2025 Praespero Impact Report

Seven years of accelerating novel science to end autoimmune disease

an autoimmune research fund praespero

MISSION

Our mission is to facilitate innovation, collaboration, and cross-disciplinary research that will lead us to the root causes of autoimmune disease.

VISION

Our vision is to end autoimmune disease worldwide.

Inside...

Message from our founder

Dear Friends of Praespero,

When I founded Praespero in 2017, it was born from a deeply personal frustration with a system that was failing autoimmune disease patients like me. But what it has grown into is a profound privilege - the privilege of working alongside some of the most brilliant and dedicated minds in biomedical science.

Relentlessly curious, courageous, compassionate and whip-smart, the people I've met over the last seven years at Praespero model what it means to challenge conventional thinking, take risks, and move the science forward. Immunologists, neurologists, micro- and molecular biologists, biomedical engineers and medical doctors, the people our Scientific Advisory Board selects for funding comprise a diverse group of experts driven to exploring the vast, uncharted depths of the immunome to find not just better treatments, but root causes and, one day, true cures.

Our network of supported scientists operate on the very frontiers of discovery, many at great personal and financial cost, yet they do so under immense pressure: facing funding cuts, closing labs, steep competition for publication and funds and the ever-present challenge of unraveling one of science's most complex puzzles with limited resources.

New knowledge comes with great cost. As a consequence, the biomedical research sector is fiercely competitive. Meanwhile autoimmune disease cases are surging, diagnosis times are unacceptably long, and the economic toll is skyrocketing. The status quo is failing us. We simply cannot afford a research environment stifled by limited resources and intense competition when the scale of the challenge demands more investment and collaboration.

The urgency of their work and the importance of your support has never been greater. This is why I am so incredibly proud of what we have built together.

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Our network of supported scientists operate on the very frontiers of discovery, many at great personal and financial cost...

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Beyond funding critical research, Praespero has become a catalyst for a new culture of collaboration and knowledge sharing. Our Research Summits have grown into a vital forum where the world's finest experts can safely share ideas, challenge each other, and forge partnerships that transcend disciplines, institutions, and borders.

The spirit of open collaboration we champion is now having a ripple effect across the entire industry, inspiring initiatives like the Autoimmune Biomedical Collaborative (ABC), proving that when we work together, we accelerate progress exponentially.

Most importantly, this collaborative engine is fueling real, tangible impact. The research we fund is making a difference, and I attribute this to the intersection of people and projects we select for support combined with our "no strings attached" funding model. We encourage the scientists we fund to follow the science where it leads.

This freedom is vital for unbiased discovery, but it is not without accountability. Recipients are meeting rigorous criteria and demonstrate meaningful outcomes that advance our mission to eradicate autoimmune diseases.

The enclosed report details this impact through specific case studies from a sample of the projects we have funded. These stories are not just about scientific advancement; they are a testament to what is possible when we empower brilliant minds to collaborate without barriers. I hope that these case studies are not only informative and educationally stimulating, but hope-inspiring.

Thank you for your continued belief in our mission. Together, we are building a brighter, healthier future towards a world free from autoimmune disease.

With hope and gratitude,

Laurie Venning
Founder, Praespero

Welcome to Praespero'

Over the seven intense years foll
Praespero's inception, the organ
advanced through the stages of
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Praespero's focus is helping exp
research projects launch, prioriti
hold promise to uncover the dee
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science by larger leaps instead
steps.

Usually in the categories of nove
research, projects Praespero fu
sometimes outside the box of co
thinking. Each project holds pote
breakthroughs — but may just n
financial boost to get the scienc

> **"We encourage the scientists we fund to follow the science where it leads.**

Note: The enclosed sample of case studies do not represent all projects funded by Praespero. Future case studies will be developed in future editions.

's first Impact Report.

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Praespero's funding helps teams demonstrate proof of concept to inspire the confidence of larger granting agencies. Many of Praespero's supported projects result in a "multiplier effect" or chain reaction of additional large scale funding.

A little can go a long way when you invest in the right people. That's why Praespero's Scientific Advisory Board comprises an assortment of trusted experts with significant pedigrees of success in immunology. Together they scan for projects and people to support.

These projects have unfolded over a diversity of short or long term timelines and are in various stages of development.

Praespero is especially pleased to report that several of the scientists supported by the organization have transitioned (or are close to transitioning) their discoveries to the clinic for human trials. You will see their stories enclosed. They hold great promise to transform treatments and change lives. We hope these stories inspire your continued support.

"Praespero is pleased to report several scientists have transitioned (or in the process of transitioning) their discoveries to the clinic for human trials.

At a glance...

$7,000,000

donated to autoimmune disease research projects, including....

50

separate gifts (seed money) given to...

30

different labs around the world, leading to...

... a multiplier effect of an estimated

$30,000,000

in additional combined funding from major granting agencies.

6

impactful summits leading to

40

collaboration projects, multiplying impact across disciplines and universities.

The human stories behind the data

While this report focuses on the impact of Praespero funding, we also believe in the importance of the journey.

Too often, the science community might focus on only the results of science, losing sight of the human story behind the polished veneer of scientific advancement. These are the struggles, uncertainties, and iterative learnings that drive progress.

At Praespero, we believe that when we lose sight of our humanity, It is at the peril of individualized healthcare and medicine, and the spheres of influence (polical, social, cultural, academic) that drive this progress.
In the spirit of honouring the person behind the impact, we asked each of our supported scientists to identify with a "story arc" that most closely reflects their journey and to share their approach, relevant challenges and outcomes.

In honoring the human stories behind every discovery, Praespero reaffirms its commitment to funding bold, transformative ideas - empowering scientists to turn aspiration into impact and ensuring that progress in immunology serves people, not just publications.

One or more of the following story arcs are attributed to each case study enclosed in this report.

The "Quest"

Research is/was driven by a clear goal or mission, facing challenges or obstacles (technical, conceptual, or resource-related) along the way. This story focuses on perseverance and the pursuit for a significant breakthrough.

The "Turning Point"

After a long journey, funding from Praespero helped the scientist experience a pivotal moment of breakthrough that accelerated research progress, funding, or changed the pace or trajectory of the research journey.

The "Discovery"

Research journey centers on uncovering something new - an unexpected finding or insight that changed the scientist's understanding or that of the scientific community.

The "Innovation"

Research involved developing new methods, technologies, tools or approaches that push the boundaries of current knowledge. This may include using a novel approach to addressing a challenge.

The "Chain Reaction" aka "multiplier effect"

Praespero funding helped the scientist prove their science and get their research off the ground, leading to a chain reaction of additional funding from other sources.

The "Collaboration Victory"

Research journey involved or resulted in collaborative partnerships, and/or interdisciplinary efforts that played (or will play) a crucial role in achieving results.

The "Conquering Hero"

Research confronted an incomplete understanding or misperception in the science surrounding autoimmune disease. This involved directly addressing or shifting an orthodoxy, or defending an unconventional thesis and persevering to demonstrate an alternative or expanded theory.

The "Transformation"

Research outcomes transformed perspectives, treatment considerations or the field itself. May also include a journey of personal or professional evolution of the scientist.

