Together We Discover

2026



Benaroya Research Institute
Virginia Mason Franciscan Health

Front cover art: Each dot represents 1,000 people. Together, the 50,000 dots represent the estimated 50 million Americans living with an autoimmune disease.

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A Letter From BRI Leadership

Dear BRI Community,

Recruiting and mentoring the next generation of researchers and leaders has been a longstanding goal at BRI. Today, we're seeing that vision become a reality. Faculty members recruited over the past decade are earning major grants and awards, making important discoveries, and building strong collaborations. They embody what BRI has always stood for: innovation, collaboration, and asking big questions that aim to predict, prevent, reverse and cure immune system diseases.

This past year offers many examples of that progress. Our scientists explored new directions in inflammatory bowel disease. We created a new generation of tetramers — building on a tool developed at BRI in the 1990s — to detect disease-causing immune cells with even greater precision. And we saw two promising therapies enter clinical trials: an engineered regulatory T cell therapy for type 1 diabetes and a new pancreas cancer therapy that BRI's Clinical Research Program helped test.

Looking ahead, we'll continue investing in the people, ideas and technologies that make advances possible. One key area of investigation will be exploring mucosal membranes of the gut microbiome and the lungs to better understand how these barrier tissues impact the immune system.

In this edition of Together We Discover, you'll see firsthand how hard work, collaboration and innovative thinking spark exciting new ideas and life-changing discoveries. You'll read stories about something BRI has understood for decades: Autoimmune diseases are deeply connected. By studying those connections — from genetics and environmental exposures to the cells and processes that drive disease — we can uncover insights that help people with all immune system diseases.

Exploring big questions like these requires sustained investment in science. As National Institutes of Health funding remains uncertain, BRI scientists are engaging with decision-makers to advocate for ongoing support of scientific research. We also know that our progress would not be possible without our donors, research participants, faculty and staff. Your commitment helps keep BRI at the forefront of studying human disease and moves us closer to our vision of a healthy immune system for everyone.

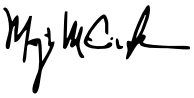
Sincerely,





Jane Buckner, MD
President
Benaroya Research Institute





Margaret McCormick, PhD
Chief operating officer and executive director
Benaroya Research Institute

Clinical Trial Highlights: Innovative Therapies Move Closer to Patients in 2026



Pictured above: members of BRI's Cancer Clinical Research Unit. Top row from left to right: Kara Turner, Rishika Das, John Willis, Kai Hinch; bottom row from left to right: Marie Hwang, Priscilla Kim, Sophia Gubbels

Across diseases, BRI is helping move promising treatments closer to patients through scientific discovery and clinical collaboration.

Engineered regulatory T cells to treat type 1 diabetes

The first therapy to treat type 1 diabetes using engineered regulatory T cells (Tregs) has entered clinical trials. Tregs act as the immune system's brakes, helping prevent it from attacking healthy tissue. Developed from decades of research from BRI's Jane Buckner, MD; Karen Cerosaletti, PhD; Alice Long, PhD; Steven Ziegler, PhD; Daniel J. Campbell, PhD; Soo Jung Yang, PhD; and other scientists. They hold the promise of addressing the underlying immune dysfunction in autoimmune diseases, not just the symptoms, and offering a more targeted treatment with fewer side effects.

"We started by understanding the fundamental biology and built on that knowledge to inform better care," Dr. Buckner said. "This is what BRI exists to do and it's a significant step toward our vision of a healthy immune system for everyone."

Targeted therapy for pancreas cancer

Pancreas cancer remains one of the most difficult cancers to treat, but the first-ever therapy that targets the root cause of the disease is now FDA-approved for some patients. A clinical trial testing this medicine as a first-line therapy is happening at Virginia Mason Franciscan Health (VMFH), in partnership with BRI's Clinical Research Program.

"This may be the most significant trial for pancreas cancer in the next decade, defining how we treat the disease moving forward," said Vincent Picozzi, MD, director of the Pancreaticobiliary Program at VMFH. "It's tremendously gratifying to be one of the sites leading this effort and it wouldn't be possible without BRI's clinical research team."

The Autoimmune Disease Branding Problem

By Bernard Khor, MD, PhD

Most people don't fully understand the biology behind colon cancer, breast cancer and pancreatic cancer. But they get that they're all cancer.

Autoimmune diseases don't enjoy that same understanding. They share a common problem: The immune system starts attacking the body, whether it's the brain, the pancreas or the gut. As an immunologist, this understanding is deeply ingrained and allows me to immediately consider how progress against one disease might help in another. However, autoimmune diseases have very different names like multiple sclerosis, type 1 diabetes and inflammatory bowel disease. They may be managed by different medical specialists. As a result, the connection is not obvious to the general public. That's why I often say that autoimmune diseases have a "branding problem."

