HARNESSING THE POLIOVIRUS
Scientists work to understand the powerful immunotherapy they have created.

SURVIVORS
Stories of hope, grit, and joy — a photo essay from Duke photographer Jared Lazarus

ONLY HUMAN
An antibody derived from lung cancer patients shows promise as a new treatment.

Help Us Rewrite the Story of Cancer

As you are probably well aware, cancer patients and their doctors have a great need for new therapies that are both more effective and less toxic. That's why immunotherapies are so promising; they have the potential to accomplish both of those goals. Rather than indiscriminately killing cells, they help the body's own immune system recognize and kill cancer cells, without the problematic toxicities to normal cells. If cancer treatments could do that consistently, with every patient, we could rewrite the story of cancer.

In this issue of our newly redesigned newsletter, Breakthroughs, you will read about several efforts from Duke scientists working to do just that. These stories—about Dr. Ned Patz's antibody therapy and Dr. Matthias Gromeier's modified poxvirus immunotherapy—don't have final endings yet. Not every promising new treatment makes it to market. For those that do, it takes a long time. Historically, it takes 13 to 16 years on average to bring a new drug from laboratory discovery to final approval.

At Duke Cancer Institute (DCI), our team of physicians and scientists have the tenacity to pursue their best ideas for new treatments, even if it takes decades. They are committed to seeing the story through to the end, and DCI is here to help them do that. Developing a new treatment is not only time-consuming, but also costly. It costs about $1.8 billion to bring a new drug to market, and those development costs are typically passed on to patients. In this issue of the newsletter, you will also learn about one of our researchers who is at the forefront of national conversations about the need to reduce the challenging cost of cancer care.

None of this work at DCI would be possible without our friends, volunteers, and supporters. Please join us in our efforts. Your support can help bring more promising new therapies to the happy conclusions that we all want to see.

"If cancer treatments could do that consistently, with every patient, we could rewrite the story of cancer."

Michael B. Kastan, MD, PhD

Women with BRCA1 Gene Mutation at Higher Risk of Aggressive Uterine Cancer

It's well known that the BRCA1 gene mutation dramatically increases risk of breast and ovarian cancers. Now a study led by a Duke Cancer Institute researcher suggests that women with the mutation are at higher risk for a deadly form of uterine cancer. Researchers from 10 institutions, led by Noah Kauff, MD, director of the Clinical Cancer Genetics Program at Duke Cancer Institute, analyzed data from more than 1,000 women with the BRCA1 and BRCA2 gene mutations. Out of 600 women with the BRCA1 mutation, 4 got an aggressive form of uterine cancer called serous endometrial cancer. That rate may seem low, but it's actually very high compared to the expected incidence of this cancer among the general population. "Even if we followed these women for 25 years, you would only expect to see no more than one serous cancer," Kauff says in a news release. Kauff points out that the findings are especially important because of how lethal serous endometrial cancer is; it has a mortality rate of 50 percent. "Our findings suggest that it may be important for women with BRCA1 mutations to consider removing their uterus at the time they are considering removing their ovaries and fallopian tubes, unless they are hoping to still have children using assisted reproductive methods or have other medical reasons," Kauff says.

The study was published online in the journal JAMA Oncology.
Almost 20 years ago, Matthias Gromeier, MD, published the idea that scientists could tame the deadly poliovirus and use it to kill brain tumors. It started with the observation that cancer cells have a receptor that the poliovirus fits into, like a key into a lock. "There were several of us who saw this at the same time. I was one of them," Gromeier says.

But Gromeier has been the one to stick with this notion. Some people would consider it outrageous. Inject a virus that can cause a paralyzing disease into someone’s brain? But Gromeier believed he could engineer a way to make the poliovirus harmless—except to cancer cells. He did that, and showed that it worked in mice. (See "How the Poliovirus Works," page 08). Then came many more years of tests in mice and primates, to prove that the modified virus couldn’t cause polio. Finally, in 2012, the Duke researchers were allowed to test the treatment for the first time in humans with glioblastoma, an aggressive brain cancer.

A BREAKTHROUGH?

In May 2016, because of promising early results from those tests, the Food and Drug Administration (FDA) designated the poliovirus as a "breakthrough therapy.

The breakthrough status means that the FDA sees evidence that the treatment may work better than current treatments for brain cancer. The FDA will give the therapy expeditious review and close attention to help ensure its progress. But it's not a guarantee that the agency will ultimately approve it for general use as a treatment for brain tumors. "This is a very challenging drug because this is a virus that replicates, infects and kills tumor cells, and that can infect certain immune cells," Gromeier says. "We know it can work. But we have to find out how we can make it work for everybody."