Back to school for T cells

How educated autoimmunity holds a cure to autoimmune disease

Hilde Cheroutre, Ph.D
Professor, La Jolla Institute for Immunology,
Center for Autoimmunity and Inflammation,
Center for Cancer Immunotherapy.

Project:
Discovering and Understanding Self-Education in the Human Thymus

The "Chain reaction"

In 2025, Dr. Cheroutre leveraged research outcomes that arose from Praespero's support to raise an $1.5 million through philanthropy.

Disease category:
All autoimmune diseases and cancer.

Published work

https://pubmed.ncbi.nlm.nih.gov/37597518/

https://pubmed.ncbi.nlm.nih.gov/40203807/

https://pmc.ncbi.nlm.nih.gov/articles/PMC8116051/

Impact category:

- ☑ Understanding root causes | prevention
- ☑ Advancing science towards a cure
- ☑ Improving diagnosis and treatment
- ☐ Increasing collaboration

Research stage

Phase 9
(late stage)

> **This belief that all self-reactivity is bad and should be suppressed has been the major roadblock in making progress in treatments of autoimmune diseases. We need to shift that mindset so we can make safer treatments.**

The "Conquering Hero"

Situation

Autoimmune diseases occur when self-reactive immune cells mistake the body's own healthy tissues for threats. Most treatments rely on suppressing the immune system, which can leave patients vulnerable to infection, cancer, and other risks. Dr. Hilde Cheroutre reasons that progress toward safer more durable autoimmune disease treatments has been slowed by the assumption that all self-reactivity— also known as autoimmunity—is harmful.

"This belief that all self-reactivity is bad and should be suppressed has been the major roadblock in making progress in treatments of autoimmune diseases," says Dr. Cheroutre. "We need to shift that mindset so we can make safer and durable treatments."

Over the years, Dr. Cheroutre has been uncovering how some forms of self-reactivity are protective. An example she provides is in how the body attacks tumors, which are detected as abnormal "self-cells" by the immune system.

Her prior research also uncovered mechanisms of self-reactivity in the gut, where immune cells must rapidly distinguish dangerous pathogens from harmless food and bacteria or malignant transformed cells from rapidly dividing healthy progenitor cells. "Abnormal Self-based immunity works faster and more precisely than conventional immune cell sensing and elimination of infected or cancer stem cells at the mucosal border," explains Dr. Cheroutre. "Therefore, the mucosal immune system uses self-reactivity-based processes to respond and eliminate infected or transformed cells quickly before pathogens or cancer cells spread."

Through her research on gut epithelial immune cells, Dr. Cheroutre made a surprising discovery: these cells are not developed and educated locally as initially believed. "Instead, we discovered that they are highly educated and pre-programmed to sense and specifically destroy "abnormal self-cells" and that "education" process originates in the thymus, an organ where all the T cells develop and mature."

Dr. Cheroutre believes that the key to ending autoimmune disease may lie in the thymus. "Understanding when and how this education goes wrong, and correcting it, could lead to safer, more durable therapies and even prevention for many autoimmune diseases without weakening the immune protection against infections and cancers," says Dr. Cheroutre.

> **I want to take understanding of self-reactivity (autoimmunity) to the next step and shift toward partnering with autoimmunity rather than suppressing it which makes patients more vulnerable to other disease threats, like infections and cancers.**

Back to school for T cells

(Continued...)

Project Overview

With Praespero funding, Dr. Cheroutre's team dissected and studied the human thymus.

A breakthrough came from understanding and testing for a proof-of-concept by manipulating molecules in genetically engineered mouse models to observe downstream effects.

The team identified a small protein expressed by immature T cell precursors during their development and education in the thymus. "But once those cells leave the thymus, that small protein is no longer expressed," says Dr. Cheroutre. "When we modified its expression, we caused major disturbance in the education of the T cells, which resulted in the generation of very auto-aggressive self-attacking T cells. We thus reasoned that this small protein must play a key role during the education process of the different types of T cells."

Soon after identifying the protein, Dr. Cheroutre's team made another discovery: "We discovered that thymic "educated" autoreactive (self-reactive) T cells, such as those residing abundantly in the gut epithelium, harbor the capacity to detect and kill pathogenic autoreactive T cells."

In animal models of autoimmune disease, they found that these T cells weren't only protective in that they detected and removed infected or cancer cells, but they were also regulatory.

"This is an extremely exciting and significant discovery for autoimmune disease research," notes Dr. Cheroutre. "With Praespero's funding, we discovered that the risk for autoimmune disease and even cancer can be set very early during the education phase of the T cells. So effective treatment of autoimmune diseases requires going back to the very beginning of T cell development and focusing on repairing or restoring proper education in the thymus," says Dr. Cheroutre.

She quickly notes that this treatment strategy would apply to "spontaneously induced autoimmune diseases" and likely not to autoimmune disease induced post-thymically by environmental threats.

For example, re-education would not apply in cases of peripheral (or post-thymically)-induced autoimmune disease, in which there is nothing wrong with the thymic "education" process. Today, Dr. Cheroutre believes the future of immunotherapy for some autoimmune diseases is re-educating T cells rather than suppressing them, using cell therapies that work with the body's natural capacity to regulate or restore the immune balance.

> **"With Praespero's funding, we discovered that restoring self-education of the T cells in the thymus holds the key to curing autoimmune disease.**

> **What is amazing about Praespero is that it makes unique out-of-the-box research possible.**

> **Shifting from fundamental research to clinical research is a major career shift and I hope it will lead to a shift in how we understand autoimmunity and how we treat autoimmune disease and cancer. It's something I would never have considered pursuing if Praespero had not provided the solid support platform to make that leap.**

Funding Impact

As a result of Praespero's support, Dr. Cheroutre's team has been able to advance their science to the next steps toward human trials and safety testing. Her team has begun isolating highly educated self-reactive T cells from peripheral and cord blood (with additional approvals in progress to expand sourcing).

They are characterizing distinct subtypes of human T cells and testing their self-sensing ability using in vitro 3D microfluidic systems and patient-derived normal healthy tissue and tumor biopsies, to dissect and compare protective and destructive autoreactivity by conventional and educated self-reactive T cells.

"We've uncovered a vast amount of new information concerning protective and destructive self-reactivity of immune cells targeting healthy or cancer cells. Much of these discoveries affirmed the thinking of the Praespero Scientific Advisory Team that both autoimmune diseases and cancers can best be treated with deeper understanding of 'the concept of autoimmunity.' We've seen some extremely promising results so far."

Dr. Cheroutre is also building the clinical collaborations needed to access patient samples and move toward trials, working with clinicians at UC San Diego's Moores Cancer Center and Rady Children's Hospital and others. Her team is preparing for initial clinical trials planned for the near future in India, the UK and the US with a dedicated local clinical team and a cohort of terminally ill patients.

She credits Praespero's model for enabling the leap from concept to clinic: "What is amazing about Praespero is that it makes unique out-of-the-box research possible. Instead of discouraging, Praespero strongly promotes pioneer thinking that breaks down barriers to address questions outside the scope of traditional medicine rooted in general assumptions and dogmas.

Unlocking the viral trigger

Targeting the B Cell at the heart of virus induced autoimmunity

Marc Horwitz, Professor,
Life Sciences Institute,
Microbiology and Immunology
University of British Columbia

Project:
Virus enhanced age-associated B cells mediate commonalities in autoimmune disease pathology.

