

Stopping the Spread of HCV and HBV in IDUs



Alan Franciscus, Editor-in-Chief

Injection drug users are at high risk for contracting hepatitis C (HCV) and hepatitis B (HBV). The Urban Health Study has been conducting an on-going study in the San Francisco Bay area since 1986 among the injection drug community. This study is looking at the effect of counseling, education, testing and medical intervention on the spread of infectious diseases such as hepatitis B and C. Data from the Urban Health Study in 1987 found that 75% of injectors tested positive for HCV antibodies within the first two years after they began injecting drugs. This was before the beginning of harm reduction interventions such as education, counseling, and the implementation of needle exchange. A debate has since ensued questioning whether these programs are effective in preventing the transmission of hepatitis C. Now a study by Fan-Chen Tseng and colleagues, *Seroprevalence of Hepatitis C Virus and Hepatitis B Virus Among San Francisco Injection Drug Users, 1998 to 2000*, may finally help to answer these important questions.

In this study the researchers used the data collected from 1986 to 2005 by the Urban Health Study. Demographic information and blood samples were analyzed for

blood-borne infections. The study authors concentrated on HCV and HBV infection in this group and compared it to data collected in 1987.

The study population included about 70% males, 49.5% African Americans, 37.8% whites (non-Hispanics) and 7.1% Latinos. The median age was 45 years, and the median age at which the subjects first injected drugs was 19 years old. Blood samples were taken from the participants and tested for a variety of blood-borne pathogens. Study participants were interviewed, given information on how to prevent infections, and referred to medical and social services. Participants were not required to disclose their names.

Data from 2,296 participants who participated in the study from 1998 to 2000 were analyzed. The authors found that 91% were antibody positive to HCV and 80% to HBV. But among injectors who recently started injecting the rates were much lower – 47% were antibody positive for HCV compared to 71% for those who had been using for 6-9 years. The authors also compared the results from this study to the 1987 study and found a dramatic decrease in new infections – of those who began inject-



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ing drugs for less than two years 76% were antibody positive for HCV compared to 91% for those who had been injecting for 6-9 years.

Looking at how many injectors in the study had been vaccinated against HBV, it was found that in those who recently initiated injection drug use the number vaccinated against HBV increased in the 1998-2000 groups to 4.6% compared to 0.09% for those vaccinated in 1987. Although the rates of vaccination were somewhat dismal, the big picture looks more encouraging among younger injectors (under 30 y.o.) – 17.5% vaccinated, and in those who had been injecting for less than 10 years the vaccination rate was 11.5%. Over-

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IDUs

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all serological evidence of HBV in the 1998-2000 group among recent initiates was 45.2% compared to 67.6% among the 1987 participants.

While it is difficult to prove what exactly caused the decrease in new infections, there is a strong correlation with the decreased sharing of syringes within the past 30 days among injectors – 34% in the participants from the 1998-2000 group compared to 59% who shared syringes in 1987.

The authors concluded that “HCV and HBV seroprevalence among newer initiates to injection drug use in the San Francisco Bay area decreased markedly between 1987 and 1998-2000. This decrease coincided with the implementation of prevention activities among this population.”

Hopefully, this study will put to rest the notion that targeted interventions do not prevent the transmission of hepatitis B and C. More importantly this study could encourage more federal, state and local municipalities to support targeted interventions or increase the types of services already in place that are directed toward the injection drug community. At present, this seems a very likely way to help stop the spread of hepatitis C and hepatitis B.

Reference: “Seroprevalence of Hepatitis C Virus and Hepatitis B Virus Among San Francisco Injection Drug Users, 1998-2000.” Tseng, Fan-Chen; O’Brien, Thomas; Zhang, Mingdong; Kral, Alex; Ortiz-Conde, Betty; Lorvick, Jennifer; Busch, Michael P.; Edlin, Brian. Hepatology; September 2007; (DOI: 10.1002/hep.21765).

Extrahepatic Manifestations: *Membranoproliferative Glomerulonephritis (MPGN)*



Alan Franciscus, Editor-in-Chief

The most common type of glomerulonephritis (kidney disease) found in people with hepatitis C is membranoproliferative glomerulonephritis (MPGN). Other less common forms of kidney disease include noncryoglobulinemic MPGN, membranous glomerulonephritis, MPGN type III, and mesangial proliferative glomerulonephritis.

