

## In this issue:

- 1 Laparoscopic Living Donor Liver Surgery
- 2 Advances in Liver Preservation Result in Improved Outcomes
- 3 Chronic Hepatitis B Update
- 4 Hypertension Management Post-Liver Transplant
- 5 Management of Bone Disease Pre- and Post-Liver Transplant
- 6 The Importance of a Caregiver and a Community Physician for the Success of a Liver Transplant
- 7 New Faculty and Staff

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Benjamin Samstein, MD, Surgical Director, Living Donor Liver Transplant Program

## Minimally Invasive Live Donor Hepatectomy

Over the past six months, the CLDT has introduced laparoscopy to all living donor surgeries performed at NewYork-Presbyterian Hospital (NYPH).

Laparoscopy promises to significantly shorten recovery in comparison to the standard procedure for living donor hepatectomy, which involves an incision extending from the breast bone to the navel. If the laparoscopic approach catches on and becomes a standard procedure, it could potentially revolutionize living donor transplantation by encouraging more donors, increasing the availability of organs, and improving outcomes.

Despite the facts that recipient outcomes for living donor liver transplant have improved during the past five years, and that living donor grafts are often superior to deceased donor grafts, living donor transplantation volume has not increased dramatically across the United States. This is because the standard adult organ donor surgical procedure requires a long recovery and has been characterized by a significant level of morbidity. In addition, long recovery time for a parent donating a portion of their liver to their child impedes their ability to care for the child, and has potential for a debilitating effect on the family.

After introduction of the laparoscopic approach for all NYPH living donor hepatectomies during the summer of 2009, the Center for Liver Disease and Transplantation (CLDT) surgeons have performed 13 laparoscopic living donor surgeries, including three that were fully laparoscopic. For adult-to-adult living donor liver transplantation, laparoscopic techniques result in incisions half the size of the standard incision for open hepatectomy and avoid the muscle division required for the standard procedure. For adult-to-pediatric living donor liver transplantation, the donor receives five small incisions, the largest of which is three inches long. In these procedures, division of the donor liver is achieved with the same techniques as in the standard procedure, and the proportion of liver removed is the same. Implantation of the liver into the recipient is performed using standard techniques.

We have been encouraged by results to date. Recovery from laparoscopic donation appears to be significantly faster than for standard donor surgery, enabling donors to get back to



Continued on page 6



Traditional incision for right hepatectomy



Minimally invasive right or left hepatectomy incision



Fully laparoscopic left lateral hepatectomy

James V. Guarrera, MD, FACS, Surgical Director, Adult Liver Transplantation\*

## Emerging From the “Ice Age” of Liver Preservation

Preserving organs on ice prior to transplantation, an approach known as cold storage or CS, has been standard practice in liver transplant for 20 years.



Now there is new evidence that the preservation technique hypothermic machine perfusion (HMP) may offer improved transplant outcomes. The first-ever study comparing the impact of CS and HMP was carried out at NewYork-Presbyterian Hospital/Columbia University Medical Center (NYPH/Columbia). We found in this study that HMP has advantages over CS in preserving donor livers — and that it most likely constitutes an advance over the traditional method. By improving preservation and reducing preservation-related injury, we may be able to expand the availability of organs for transplantation to more patients who need them.

Cold storage involves flushing out the liver with a preservation solution and storing the organ in a cooler of ice. This reduces the metabolic rate, which is a fairly effective way to keep a liver healthy en route to transplant surgery. Machine perfusion, on the other hand, provides a continuous flow of oxygen and key nutrients to the liver while diluting and removing toxins and waste products. It appears from our preclinical work and kidney transplant experience that machine perfusion promotes better function and less preservation-associated damage after transplant. Our study compared 20 transplant patients who received HMP-preserved livers with 20 patients with CS-preserved livers, finding that the first group experienced shorter hospital stays and fewer long-term complications. The HMP group also had lower levels of the blood markers indicating liver injury that may have occurred during the preservation interval.