Published work

https://www.science.org/doi/full/10.1126/sciadv.adu5110

https://doi.org/10.1007/s00018-022-04433-9

Research Phase:

Phase 3
(early/mid stage)

Impact category:

- ☑ Understanding root causes | prevention
- ☐ Advancing science towards a cure
- ☐ Improving diagnosis and treatment
- ☑ Increasing collaboration

Disease category:
General (all autoimmune diseases impacted), with focus on MS, RA, T1D and lupus.

Situation

For decades, Dr. Horwitz's research into the role of viruses like Epstein-Barr virus (EBV), coxsackievirus, and their links to autoimmune diseases such as multiple sclerosis (MS), type 1 diabetes, and myocarditis was met with criticism.

"Our approaches were considered outside the box of reality," Dr. Horwitz notes, explaining that the prevailing skepticism arose because disease models could be generated without a virus, suggesting their role was minimal. However, these

The "Conquering Hero"

"Development of mice with human immune systems that could encompass the EBV life cycle was met with cynicism and was costly, but we persevered.

The "Chain Reaction"

Praespero support had a multiplier effect of over $2 million in past and future funds to establish a core facility supporting other researchers to utilize Hu mice.

non-viral models did not represent true human disease initiation and pathogenesis.

The team's conviction was driven by patient experiences, as their disease was often described as being induced or relapsed close in time to a cold or infection.

Initially tasked with modeling EBV in mice, the team spent a decade developing models and fighting for funding. Their persistence led to a critical discovery: defining the mechanism by which EBV triggers an autoimmune response.

"Our own work revealed the important role of a unique B cell subset, age-associated B cells, which were essential in mice to drive the MS-like disease," explains Dr. Horwitz.

"Our mouse models with a mouse version of EBV were informative, but we needed to move to questions studying human EBV and human immune cells. These studies represented a significant cost and some risk but they were critical."

"Our approaches were considered outside the box of reality…

Project Overview

With validation of the team's premise and a strong argument going forward, the team presented its work at the annual Praespero meeting and received an Innovation Award.

"This award provided us with the funds to push forward as well as the confidence to gamble and to take the next steps." Pursuing the question of mechanism, the team's first experiments with human immune cells and EBV validated a new direction to generate mice with human immune systems with and without EBV present and study the B-cells driving disease.

"Development of mice with human immune systems that could encompass the EBV life cycle was met with cynicism and was costly, but we persevered, adjusted our experiments to test both arguments and the work stayed the course."

Although Dr. Horwitz' work is still in early stages, an early result has been the development of a groundbreaking novel human (Hu) mouse model. Dr. Horwitz' lab is the only one in North America that provides it and it is among only 2-3 in the world offering an insight into the role of EBV in autoimmune disease initiation and development.

"We now have a novel mechanistic animal model with a human immune system that mimics the biomarkers involved in translating EBV into MS in humans," Dr. Horwitz explains.

Unlocking the viral trigger

(Continued...)

Funding Impact

Praespero's support enabled Dr. Horwitz's team to bring their work to publication readiness and secure additional funding.

The work has also contributed to a fundamental shift in immunology. "For many years, MS and other autoimmune diseases were studied through a T cell centric lens," explains Dr. Horwitz. "B cells were thought to be rather boring. People believed their only role was to make antibodies."

Evidence from the Horwitz lab helped overturn that view. They were able to demonstrate that B cells do so much more: presenting antigens to T cells; driving immune responses by making cytokines and chemokines, making or modifying other immune cells.

"Other people found these unique B cell subsets but we showed that they were driven by the viral infection and, in turn, driving autoimmunity and therefore a prime target for future drugs, therapies, and prevention strategies." says Dr. Horwitz. "Understanding the role of B cells in driving autoimmune disease represents a huge shift in understanding over the last 3-4 years and we were one of the first groups to show that."

This new understanding has opened the door to numerous collaborations. The team's hypotheses and model systems can now be applied to other EBV-associated autoimmune diseases, fostering partnerships across the Praespero network and beyond.

"The funding came at a pivotal time allowing experiments that parlayed our work into multiple grants to better define the important cell types in MS effected by EBV and to improve the models of MS," says Dr Horwitz.

"The funding came at a pivotal time allowing experiments that parlayed our work into multiple grants to better define the important cell types in MS effected by EBV and to improve the models of MS.

A Collaboration Case Study

Inspired by Praespero's Annual Summit format, Praespero supported scientists Marc Horwitz, Maria Tokuyama, May Choi and Lisa Osborne, among others, launched the Autoimmune Biomedical Collaborative Research Cluster (ABC) with financial support from UBC to forge collaborations that would facilitate innovative solutions to global autoimmune disease health challenges.

"Praespero has created an environment of scientists that clearly want to share their work, discuss their ideas and collaborate on future ideas," adds Dr. Horwitz.

With experts from UBC, Canada and around the world, the ABC grew from 15 biomedical and clinical researchers in May 2025, to over 60 members by September. "The hope is to impact our knowledge of disease, develop diagnostics, treatments and possibly cures to stop autoimmune disease," says Dr. Horwitz.

The ABC grew from 15 biomedical and clinical researchers in May 2025, to over 60 members by September.

Uncovering the hidden drivers of disease

From genetic switches to thymus fat

Project 1: Decoding the "hidden switch" of autoimmune disease relapses.

Situation

Each cell in the body carries roughly the same 20,000 genes, yet cells can look and behave very differently. Transcription factors are proteins that control gene expression - deciding which genes are turned on or off in each cell, and when. They do this by binding to specific sites on DNA and working with other molecules to start or stop gene activity.

As with all cells, transcription factors are critical in immune cells called lymphocytes (B and T cells). They help create long-lived "memory" cells that remember infections, drive powerful antitumor responses, and - when regulation fails - can contribute to autimmune disease. Similar gene-switching processes also guide embryonic development and shape how stem cells and other cells respond to signals.

"When transcription factors or their control systems malfunction, the result can be immune disorders, developmental defects, or cancer," says Dr. Dean Tantin. "As new technologies emerge, these transcription regulators and their partners are becoming promising direct targets for future drug development."

The challenge for Dr. Tantin's project was twofold: money and what he calls the "pivot penalty." "In 2018, I was moving from pure immunology into autoimmune research," explains Dr. Tantin. "The famously conservative NIH study sections would not have given this project strong consideration were it not for the early resources I received from Praespero."

Dean Tantin, PhD,
Professor Pathology,
Division of Microbiology & Immunology University of Utah School of Medicine

Project 1: Genetic and pharmacological targeting of OCA-B in autoimmunity.

Impact category:

- ☑ Understanding root causes | prevention
- ☑ Advancing science towards a cure
- ☐ Improving diagnosis and treatment
- ☐ Increasing collaboration

Disease category: MS, T1D & all autoimmune diseases

Published work

https://pubmed.ncbi.nlm.nih.gov/31266507/
https://pubmed.ncbi.nlm.nih.gov/33295943/
https://pubmed.ncbi.nlm.nih.gov/40299553/

Uncovering the hidden drivers of disease

(Continued...)

Project 1 Overview

Through Praespero support in 2019, Dr. Tantin's research uncovered a crucial protein that – unlike traditional transcription regulators that act as on-off switches for immune-target genes – prevents certain genes from being permanently shut off. Expression of this protein allows otherwise silent genes to be induced much more strongly later.

