




**BREAKTHROUGHS**  
FOR LIFE

# When you give to science, science gives back.

The generosity of private benefactors is critical to eradicating cancer. Donations from individuals, businesses and foundations are the genesis of scientific breakthroughs that save lives. Your contributions fund innovative early stage research, recruitment of the world's top scientists, new research technologies, and services for patients and families in need. In the articles that follow, look for this icon,  a symbol of the forward momentum that private benefactors deliver to accelerate scientific breakthroughs for life.

**On the cover: Drs. Barry Stoddard and Amanda Mak made a potentially lifesaving breakthrough this year, which they are already translating into possible treatments for genetic diseases.**

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## MESSAGE FROM DR. LARRY COREY, PRESIDENT AND DIRECTOR

### Your gifts catalyze our lifesaving breakthroughs

One of the privileges of my position as president and director of Fred Hutchinson Cancer Research Center is to reflect each year on the many ways in which our latest breakthroughs lead to improved health for people around the globe. It is both rewarding and deeply humbling to know our research saves lives.

Another privilege I particularly enjoy is thanking the many organizations and individuals who support the Hutchinson Center. Breakthroughs happen here because generous benefactors like you give our pioneering researchers the freedom to create and explore innovative ideas with the potential for profound impact.

This year that exploration led to major strides—not just in one or two areas, but across the entire breadth of our research. Some of our recent advances, highlighted in this annual report, include:

- Creative new solutions to treatment challenges—including bold efforts to extend the curative power of bone marrow transplantation to wider patient populations and a game-changing breakthrough in pancreatic cancer treatment.

- Understanding the structure and mechanism of highly specific DNA-binding proteins—after investing nearly 20 years in research to unlock their secrets—which might one day be used to repair children's genetic disorders, such as cystic fibrosis, thalassemia and sickle cell anemia, through a combination of targeted gene correction and stem cell transplantation.

- World-leading cancer prevention research to unravel the links among obesity, inflammation and cancer risk, helping the medical community begin to tackle the emerging epidemic of obesity-related cancers.

- Two unprecedented discoveries—one that sheds light on why some prostate cancers are lethal and another that reveals how a single genetic abnormality leads to a form of muscular dystrophy—that researchers can leverage to craft new prevention, detection and treatment strategies for these diseases, and potentially many others as well.

- The finding that drugs used to treat AIDS can actually prevent HIV's spread—a result so

significant it was named the breakthrough of the year by *Science* magazine—and first-of-its-kind evidence of a viral vulnerability that is already changing the science of HIV vaccine development.

These achievements show how innovative thinking, catalyzed by your gifts, brings hope to patients and their families. Your contributions sustain the Hutchinson Center's unique scientific culture—one that fosters ingenuity, risk-taking and collaboration among the world's top researchers to produce major medical advances.

On behalf of everyone at the Hutchinson Center, I extend my sincere thanks to you for helping us save lives by investing in our breakthrough research.



DR. LARRY COREY





# LIFESAVING RESEARCH

Leukemia patient Christian Beattie, opposite page, is reaping the benefits of Dr. Colleen Delaney's lifesaving breakthrough in umbilical cord blood transplantation. Delaney has developed a way to multiply cord blood cells in the lab, making transplants viable for the many patients who can't find a matching bone marrow donor.

## The curative power of umbilical cord blood

Christian Beattie is a competitive cyclist who, over the course of a long career, has raced with the sport's biggest names and in the world's most prestigious events. But last year, the 39-year-old from Kansas City, Mo., went from winning races to feeling "not right."

Beattie was diagnosed with acute myelogenous leukemia. His best hope for a cure was a bone marrow transplant, the Nobel Prize-winning treatment developed at Fred Hutchinson Cancer Research Center.

But, like 40 percent of all transplant candidates, Beattie was unable to find a well-matched donor with enough shared biological characteristics to make a transplant viable. After exhausting all other treatment options, a doctor in Kansas gave him slim odds of reaching the age of 40.

Then a friend showed him an article about Dr. Colleen Delaney's research at the Hutchinson Center. Her efforts were a ray of hope to Beattie, one of the 16,000 people each year who can't

find a donor match within their families or through international bone marrow registries. For most ethnic minorities and people of mixed heritage, the chances of finding a donor are even lower. "When all else failed, I knew I had to get to Seattle," he said.