This article will concentrate on MPGN.

MPGN is a type of kidney disease that is caused by the complexes (such as HCV antibodies, hepatitis C virus, rheumatoid factor) deposited in the membranes of the kidneys. The hepatitis C virus is detected in about 60% of patients with MPGN in Japan and 10-20% of people with MPGN in the United States.

Despite the fact that HCV-related MPGN is relatively uncommon in the hepatitis C population, MPGN is considered a significant problem because of the large number of people infected with hepatitis C and the potential for serious and life threatening complications.

MPGN is usually diagnosed using various laboratory tests such as HCV antibody positive, HCV viral load, elevated liver enzymes, positive rheumatoid factor and circulating cryoglobulins. It can also be confirmed by kidney biopsy.

MPGN is a difficult disease to diagnose because there are few symptoms during the early stages of MPGN. The actual diagnosis of most cases occurs mostly in people

who are in their 50’s and 60’s. It also occurs somewhat more commonly in women than in men. Symptoms can include elevated liver enzymes, hypertension (high blood pressure), joint pain and neuropathy.

The disease progression of MPGN is generally over a long period of time and is highly variable from one person to another. About 10% of people with MPGN will develop serious kidney disease (end stage renal disease) that will require dialysis.

Treatment of HCV-related MPGN usually consists of treatment with interferon monotherapy for the underlying cause – hepatitis C. Interferon treatment has had limited success, but it has been shown to improve the condition of the kidneys, and reduce the incidence of acute flare-ups in some people. Remission of HCV-related MPGN only occurs in a minority of people treated. One of the most important strategies for managing MPGN is to control blood pressure, which will help to prevent further damage to the kidneys. Since ribavirin is mainly eliminated through the kidneys it is not generally used for the treatment of HCV in someone with kidney disease. However, a few studies have found that ribavirin (usually low dose) in combination with interferon has produced better results in helping to manage MPGN. Other treatment options included plasmapheresis (removal of circulating cryoglobulins), and the use of corticosteroids.

Currently studies are looking at the use of pegylated interferon (with and without ribavirin) and Rituximab (Anti-CD20), a drug used to treat lymphoma and some autoimmune diseases such as rheumatoid arthritis.

HIV/HCV Coinfection Updates from the Int'l AIDS Society Conference - Part 2

■■■
Liz Highleyman

About 50 oral and poster presentations at the recent 4th International AIDS Society Conference on HIV Pathogenesis, Treatment and Prevention, held July 22-25 in Sydney, Australia, provided new information on coinfection with hepatitis C or B in HIV positive individuals. Below are highlights from several of these studies. Coverage of hepatitis C disease progression and treatment in HIV/HCV coinfecting people, noninvasive fibrosis assessment, and hepatotoxicity of antiretroviral drugs appeared in the September *HCV Advocate*. For conference abstracts, see <http://www.ias2007.org/pag>.

HIV DISEASE PROGRESSION

HIV/HCV coinfecting patients tend to experience more rapid liver disease progression, but there is conflicting data about the influence of HCV on HIV disease progression. J. Tolia and colleagues (*abstract MOPEB044*) looked at immunological progression in 57 coinfecting patients with undetectable HIV viral load who received treatment for hepatitis C using pegylated interferon alpha-2b (PegIntron) plus ribavirin. Of the 57 patients who began anti-HCV therapy, 18 were excluded from the analysis (11 did not maintain undetectable HIV viral load, three discontinued due to adverse events, and four were lost to follow-up). Of the remaining 39 patients, 13 achieved sustained virological

response (SVR) and 26 experienced treatment failure. Sustained responders had smaller decreases in naive and memory CD4 T-cells and naive CD8 cells compared with nonresponders, as well as larger increase in CD8 memory cells, suggesting that SVR was associated with improved immune function. However, the differences did not reach statistical significance.