Praespero's support allowed Tantin's lab to systematically test what happens when this protein (called OCA-B) is removed or modulated in immune cells known as T cells across different multiple sclerosis (MS) mouse models.

In a relapsing–remitting MS model that closely mirrors the human disease, the team saw something unprecedented: the initial disease course and viral control remained intact, but relapses were specifically altered when the factor was removed.

"That was the first time anyone had ever seen anything like that in those models, as far as I know," says Dr. Tantin. "That's the cornerstone of this project. Because many autoimmune diseases involve relapses or flares – periods when immune responses become misdirected and repeatedly attack the body's own cells – our discovery highlights a mechanism that could inform future therapeutic interventions."

Project 1:The "Discovery"

"…our discovery of this protein activity sheds light on a mechanism that holds promise for future therapeutic interventions."

Project 2: The "Quest"

"This knowledge could provide means to combat the loss of thymic function with age, stopping impaired pathogen response and increased susceptibility to infection."

The "Chain Reaction"

Pilot data from Praespero formed the core of the preliminary that went into a funded NIH R01 grant for nearly $2 million.

Research Phase: Project 1

Phase 9
(late stage)

Research Phase: Project 2

Phase 2
(semi-late stage)

Project 2: Hidden fat cells in the thymus: Clues to aging immunity.

Situation

Again in 2024, Dr. Tantin sought support on a radically different project: studying an uncharacterized cell type – fat cells in the thymus, where T cells are made. "Importantly, work from others has shown that fat cells in different locations in the body have very different functions, but as a common feature tend to interface with the immune system," explains Dr. Tantin.

"We reasoned that fat cells in the thymus would function differently from similar cells elsewhere in the body – just as fat cells differ from one another elsewhere – and that they would interface with immune cells in the thymus, as they do in other tissues."

Dr. Tantin calls this an example of "fundamental" science: work that, although may not have an immediate practical application, informs our basic understanding of how organs like the thymus function. He notes that while fat cells have been studied extensively, information about fat cells in the thymus is very limited.

Project 2 Overview

So far, the Tantin lab has demonstrated there are multiple distinct types of fat cells in the thymus and that a likely origin for one of these subsets is a particular cell type called a thymic epithelial cell (a kind of thymic support cell). "We also found that the mix of cells in the thymus changes over the lifespan," explains Dr. Tantin, noting that the amount of one type of thymic fat cell slowly increases in ways impacted by a person's sex.

"As we age, the parts of the thymus responsible for T cell development shrink. We call this thymic involution and it leads to fewer new T cells and weaker immunity," adds Dr. Tantin noting that other work has shown thymus involution correlates with an expansion of fat cells. "We do not yet fully understand what these fat cells do but our work has been the first to characterize these cells molecularly."

Next, Dr. Tantin's team will test if thymus fat cells are truly different from other body fat, whether one subset does in fact come from thymic support cells, and whether these cells help feed and support developing T cells but do so less and less with age. "Knowing this could provide means to combat the loss of thymic function with age, something that contributes to impaired pathogen response and increased susceptibility to infection."

Funding Impact: Project 1 & 2

Seven years ago, Dr. Tantin made an argument that was outside the box of mainstream thinking. "I said if you want target something useful for future therapies, and if you want to understand what a cell is, what it's doing and where it's going, you have to understand its nuclear state including the transcription factors," says Dr. Tantin.

"Praespero funded me and let me run with that pie-in-the-sky idea and today the idea is mainstream and a major component of my lab work."

But the impact of Praespero's funding goes beyond basic research, as it enabled Dr. Tantin to initiate a drug discovery pipeline aimed at developing small molecules that can modulate the activity of the identified transcription regulator and better treat autoimmune diseases.

The first Innovation Award seeded an initial project that allowed Tantin's team to publish early findings and generate data that they leveraged into additional funding totalling near $2 million and multiple important additional research papers.

A competing renewal for that grant has been submitted and the team hopes to hear by March 2026 whether their application was successful.

> **"In this new environment, funding from organizations like Praespero is more important than ever to ensure these kinds of fundamental research projects get a fair shot.**

Shielded from the start

How early life protects against type 1 diabetes

Sue Tsai, Associate Professor, University of Alberta

The "Quest"

"By uncovering the natural protective mechanisms that mothers provide during gestation and early postnatal life, we can identify new strategies to prevent type 1 diabetes from the very start of life."

The "Chain Reaction"

After Praespero support, Professor Tsai's team secured additional funding from CIHR ($1M, 2024-2029) to expand this project, and was awarded a Weston Family Foundation grant ($300k) to study breastfeeding in T1D.

Situation

Type 1 diabetes is a serious autoimmune disease that typically develops in childhood, but its origins may begin much earlier in life. While much research has focused on what increases disease risk, according to Dr. Sue Tsai, we know surprisingly little about what protects against it, especially the biological processes during and after pregnancy that may help shield children from developing diabetes later in life.

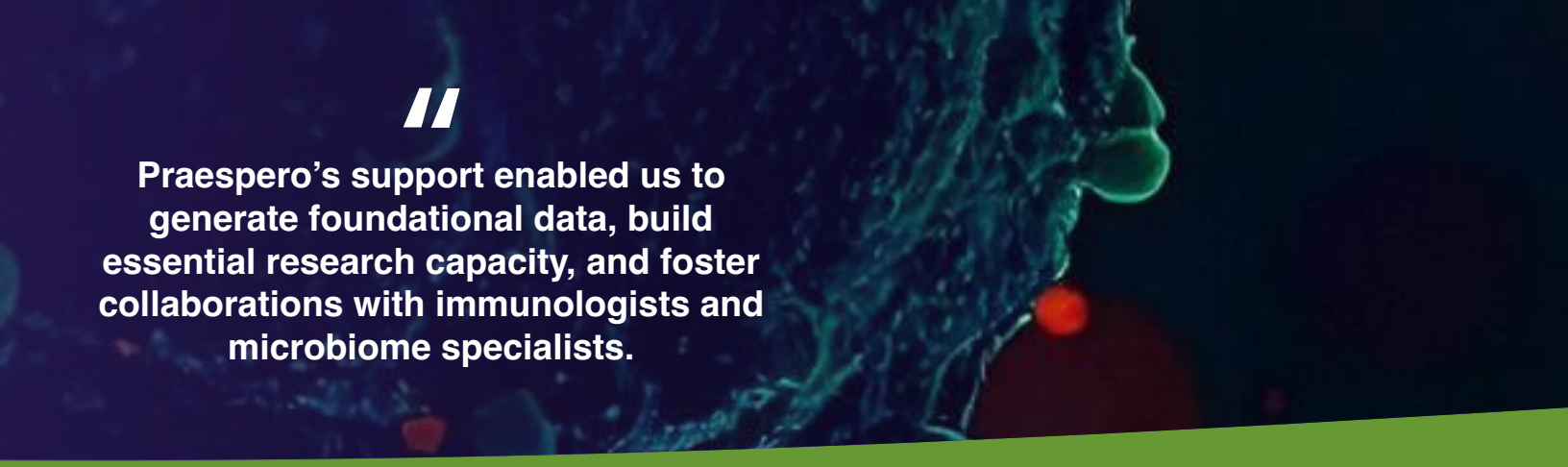
"My research addresses this gap by exploring how maternal factors such as microbes, hormones, and immune factors shape offspring health both before and after birth," says Dr. Tsai. By uncovering the natural protective mechanisms that mothers provide during gestation and early postnatal life, Dr. Tsai believes we can identify new strategies to prevent type 1 diabetes from the very start of life.