LESS IS MORE

In the first human trials, a few patients responded dramatically. Three appear to be cancer free, including Stephanie Lipscomb. She was in college when she received the treatment and is now working as a cancer nurse (see "Survivors," photo essay, page 10).

But some other patients, particularly those who received higher doses of the treatment, had debilitating inflammatory reactions. Nancy Justice, a wife and a mother of two sons, was one of...
Nair has begun testing the poliovirus in mice with breast cancer. Just seven days after she injects the poliovirus into the tumor, she sees an increase in immune-system activity. “After we administer the poliovirus, we are seeing immune cells—T cells—coming to the tumor,” Nair says. “This is not in one tumor model, but in three models.” These and other early animal studies also suggest that the treatment may kill not only brain tumors, but other types of cancer, too.

**TAking it Slow**

The possibility of treating other cancers with poliovirus is exciting, but the Duke team is proceeding cautiously. With a $2.5 million grant from the Department of Defense, Nair plans to conduct two to three years of mouse studies. If all goes well, at the end of that time, the team will conduct a pilot tissue study in six women with breast cancer. The women will receive standard of care—chemotherapy plus surgery. Two weeks before surgery, they will get an injection of the poliovirus directly into their tumors. After the tumors are removed, Nair will compare the tissue to a biopsy taken before the poliovirus injection.

Nair will look for changes in immune system cells and in genes linked to inflammation and immune responses. Those findings should help those patients. She responded well to the treatment at first, then, after a series of setbacks and improvements, declined and passed away from the brain tumor in April 2016.

Patients like Justice taught the researchers that controlling inflammation, by lowering the dose of the treatment and possibly by adding other medications, will be crucial.

“We are still puzzled by the fact that we have the best responses in the patients who got the least amount of virus,” Gromeier says. The less-is-more effect may happen because the poliovirus works mostly by unleashing the body’s powerful immune defenses.

“I think of cancer as a result of a failing immune system,” says Smita Nair, PhD, a Duke immunologist who is studying the poliovirus. “A tumor has some sort of marker on it—a protein or an antigen—that the immune system can see.” But cancer finds a way to hide. “With the poliovirus,” Nair says, “the idea is that it will kill the cancer cells, and at the same time, it will release the tumor antigen and cause inflammation that will hopefully drive an immune response.”

<table>
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<tr>
<th>Breakthroughs</th>
<th>1994</th>
<th>Matthias Gromeier modifies the poliovirus by replacing a segment of the virus’s RNA with a piece of human cold virus. In mice, he demonstrates that the modified virus can’t cause polio.</th>
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<td>1996</td>
<td>Gromeier and colleagues publish their findings.</td>
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<td>1999</td>
<td>Gromeier and colleagues of the FDA show in an animal study that the modified poliovirus can be safely administered in primates without causing polio.</td>
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<td>2000</td>
<td>At Duke, Gromeier shows that, in mice, the modified poliovirus locks onto brain tumor cells and kills them, and that it can grow and spread in human brain tumor cells in culture. For the first time, he publishes the idea that the poliovirus could be used as a brain tumor treatment.</td>
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<td>2001</td>
<td>The Rapid Access to Intervention Development (RAID) program of the National Cancer Institute begins performing screening, manufacturing, and regulatory work to support development of the poliovirus treatment.</td>
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<td>2012</td>
<td>Out of 24 patients: 11 are doing well with no disease progression; of these, there are 3 long-term survivors (22 months or longer after treatment). 2 have no evidence of disease.</td>
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<td>2016</td>
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the researchers design the first clinical trials of the poliovirus in women with breast cancer. Gromeier says, “We have to fully understand how our strategy works before we can move into cancers that occur outside the brain. Moving too fast with clinical trials that are poorly conceived can damage the prospects of a new, promising therapy.”

BEYOND DUKE

Meanwhile, trials in brain cancer patients continue. A new trial at Duke for children with brain tumors is planned and is awaiting final approval.