ANTIRETROVIRAL TREATMENT INTERRUPTION

Intermittent interruption of antiretroviral therapy based on CD4 cell counts has been explored as a strategy for reducing drug-related toxicities. One of the largest treatment interruption trials, known as SMART, was discontinued last year after it was shown that, compared with those who remained on continuous therapy, patients who interrupted treatment when their CD4 cell counts fell below 350 cells/mm³ were more likely to experience both AIDS-related opportunistic illnesses (those due to compromised immune function) and non-opportunistic complications including heart, liver, and kidney problems.

E. Tedaldi and colleagues (*abstract TUAB203*) assessed outcomes among HIV/HBV and HIV/HCV coinfecting individuals in the SMART study. Out of nearly 5,500 total participants, about 17% were coinfecting (2.0% with HBV, 14.6% with HCV, and 0.25% with both

HBV and HCV). Coinfecting individuals and those with HIV alone had a similar relative increase in the risk of opportunistic or non-opportunistic illnesses or death in the treatment interruption arm compared with the continuous therapy group. Overall, there were few deaths due to opportunistic illnesses among either coinfecting or HIV monoinfecting patients, and death rates were similar. However, the coinfecting patients had almost a four-fold higher risk of death due to non-opportunistic conditions. Although they made up just 17% of the total study population, coinfecting patients accounted for nearly half of all non-opportunistic deaths. The most common non-opportunistic causes of death were substance abuse and non-AIDS defining cancers. Coinfecting people were more likely to die of liver or kidney disease, but those with HIV alone had a slightly higher rate of cardiovascular disease. In addition, coinfecting patients were significantly more likely to have an unknown cause of death. The researchers concluded that interruption of antiretroviral therapy may be particularly harmful for HIV/HBV or HIV/HCV coinfecting patients, who already have a higher underlying risk of death.

MONITORING DISEASE PROGRESSION

Absolute CD4 T-cell count (the number of CD4 cells in a small amount of blood) is generally used to monitor HIV disease progression, but CD4 cell percentage (the proportion of all lymphocytes that are CD4 cells) is preferred in some circumstances. One recent study found that HIV negative people with liver cirrhosis had unusually low CD4 cell counts but normal CD4 percentages, suggesting that

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Hiv/Hcv COINFECTION

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CD4 percentage might be a more accurate marker of disease progression in cirrhotic patients with HIV.

M. Bongiovanni and colleagues (*abstract MOPE063*) looked at absolute CD4 cell counts and CD4 percentages in about 6,000 HIV positive participants in an Italian cohort; 38% were coinfecting with HCV and 5% had hepatitis B surface antigen. About 3% had cirrhosis at enrollment, and an additional 1% progressed to cirrhosis during follow-up. The researchers divided the participants into three groups: those with evidence of cirrhosis; HIV/HBV or HIV/HCV coinfecting patients without cirrhosis; and people without HBV or HCV, normal ALT, and no cirrhosis. Among patients with cirrhosis, having a higher CD4 cell count – but not a higher CD4 percentage – was associated with a lower risk of progression to AIDS. Among the non-cirrhotic patients (with or without HBV or HCV coinfection), lower absolute CD4 cell count and lower CD4 percentage were both associated with a higher risk of progression to AIDS. After controlling for both CD4 values, people with cirrhosis were significantly more likely to develop an AIDS-defining illness. The researchers concluded that absolute CD4 cell count is a better predictor of progression to AIDS than CD4 percentage in HIV positive patients with cirrhosis, and therefore should be used to guide decisions about anti-HIV treatment.

SEXUAL TRANSMISSION OF HCV

Several studies at the conference looked at sexual transmission of HCV. Starting around 2000, clinicians in the United Kingdom and elsewhere in Europe began report-

ing outbreaks of apparently sexually transmitted acute hepatitis C, mostly among HIV positive men who have sex with men (MSM). To date, nearly 400 such cases have been reported in London and Brighton, with smaller clusters in France, Germany, and the Netherlands; small outbreaks have also been reported in Australia and the United States. These cases are assumed to be sexually transmitted since they have been linked to high-risk sexual practices but not to injection drug use.