He did just that and is now reaping the benefits of Delaney's lifesaving breakthrough.

Delaney is pioneering research focused on the healing powers of umbilical cord blood transplants. Collected from the discarded placenta after a baby is born, cord blood is rich in helpful cells that have the potential to cure cancer.

"It really is the ultimate recycled product," Delaney said. "You can give life to a child and by reusing what is normally thrown away, save the life of someone else."

Because the cells found in cord blood are less developed than adult blood stem cells, they don't need to be as closely matched to a patient, allowing a match for nearly all patients. But cord blood transplantation has one problem—a single unit contains so few cells that a patient is vulnerable to infection for a long time before the transplant can rebuild the immune system. This has made it

particularly difficult to transplant adult patients with cord blood.

Delaney's research has broken through this barrier by developing a way to multiply the tiny number of umbilical cord blood cells 200-fold in the laboratory so they can be transplanted in adults.

Beattie received an expanded cord blood transplant and left the hospital in just 10 days, with the underpinnings of a healthy immune system—far faster than with a traditional transplant and with few side effects.

Now, Delaney and colleagues are taking their work further, developing an approach to cord blood transplants which doesn't require patient-donor matching. Under this new study, she is working to develop expanded, "off-the-shelf" cord blood units that can be frozen and made available to all patients.

"I want people to know *everyone* has a match," said Beattie, whose plans now include cycling into his 40s and beyond.



CHRISTIAN BEATTIE

**▶ Gifts from the Bayley Family Foundation and other unrestricted donations support Dr. Colleen Delaney's lifesaving cord blood transplantation research. Additional philanthropy helps patients, like Christian Beattie, go through the rigors of the transplant procedure and live in the furnished apartments of Pete Gross House during their treatment.**



## Significant breakthrough in pancreatic cancer treatment

Pancreatic cancer is a formidable foe. It grows fast and spreads quickly, often before any symptoms appear. And because of its notorious resistance to treatment, 97 percent of patients aren't alive five years after diagnosis.

Dr. Sunil Hingorani understands all too well the pain inflicted by a pancreatic cancer diagnosis.

He lost his father to the disease, leading him on a personal and professional mission to find ways to beat it.

His dedication is paying off with some of the most significant contributions in the fight against pancreatic cancer—including a

breakthrough discovery that could change the way it is treated.

A few years ago, Hingorani developed the first mouse model that faithfully mimics human pancreatic cancer, from its precancerous inception

to its advanced stages. This powerful tool has enabled researchers to get closer to understanding pancreatic cancer's defenses better than ever before, opening the door to more effective ways to detect and treat the disease.

Using this model, Hingorani recently made two potentially momentous discoveries. He unraveled the secrets of how pancreatic tumors thwart therapies and then developed a way to penetrate those defenses.

Most cancerous tumors grow blood vessels to deliver nutrients and oxygen to them, providing an entrance for chemotherapy drugs to gain access to the tumor.

In pancreatic cancer, however, Hingorani discovered that pancreatic tumors build a tough, protective shell—similar to scar tissue—that physically keeps chemotherapy from entering the tumor. This revelation explains why treatments that eliminate other cancers are ineffective here; the cancer drugs never reach the pancreatic cancer cells.

With this knowledge, Hingorani and colleagues

found a way around the protective shell: Using their mouse model, the team employed an enzyme that digests and removes the scar-like tissue around the tumor. With the tumor defenses breached, the chemotherapy can enter and attack the cancerous tissue.

The results were so successful, Hingorani worked to get clinical trials fast-tracked.


Hingorani, with his unwavering dedication to patients, is now leading an international trial for patients with metastatic pancreatic cancer, using

a combination of the enzyme and chemotherapy.

Already, his work is bringing new hope to patients at Seattle Cancer Care Alliance, the treatment arm of the Hutchinson Center.