> ***I believe this "branding problem" has real consequences and addressing it could fuel significant progress.***

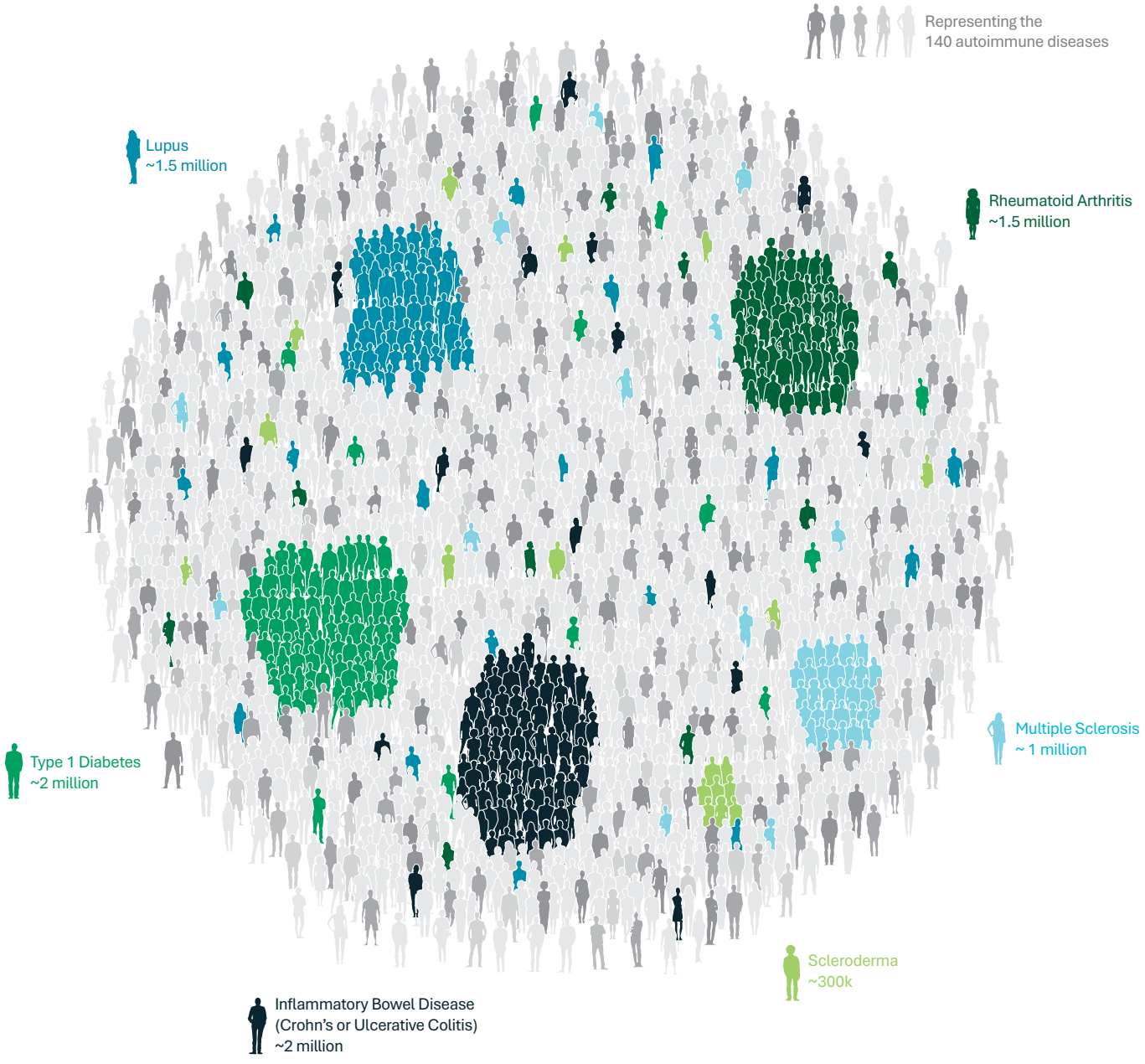
Individuals and families coping with a cancer diagnosis find strength and community in each other, often regardless of what type of cancer. Building that shared connection in autoimmunity could make each individual diagnosis more relatable and less lonely. Appreciating the magnitude of the problem that autoimmunity poses could unlock a new level of awareness, connection, urgency and research that serves the entire community.

At BRI, we believe there is power in studying these diseases — and the entire immune system — as a whole. We explore fundamental processes and shared genetics that underlie many autoimmune diseases, and we study therapies that could help treat multiple autoimmune diseases.

BRI's team has spent the past decade studying immune system diseases through this lens — because progress against one disease should mean progress against them all.

~50 MILLION

More than 140 autoimmune diseases affect people across the United States, with an estimated 27 million Americans diagnosed and 23 million more living with an undiagnosed condition.



Getting to the Root of Autoimmune Disease

Featuring Daniel J. Campbell, PhD and Estelle Bettelli, PhD

How fundamental immunology uncovers shared causes and solutions

When Daniel J. Campbell, PhD, is teaching graduate students about autoimmune diseases, he gives them a thought experiment: Imagine you're a doctor 150 years ago. One person comes in with digestive issues. Another comes in with a skin rash, then another with muscle weakness and vision loss.

> ***"Based on the symptoms alone, you'd never think their illnesses were related."***

For a long time, no one did. But decades of work to better understand how the immune system works — a branch of research known as fundamental immunology — surfaced a key realization: These diseases may look very different on the outside, but they all begin when the immune system spirals out of control and starts attacking healthy tissue.

The Center for Fundamental Immunology at BRI is asking questions to advance this work: How does the immune system ramp up to fight off infections? How does it pump the brakes once that infection is gone — and why does it sometimes attack healthy tissues?

"Our center focuses on the building blocks of the immune system and how they all fit together," Dr. Campbell said. "One of our key goals is to find ways to turn on the brakes when the immune system becomes overactive."

Tregs: Pumping the brakes on the immune system

Regulatory T cells (Tregs) are a core area of Dr. Campbell's research. These cells are key "brakes" on the immune system, telling other cells to stop attacking when an infection is gone. They're also supposed to tell your immune system not to attack healthy tissue, but something goes wrong in autoimmune disease.

Because knowing how the immune system works in healthy people can shed light on what goes wrong in disease, much of Dr. Campbell's work involves studying Tregs in people without autoimmune diseases.

