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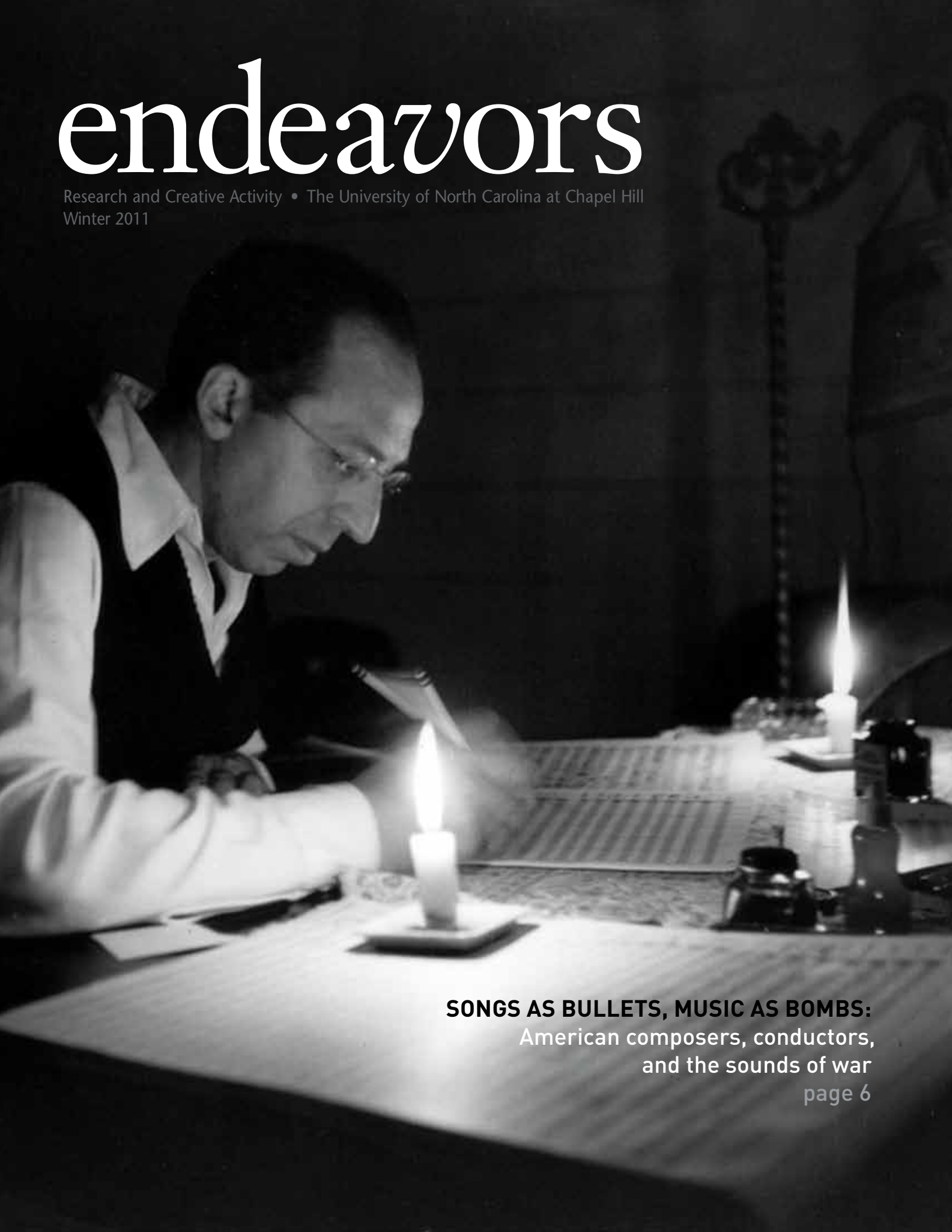

SONGS AS BULLETS, MUSIC AS BOMBS:
American composers, conductors, and the sounds of war
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On the cover: Aaron Copland, American composer, 1946. His *Lincoln Portrait* was commissioned as part of the war effort. Photo courtesy of the Library of Congress.

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THE UNIVERSITY of NORTH CAROLINA at CHAPEL HILL

In the thick of World War II, one low, sad, and sweet sound rose out of Belgrade and wove itself around and through the drone of bombers, the bark of infantry fire, and the thudding of shells. Eventually it grew strong enough to be heard all over Europe and North Africa, and to bring together soldiers on both sides of the conflict, at least in spirit: the sound was a pop song called "Lili Marlene."

Written in 1918 by a schoolteacher who'd been conscripted into the Imperial German Army, the song tells the story of a German soldier's love for a young woman whom he meets every night at a lamppost outside his barracks. A German cabaret singer named Lale Andersen recorded the song in 1939. It sold about seven hundred copies and was promptly forgotten.

When the Germans occupied Belgrade in 1941, they set up a radio station to broadcast to their troops throughout Europe and the Mediterranean. A lieutenant who worked at the station picked up a stack of records at a secondhand shop in Vienna. In the stack was "Lili Marlene." Because Radio Belgrade had only a few records, it played the song often, and "Lili Marlene" became wildly popular among Axis troops and in German homes and cafés. Radio Belgrade signed off with the song, playing it every night at five minutes till ten. Hitler liked it. But Nazi propaganda minister Joseph Goebbels could not abide it. "A dance of death lingers between its bars," he wrote. In their book *Lili Marlene*, Liel Leibovitz and Matthew Miller write that Goebbels, "so accustomed to stage-managing every Nazi invasion with a soundtrack full of appropriate pomp . . . was apoplectic at this sentimental love song that was lulling Germany's vaunted Aryan warriors to sleep each night on the battlefields." It didn't help that Lale Andersen had Jewish friends. Goebbels banned the song from German radio and tried to get Radio Belgrade to stop playing it, too. But Axis soldiers flooded the station with letters supporting the song, and Goebbels eventually relented.

The song quickly caught the ears of Allied soldiers, who could also pick up Radio Belgrade. Fitzroy MacLean, fighting for the British, wrote: "Husky, sensuous, nostalgic, sugar-sweet, her voice seemed to reach out to you, as she lingered over the catchy tune, the sickly sentimental words. Belgrade . . . the continent of Europe seemed a long way away. I wondered when I would see it again." Allied fighter pilots painted "Lili Marlene" on the noses of their planes. Eventually, it became one of the world's most recorded songs. Today, web pages catalog hundreds of versions of "Lili Marlene," and iTunes carries Andersen's original—which, after a few listens, is foxholed in my brain. But in the 1940s, write Leibovitz and Miller, "in a war remembered mostly for its stark divides and brutal, dehumanizing crimes, this song emerged from the ashes as a tiny reminder of unity, hope, and brotherhood." The sounds of war weren't just explosive, percussive, and traumatic. Some of them, whether classical or pop, could help heal a world split apart.

—Jason Smith

LILI MARLENE RECORD IMAGE: MEDIATUS

contents

Detail, "Double Caps II," a color photogram by Mike Sonnichsen. Story on page 30.

FERTILITY

Don't trust the stick

by Stephanie Soucheray-Grell

Meet the crystal balls of modern medicine: at-home fertility tests. Women contemplating pregnancy can use these pee-on-a-stick tests to take instant snapshots of their reproductive ability. The tests, which work like at-home pregnancy tests, measure urine levels of FSH, a hormone related to ovarian aging and function.

But a new study from UNC shows that over-the-counter fertility predictors may be inaccurate. Moreover, the tests may not be measuring the strongest predictor of female fertility.

Anne Steiner studies the fertility of women in their thirties and forties. She recreated the home fertility tests while studying one hundred women and their chances of getting pregnant in any given cycle. High urine FSH levels are thought to be a sign of infertility, but Steiner found that women with higher FSH levels were no less likely to get pregnant than women with normal levels.

Steiner says another hormone, AMH, may be a better measure of a woman's fertility. Unfortunately, AMH can only be measured through blood, and a blood test has not yet been approved for clinical use.

In Steiner's study, women with low AMH levels were 60 percent less likely to get pregnant in any given cycle. "AMH comes directly from the ovary, whereas FSH comes from the pituitary gland," Steiner says. "AMH is the first to decline, which means AMH may be a more sensitive marker in egg decline."

But the best fertility predictor a woman has can't be found in the drugstore. "I can't support or discredit fertility tests," Steiner says. "But I can say that age is the best predictor we have. We know that fertility begins to decline in the early thirties, then more rapidly by the time a woman is in her forties. But what we don't have yet is a test to give thirty-eight-year-old women that says, 'Yes, you're still fertile.'"

Steiner hopes her research will lead to more choices for women. "We want to find fertility markers," she says. "If a woman can have a better measure, we can ultimately allow women, and couples, more reproductive control."

Anne Steiner is an assistant professor of obstetrics and gynecology in the School of Medicine. Steiner's study tracks hundreds of women between the ages of thirty and forty-five from the time they begin trying to get pregnant until they achieve pregnancy.

FERTILITY STICK: LIBBY CHAPMAN

MUSCULAR DYSTROPHY

Delivery refused

by Stephanie Soucheray-Grell

Sometimes the "Eureka!" moment for a scientist doesn't happen during the course of a carefully planned clinical trial. Geneticist Jude Samulski found that the moment of discovery can happen before the trial even begins.

In 2006, Samulski and collaborators across the country, including UNC's Xiao Xiao, conducted a new gene therapy trial on children with Duchenne muscular dystrophy. The idea was to deliver a healthy dystrophin "minigene" to patients. In a person with muscular dystrophy, a gene on the X chromosome contains faulty instructions for encoding the protein dystrophin, which keeps healthy muscles from deteriorating. Scientists have believed that people with muscular dystrophy couldn't produce any dystrophin. Giving patients new, functional genes, the scientists thought, could treat the disease.

But when the researchers delivered new minigenes to study participants, the patients' bodies showed an immune response to the protein—their bodies had encountered it before. "When we sent our FedEx truck of the vector and gene package to the patients, they sent us a message back that says, 'Already seen it,'" Samulski says.

This may mean that people with muscular dystrophy have an inherent tendency to destroy their bodies' natural dystrophin. In the study, researchers were surprised to find that people with the disease had different trace amounts of dystrophin in their muscle fibers. Samulski says that before the study, scientists knew that people with muscular dystrophy had these muscle fibers, but no one knew what the fibers were doing.

"The trial told us that the patient's fibers may be inadvertently vaccinating the patient against the therapeutic protein we were trying to deliver," Samulski says.

Samulski likened the response seen in patients to someone rejecting an organ after a transplant. He also says immunosuppression may be used in future muscular dystrophy trials.

For now, this study raises more questions about the nature of muscular dystrophy and the usefulness of gene therapy in treating the debilitating and deadly disease. "We don't know exactly what this means for future therapies," Samulski says. "But the discovery did raise this question: will our therapies be therapeutic?"

Stephanie Soucheray-Grell is a master's student in medical and science journalism.

Jude Samulski is the director of the Gene Therapy Center at UNC. Xiao Xiao works in the Eshelman School of Pharmacy. These findings were published in October 2010 in the New England Journal of Medicine.

CAROLINA FINDINGS

Women with anorexia nervosa are much more likely to have unplanned pregnancies and induced abortions than women who do not have an eating disorder. Women aged sixty-seven and older with normal bone-mineral density scores may not need screening again for ten years. Radio frequency tags may help prevent surgical sponges from being left in patients after operations. A team approach to cancer treatment

People whose bodies react strongly to alcohol are less likely to become alcoholics. A newly discovered gene may explain why.

ROBERT BYRON

GENES AND ALCOHOLISM

The lightweight trait

by Mark Derewicz

In Australia they call it the tipsy gene, and people who have it feel inebriated after just two or three drinks. UNC geneticist Kirk Wilhelmsen discovered it. "I did a couple interviews with Australian radio stations, and they talk about wanting to cure these people," he says. "They're concerned because they think not being able to drink a lot is a big social disability."

They've got it backwards, Wilhelmsen says. We should *want* this genetic variant. It could save us some heartache down the road. And some brain cells.

Thirty years ago, when Wilhelmsen was an undergraduate at UC-San Diego, psychiatrist Mark Schuckit recruited college students and their siblings to determine how having a few drinks affects people differently. Schuckit kept track of the students, their siblings, and their parents for more than twenty years. He found that people who felt more inebriated after a few drinks were less likely to become alcoholics later in life. "There are lots of reasons why people drink, and alcoholism is a complicated disease," Wilhelmsen says. "But not having the trait was a strong predictor of alcoholism—stronger than family history."

By then Wilhelmsen had his own lab. He teamed with Schuckit to see if alcohol sensitivity had a genetic basis. Using blood samples from Schuckit's study participants and genetic analyses called linkage and association, Wilhelmsen's lab pinpointed a gene called CYP2E1 that caused at least 10 percent of the research subjects to be more sensitive to alcohol's effects.

For years scientists have known that CYP2E1 produces an enzyme that metabolizes alcohol, but they had never studied the gene in relation to alcohol sensitivity. Turns out, people who have the tipsy gene express more of the metabolizing enzyme. Wilhelmsen says this is where the science gets counterintuitive.

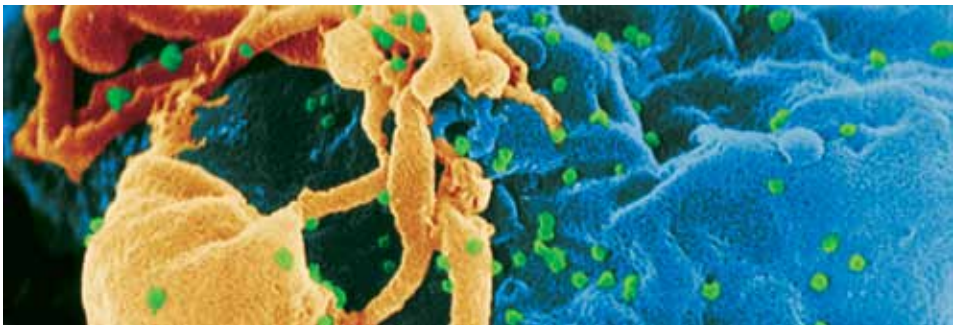
You might think that having more of the enzyme would help people metabolize alcohol faster, which would keep them from getting tipsy. But that's not how the CYP2E1 enzyme works. First, it's in the brain. And unlike liver enzymes, which metabolize most of the alcohol, this enzyme uses oxygen to break down alcohol. "That's oxidation," Wilhelmsen says. "The enzyme just ruins the alcohol instead of turning it into energy." This kind of metabolism releases electrons, which can attach to other molecules to form free radicals and attack sensitive structures such as brain cells.

"I think when you drink you're feeling that buzz because of free radicals," Wilhelmsen says. That flies in the face of conventional thinking. For decades, scientists have thought that alcohol disrupts how neurotransmitters do their jobs. "That *does* happen," Wilhelmsen says. But he says it's tough to prove that disrupting neurotransmitters—the molecules that communicate between neurons—causes drunkenness. Wilhelmsen thinks it's just as likely that inebriation is a result of metabolic enzymes interacting with alcohol. The fact that the CYP2E1 genetic variant causes people to feel buzzed quickly, *and* that variants of another gene that also metabolizes alcohol protect individuals from developing alcoholism, supports his theory.

At the very least Wilhelmsen's finding suggests that the chemistry of drunkenness and the causes of alcoholism are more complex than previously thought. The tipsy gene is real. And it's not such a bad thing.

Kirk Wilhelmsen is a distinguished professor of genetics in the School of Medicine. His team, which includes UNC graduate student Amy Webb, received funding from the National Institute on Alcohol Abuse and Alcoholism, the CompassPoint Addiction Foundation, and the Bowles Center for Alcohol Studies at UNC.

benefits patients by providing more precise diagnosis and staging as well as more comprehensive treatment recommendations. Routinely giving an antinausea drug to children with gastroenteritis-induced vomiting would prevent thousands of hospitalizations and save millions of dollars each year. Patients with x-ray evidence of knee osteoarthritis who inherit a specific pattern of genetic variations are almost twice as likely as other patients to progress to severe osteoarthritis.



HIV-1 budding from a cultured lymphocyte. Image: Centers for Disease Control and Prevention

INFECTIOUS DISEASE

HIV in semen differs from HIV in blood

by Noor White

A team of UNC researchers has discovered that populations of HIV strains differ between blood, which is the focus of most HIV research, and semen, the main point of HIV transmission.

HIV is adept at evading the immune system because it mutates into many strains within one person's body. By the time the immune system creates cells to target one strain, another strain is poised to multiply and take the target's place. It's a never-ending competition between the immune system and virus evolution.

The team, led by virologist Ron Swanstrom, looked at sixteen HIV-positive men and found three different ways that populations of the virus formed in their blood and semen.

In the first scenario, the virus was identical in the bloodstream—where HIV goes first after it enters the body—and in the seminal tract.

In the second scenario, say that the person's blood contained strains A, B, and C in roughly equal amounts. In this scenario, the person's semen also contained strains A, B, and C, but C was much more abundant. Swanstrom believes the virus underwent rapid replication but did not have enough time to evolve after it entered the seminal tract.

In the third scenario, the person's semen contained strains D, E, and F, and not the A, B, and C strains found in the blood. The strains were different because the virus had replicated for a long time (months to years) after it entered the seminal tract, and it evolved as a separate population from the virus in the bloodstream.

To explain why HIV may be able to replicate in the seminal tract, Swanstrom's team looked at immune signaling molecules—cytokines and chemokines—in semen. The team found that some of these molecules are at higher concentrations in HIV-infected men. HIV lives in immune cells, so the more cells that are recruited and activated by signaling molecules, the better the environment for HIV to replicate and evolve. The greater concentration of the signaling molecules in semen than in blood could explain how the target immune cells are attracted to the seminal tract, where they can support the different viral populations.

"Understanding how HIV establishes separate populations in the body will help us understand disease progression and pathogenesis," Swanstrom says. "In this case, independent replication of the virus in the seminal tract can change the nature of the virus that is transmitted."

Next, Swanstrom's lab will focus on finding the properties of HIV that cause the differences in viral populations in blood and semen.

Noor White is a research technician in biology at UNC.

Ron Swanstrom is a professor of biochemistry and biophysics in the School of Medicine. His paper was published in PLoS Pathogens *in August 2010. Participants in this study were from the Kamuzu Central Hospital, part of the UNC Project in Malawi, and from the CHAVI 001 Cohort in North Carolina.*

EPIDEMIOLOGY

Anemia, dialysis, and risk of death

by Margarite Nathe

Anemia, which happens when the body doesn't produce enough red blood cells to provide oxygen to all its tissues and organs, can be miserable. Lightheadedness, fatigue, fainting, heart pains and murmurs—the symptoms combine to devastate quality of life for many and can lead to hospitalization or death for dialysis patients (the condition often goes hand in hand with kidney disease).

To prevent anemia in dialysis patients, doctors routinely use iron and ESAs (erythropoiesis-stimulating agents), which drive the body to make more red blood cells and reduce the number of blood transfusions a patient will need. But ESAs don't have the same results for everyone—they can be risky for some patients. Some studies have shown that the treatment can increase a patient's risk of heart attack, heart failure, stroke, and blood clots. Now a study led by M. Alan Brookhart has shown that while ESAs may lower the risk of death for dialysis patients with severe anemia, the drugs may *increase* the risk for patients with milder anemia.

JASON SMITH

Brookhart and his colleagues studied nearly 270,000 patients to determine how dialysis centers' use of ESAs and iron affected mortality risk among patients. They found that dialysis units that treated severe anemia more aggressively with ESAs and intravenous iron had a one-year mortality rate that was 5 percent lower than in units that treated more conservatively, Brookhart says. But the same aggressive treatment for milder anemia brought a 10 percent increase in the rate of mortality.

Brookhart is an associate professor of epidemiology in the UNC Gillings School of Global Public Health. The study was published in the March 3, 2010, issue of the Journal of the American Medical Association.

Spanking has declined in the United States since 1975, but nearly 80 percent of U.S. preschool children are still spanked. Thirty-one radiologists who reviewed the CT scans used in a medical malpractice lawsuit did not agree with the conclusions reached by four paid medical expert witnesses in the case. A normally benign protein in the human body, when paired with nanoparticles, appears to be able to zero in on and kill certain cancer cells without the use of chemotherapy drugs. Tai chi

VIEWPOINT

Believe Nothing They Tell You

by Noor White

ELKE DENNIS

They, the dispensers of well-meaning knowledge. Attendants of my college graduation party: family, friends, and neighbors. People I've come to associate with Home and the places where I grew up. The people that, by virtue of our history together, I trust most.

