

2026 CANADA GAIRDNER AWARD WINNERS

Explore the science behind today's biggest
breakthroughs



ABOUT GAIRDNER

The mission of the Gairdner Foundation is to celebrate, inform and inspire scientific excellence around the globe.

Established in 1957, the Gairdner Foundation is dedicated to fulfilling James A. Gairdner's vision to recognize major research contributions to the treatment of disease and alleviation of human suffering. Through the prestigious annual Canada Gairdner Awards, the Foundation celebrates the world's most creative and accomplished researchers whose work is improving the health and wellbeing of people around the world. **The Canada Gairdner Awards are recognized internationally as being among the most prestigious in the world, based on our reputation for rigorous review and early recognition of breakthroughs in science.**

The Gairdner Foundation brings people together to openly discuss science in order to better engage the public, understand the problems we face, and work together to find solutions. Through Gairdner Connects, our national outreach program, we bring science to communities across Canada to inspire future innovators and spark public dialogue about the role of research in addressing the world's most pressing health challenges.

BY THE NUMBERS

- 5** CANADA GAIRDNER **International Awards**
For excellence in biomedical research
- 2** PETER GILGAN CANADA GAIRDNER **Momentum Awards**
For mid-career investigators with exceptional research
- 1** JOHN DIRKS CANADA GAIRDNER **Global Health Award**
For outstanding achievements in global health research

443 AWARDS to scientists from over **40** countries

103
Gairdner awardees have gone on to win the **Nobel Prize**



20 CANADIAN UNIVERSITIES | **6000+** ENGAGING students and researchers annually through outreach programs that give Canadians access to world-renowned scientists



ABOUT THE COLLECTION

This article is part of a larger collection introducing the discoveries of the 2026 Canada Gairdner Award winners, among the world's leading biomedical and global health researchers. From the inner workings of our cells to new approaches to preventing disease and improving treatment, these stories explore research shaping the future of human health.

To explore the other stories in this series, [check out the full collection here](#). By viewing the full collection, you'll discover the science behind this year's award-winning research and explore big questions such as:

- How can viruses help us fight antibiotic-resistant bacteria?
- How can technology help regulate blood pressure after spinal cord injury?
- How can we stop misfolded proteins from causing disease?
- How can scientists identify, measure and study thousands of proteins?
- How can scientists use 3D images to see inside cells?
- How can oral vaccines help prevent cholera?

ABOUT THE CREATORS

Each article was written by Gairdner student intern **Leanne Cherry**, an undergraduate student at McGill University studying Cognitive Science and Communication Studies, and developed in collaboration with the Award winners themselves. Passionate about science communication, Leanne is interested in making research accessible and combating science and health misinformation.

The articles were also reviewed by the students from the **Canadian Association of Girls in Science (CAGIS)**, whose thoughtful feedback helped make these stories clear, accurate and engaging for young readers.

Illustrations were created by **Heather Ng-Cornish**, an illustrator and communicator based in Ontario. Heather brings her science communication experience to her illustrations, creating clear, colourful and accessible visuals that help bring scientific ideas to life. To see more of her work, visit www.heatherngcornish.com

The Gairdner Foundation is committed to sharing the excitement of discovery with people of all ages. We hope this collection sparks your curiosity, encourages you to ask big questions, and inspires you to imagine the discoveries still waiting to be made.



LEANNE
CHERRY



HEATHER
NG-CORNISH



PREVENTING PROTEIN MISFOLDING: THE BATTLE AGAINST TOXIC PROTEIN CLUMPS

PROF. JEFFERY W. KELLY

CANADA GAIRDNER INTERNATIONAL AWARD WINNER

H. Lutchter Brown Professor of Chemistry, Department of Chemistry, Scripps Research, La Jolla, United States

SUMMARY

Prof. Jeffery W. Kelly was the first person to develop a medication that successfully slowed the progression of a human amyloid disease—a kind of disease which occurs when our proteins undergo shape or structural changes, allowing them to misassemble and accumulate in clumps throughout the body causing organ damage. Kelly was also the first to show that we can prevent some of the harmful effects of these diseases if we can stop the proteins from undergoing shape changes in the first place. Kelly's work represents a massive step forward in our conceptual understanding and our treatment methods for these diseases.

STUDENT REVIEWERS:



ARSHIA
Grade 10



CYNTHIA
Grade 10



JUNE
Grade 11



VALINI
Grade 11

Amyloid diseases occur when abnormally-shaped proteins build up around the brain and body and form toxic clumps, called **amyloid aggregates**. Over time, amyloid aggregates degrade a person's health, often leading to bodily dysfunction and deterioration, and eventually, death. While amyloid diseases are difficult to study, researchers have made huge progress in understanding their underpinnings, in some cases even treating them.

Prof. Jeffery W. Kelly, a chemistry professor at *Scripps Research Institute* in California, created the first ever medication to successfully slow the progression of a human amyloid disease. More specifically, Kelly designed a drug to help manage **Transthyretin amyloidosis (ATTR)**—the third most common amyloid disease, behind Alzheimer's and Parkinson's diseases. ATTR occurs when the protein transthyretin forms into amyloid aggregates around the heart and nerves.