"In healthy immune systems, we see this delicate balance where Tregs are very good at keeping other cells quiet when they don't need to do anything, but allowing them to respond when you do need them to, like when you have a virus," Dr. Campbell said.

That's why Dr. Campbell's team is working to learn more about exactly how and when Tregs pump the brakes, asking:

- How do Tregs tell other immune cells to slow down or stop?
- Do Tregs always tell other immune cells to slow down in the same way? Or do they use different strategies in different locations (e.g., the gut, the brain, the skin)?

The answer so far: It's complicated.

"For example, we've learned that an immune response that's neutral or even helpful in type 1 diabetes could make inflammatory bowel disease (IBD) worse," Dr. Campbell said. "That tells us that Tregs might not just use one 'off' switch; they may use different strategies depending on different situations."

Th17: An accelerator in the immune system

Tregs aren't the only cell type that play a role across multiple diseases. Another group of cells, called Th17 cells, can play a more inflammatory role, acting like an accelerator on the immune system.

BRI's Estelle Bettelli, PhD, has long studied Th17 cells. Her early work helped establish them as a key driver of inflammation in multiple sclerosis. Now, she's working to better understand exactly how these cells cause disease and if they behave differently in different tissues.

Understanding players, like Th17 cells, that ramp up the immune system is crucial because it provides a potential target for treatment. For example, now we're seeing treatments that target IL-23 (a molecule that amps up Th17 cells). Those drugs treat psoriasis and IBD by slowing down inflammation driven by Th17.

"That's just one powerful example of how one discovery can make an impact across multiple autoimmune diseases," Dr. Campbell said.

Autoimmune Diseases by the Numbers

As many as

80%

of people with autoimmune disease are female

National Institutes of Health, 2025

Nearly

25%

of people with an autoimmune disease will develop at least one more during their lifetime

Autoimmune Association, 2026

An estimated

1 in 7

people in the United States live with an autoimmune disease

National Institutes of Health, 2026

More than

140

autoimmune diseases identified

National Institutes of Health, 2025

Autoimmune Disease Prevalence in the United States*

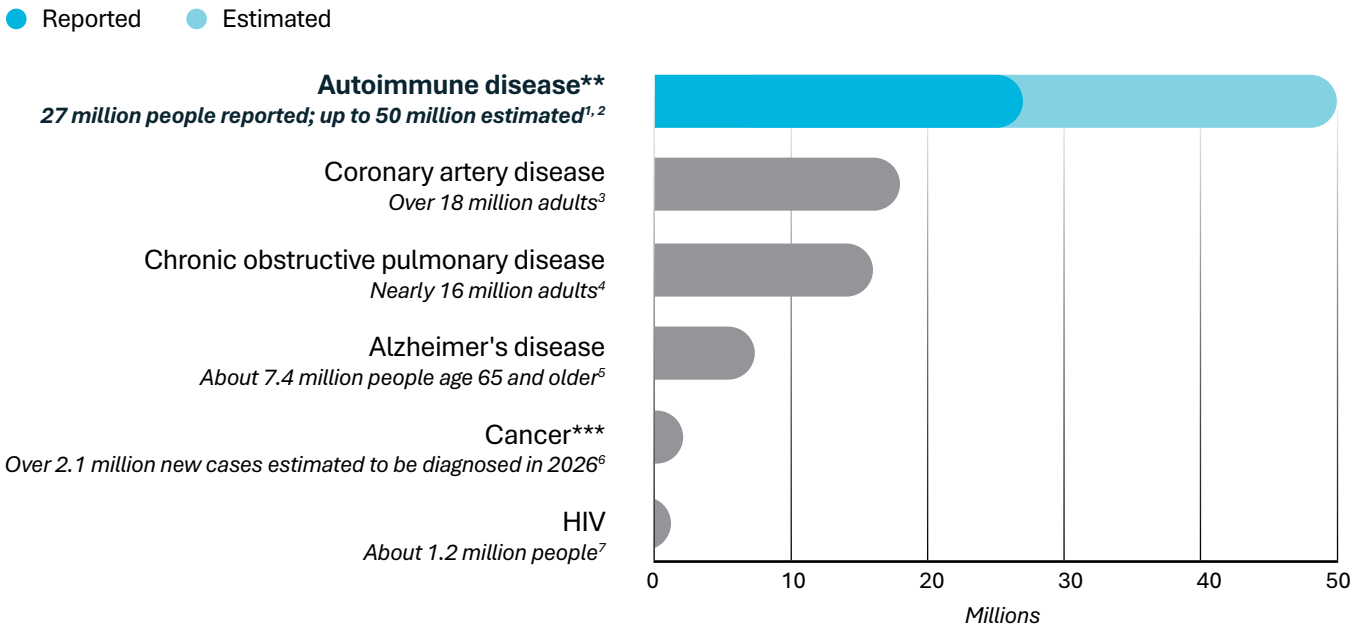


Chart adapted from the National Institutes of Health-Wide Strategic Plan for Autoimmune Disease Research: Launch Video Slides — Autoimmune Disease Prevalence in United States, 2026.