At said graduation party, I announced that I had made a decision on my first real-world job. I would move down to Chapel Hill, North Carolina, to begin work as a research technician and start a new life in a place where I had not one friend, relative, or connection. Yes, I was afraid, but I believed it was the best decision for me and my career path, and I wanted to go.

Chapel Hill is one of those places that everyone has a connection to and an opinion about. Everyone besides me and my immediate family. The rest of my particular They, on the other hand, all loved it. Being the experts that they were, they spared no time in defining what I should expect. First, the fact that I was moving to a completely new area completely alone was declared unimportant. Chapel Hill is, after all, the South. People are friendly, warm, welcoming. I would have no problem making friends, finding a niche, joining the community.

My second mistake was listening to Them. While my personal life was supposed to be rife with good times and noodle salad, They had told me not to expect too much of my professional life. Research technicians are supposed to be the busboys of academia, the ones given the tedious, messy maintenance work that no self-respecting graduate student would do. I would be required to work long, hard hours, and not to have much contact with my busy, important boss. I didn't think I would take to the fruit flies either—nasty, buzzy things.

Almost a year later, I still know few people other than my coworkers, who groan when I listen to country music on the lab speakers. The misconception that upon my arrival to Chapel Hill I would be immersed in a pool of southern accents is largely my fault. First, I should have expected that I wouldn't find many of the locals in academia. An academic career requires travel to wherever vacancies lie. Few faculty and graduate positions are filled by locals; it's an effort against what my boss calls "intellectual inbreeding" that those pursuing academic paths get their training from many different leaders in the field, and thus in many different places. Other than the fact that it hit ninety degrees in April, and that the air smells of honeysuckle and wisteria as I walk back to my apartment in the late afternoon, I may as well be in any other state, in any other part of the country.

My second mistake was listening to Them. While my personal life was supposed to be rife with good times and noodle salad, They had told me not to expect too much of my professional life. Research technicians are supposed to be the busboys of academia, the ones given the tedious, messy maintenance work that no self-respecting graduate student would do. In return for application-enhancing experience as well as invaluable insight into the fields I may want to pursue my next degree in, I would be required to work long, hard hours, and not to have much contact with my busy, important boss. Personally, I didn't think I would take to the fruit flies either—nasty, buzzy things.

Almost a year later, I still look forward to going in to work every morning. My boss, whom I speak with every day, promotes my development as a scientist. I work independently and make decisions on how to carry out my own projects. I am challenged, engaged, and appreciated. I work in a setting where the thrill of the quest for knowledge runs in the drinking water. And the flies? If my brain were capable, I would give each and every one a name (there are thousands).

So apparently you really can't trust anyone nowadays, especially those who want the best for you. Luckily for my sanity and the relationships I have with Them, I made the decision before that party to pack up and head south. And the only things that I could really have expected to find, I have—independence, a fresh start, and a new horizon.

Noor White is a research technician in biology at UNC. A version of this essay appeared on The Bucknell Afterword, *a blog for alumni of the creative writing program at Bucknell University.*

relieves arthritis pain and improves reach, balance, and well-being in patients being treated for fibromyalgia, rheumatoid arthritis, and osteoarthritis. There are at least four molecular subtypes of squamous-cell lung cancer. A person's height is determined by a combination of a great number of genes rather than by a simple "tall" gene or "short" gene. People who have melanomas on the scalp or neck die at almost twice the rate of people who have melanomas on other areas of the body.


USA
SONGS AS BULLETS,
MUSIC AS BOMBS

THEY HELPED WIN WORLD WAR II,

YET THEY'VE BEEN IGNORED BY MOST MUSIC SCHOLARS:

AMERICAN COMPOSERS, CONDUCTORS, AND THE SOUNDS OF WAR

BY MARK DEREWICZ

"LILY AND I AUTOGRAPHED A FIVE-HUNDRED-POUND BOMB WITH AN INSCRIPTION, 'GREETINGS TO JAPAN.' I HOPE IT BLASTED THE HELL OUT OF THE JAPS."

CONDUCTOR Andre Kostelanetz wrote that in 1945 when he and his wife, soprano star Lily Pons, toured combat zones with their orchestra, often competing with noise from bombers at nearby airfields. In Germany, days after American troops stormed toward Berlin, Kostelanetz wrote to a friend that antiaircraft canons provided "a reasonable replica of the bass drums at the most unexpected moments during the concert."

Left: This illustration is modeled on a World-War-II-era poster titled *More Production* by an artist named Zudor. The original, which aimed to rally enthusiasm and industry for the war effort at home, was printed by the Government Printing Office for the War Production Board. It is now held in the National Archives. Illustration by Jason Smith. **Above:** Conductor Andre Kostelanetz and soprano opera star Lily Pons, who were married, performed for Allied soldiers all over the world. Image courtesy of Lucy Kostelanetz.

Kostelanetz was one of many conductors and musicians who toured the front lines during World War II. But tales of their adventures and their music have gone largely untold. Musicologist Annegret Fauser says, "Never before had classical music received so much financial and ideological support from the U.S. government."

The government believed in the power of music, she says. So did musicians. And so did soldiers. Music as therapy. Music as entertainment. For morale. As a weapon. For Fauser, conductor Serge Koussevitzky said it best in 1942: "We, as musicians, are soldiers, too, fighting for the ever-growing spiritual need of the world. If music is our life, we give it joyfully to serve the cause of freedom."

FAUSER WAS BORN IN GERMANY almost two decades after World War II. "I'm a very typical German in that I don't want to be German," she says. "My generation is still part of that postwar world where the idea of national identity is hugely problematic." The first chance she got, in 1987, Fauser left Germany to study in Paris, which is where she discovered letters between French composer Nadia Boulanger and her star pupil, the American composer Aaron Copland. Years later, after Fauser arrived at UNC in 2001, she started researching Copland's era to serve as context for a book about those letters.

"I kept coming across really interesting texts about American music in the 1920s, the 1930s, 1950s, the 1960s," she says. "There was this big hole—the 1940s. I thought that was strange; you could fill a whole library with books on music from Nazi Germany. So I thought, 'This is not possible; I'm just a bad scholar and haven't found anything yet.'"

She knew that some of Copland's most iconic compositions were commissioned during the war. She found plenty on Copland but hardly anything about classical music during the war. Fauser, astonished, consulted with colleagues. They said, "All that stuff is just propaganda music."

Fauser says, "I think some scholars didn't want to embarrass themselves, because an awful lot of that music was blatant Americana. They'd say, 'John Cage and others came afterwards, so we don't have to deal with Morton Gould's *American Salute* and stuff like that.' But I'm not American. To me it's an interesting cultural phenomenon."

For five years Fauser spent summers, holidays, and long weekends rummaging through archives full of wartime corre-

July 13, 1944, Saipan, Marianas: Soldiers of the 2nd Marine Division listen to music on a Japanese phonograph. During World War II American soldiers craved music—live or recorded. To musicologist Annegret Fauser's surprise, many preferred classical music. Photo: Corbis

THE FEDERAL GOVERNMENT COMMISSIONED CLASSICAL MUSIC IN PART TO COMBAT THE NAZI PROPAGANDA MACHINE. "THE NAZIS MADE A HUGE POINT ABOUT THE BARBARIANS ACROSS THE POND WHO LISTENED TO JAZZ AND HAD NO REAL CULTURE," FAUSER SAYS.

spondence, journals, news reports, and declassified documents. She realized that the kind of classical music being made was just part of the story, one she tells in her forthcoming book, *Sounds of War*.

"FOR THE FIRST TIME in their lives composers had commissions coming out of their ears," Fauser says. And the government was a major funding source. There were Copland, Samuel Barber, Elliot Carter, Henry Cowell, and others, many of whom wrote classical music for the propaganda missions of the Office of War Information (OWI). Dissidents from Germany and the Soviet Union worked for the OWI too. Kurt Weill, for instance. He dubbed himself "formerly German" and worked hard for the American war effort, Fauser says, even though the government classified him as an enemy alien. Some musicians were classified the same way, and Fauser says many of them had to work harder than other musicians to prove their loyalty to the United States.

"Marc Blitzstein, whose music I absolutely adore, was a great composer who enlisted in the U.S. Army because he wanted to fight the Germans, especially after they attacked Russia," Fauser says. Blitzstein, after all, was a communist sympathizer. He was attached to the Eighth Airborne in London, where he came up with the idea to write *The Airborne Symphony*, which the U.S. Army Air Forces commissioned for use in propaganda films.

One reason the federal government

CONDUCTOR ANDRE KOSTELANETZ (RIGHT) WANTED SO BADLY TO SERVE THE WAR EFFORT THAT HE AND HIS WIFE, SOPRANO STAR LILY PONS, TOURED AS CLOSE TO THE FRONT LINES AS POSSIBLE. THEY ENTERED GERMANY ON THE HEELS OF AMERICAN TROOPS.

commissioned so much classical music was to combat the Nazi propaganda machine. "The Nazis made a huge point about the barbarians across the pond who listened to jazz and had no real culture," Fauser says. This was a propaganda war about who had cultural superiority.

The U.S. government and musicians considered the United States to be the last bastion of freedom. "They'd say, 'This is where all the great arts have now come to take refuge,'" she says. "'We have the great orchestras, the great operas, the great singers.'" And the government had a point. Many composers and musicians had fled Europe for the States before the war.

But Fauser also found that combating Nazi propaganda wasn't the only reason why the U.S. government poured money into classical music.

July 1944, Italy: Andre Kostelanetz conducts an orchestral performance for Allied troops less than a year after U.S. troops invaded mainland Italy. Image courtesy of Lucy Kostelanetz.

AFTER THE ATTACK on Pearl Harbor on December 7, 1941, the military ballooned in size thanks to massive enlistments and the draft, which brought an influx of men from all walks of life.

"For the first time the government had about 17 percent of the population under its control, mostly in the military," Fauser says. That's ten million people, an armed force that mirrored the diverse American population. She says the military and OWI realized they could survey military men to get a good idea of what average citizens—at least the men—were thinking. In declassified documents, Fauser found that the military was curious about musical tastes. Swing was the most popular form of music—no surprise there. But number two was classical. "I had no idea that would be the case," Fauser says. As she researched further, it made sense. After all, she says, "the Metropolitan Opera broadcast every Saturday afternoon to something like thirty million listeners."

Fauser says that classical music, which was called "good music" in the 1940s, had a deeper appeal than other genres. Swing was for parties, and soldiers loved it. But when they wanted to feel uplifted, when they craved a sense of peace or calm, when they desired spirituality, they turned to classical music.

The army noticed. Fauser says that the first clinical trials of music therapy were held at Walter Reed Army Medical Center during the war. Results showed that music helped recondition soldiers. Not just any music, though. "Good music."

In the National Archives and the Library of Congress, Fauser found the story of Harry Futterman, a professional accountant and amateur musician who started a charity to send libraries of classical albums to military camps stateside and abroad. Well-known conductors, such as Toscanini and Koussevitzky, sponsored the purchase of libraries.

Soldiers, chaplains, and medical officers wrote to Futterman, calling classical music a mainstay of their morale. Captain Revel Lahmer told Koussevitzky, who conducted orchestral performances for radio broadcast: "Living in an army camp looking to a future of uncertainty, we need more than words and promises to bolster our morale. We need expressions of strength, health—tangible expressions of belief and faith in living—to make us fight for a future that will be better than any past." Referring to Koussevitzky's performance of Roy Harris's Fifth Symphony, Lahmer wrote: "Please give us a repeat performance . . . this music expresses [who we are as] Americans. And today more than ever we need the strength that comes through this expression."

At first Futterman decided which albums to send, but he eventually let enlisted soldiers make selections. "To his surprise," Fauser says, "they chose a collection of heavies, starting with Elisabeth Schumann's recording of Bach's 'Bist du bei mir,' Edward Elgar's *Enigma Variations*, Beethoven's Fifth Symphony, and Richard Strauss's *Rosenkavalier* waltzes." Germans all.

Also, Fauser discovered that soldiers loved Richard Wagner, arguably the epitome of German music and Adolf Hitler's preferred composer.

"Soldiers were actually listening to Wagner quite a bit but not in the same way you see in that helicopter scene in *Apocalypse Now* with Wagner's 'Ride of the Valkyries,'" Fauser says. "They used it for recreation and uplift." They often listened to it in groups at their camps, not while taking a hill or shooting down enemy aircraft.

One soldier wrote home: "It doesn't make any difference what composers' music is played. We are not fighting individuals, nor

Harold Spivacke directed the Library of Congress's Music Division and fulfilled a unique role in the war effort, including making sure soldiers received libraries of albums. To his surprise, soldiers clamored for classical music. Photo courtesy of the Library of Congress.

CAPTAIN COPLAND?

Aaron Copland wanted to serve the war effort and considered enlisting, as long as the army could find him an appropriate role. He nearly became a musical advisor—an officer who would be in charge of all things musical for an army unit. Fauser found letters between Copland and Harold Spivacke, the director of the advisor program. After Copland expressed his reluctance to join the army and give up composing, Spivacke responded: "I wish to advise you to change your attitude toward service in the army. I admit that you will not have as much time for composing, but I see no reason why you will have to give it up entirely."

In his letter back, Copland wrote: "You can't imagine how right I hope you are but I should warn you that composing to me means a private room with a piano and some consecutive time for writing. (Unlike Beethoven and Hindermith, I don't work in the fields.) If the Army can provide that, its set-up is even more intricate than I thought."

But Spivacke, who had no say where the army would assign Copland, responded: "I do not want you to get a false impression of the possibilities of composing in the army. Please do not expect the Army to make any special arrangements. Perhaps you had better start learning to compose on something portable like an accordion."

Copland never enlisted. And he was ineligible for the draft. He did, though, participate in the war effort. Conductor Andre Kostelanetz commissioned composers to write musical portraits of famous Americans. Copland penned the most well-known of these, *Lincoln Portrait*. It has held up better than any other piece of wartime propaganda: President Obama used it during his inauguration.

—Mark Derewicz

are we fighting against the products of the culture of our enemies. Beautiful music should be regarded and cherished for what it is—just beautiful music. If it is written by an enemy sympathizer that is unfortunate. But if it is good music it will live and be loved long after our hates have become a thing of the past."

Fauser found that Futterman's work inspired formal listening sessions, sometimes in chapels. Soldiers would sit in rows, as if in a concert hall, and face the music—a hand-cranked phonograph under candlelight. "You could argue that soldiers were so ready for any kind of entertainment that they'd have listened to anything," Fauser says. "But letters home tell another story. The soldiers clamored for more classical music."

In a letter to radio journalist and composer Deems Taylor, an anonymous soldier wrote: "I know that I express the feelings of thousands who perhaps don't have the time to sit down and write to you. We who are busy seven days a week training and fighting for our ultimate victory want and need good music. We need it because it clears our minds and gives us relaxation; we need it because it gives us purpose, courage, and hope in the cause for which we are fighting."

FUTTERMAN WORKED with Harold Spivacke, the director of the Library of Congress's Music Division, to make sure that crates of records found their way onto submarines in the Atlantic and aircraft carriers in the Pacific, into army hospitals in the Philippines and training camps in Alaska.

Spivacke was involved because he was chairman of the Sub-Committee on Music of the Joint Army and Navy Commission on Welfare and Recreation. The chairmanship was not a typical librarian duty, but Spivacke was not a typical librarian.

Before Pearl Harbor, the Library of Congress's Music Division was like any other library department: interested in collections and acquisitions. But after the United States declared war, Fauser says, "the division became the hub of all wartime music endeavors, and Spivacke served as the mediator between the military and the country's musical community."

The U.S. Marine Corps came to Spivacke to borrow two reel-to-reel tape-recording machines to train soldiers to identify enemy planes and artillery. Spivacke gladly lent the machines, Fauser says, and then never saw them again.

Spivacke created the Army Song Leader program and the Army Hit Kit, pamphlets of songs that were mailed to soldiers so they could sing popular songs in groups. The idea, Fauser says, is that the army preferred singing soldiers to idle ones. "The employment of music by the German Army has received considerable publicity as part of its daily routine," wrote Major Howard Bronson, who worked with Spivacke. "Thus if mass singing worked for Germans, maybe it might also benefit the American Army."

Spivacke and Bronson also created a music advisor program that recruited musicians, performers, and composers for army duty. Fauser says that the advisors were in charge of musical activities wherever the army assigned them with the purpose of using music as a psychological weapon to win the war. Composers William Schuman and Blitzstein joined. So did band leaders Glenn Miller, Wayne King, and Joe Jordan.

In the Library of Congress archives, Fauser found a letter in which Copland explains to Schuman why he was reluctant to en-

In India, Andre Kostelanetz and Lily Pons pause for a photo with part of their orchestra. During their India tour, the couple took the Bengal-Assam Railroad through jungle areas to reach troops. Image courtesy of Lucy Kostelanetz.

list: "I hate like hell the idea of giving up composing at least until New York is under attack." (*See "Captain Copland?" at left.*)

Copland never enlisted. In 1943, as men in their forties were being drafted, he was designated 3-A—ineligible for the draft because his mother was dependent on him. But he made his mark with several compositions that supported the Allied cause.

Still, at the onset of war some people were worried that composers would have little place in the war effort or would need to contribute in other ways.

FAUSER FOUND some documents from two musical administrators—Ross Lee Finney at Smith College and Claire Reis at the League of Composers—who sent questionnaires to composers to find out what they thought their function should be during the war. Some responded that they had little to offer. Others, such as Earl Robinson, said that "songs can be bullets." That is, propaganda. Composer Marion Bauer's response was pragmatic: her colleagues should "compose works that would be timely, principally choral numbers" that might be used for concerts throughout the country. During those concerts, the U.S. Department of the Treasury could sell war bonds.

Reis wanted to know composers' individual abilities in case they were drafted. She asked if they could teach an instrument. Most composers, including Elliot Carter, Nathaniel Dett, Ernst Krenek, Richard Rodgers, William Schuman, William Grant Still, Barber, Copland, Cage, and Gould, said they could not. Only William Handy, Werner Janssen, and Robinson said they could. Cowell admitted, "Not too well." Arnold Schoenberg answered, "I guess." And so, Fauser says, Reis's goal of creating a national talent pool was unsuccessful.

ANNEGRET FAUSER: "THE WAR WAS A BOON FOR RANK-AND-FILE MUSICAL PROFESSIONALS WHO SUFFERED THROUGH THE GREAT DEPRESSION."

Composers did what they did best: they wrote music. And they had the support of First Lady Eleanor Roosevelt, who said composers and musicians should continue

MARK DEREWICZ

IN A LETTER FROM KOSTELANETZ TO A FRIEND, THE CONDUCTOR WROTE THAT UPON CROSSING INTO GERMANY ON THE HEELS OF INVADING AMERICAN TROOPS, PONS LEANED OUT OF HER CAR AND SPAT ON THE GROUND AS IF THE NAZIS HAD INFECTED IT WITH EVIL.

In Cologne, Germany, Kostelanetz and Pons posed in front of a burnt-out German tank and the Cathedral of Cologne, one of the few structures left standing after the Allies bombed the city. Image courtesy of Lucy Kostelanetz.

making music because it was "one of the finest flowerings of that free civilization which has come down to us from our liberty-loving forefathers," and that music was "a force of morale."

But composers didn't wait to be told what sorts of music to make or which songs to conduct. The Juilliard School's Ernest Hutchenson held victory concerts at the Metropolitan Museum, with famous musicians such as Marian Anderson, Vladimir Horowitz, Lotte Lehmann, Yehudi Menuhin, and Arturo Toscanini drawing big audiences. In January 1943, the fiftieth performance was attended by nineteen hundred people—double the capacity of the Met's auditorium. These musicians, in turn, helped the government sell war bonds. Fauser found a photo of Pons selling bonds at the Met two months after the United States entered the war.

Hutchenson said that the purpose of the concert series was "to meet evil with superior morale."

Koussevitzky said that musicians and composers had to focus on putting "better music than ever within the reach of the multitudes who will need its divine solace in grave days to come." American musicians had to become "the new bards, new Orpheuses—inspiring, invigorating, ennobling, and consoling."

Andre Kostelanetz was the first to respond to the call. He commissioned a series of musical portraits of famous Americans just eleven days after Pearl Harbor. Kostelanetz said his series was "a reaffirmation of the democracy in which we live and the people who have made our country great." Copland met the challenge with *Lincoln Portrait*. Jerome Kern composed *Mark Twain, A Portrait for Orchestra*. And composer Virgil Thompson used New York mayor Fiorello LaGuardia and the writer Dorothy Thompson as inspiration for musical portraits.