AMYLOID AGGREGATION

The process of misfolded proteins becoming the toxic clumps called amyloid aggregates.

AMINO ACID

Chemical building blocks which join together to form protein. In the case of transthyretin, the amino acid chains first form into the smaller monomers, and then the four monomers come together to create transthyretin.

Kelly developed the drug after a foundational discovery made in his laboratory: that changes in a protein's shape or structure are enough to kickstart toxic protein build-up. Beyond slowing the progression of ATTR, Kelly was also the first to definitively prove that **amyloid aggregation** fuels neurodegeneration—the loss of neurons in the brain and/or outer nervous system often seen in amyloid diseases. Kelly's discoveries have propelled the study of human amyloid disease forward, fundamentally changing how we think about research and drug development in this field.

WHEN A PROTEIN'S STRUCTURE BECOMES HARMFUL

The **transthyretin protein** is made of four identical pieces, called monomers, that join together to form a single protein. Transthyretin is a kind of transport protein, and its function is to deliver hormones and vitamins around the body.

Our cells make proteins by stringing together several small molecules called **amino acids**. Normally, this string of amino acids folds to produce a protein of a specific 3D structure which ultimately allows a protein to do its job. With ATTR, however, a person's transthyretin protein becomes unstable and splits into individual monomers, meaning transthyretin loses its proper structure. When detached, the monomers can misfold into abnormal shapes which form large assemblies or clumps with other misfolded monomers (**Figure 1**). The bonds holding these clumps together are incredibly strong, making it difficult for our bodies to break them down.

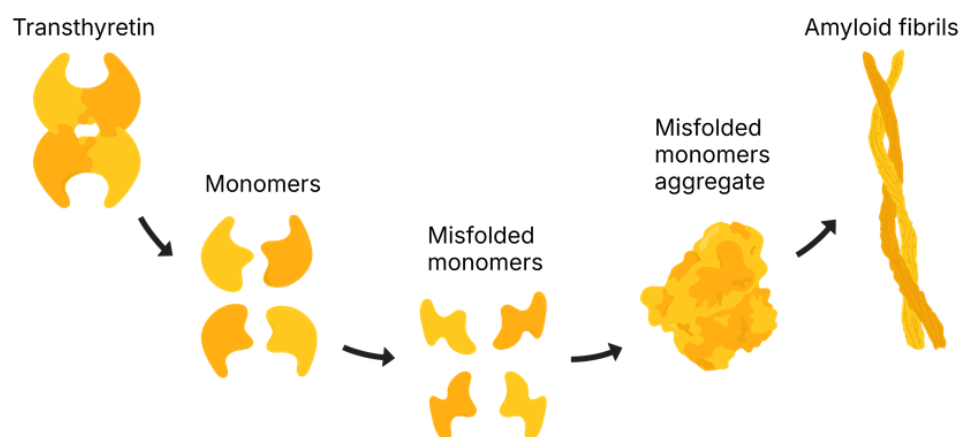


FIGURE 1

Transthyretin proteins split into monomers. The monomers misfold and aggregate which create amyloid fibrils.

Over time, these misfolded protein clumps build up around the heart and throughout the nervous system, forming into long, twisting amyloid fibrils (Figure 1). These aggregates can lead to issues like an irregular heartbeat, shortness of breath, and tingling or numbness in the toes and feet (**Figure 2**). Because these symptoms are common to many other illnesses, ATTR can be difficult to diagnose. If left untreated, the condition is ultimately fatal.

UNCOVERING THE HARM OF AMYLOID AGGREGATES

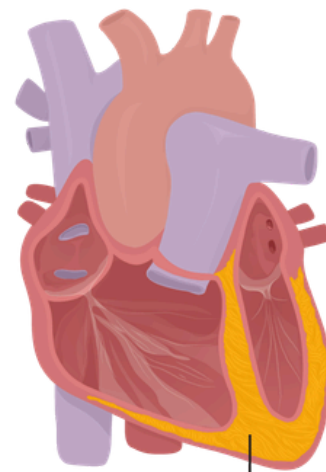
Kelly was the first to prove that transthyretin aggregation is responsible for the neurodegeneration seen in ATTR. He discovered this by revealing something about our DNA—the molecular information encoded deep within us that makes us who we are.

DNA is grouped into segments called genes. Some genes act as instruction manuals for building particular proteins, telling our cells which amino acid to use and where to place it when creating a protein. A protein's amino acids determine the protein's final structure and function, and any change to the order of amino acids—or the underlying instruction manual—can completely alter how the protein will form.

Humans generally have two copies of every gene, with one coming from each parent. One of the ways a person can develop ATTR is through a mutation to one copy of the gene which codes for the transthyretin protein, passed down through their family tree.

This version of ATTR is called hereditary or **variant ATTR (ATTRv)**. For someone with ATTRv, a genetic mutation on one copy makes the transthyretin protein highly unstable. It breaks apart into monomers more easily, which can ultimately lead to the formation of amyloid aggregates that damage the body.