***The National Institutes of Health reports approximately 27 million people living with autoimmune disease in the United States (U.S.), up to an estimated 50 million. The one in seven statistic in the Autoimmune Diseases by the Numbers section is calculated using the 50 million estimate and U.S. population data from October 12, 2026.*

****Excludes basal cell and squamous cell skin cancers and in situ carcinoma, except urinary bladder.*

***Sources:** [1]National Institutes of Health, 2025; [2]National Institutes of Health, 2026; [3]Cleveland Clinic, 2026; [4]Centers for Disease Control and Prevention, 2024; [5]Alzheimer's Association, 2026; [6]American Cancer Society, 2026; [7]U.S. Department of Health and Human Services, 2026.*

The Shared Path to Autoimmune Disease

Every autoimmune disease is different, but the path to developing these diseases is similar. It starts with genetic risk, followed by a trigger, like a virus or exposure to pollution or smoke. That trigger can upset the balance of the immune system, and it can lose

GENETIC RISK

Genetics can make a person more likely to develop an autoimmune disease. But not all people with genetic risk will actually develop disease.

MULTIPLE SCLEROSIS (MS)

TYPE 1 DIABETES (T1D)

INFLAMMATORY BOWEL DISEASE (IBD)

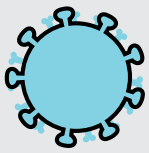


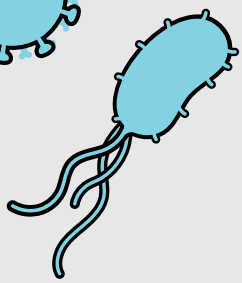
Genetic variants that increase the risk of MS can decrease the risk of T1D.

TRIGGER

If a person with genetic risk encounters a trigger, they may develop disease. Triggers can be viruses, bacteria, air pollution and other things in our environment.

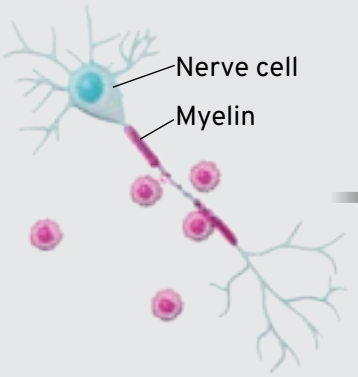


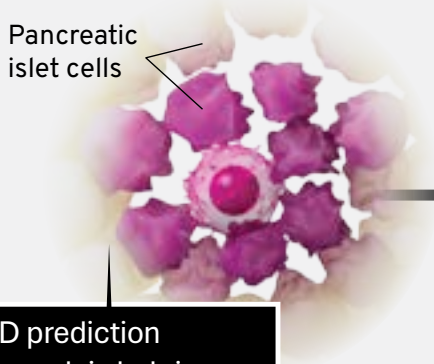




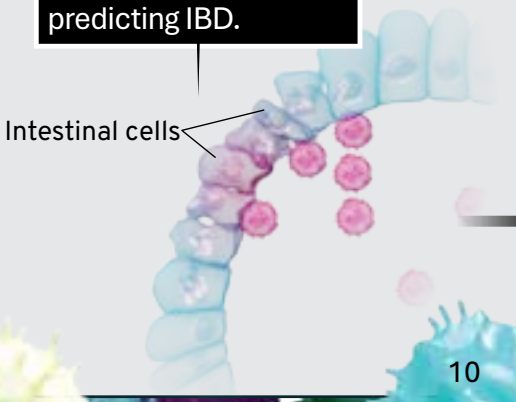
BREAKING TOLERANCE

When tolerance is broken, the immune system loses its ability to distinguish what to protect from what to attack.





T1D prediction research is helping shape a framework for predicting IBD.

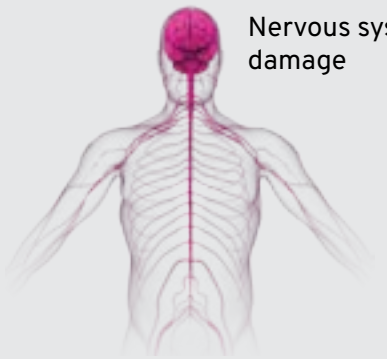


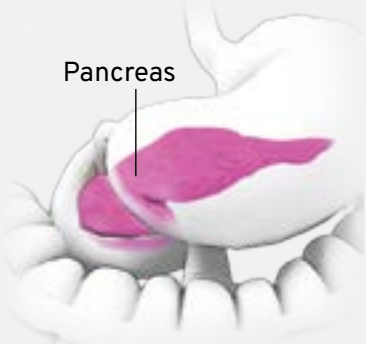
its ability to distinguish what it should protect from what it should attack. As a result, tolerance is broken and immune cells may start attacking healthy tissue, leading to autoimmune disease.

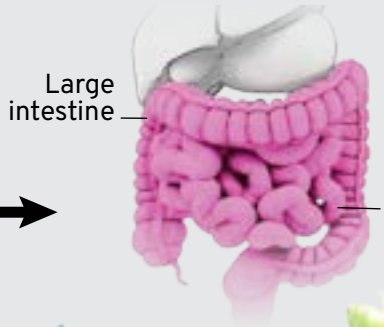
The path ends in "treatment" because there is no cure for any autoimmune disease. BRI is working to change that by studying every step on this path, in different diseases and across all of them.

TISSUE DAMAGE

The immune system mistakenly attacks healthy tissue. Different autoimmune diseases affect different parts of the body, causing tissue damage and symptoms.



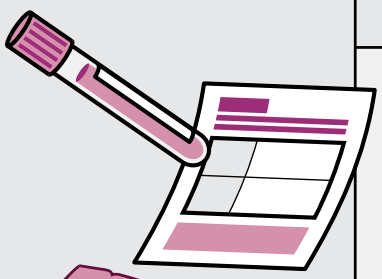




DIAGNOSIS

For many autoimmune diseases, it can take years and visits to many doctors to get the right diagnosis.