Others took a more propagandistic approach. Barber, for instance, composed the distinctly militaristic "Commando March" for military bands while serving in the air force. But Fauser found that Barber, because of his military duties, had little time to compose other works until he gained a different assignment. The same was true of Lehman Engel, who was put in charge of a sixty-eight-piece band at the Great Lakes Naval Station near Chicago. Engel was lat-

er promoted to lieutenant and led the music division of the navy's Photographic Science Laboratory, where he helped produce propaganda films.

Engel was one of several composers and conductors who led bands that sprouted up during the war. Some were stationed stateside. Some wound up touring with the USO. All were grateful for work. "The war was a boon for rank-and-file musical professionals who suffered through the Great Depression," Fauser says. In the Library of Congress archives, she found that over 95 percent of all USO performers were little-known musicians, thankful for jobs in music. And servicemen, in turn, were thankful for the music—especially classical.

Soldiers at Fort Monmouth in northern New Jersey sent telegrams to the army's Special Services Division requesting that it cancel a theater show to make room for classical pianist Vladimir Horowitz.

Abroad, the USO took classical music to war zones even though at first, Fauser found, the organization doubted soldiers would appreciate orchestral arrangements and arias. She found a memo to Colonel Marvin Young in the Special Services Division: "With the exception of Marian Anderson, Jascha Heifetz, and people of that caliber, I do not recommend offering concert people to posts. Women might be more acceptable (because they are women), but men would not be wanted."

Young disagreed and pushed for the classical artists to entertain troops. Fauser found that some military companies contacted classical performers directly because the USO couldn't keep up with demand.

BY 1943, performing classical music at military bases became a badge of honor for musicians, Fauser says. Letters home and to the USO show that troops were attending concerts because they loved the music, not just because there was little else to do. As one seaman said about violinist Yehudi Menuhin: "Jeez! That guy can do more with a G-String than Gypsy Rose Lee!"

Menuhin was twenty-seven years old. He could've enlisted but didn't. He could've been drafted but wasn't. His personal guilt, Fauser says, may have contributed to his extreme schedule. When not performing for the USO, Menuhin scheduled independent concerts near the front lines. In a letter to his father, Menuhin wrote that playing for soldiers allowed him to know his "American brothers" and his own generation in a way that previously hadn't been possible. Fauser says, "It was a poignant remark for a Jewish musician and former child prodigy long cloistered within a virtuoso career."

There were many tireless USO performers, Fauser says, but few traveled longer or performed harder than Kostelanetz and Pons. The couple gave concerts for an estimated two million GIs, including a Christmas Eve concert in India. They played in Burma, North Africa, France, Germany, China, and Italy. In Iran they performed for Soviet soldiers.

Pons, used to performing in grand halls for society's upper crust, wasn't sure how troops would respond to her. In a letter to a friend, Pons wrote that she was surprised that "these vast audiences of [soldiers], many of whom have never listened to a coloratura soprano, sit so intently and reverently as I sing."

Kostelanetz's assistant conductor, Lieutenant Don Taylor, was a pilot with more than three hundred combat hours. Their orchestra was full of pilots, navigators, and gunners, Fauser says. They stashed their upright piano in a military plane's bomb bay when traveling from war zone to war zone.

February 1945, Burma: U.S. Air Force Major General Howard Davidson greets Pons and Kostelanetz, who had flown in after concerts in China and India. Image courtesy of Lucy Kostelanetz.

Fauser says that for weeks on end, the couple put on concerts every day in unforgiving climates. "And that took a toll on the forty-six-year-old diva," Fauser says. "She prided herself on performing to the same standards for soldiers as she would at Carnegie Hall."

They did this, according to Fauser's findings, because they loved the troops nearly as much as they hated the Nazis.

In a letter from Kostelanetz to a friend, the conductor wrote that upon crossing into Germany on the heels of invading American troops, Pons leaned out of her car and spat on the ground as if the Nazis had infected it with evil. And Kostelanetz wrote, "Now that we are deep in Germany, we are even more pleased with the devastation—most cities are level with the ground or at least not habitable."

Fauser says, "Not even glamorous sopranos and orchestra conductors were immune to military triumphalism."

There's no veiling the fact that Pons and Kostelanetz loved the adoration of entertainment-starved soldiers. But Fauser says that their deep desire to serve the war effort played a far greater role in their motives.

After reading letters, USO reports, and personal travel logs, Fauser is convinced that Kostelanetz—too old to enlist or get drafted—considered his USO touring a substitute for combat duty. He said he was in awe of his audience decked out in battle regalia. In a letter to his orchestra, Kostelanetz wrote: "Anyone who sees and hears the rapt attention and wild enthusiasm of our audiences will realize what music means to our fighting men."

Annegret Fauser is a professor of music and adjunct professor of women's studies in the College of Arts and Sciences. She received a fellowship from the National Endowment for the Humanities to conduct her research, which will be published in her forthcoming book, Sounds of War: Music in the United States During World War II.

Map Quest

The Peutinger map is the only surviving map from the Roman Empire, and for over five hundred years it's brought historians one headache after another. Why is it sausage-shaped? What do those bizarre numbers mean? Who made it? And why? Richard Talbert may finally have some answers.

by Margarite Nathe

DONN YOUNG

"There's an argument about *everything* concerned with the map—absolutely everything," says historian Richard Talbert. But this is what he thinks happened:

It was AD 1500 in Germany. Konrad Celtis—a treasure hunter who often borrowed valuable antiquities from acquaintances and never returned them—was poking around in an out-of-the-way monastery when he came across what seemed to be a fabulously old map. Nailed to a classroom wall, it was a painted strip of calfskin vellum twenty-two feet long and one foot high. The entire Roman Empire stretched across it. Every river, island, region, and town from Britain to Sri Lanka was carefully labeled. A glorious Rome was especially prominent, a network of deep-red roads radiating from it like arteries.

Celtis wanted it.

What he had found was actually a three-hundred-year-old copy of the original map, Talbert says, "or probably a copy of a copy of a copy." But the relic was—and still is—the only surviving map made by the Romans of their own world during antiquity. It gives us a rare glimpse into how the ancient Romans saw their world, Talbert says, and how they felt and thought about it. For centuries, we've known almost nothing about the original map. Now Talbert tells the story in his new book, *Rome's World: The Peutinger Map Reconsidered*.

There were undoubtedly other copies made along the way, but the one that Celtis found is all we've got of the first map, which Talbert believes was made around AD 300 (just one of the many details historians argue about). Most historians say that sometime before Celtis found the map it lost about two feet of its length. But Talbert disagrees. The map has lost at least six feet, he says. That's how much room it would take to fill in the missing portions of Britain and the Iberian Peninsula. And that would put Rome squarely in the center, which Talbert believes was the mapmakers' intention.

Celtis was a fanatic and unfettered by scruples, which made him very good at his day job: collecting rare materials for Emperor Maximilian's library. "But some things he kept for himself," Talbert says. "The map was one for him."

Illegal trade in antiquities was just as common then as it is today, Talbert says, and Celtis knew how to play the system. Although no one knows the details of the transaction—no record of a sale exists, Talbert says—Celtis became the map's new owner. He rolled it up, took it home, and showed it to his educated friends, many of whom were just as voracious for antiquities as he was.

They could all see that the map looked nothing like the earth's true geography. Whoever had drawn it had squashed the whole arc of the known world into a sausage-like frame, draining the seas and disfiguring the landmasses to make everything fit. And

it showed Rome at a preposterous point halfway between Spain and India. The roads, though, seemed about right—very detailed and practical-looking. Ever since the Middle Ages, Talbert says, history buffs and common travelers alike have been infatuated with Rome's roads and all the long distance trips they made possible.

Celtis's friends were excited. They might actually be able to reconstruct the ancient routes, if they could just interpret all those symbols and numbers. But Celtis died before they could decipher the map. And for some reason he left the map to his bibliophile friend Konrad Peutinger—maybe Celtis owed him money, Talbert says—on the condition that Peutinger try to have it copied and published. From then on it was known as the Peutinger map.

The printmaking and engraving industry was up and running by the time Peutinger took over the map, and he hired three artists in succession to take the initial step of copying it. The first two were unsatisfactory, and the third died before finishing. Peutinger never got the job done.

"This thing is a copyist's nightmare," Talbert says. It could take years of meticulous copying, lettering, engraving, and hand-coloring to reproduce even small maps. Even today, mass-producing a twenty-two-foot-long map isn't cheap. Cartographers have been pulling their hair out for centuries trying to make affordable, precise, full-scale, color duplicates of the Peutinger map. But no one has succeeded.

One of the first attempts was a half-size engraving in 1598; unfortunately, the copyist took upon himself to "improve" it, Talbert says, and he made various changes here and there. Another tried in 1753, but the engraver often misunderstood the lettering, and he made a mess of it. Of course, almost no one knew about the copies' errors because so few people could see the Peutinger map firsthand. For centuries, the copies were reproduced in books and were the only images scholars had to work with. No one realized just how inaccurate they were until the first color photographs of the map appeared in the 1970s.

After Peutinger died, the map stayed in his family, although they didn't really have much interest in it. They tucked it away, and occasionally moved it during wartimes. Luckily for us, Talbert says, it slept quietly in storage, protected from the light, for decades at a time. It was even thought to be lost.

Today the Peutinger map is in the Austrian National Library in Vienna, where the staff have guarded it fiercely since 1738. "They managed to keep it away from Napoleon, and it survived World War II," Talbert says. But it was a popular item there, and suffered some wear and tear. For decades the library kept it on a sort of roller, he says. "You could wind it and unwind it. It was common for bits to fall off the edges."

Eventually the library took the map off the roller and separated it into its original eleven segments, which had been gummed together before the map was painted. Unfortunately, Talbert says, vellum has a tendency to warp. While separating the sections saved the map from further damage, the pieces will never fit perfectly together again. Each one is now stored in its own plastic case, and they seldom come out of storage for any patron. Even while Talbert was writing his book about the map, he was never allowed to see all eleven segments together—just one at a time over the course of two years.

The idea that the Peutinger map is merely a sort of ancient AAA guide has persisted, Talbert says, possibly because many of the names and numbers painted along the roads to indicate distances are actually correct, and have helped modern mapmakers create entire atlases of the Roman Empire. The distances are almost always in Roman miles (equal to a thousand paces), although Talbert found that sometimes the miles are swapped without warning for leagues (one and a half times the length of a Roman mile). And the farther east the numbers go, the more bizarre they become, meaning the mapmakers probably used eastern reference sources that listed distances in Persian *parasangs* or Indian *kos*.

But we know today that even the paths that the roads follow are often all wrong, Talbert says—and that's not just copyists' mistakes. "Some scholars have gotten tremendously worked up over this, and they write long screeds correcting the mapmaker," he says. "But they're missing the point. Ultimately, all the road detail is filler." If the map didn't have a practical purpose, he says, it must have had a cultural one.

So why would a culture that knew so much about the far reaches of its empire muck up Rome's place in things so egregiously? And why did the original makers choose that particular shape? Why does it look like a fun-house mirror image of the Western world? How could the map have been displayed? And whose was it?

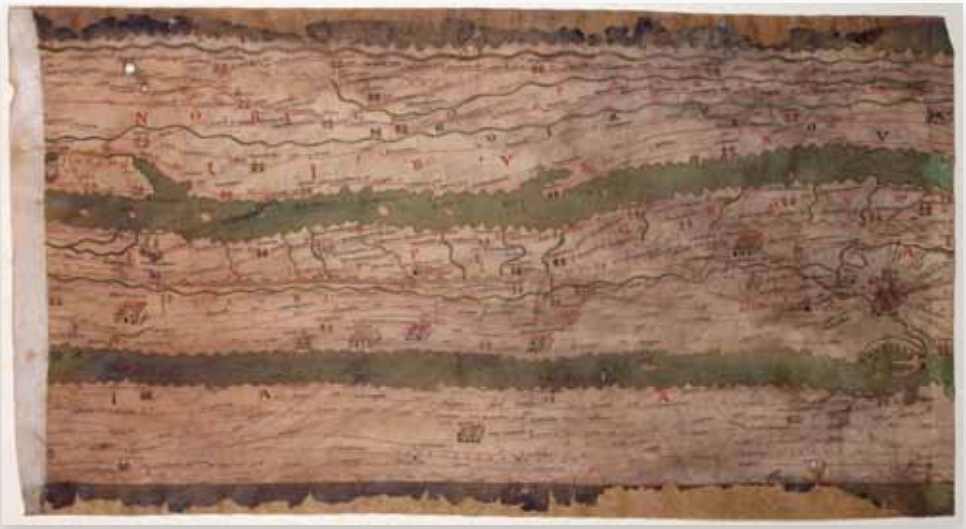
The purpose of the Peutinger map, Talbert claims in *Rome's World*, was not to help anyone plot a route from Gibraltar to Persia—it was to brag about the glory of Rome and show off how posh and civilized the empire had become. "The overall message is 'Yippee, Rome rules the world. Rome is literally the center of the world,'" Talbert says. "Geographically, that's ludicrous, but that's not the point." The point was to make it impressive and ostentatious, and to bring a satisfied smirk to the faces of the educated people who saw it. "There's this in-joke, a sort of 'Tee-hee-hee-ha-ha-ha, don't you know that the world isn't *really* like this.' This kind of sense of humor was very popular among educated, aristocratic people."

Globe maps were made up of five or more horizontal zones. The middle zone at the equator was boiling hot and uninhabitable. Above that was the great northern zone controlled by Rome. And above that, a horrible, cold zone. There were also thought to be southern equivalents, but since the middle zone was impassable, no one gave much thought to those.

In *Rome's World*, Talbert makes the case that the map is probably just one segment of a globe map, an old Greek concept that was still going strong in Roman times. Globe maps were made up of five or more horizontal zones. The middle zone at the equator was boiling hot and uninhabitable. Above that was the great northern zone controlled by Rome. And above that, a horrible, cold zone. There were also thought to be southern equivalents, but since the middle zone was impassable, no one gave much thought to those.

If the Peutinger map *was* part of a globe, all the zones stacked together would have made a chart six feet high. Each section

For years, the Austrian National Library kept the map on a kind of roller device that allowed for convenient viewing but also tended to cause wear and tear. Eventually the library separated the map back into eleven segments of vellum (the segments had originally been gummed together before the map was painted). Right: segment four contains Rome, denoted (at the far right of the segment) by a female figure on a throne, holding a globe, a spear, and a shield.



There are no military fortifications, bases, or legions anywhere on the map. The message, Talbert says: "You don't have to worry about that stuff now! You can relax. Big Brother is looking out for you." The pictorial symbols, like those for cities, temples, baths, and other buildings that pepper the Peutinger map, were common in maps and artwork at the time. Here they're ornamental, Talbert says. If the makers of the Peutinger map had been aiming for absolute accuracy, he says, there would be far more symbols, such as for forts, bridges, mountain passes, aqueducts, and fortification lines.

Few things about the map can be taken literally. "You're not meant to work with this map in a practical way," Talbert says. The mapmakers knew all about geography and latitude and longitude, so they weren't acting out of geographical ignorance. "Quite the reverse," Talbert says. "It's about geographical sophistication. Because unless you have an accurate sense of what the world looks like, you won't get this joke."

would probably have been painted on panels, not vellum, Talbert says, so they could be moved or copied. And such a monstrously huge artwork would need a prominent spot to occupy.

Talbert says it just so happens that around the time the original map was made, the new fashion was for an emperor to sit on a throne in a special cove of the imperial palace called an apse. That's where he received homage. Imagine, Talbert says: "People would grovel to approach him, and there he is in splendor."

Talbert opens *Rome's World* and points to a picture of one such room found in a ruined palace in Croatia. "A globe image would work remarkably well in the apse there behind him," he says. From that spot, the Roman zone—with Rome in the center—would appear just above the emperor's head, and create a very impressive sight. Anyone approaching the emperor would be humbled by this vision of peace, control, and order.

"Now, you can say 'That's brilliant,' or you can say, 'That's twaddle,'" Talbert says. And although he thought his ideas would cause an uproar, so far many of his colleagues have liked this theory—even if they do still disagree on the details.

Rome's World comes with access to digital views of the entire Peutinger map. Any cartographer or Roman history enthusiast can visit a website at Cambridge University Press to study the map in ways that were never before possible. You can use the site's database to look up every mountain, river, and symbol, and read every red-inked word on the entire map. There's even a list of illegible symbols, each linked to its own spot on the map so that you can peer at it closely.

Talbert believes the original map was part of a larger, globe-shaped map that was installed behind the emperor's throne in the imperial palace. Sketch by Daniel Talbert.

After years of studying the Peutinger map, Talbert is convinced his theories are plausible. But there are some things we'll probably never know for sure, he says. Just how much of the map is missing? When and where was the original actually made? How faithful is this only remaining copy to the original? "Whether we'll ever know more about the Peutinger map than we do now is a faint hope at best," Talbert says, "but still a far from inconceivable prospect."

Richard Talbert is the William Rand Kenan Jr. Distinguished Professor of History in the College of Arts and Sciences. Much of the data entry for the online database was contributed by student assistants in UNC's Ancient World Mapping Center. To see the Peutinger map online, visit: cambridge.org/us/talbert/.

In 2007, the Peutinger map was added to the UNESCO Memory of the World Register.

Richard Feins wants cardiothoracic surgery residents to learn skills by practicing on a realistic simulator before they start their operating-room training. "It's a terrible feeling," he says, "to have a patient's life in your hands and not be as prepared as you possibly can be to take care of that patient." Photo by Michelle Morgan.

TO LEARN IT BY HEART

by Susan Hardy

An operating room: bright lights, trays of instruments, people in masks and blue gowns. A monitor on the wall shows a live image of a patient's heart. The resident standing next to the operating table has just performed an artery bypass graft; he's waiting while the more experienced surgeon who guided him through the procedure inspects his work.

Good job, the surgeon tells him. Now do it again—this time, with the heart beating.

The heart is from a pig, and the patient is an old mannequin from a clothing display. The blood inside it is fake, from a store that supplies it to Hollywood studios. But this is real training for residents specializing in cardiothoracic surgery. The heart is positioned in the mannequin's chest like it would be in a human's. It can produce arrhythmias if a surgeon in training handles it the wrong way, or when a computer tells it to. It can go into cardiac arrest. It's not forgiving of mistakes. "If you don't do the right thing," surgeon Rick Feins says, "there's going to be blood all over the place."

Feins heads up a team that trains residents in common procedures such as coronary bypass, heart valve replacement, and lung resection. Simulators like this one aren't part of the traditional curriculum for surgeons specializing in cardiothoracics; the surgical community hadn't seen anything like it until Feins started using it in 2008. "At that time, no one in cardiac surgery really thought there could be anything that was like learning in the operating room," he says.

Feins first started thinking about simulation in 2007, when one of his sons was in medical school and the other was training to be a navy pilot. Before Jonathan could get into a plane, he had to log hundreds of hours in a simulator. Wouldn't it make sense, Feins thought, for surgical residents to have to do the same before they were allowed in the OR?

He started researching cardiac simulation, and at first the results didn't seem too promising. Plastic models of the thorax (the organs in the chest) cost a lot and aren't all that much like the real thing. Digital simulation is also expensive, and it lacks haptics—the quality of weightiness that tissue has when you pull on it. Sometimes surgical residents learn on live pigs, but that means killing a pig in order to practice with organs that aren't even arranged like human anatomy.

But then Feins found a journal article about a cardiac simulator, complete with a video of it in action. It really looked like surgery being done on a human. He tracked down the article's first author and called him up. "This is Dr. Feins. I'm from UNC and I'm the chair of the American Board of Thoracic Surgery. I read about your simulator, and I'd like to come down and see it."

Is this a joke? Paul Ramphal asked. The article was two or three years old, and Feins was the first person to contact him about it. Ramphal, also a surgeon, had since moved on to other things. He'd also moved from Jamaica, where he built the simulator, to the Bahamas; all he had left of it were a few pieces sitting in his garage.

So come to Chapel Hill, Feins said. Bring anyone you need with you, and we'll rebuild it here. They had only a small grant, but they made it stretch, getting free pig hearts from a North Carolina farm and scavenging old surgical equipment discarded by UNC Hospitals. Residents volunteered their time and labor (it turned out that they weren't allowed to work for free under the terms of their contract, so they got minimum wage).

Feins started researching cardiac simulation, and at first the results didn't seem too promising. Plastic models of the thorax (the organs in the chest) cost a lot and aren't all that much like the real thing. Digital simulation is also expensive, and it lacks haptics—the quality of weightiness that tissue has when you pull on it. Sometimes surgical residents learn on live pigs, but that means killing a pig in order to practice with organs that aren't even arranged like human anatomy.