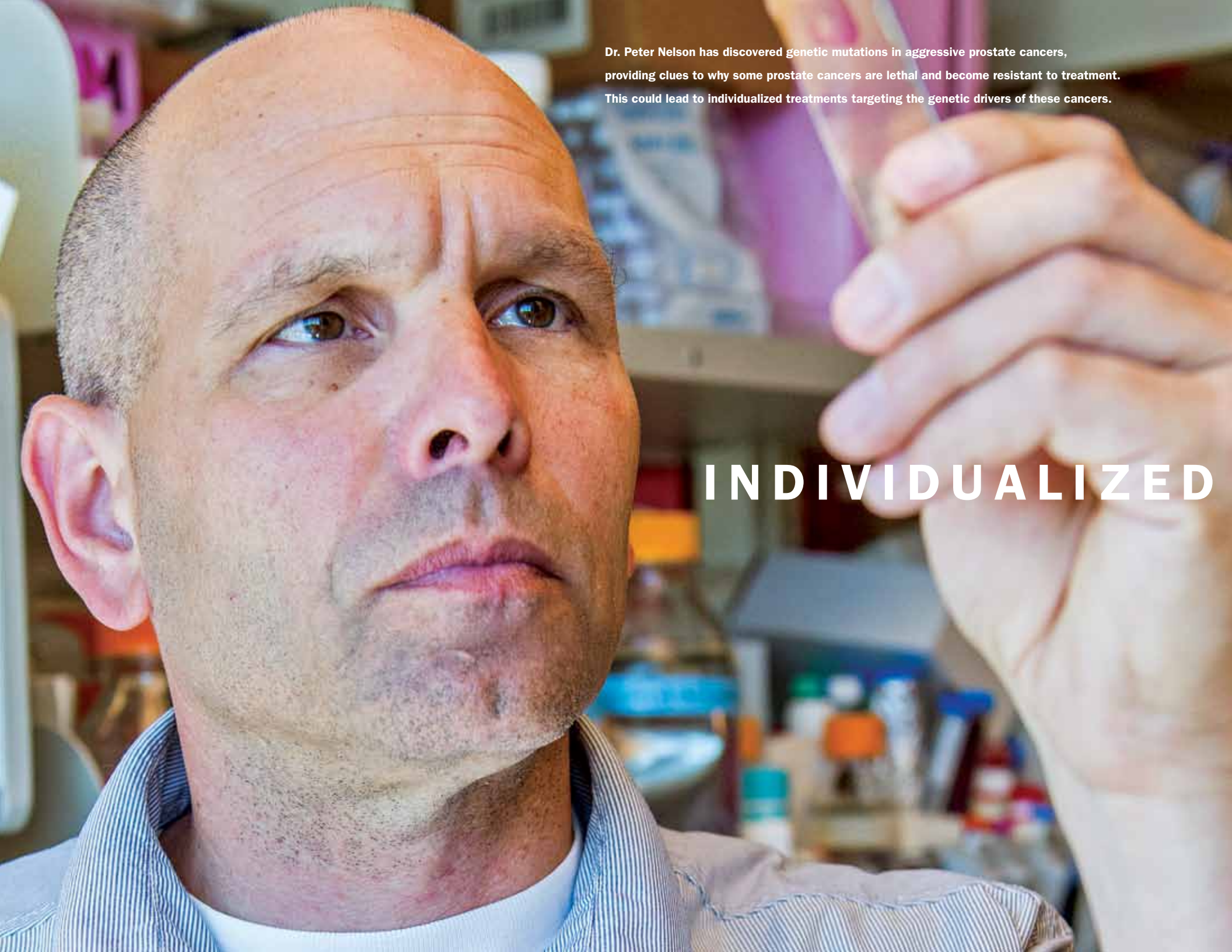
"We're not just treating a disease," he said. "We are treating a person. Patients are not statistics. We think about this disease day and night."

# BREAKING THROUGH BARRIERS

 A generous donation from Maryanne Tagney-Jones and David Jones challenged a community of supporters, and collectively their gifts helped make possible Dr. Sunil Hingorani's potential breakthrough in pancreatic cancer treatment.

Dr. Sunil Hingorani, pictured with Dr. Vikas Goel, right, unraveled the secrets of how pancreatic tumors thwart chemotherapy and developed a way to penetrate those defenses. These breakthroughs are so significant, he is fast-tracking this new treatment to help patients with metastatic pancreatic cancer.





Dr. Peter Nelson has discovered genetic mutations in aggressive prostate cancers, providing clues to why some prostate cancers are lethal and become resistant to treatment. This could lead to individualized treatments targeting the genetic drivers of these cancers.