The findings are reported in the February issue of the *American Journal of Transplantation*.\* The study was supported by a grant from the Health Resources and Services Administration (HRSA), Division of Transplantation.

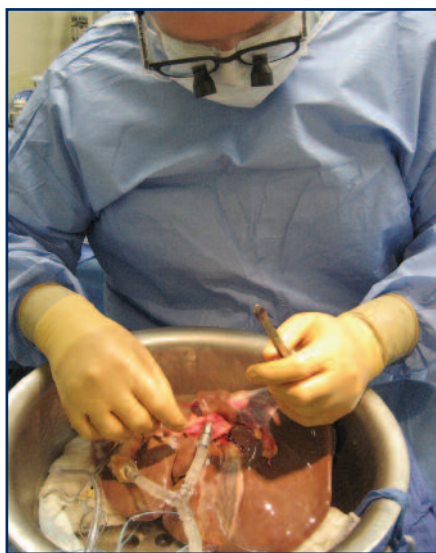
### Expanding Availability of Livers for Transplant

Improving organ preservation is especially important in light of major changes in the transplantation landscape during the past two decades and could broaden the availability of donor organs.

In the early days of liver transplantation, high-quality organs were plentiful for two reasons: First, liver transplantation had not yet become widespread, so demand was relatively low; and second, there was a greater supply of livers from young trauma victims. Thanks to a significant drop in violent crime and to public safety measures such as mandatory seat belt use, the pool of young

donors has shrunk — and that's obviously a good thing. But there is no denying the stark fact that the median age of the average liver donor is higher today, which means that the quality of available organs is reduced.

The HMP technique dates back to the 1960s, when it was introduced for kidney preservation. It was soon dropped in favor of cold storage, a method that was deemed simpler. But in the 1990s, HMP made a comeback in kidney transplantation, coinciding with greater reliance on “imperfect” kidneys from older donors with more comorbidities.



When we began our research, investigators in the NYPH/Columbia-based HMP study strongly suspected that HMP could be adapted to the liver transplantation setting, particularly because the liver is inherently more vulnerable to injury than the kidney. We began using a pump produced by Medtronic originally designed for use in cardiopulmonary bypass, combined with a preservation solution called Vasosol.

We have now received FDA approval to conduct a phase II study focusing specifically on the effects of HMP in livers from extended-criteria donors, a group that makes up a growing proportion of the total number of donors today. Organs from these older, sicker donors are the ones most likely to benefit from machine

perfusion. This trial is also supported by the HRSA Division of Transplantation grant.

Establishing the benefits of HMP over CS will depend on the results of larger clinical studies, but it is equally important to clarify the way the two techniques play out at a cellular and molecular level. Molecular and mechanistic studies also are underway at NYPH/Columbia.

Through these studies, our team aims to show that even imperfect livers can be maintained in peak condition via HMP during the critical period when they are in transit from donor to recipient. This is the kind of “quality improvement” that will translate into long-term benefits for patients. ■

\* Dr. Guarrera is Principal Investigator of the Liver Machine Preservation Trial. Findings of the trial were published in the *American Journal of Transplantation* in February 2010:

Guarrera JV, Henry SD, Samstein B, Odeh-Ramadan R, Kinkhabwala M, Goldstein MJ, Ratner LE, Renz JF, Leeb HT, Brown, Jr., RS, and Emond JC. Hypothermic Machine Preservation in Human Liver Transplantation: The First Clinical Series. *American Journal of Transplantation*, 2010; 10 (2): 372-381.

Damaris C. Carriero, CNP, MHA, Hepatology Nurse

## Review of Chronic Hepatitis B: First in a series of articles about hepatitis B

An estimated 350 million individuals are chronically infected with hepatitis B virus (HBV) worldwide.