Project Overview

Dr. Tsai's research journey began with a simple question: Why do some children develop type 1 diabetes while others, even in similar conditions, remain healthy? She became intrigued by the possibility that protection might begin before birth, that mothers might pass on more than genes, perhaps also biological "shields" that help their children resist disease.

"To uncover these protective mechanisms, my team uses an animal model that allows us to study the earliest stages of life in a controlled way," explains Dr. Tsai. "We focus on two key clues: maternal antibodies, which provide early immune protection, and the microbiome, the community of microbes that helps train the immune system."

By selectively changing these maternal factors, Dr. Tsai's team can observe how each one affects the offspring's immune development and risk of diabetes. "In many ways, this process feels like being



"Praespero's support enabled us to generate foundational data, build essential research capacity, and foster collaborations with immunologists and microbiome specialists.

Project: Early life determinant of T1D

Disease category: T1D

Impact category:

- ☑ Understanding root causes | prevention
- ☐ Advancing science towards a cure
- ☐ Improving diagnosis and treatment
- ☑ Increasing collaboration

Research Phase

Phase 5 (mid stage)

Published work

https://www.biorxiv.org/content/10.1101/2024.08.28.604371v1

connect mother and child," says Dr. Tsai, explaining that each experiment helps rule in or rule out possible explanations, guiding her team closer to understanding how maternal factors shape lifelong health.

"The journey has taught me that science advances step by step, through curiosity, patience, and persistence, and that the smallest discoveries can illuminate the path toward preventing disease before it ever begins."

"In many ways, this process feels like being a detective, following a trail of biological clues that connect mother and child.

Funding Impact

Dr. Tsai believes that support from Praespero has been instrumental in advancing her work. "With this funding, we established and refined an animal model to test how maternal antibodies and the microbiome influence immune development and diabetes risk in early life," notes Dr. Tsai, adding that the experiments have helped her team identify specific maternal signals that appear to guide immune tolerance and protect against autoimmune attack.

The results have already highlighted the critical role of maternal factors during gestation and early postnatal life in shaping long-term immune health and disease susceptibility. Their next steps will focus on defining the molecular pathways through which these maternal protective signals operate and exploring whether similar mechanisms exist in humans. Ultimately, this work aims to uncover new strategies for preventing autoimmune diabetes by bolstering resilience and tolerance from the very start of life.

Cracking the code of T1 diabetes

How risky immune genes trigger the attack

Eric S. Huseby, Ph.D.
Professor of Pathology.
University of Massachusetts Chan Medical School

The "Quest"

"We were puzzled because while MHC class II mainly affects CD4 T cells - a type of helper immune cell - the actual damage in the pancreas is mostly done by CD8 T cells."

The "Chain Reaction"

As a result of Praespero supported work, Dr. Huseby's team was able to secure an NIH RO1 totaling US $2.67 million.

Situation

Type-1 diabetes (T1D) is a condition where the body's immune system, specifically certain T cells, mistakenly attacks and destroys the cells in the pancreas that produce insulin, leaving people unable to regulate their blood sugar naturally.

This disease has a strong genetic component, with about half the risk tied to variations in genes within the major histocompatibility complex (MHC), particularly MHC class II genes. These genes influence how the immune system recognizes threats, and specific versions (alleles) are linked to T1D and other autoimmune diseases like lupus or rheumatoid arthritis.

"We were puzzled because while MHC class II mainly affects CD4 T cells - a type of helper immune cell - the actual damage in the pancreas is mostly done by CD8 T cells, also known as killer immune cells," says Dr. Huseby. "Our team wondered why having two copies of these risky MHC-II alleles dramatically increases T1D risk, while one copy doesn't, suggesting that these genes might indirectly trigger the destructive immune response through a chain reaction involving both types of T cells, combined with other genetic and environmental factors."

Project Overview

To explore the impact of the aforementioned T cells, Dr. Huseby's team compared T cells from people with zero, one, or two copies of the risky MHC-II alleles associated with T1D. They also created mouse models genetically engineered to mimic these scenarios, allowing detailed mechanistic studies in a controlled setting that mirrored human T1D. The journey involved analyzing how these alleles shaped the immune cell lineup, focusing on why two copies led to disease while fewer did not, through experiments tracking T cell development and behavior.

"Our team wondered why having two copies of these risky MHC-II alleles dramatically increases T1D risk, while one copy doesn't…

Project: Fate-mapping T cell responses in type-1 diabetes

Disease category: T1D and all autoimmune diseases

Impact category:

- ☑ Understanding root causes | prevention
- ☑ Advancing science towards a cure
- ☐ Improving diagnosis and treatment
- ☐ Increasing collaboration

Research Phase

Phase 6 (mid stage)

Published work

https://www.nature.com/articles/s41590-023-01441-0,

https://www.nature.com/articles/s41590-023-01446-9

Funding Impact

The key outcomes revealed that having two copies of these MHC-II alleles uniquely disrupts the normal mix of CD4 T cells, allowing a special group of highly autoimmune CD4 T cells to emerge. "These aren't directly harmful but act as "enablers" that orchestrate aggressive CD8 T cells to attack the pancreas," explains Dr. Huseby. "This doesn't happen with one or zero copies, explaining the higher risk in individuals with the two copies of the risky MHC alleles."

These insights map out a step-by-step process of how T1D develops genetically, paving the way for targeted therapies that could halt the autoimmune attack without weakening overall immunity to infections.

"Praespero funds contributed to two critical aspects of our research," notes Dr. Huseby. "First, they supported access and support for studies of individuals with T1D. Studies of human patients was a new direction for us. Second, the Research Summit has been a boon for establishing new collaborations with scientific experts from around the world."

"With recent changes in funding for USA-based research, private/foundation-based support will be more critical than ever to keep progress moving forward to stop autoimmunity from debilitating people's lives.

Dr. Huseby notes that collaborative studies between his lab and two other members of the Praespero community have recently been published.

Stopping MS at the gate (Pt 1)

How specialized immune cells at the brain's border shape inflammation and influence autoimmune disease

Vanja Lazarevic, Ph.D.
Senior Investigator
Immunopathogenesis Unit, National Cancer Institute, National Institutes of Health

The "Collaboration Victory"

"Their questions and perspectives have encouraged us to think more broadly about how immune cells communicate in these border regions and how those interactions contribute to neurological disease."

The "Innovation"

"We developed and spent years refining a highly specialized technique for visualizing immune cells within the meninges, the thin membranes that surround the brain and spinal cord. Imaging immune cells inside them requires a precise, labor-intensive method that few labs have mastered."

Situation

In earlier work, Dr. Lazarevic's team identified a rare group of immune cells known as NKp46 innate lymphoid cells (ILCs).

These cells patrol the meninges, the protective membranes surrounding the brain and spinal cord, where the immune and nervous systems intersect.