As part of the breakthrough therapy designation, studies for adult brain cancer patients will open at a handful of other cancer centers outside of Duke, though Gromeier can’t say exactly when. Those multi-center trials will likely combine the poliovirus treatment with chemotherapy, thanks to patients like Brendan Steele. As CBS News program 60 Minutes reported, Steele’s tumor initially grew larger after the poliovirus injection. He was given chemotherapy in hopes of gaining a few more weeks. Soon, his tumor was shrinking. After a second dose of chemotherapy and a few more months, it had disappeared. Gromeier was excited but puzzled. “Scientifically, no one really knows what’s going on,” he says. “But it’s so attractive because these chemotherapy drugs have been used for decades. We have other patients, not just Mr. Steele, who are responding very well to the combination of the poliovirus with chemotherapy. We are performing laboratory studies combining our virus with traditional chemotherapy to identify the mechanisms at work.” Today, Steele is doing well three years after first receiving the poliovirus.

"MY GOAL IS THAT NO CHILD WITH LEUKEMIA WILL HAVE TO GO THROUGH CHEMOTHERAPY"

Tom Tedder

"SOME B CELLS DAMAGE NORMAL CELLS AND CAUSE PROBLEMS."

Stefanie Sarantopoulos

"WE ARE PERFORMING LABORATORY STUDIES COMBATING OUR VIRUS WITH TRADITIONAL CHEMOTHERAPY TO IDENTIFY THE MECHANISMS AT WORK."

Matthias Gromeier

THE MANY SIDES OF B CELLS

Immune-system cells called B cells can either ramp up the body’s immune defenses or calm them down. Duke researchers are using them to fight cancer and autoimmune disease.

TARGETING CHILDHOOD CANCERS

The technology: MEDI-551 (Inebilizumab, Medimmune/AstraZeneca)

The promise: A gentler, more specific treatment for childhood leukemia and lymphoma. Duke’s Tom Tedder, PhD, whose team originally developed the drug that forms MEDI-551, aims to use it to selectively wipe out the majority of leukemia cells, then follow up with a second immunotherapy treatment.

“My goal is that no child with leukemia will have to go through chemotherapy,” Tedder says.

How it works: The drug latches on to a protein present on immature B cells, such as those in most childhood leukemias. Depleting most of the cancer B cells paves the way for new cellular therapies being developed by collaborators at the Fred Hutchinson Cancer Research Center to kill the rest of the cancer, reducing or eliminating the need for chemotherapy.

Next steps: Tedder seeks support for safety studies to prepare for eventual clinical trials.

"TURNING BAD B-CELLS GOOD"

The technology: Fostamatinib

The promise: Prevent graft-versus-host disease

How it works: Fostamatinib blocks B cells that have been abnormally activated. “When a foreign protein is seen, the B cell should fight it off,” says Stefanie Sarantopoulos, MD, PhD. But in patients with chronic graft-versus-host-disease, a potential side effect of bone marrow transplant, some B cells damage normal cells and cause problems. Sarantopoulos has found.

Next steps: Sarantopoulos is testing Fostamatinib in a Phase 1 clinical trial for Duke bone marrow transplant patients.

FIGHTING GRAFT-VERSUS-HOST DISEASE

The technology: Regulatory T cells (in development with Collective Biotherapeutics)

The promise: Prevent a debilitating side effect of bone marrow transplant called graft-versus-host disease. After some transplants, the bone marrow donor’s immune cells attack the patient’s body, as if it were a foreign invader.

How it works: B10 cells can tamp down an immune response such as graft-versus-host-disease, Duke researchers have found. But these cells are rare; a human body contains only a handful. The lab of Tom Tedder, PhD, has developed a way to take B10 cells from the blood, massively increase their numbers in culture, then put them back into the person. Tedder’s team, including collaborators Anthony Sung, MD, and Nelson Chao, MD, has effectively treated graft-versus-host disease in mice, using mouse B10 cells.

Next steps: After translation into humans, Tedder aims to launch clinical trials for adults with graft-versus-host-disease and children with a disease called humoral immunodeficiency.

HOW THE POLIOVIRUS WORKS

The poliovirus is modified to cause polio. The RNA is replaced with RNA from a tumor initially grew larger after the poliovirus injection. He was given chemotherapy in hopes of gaining a few more weeks. Soon, his tumor was shrinking. After a second dose of chemotherapy and a few more months, it had disappeared. Gromeier was excited but puzzled. "Scientifically, no one really knows what’s going on," he says. "But it’s so attractive because these chemotherapy drugs have been used for decades. We have other patients, not just Mr. Steele, who are responding very well to the combination of the poliovirus with chemotherapy. We are performing laboratory studies combining our virus with traditional chemotherapy to identify the mechanisms at work." Today, Steele is doing well three years after first receiving the poliovirus.
STEPHANIE LIPSCOMB, 25, Greenville, South Carolina, was diagnosed with stage 4 glioblastoma (brain tumor) in 2011. After receiving an experimental poliovirus therapy she is in remission and works as a pediatric oncology nurse. She was photographed with her boyfriend, Matthew Hopper, at Bald Rock Heritage Preserve in Cleveland, South Carolina.