Persons with chronic HBV (CHB) are at greater risk for hepatocellular carcinoma (HCC) than uninfected individuals, and the relative risk of HCC has wide variability in both case-control and cohort studies.<sup>1</sup> Results from a large population-based prospective cohort study of untreated individuals with CHB — The Risk Evaluation of Viral Load Elevation and Associated Liver Disease/Cancer-In HBV (REVEAL-HBV) study — demonstrate the need for prolonged suppression of HBV replication to prevent CHB-related liver disease. Data show a strong association between progression to cirrhosis and viral replication.<sup>2</sup> The cumulative incidence of cirrhosis increased with the HBV DNA level and ranged from 4.5 to 36.2% for patients with serum HBV DNA of less than 300 copies/ml and greater than 106 copies/ml, respectively. Additionally, elevated viral DNA was shown to be an independent risk factor for the development of HCC. Corresponding cumulative rates of HCC were 1.3 and 14.9% for patients with serum HBV DNA less than 300 and greater than 106 copies/ml, respectively.<sup>3</sup> This association underscores the importance of durable HBV DNA suppression with concomitant histological improvement as a primary objective to prevent cirrhosis, decompensation, HCC, and death.

### Natural History

The natural course of CHB is an actively changing process, and viral, host and external factors determine disease progression. CHB presents as either hepatitis Be antigen (HBeAg)-positive ('wild type') or HBeAg-negative disease. 'Wild type' CHB represents an early phase of chronic infection, while HBeAg-negative disease represents a later phase and is associated with naturally occurring HBV variants. The natural history of CHB is divided into five phases. The 'immune-tolerant' phase is characterized by HBeAg positivity, high serum HBV DNA levels, normal or minimally elevated alanine transaminase (ALT) levels, and mild inflammation, with absent or limited fibrosis. High levels of viremia make these patients highly contagious. The 'immune-reactive' phase is characterized by HBeAg positivity, lower serum HBV DNA levels, persistently or intermittently elevated ALT levels, moderate or severe active inflammation, and more rapid fibrosis progression. This phase may last several weeks to several years. Very low or undetectable serum HBV DNA levels, normal ALT levels, and minimal fibrosis characterize the 'inactive HBV' or 'HBsAg carrier' state. This state may follow seroconversion from HBeAg to anti-HBe antibody and, due to immunologic control of infection, confers very low risk of cirrhosis or HCC. 'HBeAg-negative CHB' may follow seroconversion from HBeAg to anti-HBe antibodies during the immune-reactive phase. It is charac-

I have chosen to present our review of hepatitis B in parts to deliver the best comprehension of the disease, sequelae, and implications for community as well as global health policies. This article is an excerpt from the recently published chapter "Hepatitis B therapies and antiviral resistance detection and management" in Expert Review of Gastroenterology and Hepatology.\*

terized by periodic reactivation with a pattern of fluctuating HBV DNA and ALT levels, and active inflammation. Patients with HBeAg-negative disease have HBV variants with nucleotide substitutions in the precore and/or the basal core promoter regions that express no or low levels of HBeAg. Furthermore, they have low rates of prolonged spontaneous disease remission and have active liver disease with a high risk of fibrosis progression, decompensated cirrhosis and HCC. The 'resolution' phase is characterized by anti-HB surface (HBs)Ag loss, undetectable serum HBV DNA, although low-level HBV replication may persist with detectable HBV DNA in the liver; and detectable anti-HB core (HBc) anti-bodies with or without HBsAg. HBsAg loss is associated with reduced risk of cirrhosis and HCC, although immunosuppression may lead to HBV reactivation.<sup>4,5</sup> Hepatitis B virus has a high rate of replication,<sup>6</sup> and unlike most DNA viruses, replicates via reverse transcription and is distantly related to the retroviruses. The HBV replication process is mediated by the viral polymerase. Rapid viral turnover and the distinctively error-prone retroviral life cycle lead to the development of HBV polymerase mutants. Virus mutations also cause the emergence of quasispecies that evolve during the course of infection secondary to host selective pressure.<sup>7</sup> Overlapping HBV genes, the poor replication accuracy

of HBV polymerase and the reverse transcription strategy of HBV lead to genetic variations that affect infectivity, vaccine efficacy, pathogenesis, the response to antiviral therapy, and transmission.<sup>7,8</sup> ■

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## Cardiovascular Management Post-Liver Transplant

As long-term survival after liver transplantation (LT) increases and outcomes continue to improve, metabolic complications have become more prevalent.