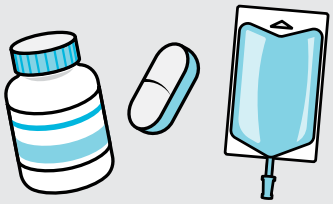




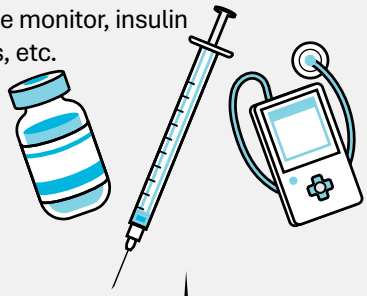
TREATMENT

Finding the right treatment can be complicated. People may need to try different treatments over time to find the right option for them.

MS: Corticosteroids, biologics targeting B cells, etc.

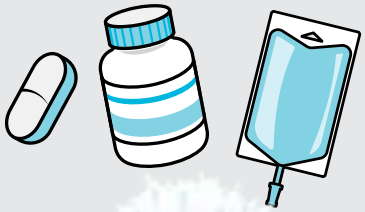


T1D: Insulin, continuous glucose monitor, insulin pumps, etc.



Treatments for IBD are being tested to prevent T1D

IBD: Corticosteroids, biologics, etc.



The Immune System's Hidden Controls

Featuring John Ray, PhD

How noncoding regions of the genome influence autoimmune disease

You've probably heard people say things like "autoimmune diseases run in my family" or "certain genes can make someone more likely to develop type 1 diabetes."

Researchers have known for some time that our genetic makeup plays an important role in whether we acquire autoimmune diseases.

But scientists are increasingly realizing that the parts of our genetics that lead to disease tend not to be coded within genes or regions of the genome that contain instructions for building proteins. Instead, most of the genetic differences that lead to disease are located in the vast stretches of DNA surrounding genes known as "noncoding regions."

For years, some scientists dismissed these regions as "junk DNA" because they don't contain genes. But researchers now know that certain parts of noncoding regions, called "regulatory regions," can play an important role in regulating how genes behave — turning them on, off, or changing how strongly they function.

"We still don't fully understand how noncoding regulatory regions of the genome work," said BRI's John Ray, PhD. "But advances in technology are finally giving us tools to explore how these regions influence genes and the role they may play in autoimmune diseases."

The light and the dimmer

Dr. Ray is working to better understand how noncoding regulatory regions influence the immune system. Think about it like this: If a given gene is a light, a nearby noncoding regulatory region acts as a dimmer system that helps control when the light turns on and how bright it is.

In a healthy immune system, those controls help immune cells respond at the right time and with the right intensity. They turn on genes that cause cells to attack germs when needed and then turn them down or off once the threat is gone.

Genetic changes, called genetic variants, in noncoding regions may help explain why autoimmune diseases happen. Autoimmune-associated genetic variants might cause immune cells to ramp up inflammation at the wrong time or stay activated for too long.

Studying thousands of genetic variants at the same time

Noncoding regions contain enormous amounts of genetic information, along with millions of genetic differences between people. Because of how genetic variation is inherited from your parents, one of the biggest challenges is figuring out the exact genetic variants that influence how the immune system behaves.

To tackle that problem, Dr. Ray uses an innovative tool called a massively parallel reporter assay. It allows his lab to test thousands of genetic variants at once and identify the ones that may influence gene activity.

One recent study performed this assay on more than 18,000 genetic variants associated with five different autoimmune diseases. Alex Ho, PhD, staff scientist in the Ray Lab, found that more than 500 genetic variants across the genome influenced gene behavior. Using CRISPR, a tool used to modify the genome, Dr. Ho identified the specific genes controlled by 56 of those variants.

The lab is now scaling these studies to test over 100,000 variants associated with almost 300 immune diseases and traits. The hope is that through defining how genetic variation guides one disease, we may be able to better understand how other diseases are acquired.

“Existing medicines that are already FDA-approved may be able to be repurposed for other diseases. That means we can get people effective treatment faster,” Dr. Ray said.

The study will also uncover some important genetic variations that are unique to specific diseases. That’s important too, because defining the genes regulated by those variants could open the door to more personalized treatment targets, prevention strategies, and new avenues for hard-to-treat diseases.

> ***“What excites me most is that we can now study what causes human immune disease with a level of precision that simply wasn’t possible 10 years ago.”***

“The more precisely we understand how immune diseases develop, the better positioned we are to ultimately deliver better therapies to patients.”



How Do Exposures Like Wildfire Smoke and 'Forever Chemicals' Influence the Immune System?

Featuring Megan Smithmyer, PhD

Exploring how the environment we live in influences health and disease

While many people have "genetic risk" for autoimmune disease, not everyone with that risk will actually develop disease. A person's environment may help explain why.

"Environmental exposures — air pollution, what we eat, the viruses we get — likely push the immune system in one direction or another," said Megan Smithmyer, PhD.

That's why Dr. Smithmyer and her team are studying how exposures like wildfire smoke and a group of chemicals known as PFAS (an abbreviation for perfluoroalkyl and polyfluoroalkyl substances) and often described as forever chemicals, affect the immune system and potentially contribute to autoimmune disease.

"Environmental exposures aren't easy to study, but we've partnered with collaborators to explore how to measure and analyze their effects," Dr. Smithmyer said. "Now we have some methods in place and we're moving forward with some very exciting work."

Wildfire smoke and immune cell activity

While wildfire smoke is becoming more prevalent in the Pacific Northwest and around the world, scientists still know very little about how it impacts the immune system.