# INDIVIDUALIZED THERAPIES

## Targeting the genetic drivers of lethal prostate cancers

Nearly all men will develop prostate cancer— if they live long enough. Yet its progression remains difficult to predict. In some men, the disease lies dormant for many years; in others, tumors rage in spite of treatment, spreading to the bones, where a cure is exceedingly rare.

Identifying new, more precise ways to individualize care for the disease’s varied forms is one of the major challenges confronted by the Hutchinson Center’s Dr. Peter Nelson, one of the

nation’s leading prostate cancer researchers.

This year, Nelson and colleagues at the Hutchinson Center and University of Washington discovered three types of aggressive prostate tumors that mutate at a much faster rate than others and become resistant to treatment.

The discovery of these genetic mutations provides clues to why some prostate cancers are

lethal, and potentially could lead to prevention methods, screening tests for early cancer detection or drug targets to slow or halt cancer growth, Nelson said.

The scientists found these “hypermutated” cancers after mapping the genomes of 23 lethal and drug-resistant forms of the disease, which until now had been poorly defined.

“The most interesting finding was the discovery of the three aggressive tumor types, which had 10 times the number of mutations compared to the other advanced prostate cancers we studied,” Nelson said. “That was very surprising and unusual. We don’t know the cause of these hypermutated tumors, but the frequency of the mutations suggests these tumors evolve very rapidly to develop resistance to therapies.”

The genetic knowledge of these lethal cancers could enable researchers to tailor treatments that target the genetic drivers of the cancers.


Nelson likens the aggressive tumors to Ferrari racecars. “If we can identify the molecular engines that drive the cancers, we may be able to block the fuel that runs those engines, which could slow or stop the cancer from progressing,” he said.

Nelson is hopeful this line of research could be one of the first successful steps to treat aggressive prostate cancer through precision medicine.

The next goals are to expand this research to other cancers and to help define its use in determining which patients to treat and which to watch closely. These are the types of big ideas Nelson and his research team are undertaking.



DR. PETER NELSON

 Dr. Peter Nelson’s discoveries on lethal prostate cancers were fueled by gifts from the Thomas C. Wright Foundation, the Richard M. Lucas Foundation and the Prostate Cancer Foundation.



Sally von Bargen lost 75 pounds, nearly one-third of her body weight, during an exercise and diet study conducted by Dr. Anne McTiernan. Her transformation illustrates how Hutchinson Center researchers are at the forefront of understanding—and potentially preventing—obesity-fueled cancers.

# LIFE-CHANGING SCIENCE

## Breaking the link between obesity and cancer

Sally von Bargen once described herself as a “jolly, overweight, grandmother type” who watched her mother struggle with breast cancer and her father battle heart disease and diabetes. Overweight since childhood, she knew she was on a slippery slope toward weight-related illnesses, including cancer.

Then last year, she signed up for a Hutchinson Center study led by Dr. Anne McTiernan examining diet and exercise in overweight postmenopausal women.

For the scientifically rigorous study, von Bargen’s calorie intake was scrutinized, her movements meticulously recorded, her body fat and bone density carefully scanned. Her study goal was to lose 23 pounds. She lost 75, nearly one-third of her body weight.

“I can say—unequivocally—that I will never gain that weight back again because now I know how to exercise and eat to be healthy,” she said.

Von Bargen’s transformation illustrates how Hutchinson Center researchers, like McTiernan and her colleague Dr. Mario Kratz, are at the forefront of understanding—and potentially defusing—what is becoming a new cancer epidemic. Being overweight or inactive is known to increase a person’s risk of some of the most common cancers, including colorectal cancer, breast cancer and certain prostate cancers, and is also linked to cancers of the esophagus, pancreas, kidney and gallbladder.

The threat is so severe that McTiernan, whose numerous groundbreaking studies over the last 20 years have illuminated much of what we now know about obesity and cancer risk, likens obesity to another insidious problem that took decades to diminish.

“Obesity is almost like the new smoking,” McTiernan told the *Los Angeles Times*. “The effect

isn’t as big for most cancers, but it’s so prevalent that it will have a huge impact.”