When compared with age- and gender-matched controls, liver allograft recipients have a higher risk for cardiovascular and cerebrovascular events, and death.<sup>1</sup> Early recognition and treatment of comorbid conditions, specifically hypertension, diabetes, renal impairment, and hyperlipidemia are essential to healthy survival.

Hypertension is a silent killer, often requiring decades before its implications for morbidity and mortality are fully realized. The negative impact of cardiovascular disease on LT recipients is brought to light by recent data that indicate it is a significant cause of late mortality, second only to malignancy.<sup>2</sup>

Data from previous studies show that the incidences of hypertension, hyperlipidemia, and diabetes post-transplant are as high as 85%, 66%, and 60%, respectively. Prevalence rates of cardiovascular events range from 9.4% at 5 years to 25% at 10 years post-liver transplant.<sup>1</sup>

Currently, there are no specific guidelines for the treatment of cardiovascular complications in LT recipients.<sup>3</sup> JNC 7 guidelines (2003) define blood pressure of 140/90 in the general population and 130/80 in persons with DM or chronic kidney disease (CKD) as thresholds for pharmacologic intervention.<sup>4</sup>

Studies of patients with hypertension have shown the benefits of therapeutic lifestyle changes in the nontransplant population. Calcium channel blockers (CCB), beta blockers, and angiotensin-converting enzyme inhibitors (ACEI) are efficacious in the LT patient. However, comorbid conditions such as diabetes, hyperlipidemia, and CKD; history of MI or heart failure; and drugs that undergo CYP metabolism are important considerations for appropriate management. Choice of pharmacotherapeutic agent(s) should be guided by side effect profile and potential drug-drug interactions.

Renal impairment after liver transplant is associated with CNI-dependent hypertension that results from hemodynamic and histological abnormalities. Anti-hypertensive agents may improve CNI-associated renal impairment. Management of CNI-induced nephrotoxicity may require dose reduction of immunosuppression, which is guided by graft function, drug levels, and optimal treatment of hypertension.

The criteria for diagnosis and management of diabetes in the nontransplant population are applicable to LT patients. Risk factors for diabetes mellitus after transplantation include older age, Black race or Hispanic ethnicity, obesity, hepatitis C infection, corticosteroids (standard of care therapy post-transplantation) and CNI, specifically tacrolimus, a more diabetogenic agent than cycloSPORINE.<sup>5</sup> Therapeutic management for most patients requires daily insulin. Patients with persistent hyperglycemia despite withdrawal of glucocorticoids may remain on insulin or change to oral antidiabetic agent(s).

Treatment recommendations for dyslipidemia are set forth in The Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III, or ATP III) from the National Cholesterol Education Program (NCEP).

Reversible risk factors for dyslipidemia after liver transplant include obesity and immunosuppression. Glucocorticoids are associated with increased total cholesterol (TC) and LDL cholesterol (LDL-C), decreased HDL cholesterol and increased triglycerides.<sup>6</sup> CycloSPORINE is associated with increased LDL-C and TC, and sirolimus is strongly associated with hypercholesterolemia. There is no evidence to date that tacrolimus has this lipogenic effect.<sup>7</sup>

CycloSPORINE and tacrolimus, as well as fibrates and most statins, undergo CYP metabolism. Statins and fibrates should not be combined in patients receiving CNI.

Cholestyramine, a resin and bile acid sequestrant used to treat dyslipidemia, may decrease absorption of CNI, and doses should be separated by at least two hours. Hypertensive LT patients who are managed with CCB must be closely monitored when statin therapy is initiated, because drug-drug interactions may increase serum concentration of the statin and risk for toxicity.

The management of metabolic complications in the LT patient should be a joint collaboration between the transplant team and primary care physician. We welcome involvement by community physicians in the effort toward improving long-term outcomes in this patient population. ■

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## Management of Bone Disease Pre- and Post-Transplant

Diseases of the bone associated with chronic liver disease (termed hepatic osteodystrophy) are common both pre- and post-liver transplantation.