"It's possible that it could have similar negative health effects to cigarette smoke or air pollution. But studying the impact of wildfire smoke is tricky because you don't know when and where the smoke will happen or how bad it will be," Dr. Smithmyer said.

As luck would have it, BRI's team happened to collect samples from a group of research participants during one of the worst wildfire smoke events Seattle has ever seen in 2020.

"The city was inundated with smoke. The sky was orange, it looked like the apocalypse and our participants still trucked all the way to BRI for their scheduled blood donations," Dr. Smithmyer said.

Those participants were part of the Sound Life Project, a study of healthy immune systems. Dr. Smithmyer was able to zero in on those samples to get one of the first detailed looks at immune cells during a wildfire smoke event.

Her team observed a number of changes in immune cells, including increased activation of T cells, particularly in samples from older people. These cells play an important role in fighting germs, but when they mistakenly attack the body's own cells, they can also drive autoimmune disease. While it's not yet clear what these changes mean for long-term health or autoimmune disease, they offer an intriguing starting point for understanding how wildfire smoke may affect the immune system.

Now, the team hopes to explore these changes in a larger population, including firefighters who experience greater exposure to wildfire smoke.

"Down the road, this work might help us better understand who is at the highest risk for long-term effects from wildfire smoke exposure and if those effects can be prevented or mitigated," Dr. Smithmyer said.

Exploring forever chemicals and autoimmunity

Dr. Smithmyer's team is also exploring the impact of PFAS on the immune system. PFAS are used in many products like nonstick cookware and water-resistant clothing and have been linked with health issues such as decreased fertility, cancer and difficulty fighting infections.

Dr. Smithmyer's team started by figuring out how to measure PFAS in existing biorepository samples. Now she's building on that work, hoping to ask questions like: Could exposure to PFAS trigger autoimmune disease in someone who is genetically susceptible? Might it accelerate the onset of disease in someone already on track to develop it?

Work like Dr. Smithmyer's wouldn't be possible without BRI's biorepositories, which give scientists access to blood and tissue samples that enable them to ask big questions and find life-changing answers.

"Our biorepositories make it possible to answer questions that otherwise may not be feasible or take too much time and money to carry out," Dr. Smithmyer said. "Every sample donated makes a huge impact, not just in one study but in many."



Same Treatment, Different Result: Exploring Why Medicines Work and Who They Work Best For

Featuring
Alice Long, PhD

How understanding the immune system can lead to more effective treatments across diseases

In the 1980s, scientists identified Tumor Necrosis Factor Alpha (TNF-α), a protein that plays a key role in inflammation. By the early 2000s, anti-TNF medicines that target this protein became widely used to treat rheumatoid arthritis (RA). Now, they're also used to treat ankylosing spondylitis, inflammatory bowel disease and other autoimmune diseases.

But as useful as anti-TNF medicines are, they are not a one-size-fits-all solution. For example, they don't work in multiple sclerosis. And even in diseases where they're widely used, they don't work for everyone.

That's where Alice Long, PhD, comes in. Her research explores how medicines work and why they might work differently in people with the same disease.

"A lot of studies ask: Does this medicine work, yes or no?" Dr. Long said. "But I focus on questions like how or why does a medicine work? This helps us learn how to develop more effective medicines, and how to select the right treatment for each person."

Boosting Tregs to fight disease

One promising way to treat various autoimmune diseases is to boost the activity of Tregs — the cells that act like brakes on the immune system. One way to boost their activity is with IL-2, a molecule that helps them grow and function.

"This approach has worked well in systemic autoimmune diseases like lupus and in graft versus host disease, where the immune system attacks the body after a transplant," Dr. Long said. "But it hasn't worked the same way in every disease."

Which begs an important question: Why do treatments boosting Tregs help in some autoimmune diseases, but not others?

One possibility is timing. In some diseases, the immune system may go off track in the early stages of disease, before a person even develops symptoms or is diagnosed. Treatments that boost the brakes may come too late to fully slow it down. Or perhaps there aren't enough Tregs or an individual's Tregs don't effectively slow their immune system.

To get to the root of the problem, Dr. Long's team studies how Tregs respond to immune signals, which genes they turn on or off, and why responses vary from person to person.

"The biology is complex and we're honing in on the right thing to fix," Dr. Long said. "The more we understand how these cells behave in different diseases and in different people, the better we can design treatments that target the right problem."

Same medicine, different response

Abatacept is a medicine that could also help treat a variety of autoimmune diseases. It's proven to effectively treat RA in around 60% of patients. It also holds promise in type 1 diabetes (T1D): Studies have shown that abatacept can keep the pancreas producing insulin in some recently diagnosed people.

Even though we have a basic understanding of how abatacept works, it remains unclear why abatacept works for some people and not others. BRI's DREAMT study aims to find out by asking:

- What do the immune systems of people who respond to the drug have in common?
- What do nonresponders have in common?
- Are there similar patterns between responders and nonresponders across T1D and RA?

"These questions can teach us about disease biology. And the better we understand disease biology, the more effectively we can treat disease," Dr. Long said. "Just as important: if we can find patterns among nonresponders, we may help identify effective treatments for them too."



The Multiplier Effect



Featuring Elliott and Lynn Green

Be a Multiplier

Donate to the Innovation Fund by scanning the QR code above.

Believing in Scientific Potential

"Their potential feels unlimited." — Lynn Green

Lynn was a librarian and Elliott was an engineer. They met when Elliott was working on a research project in 1974.

"My colleague encouraged me to go back and thank the librarian who helped me," Elliott said. "Now Lynn and I have been married for 50 years."