McTiernan’s research measures how exercise and weight loss affect the levels of estrogen, inflammation and other so-called “biomarkers” linked to cancer. Her studies have found that people who get regular, moderate exercise—even later in life—can reduce their risk for breast cancer, colon cancer, diabetes and other major diseases. This year alone, she published findings from several studies that explain how weight loss can reduce breast cancer

risk and describe evidence-based factors that contribute to successful weight loss.

Kratz has also made significant strides with some of the world’s most innovative feeding studies, which he believes will redefine the guidelines of what we should and shouldn’t eat to reduce our cancer risk. His research focuses on inflammation, one of the big keys in the obesity-cancer equation.

Obesity, he explains, puts the body in a state of

constant, low-grade inflammation, which can cause progressive damage linked to a variety of health problems, including cancer, heart disease and diabetes.

A current study investigates how sweetened beverages, such as soda with high-fructose corn syrup, impact levels of C-reactive protein (CRP) in a person’s blood, which signals the presence of inflammation.

“Our pilot studies showed that some sweeteners could cause a significant increase in a person’s CRP levels in just over a week,” Kratz said. “If that happens in eight days, imagine what it does over your entire life—it could have a substantial impact on your risk of developing many kinds of chronic disease.”

Kratz’s studies could ultimately reshape dietary guidelines to reflect new knowledge about which foods affect cancer risk. It’s all part of stopping obesity-related cancers before they strike.



DR. ANNE MCTIERNAN



DR. MARIO KRATZ



The cancer prevention research of Drs. Anne McTiernan and Mario Kratz is supported by the Hutchinson Center’s President’s Circle, a group of individuals and corporate benefactors whose aggregate funding speeds the development of innovations in all areas of our research.





Using a unique form of computational and X-ray crystallographic analyses, a team of researchers, including Drs. Amanda Mak and Barry Stoddard, solved the structure of a unique type of DNA-targeting protein. This will enable them to engineer the protein to fix genetic diseases, such as sickle cell anemia and cystic fibrosis.

# TARGETING GENETIC DISEASE

## Correcting genes that cause inherited diseases

In his Hutchinson Center laboratory, structural biologist Dr. Barry Stoddard is engineering biochemical guided missiles to fix genetic diseases.

After almost 20 years analyzing unique types of proteins that seek out and bind to precise gene targets, Stoddard's laboratory made a potentially lifesaving breakthrough this year, which they immediately began translating into tools to provide better care for patients.

Stoddard and his team of researchers were able to take this leap forward when they determined the

three-dimensional structure of a protein called a TAL effector—transcription-activator-like effector—which, along with a group of DNA-targeting enzymes called homing endonucleases, can be used to treat genetic diseases. Stoddard and colleagues are now attempting to do just that, by combining both types of proteins to potentially treat and cure inherited disorders, such as cystic fibrosis and sickle cell anemia through a technique known as targeted gene correction.

Already, he's collaborating with Dr. M.A. Bender to develop therapies for sickle cell anemia—a devastating blood disorder that causes excruciating

pain and leads to a host of chronic, sometimes fatal health problems.

Bender, an affiliate researcher at the Hutchinson Center, is an associate professor of pediatrics at the University of Washington and director of the sickle cell clinic at Odessa Brown Children's Clinic. After Bender identified a DNA sequence associated with the disease, Stoddard engineered a targeting protein to moderate the genetic defect.

Targeting sickle cell is just the first step. By determining the TAL effector structure, Stoddard has opened the door to alter the protein for a variety of biotech and

medical applications. "The upcoming years will see an explosion in the development and use of TAL effectors for corrective gene therapy," he said.

Although still in the developmental stage, targeted gene correction is a giant advance from existing gene therapy, which involves introducing a corrected version of a defective, disease-causing gene with the hope that it assumes the role of the defective gene. The problem is that the corrected gene doesn't always find its way to a safe or useful location in a patient's DNA—a drawback that limits the therapeutic benefits and can cause harmful side effects.

Dr. Barry Stoddard's breakthrough was aided by Dr. Phil Bradley's computer modeling of the TAL protein, pictured behind Stoddard.




Targeted gene correction with TAL effectors involves directly repairing disease-causing genes, which promises more predictable results without harmful side effects because the TAL effector can lock onto precisely the right gene.