“I never thought that I was going to die. Just going back for my MRI scans, I had complete faith in God and my doctors. It was just showing me, look, you’re trusting me and you’re trusting your doctors, and you’re going to change the world.”

STEPS ON COMB, 25, Greenville, South Carolina was diagnosed with stage 4 glioblastoma (brain tumor) in 2011. After receiving an experimental poliovirus therapy she is in remission and works as a pediatric oncology nurse. She was photographed with her boyfriend, Matthew Hopper, at Bald Rock Heritage Preserve in Cleveland, South Carolina.

DUKE PHOTOGRAPHER JARED LAZARUS HAS CHRONICLED THE STORIES OF 14 CANCER SURVIVORS. HERE ARE JUST A FEW OF THEM.
Two years ago, I began a journey to show some of the faces and voices of Duke Cancer Institute patients and survivors, to give hope and inspiration to those battling the disease. I photographed and interviewed 14 brave men, women, and children in their own environments doing what they love. Unexpectedly, all these people have given me courage, especially last summer, when a large tumor was discovered in my 12-year-old daughter’s abdomen. (The tumor was found to be benign, though not before it crushed her ovary.)

So after taking an extra few minutes to hug and talk to my younger daughter about her day and the new Supergirl episode on TV that night, I pull up a photo of Stephanie Lipscomb, the young woman who volunteered to be the first patient to receive an injection of an experimental poliovirus therapy to treat her brain tumor. I tell my daughter, “Now this is a real-life superhero.”

Lazarus donated his time to this project. The Duke Cancer Patient Support Program plans to display the portraits and accompanying audio in the Duke Cancer Center in the near future.

By Jared Lazarus

MEET MORE SURVIVORS
See more faces and hear their voices at bit.ly/dcifaces

JOHNNY ALSTON, 70, Durham, North Carolina, is in remission from prostate cancer that spread to his rib, lymph nodes, and spine. He was photographed while directing a student production at North Carolina Central University, where he works part-time after serving as a theater professor for 43 years.

“If you don’t let it get you in the beginning, then every little upswing, every little positive thing that you hear or that happens to you, makes you stronger.”

BOB NORRIS, 83, New Bern, North Carolina, was diagnosed with lung cancer in 2003. Before that, his wife and adult son both died from cancer. Currently cancer-free, he skydives to raise money for cancer research.

“I just want my great grandkids to not even know what cancer is, unless they look in the dictionary or hear it on a history program. That’s why I’ve decided to do things to help raise funds.”

LAYLA SMITH, 6, Hope Mills, North Carolina, is in remission from acute lymphoblastic leukemia. She is halfway through a two-and-a-half year treatment regimen. She likes singing, dancing, and playing with her puppies and her toys.

“My hair was past my shoulders. I got sick, and after chemo, my hair fell out. People think I’m a little boy. But I like my hair short. It doesn’t hurt my feelings. I know I’m a girl and I’m still pretty.”

MELISSA CULBRETH, 42, Louisburg, North Carolina, was diagnosed with breast cancer in 2009. She is currently cancer free. Retired from her career as a chaplain in the N.C. Army National Guard, she was photographed with her dog Rocky.

“You have to pace yourself, you have to take it one day at a time, one hour at a time, and you have to have a support system and lean into it. Being in nature is where I find peace.”

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EVE GRIFFITH, 8, Apex, North Carolina was diagnosed with Wilms tumor (kidney cancer) in 2009. She has been cancer-free for five years. She likes to cook with her mother, Christy, and to dance (ballet, hip hop, and modern). She wears a tutu to her clinical visits.

“Just because you have cancer doesn’t mean you can’t show up to Duke looking fabulous. Remember you are strong, and you are brave. I kicked cancer’s butt.”

“Hope”
In his work as a cardiothoracic radiologist, Edward Patz Jr., MD, looks at a lot of X-ray film from patients undergoing cancer treatment. What he sees there has convinced him that some new ideas are needed.

"Although we’ve come a long way, we still have a long way to go," says Patz. "We keep trying variations on the same processes, and we get a little bit of improvement here and a little bit there, but it’s not enough. A lot of current drugs just are not having great impacts on many of these diseases. We need some new and different approaches."