When it was done, Feins took the simulator to a conference of cardiothoracic residents, where it was a hit.

"All it takes is for other surgeons to see the thing, and they say, 'Wow, this is it,'" Feins says. "Most of them haven't even really seen it—they've just seen our videos, and I get one or two calls a week asking where they can get one." The University of the West Indies in Mona, where Ramphal first designed the simulator, is producing it for commercial sale; seven major hospitals, including Johns Hopkins and the Mayo Clinic, have already bought in. Meanwhile, Feins has led a yearly surgical boot camp at UNC, bringing residents from around the country along with surgeons to teach them.

Then in 2009, Feins and his colleagues designed a new simulator focused on the lungs. It's also made of pig parts—heart, lungs, esophagus, and so on—arranged like they would be in a human. The lung simulator started when a biomedical engineering student from Vanderbilt, Alec Grubbs, came to work for Feins for the summer. Grubbs's father, Andy Grubbs, was also an engineer, and happened to be a lecturer in the Kenan-Flagler Business School. The father-and-son Grubbses came up with solutions to make a lung simulator run cheaply—it has to look as realistic as the Ramphal simulator, but in this case, the heart simply has to beat while a student operates on the lungs.

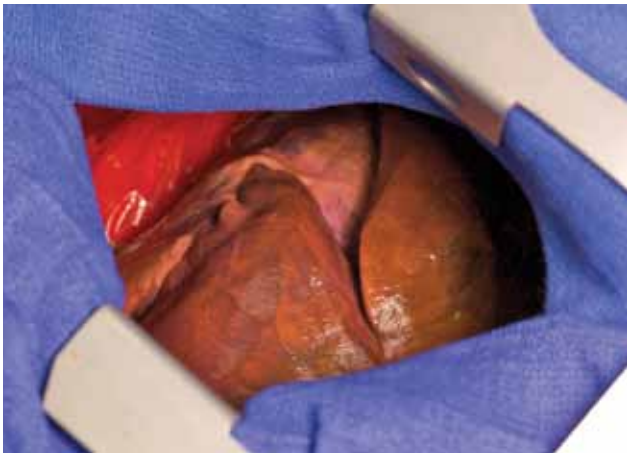
They used a motor designed to power the windshield wipers of a car to drive a puff of air into the heart seventy-eight times a minute—the same rate at which a human heart might beat.

Andy Grubbs is starting a spin-off company to develop the lung simulator commercially. He and Feins want to produce it in rural North Carolina, where unused pig organs are plentiful and jobs are needed. They're patenting a dummy that has removable sections that surgeons can replace with the organ systems they need, such as the liver, spleen, and kidneys (also made from pig parts). The U.S. military is interested in the dummy as a training tool for medics and Hospital Corpsmen.

Feins is also working on a curriculum to make surgery simulators part of the standard program for surgical residents. The traditional education that takes place in the operating room is safe for patients, he says, but it's not the best way for residents to learn. "Operating rooms nowadays are very, very busy," he says. "Teaching may not be as easy to do there as it was before. And in the operating room you can't control the curriculum—whatever the patient has that day is what you're teaching. That's particularly difficult with what we call adverse events, because you can't know when those will happen or inflict them at any time."

Also, Feins says, in the OR you can't practice a skill over and over the way you'd practice swinging a golf club or playing a piece on the flute. Surgical simulation could relieve some of the pressure on operating rooms by delivering residents who already have basic skills from working with real tissue.

"If you can teach those things in a controlled environment before you move on to the operating room," he asks, "then why would you want to do it any other way?"



A pig heart is similar in size and anatomy to a human's. Storing it in 40 percent pharmaceutical-grade ethanol (that's vodka with fewer impurities) keeps the tissue supple and lifelike. Photo by Michelle Morgan.

Richard Feins is a professor of surgery in the School of Medicine. The Ramphal Cardiac Surgery Simulator was built with funding from UNC's Department of Surgery and the American Board of Thoracic Surgery. UNC medical students Matthew Dedmon, Melissa Readio, and Aaron Webel; UNC surgical residents Leora Tesche and Phil Pepple; and Vanderbilt engineering undergrad Alec Grubbs helped develop the Thoracic Surgery Simulator.

THE GOOD, THE BAD, AND THE UNKNOWN:

BACTERIA IN YOUR BODY

FIVE STORIES BY MARK DEREWICZ

ILLUSTRATIONS BY ERIC KNISLEY

INSIDE YOU RIGHT NOW THERE ARE MORE BACTERIAL CELLS THAN HUMAN CELLS. IN FACT, IF YOU CRACKED OPEN EVERY CELL IN YOUR BODY, UNWOUND THE DNA, AND STITCHED IT TOGETHER, YOU'D FIND TEN TO A HUNDRED TIMES MORE BACTERIAL DNA THAN HUMAN DNA.

In our mouths, on our skin, and in our digestive and reproductive tracts, bacteria have been with us since the beginning, evolving with us. We know some of them. Researchers identified *Lactobacillus acidophilus* many years ago. You might know it from reading yogurt labels. It's supposed to be good for us. *E. coli* live inside us. We know a lot about them. They're not so good.

Most of the bacteria in our intestines, though, can't survive exposure to oxygen, which means they can't be cultured on a plate and studied like other bacteria. Scientists estimate that there are hundreds, maybe thousands, of species inside us, but we know little about them because we haven't been able to study them—until now.

As part of the Human Microbiome Project, which is a National Institutes of Health mandate to identify and characterize the bacteria that call humans home, scientists use new methods that allow them to isolate and sequence bacterial DNA. This means they are finally identifying more strains of bacteria. At UNC, some of these new technologies are housed in the Microbiome Core Facility, where researchers can figure out the bacterial composition of, well, anything. And there are plenty of reasons why we want to figure out what bacteria are doing inside of us, especially in our bellies.

For centuries scientists didn't think our relationship with bacteria was terribly important, except in those rare cases when bugs escape the GI tract, enter the bloodstream, and cause a life-threatening infection. Then researchers started figuring out that our intestines are full of known and unknown species that compose unique bacterial ecologies. And each of us, they're finding, has a symbiotic relationship with these bacteria.

Today we're learning that diverse groups of bacteria play major roles in several ailments, including colitis, Crohn's disease, irritable bowel syndrome, obesity, diabetes, cystic fibrosis, and even colon cancer. Scientists at Carolina and elsewhere are proving that probiotics—the so-called good bacteria sold at health-food stores—can help millions of people, including kids. And they've found that bacteria's effect on health starts when we're babies.

In the following pages, UNC researchers tell of their findings even as they continue to explore uncharted territory—inside us.

ON THE ATTACK

What triggers the inflammation that causes gut problems?

Balfour Sartor couldn't shake the fevers. It was 1971 and he was a college senior. He had lost weight, felt tired all the time, and had no idea why. His doctor came up with an unusual diagnosis: Crohn's disease, a serious inflammation of the small intestine. "I had never heard of it," Sartor says. "The doctor gave me anti-inflammatory pills and steroids, totally nonspecific medications that dampen the immune system response."

The doctor knew that an overactive immune system could trigger inflammation, but he didn't know what caused Sartor's T-cells and B-cells to attack the walls of his small intestine. No other doctor did, either—a fact that inspired Sartor to specialize in gastrointestinal disorders during medical school. He became a GI fellow at UNC.

One day Sartor ran into his boss, who handed him a journal article. "It was about how a purified bacterial product caused arthritis in rats," Sartor recalls. The bacterial product was a polymer that's found in the cell walls of virtually all bacteria. Immediately, Sartor had an idea. Arthritis, essentially, is a kind of inflammation. Bacteria live in our digestive tracts and are in their highest concentrations in the parts of the intestine where Crohn's usually occurs. As he thought about chronic intestinal inflammation, Sartor said to himself, "It's the bacteria, stupid!"

He sought out the author of that article, UNC's own John Schwab, a microbiologist. "I told him I wanted to test his bacterial product to see if it could cause gut inflammation," Sartor says. "Most people thought I was out of my gourd. I had no research background. I was probably headed into private practice back home in Louisiana. But Dr. Schwab is a marvelous guy." He didn't think Sartor was crazy, and agreed to let him test his theory. And luckily Sartor had a little surgery background, so he was comfortable operating, even on a rat.

Working with Schwab, Sartor injected the bacterial product into a rat and waited. Within a month, the rat developed inflammation that looked a lot like Crohn's disease. It was a seminal finding that turned Sartor into a pioneer in a previously nonexistent field—normal intestinal bacteria implicated in inflammatory bowel disease (IBD). He never entered private practice, and he stayed at UNC, where he works with patients who struggle with gastrointestinal problems.

For years Sartor's team has bred germ-free rats and mice that scientists from around the world have used for research. In many experiments Sartor used mice deficient in the protective gene interleukin-10; these mice developed colitis, or inflammation of the large intestine. But IL-10 deficient, germ-free mice didn't get any inflammation. "Then, when we exposed those mice to typical gut bacteria, they got the disease within a week," he says.

UNC molecular biologist Ian Carroll, who works with Sartor, used the IL-10 knockout mice to see if a probiotic could help prevent inflammation. He gave mice *Lactobacillus*, a common beneficial bacterium thought to keep detrimental bacteria in check. The mice didn't experience as much inflammation. Then Carroll gave *Lactobacillus* to another set of mice, except this time the probiotic was engineered to release antioxidants in the digestive tract. The mice experienced even less inflammation.

The upshot is that certain strains of *Lactobacillus* might protect people from developing the worst kinds of inflammation associated with GI disorders. But that doesn't mean that probiotics help people if they already have Crohn's disease or ulcerative colitis, the causes of which are complex. Part of what's going on, Sartor says, is that Crohn's patients have genetic makeups that cause the innate immune system to react inadequately to the so-called bad gut bacteria. As a result, the adaptive immune system overcompensates—T-cells go on the attack against the normal intestinal bacteria and don't stop—causing inflammation that leads to symptoms such as abdominal pain, lethargy, diarrhea, fevers, weight loss, and bleeding. Most Crohn's patients are forced to have parts of their small intestines and colons removed. Eighty-five percent of the time patients require a second surgery within twenty years of their first operation.

Sartor had surgery in 1993 and hasn't needed another one. "I treat it traditionally with immunosuppressive agents, but I'm also compulsive about my diet and take an antibiotic directed against aggressive gut bacteria," he says. "I used to chug Coke and lots of sugary fruit juices. I don't do that anymore. I've pretty much cut out refined sugar from my diet."

Using the IL-10 mouse model, Sartor's team found that a high-sugar diet makes inflammation worse. "We also found that some bacteria groups grow in the presence of sucrose and fructose and others don't," he says. Sartor's group now uses the Microbiome Core Facility to profile these bacteria. The idea is to find out which species feed on sugar and cause bacterial imbalance in the gut.

To help limit symptoms and promote beneficial bacterial populations, Sartor eats prebiotics—fibrous foods that beneficial bacteria consume to grow their colonies. Leeks, garlic, onions, artichokes. If prebiotics help, what about probiotics? I asked Sartor if he took one. "I don't," he says. "There's no evidence that probiotics are effective for postoperative Crohn's disease." One reason for that, he says, is that a lot of commercial probiotics don't contain the same species found in our guts. For instance, *Lactobacillus* and *Bifidobacteria* are genera that contain largely beneficial species. But the Lactobacilli and Bifido species found in probiotic pills and yogurt are not the same species found in us. Most don't colonize in our intestines, Sartor says, and don't necessarily alter the microbiota. What Crohn's and colitis patients might need is a bacterial transplant, though the most current study results are not yet verified. In studies without control groups, Australian doctor Thomas Borody successfully treated colitis patients with human probiotic infusions—also known as fecal transplants. Without getting into details, the procedure involves repopulating the patient's colon with a healthier bacterial ecology (one found in a family member's fecal matter).

Brock Miller, a GI fellow at UNC, has used fecal transplants to help patients with *Clostridium difficile*—a nasty bacterial infection. In 2009 Miller had a patient who kept getting C. diff, so he recommended a fecal transplant. "She felt better the next day and hasn't had a relapse," Miller says. "A transplant almost always works for C. diff." Though the treatment is not yet approved for Crohn's or colitis patients, Sartor thinks IBD patients might benefit from altering their bacterial ecologies with a more specific approach using certain strains of bacteria.

Researchers in France, Sartor says, have found that Crohn's patients lack normal amounts of *F. prausnitzii*, a beneficial bacterium. Patients who relapse soon after surgery have especially low amounts of that bacterium. Sartor says, "In mice, those researchers found that *F. prausnitzii* protected against colitis."

Right now there's no *F. prausnitzii* pill at the health-food store, and doctors don't routinely prescribe other probiotics to IBD patients. But probiotics still might help the millions of people who scour drugstore aisles a little too often for relief from digestive problems.

THE "GOOD" BUGS

And why you want them

Probiotics date back to the early 1900s, when Russian scientist Elie Metchnikoff observed that Bulgarian peasants lived unusually long lives. He thought it was because they ate milk fermented by lactic acid bacteria. Metchnikoff started eating fermented sour milk made with a bacterium he called *Bulgarian Bacillus*, and reported that his health improved. His friends began eating it. Doctors in Paris, where Metchnikoff worked at the Pasteur Institute, began telling patients about it. After his death, Metchnikoff's bacterium was renamed *Lactobacillus delbrueckii subsp. bulgaricus*, which is one of the bacteria in some yogurts.

In 1920 scientists found that *L. d. bulgaricus* could not live in the human intestines; they doubted it had any benefit. The fermented milk fad faded in Paris, but research was just getting underway in the United States. In 1935 U.S. researchers surmised that *L. acidophilus*, a naturally occurring gut flora, would be more beneficial than *L. d. bulgaricus*. Clinical trials concluded that *L. acidophilus* helped relieve constipation. In 1989 probiotics were defined as live microbial feed supplements that beneficially affect the host animal by improving its intestinal microbial balance. Since then companies have cultured various bacterial strains and sold them as digestive aids.

"But not all probiotics are equal," Sartor says. Many of the probiotic products you can buy in stores have not been tested.

Tamar Ringel-Kulka, a UNC maternal and child health researcher, studied literature about the health benefits of probiotics marketed for kids and found that very few products had been investigated for effectiveness. Through a series of clinical studies, she and gastroenterologist Yehuda Ringel, her husband, have been investigating the beneficial effects of probiotics in children and adults. They're also trying to figure out how exactly these good bacteria work.

Ringel sees patients who have functional GI disorders for which there are no clear causes, such as inflammation or identified infections. Symptoms can include diarrhea, gas, bloating, nausea, constipation, and abdominal pain.

In one study, Ringel gave participants *Lactobacillus acidophilus* NCFM, a probiotic developed at NC State, and *Bifidobacterium Lactis* Bi-07. "The main effect was on bloating," Ringel says. "It was significantly reduced in patients who received probiotics compared to those who received placebos." The Mayo Clinic tested another blend of probiotics and also found that patients experienced significantly less bloating and gas. And, Ringel says, an RTP company got similar results after treating patients with an antibiotic that works only in the digestive tract.

Ringel and Ringel-Kulka are now studying the possible mechanisms by which probiotics relieve bloating. Previous animal studies revealed that L-NCFM can increase the activation of the colon's opioid receptors. "It may work like morphine," Ringel says. "If you activate the opioid receptors, you reduce sensation." You reduce the feeling of bloating. Ringel and Ringel-Kulka are now investigating whether something similar happens in humans.

Meanwhile, molecular biologist Ian Carroll is using UNC's Microbiome Core Facility to characterize bacteria taken from the people in Ringel and Ringel-Kulka's studies. The goal, Ringel says, is to gain a better understanding of the intestinal microbiota, especially in patients with functional GI disorders. Maybe a different probiotic would help more patients or alleviate different symptoms. Maybe a specific bacterial concoction can help prevent digestive problems.

Ringel-Kulka, a pediatrician, is doing that kind of work with children. She gave healthy kids aged one to four a yogurt drink containing *Streptococcus thermophilus*, *Lactobacillus bulgaricus*, *Bifidobacterium lactis* (BB-12), and the prebiotic inulin for sixteen weeks. After collecting biweekly journals that the children's parents completed, Ringel-Kulka found that kids who drank the concoction had fewer days of fever and significantly better quality of life, which in this context means that the kids felt better than usual and behaved better in day care than kids who were given a placebo.

"The changes we found aren't the major kinds of changes you see in patients who are ill," Ringel-Kulka says. "But that's why it's meaningful. The fact that you can improve health even further in healthy kids is pretty interesting."

That's especially true when you factor in that our symbiotic relationship with bacteria begins at birth.

THE BATTLE FOR BABY'S BELLY

Not all newborns have the same bacterial compositions.

As a dad of two young kids, I was fairly confident that dirty diapers had no redeeming qualities. Apparently I've been misguided. These days, soiled nappies are good as gold to researchers interested in gut microbes.

Germ-free while in the womb, babies are exposed to beneficial bacteria, such as *Lactobacillus*, in the birth canal during delivery. Researchers are linking increased rates of childhood allergies and asthma to Caesarean section and the lack of a typical bacterial ecology at birth.

Breastfeeding also seems beneficial for bacterial balance. Sartor says that premature infants are susceptible to necrotizing enterocolitis, a devastating overgrowth of bad gut bacteria. "But that's almost universally found in formula-fed babies, not breastfed babies," Sartor says. Breast milk provides a steady influx of beneficial bacteria, he adds, "and there's abundant evidence that gut bacteria colonized early in life induce protective immune responses."

The theory is that vaginal birth and breastfeeding help babies acquire a healthy gut bacterial ecology, which helps the immune system ward off chronic ailments later in life. Right now findings are mostly correlative, not causative. But researchers, including some at UNC, have already found that diet affects babies' bacterial balances. The evidence is in the diapers.

Andrea Azcarate-Peril, a microbiologist who directs the Microbiome Core Facility, studied bacterial compositions in baby poop to find that breastfed babies have many more Firmicute bacteria than formula-fed infants. Firmicutes is a large phylum that contains classes and genera of beneficial bacteria. Azcarate-Peril also found that formula-fed babies have bacterial patterns similar to adults'. That's not good. Other researchers have found that too many adults have bacterial imbalances, which are being linked to chronic conditions, such as diabetes associated with obesity.

"Not all diseases are a result of the microbiota," Azcarate-Peril says. "But if there's an imbalance, it affects you." If babies don't get proper amounts of so-called good bacteria early in life, there could be consequences later on.

Anthropologist Amanda Thompson is trying to connect the dots. Every week for fifteen months, she collected soiled diapers from thirty-two families. Back at her lab she analyzed the poop and found that babies who are fed formula and solids in addition to breast milk have higher levels of the hormones that promote body fat. Now she's using the Microbiome Core Facility to analyze bacterial composition. Her preliminary results show that babies who drink formula and eat solid food early in life have less Firmicutes. And because she got samples from babies every day or every week, she could see when the changes in bacterial composition happened over time and when parents altered their babies' diets. She will use more detailed DNA tests to pinpoint which genera and species are most involved, but the fact that formula-fed babies have fewer good bacteria and higher hormone levels doesn't bode well.

Those are two of the underlying factors in obesity. Beneficial bacteria help us digest food. As endocrinologist Kay Lund points out, lean people have different bacterial ecologies than overweight people.

Lund has implicated bacteria in type 2 diabetes, which can be a consequence of obesity. She found that a high-fat diet triggers inflammatory responses in the small intestines and colon long before the animals gain weight. Germ-free animals that don't have bacteria don't get the intestinal inflammation, insulin resistance, or weight gain even when they eat as much fat as regular rats. She also found a strong correlation between bacteria-induced inflammation and insulin resistance, which can lead to diabetes.

It may seem strange that bacterial imbalances in our guts play roles in diabetes, asthma, and allergies. But that's what researchers find fascinating about this particular line of work. As gastroenterologist Yehuda Ringel says, because we know so little about the human microbiota scientists can check how bacteria relate to just about any disease or disorder they can think of. And the correlations and causations between bacteria and disease are piling up.

BIG, BAD BACTERIA

Are gut microbes implicated in the worst kind of colon disease?

Five years ago I changed my diet and started taking probiotic pills to help with digestion. Within days I felt like a new man. But I never thought that a beneficial bacteria supplement might someday be able to help me avoid disease.