After spending many years in Silicon Valley, it's no surprise the couple is passionate about exploring new ideas that have the potential to change the world.

Lynn and Elliott retired and moved to Bainbridge Island, Washington, and learned about BRI through Elliott's care team at Virginia Mason Franciscan Health. BRI's Innovation Fund, which awards teams of BRI investigators funding to explore new ideas and technologies, quickly piqued their interest. They've been supporting the fund since its inception in 2023.

"We enjoy seeing which projects are chosen and watching their success," Lynn said. "It's extraordinary to see BRI's scientists develop innovations that change the field, not only in our lifetime but in just a few years."

Exciting projects so far include a state-of-the-art microscope that allows scientists to see cells in 3D and a sequencing tool that enables scientists to collect more data from rare, disease-causing cells.

"There's a lot of personal satisfaction in supporting the Innovation Fund because it serves as venture capital for exploring promising research ideas," Elliott said. "It is a place where our donations, quite modest in terms of the BRI budget, are highly leveraged. Innovation grants have helped scientists develop proof of concept and have helped establish junior staff as independent researchers."

Elliott spent much of his career helping organizations run more efficiently and effectively. He appreciates how BRI is built on collaboration, breaks down silos and prioritizes supporting the next generation of research leaders.

"I like the way they run things," he said. "They're very intentional about investing in retention and bringing on new, talented people, which is crucial to the long-term success of an organization."

Lynn describes what BRI means to her in a single word: potential. She loves learning about research advances, like the noncoding region of the genome and AI and machine learning in immunology. "BRI has the power to conquer debilitating diseases," Lynn said.

Innovation Fund Helps BRI Scientists Land Larger Grants

Featuring Eddie James, PhD

Type 1 diabetes (T1D) begins when the immune system starts producing proteins called autoantibodies. For years, Eddie James, PhD, has wanted to zero in on the period when people first develop autoantibodies to learn more about how T cells that attack the pancreas behave before T1D symptoms appear.

But these cells are few and far between. When Dr. James first applied for funding for the project, he was denied. Funders weren't convinced he could collect and study enough of these rare cells to find meaningful answers.

A few years later, Dr. James and Bill Kwok, PhD, pioneered a more powerful version of tetramers, a tool Dr. Kwok originally developed in the 1990s at BRI. With support from BRI's Innovation Fund, Dr. James and Dr. Kwok launched a pilot study using the new technology.

"Second-generation tetramers enable us to collect and study ten times as many disease-causing cells," Dr. James said. "When we reapplied for funding using data generated from our Innovation Fund project we received a $2.6 million grant."

That's the power of BRI's Innovation Fund: It gives scientists the opportunity to test promising ideas, implement new technologies and collect data that can help them compete for and secure much larger grants.

With new NIH funding, Dr. James is using second-generation tetramers to fish out self-reactive T cells (the cells that attack the pancreas in T1D). Then, he uses a tool called single-cell RNA sequencing to look inside those cells and see what they're doing.

"It's like pulling up the control panel on your computer screen and seeing all the programs running, like an internet browser, a music player, a notes app," Dr. James said. "Cells run programs too, and some of those programs are linked to autoimmunity."

Dr. James' hypothesis is that after autoantibodies appear, T cells start running more disease-related programs. If that's true, it could open the door to a therapy that selectively kills those cells or turns off their disease-driving programs.

"Right now, we have one therapy that can delay the onset of T1D by a few years, and that's great," Dr. James said. "We hope this research leads to treatments that slow the disease for longer — or prevent it altogether."

What is the Innovation Fund?

BRI's Innovation Fund supports early-stage projects and technologies, filling funding gaps and advancing scientific discovery.

Each project:

- Brings together at least two BRI scientists from different Centers of Discovery, often including an early-career investigator.
- Develops, tests and implements new technologies.
- Shares tools and findings across BRI, fostering collaboration and supporting grant proposals.

The Multiplier Effect: Fall 2023 Innovation Fund Project

Focus: Generating Beta Cells at BRI

Led by Eddie James, PhD, and Caroline Stefani, PhD

$100,000 → **6** → **>$7.6 mil.**

Innovation Fund project investment

Number of BRI labs now using data and tools developed during this project

received to date in federal grants using data and tools developed from this project

BRI Faculty

Members

BRI members are faculty scientists who lead labs, develop innovative approaches to studying the immune system, and advance BRI's leadership in human immunology.

★ *Denotes an individual who has joined our faculty within the past year.*
◆ *Denotes an individual who has retired within the past year.*



Estelle Bettelli, PhD
Center for Fundamental Immunology
Member



Jane Buckner, MD
Center for Translational Immunology
Member
President, BRI



Allyson Byrd, PhD
Center for Systems Immunology
Assistant member



Daniel J. Campbell, PhD
Center for Fundamental Immunology
Center director and member



Karen Cerosaletti, PhD
Center for Translational Immunology
Center director and associate member



Carla Greenbaum, MD
Center for Interventional Immunology
Member



Kurt Griffin, MD, PhD
Center for Interventional Immunology
Associate member



Jessica Hamerman, PhD
Center for Fundamental Immunology
Member
Director, BRI academic affairs



Oliver Harrison, DPhil
Center for Fundamental Immunology
Associate member



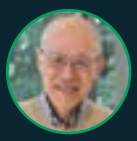
Edward Irvine, PhD ★
Center for Systems Immunology
Assistant member