He is exploring one such approach that involves developing an antibody from the body’s own immune system that selectively attacks cancer cells and leaves surrounding cells alone. It’s the first ever completely human-derived antibody to be used against cancer, and while Patz cautions that studies have thus been done only in vitro (in a test tube or culture dish) and in mice, the results in those tests have been encouraging. He is now preparing to conduct human clinical trials, hopefully within the year.

The idea grew out of his work examining molecular markers that provide an indication of how well, or how poorly, individual cancer patients are likely to fare. These markers are frequently used to help inform the diagnosis and prognosis of disease, but Patz wondered whether they could also be used to suggest therapeutic uses.

He knew that some patients with early-stage lung cancer do much better than others, even factoring out the effects of treatment; in these people, the tumors simply don’t metastasize or progress to advanced disease.

“So we wondered, what is it about the immune systems of those patients that provides that protection?” says Patz. “And if we can find that, can we then figure out how to activate the same sort of response in other patients? That was the fundamental question.”

He and his colleagues discovered that one thing the patients who did well had in common was an antibody that targets a specific protein called Complement Factor H (CFH), which protects cancer cells from immune system attack. The antibody disables CFH and, in a way, opens the door for the immune system to attack and kill the tumor cells.

Patz and colleagues at the Duke Human Vaccine Institute developed an innovative and complex but effective technique for producing quantities of the antibody, in effect cloning it from cells drawn from patients who produced it naturally. In tests in vitro and in mice, the antibody killed tumor cells of multiple types, and it killed them with surgical precision, targeting only the cancer cells without doing any damage to non-cancer cells.

The researchers have seen no evidence of toxicity or side effects. And in the bargain they discovered a bonus benefit: the antibody also appears to trigger an ancillary attack on the cancer from another part of the immune system.

“We don’t have to kill every cancer cell directly, because it appears that the process activates the immune system, and then that takes over and kills the rest,” Patz says.

He is working to secure funding, regulatory approvals, and all the other steps necessary to take the work to clinical trials.

Patz is cautious, but optimistic. The work is still in its early stages, but the data thus far are encouraging, he says, and the need for new therapies that target cancer without causing collateral damage is so great that it’s worth the effort.

“Will this work in humans? We don’t know yet,” says Patz. “But it gives us a new paradigm, a new way to think about attacking cancer. We’re hoping, of course, that it will work. How cool would that be, to get a drug to patients that takes care of the tumor and doesn’t have side effects? Our hope is to come up with something that’s a real benefit for patients.”

Edward Patz Jr. is the James and Alice Chen Professor of Radiology and a professor in pharmacology and cancer biology.
But only...

Margaret Stoffregen presents a check to her surgeon, Brian Brigman, MD, PhD, alongside her mother, Molly, and father, Eric. Margaret and five friends from her Chapel Hill, North Carolina, middle school held a bake sale that raised more than $2,000 for Duke cancer school held a bake sale that raised more than $44,000 for the Duke Multidisciplinary Sarcoma Research Program.

THE SWEET SIDE. Osteosarcoma survivor Margaret Stoffregen presents a check to her surgeon, Brian Brigman, MD, PhD, alongside her mother, Molly, and father, Eric. Margaret and five friends from her Chapel Hill, North Carolina, middle school held a bake sale that raised more than $2,000 for Duke cancer research and the Be Loud Sophie Foundation, which supports teen and young adult cancer patients.

GIVE 1 FOR DAD. In honor of his late father, Neil Poley, who battled prostate cancer, Sam Poley has raised more than $72,000 to support a clinical trial of a potential new treatment for patients with advanced prostate cancer, based on Duke research. The trial, to be led by Dan George, MD, should start enrolling patients by early 2017. Learn more or donate at give1fordad.com.

CLIMBING TO BEAT CANCER. Barrett Whitten stops for a DCI photo op on Mount Whitney, the highest mountain in the continental United States. The climb was part of his training for his “Summit for Cancer” campaign, in which he aims to climb all Seven Summits of the world to raise funds and awareness for Duke colon cancer research. Whitten climbed the first of the seven—Mount Kilimanjaro in Africa—in 2013. Next, his sights are on Russia’s Mount Ebrus.