For most of his twenty-five-year career, Robert Sandler has been researching the causes of colorectal cancer, the second leading cause of death by cancer in the United States. "I was studying the same things everyone else was—diet, lifestyle, physical activity, family history, medication," he says. "But the more I studied, the more obvious it was that there was something missing, because the data on diet are pretty inconsistent. Everyone thinks fiber protects against colon cancer. But if you look at the studies, some say it does and some say it doesn't. About eight years ago I began wondering if gut bacteria play a role."

Sandler says fiber might protect some people but not others against colon cancer because we all have different bacteria in us that react differently to fiber. He began working with Tope Keku, a UNC researcher who took the lead on clinical studies that try to determine whether bacteria have any say in whether someone gets colon cancer.

In 2010, Keku and Sandler used the Microbiome Core Facility to find that patients with precancerous growths or polyps in their colons have more Proteobacteria than people without the growths. Proteobacteria is a phylum that includes hundreds of species, including *E. coli* and other pathogenic bugs. Other researchers found that *E. coli* were more prevalent in biopsies taken from people with precancerous polyps and malignant tumors. And because bacteria fight each other for space in our bellies, the patients with more Proteobacteria and *E.coli* have fewer beneficial bacteria.

Still, Sandler and Keku can't yet say that bacteria caused the tumors. It could be that the tumors formed and then the bacterial balance was altered. "I think that's kind of far-fetched," Sandler says. "It's much more likely that bacteria play a role in the creation of the precancerous polyps."

Keku says, "*Something* upsets the balance of bacteria. It could be diet that leads to some bacteria growing faster than others. And then maybe those bacteria promote colon cancer."

To find out, she's expanding her study to include hundreds of participants, and she'll use DNA sequencing to get a more precise view of which bacterial species are prevalent in patients with the kinds of polyps that often turn into cancer. She's also creating animal models to see if certain bacterial genera and species promote cancer growth. And if some do, then there ought to be a way to limit those so-called bad bacteria and promote the good kind to take up residence in our intestines.

It's not so crazy to think that someday those capsules full of bacteria and those creamy fermented snacks could include the sorts of good bacteria that are proven to prevent the fourth most common form of cancer in the United States.

"It would be great to find out what bacteria are missing and introduce them back into our diets," Keku says. "What a great way to address colon cancer—you could know your risk and lower it by eating yogurt every day."

BUGS IN THE LUNGS

More than a lone gunman

Matthew Wolfgang was fascinated by two simple truths: people with a specific genetic mutation will get cystic fibrosis (CF), and eventually, a nasty bacterium called *Pseudomonas aeruginosa* will dominate their lungs. Why *that* bacterium and not a different but equally bad bug? "We have no idea," Wolfgang says. "We all encounter *Pseudomonas* every day. It's abundant in soil and on plants. It likes to live in sink taps and shower heads. It doesn't affect healthy people at all. But as soon as you become immune-compromised it becomes a problem."

For years, CF researchers thought *Pseudomonas* was a lone gunman. But now they know that it has accomplices. Wolfgang used UNC's Microbiome Core Facility to find dozens of bacterial species in the lungs of CF patients. "One is *Prevotella*," Wolfgang says. "It normally resides in the mouth and doesn't really do anything in healthy people. Occasionally it's associated with abscesses. We've been finding *Prevotella* in high numbers in a lot of our CF patients."

Wolfgang's lab also found bacteria that can't survive in oxygen-rich environments. "This tells you about where the disease has progressed in a patient," he says. Some CF lungs are so damaged that oxygen can't reach every part of the lung. Anaerobic bugs infect those areas, causing more inflammation.

Wolfgang says that the antibiotics CF patients take to kill aerobic bacteria are not as effective against anaerobic bacteria. "Maybe patients need combination therapies," he says. "Maybe those *Prevotella* bacteria and others play some beneficial role."

Wolfgang admits it seems crazy to think that *any* bacteria in the lungs would benefit anyone. Having no bacteria would be best. But a diverse community would be better than one dominated by pathogenic bugs. "It's like in the gut," Wolfgang says. "You want a balance. Bacteria fight for space, for nutrients." You don't want a really bad bug, such as *Pseudomonas*, to win that battle.

Wolfgang teamed with researchers in Ireland to study how bacterial communities change over time in CF patients. Turns out that as symptoms worsen and lung function decreases, the bacterial ecology of CF lungs gets less diverse. *Pseudomonas* becomes more dominant. The correlation is clear, but what to do about it isn't.

CF patients typically take antibiotics that can have long-term side effects, such as kidney problems. Wolfgang points out that some bacteria in CF lungs are highly resistant to antibiotics. In the end, *Pseudomonas* dominates and causes major respiratory failure.

Still, trying to keep a CF patient's lungs bacterially diverse might not be a good idea either. "I'm nowhere near advocating the use of probiotics—colonizing someone's lungs with bacteria—because we know that the inflammation response to bacteria is doing a lot of damage," Wolfgang says. "We're getting a good handle on what's going on in the lung, but we're a ways off from figuring out how to intervene in a useful, therapeutic way."

Balfour Sartor is a distinguished professor of medicine and microbiology and immunology in the School of Medicine, and director of the National Gnotobiotic Rodent Resource Center. Andrea Azcarate-Peril is an assistant professor of cell and molecular physiology and director of the Microbiome Core Facility. Ian Carroll is an assistant professor of medicine, Yehuda Ringel is an associate professor of medicine, and Brock Miller is a postdoc, all in the Division of Gastroenterology and Hepatology in the School of Medicine. Tamar Ringel-Kulka is a research assistant professor in the UNC Gillings School of Global Public Health. Amanda Thompson is an assistant professor of anthropology in the College of Arts and Sciences and a fellow at the Carolina Population Center. Kay Lund is the Sarah Graham Kenan Professor of Cell and Molecular Physiology. Tope Keku is an associate professor of medicine in the School of Medicine and an adjunct associate professor of nutrition in the UNC Gillings School of Global Public Health. Robert Sandler is a distinguished professor of medicine and epidemiology, and chief of the Division of Gastroenterology and Hepatology in the School of Medicine. Matthew Wolfgang is an associate professor of microbiology and immunology in the School of Medicine and a member of the Cystic Fibrosis/Pulmonary Research and Treatment Center.

When her phone rang in 2004, Carol Otey had no idea it would change the course of her research.

The call was from a stranger, Teri Brentnall at the University of Washington. Brentnall had done a genetic analysis of a Washington State family, referred to by researchers as Family X, that had an extremely high rate of pancreatic cancer.

Seven members of Family X had already chosen to have their pancreases removed—giving them instant diabetes—to prevent this deadly cancer. Brentnall knew generally where the mutation was that was causing their disease, but that left about 250 candidate genes. Number twenty on the list was palladin.

Otey had discovered palladin years before. Now a stranger from across the country was calling to ask whether she thought it was possible that a mutation in palladin could be causing pancreatic cancer in Family X. Otey hesitated. A cold call like this is rare in science, where competition for funding and publications discourages tipping one's hand.

Cold-called

story and photos by Alex Raines

At a meeting of the Pancreatic Cancer Action Network, Carol Otey met a woman whose husband had died of pancreatic cancer. The disease had also claimed other members of the man's family, and the woman was terrified for her children. "It really raised my consciousness about the urgent need for more research in this area," Otey says. "It motivated me to work on this."

Brentnall didn't know that Otey had brand-new evidence that palladin affects the behavior of cultured breast cancer cells. "I had to make a decision on the spot about whether to confide in someone I had never met about our unpublished results," Otey says. "But I felt it was necessary that I tell her." She passed along what they knew and offered to help. Then she didn't hear back from Brentnall for months.

Brentnall's lab was having trouble with the palladin gene, which is huge and complex. So a technician from Brentnall's lab came to work with Otey, staying in her house while they worked together. After a few more months, the collaboration paid off: Family X carried a mutation in the palladin gene that changed just one amino acid from a proline to a serine. All of the affected family members had the serine—the others had the proline. This was the first clue to what was causing pancreatic cancer in Family X, and it would eventually lead Otey to a possible tool for early diagnosis.

An unexpected discovery

Otey discovered palladin by accident in 1991. She was a postdoc at UNC studying the cell's cytoskeleton. Her research focused on alpha-actinin, which can bundle and anchor filaments of actin, one of the main components of the cytoskeleton. Otey had an antibody that she thought recognized alpha-actinin, indicating when it was present in cells or tissue. It took four years before she figured out what it was actually recognizing: a new protein that binds to alpha-actinin.

Otey soon figured out that, like alpha-actinin, the new protein regulates actin and the cytoskeleton. That's where she got the idea for the name. Because the protein was controlling the architecture of cells, Otey named it after the Italian Renaissance architect Andrea Palladio.

Before she could publish on palladin, Otey had to clone the gene that produces the protein. Cloning in this context means copying the gene from the genome into a form that can be sequenced and manipulated to study its function. This was in the days before the human and mouse genome projects; it took a long time to clone the palladin gene. But along the way, Otey began studying the function of the protein, eventually finding that it was activated by cells during wound healing. This was the first hint that palladin might be involved in cancer.

Otey says tumors are often described as wounds that don't heal. And when tumor cells metastasize they can behave a lot like cells responding to an injury. So Otey began to look at palladin in breast cancer cells. Later, she got the call from Brentnall.

After a couple years of work, Otey and Brentnall published their discovery of the palladin mutation in Family X. Otey continued studying palladin in breast cancer cells, but knew she had to follow this lead on pancreatic cancer. The problem was, Otey didn't know much about pancreatic cancer.

Rare but deadly

Pancreatic cancer is the fourth leading cause of cancer deaths, but it doesn't have the visibility of the others in the top five, like breast cancer or prostate cancer. Patrick Swayze, who in 2009 died from pancreatic cancer twenty months after his diagnosis, helped raise the profile of the disease. But it still doesn't get the research funding and attention of the more high-profile cancers. It's not associated with a certain behavior, like lung cancer is. There's no screening test. Only about 5 percent of patients live five years. Many only make it a few months.

The problem is that pancreatic cancer is almost never found early. The pancreas, which is responsible for controlling glucose levels in the blood and helping digest our food, is tucked away at the back of the abdomen, just beneath the stomach. It's so buried that it's hard for doctors to check during physical exams. Symptoms of pancreatic cancer, including back pain and weight loss, are vague and can be mistaken for other diseases.

Once pancreatic cancer is diagnosed, it is hard to treat. Chemotherapy and radiation are ineffective. Surgery is the only hope, but often the cancer has spread by the time it's diagnosed, making it impossible to remove. Even when patients are eligible for surgery and surgeons think they have removed all of the cancer, only 25 percent of those patients live five more years.

A clinical-science partnership

Luckily for Otey, as she was trying to figure out how to direct her research, she met with a few other researchers on campus interested in pancreatic cancer. One was H. J. Kim, a surgical oncologist who specializes in pancreatic cancer. In addition to seeing patients and operating, Kim was studying tumor resistance to chemotherapy. He was immediately intrigued by Otey's work.

Kim invited Otey to join him at a meeting of the local chapter of the Pancreatic Cancer Action Network. Kim was there to give a presentation, but it also gave Otey a chance to meet the people she was trying to help. One woman's husband had died of pancreatic cancer—so had other members of his family, and his wife was terrified for her children. "It really raised my consciousness about the urgent need for more research in this area," Otey says. "It motivated me to work on this."

Together, Otey and Kim have found some surprising results. There are at least nine so-called isoforms of palladin; other labs had shown that one form is much more prevalent in pancreatic tumors than in normal tissue. But Otey and Kim were shocked to find that this isoform wasn't in the cancer cells—it was in the cells surrounding the cancer.

These surrounding cells, called tumor-associated fibroblasts (TAFs), may hold the key to how tumors grow and metastasize, Otey says. "They also secrete a dense layer of collagen around the tumor that may be acting as a barrier to chemotherapy drugs, walling off the tumor."

Kim says that pancreatic cancer is particularly resistant to chemotherapy and is known for a prevalence of TAFs. So palladin may be influencing both how the tumors spread and how they respond to treatment. Someday doctors may be able to use this knowledge to improve chemotherapy or slow the metastasis of pancreatic cancer.

But Otey is excited that another aspect of their work may benefit patients much sooner. The TAFs, and palladin along with them, show up very early in the disease. Using mice that were genetically engineered to develop pancreatic cancer, Otey found this isoform of palladin when the tumors were just beginning to form. This could help detect the disease earlier using a technique already in practice.

When a doctor sees a suspicious mass in the pancreas, she can use ultrasound to guide a needle to the mass. A pathologist has to look at the extracted tissue and determine

"There are two completely different ways of doing science," Otey says. "Basic scientists mostly try to unravel underlying mechanisms." They want to understand how a disease works so doctors can know how to treat it. Clinical scientists care about mechanisms too. "But what they really care about," Otey says, "is does this information tell us a better way to diagnose or treat this disease? And they care about it **now.**"

if it's cancerous, a challenging prospect. Instead, the pathologist could just stain the tissue with an antibody for this isoform of palladin. Because the TAFs surround the tumor, the needle would collect some of these cells along with the cancer cells. This isoform of palladin is specific to TAFs, allowing the doctor to rule out other diseases that can be mistaken for pancreatic cancer.

It will take clinical trials to prove that this process will work with needle biopsies from patients, but Otey and Kim got close: they could detect this isoform of palladin in needle samples from tumors that Kim had removed from his patients. And anything that improves early detection of pancreatic cancer is a welcome development, especially for families waiting for a diagnosis.

A tale of two approaches

Otey says their work is a perfect example of the different approaches of basic scientists and clinicians. "There are two completely different ways of doing science," she says. "Basic scientists mostly try to unravel underlying mechanisms." They want to understand how a disease works so doctors can know how to treat it. Clinical scientists care about mechanisms too. "But what they really care about," Otey says, "is does this information tell us a better way to diagnose or treat this disease? And they care about it *now*."

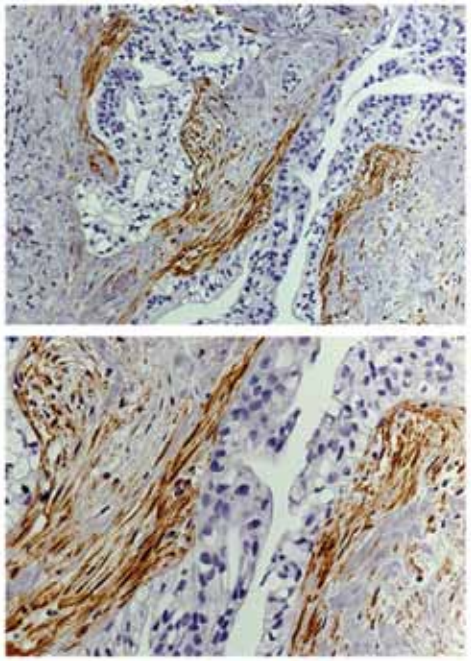
Otey and Kim may have found a new diagnostic tool for pancreatic cancer. But they also understand a little better how the disease works and possibly why it is so aggressive and so resistant to treatment. That's the power of collaboration. "It has shifted my priorities," Otey says. "I'm still a basic scientist and I still want to understand underlying mechanisms, but I'm constantly on the alert—is there something here that would help us immediately?"

Kim also makes it clear how much he depends on his collaborations. He spends one day a week seeing patients, and one or two days in the operating room. Whatever time is left is for research. That would never be enough to compete for grants and publications without collaborators such as Otey. But if Kim spent any less time in the operating room, he says, he would not be the kind of surgeon you would want operating on your family member. "You can't turn that on and off," he says.

Kim has helped expand their collaborations, including what he describes as "mini clinical trials" with human tumor samples grown in mice in the lab of fellow surgical oncologist Jen Jen Yeh. This could dramatically accelerate drug discovery.

But Kim and Otey are realistic about the pace of research. It's been over a decade since Otey discovered palladin and six years since that call from Brentnall sparked Otey's interest in pancreatic cancer.

In fact, they still don't know exactly why the palladin mutation in Family X caused pancreatic cancer. Otey thinks it may not be affecting the cell's architecture via actin, but palladin may be entering the nucleus and changing which genes are turned on. Otey and Kim will keep trying to figure out what palladin does and how pancreatic cancer works, but always with an eye to how they can help patients. *e*



Tissue from the pancreas of a patient with pancreatic cancer, viewed at 20x (top) and 40x magnification (bottom). Islands of cancer cells are surrounded by tumor-associated fibroblasts, which stain strongly for palladin (in brown). The fibroblasts respond to the cancer cells from the earliest stages of disease, making palladin an early indicator of pancreatic cancer. Image courtesy of Carol Otey.

Alex Raines is an MD/PhD student studying neurobiology.

Carol Otey is the interim chair of the Department of Cell and Molecular Physiology in the School of Medicine. Hong Jin Kim is an associate professor of surgery in the Division of Surgical Oncology. Otey and Brentnall published their discovery of the mutation carried by Family X in PLoS Medicine *in December 2006. Otey and Kim published their work on pancreatic cancer in April 2010 in* PLoS One, *and their work with breast cancer cells appeared in* Oncogene *in January 2009.*

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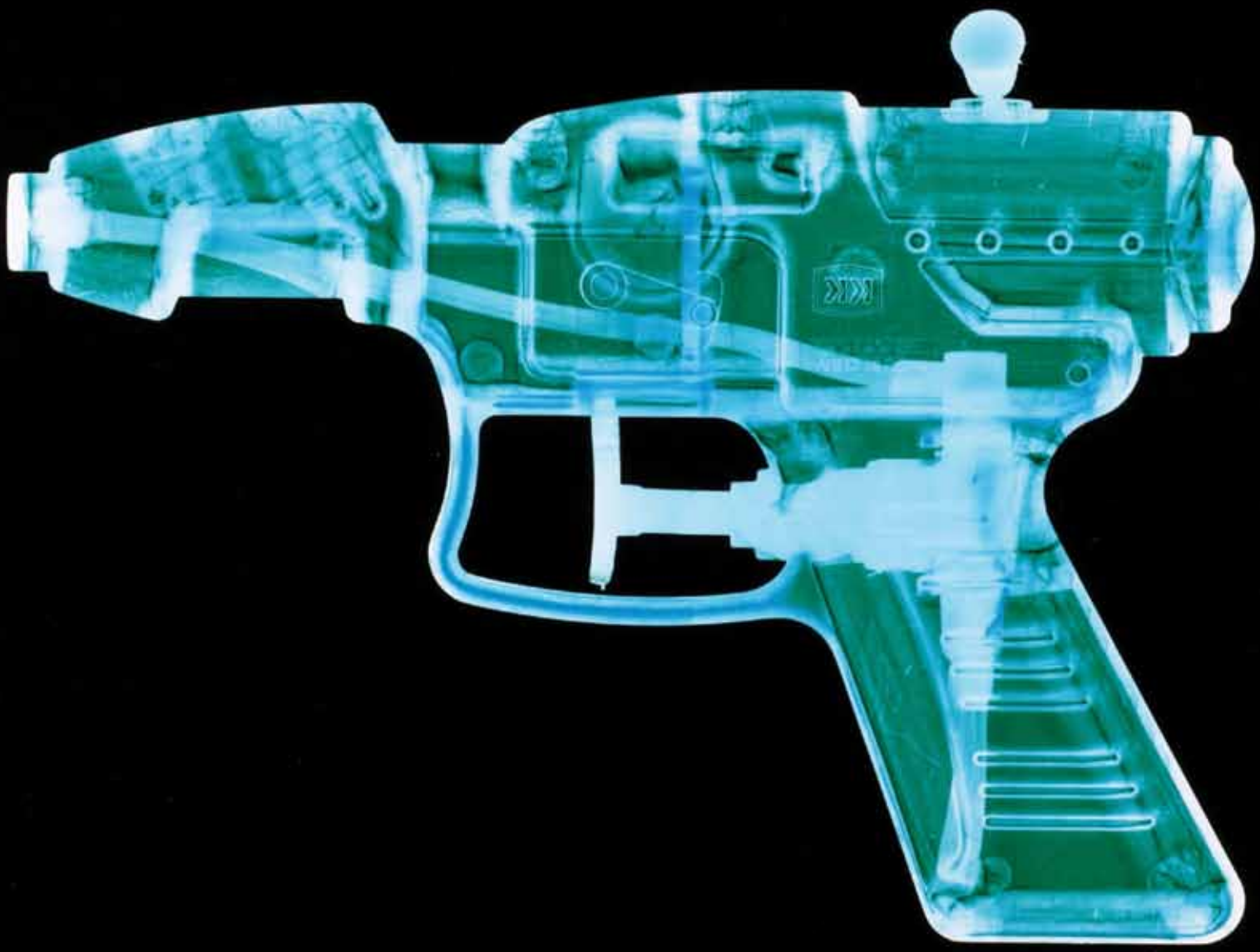