M. Danta and colleagues (*abstract TUAB201*) constructed phylogenetic “family trees” of HCV isolated from the men involved in the European outbreaks to determine how the virus strains are related. The analysis included 107 men from the UK, 51 from the Netherlands, 24 from Germany, and eight from France. Half had genotype 1a HCV, 23% had genotype 4d, 7% had genotype 3a, 5% had genotype 1b, and 2% had genotype 2. An unusually high proportion had genotype 4, which is the predominant type in Africa and the Middle East, but relatively uncommon in Europe. Genotypic analysis revealed that 10 clusters of related HCV strains accounted for 88% of all analyzed infections. The smallest cluster included three men, while the largest included 36. Seven clusters contained HCV strains from more than one country, and four clusters included isolates from more than two countries. One of the genotype 4d clusters, which comprised 31 men, included isolates from all four countries. The researchers said their findings reveal a large HCV transmission network among HIV positive MSM in Europe, likely facilitated by travel between cities. They recommended that public health agencies should implement targeted hepatitis C prevention strategies for

high-risk HIV positive MSM.

In a related study, A.J. Schmidt and colleagues (*abstract MOPEB037*) assessed social, behavioral, sexual, and other risk factors associated with hepatitis C among MSM in Germany. The study included 22 HIV/HCV coinfecting MSM without a history of injection drug use and 44 matched control subjects who had HIV but not HCV. The researchers found that significant risk factors for HCV infection included use of intranasal drugs such as cocaine, 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 logistic regression analysis, however, only intranasal drug use and bleeding anal injuries due to sex were significant risk factors.

Finally, J. Fox and colleagues (*abstract MOPEB036*) looked at the incidence of acute hepatitis C among 155 MSM with primary HIV infection (i.e., around the time of HIV antibody seroconversion) recruited at Mary’s Hospital in London between 2000 and 2006. One man had HIV/HCV coinfection at enrollment, and 12 others experienced HCV seroconversion during follow-up. Among these 12, the median time between acquiring HIV and acquiring HCV was 17 months (range 5 to 41). One new HCV diagnosis was made in 2003 and one in 2004, but the number rose to six in 2005 and four in 2006. All of the coinfecting men reported unprotected sex with individuals presumed to be HIV positive and all had used recreational drugs (including intranasal use) within the preceding three months. Most patients (83%) had HCV genotype 1a, but the remainder had genotype 4d. The researchers suggested that high-risk MSM should be tested more regularly for HCV.



HealthWise:

Protection from Viruses: The Mighty Immune System (Second in a four part series)



Lucinda K. Porter, RN

Last month's *Healthwise* presented basic information about viruses; this month's column explores defenses against them. The most effective weapon against viruses is prevention. Vaccines, good hygiene and safety precautions are some ways to do this. The next best weapon is a good immune system.

Viruses are transmitted in multiple ways. Some viruses are spread in the air; landing on us after someone has coughed or sneezed. We are susceptible to stomach viruses if we eat food that hasn't been properly cleaned or cooked. Viruses are transmitted by touch, whether casual or intimate. Some are passed by blood-to blood contact with someone who has a virus, such as hepatitis C (HCV). Animals may also transmit viruses, such as West Nile.

If prevention fails and a microbe makes human contact, then our immune system takes over. The immune system is not a connected link like the digestive or nervous systems. It is a complex network of organs, cells and chemicals that protect our health. Nearly every part of the body is part of the immune system, including single molecules as well as large organs such as the liver and skin.

The immune system is much like a game of chess. There are lots of options and strategies, each changing depending on the opponent's move. Although it is complex, this article will explain the immune system in more simple terms.

Humans are born with some immunity. This is called *natural* or *innate immunity*. Our bodies have external and chemical barriers that keep out foreign matter. These include skin, sweat, mucus, nose hairs, eyelashes, stomach acid, coughing and sneezing. Microorganisms that live in our bodies also protect us.

We have an *immune surveillance* mechanism. Our body is equipped with the capacity to find and destroy foreign matter. It does this by producing an *immune*

response. An immune response is the body's counterattack to foreign invasion. Foreign matter may be viruses, bacteria, pollens, fungus, parasites, etc.

There are many categories of immune responses. One is a *nonspecific immune response*. Not caring what the attacker is, this type of response is armed and ready to destroy all invaders. The *specific immune response* is particular. It uses a recognition system and sends out a specialist to finish off the intruder. Foreign microbes that trigger a specific immune response are called *antigens*.

