendle NC, Inc.
Ray Kurdziel
Marisa Lakind
Iris Lior & Gary Posternack
Peter McCourt
NAPO Pharmaceuticals, Inc.
Noreen O’Grady
Parexel International
Saara Pritchard
Sam Rabin, Jr.
Ramsay Fairs, LLC
Susannah Z. Ringel
Alvin Rodolfo
Kent Rogowski
Susan Rothstein
Marc & Carolyn Rowan
Leslie and Eric Seid
Nathan Serphos
Stephen Snyder
Sharon Socol
Gerardo Suarez
Dr. Pritinder K. Thind
Vidacare Medical Services
Joanna Wiederhom
Vaughn C. Williams
Howard Wolfson
Alice Zimet
Robert Mapplethorpe Foundation
Tibotec Therapeutics

ACRIA Update is sponsored in part by an unrestricted educational grant from:

GlaxoSmithKline