ALBUM FOR A CURE. Duke Raleigh ICU nurse Daniel Nickels is pouring his love of singing and songwriting into his first solo album, The Answer. In honor of his late aunt’s battle with breast cancer, he will donate sales until November 15 to support breast cancer research in the lab of pharmacologist and cancer biologist Donald McDonnell, PhD. Donors will receive a copy of the album for a minimum $10 donation. Visit danielnickelsmusic.com.

STRIKE OUT FOR SARCOMA. The annual 5K run and family fun walk in September 2016 raised more than $44,000 for the Duke Multidisciplinary Sarcoma Research Program.

CANCER patients with high-cost treatments such as oral chemotherapy are at greater risk of non-adherence.

- 70% greater risk of non-adherence.
- 51% of patients in cancer treatment want doctors to take cost into account.
- But only 19% had talked costs with their doctors.

Patients with cancer are 2.65 times more likely than others to declare personal bankruptcy.

- 27% of cancer patients reduced spending on basic needs like food and clothing.
- Gleevec, a medication for the treatment of chronic myeloid leukemia, saw a 249% price increase from 2001 to 2014.

The costs to patients continue to rise as co-pays, drug prices, and administrative costs increase. Still, Zafar says there are steps that could be taken by virtually all health system stakeholders to minimize the financial burden on patients. That is true not only of major players such as government, insurance companies, and pharmaceutical firms, but also of health care providers and patients themselves.

"The health care system should have greater transparency to help minimize unexpected bills," he says. "Providers should discuss financial considerations with patients just as they do medical considerations, and patients need to learn how to bring up financial matters with their doctors. There are resources available that can help. We all need to do a better job of linking patients with those resources." You can follow Zafar on Twitter @yzafar.

Yousuf Zafar is an associate professor of medicine and director of the Center for Applied Cancer Health Policy.

By Dave Hart
Quitting for Good

Tim Kinnamon, 58, has been a smoker for more than 25 years. He started when he was 21 and soon was smoking three packs a day. In his early 30s, he decided to quit. “I just did it cold turkey,” he recalls. “I put the cigarettes down and did not pick them up for almost 12 years.”

He does not remember what triggered it, but after 12 years he found himself smoking again. “I was an idiot. I tried those little cigars, and next thing I know, I started smoking again,” he says. For the past 15 years, Kinnamon has been smoking two packs a day. Five years ago, he was diagnosed with prostate cancer, and recently he decided to try quitting again. He was hoping it would be as easy as it was 27 years ago. “But this time it was not happening,” he says.

Kinnamon’s oncologist at Duke Cancer Institute referred him to Quit At Duke, a new smoking cessation program that launched in May 2016. According to James Davis, MD, the program’s director, it is very common that smokers will make several attempts to quit without success. The cessation rate for people who try to quit on their own is less than five percent, and with some additional help they may reach a 10 to 15 percent success rate. The Duke Center for Smoking Cessation has developed a unique program to treat smokers more effectively. The first step is a thorough evaluation of what causes a patient to smoke. Treatment includes evidence-based medications, behavioral therapy, and close follow-up with patients for six to twelve months after they complete the program.

The program offers a novel behavioral intervention called mindfulness training for smokers. “Mindfulness is a skill that trains patients to pause and bring close attention to their experiences, thoughts, emotions, or feelings. It is particularly effective for people who have difficulties with stress and anxiety,” Davis says. Studies have shown that when smokers experience urges or negative emotions, they react automatically by smoking. “Mindfulness is a way to disrupt this connection, bring awareness to the triggers of smoking, and let them pass,” Davis says.

Cancer patients who quit smoking will respond better to treatments. “We know that surgery is more successful in patients who quit smoking. Radiation is less effective and has greater side effects in smokers, and chemotherapy side effects are worse for them,” Davis says.

Kinnamon is making his first steps in Quit At Duke. “I hope it will allow me to breathe better and get some more energy to start exercising,” he says.

By Aliza Inbari

TO ENROLL in the Quit at Duke program, please call the Duke Center for Smoking Cessation at 919-668-3842.
SOOTHING SOUNDS

WILLIAM DAWSON, musician in residence at Duke University Hospital, plays the Steinway and Sons piano near the healing path on the lower level of the Duke Cancer Center. The piano was a gift of Alison and Carl Ravin, MD, in honor of Kevin Sowers, President, Duke University Hospital.

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DCI Office of Development / Kathi Dantley Warren, Senior Executive Director
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30th Anniversary of the Shingleton Society Luncheon and Awards

Thursday, October 27, 2016
12:00 – 2:30 pm • Washington Duke Inn
For information contact 919-385-3176 or dukehealthrsvp@duke.edu