"Under normal conditions, their activity helps maintain immune balance at these critical borders," explains Dr. Lazarevic.

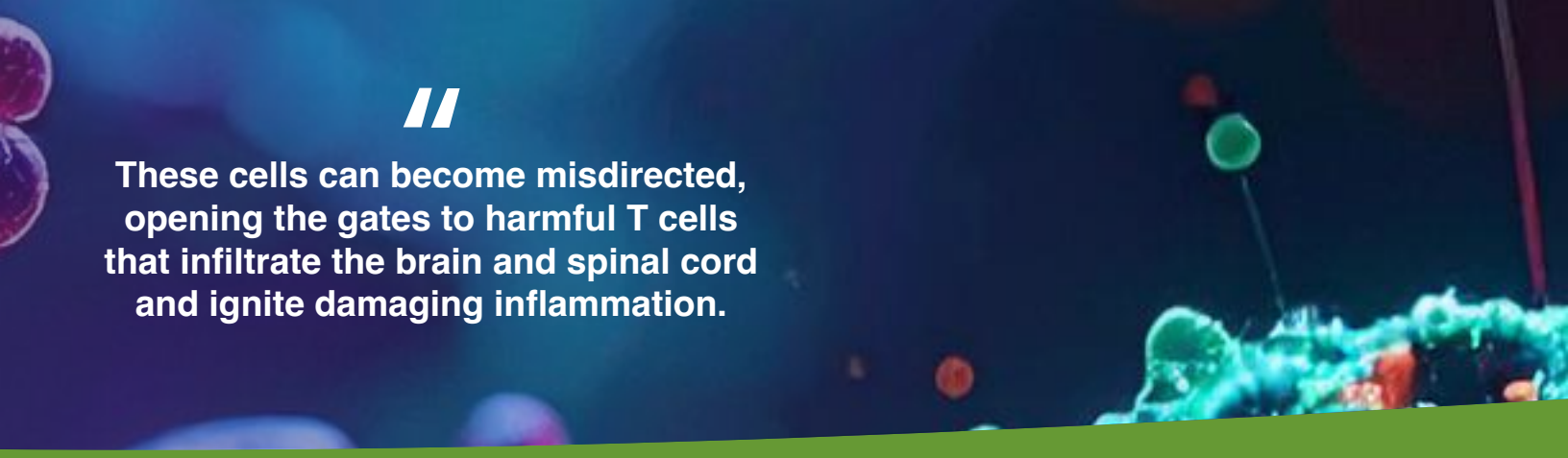
"In autoimmune diseases such as multiple sclerosis, however, these same cells can become misdirected, opening the gates to harmful T cells that infiltrate the brain and spinal cord and ignite damaging inflammation."

Project Overview

Understanding how these gatekeepers transition from protective sentinels to drivers of disease remains a central focus of Dr. Lazarevic's research. To understand how this shift from protection to disease occurs, Dr. Lazarevic's team combines genetically modified models with advanced tools such as single-cell RNA sequencing, flow cytometry, and confocal microscopy.

"Together, these approaches make it possible to trace molecular signals that allow these cells to either promote or restrain inflammation at the brain's borders," explains Dr. Lazarevic.

"By revealing how these immune circuits go awry, we hope to uncover new therapeutic opportunities to interrupt the process early, before inflammation spreads into the brain and spinal cord and causes irreversible damage."



> **These cells can become misdirected, opening the gates to harmful T cells that infiltrate the brain and spinal cord and ignite damaging inflammation.**

Project: Novel function of NKp46+ innate lymphoid cells in CNS inflammation and autoimmunity

Disease category: Multiple Sclerosis

Impact category:

- [x] Understanding root causes | prevention
- [ ] Advancing science towards a cure
- [ ] Improving diagnosis and treatment
- [ ] Increasing collaboration

Research Phase

Phase 7 (mid/late stage)

Published work

Two manuscripts are currently in preparation.

Funding Impact

Praespero's support provided the laboratory reagents needed to carry out these experiments, including antibodies to identify NKp46 innate lymphoid cells, cytokines for growing T cells, and specialized kits to isolate specific immune cell types.

"These resources enabled our team to perform studies that deepened our understanding of how immune cells at the brain's borders can fuel autoimmune inflammation," says Dr. Lazarevic.
The knowledge gained through this work continues to guide her team's current efforts to pinpoint new molecular targets and develop strategies to prevent or treat diseases such as multiple sclerosis.

> **By revealing how these immune circuits go awry, we hope to uncover new therapeutic opportunities to interrupt the process early.**

Collaboration Case Study

Praespero's Research Summit opened doors for Dr. Vanja Lazarevic and Professor Edwin Wan (see next) to study immune cell behaviour at the brain's border. "After hearing my presentation at a Praespero meeting, Edwin approached me to ask whether my lab could help his team with a highly specialized technique we had developed for visualizing immune cells within the meninges, thin membranes that surround the brain and spinal cord," says Dr. Lazarevic noting the delicate nature of these membranes. "Imaging immune cells inside them requires a precise, labor-intensive method that few labs have mastered."

(Continued on page 23)

Stopping MS at the Gate (Pt2)

Preventing harmful immune cells from entering the Central Nervous System

Edwin Wan, PhD.
Associate Professor, West Virginia University School of Medicine

The "Collaboration Victory"

"Dr. Lazarevic's and my research groups are both interested in studying immune cell interactions in the meninges during neuroinflammation…After a few years, our angle to study MS has greatly shifted."

The "Chain Reaction"

As a result of Praespero supported work, Dr. Wan's team was able to secure an additional USD $2.5 million, 5-year grant award from the National Institutes of Health in the US.

Situation

MS is an autoimmune disease initiated by the activation of central nervous system (CNS)-targeting T cells. CNS is ensheathed by three layers of cell-based layers (meninges) and is protected by the so-called blood-meningeal barrier (and a few other barriers) so T cells normally cannot enter the brain. However, during inflammatory events such as MS, T cells entered the meningeal area where they interact with other immune cells. "This interaction generates factors that compromise the blood-meningeal barrier and direct cell trafficking so that immune cells can enter and damage CNS," says Professor Edwin Wan.

Project Overview

The overarching goal of Dr. Wan's research is to identify cellular factors that promote the initiation and progression of multiple sclerosis (MS).
His team is using animal models to identify the signals that initiate cell interactions in the meninges, the molecular pathways involved, and the effector molecules that are responsible to control immune cells entering CNS.

"The moment my student showed me that T cells lacking a certain molecular signal could not leave the blood vessels and enter the meningeal area, I knew this finding held the potential to transform our strategies to treat MS in the future," says Dr. Wan, noting that future therapies targeting proteins regulated by this molecular signal could prevent T cells going into the central nervous system.

"The process of research is long and full of failures, and sometimes very discouraging,' he continues, noting that challenges include: finding the key and addressable scientific questions; securing research funding; recruiting, motivating, and training the next generation of scientists; time management, and many others. "What I learned through the process is patience, resilience, and keeping positive. Celebrate even small successes. Be passionate not only about the science but the people around me."

"The moment my student showed me that T cells lacking certain molecular signal could not leave the blood vessels and enter the meningeal area, I knew this finding might transform our strategies to treat MS in the future.