Hepatic osteodystrophy encompasses osteoporosis and less commonly, osteomalacia from poor bone mineralization due to vitamin D deficiency. Historically, osteomalacia has been reported in a high percentage of individuals with primary biliary cirrhosis, but today is rarely seen in adult patients with chronic liver disease (LD). This shift is possibly due to earlier diagnosis, recognition, and improved nutritional management. It was reported that 96% of patients awaiting liver transplantation have low 25-hydroxy vitamin D levels in the absence of osteomalacia, suggesting that osteomalacia in this patient population is uncommon.<sup>1</sup> Osteoporosis is more common in patients with LD and it is important that clinicians understand surveillance and treatment of osteoporosis.

Osteoporosis is reported to have up to twice the prevalence in patients with LD, compared to age-matched controls.<sup>2</sup> It is unclear whether cirrhosis and cholestasis are independent risk factors for osteoporosis, as studies have not been adequately powered to study this relationship.<sup>3</sup> However, commonly identified risk factors coexist in this population and include an age > 40, poor nutrition, excess alcohol intake, hypogonadism, and previous corticosteroid use.

Bone mineral density (BMD) as measured by DEXA is currently the best predictor of fracture risk, which increases 2-fold for each standard deviation below the mean of normal controls (T-score). Osteoporosis is defined by a BMD on DEXA of less than 2.5 standard deviations (or T-score of -2.5) below normal peak bone mass. Independent of BMD, an additional risk for fracture is a previous history of vertebral fracture which further increases vertebral fracture risk by 10-fold and subsequent hip fracture risk increases 2.3-fold.<sup>4</sup> Often, underlying vertebral fractures present without symptoms and are not recognized in many patients.

Prevalence of osteoporosis depends on the severity of underlying LD. Cirrhosis increases fracture risk 2-fold with a reported prevalence of 12-55% depending on the population examined. In addition, risk of osteoporosis increases with severity of LD, with Child's C patients having a lower BMD than Child's A.<sup>5</sup> In PSC, prevalence of osteoporosis varies from 8-32% and risk of fracture has been linked to advanced age, duration of coexisting inflammatory bowel disease, and more advanced biliary disease. Ursodiol use has not been shown to improve BMD. In PBC, recent studies have shown a 4-fold risk of osteoporosis and a 2-fold increased fracture risk. Patients with end-stage PBC who undergo transplantation have an even higher prevalence of osteoporosis and fracture risk.<sup>3</sup>

Understanding of changes in BMD post-transplant, both early and late (after 2 years), is important to prevent vertebral and hip fractures. Following liver transplant, the BMD falls for the first few

months, then rises again to pre-transplant levels within 2 years. As a result, fracture rate is high within the first 2 years with reported rates of 15-27%. Pre-transplant vertebral fracture is more predictive of post-transplant fracture than BMD but a reduced BMD post-transplant correlates with post-transplant fracture risk. Cumulative steroid exposure in the first few months after transplant increases the fracture risk. Over the last decade, the trend towards reducing prolonged steroid use post-transplant has led to fewer fractures.<sup>6</sup> The role of calcineurin inhibitors (ie, tacrolimus or cyclosporine) and fracture risk remains controversial.

There are no published guidelines regarding screening for BMD with DEXA in patients with chronic LD. This is due to the difficulty of predicting an individual's risk for osteoporosis.<sup>3</sup> There is a clear consensus that patients who have had previous osteoporotic fractures, those taking prolonged corticosteroids (>5mg daily for > 3 months), patients with cholestatic LD and elevated bilirubin, and all patients being assessed for liver transplantation be screened. At Columbia, we perform DEXA scans every 1-2 years on the above patient populations, post-menopausal women, and all patients post-transplant.