Eddie James, PhD
Center for Translational Immunology
Associate member



Bernard Khor, MD, PhD
Center for Translational Immunology
Associate member



Bill Kwok, PhD
Center for Translational Immunology
Member



Adam Lacy-Hulbert, PhD
Center for Systems Immunology
Center director and member



Peter Linsley, PhD
Center for Systems Immunology
Member



S. Alice Long, PhD
Center for Translational Immunology
Member



James Lord, MD, PhD
Center for Translational Immunology
Research associate member



John Ray, PhD
Center for Systems Immunology
Associate member



Sandra Lord, MD
Center for Interventional Immunology
Clinical director
Director, BRI Clinical Research Center



Cate Speake, PhD
Center for Interventional Immunology
Center director and associate member



Meg Mandelson, PhD, MPH
Center for Interventional Immunology
Director, pancreatic cancer research



Caroline Stefani, PhD
Center for Systems Immunology
Research assistant member



Carmen Mikacenic, MD
Center for Translational Immunology
Member
Associate director, BRI



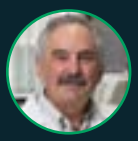
Soo Jung Yang, PhD
Center for Translational Immunology
Research assistant member



Peter Morawski, PhD
Center for Fundamental Immunology
Research assistant member

Emeritus

BRI's emeritus faculty are retired members whose scientific leadership and discoveries have helped shape the field of immunology and advance BRI's mission.



Gerald Nepom, MD, PhD
Emeritus member



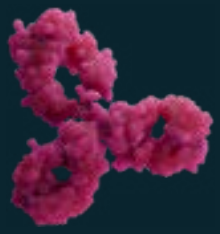
Thomas Wight, PhD
Emeritus member



Steven Ziegler, PhD ◆
Emeritus member

Celebrating Dr. Ziegler and His Retirement

After nearly three decades at BRI, Dr. Ziegler retired, leaving a lasting legacy of groundbreaking discoveries in immunology.



Welcome to BRI's Newest Principal Investigator, Dr. Irvine

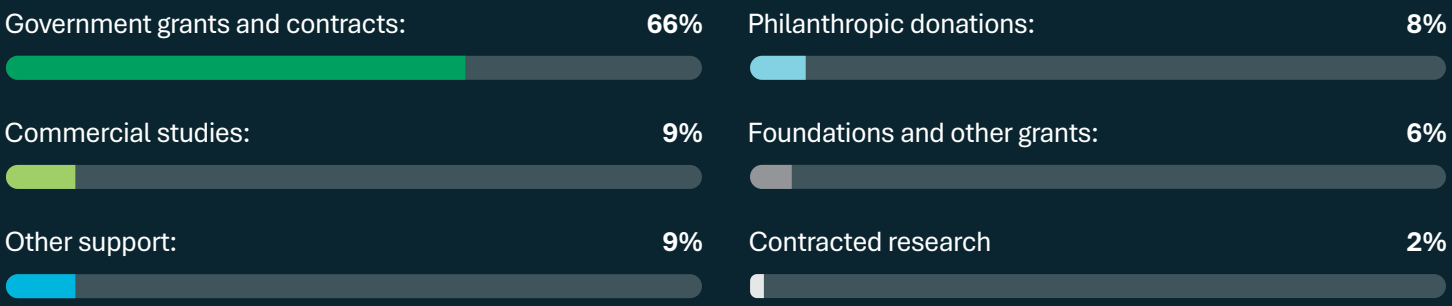
Dr. Irvine's lab combines protein engineering, machine learning and immunology to study how antibodies and other proteins communicate with immune cells. His team uses these insights to design proteins that control immune responses with greater precision.

BRI by the Numbers

For 70 years, BRI scientists have worked to better understand the immune system and improve the lives of people affected by diseases like type 1 diabetes and rheumatoid arthritis. From the start, we've known discovery doesn't happen alone. Together with partners in science, government and industry — and with the support of our generous donor community — we're uncovering the root causes of immune system diseases and building on discoveries across diseases to advance better treatments and new approaches to prevention.

2026 BRI Components of Support

$78 million



2025-2026 Awards and Recognition

Shubham Bansal
Student Intern, Linsley Lab
2026 Husky 100 Award, University of Washington

2026 Rhodes Scholar, University of Oxford

Jane Buckner, MD
2025 Top Doctor*, Seattle Magazine
**Four years running*

Linda Dement
Grant and Finance Program Manager, Grants and Contracts
2025 Excellence Award, Society of Research Administrators International

Sarah Holton, MD, PhD
Visiting Scientist, Mikacenic Lab
2026 Chair of Medicine Scholar Award, UW Medicine

Peter Linsley, PhD
2025 Health Care Leadership Lifetime Achievement Award, Puget Sound Business Journal

Carmen Mikacenic, MD
2026 Assembly on Allergy, Immunology, and Inflammation Mid-Career Award, American Thoracic Society

2026 Physician Enterprise Vision Overall Academic Excellence Clinical Research Award, CommonSpirit Health

Kristin Weinstein
Graduate Student, Campbell Lab and Ziegler Lab
2026 Husky 100 Award, University of Washington

About BRI

Progress against one immune system disease is progress against them all

At BRI, we study the immune system and the many diseases that affect it — including autoimmune diseases, allergies, asthma and cancer. By studying the immune system in both health and disease, we're uncovering how these conditions begin, what they have in common, and how to rebalance the immune system back to health.

As a nonprofit institute within Virginia Mason Franciscan Health, we work closely with physicians and patients to accelerate the path from scientific discovery to life-changing care.

Our mission:	Advance the science to predict, prevent, reverse and cure diseases of the immune system
Our vision:	A healthy immune system for everyone
Our values:	Persistent inquiry; innovation and agility; constant collaboration; integrity and respect

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