If a microbe penetrates any of our natural barriers, the immune surveillance mechanism triggers various reactions. Patrolling cell eaters devour the invaders. These cell eaters are large white blood cells that clean up debris. Some white cells use chemicals to destroy microbes.

White blood cells (WBCs) are the army of the immune system. WBCs originate in the bone marrow. When the immune system needs help, it recruits more WBCs. These multiply in the *lymphatic system*, which is part of the immune system. The lymphatic system includes nodes, organs and vessels.

As the WBCs are multiplying, lab tests will show an elevated white count. This usually indicates an infection. There are five types of white blood cells – *neutrophils*, *monocytes*, *lymphocytes*, *eosinophils* and *basophils*. Each of these has their own function, but some of them kick in if one of the others fails to destroy unwanted microbes.

Inflammation weakens or wipes out foreign invaders. Inflammation is primarily a nonspecific immune response, although inflammation can occur during a specific response. The signs of inflammation are redness, swelling, heat, and pain.

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IMMUNE SYSTEM

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Imagine you have scraped your knee on the ground. Blood flow increases to the knee. The area swells because tiny vessels allow more proteins and fluids to flood the area. White blood cells engulf invaders and debris, permitting the tissue to repair itself.

Meanwhile, the lymphatic system is organizing its defense. A clear lymphatic fluid flows throughout the body, transporting *lymphocytes* as they patrol for antigens. Lymphocytes are white blood cells and are vital to immune function. There are various types of lymphocytes, each with a specific job to do. *B cells* and *T cells* are important lymphocytes.

B cells monitor the body, looking for foreign matter. B cells are highly specific. Each B cell has a sensing device for only one type of antigen. If a B cell senses an antigen, it needs help to deactivate the antigen. The B cell transforms into a bigger cell and these cells produce millions of *antibodies*. Antibodies latch on to the antigens and direct the immune system to eliminate them.

An amazing feature of the antibody process is that after the first encounter, the number of antibodies drops. However, after each subsequent attack, the antibodies reappear faster and in greater numbers. This is because B cells have produced *memory cells*. Memory cells know how to produce antibodies, but they don't do this until the second exposure to the antigen.

If the invading microbe is a virus, the immune system may use a different strategy. It will use T cells in a direct attack on antigens. These cells depend on a recognition system that can tell the difference

between what belongs to the body and what doesn't. T cells destroy cancer cells. Organ rejection in transplant patients occurs because T cells don't recognize the new organ.

The immune system relies on a fundamental concept – that it can recognize the cells it needs to protect. Unfortunately, sometimes it gets confused and starts to attack itself. This is how autoimmune diseases start. Type 1 diabetes, lupus and rheumatoid arthritis are autoimmune diseases.

Important weapons in the immune system's arsenal are the interferons. Their job is to prevent viral replication in the host cells. The interferons direct the cells to make enzymes. These enzymes interfere with the virus's attempt to multiply. Interferons provide a nonspecific immune response. Although they appear naturally in our bodies, interferons can be manufactured. Alpha-interferon is used to treat HCV infection.

This brief introduction to the immune system describes some of the mechanisms that fight viruses and other microbes. This elaborate system protects us in wonderful ways. Next month we will discuss a simple and more effective weapon against viruses – prevention.

Resources:

National Cancer Institute www.cancer.gov/cancertopics/understandingcancer/immunesystem

National Institute of Allergy and Infectious Disease www.niaid.nih.gov/final/immun/immun.htm#How

How Stuff Works www.howstuffworks.com/immune-system.htm



Disability Programs from Social Security

A new HCSP Training Module

This July, the first of a new series of HCSP Training Modules focussing on Disability, Insurance and Benefits issues was posted to the HCV Advocate website.

The online modules have been designed and written by Jacques Chambers, CLU, a Benefits Consultant and Counselor, who has been a contributing writer at the HCV Advocate for many years.

Jacques has spent the last ten years helping people dealing with disabilities understand and access their benefits. Prior to that he spent twenty-five years in the insurance industry, designing, selling, and servicing employee benefits programs.