The treatment is based on trials of postmenopausal osteoporosis which revealed reduced fracture risks. This has not definitively been translated into LD patients. All patients need to be encouraged to perform weight-bearing exercises and avoid alcohol and tobacco use. In patients with osteopenia, we start supplementation with calcium 1000 mg and with vitamin D 800 IU daily. Patients with osteoporosis should be given a bisphosphonate (such as alendronate or risidronate). In cholestatic LD and post-transplant, studies have shown a benefit with bisphosphonates by increasing BMD. However, studies have been too small to show a benefit in reducing fracture risk. After starting therapy, DEXA should be repeated annually to assess response. Patients not responding to therapy may benefit from referral to an endocrinologist. ■

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Maura Hagan, LMSW

## The Importance of a Caregiver and a Community Physician for the Success of a Liver Transplant

“No man is an island.” – John Donne



Studies have shown that the support from a caregiver is one of the most important factors in predicting the success of a transplant. Patients with a caregiver are more likely to be medically compliant, thus reducing the risk of medication errors, complications, and re-hospitalizations. Adherence to the medical regimen both before and after transplantation is of critical importance and is more easily accomplished when the patient has a person or persons dedicated to taking care of them.

### The Caregiver Role Pre-Transplant

Pre-transplant, helping the patient means accompanying them to the psychosocial evaluation and various other medical appointments. A caregiver will also need to accompany the patient to the educational workshops. Should the patient's condition deteriorate pre-transplant, a caregiver may need to increase their level of support by helping the patient manage medications, assisting them in getting to their appointments, and helping with activities of daily living and household chores.

### The Caregiver Role Post-Transplant

Post-transplant, the caregiver continues to play a vital role in the care of the patient. Initially after transplant, the patient will need emotional support in the hospital as they move from the ICU to the step-down unit to the floor bed. The caregiver can be instrumental in helping smooth these transi-

tions and in being the patient's voice during this time, allowing the patient to focus on his or her recovery.

### The Caregiver Role at Home

A caregiver familiar with transplant and medications will need to be at home with the patient for a minimum of three weeks. While the patient is still recovering in the hospital, the caregiver will need to learn the patient's complicated medication regimen, which normally includes 10-14 new medications. The patient will not be allowed to leave the hospital before an adequate care plan has been established at home and the caregiver feels confident with managing the medication regimen. By the time the patient is home, the caregiver must be familiar with the various side effects of the medications and monitor these in the home environment.

The caregiver must assist with additional tasks the liver transplant patient cannot accomplish on their own. The caregiver will need to monitor the surgical incision for signs and symptoms of infection. The caregiver will need to monitor vital signs and may need to learn to check the patient's blood sugar, administer insulin, administer IV antibiotics, or learn wound care, including dressing changes or how to manage a wound vac in the home environment. The patient will need transportation and will need to be accompanied to and from their post-transplant follow-up appointments.

### The Role of the Primary Care Physician

A primary care physician is an integral part of the caregiving team, and the importance of a patient establishing a relationship with one cannot be overemphasized. Medical decisions are commonly made by the transplant team for the specific purpose of achieving the best results for the patient. The transplant team, however, cannot replace a primary care physician. If a transplant candidate does not have a primary care physician, it is best to put this relationship in place during the transplant evaluation process.

While it is understandable that liver recipients develop an emotional connection with those who have cared for them through their transplant, it is unrealistic not to have a transition to medical decisions being made by the primary care physician regarding common ailments, such as colds, gout, stomach upsets, skin problems, etc. A skilled primary care physician will be able to make the determination if a particular problem falls under the transplant umbrella and will not hesitate to inform the patient/caregiver that they should contact the transplant team.

In summary, a successful caregiving team should include a dedicated caregiver and a primary care physician. ■

### Minimally Invasive Live Donor Hepatectomy ~ Continued from Page 1

life more quickly. In most cases, donors have been able to return to work in a matter of weeks rather than months.