In nature, TAL proteins exist in a type of bacteria that infects plants such as soybeans, tomatoes and peppers. The bacteria use them to target specific DNA sites in the plants.

Stoddard's breakthrough enables scientists to see exactly how TAL effectors recognize specific DNA sequences and bind to them. "By solving their structure, we can manipulate TAL effectors to find human DNA targets, stay active longer, or deliver additional molecules that drive a desired change in the gene target," he said.

This is exactly what Stoddard envisioned when he began studying gene targeting proteins almost 20 years ago—and what he and Bender are doing to correct the genetic defect that causes sickle cell anemia.

"We're getting close to being able to test it on human cells in the lab. If it works, we'll move forward with improved versions," said Bender. "Our ultimate hope is that we can dramatically reduce the symptoms of sickle cell anemia—and possibly even provide a cure."

 Dr. Barry Stoddard's advances in targeted gene correction were made possible in part by a generous gift from the Cuyamaca Foundation.



## Hope for patients with a common muscular dystrophy

It is an insidious muscle disease whose symptoms often begin in the mid-teens. Arm weakness and shoulder deformities are common. In some cases, it can lead to hearing loss, abnormal heartbeat and leg weakness. Some patients become severely disabled and wheelchair-bound.

It is one of the most common inherited forms of muscular dystrophy, known as FSHD—facioscapulohumeral dystrophy—named for the

muscles of the face, shoulder blade and upper arm it attacks.

FSHD and other muscular dystrophies have proven to be extremely confounding for biomedical researchers, yielding so little information that the very idea of coming up with drugs to treat them has

seemed like a dream. For more than two decades, researchers have known that FSHD is caused by a genetic mutation, but no one understood the mechanics of the disease and exactly what was causing the muscles to deteriorate.

Then, after several years of intensive research, a team led by Hutchinson Center neurologist

and molecular geneticist Dr. Stephen Tapscott broke through the decades-long stalemate with FSHD. This year, they identified the genes and proteins that damage muscle cells, as well as the mechanisms that can cause the disease.

These discoveries open the door for developing a biomarker-based diagnostic test and future treatments for FSHD—as well as for potential new ways to treat cancer.

Tapscott's team first showed that it took two genetic changes on chromosome 4 to cause FSHD. Later, they determined that a certain protein called DUX4—normally found in germ cells that give rise to sperm in males

and egg development in females—was abnormally active in muscle cells, causing muscle mayhem.

Because the protein is not supposed to be active in mature muscle tissue, it blocks normal functions and regeneration of muscle cells, leads to muscle cell death, and likely triggers an immune response, with the body attacking its own muscle cells.

With this information in hand and newfound interest from pharmaceutical companies, Tapscott hopes that new therapies and biomarker-based diagnostic tests for FSHD patients are not far off.

“Getting here was not easy,” Tapscott said.

“There was not a ‘eureka’ moment, but rather it played out over several years of hard work that gradually revealed the answers. It has been extremely gratifying to put all the different pieces together and see a bigger picture reveal itself that could have a profound impact on patients suffering from this disease.”

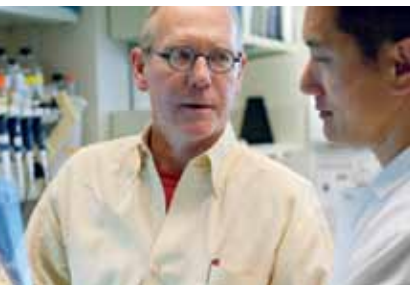
In addition, Tapscott's research has the potential to lead to a vaccine to treat cancer.

One of the major challenges in developing cancer vaccines is finding immune targets present


in cancer cells but absent in healthy cells. DUX4, the protein Tapscott identified as the cause of FSHD, induces the expression of proteins that are targeted by the immune system in several types of cancer. Now, he and Dr. Stanley Riddell are working to determine whether they can use DUX4 to stimulate the immune system against these proteins.

“This knowledge gives us a way to manipulate the expression of these proteins, opening the door to developing a new type of vaccine capable of targeting multiple cancers,” Tapscott said.