Disability Programs from Social Security, will focus on

- Differences and similarities between Social Security Disability Insurance (SSDI or SSD) and Supplemental Security Income (SSI)
- Definition and determination of disability
- Financial eligibility requirements
- Navigating the application process
- Dealing with a claim denial
- Appealing a decision

To take this module, go to www.hcvadvocate.org and follow the instructions.

Disability & Benefits:

Talking to the Disability Examiners



Jacques Chambers, CLU

There seems to be a belief among people filing for disability benefits that talking to anyone working on your case should be avoided at all costs. People applying for disability benefits are afraid that they may say something that will ruin their chances for approval. People already collecting disability think if they keep quiet and don't contact anyone, their checks will keep coming.

While there may be a grain of truth in some of these concerns, it is a very small grain, and many people take that to the extreme of avoiding all possible contact with the "disability people" – frequently to their own detriment.

The general rule is that there is nothing wrong with talking to the people handling your disability claim whether it is from an insurance company or from Social Security or another government program. But there are exceptions to that general rule in both directions; there are times when it's best not to say too much, but there are times when it is important to stay in contact.

An important part of that general rule is that when you do talk to someone about your disability claim, you should keep a written record of it. Have a notepad or notebook at hand when you make a call, or receive one. You should record the following:

1. Time and date of the call;
2. Phone number called including the extension;

3. The name of the person spoken to (Don't be embarrassed to ask him or her to repeat it or spell it);
4. What was said; and,
5. What the next step is. (Is something being sent to you; are you sending something; do you have to contact someone else; when do you need to look at this again)

(See the other suggestions at the end of this article.)

DISABILITY INSURANCE CLAIMS

Insurance companies are different from the government. Whether the people you speak with are conscious of it or not, the insurance company would be much happier (because it would be more profitable) if your claim were denied or terminated. Because of this you do want to be more careful when contacting an insurance company.

I do not recommend detailed descriptions of your condition or work problems by phone. Those should be in writing. If an insurance company wishes to conduct a telephone interview with you, ask if you can review a copy of the transcript or summary for accuracy before it is put into your claim file. If not, take extra good notes. There are times, however, when you *should* talk with insurance company personnel, even contact them.

- First and foremost, you should contact the insurance company every time you send them something, just to confirm that

they received it. Even when mailing the initial claim form, you should call the carrier after about one week, and ask if they received it. If they have already assigned it to a claims adjuster, speak with that adjuster, introduce yourself, get his or her name and direct phone extension and offer to help get any medical records or other documents they need.

- Contact the insurance company if you receive some correspondence that you do not totally understand. However, keep in mind that what you are told by the insurance company over the phone doesn't mean a thing. Some people will say almost anything over the phone, knowing that they won't be held responsible. If they have to put it in writing, chances are they will make sure they are right before writing it down.
- For substantive information the best record is the always a written record. If you are in a phone conversation and the information or questions get somewhat complicated, or if you just want verification of what is being said, try asking something like, *"I have trouble remembering things and this is so complicated. Could you put that in writing and send it to me?"*

SOCIAL SECURITY AND STATE DISABILITY CLAIMS

Despite what rumor mills, blogs, and bulletin boards may say, Social Security is really only concerned that their rules are being followed; they really don't care whether benefits are approved or denied, continued or stopped, as long as they

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TALKING TO EXAMINERS

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followed proper procedure. Besides, it's not their money they're spending. The same seems to be true of the five state disability programs.

I strongly encourage staying in contact with Social Security as much as necessary without becoming a pest while a claim is pending. The Claims Representative that takes your initial claim will give you the name and phone number of the Claims Analyst who will be requesting and reviewing your medical evidence, once it is assigned.

The biggest problem that Social Security staff has is dealing with the number of claimants in their caseload. Social Security is operating at almost the same level of employment as they were thirty years ago, even though the number of claimants and programs (remember Medicare Part D?) have dramatically increased.

When your disability claim is being processed, the analyst can only follow up on outstanding medical records on a monthly basis. You can speed up your claim by working with the analyst and making sure the doctors all get the requests and send their records promptly.