The new surgical advance represents the latest chapter in a history of innovations in living organ donation by New York-Presbyterian/Columbia surgeons. Dr. Jean Emond, Chief of Transplantation at NYPH/Columbia, was a key member of the team that performed the first pediatric living donor liver transplantation in North America in 1989 while at the University of Chicago Medical Center. Dr. Lloyd Ratner, Director of Renal and Pancreatic Transplantation at NYPH/Columbia, performed the nation's first adult-to-adult laparoscopic living donor kidney transplant in 1995 while at Johns Hopkins

Medical Center. Today more than half of kidney transplants involve a living donor, and nearly all of these donor kidneys are retrieved laparoscopically.

Laparoscopic living donor liver retrieval for pediatric transplant was developed by Dr. Daniel Cherqui, Professor of Surgery at the Hospital Henri Mondor/University of Paris XII, who reported the first case in 2002. Dr. Cherqui trained under Dr. Emond in the late 1980s. With a reputation as one of the most experienced laparoscopic liver surgeons in the world, Dr. Cherqui has been a valuable resource to our center in making laparoscopic living donation available in New York. ■

## Announcing New CLDT Faculty and Staff



Megan Sykes, MD



Sonja Olsen, MD



Cynthia Sterling-Fox,  
BSN, MSN, FNP



Judith Gang, BSN, MS, FNP



Teresa Lukose, PharmD

### Megan Sykes, MD

Professor of Medicine and Microbiology & Immunology and Surgical Sciences (in Surgery), Columbia University College of Physicians and Surgeons

Director, Columbia Center for Translational Immunology, Columbia University College of Physicians and Surgeons

Director of Research, Transplant Initiative, NewYork-Presbyterian Hospital

Megan Sykes, MD, joined Columbia University in April, 2010. She comes to us from Massachusetts General Hospital/Harvard Medical School, where she was the Harold and Ellen Danser Professor of Surgery and Professor of Medicine (Immunology) and Associate Director of the Transplantation Biology Research Center.

Dr. Sykes' research career, during which she has published 365 papers and book chapters, has been in the areas of hematopoietic cell transplantation, achievement of graft-versus-leukemia effects without GVHD, organ allograft tolerance induction, and xenotransplantation.

Her current research focuses on utilizing bone marrow transplantation as immunotherapy to achieve graft-versus-tumor effects while avoiding graft-versus-host disease, the common complication of such transplants. Her laboratory studies in this area have led to novel approaches that have been evaluated in clinical trials. Another major area of her current research focuses on utilization of bone marrow transplantation for the induction of transplantation tolerance, both to organs from

the same species (allografts) and from other species (xenografts). This work has resulted in the first successful trials of intentional allograft tolerance induction in humans. At Harvard, Dr. Sykes' laboratory worked toward the development of clinically feasible, non-toxic methods of re-educating the T cell, B cell and NK cell components of the immune system to accept allografts and xenografts without requiring long-term immunosuppressive therapy. Her work has also extended into the area of xenogeneic thymic transplantation as an approach to tolerance induction, and into the mechanisms by which non-myeloablative induction of mixed chimerism reverses the autoimmunity of type 1 diabetes.

She is the Immediate Past President of the International Xenotransplantation Association and is Vice President of The Transplantation Society. She has recently been elected to membership in the Institute of Medicine. ■

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### Sonja Olsen, MD

Assistant Professor of Medicine

Born in Brooklyn, Dr. Olsen attended Stuyvesant High School in New York City and graduated from Williams College in 1997 with a BA in biology and economics. A graduate of Dartmouth Medical School, she returned to New York for her residency and completed her internal medicine training at NewYork-Presbyterian Hospital/Weill Cornell Medical Center (NYPH/Weill Cornell). Her interest in hepatology led her to NewYork-Presbyterian Hospital/Columbia