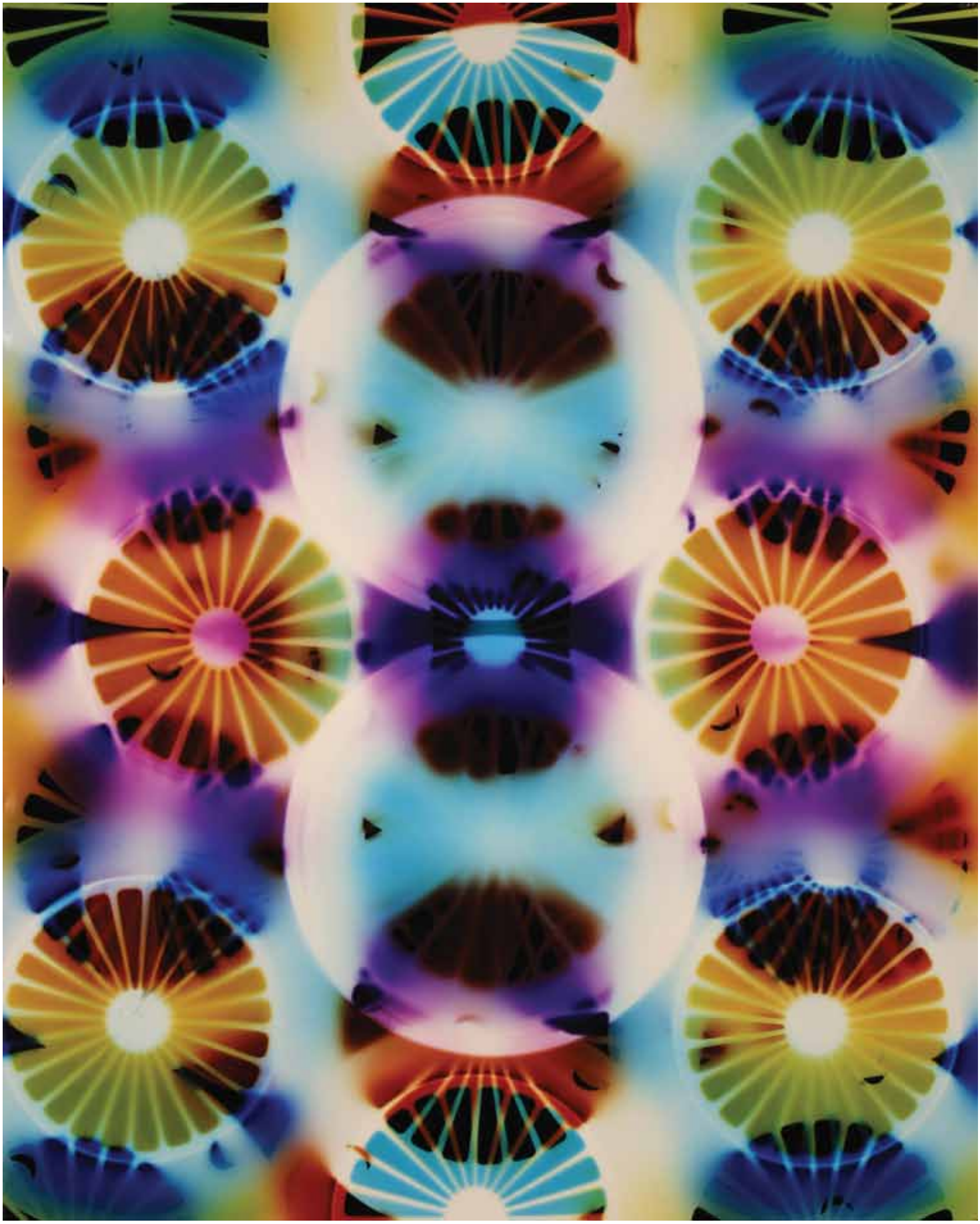
Plastique Fantastique

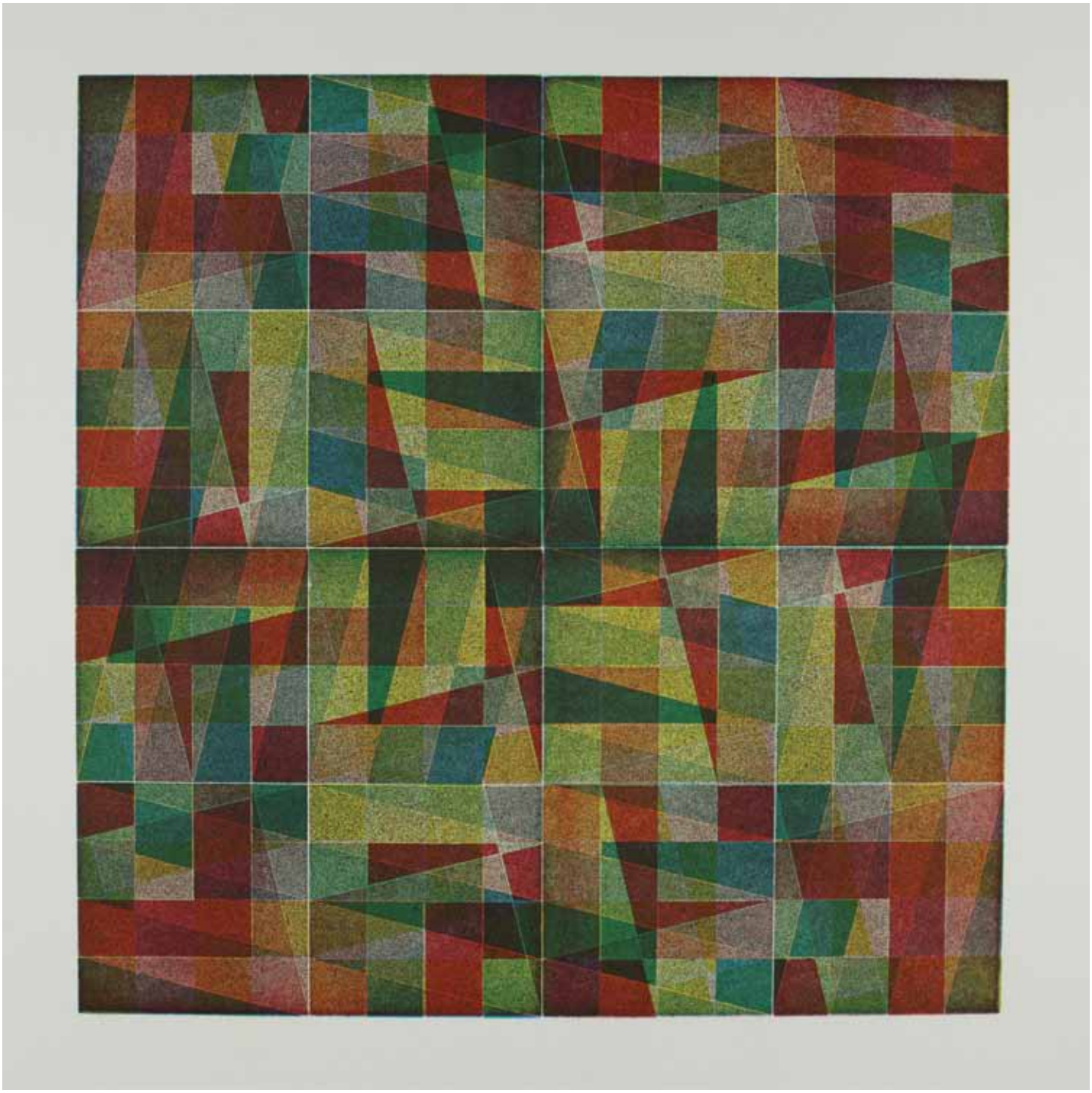
Photograms and prismatic prints from Mike Sonnichsen's visual laboratory

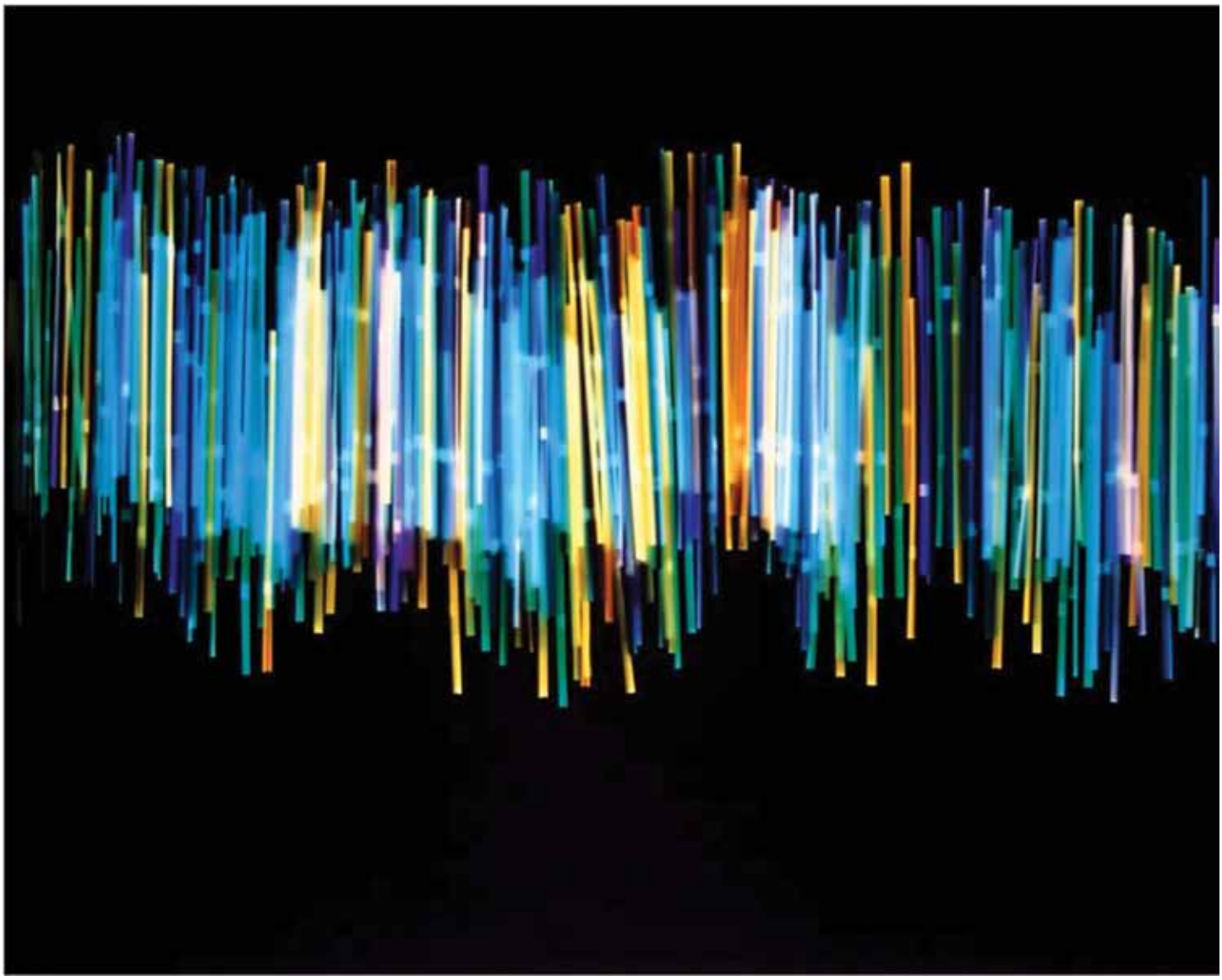
Left and above: Details from the *Watergun* series. These candy-colored plastic water guns make artist Mike Sonnichsen nostalgic for childhood toys and play. He finds them at thrift and dollar stores and brings them back to his studio to make photograms. Find out how he does it on the next page.

You can't always predict what you're going to end up with when you make a photogram, says artist Mike Sonnichsen. He uses a sheet of light-sensitive color photo paper, a darkroom, and translucent plastic objects from his peculiar collections. "The room is completely dark because the paper is sensitive to all light," he says. "You can't even use a safelight. So I set things up by feel. I can feel the edge of the paper and I can feel the objects that I'm assembling on the paper. Then I begin the exposure. When the light shines on the objects, the paper responds." What he gets are jewellike objects whose luminescent innards are suddenly visible. The paper prints the complementary, or opposite, color of the object, he says. A blue water gun materializes on the paper as a gold water gun. "These are objects from our world that we don't usually give a lot of thought to," Sonnichsen says, "even though some industrial designer created the form of the bottle and the spout, and determined what color the soap should be." He constantly asks himself: "Who designed this and why? Why are there so many different types, forms, and colors of, say, lids to go on liquid laundry detergent? Will they ever get recycled into something else? Do we need all this stuff?"

This page: Details from the *Dish Detergent Bottles* series. The liquid soap inside the bottles formed a crude sort of lens that bends the light and reveals strange reflections and hotspots, Sonnichsen says. **Opposite page:** "Untitled" from the *Paper Plate Holder Mandala* series.







Sonnichsen grew up in Puerto Rico and spent much of his childhood snorkeling. "There were brilliantly colored fish and living things in the reefs. There was all this sunlight shining down and optical brilliance from waves and light on the sea floor. It really snuck into my subconscious," he says. He uses only three colors in his relief prints (opposite page), a series he first started twenty years ago while in architecture school, but the etching technique he uses—aquatint—can create an array of vivid, watery hues. To make each print, Sonnichsen sprinkles acid-resistant dust onto a small metal plate. Then the plate goes into an acid bath. How light or dark certain areas will eventually print, he says, has to do with how long the plate stays in the acid bath. "You can get really light tones, or velvety gradations," he says. Then he inks the plate, prints onto paper by running the plate through a press, re-inks it with a different color, rotates the plate, and so on until new colors, order, and hidden geometries reveal themselves. "I can barely keep track of the complexity sometimes. It helps to be systematic and machinelike about it, though that's not how we usually think of artists. I love the way that both of these processes play out in surprising ways. The results are almost always beyond what I could have imagined."

—Margarite Nathe

Mike Sonnichsen is a lecturer and manager of the print and photo labs in the art department. See more at www.mikesonnichsen.com.

This page: "Short Straw Draw II." Plastic drinking straws.
Opposite page: Aquatint relief prints from the *8x8* series.

JASON SMITH

LEGISLATION BY DONATION?

In the lobbies of congress, money matters. But not as much as you might think.

by Mark Derewicz

"Ten people who speak make more noise than ten thousand who are silent."
—Napoleon Bonaparte

THEY'VE BEEN CALLED HIJACKERS of democracy, widely viewed as scoundrels who spend millions of dollars to curry favor with politicians while regular people go unheard outside of Election Day. In public opinion polls that rank honest professionals, lobbyists finish dead last—right behind car salesmen.

Each year, the antilobbying chorus gets louder, but each year lobbyists pack Washington, D.C.—there were 13,694 of them in 2009. Lobbyists are required to register with the federal government and release records that include how much money they spend: a record $3.5 billion in 2009, not including campaign donations. Most of that money is spent on salaries for lobbyists. They draft reports full of facts and opinions to support their arguments. They meet with politicians and other government officials. They create ad campaigns and spur public support for their causes. And, as the cliché goes, they wine and dine politicians, paying for junkets—including the occasional golf outing—across the country and around the world.

Most people agree there's a problem; the First Amendment right to petition the government shouldn't depend on cash flow. Even campaigning politicians often blame corruption on corporate slush funds. But Frank Baumgartner and four other political scientists found that almost every public policy debate can be boiled down to two sides, and the side with more lobbyists, more political action committee donations, and bigger budgets wins only half the time.

"It's not that money doesn't matter," Baumgartner says. Our political system is biased toward the wealthy, he says. But in the book *Lobbying and Policy Change: Who Wins, Who Loses, and Why*, Baumgartner's team reveals that there's a lot more to lobbying than just dollars.

"Lobbyists . . . woo lawmakers with facts."
—Jeffrey Birnbaum, lobbyist

You can't generalize about lobbying by reading newspapers, Baumgartner says. That's one lesson he learned from his team's research. The front page is filled with controversies and crises that journalists and scholars use for case studies about corruption. Think Jack Abramoff, the infamous lobbyist who defrauded his own clients. "But by definition those things are not normal," Baumgartner says. Or else they wouldn't be news.

To generalize about lobbying, Baumgartner's team randomly picked ninety-eight public policy issues for which there are many active lobbyists. Some issues, such as normalizing trade relations with China, involved thousands of people, including former President Bill Clinton. But many issues involved ten or fifteen people, some of whom were government officials. For each interest group—such as the U.S. Chamber of Commerce, Exxon Mobil, or the Sierra Club—researchers gathered all available public information, including annual corporate sales, number of employees, how many lobbyists the group had, and how many political action committee contributions it had made. Baumgartner's team, which included several graduate students, interviewed more than three hundred lobbyists, interest group advocates, government officials, and congressional chiefs of staff and aides who worked to change a policy or preserve the status quo between 1999 and 2002. The researchers studied twenty thousand lobbying reports.

"We did that for 2,221 major players in Washington," Baumgartner says. "Then we followed these issues for four years and asked, well, who won?" After analyzing the data for six more years, the researchers found that there was no correlation between money spent and which side won.

"We were all shocked," Baumgartner says. "Anyone who grew up in America would be shocked."

On average, each corporation, trade organization, or professional association spent two million dollars a year on lobbying between 1999 and 2002. That's five times as much as each citizen group and twice as much as each union. But there are many more corporations, trade groups, and professional associations; taken together they spent nineteen times as much as unions and twelve times as much as citizen groups. Those statistics don't include political action committee donations or groups with little lobbying power—college students, the poor, the uninsured. The vast difference in financial resources has helped shape portions of society through the decades.

The problem, Baumgartner says, is not that wealthy lobbyists pay to get policies in place that are advantageous to their industries; most major policies already favor rich corporations and major trade organizations. "The problem is the accumulation of bias already in the political system," he says. "Public officials are hearing a steady drumbeat of news and information that doesn't come from a random sample of citizens; it comes from the constitutional right to petition the government, which is used by professionals and corporations more than it's used or ever could be used by individual people."

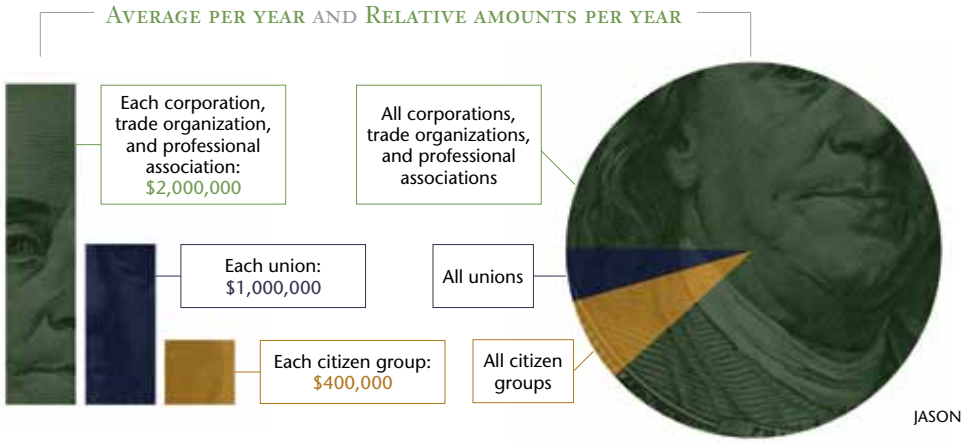
Still, there's this idea that lobbyists are all-powerful, that money *always* rules, he says. "And that's just not the case."

"You can have all the money in the world behind you. But if public perception cuts against you, it's an awful major hurdle to overcome."
—David Levinthal, Center for Responsive Politics

Baumgartner says that studying lobbying is like studying the stock market. One day a company's stock might fare well; another day its value might drop. But fluctuations occur for all kinds of reasons that don't speak to the stock's true value. Same with lobbying—sometimes wealthy lobbyists fare well and get their way in Congress, and sometimes they don't. But their stock is always high; their influence is always greatly felt.

Consider the oil lobby. Big Oil already has most of what it wants, including an economy based on its product. So it works

Spending on lobbying, 1999 to 2002



JASON SMITH

Some things money can't buy

hard to maintain the status quo. In 2009 oil companies spent $169 million on lobbying. Environmentalists spent $22 million. Yet the oil industry has always lost the battle to drill in Alaska's Arctic National Wildlife Refuge.