# GAME-CHANGING DISCOVERIES



DRS. STEPHEN TAPSCOTT  
AND ABRAHAM FONG

 **Dr. Stephen Tapscott's research on FSHD and cancer is supported by a pilot grant from the Hutchinson Center's Hartwell Innovation Fund, created by Craig and Marie Mundie, which allows scientists to rapidly respond to novel and sometimes unexpected research opportunities. The Geiger Family Foundation and Orin and Charlene Edson are longtime supporters of the fund.**



**Dr. Stephen Tapscott's team broke a decades-long stalemate with a common form of muscular dystrophy when they identified the mechanisms that cause it. This opens the door for future treatments for the disease and, potentially, some cancers.**



## Discovery of an HIV vulnerability is advancing vaccine development

In 2009, the HIV research field was rocked by the results of a vaccine study conducted in Thailand, the first HIV vaccine success in the epidemic's 30 years of existence. Finally, scientists had a real starting point to fight this devastating disease.

The vaccine in the RV144 HIV study—often called the “Thai Trial”—protected 31 percent of heterosexuals from new HIV infections, a huge step forward in the hunt for a successful vaccine against the virus that causes AIDS.

But the trial's partial success meant nothing without understanding why this vaccine succeeded where others had failed. This year, Fred Hutchinson Cancer Research Center scientists helped answer that question.

The Hutchinson Center has the largest team of

infectious disease researchers of any U.S. cancer center and is a global leader in HIV research. Our scientists manage the world's largest HIV vaccine program, the HIV Vaccine Trials Network.

Statistician Dr. Peter Gilbert is an expert at detecting “correlates of protection” in HIV vaccine trials. These correlates are signals from our immune system that it is successfully protecting us from infection. Identifying correlates of protection could dramatically speed up trials to test new candidate HIV vaccines and enable scientists to build a vaccine that specifically elicits those signals.

Gilbert, collaborating with a team of HIV vaccine researchers, including the Hutchinson Center's Dr. Julie McElrath and scientists at Duke University, led an effort to compare the immune signals of Thai Trial participants who had become infected and

those who had not. The team found that uninfected people had higher levels of an antibody that binds to one tiny spot in HIV's bumpy outer shell.

Gilbert and his Hutchinson Center collaborators also found the HIV strains that slipped past the Thai Trial vaccine had mutations in the exact spot where the antibody would normally bind.

The team's findings are the first evidence of this kind of weakness in HIV's seemingly bullet-proof armor.

“If this is, indeed, a real correlate of protection, then it changes everything,” Gilbert said.

Our vaccine researchers are already designing their upcoming trials to focus on this important antibody.

“This is not just academic data,” Gilbert said. “This is changing the science of HIV vaccines.”



Dr. Ying Chen and colleagues designed and analyzed the groundbreaking study that proved the drugs used to treat AIDS can prevent the spread of HIV.

## HIV treatment as prevention: breakthrough of the year

Stopping—or even slowing—the AIDS pandemic has proven to be a difficult undertaking for more than three decades. But this year a team of researchers, including biostatisticians from the

Hutchinson Center, made an advance with immediate lifesaving impact. The researchers

showed drugs used to treat AIDS can prevent the spread of HIV, the virus that causes AIDS.

The HIV Prevention Trials Network study known as HPTN 052 was so profoundly significant, *Science* magazine chose it as the world's scientific breakthrough of 2011.

More than 1,700 couples—one partner with

HIV, the other without—participated in the study spanning nine countries on four continents. The results showed that the early use of antiretroviral drugs reduced HIV transmission to uninfected heterosexual partners by a stunning 96 percent.

Half of the HIV-positive participants were given antiretroviral drugs at the outset. The dramatic effectiveness of the drugs—a 20-fold reduction of HIV transmission to uninfected partners and a 41 percent decrease in serious HIV-related health problems for infected partners—settled a long debate over whether HIV treatment could do double-duty and cut transmission rates.

“The results have galvanized efforts to end the world's AIDS epidemic in a way that would have

been inconceivable even a year ago,” wrote Dr. Bruce Alberts, editor-in-chief of *Science*. “This is not to say that we can abandon the search for an AIDS vaccine. Nor will profound change come overnight from the promise of using treatment as prevention. But for its role in making success conceivable, we have chosen the results of this trial as our breakthrough of the year.”

Hutchinson Center biostatistician Dr. Ying Chen, the second author, and his colleagues in the Center's Vaccine and Infectious Disease Division played a pivotal role in the study. They helped design, monitor and analyze the study and provided vital statistical support, processing more than a half million forms of study participant medical data.