University Medical Center (NYPH/Columbia) for a gastroenterology and hepatology fellowship. Dr. Olsen then returned to NYPH/Weill Cornell where she was named Chief Medical Resident, and was charged with training NYPH/Weill Cornell medical housestaff and overseeing the residency program. During 2008-2009, Dr. Olsen was awarded an American Association for the Study of Liver Diseases (AASLD) training grant based on her fellowship work in hepatitis B and joined the team at the CLDT NYPH/Columbia campus to study transplant hepatology for one year. "I feel very fortunate to be part of such a wonderful team and enjoy both campuses with my outpatient office at Cornell and my time at Columbia on the inpatient service," she says. "I am delighted to be caring for patients in partnership with dedicated and skilled clinicians Leah Bucu, NP, and James Spellman, NP, at the Weill Cornell campus of the CLDT." Dr. Olsen's clinical specialties are liver cancer, non-alcoholic fatty liver disease, and viral hepatitis. ■

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### Cynthia Sterling-Fox, BSN, MSN, FNP

Clinical Coordinator

Cynthia completed her graduate training at the Columbia University School of Nursing, where she earned an MSN (cum laude) as a family nurse practitioner. She holds a BSN degree from SUNY Health Science Center (Downstate Medical Center).

Early in her career, Cynthia worked as a registered nurse in the medical surgical

Continued on page 6

## Announcing New CLDT Faculty and Staff ~ Continued from Page 7

unit and emergency room of a major New York hospital for five years, and as an emergency room NP for six years. She discovered her true career passion in transplantation nursing in 2002, when she joined the NYPH/Columbia Center for Advanced Cardiac Care, caring for pre- and post-cardiac transplant patients. At the CLDT, Cynthia works collaboratively with hepatologist Dr. Eva Sotil, to provide care for pre and post liver transplant patients.

Cynthia's other interests include clinical research and teaching. She has provided research support to many studies at Columbia University Medical Center, including in the areas of immunosuppression and molecular expression testing in transplant patients. She is an adjunct assistant professor of nursing at CUNY Medgar Evers College. ■

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### **Judith Gang, BSN, MS, FNP**

Transplant Coordinator

Judy Gang, who has a special interest in hepatocellular carcinoma research and treatment, comes to the CLDT from Mount Sinai Medical Center's Racanati/ Miller Transplantation Institute, where she worked from 2000 through 2009. At Mt. Sinai, Judy

worked as a floor nurse, a liver transplant coordinator, and a hepatobiliary/HCC coordinator. She also served as the primary liaison between Mt. Sinai's hepatobiliary/surgical oncology and liver transplant services, where her objective was to streamline the referral and evaluation processes of patients with HCC and expedite their transplant listing.

A graduate of Yeshiva University, in New York City, where she received a BA in Jewish History, Judy completed her nursing training in 2007 at Pace University's Lienhard School of Nursing. At the CLDT, Judy works collaboratively with NP Ariana Rose and Dr. Scott Fink to provide care for pre- and post-transplant liver transplant patients. ■

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### **Theresa Lukose, PharmD**

Director, Clinical Transplant Research

Theresa joined the CLDT and the New York-Presbyterian Hospital Transplant Initiative in September, 2009. She oversees the ongoing clinical trial activities of the center and identifies and applies for federal and foundation grants. She also oversees transplant protocol development and execution, and manages the CLDT research staff. One of Theresa's main initiatives is facilitating a centralized support struc-

ture for the multidisciplinary transplant research center of the NYPH solid organ transplant programs.

Before coming to the CLDT, Theresa spent five years as a clinical pharmacy manager specializing in lung and heart transplantation, working with the Columbia and Cornell transplant teams at New York-Presbyterian Hospital. During that time, she guided the inpatient transplant team in medication management of the post-transplant patient, instructed transplant recipients about medications, developed policies and protocols related to transplant, and participated in transplant related research. She also served as Director of NYP's PGY2 Solid Organ Transplantation Pharmacy Residency and taught pharmacy residents and students.

Theresa graduated from Albany College of Pharmacy in 2002 and completed a PGY-I pharmacy residency at NYPH/Columbia in 2003 and a PGY-2 Specialty Residency in Solid Organ Transplantation at Duke University Hospital in Durham, North Carolina in 2004. It was during her liver transplant rotation as a PGY-I Resident at Columbia that Theresa discovered her interest and passion for transplant pharmacy. ■

## **New York-Presbyterian**

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