In 2010 President Barack Obama lifted the moratorium on drilling in federal waters. Oil companies rejoiced. Come summer, though, the BP oil spill forced the president to reinstate the moratorium, and few politicians were eager to give oil companies anything. Big Oil's stock was down. But the stock's value—the industry's overall influence—remained higher than that of all the alternative energy companies combined.

Baumgartner knew that politics and current events can hinder even the richest lobbyists. But he found another reason why the rich win only half the time: lobbyists have strange bedfellows. Coalitions of interest groups form on each side of just about every issue, and the rich ones don't always lobby with wealthy allies. Sometimes poor interest groups team up with a rich corporation. Sometimes a powerful trade organization supports a policy change for one reason while another trade group supports it for a different reason. And sometimes small but typically disparate interest groups band together to battle a huge trade organization.

Such was the case in 1999 when ACT UP—a tiny AIDS activist group with no lobbyists, no staff, and no money to give to politicians—squared off against the Pharmaceutical Research and Manufacturers of America (PhRMA).

ACT UP wanted Congress to allow companies to produce generic AIDS drugs for patients in Africa without paying high fees to the pharmaceutical companies that created the original drugs. PhRMA, which had constituents who would lose millions in profits if Congress passed such a provision, was by far the richest of its small coalition of lobbyists. ACT UP, meanwhile, called on hundreds of other interest groups and players in Washington, D.C.

"PhRMA still had more resources, but the discrepancy wasn't as big as you'd think," Baumgartner says. "And that's because ACT UP had powerful allies." Congress sided with ACT UP.

Baumgartner was surprised at how often diverse coalitions formed on two sides of even the most complicated issues. There might have been seven ways to address an issue, he says, but instead of each interest group arguing separately for specific policy changes, they often joined together to fight for policy change in a general direction. And Baumgartner points out that an issue that brings together, say, corporations may also unite environmentalists, citizen groups, labor unions, and government officials in opposition.

Because of this coalition building, only nineteen of the ninety-eight issues that the researchers studied were heavily skewed in terms of financial support and lobbying power. Maybe that's nineteen too many, but Baumgartner had predicted a much wider gulf between the haves and have-nots. The rich won most of those nineteen but not all of them. And Baumgartner's research shows that if given the choice, those richest lobbyists would trade their sizable advantages for the support of one particular man.

"The president is the people's lobbyist."
—Hubert Humphrey, former vice president

Trying to regulate lobbyists isn't the problem, Baumgartner says. "The problem is trying to educate politicians so they can recognize the difference between what they're hearing constantly from lobbyists and what's happening out there in society."

According to public opinion polls, the most pressing issue at the end of the Clinton administration was the state of the economy—the unemployment rate in particular. "But there weren't any lobbyists

lobbying on the issue of increasing jobs," Baumgartner says. "Even labor unions don't focus on that."

Of the ninety-eight issues in the study, seventeen heavily involved health-care lobbyists. "But they weren't lobbying to improve health care for you and me; they lobbied to protect the financial interests of professionals in the health-care industry," Baumgartner says. There's a huge disconnect between what the public needs and what lobbyists work for.

Still, following the richest lobbyists didn't help the researchers figure out which ones were most influential. But they found one person lobbyists always wanted on their side—the president. About 78 percent of the time, the lobbyists on the winning side shared the president's view. In fact, when party leaders didn't already share an interest group's view, that interest group was rarely successful in swaying politicians, according to Baumgartner.

"That's one of our nondepressing findings," he says. "Elections do matter." And lobbyists know this, which is why their money finds its way into our electoral process, too.

"The only way to stop the system of legalized bribery is to cut it off at its roots."
—Robert Reich, former secretary of labor

Baumgartner found that most lobbyists work tirelessly to maintain the status quo. And 60 percent of the time they're successful. As a result we see a lot of gridlock in Washington.

But 40 percent seems like a lot of policy change over the course of four years. "It *is* a lot," Baumgartner says. "And had we extended our study ten or twenty years, we might have seen a lot more."

Of the policies that changed between 1999 and 2002, seventy percent were substantial shifts, such as when President George W. Bush revoked President Clinton's ergonomics reform to improve workplace safety.

"Sometimes we think that people in Washington aren't doing anything, but that's not really true," Baumgartner says. In a typical two-year congressional term, eight thousand bills are introduced, and four hundred are passed. Between 1999 and 2002, bills addressing forty-seven of the study's ninety-eight issues never reached the floor of the House or Senate for a vote; "died in committee" is a common refrain on Capitol Hill. Many proposals never got any attention because there was no time—even issues that had no active opposition, such as a proposal to mandate hearing tests for all newborns. Advocates for that bill did not have much lobbying support, and Congress never voted on it.

This is the crux of the money problem: cash doesn't decide short-term policy outcomes, but you still need money to get on the policy agenda. You need money to make noise. Baumgartner doesn't think the government should limit lobbyist money. But he thinks the government can reduce the advantage that money gives lobbyists. The best big step, he says, would be election finance reform.

"Politicians are just constantly begging for money," Baumgartner says. "They have to raise tens of millions of dollars in small bits. It's humiliating. And at worst it's corrupt, because people will say, 'I'd be happy to give you money if you do *this* for me.' That's illegal." But it still happens, and not all corruption boils down to quid pro quo deals. Political action committees and lobbyists can bundle small donations to amplify an interest group's voice. "The system lends itself to extra access to the wealthiest among us," Baumgartner says. And politicians often ask donors not to give money to opponents. "So we end up with underfinanced challengers."

What about term limits? The idea is that if a senator only has two six-year terms, then the pursuit of a Senate seat would be viewed as a public service instead of a career choice dependent upon raising huge sums of cash. But Baumgartner thinks term limits would be a big mistake. "We'd have citizen legislators, but lobbyists are not citizen lobbyists," he says. "They're real pros. The learning curve on these issues is huge." He says Congress needs people who know policies inside and out, who were there when policies were created, who have experienced staffs.

Some critics say a system that allowed Senator Ted Kennedy to keep his seat for forty years is a system that's broken. Baumgartner, though, says that such long tenures are rare. And, more importantly to Baumgartner, Kennedy's staffers had become real health-care experts. "When his staff made a proposal it was taken seriously. They were as expert as insurance company lobbyists. They could go toe-to-toe. That's an important thing people don't think of."

If our government were full of fresh faces and naïve staffers, then our leaders would be even more susceptible to the arguments of lobbyists who, as Baumgartner says, will always have more information and money: "It would be like babes versus wolves."

THIS IS THE CRUX OF THE MONEY PROBLEM: cash doesn't decide short-term policy outcomes, but you still need money to get on the policy agenda. You need money to make noise.

Frank Baumgartner is the Richard J. Richardson Distinguished Professor of Political Science in the College of Arts and Sciences. The coauthors of Lobbying and Policy Change *are Jeffrey Berry from Tufts University, Marie Hojnacki from Penn State University, David Kimball from the University of Missouri-St. Louis, and Beth Leech from Rutgers University.* *They received funding from the National Science Foundation. You can see the full report on the web at lobby.la.psu.edu/.*

Two Carolina scientists want to diagnose diseases such as tuberculosis in places like remote African villages by having people

SPIT ON SEEDS.

Bill Gates gave them money. They have one year to figure out whether their idea is

SCIENCE FACT OR SCIENCE FICTION

BY BETH MOLE

Left: *Arabidopsis thaliana,* commonly called thale cress, was the first plant to have its genome sequenced. Vyas Sharma and David Lawrence want to turn *Arabidopsis* seeds into a kind of cheap, go-anywhere diagnostic kit by tricking the seeds into germinating only when they come into contact with an infected patient's saliva. Photo by Dra Schwartz. Seed illustration (above) by Robin Corp.

In 2009, the Bill and Melinda Gates Foundation challenged researchers to come up with new diagnostic tests for diseases that threaten global health. The proposed tests had to be cheap, simple, and fit for even the remotest village in Africa. The twenty-six winning ideas seem like something you might see on *House* if MacGyver were a new character. A kit that can separate out infected cells in blood samples using an old-fashioned eggbeater. Cell phones with built-in microscopes that identify parasites. And seeds engineered to germinate when spit on by patients who have tuberculosis.

The point of the Gates challenge is to put current scientific advances to work in developing countries. In developed countries, we kicked diseases such as malaria, tuberculosis, and polio decades ago, and any modern hospital could diagnose them with blood or molecular tests. But in countries where those diseases are still endemic, there may not be the equipment, facilities, or even electricity needed to run the tests.

Despite the need, the Gates challenge is still a gamble. Unlike most scientific grants, the challenge doesn't require calculated experiments or preliminary data to back up the winning ideas. But if the ideas are successful, they could have huge impacts on public health.

Vyas Sharma and David Lawrence, chemists who normally work on ways to track molecular activities in cancer, have already collected encouraging data for their diagnostic seeds.

Sharma's inspiration to use seeds came from the ancient Egyptians, who found out whether women were pregnant by having them "water"—or so it was translated from a recovered papyrus—bags of barley and wheat seed.

"It's the original pregnancy test," Sharma says. "They said they could predict whether the lady was going to have a child, as well as the sex of the offspring." If the wheat germinated, the baby was supposedly a girl; if barley, a boy. Scientists now believe elevated levels of estrogens in pregnant women's urine can induce germination, and modern trials found that the seed test accurately predicts whether a woman is pregnant about seventy percent of the time.

Seeds are cheap, easy to transport, and completely inert. "They're basically sleeping organisms," Lawrence says.

To figure out how to wake the seeds, Sharma started collaborating with Alan Jones, a biologist at Carolina who works with *Arabidopsis thaliana*, a mustard plant relative that's a good model organism for learning about growth regulation. Like all animals and plants, *A. thaliana* needs hormones that signal the seed to start growing. Usually the seed makes these hormones itself when conditions are right. But other researchers have engineered seeds that can't make hormones.

"These seeds won't germinate until we add a hormone," Sharma says. The goal is to have the seed release the hormone only when there's a specific cue, or signal, from a sick patient. Getting a signal from blood samples would require sterile needles and clinical training, which aren't always available in remote places. But getting a signal from saliva would be easy. Everyone could give it. Anyone could collect it. Anywhere.

Luckily, saliva contains the perfect signal of disease. "When people have infections they have very, very small quantities of DNA from microorganisms present in their saliva," Lawrence says.

Sharma and Lawrence first had to design a way to multiply the DNA from saliva to create a strong enough signal. They built on previously published methods for copying DNA at room temperature (no electricity needed).

But how could they link the signal to seed germination? Sharma and Lawrence had to come up with a couple of chemical tricks, the details of which are still under wraps. Basically, if there's no DNA from an infectious microorganism in the saliva, no copying will take place—a hormone won't be released and the seed will stay a seed. But if there is DNA from the microorganism in the saliva, the DNA will be copied, and a hormone will be released to kick off germination.

"Now, you're not going to wait ten years for a seed to sprout into an eight-foot tree," Lawrence says. But full germination of *A. thaliana*, which is relatively fast, still takes days. "The idea is to do the diagnosis in the field," Sharma says, "right in front of people."

To speed things up, Sharma and Lawrence decided to use seeds that are engineered to show when they're germinating. When the hormone tells the seed to start germinating, the seed will also start producing a protein that creates a color that can be seen by the naked eye within a few hours.

So far Sharma and Lawrence have some positive preliminary data that suggest the idea is workable. "We're a lot closer to it being science fact," Lawrence says.

They're trying to diagnose tuberculosis, a disease that kills more than a million people worldwide each year and spreads easily because most people don't know they're infected. If Sharma and Lawrence can get the idea working, they plan to move it to other so-called silent diseases, such as malaria. "Malaria can fester without any obvious signs for months and months," Lawrence says.

Sharma wants to go further and diagnose a whole set of diseases in a single go. "We could have an entire disease profile on a single plate," he says. He's even considered moving away from *A. thaliana* to plants that are native to each country in which the test could be used.

"If we get our idea to work, it could help a lot of suffering people," Sharma says. But he and Lawrence realize that the odds may be slim.

In May 2011 they'll apply for the Gates challenge's next round of funding: a million dollars to fund two years of work. The challenge money they initially won lasts for just a year and covers little more than one salary and some supplies.

"Now is like an exploration period for us," Sharma says. "We're trying to answer the questions, 'What's the right seed? How can we reduce the response time? How quickly can we get the amplification going? How do these three things tie together?'"

If they can get more preliminary data to back up the idea by May 2011, they'll have a shot at more funding for their diagnostic. That's not a lot of time to tweak the idea. But the Gates challenge is one of few ways to fund research on infectious diseases in developing countries. "There's a lot of suffering and risky ideas," Lawrence says.

If nothing else, Sharma hopes that the opportunity encourages researchers to start thinking about solutions, even if they sound at first like science fiction.

Beth Mole is a postdoctoral fellow in the medicinal chemistry and natural products division of the Eshelman School of Pharmacy. David Lawrence is the Selman Endowed Professor, and Vyas Sharma is a research assistant professor, both in the Eshelman School of Pharmacy.

The Making *of a* Beast

Wolf, hyena, hybrid—for centuries the French have argued about the identity of a creature that killed more than a hundred people in the southern countryside. But the real mystery is how the Beast of the Gévaudan was transformed from history into myth.

by Susan Hardy

The first victim of *la Bête* was a fourteen-year-old girl named Jeanne Boulet, killed in the summer of 1764 while she tended livestock near her village. Peasants back then knew that sheep and cattle sometimes drew hungry wolves and other predators, says historian Jay M. Smith. But a few weeks later, two more teenagers were killed the same way. A month after that, four people died, and word started to spread across France that a fearsome beast was on the loose in the hills of the small, south-central province called Gévaudan.

This went on for more than a year: people dying gruesome deaths, often out in the fields where there were no witnesses. Hunters failed to track the beast. The crown sent a military captain, a pair of professional wolf hunters, and finally the king's lieutenant of the hunt to catch the beast. Newspapers followed the story closely. By the time the attacks petered out in 1767, the beast had struck more than two hundred times, killing (and eating) more than half of its victims.

"Almost certainly, it was a wolf, or wolves," Smith says. "That's what the physical evidence overwhelmingly points to." On the shelf next to him is a thick volume; it catalogs the evidence for up to nine or ten thousand deaths by wolf attack in France between 1500 and the early nineteenth century. "The practice in France was to send women and adolescent children into the fields with the flocks and the herds," he says. "So there were many frail and isolated people, usually armed with nothing more than a staff, available for hungry wolves." The real question isn't what killed them, Smith says—it's why an episode that enthralled France for a year and even involved the crown has been shunted into the realm of folklore.

Today, the beast is known in France as a supposedly unsolved mystery. It's fodder for cryptozoologists and for the tourism industry. But as part of the country's history, the Beast of the Gévaudan might as well not exist. "At the time, the whole episode was densely documented," Smith says. "The people involved—the hunters who were sent to kill it, the administrators of the region—left extensive correspondence, and the newspapers were full of stories. But it's been almost entirely ignored by serious historians."

Smith is an expert on eighteenth-century France, and he'd been studying the period for nearly two decades when he first learned about what had happened in the Gévaudan. He was at the Bibliothèque nationale in Paris, looking for images for a book he was writing on French nobility, when he stumbled across engravings depicting the beast. "Most of them had detailed captions, so as I sat there I was able to piece together this story that had held the attention of France for a year," he says. "The images were so interesting that I was immediately drawn to the story—but I was also intrigued by my own ignorance, because why I'd never heard about this before was a mystery in itself." He checked with other American historians of France; they had never heard about the beast, either. So he decided to write a book—not just to give the story its proper place in history, but also to figure out why it wasn't already there.

If wolf attacks were so common in France, why would anyone have thought that the deaths in the Gévaudan were caused by anything more than ordinary, hungry wolves? From what Smith found, the episode has

La Bête du Gévaudan, watercolor by Johanna Öst, September 2010.

always been blamed—wrongly—on the superstitious peasants of the Gévaudan. As early as 1766, just months after the craze over the beast had died down, Enlightenment philosopher Immanuel Kant bemoaned how easily "common country folk" had led "a substantial number of intelligent men to take a common wolf for a hyena . . . in spite of the fact that any sensible person could see that there are not likely to be any African predators prowling around the forests of France." Over the next century, French intellectuals who wrote about the beast blamed irrational women and children for creating it. And the very few French historians who talked about the beast in the twentieth century described it as a magnification of peasant superstition.

That isn't what happened at all, Smith says. "First, some peasants even at the time did say it was a wolf, and you get that report in the newspapers. They say it resembles a wolf, or it's like a wolf but it has a longer tail, or something like that. Then you have these other reports that are more fantastic. It would have been easy enough, especially in a moment of terrible fright, to merge the characteristics of this large wolf with creatures that filled local lore, so there are some accounts that this may be a werewolf, or that witches are somehow involved."

As part of France's history, the Beast of the Gévaudan might as well not exist. "At the time, the whole episode was densely documented," Jay M. Smith says. "The people involved—the hunters who were sent to kill it, the administrators of the region—left extensive correspondence, and the newspapers were full of stories. But it's been almost entirely ignored by serious historians."

These more fantastic stories were a great opportunity for journalists who, Smith says, were looking for something gripping to fill pages that until the year before had been taken up with dramatic news of the Seven Years' War against Britain. Apart from war news, papers of the time mainly ran boring accounts of political happenings in the European capitals. One editor in particular, François Morénas of the *Courrier d'Avignon*, was trying to distinguish

his paper by breaking up the dry political news with human interest stories. "He saw early on that this story had real potential," Smith says, "and he did pretty sensationalistic reporting in the fall of 1764 that spread the word and that got the attention of other journalists." In those early months of the attacks, Morénas referred to the Gévaudan as "a theater of war," compared the beast to the giants and hydras of Greek myth, and spread the story that it might be a hyena.

Not long after, the crown got behind the idea that there was something strange about the situation in the Gévaudan. Louis XV had good reason to pay attention to the beast, Smith says. The king's reputation was in bad shape: he'd lost the Seven Years' War, he had levied too many taxes on the provinces, and he was known for wasting his time with hunting and womanizing. He saw an opportunity to appear concerned for his people and took it, sending hunters and offering a large bounty for the beast.

The leader of the first group of hunters, Jean-Baptiste Duhamel, had been a French captain in the Seven Years' War. During his time as a hunter of the beast he wrote constantly, Smith says, and he described the hunt for the Beast of the Gévaudan as a way to regain honor for himself and for the supreme commander during the war. He told the commander's secretary: "I sincerely hope that I will be able to announce in my next letter . . . that the monster is no more, and that it was the dragoon company of His Serene Highness that put it to death."

Smith says the king's hired hunter, Jean-Baptiste Duhamel, "perpetuated the idea that it was this particularly ghastly creature, to explain his failures and account for his own disappointments. He described the beast as this weird hybrid animal that was part horse, part lion, part hyena—jaws like a vice, six talons on its paws." But the key thing that Duhamel did to perpetuate the story of the beast was fail, obviously and publicly.

According to Smith, Duhamel did more than anyone else to spread the idea early on that the Beast of the Gévaudan was a fantastic creature. "He'd been commissioned to go find this thing, and for more than four months he was fighting through terrible conditions, braving the winter of the Gévaudan, and he was frustrated by this animal many times," Smith says. "There was one episode, he said, where he had the beast in his sights and was about to shoot him, and his men came up behind him on horseback and startled the beast, and he was unable to get off a shot. That was, as he put it, 'the most crushing disappointment of my life.'"

In the letters he wrote while leading the hunt, Duhamel really seems to have believed that the Beast of the Gévaudan was one creature, Smith says. "And he then perpetuated the idea that it was this particularly ghastly creature, to explain his failures and account for his own disappointments. He described the beast as this weird hybrid animal that was part horse, part lion, part hyena—jaws like a vice, six talons on its paws." But the key thing that Duhamel did to perpetuate the story of the beast was fail, obviously and publicly. In February 1765, Duhamel led a hunt by thousands of villagers, who marched through a winter storm only to find no trace of the beast. This image "impressed even the most jaded readers of news," Smith writes. Throughout France and beyond, people became hungry for any news from the Gévaudan.

Of course, if the beast could be used as a political tool, it could be used as a religious one as well. A powerful bishop in the area wrote to all his parishes warning that the beast was a monster "drawn from the arsenal of God's anger to execute the death sentences that his justice has pronounced." It would be easy now to dismiss the bishop as a superstitious crank, Smith says, but actually he was putting the beast to good use. The bishop belonged to a school of Catholic thought called Jansenism, which had been declared heretical by the Church because it was too close to Reformation ideas about human depravity and the grace of God. The bishop used the "scourge" of the beast as a pretext to set up special public prayers in the Jansenist style of worship.