The toll-free number for Social Security (800-772-1213) can answer most questions once a claim has been approved and payments have started, and that number is active from 7AM to 7PM regardless of your time zone.

Whether it is a government agency or an insurance company, there is no reason to try to hide or avoid talking to the representatives working on your claim. Just follow the guidelines given above. However, *you should review the following suggestions before calling them:*

- **Don't be put off by voicemail** – As aggravating as it may be to deal with voicemail, it is very efficient and you can put it to good use. Many times you will always get voicemail when trying to call your analyst, representative, or adjuster. That's OK. Leave a message, but don't just ask for a return call.
 - In the message, ask the question you want to get answered. That will give the person a chance to research the answer before calling you, and, if necessary, leave the answer on your voicemail.
 - There are times that a person and my office will only exchange voicemails; and we will never speak directly to them, but the business still gets accomplished and the questions get answered.
 - Do not be impatient with the person if your call isn't returned. They really are busy. Our office policy is to let one day go by, then call again. We also never mention unreturned calls. We simply leave the message again and apologize for bothering them since they are so busy. This may happen three or more times before a call is returned, but it will be returned eventually, and there will be no hard feelings because we did not complain about the delay.
- **Attitudes can reflect like a mirror** – Many times, a client has complained that the people they contacted had a "bad attitude" and "talked down" to him or her. I am convinced that many times, this is just a reflection of the attitude of the caller when placing the call. Our office finds that when we are upbeat and friendly, the persons we speak with are usually the same and much more helpful.
- **Talk to the person, not the office** – It's easy to picture monsters and ogres working for the companies and agents and to imagine them squealing with glee when they can deny your claim, but these people are mostly human and just trying to do their jobs. Treat each one as a person, try to be friendly, try to personalize the conversation, and you may find you have an ally who will help and not be an obstacle. Then again, don't expect miracles; they really don't have time to become your phone pal.
- **Be generous with compliments** – If the claims representative goes out of his/her way or gives you better than expected service, let them know. In fact, if they are especially helpful, get the name and number of their supervisor and let him or her know.
- **Play dumb** – You're much more likely to get the attention and advice of a claims representative by playing the helpless, ill, lost-in-the-system role. Demands, orders and threats won't help your case move any faster, at least not initially.
- **Don't bother with threats** – These people are regularly threatened with lawsuits and insurance department complaints. It doesn't scare them, but it does make you "an enemy." Also, **DO NOT ARGUE**; arguments belong in written appeals not telephone conversations.



Exercise Guidelines

Alan Franciscus, Editor-in-Chief

In the past couple of years, information about the relationship between many lifestyle conditions such as metabolic syndrome, insulin resistance, obesity and steatosis, and HCV disease progression and treatment outcome has come to light. For most people, healthy lifestyle choices, such as eating nutritious meals and a regular exercise routine can help reverse these conditions and even improve or compensate for the overall negative impact of these conditions on HCV disease progression and treatment outcome. Exercise has been found to be one of the most important components of staying healthy and living well with hepatitis C.

The American College of Sports Medicine (ACSM) and the American Heart Association (AHA) recently updated the exercise recommendations for healthy adults and adults with chronic conditions. This is the first update since 1995 and includes recommendations for resistance trainings as well as aerobic exercise. The new guidelines will help us all to reach an exercise goal that will improve our overall health and help our bodies fight Hep C.

As always, check in with a medical provider about starting any exercise routine. The updated guidelines are for different ages and are also based on chronic health conditions. This article will focus on the recommendations both for healthy adults under age 65, and for adults over age 65. The recommendations for adults over age 65 are also the same recommendations for people living

with a chronic condition. Check in with a medical provider about the level that is appropriate for you based on the degree of HCV disease progression and/or HCV symptoms.