Project: Novel function of NKp46+ innate lymphoid cells in CNS inflammation and autoimmunity

Disease category: Multiple sclerosis

Impact category:

- ☑ Understanding root causes | prevention
- ☑ Advancing science towards a cure
- ☐ Improving diagnosis and treatment
- ☐ Increasing collaboration

Research Phase

Phase 2 (early stage)

Published work

https://www.pnas.org/doi/10.1073/pnas.2116256118

Funding Impact

Praespero's funds helped Dr. Wan establish a "wonderful" collaboration with Dr. Vanja Lazarevic at the National Institutes of Health.

"Dr. Lazarevic's and my research groups are both interested in studying immune cell interactions in the meninges during neuroinflammation," says Dr. Wan.

"She helped train my students in critical techniques to address our scientific questions. Now after a few years, our angle in studying MS has greatly shifted."

"In the end, remember I am part of the puzzle, not the whole, but I can do my part to finish it.

Collaboration Case Study

(Continued from page 21...)

Drawing from years of refining their technique, Dr. Lazarevic's team was able to train Dr. Wan's graduate student to perform this imaging which allowed their group to directly observe how specific immune cells behave at the brain's borders during inflammation. This arrangement not only helped advance Dr. Wan's work but also led to what she described as a very positive scientific exchange.

"Their questions and perspectives have, in turn, encouraged us to think more broadly about how immune cells communicate in these border regions and how those interactions contribute to neurological disease."

From microbes to medicines

Mining the aging gut to stop progressive MS.

Professor Jen Gommerman, University of Toronto, Department of Immunology

The "Innovation"

We validated our hunch by transferring microbiomes from old versus young humans into our model and we found that an aged microbiome could make young mice look "old" in terms of MS progression.

The "Chain Reaction"

Praespero support for this project resulted in additional funding from MS Canada.

Situation

While relapsing MS patients have a lot of treatment options, those patients who progress in their disease face a future of disability and illness for which there is no effective treatment. Part of the reason for this is that there are no good animal models that capture the transitional moment of MS progression.

Project Overview

With a lack of animal models capturing the transitional moment of MS progression, Dr. Gomerman's team developed a new model that develops progressive MS. "We noticed that this development was age-dependent," notes Dr. Gommerman.

"The breakthrough came when we theorized that the ageing process was impacting the microbiome. We validated this hunch by transferring microbiomes from old versus young humans into our model and we found that an aged microbiome could make young mice look "old" in terms of MS progression."

With that observation a pipeline was born. The team was then able to then ask why the aged microbiome promoted disease progression in a young animal, allowing them to isolate a small molecule that was responsible for this finding.I

Funding Impact

Because of Praespero funding, the team is now submitting their findings to high profiles journals.

"Praespero seed funding was exactly what we needed at the right moment to fuel our research," says Dr. Gommerman. "Ordinary funding agencies would not have wanted to fund a risky project like ours. The investment paid off!"

"The breakthrough came when we theorized that the ageing process was impacting the microbiome.

Project: Mining for microbial metabolites for treatment of progressive MS

Disease category:
Multiple sclerosis

Impact category:

- ☐ Understanding root causes | prevention
- ☑ Advancing science towards a cure
- ☐ Improving diagnosis and treatment
- ☑ Increasing collaboration

Research Phase

Phase 8 (mid/late stage)

Published work

https://www.biorxiv.org/content/10.1101/2024.05.27.595846v1

Collaboration Case Study

Dr. Gommerman calls collaboration at Praespero a "significant accelerator" for her work.

As science advances, new tools are revealing how the immune system interacts with tissues targeted in autoimmune disease, but many of these methods are "super cumbersome, very expensive, and very laborious" to set up in a single lab.

Collaboration, she says, helps bring these technologies together by pairing teams that share a question but use complementary approaches.

She adds that collaboration also strengthens reproducibility, especially when labs operate under different conditions.
Through Praespero's Innovation Award grants, Dr. Gommerman's team partners with Dr. Lisa Osborne's lab at the University of British Columbia. For every sample Dr. Gommerman tests in her model, Dr. Osborne tests it in hers.

"Whenever we think we have a really cool lead, we can be more confident that what we're seeing is real because we've cross-replicated these findings across two different labs," Dr. Gommerman says. The partnership expands the techniques they can apply and enables cross-validation—critical, she notes, for autoimmune disease research.

"We don't want to waste time and money down rabbit holes. We want to ensure we're studying something with solid foundational data, and collaboration is key to that."

"Ordinary funding agencies would not have wanted to fund a risky project like ours. The investment paid off!

When balance breaks

How Treg plasticity drives chronic inflammation in IBD and colon cancer.

Dr. Fotini Gounari & Dr. Khashayarsha Khazaie, Professors of Immunology, Mayo Clinic

The "Turning Point"

"The initial findings were not immediately embraced. Today, Treg plasticity is recognized as an important and actively investigated concept across leading immunology laboratories."

The "Chain Reaction"

These insights, which connected Treg instability to disease progression, formed the foundation for an NIH R01 award and continue to guide ongoing investigations.

Situation

This collaboration brought together two investigators with complementary motivations: Dr. Khashayarsha Khazaie is focused on translating discoveries to patient benefit and Dr. Fotini Gounari is drawn to uncovering the fundamental mechanisms of life.

Their joint effort began with a shared question - how chronic inflammation develops in inflammatory bowel disease and colorectal cancer, and the role of the immune system in those pathologies.

Project Overview

"As with many new concepts, the initial findings were not immediately embraced," says Dr. Gounari. "Novel ideas often face skepticism, but at Praespero the challenge was met with interest and constructive dialogue," adds Dr. Khazaie.

Their work led to the unexpected finding that regulatory T cells (Tregs), key controllers of immune balance, exhibit functional plasticity. They demonstrated that distinct Treg functions can be modulated independently, and that the equilibrium among these functions is crucial for maintaining immune homeostasis. Mechanistic studies revealed how tissue environments influence Treg stability and identified molecular networks governing their regulation and dysregulation in chronic inflammation.

Novel ideas often face skepticism, but at Praespero the challenge was met with interest and constructive dialogue.

Project: Mechanistic insight into de-regulation of inflammation and immunity by Tregs and microbiota.

Disease category: IBD, all autoimmune diseases, and cancer.

Impact category:

- ☑ Understanding root causes | prevention
- ☑ Advancing science towards a cure
- ☑ Improving diagnosis and treatment
- ☐ Increasing collaboration

Research Phase

Phase 7 (mid/late stage)

Published work

https://pubmed.ncbi.nlm.nih.gov/34385712/,

https://pubmed.ncbi.nlm.nih.gov/33664518/

Funding Impact

Faculty engagement and seed funding enabled completion of key preliminary experiments, leading to high-impact publications and subsequent NIH support.

"Today, Treg plasticity is recognized as an important and actively investigated concept across leading immunology laboratories," says Dr. Gournari. "It remains a central focus for translational immunologists seeking to harness or stabilize regulatory T cells for the treatment of autoimmunity and for improving outcomes in adoptive Treg therapy for transplant rejection."

These insights, which connected Treg instability to disease progression, formed the foundation for an NIH R01 award and continue to guide ongoing investigations.

Both scientists are now active members of the Mayo Clinic Arizona Treg Group, established by the Dean of Research to advance understanding of autoimmunity and transplant rejection.