These landmark HIV breakthroughs were made possible in part by private support, including grants from the Bill & Melinda Gates Foundation and a planned gift from the Richard W. Weiland Estate.



Dr. Peter Gilbert and colleagues have found evidence of weakness in HIV's armor, which is changing the science of vaccine development.

# LIFESAVING IMPACT



## Without research, there are no cures

Without research, there are no cures. That's why every day for nearly 40 years, Fred Hutchinson Cancer Research Center's investigators have dedicated themselves to world-class science. The payoff has been immense—breakthroughs in virtually every field of cancer research, discoveries that save thousands of lives each year. And the promise for the future is even greater.

Your support is critical to our ongoing efforts to eradicate cancer and other life-threatening diseases. It is your investment that propels Hutchinson Center science forward, accelerating progress along the entire research pipeline, whether by funding an early stage study, nurturing the career of a talented young investigator, or jump-starting a clinical trial that will bring

tomorrow's therapies to today's patients.

When you give to science, science gives back, and this report illustrates how Hutchinson Center researchers are turning your investment into innovation. We thank you for your partnership and for inspiring all of us with your generosity.



STEVE DAVIS

# YOUR SUPPORT HELPS FUND—AND FIND— CANCER BREAKTHROUGHS.

## MAKING RESEARCH PROGRESS POSSIBLE

### PRIVATE CONTRIBUTORS FISCAL YEAR 2012

Fred Hutchinson Cancer Research Center relies on private contributions to accelerate scientific advances. This crucial support aids in the pursuit of novel ideas, sustains promising research and enables scientists to explore new directions as they emerge; all paramount in the fight against cancer and related diseases. We are grateful to each of the 22,824 donors who provided \$28.8 million in contributions and in-kind gifts between July 1, 2011, and June 30, 2012. Each of the individuals, corporations, foundations and organizations listed below contributed \$1,000 or more within the 2012 fiscal year. We are grateful for the generous investment from all of our supporters in the Hutchinson Center's relentless pursuit to improve the prevention, detection and treatment of cancer and related diseases.

#### Nobel Laureates Circle

The Nobel Laureates Circle recognizes contributors who have made cumulative gifts of \$1 million or more. These key benefactors are permanent members of the Circle which was established in 2001 to honor the Hutchinson Center's Nobel Prize recipients, Drs. E. Donnall Thomas (1990), Lee Hartwell (2001) and Linda Buck (2004). By giving at this level, members of the Nobel Laureates Circle are at the forefront of philanthropic leadership and make a significant impact on the Center's mission to eliminate cancer and related diseases as causes of human suffering and death.

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*Nobel Laureates Circle membership includes three anonymous benefactors.*

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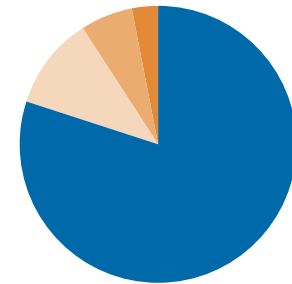
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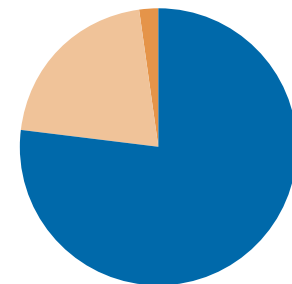
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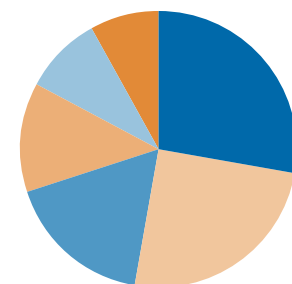
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Category	Amount	Percentage
Research Grants and Contracts	\$350,372	(80%)
Other Income	50,595	(11%)
Net Contributions	27,123	(6%)
Investment Income	11,197	(3%)



Category	Amount	Percentage
Program Services - Research	\$336,658	(77%)
Facilities and Administration	92,231	(21%)
Fundraising	6,601	(2%)



Category	Percentage
Planned Giving	28%
Major Gifts	25%
Foundation Relations	17%
Annual Giving	13%
Special Events	9%
Special Gifts	8%

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