GUIDELINES FOR HEALTHY ADULTS UNDER AGE 65:

- Do moderately intense cardio 30 minutes a day, five days a week, or
- Do vigorously intense cardio 20 minutes a day, 3 days a week,

AND:

- Do eight to 10 strength-training exercises, eight to 12 repetitions of each exercise twice a week

GUIDELINES FOR ADULTS OVER AGE 65 (OR ADULTS 50-64 WITH CHRONIC CONDITIONS, SUCH AS ARTHRITIS):

- Do moderately intense aerobic exercise 30 minutes a day, five days a week, or
- Do vigorously intense aerobic exercise 20 minutes a day, 3 days a week

AND:

- Do eight to 10 strength-training exercises, 10-15 repetitions of each exercise twice to three times per week

AND:

- If you are at risk for falling, perform balance exercise,

AND:

- Have a physical activity plan.

Moderate-intensity aerobic exercise means working hard at about a level-six intensity on a scale of 10. **Important:** you should be able to carry on a conversation during exercise.

Some additional information on exercise was also included:

- You can perform the moderate-intensity workouts throughout the day in 10-minute bouts.
- Mix up your combinations of moderate- and vigorous-intensity.
- It is also important to mix up your routines or types of exercise to prevent boredom from setting in.
- Include flexibility or stretching routines before and after exercise.

Exercise is a lifelong commitment so try to find a variety of exercises that you enjoy. This will ensure that you succeed in your lifelong goal to become healthier.

For more detailed information about the recommendations go to www.acsm.org

Reference:

Physical Activity & Public Health Guidelines by the American College of Sports Medicine (www.acsm.org) and the American Heart Association (www.americanheart.org)



Kentucky Organ Donor Registry

October is National Liver Awareness month. Being willing to donate organs and tissue may be the perfect way to honor the complex, hard-working liver that we can't live without. The state of Kentucky has an easy, confidential process for doing this. Those living in the state of "unbridled spirit" can sign up for the Kentucky Organ Donor Registry at www.donatelifeky.org. Signifying your intention to become a donor may also be done at driver license renewal time. Kentucky does not require family consent to carry out the wishes of adults. However, advance notification of family is highly advised. For more information: www.donatelifeky.org
www.trustforlife.org
 (866) 945 LIFE (5433).

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Interferon and Liver Cancer

Alan Franciscus, Editor-in-Chief

In the United States, hepatocellular carcinoma (HCC or liver cancer) is one of the fastest growing causes of cancer-related death. The primary causes of HCC in this country are hepatitis B, alcoholic liver disease and hepatitis C. Hepatitis C now accounts for the largest increase in HCC while the rates of HBV- and alcohol-related HCC have remained the same over time.

In the last 5 years we have seen clear evidence that interferon therapy (with and without ribavirin) can help to slow down or even reverse some of the damage caused by hepatitis C. But does this effect translate into preventing HCV-related HCC? Since HCV-related HCC usually only occurs after the development of cirrhosis it would seem likely that HCV therapy should slow down or stop the development of HCC. Unfortunately, there has been limited data that can answer this question until now – a new study released in the August issue of the *Journal of Medical Virology* may help to answer this important question.

Dr. Yasuji Arase and colleagues at Toranomon Hospital in Tokyo, Japan, studied 120 HCV positive patients with biopsy-proven chronic hepatitis C or liver cirrhosis. Study participants were 60 years and over with elevated serum aminotransferase. The 120 patients were treated with standard interferon (IFN-3 million units, two to three times a week for between 0.5 to 15.5 years – median treatment duration was 2.47 years). The control group consisted of 240 patients who were treated with herbal medicines, but did not receive interferon (non interferon group). The patients who were

not treated with interferon were matched 2 to 1 with the interferon group for gender and age.

The results found that serum alpha-fetoprotein (AFP-an indicator of liver cancer) level decreased significantly after starting interferon treatment compared to those who received no interferon treatment. It was also found that the rates of HCC were significantly lower in the interferon group compared to the non-interferon group. The 5 year cumulative rate of HCC was 5.9% in the interferon treated group compared to 13.7% in the non-interferon group. The 10-year cumulative rate was 17.1% in the interferon treated group compared to 32.8% in the non-interferon group.

The authors concluded that “long-term IFN therapy for aged patients with chronic HCV infection is effective in decreasing the serum AFP level and preventing hepatocarcinogenesis.”

Reference:

“Prolonged-interferon therapy reduces hepatocarcinogenesis in aged-patients with chronic hepatitis C.” *J Med Virol.* 2007 Aug;79(8):1095-102



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