Above left: "The furious beast that is supposed to be a hyena." The text tells of two peasants who were made into national heroes for fighting the beast—a twelve-year-old boy who led an attack on the creature on January 12, 1765, and a mother who managed to wrest her six-year-old son, still living, away from the beast on March 12, 1765. (The child later died of his injuries.) **Above right:** This drawing depicts the killing of a large wolf that representatives of the crown declared was the Beast of the Gévaudan. Images: National Library of France

Eventually, the beast started to outlive its usefulness, at least in the eyes of Louis XV. "The crown began to worry about the embarrassment that was being caused by this ongoing crisis that it was unable to solve," Smith says. "From January through August, the crown had become very visibly engaged in the hunt, and they hadn't been able to bring the tragedy to an end." After Duhamel, another pair of hunters failed. That fall, more than a year after the attacks in the Gévaudan had begun, a fourth hunter sent by Louis XV killed an especially large wolf. Several victims confirmed that it had been their attacker—they were probably under a good bit of pressure to say what the king's representative wanted to hear, Smith says. And so the animal was embalmed and sent to Versailles to be paraded before the king, even though, a civil servant later said, the surgeon who had examined the beast "saw nothing indicating that this animal had eaten human flesh." In Versailles, "the recorded reactions to the show suggest that the beast fell short of expectations," Smith writes. "Its appearance failed to dazzle."

The crown used the wolf's ordinary appearance to bring the saga of the beast to an end. The official paper *Gazette de France* stated: "The most experienced hunters have concluded that it was a true wolf that boasted nothing extraordinary, neither in its size nor in its composition." After that, the paper stopped reporting on the attacks in the Gévaudan, which continued off and on for two more years. The other papers eventually either followed the *Gazette's* lead, Smith says, or just decided that the public was tired of the story.

The beast "prefers to attack women and children," drinks their blood, and cuts off their heads, warns this early 1765 drawing. A reward of 2,700 livres is offered for the creature's death.

Fear of shame was a powerful motive for French intellectuals, Smith says. "They had either participated in the frenzy surrounding the beast or lent silent support by looking on and not offering skeptical words. Then they were embarrassed after it sank in that the killings probably resulted from wolf attacks."

Fear of shame was a powerful motive for French intellectuals, Smith says. "They had either participated in the frenzy surrounding the beast or lent silent support by looking on and not offering skeptical words. Then they were embarrassed after it sank in that the killings probably resulted from wolf attacks." Intellectuals in the following decades claimed that rumors from the villages had been to blame, but Smith found that the intellectuals themselves had helped create the climate that made a monster seem like a plausible theory. "Speculation about freakish or preposterous products of nature took place not on the margins of scientific inquiry but at its very center," Smith writes. "From the late seventeenth century through the 1730s, the French Academy of Sciences showed an almost obsessive interest in monsters, those creatures defined as 'contrary to the order of nature.'"

And the theory that the beast was an African hyena? Smith thinks that it probably came from a bestselling encyclopedia of natural history that was circulating around France at the time; physical descriptions of the beast-as-hyena read like those from the book. The author's summary of hyena behavior probably encouraged this interpretation: hyenas were "extremely ferocious," fond of digging up graves and breaking down stable doors and fences, and sometimes attacked men.

Having already been reinterpreted as a peasant fantasy, the real story of the beast couldn't survive the nineteenth-century French emphasis on reason and progress. It might have disappeared from academic writing altogether, Smith says, except that late in the nineteenth century France started to idealize its preindustrial past. "Folklore emerges as an academic discipline then, and there's this new interest in rural culture and peasants—the dress they wore, the farm implements they used, the stories they told, the crazy beliefs they had," Smith says. "So the Beast of the Gévaudan, even though it's a fairly complicated story, becomes just one more remnant of an archaic French past that doesn't require much explaining, because it's just an emanation of peasant superstition."

Smith's book, *Monsters of the Gévaudan*, will be coming out in early 2011. A historian friend who is French has warned him that French academics may be skeptical. To talk about the beast is to risk being grouped together with people who speculate that it was a human-trained, wolf-dog hybrid—or perhaps a Mesonychid, a wolflike species that's been extinct for millions of years. Pretty much everyone agrees that the last thing the story of the beast needs is another theory.

Unless, maybe, it's the right one. e

Jay M. Smith is the John Van Seters Distinguished Term Professor of History in the College of Arts and Sciences. His book, Monsters of the Gévaudan: The Making of a Beast, *is being published by Harvard University Press.*

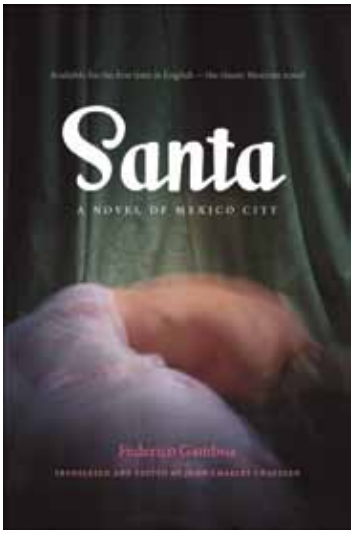
in print

LITERARY NATURALISM

A south-of-the-border classic, now in English

by Margarite Nathe

Santa: A Novel of Mexico City. By Federico Gamboa (translated by John Charles Chasteen). UNC Press, 238 pages, $22.95.



UNIVERSITY OF NORTH CAROLINA PRESS

Santa, says historian John Chasteen, is sort of like Mexico's *Huckleberry Finn.* Most Mexicans who haven't even read the book know the story. It's been made into four movies (including Mexico's first nonsilent film), stage plays, a hit song, a telenovela, and even a pornographic comic. The novel's main character is a prostitute named Santa.

Everyone who meets Santa—"saint," in Spanish—is enchanted by her beauty and sweetness. (The author Federico Gamboa is no exception, considering the number of paragraphs devoted to her "lovely body," her "provocative beauty," her "perpetual, shameless nudity.") But she has a tragic story. Her family threw her out. The sleazy police officer she fell in love with abandoned her. She was forced from her idyllic, churchgoing life in the country into a brothel in Mexico City and, eventually, to worse. One historian found that young Mexican prostitutes in the 1930s often compared their own life stories to Santa's, Chasteen tells us in the introduction to his new translation.

Chasteen first read Gamboa's novel many years ago and was amazed, first of all, that it was so racy, but also that no English version had ever been published. Although Chasteen's work usually involves writing academic books and articles about Latin America, he moonlighted for a year to produce the first-ever translation of the novel from Spanish into English.

"Translation is not like other writing that I do," Chasteen says. "With translation, you don't have to decide what to say. You have the more technical problem of deciding how to say it. And it's your responsibility to communicate as many levels of meaning as possible." That includes vocabulary, he says, but also tone and cultural interpretation. In the case of *Santa*, it sometimes meant knowing when to let go. For instance, certain types of plants or garments don't appear in the English version, he says, "because it would take a paragraph to describe an article of clothing that they simply have a name for."

Santa can be a real tearjerker, Chasteen says, but it also tells us a lot about Mexico City at the turn of the twentieth century. Gamboa, an upper-class Mexican gentleman, published the novel in 1903. It was so popular, Chasteen says, that when Gamboa ran for president ten years later as a member of the Catholic Party, *Santa* was the reason for a great number of his votes. (He didn't win.) And because Gamboa was part of a group called the literary naturalists—writers who thought of themselves as scientific observers of human society—*Santa* gives us thousands of details of everyday life in a Mexican metropolis.

Gamboa takes us all over the city with Santa, through ritzy restaurants and hideous dives, where walls are held together by "bits of wire, wood, and greasy cord." We sit with Santa and her coworkers through their miserable medical checks at the health department. We hear every detail of the hospitals, police departments, and courtrooms she visits. We find out what prostitutes' bedrooms looked like inside the most chic and the shabbiest brothels in Mexico City.

Literary naturalists, Chasteen says, "became historians for people without a history." It's thanks to Gamboa that we know how life was in that time and place for women like Santa, women Gamboa described as "a legion of slaves, teeming, never ending."

John Charles Chasteen is a professor of history in the College of Arts and Sciences.

Mexico City circa 1890, as it appeared in the book *A Photographic Trip Around the World* (1892). "Perhaps the streets of no other city present so diversified a picture as those of the city of Mexico," read the original caption. "Every variety of costume, civil and religious, Indian and European, of the city and country, is intermingled in the crowd. The native Mexicans, men and women, are easily distinguished by their garments, which are of the lightest description. The streets, in the early morning, are overrun with peddlers, whose cries are peculiarly discordant. In the afternoon the fashionables resort to the parks, which are in the best part of the city. The avenue in the photograph represents one of the most fashionable thoroughfares."

BOOKISH VERSE

Poems from the stacks

by Susan Hardy

The Librarian and Other Poems. By Ruth Moose. Main Street Rag Publishing Company, 85 pages, $14.00.

Ruth Moose was once a librarian—years ago at Pfeiffer University, a small school near Charlotte, North Carolina. "But I was a bad librarian," she says. "I'd get too involved. Students would come in to do research, and I'd get too excited about their papers. I'd end up going to class to hear them present."

Clearly she belonged in the classroom rather than the stacks, so Moose left the library to teach creative writing at UNC. She is not the character who narrates her collection of poems, *The Librarian*, she says. That librarian is a younger, single woman; Moose was married for more than forty years and raised two sons. The Librarian is a cat person, finding dogs too fawning and needy ("I had two dogs when I wrote that," Moose says).

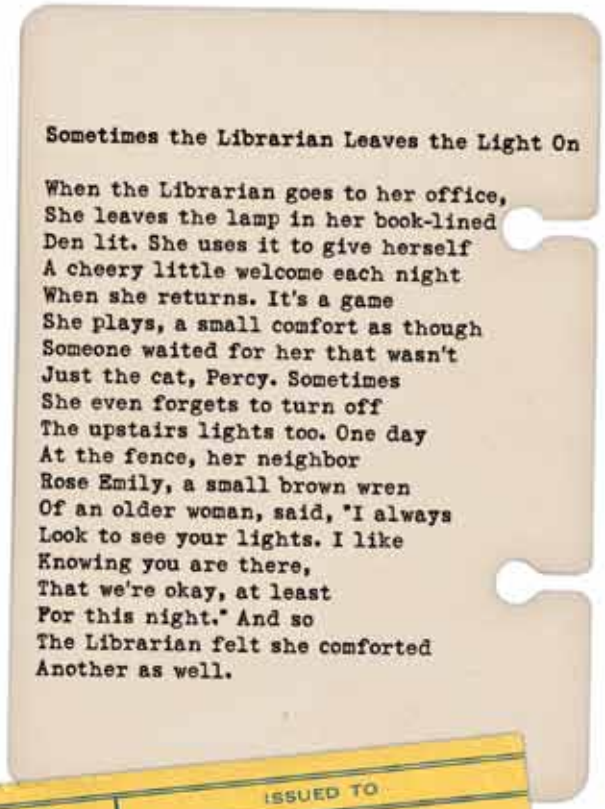
But like Moose at the time she wrote the poems, the Librarian is in mourning—over HWLWG (He Who Left Without Goodbye). Moose says she may have come up with the Librarian to help her cope with her own grief in the years after her husband's death. When Moose showed a few of the poems to her writers' group, they demanded to hear more about this unorthodox librarian—a Jane Austen fan who listens to country music, a bookworm who loves her Jim Beam, a woman who disdains jewelry but loves lace.

"I don't know this person," Moose says. "I don't know where she came from. But people—all different kinds of people—have told me that she's someone they can relate to."

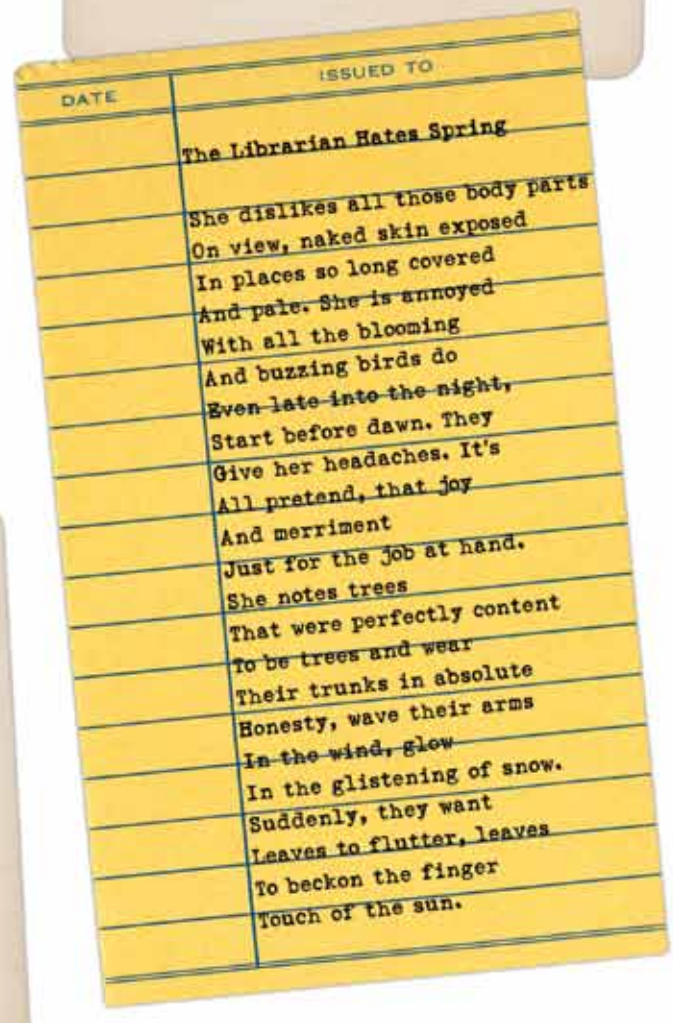
Ruth Moose is a lecturer in the Department of English and Comparative Literature in the College of Arts and Sciences. Her latest collection of poetry, TEA and Other Assorted Poems, *was published in December 2010 by Main Street Rag.*


The Librarian Loves Men
In Seersucker Suits

The kind her father wore summers
Sometimes with a white hat.
He was a good guy. The kind
The Scholar wears when he comes
And she hangs his coat
On her tall hall tree. Then,
She pauses a moment, takes
A sniff of his sweet scent
Still in the round damp
Ring of his collar.


Sometimes the Librarian Leaves the Light On

When the Librarian goes to her office,
She leaves the lamp in her book-lined
Den lit. She uses it to give herself
A cheery little welcome each night
When she returns. It's a game
She plays, a small comfort as though
Someone waited for her that wasn't
Just the cat, Percy. Sometimes
She even forgets to turn off
The upstairs lights too. One day
At the fence, her neighbor
Rose Emily, a small brown wren
Of an older woman, said, "I always
Look to see your lights. I like
Knowing you are there,
That we're okay, at least
For this night." And so
The Librarian felt she comforted
Another as well.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The Librarian Hates Spring

She dislikes all those body parts
On view, naked skin exposed
In places so long covered
And pale. She is annoyed
With all the blooming
And buzzing birds do
Even late into the night,
Start before dawn. They
Give her headaches. It's
All pretend, that joy
And merriment
Just for the job at hand.
She notes trees
That were perfectly content
To be trees and wear
Their trunks in absolute
Honesty, wave their arms
In the wind, glow
In the glistening of snow.
Suddenly, they want
Leaves to flutter, leaves
To beckon the finger
Touch of the sun.

Poems reprinted with permission from *The Librarian and Other Poems* by Ruth Moose, Main Street Rag Publishing Company, 2009. Images: Marie-France Bélanger (catalog card) and Jamie Carroll (check-out card)

GRAZVYDAS

Test your nutrition cognition

Q: What can turmeric do for you?
A: Tea made from turmeric is thought to lubricate the joints and help relieve pain from arthritis, bursitis, and tendonitis, and other chronic joint pain.

Q: Why should you avoid talking on the phone in the kitchen?
A: It leads to unintentional, mindless grazing.

Q: What is molybdenum?
A: It's an essential trace element that's necessary for keeping several enzymes active, including one that's involved in your body's production of protein and genetic material.

Q: If weight-bearing exercise is supposed to be good for bone density, why does carrying extra pounds of fat lead to lower bone density and a weaker skeleton?
A: Adipose tissue (fat) produces toxic substances that impair our ability to maintain strong bones. Excess fat also causes an inflammatory response and multiplies osteoclasts, or cells that break down bones. Extra muscle weight, on the other hand, won't cause this.

Q: What does vitamin B5 do?
A: It helps regulate the stress hormone cortisol. If you don't get enough B5, you may have headaches, fatigue, and insomnia. Find it in mushrooms, liver, wild salmon, lentils, cereal, and turkey breast.

NUTRITION

Hungry for the good stuff

by Margarite Nathe

The Dole Nutrition Handbook: What to eat and how to live for a longer, healthier life. By the Dole Nutrition Institute. Rodale Books, 352 pages, $19.95.

The Dole Nutrition Handbook is based on the idea that many of us overeat because we are undernourished. We tend to eat the same things over and over again, the nutritionists say. We take in all the calories without getting the variety of nutrients that could keep our bodies humming along, say, well past the one-hundred-year mark.

Can you eat pineapple to heal wounds? Collard greens for your bones? Bananas to make baby boys?

Page through the handbook and you can learn all kinds of interesting things. It's stuffed with food profiles, recipes, basic exercise programs, and interviews with nutrition scientists. According to nutritionist Steven Zeisel, if you eat a little extra choline around twenty-five weeks into pregnancy, your baby's memory will improve 30 percent—an improvement that will last for the rest of your child's life. (Find choline in eggs, cauliflower, peanuts, potatoes, oranges, wild salmon, wheat germ, beef liver, and cod.)

But can't you just take a supplement? No, nutritionists say: supplements and multivitamins don't give us the same benefits that come from whole foods. The nutrients in a serving of broccoli, for example, interact with and enhance one another to give an amazingly efficient boost to bone health. Broccoli is packed not only with calcium but with other bone-strengthening minerals such as potassium, folate, and vitamins C, D, and K. (These are also in kale, spinach, collard greens, chicory, kiwifruit, and asparagus.)

According to nutritionist John Anderson, whose research on bone health is also featured in the book, bone cells and tissues use calcium, phosphorous, magnesium, potassium, zinc, omega-3 polyunsaturated fatty acids, and vitamins D, B, and C to keep the skeleton strong. Luckily for us, Anderson says, we can get almost all of these nutrients in a single serving of fruits, vegetables, whole grains, or nuts. Processed foods, on the other hand, are often too heavy on the phosphorous. And too much of that leads to bone breakdown, he says.

Make sure to check out the section on how pineapple can help heal wounds. And the part about how quercetin-rich berries can boost your immunity. And that old myth that bananas can lead moms to have baby boys? Find out if it's true on page 177.

Steven Zeisel is director of UNC's Nutrition Research Institute, part of the Dole Nutrition Institute's North Carolina Research Campus; Zeisel helped in the preproduction of the book and reviewed its contents for scientific accuracy. John Anderson is an adjunct professor of nutrition in UNC's Gillings School of Global Public Health. The Dole Nutrition Handbook *also features several researchers from UNC-Greensboro, North Carolina State University, and Appalachian State University.*

endview

Above: In a migrant village in Beijing, a child plays in a puddle formed by recent rains. His brother rides down the road on his way back home from working construction in the city. **Below:** A young girl at Hope Primary school. Photos by Wyatt Bruton.

TRAVEL JOURNAL

Wyatt Bruton was already in love with Beijing when his Chinese teacher took him to a part of the city that many tourists don't see. "After driving down a wide street lined with skyscrapers and luxury apartments," Bruton says, "our bus turned onto a barely noticeable gravel road, and we entered a dramatically different world that would change my life." Many Chinese move to Beijing to work. They live in small migrant villages outside the city and work in the inner city, often leaving their children behind for the day. Because the children don't formally live in Beijing, they typically can't attend public schools. Bruton discovered Hope Primary, a makeshift school for village children started by a former Beijing schoolteacher. "I spent almost every Saturday with the children at Hope Primary," he says, "as we practiced English, played games and simply experienced life together."

Wyatt Bruton is a senior majoring in journalism and mass communication and minoring in entrepreneurship. He studied in Beijing as part of the Phillips Ambassadors Program.

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endeavors

The Peutinger map is the only surviving map from the Roman Empire. Story on page 14. Image: Austrian National Library