Today Treg plasticity remains a central focus for translational immunologists seeking to harness or stabilize regulatory T cells for the treatment of autoimmunity…

"Support from Praespero was instrumental in enabling us to help establish a new area of immunology research with significant translational relevance for the diagnosis and treatment of autoimmunity," adds Dr. Khazaie.

Why some RA patients flare after COVID infection or vaccination

How stimulated double-negative T cells translate to disease activity.

Alessandra Franco MD PhD
Professor, University of California San Diego.

The "Discovery"

"An unexpected moment in the study was discovering a large number of unusual immune cells in RA patients."

Research phase

Phase 9 (end stage)

Published work
https://pubmed.ncbi.nlm.nih.gov/41416941/

Situation

Although mRNA vaccines for COVID-19 protection have been effective in protecting patients with rheumatoid arthritis (RA) against SARS-CoV-2 infection, some patients with the disease had flare-ups of their disease after vaccination, and some people developed signs of autoimmunity after getting COVID-19 itself. There were also people with no prior autoimmune problems who showed temporary or lasting autoimmune reactions after mRNA vaccines.

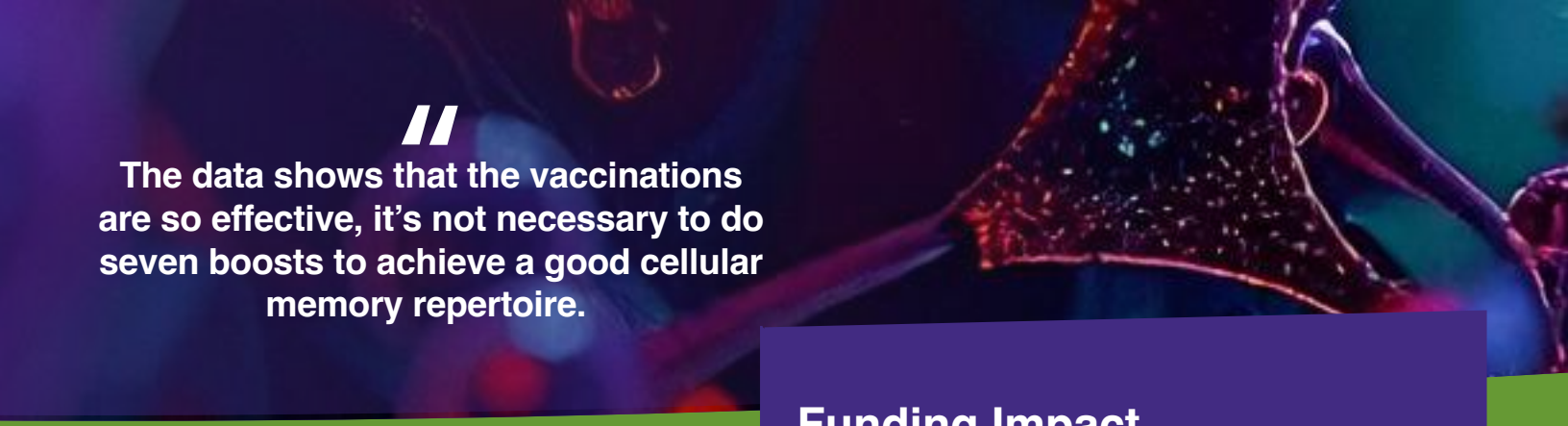
With support from Praespero, Dr. Franco's team studied how the immune system of people with RA responds to the virus after vaccination.
The goal of the research was to determine: a) the impact of medications that suppress the immunity in the ability to respond to vaccinations; b) the development of cellular T cell memory depending upon number of vaccine injections, c) the expansion of virus-specific regulatory T cells after vaccination; d) the immune phenotype of RA patients after mRNA-based vaccination for COVID-19 protection.

Project Overview

The study population included subjects that uptake diverse medication to suppress inflammation, and that received different numbers of vaccine boosts.

The researchers took T cells from the participants' blood and grew them in a lab setting. They exposed these T cells to tiny protein pieces from different parts of the SARS-CoV-2 virus (both the spike protein, which helps the virus enter cells, and other non-spike areas). Afterward, they collected the T cells and examined how they behaved and what characteristics they showed.

"The results showed the different types of immune suppressive medications that RA patients were undergoing at the time of vaccination had no negative impact on the effectiveness of the vaccine nor on the development of virus-specific regulatory T cells," says Dr. Franco.
They also found that the number of vaccine doses a patient received—from just one up to seven—didn't affect the strength of the immune system's cellular



> **The data shows that the vaccinations are so effective, it's not necessary to do seven boosts to achieve a good cellular memory repertoire.**

Project: How SARS-CoV-2 spike-specific regulatory T cells control auto-immunity and prevent clinical relapses in mRNA vaccine recipients with rheumatoid arthritis.

Disease category: Rheumatoid arthritis

Impact category:

- ☑ Understanding root causes | prevention
- ☐ Advancing science towards a cure
- ☐ Improving diagnosis and treatment
- ☐ Increasing collaboration

Funding Impact

"Praespero's support allowed the stability of our laboratory," says Dr. Franco. The funds helped her explore the cellular immune response (in autoimmunity) to the SARS-CoV-2 virus that causes COVID-19. The work suggested that immune suppressive therapies did not affect the response to vaccination in rheumatoid arthritis patients: T cell memory and spike-specific regulatory T cells developed as early as after a single vaccine injection. However, repeated T cell stimulations (multiple vaccine doses or COVID-19) may cause the differentiation of double negative T cells into cytotoxic T cells, useful for anti-viral protection but also potentially harmful in RA. The results may explain clinical relapses after vaccination in some, and the occurrence of autoimmunity after vaccination (or COVID-19 infection) in previously healthy subjects.

"memory" of the virus, meaning how well it could recall and respond to it later.

"The data shows that the vaccinations are so effective, it's not necessary to do seven boosts to achieve a good cellular memory repertoire," says Dr. Franco.

An unexpected moment in the study was discovering a large number of unusual T cells in RA patients. These are called double-negative T cells (or DN T cells), which lack two common T cell markers (CD4 and CD8) that help classify T cells.

In traditional immunology, Dr. Franco explains, these T cells are usually seen as immature and not yet fully differentiated in mature T cells. But in this study, DN T cells were active, circulating in the blood, and they could recognize pieces of the SARS-CoV-2 virus. When exposed to those virus fragments in the lab, DN T cells differentiated into a type of killer T cell (CD8+ cytotoxic T cells) that may cause inflammation.

"Cytotoxic T cells are harmful cells, in the sense that they are designed to clear tumors and viruses," explains Dr. Franco. "What we saw is that, in responding to the SARS-CoV-2 virus the DN T cells are becoming pro-inflammatory T cells, which might lead to tissue damage in affected patients."

These astonishing and unexpected findings suggest that this process could make autoimmune conditions like rheumatoid arthritis worse. It might even trigger new inflammation in otherwise healthy people, especially after getting multiple vaccine boosters – a finding that can potentially guide doctors to more patient-centered protection and treatment protocols.

"Every patient is different; what I describe is a potential harm for some patients but it doesn't mean that it will happen in everyone," notes Dr. Franco.

> **Praespero's support allowed the stability of our laboratory.**