However, during his research, Kelly noticed a different mutation that can occur on the transthyretin genes—one which seemed to prevent the harmful effects of ATTR from developing. In rare cases, people with ATTRv can have mutations in both gene copies: one mutation that leads to amyloid aggregation and one that is protective against amyloid disease. Kelly found that people who had both mutations did not show the typical neurological symptoms of the disease. It turns out that this second mutation counteracted the effects of the first by stabilizing the transthyretin protein. Because the protein remained stable, it was much less likely to break into monomers, therefore preventing it from misfolding and clumping with other monomers to form amyloid aggregates.



Amyloid aggregates
in the myocardium

FIGURE 2

Amyloid fibrils (yellow) build up in the myocardium of the heart.

By showing that preventing aggregation also prevented disease symptoms, Kelly demonstrated that amyloid aggregates are a key driver of nerve damage in ATTR. Using what he discovered here, Kelly was then able to figure out how to create a medication that would slow ATTR's progression down—and he was the first person to ever do so.

THE IDEA BEHIND KELLY'S MEDICATION

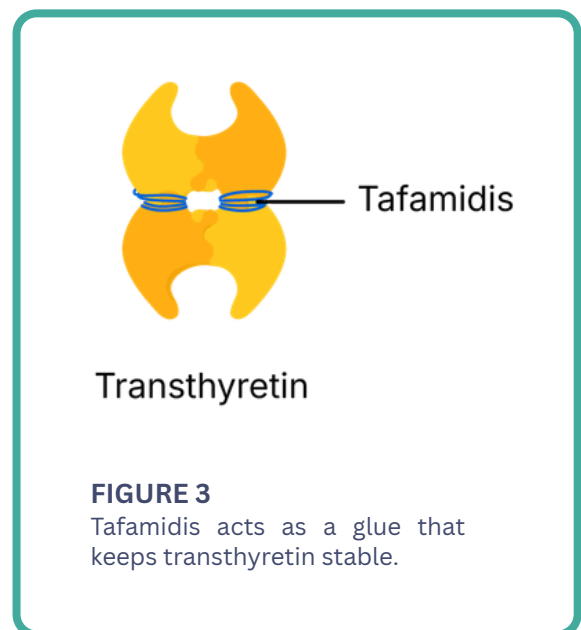
Tafamidis, the ATTR drug that Kelly created, essentially keeps its target protein transthyretin intact. Because transthyretin is a transport protein, it contains various sites that different molecules can bind to. Two of these sites are right at the centre of the protein's four subunits. When one or two small molecules bind here, they act almost like a glue for transthyretin, holding its four subunits together. When this site is empty, the protein is naturally less stable, but under normal conditions this does not cause any issues. However, for someone with ATTR, it is a completely different story.

As mentioned, ATTRv is caused by an inherited genetic mutation. A person can also develop ATTR as a result of aging, and in this case, it is called wild-type ATTR (ATTRwt). For these patients, their transthyretin is normal in terms of the amino acids comprising it, but it nonetheless misfolds and leads to amyloid aggregation upon aging for reasons that are not yet understood.

Kelly's idea behind his drug, tafamidis, was that it would bind to transthyretin's empty binding site(s). By acting like a molecular glue, it helps hold the transthyretin protein together to prevent it from falling apart, becoming misshapen, and misassembling into toxic amyloid aggregates (**Figure 3**). After formulating this theory, Kelly and his team ran several human clinical trials to find out how well the drug would work in practice.

They found that when ATTR patients took tafamidis, it slowed or stopped amyloid aggregation around the body, including in the nervous system and the heart. The early phases of these trials demonstrated that tafamidis helped slow neurodegeneration, while the final phases confirmed that tafamidis decreased death rate and reduced the number of hospitalizations due to ATTR-related heart issues.

Tafamidis has since been approved for usage in over 50 countries. Its effectiveness further confirmed the idea that amyloid diseases can be slowed by stabilizing proteins before they have a chance to fall apart and form aggregates.



BIGGER PICTURE: IMPACTS ON OTHER AMYLOID DISEASES

Beyond transforming the treatment of ATTR, Kelly's work fundamentally changed how scientists and physicians think about amyloid diseases more broadly. Researchers have long debated whether amyloid aggregation is the cause of the bodily degradation and organ system deterioration characterizing these diseases, or merely a symptom of a different underlying issue. Skepticism toward this "**aggregation hypothesis**"—which blamed a complex spectrum of protein clusters for the neurodegeneration seen in Alzheimer's—grew following repeated failures to treat the disease with aggregation-targeting medications.

Part of the difficulty with proving or disproving the amyloid hypothesis also has to do with where the amyloid aggregates form. In Alzheimer's disease, they form directly in the brain, which is part of the central nervous system. Brain tissue is not easy to study in living patients, and often we must wait until after a patient has died to actually see the aggregates. While we have imaging techniques that help us get around this today, many questions about Alzheimer's and amyloid aggregation remain unanswered.

However, ATTR is different from other amyloid diseases. Amyloid aggregation in ATTR starts in the heart and in the peripheral nervous system—the network of nerves that lies beyond our brain and spinal cord—only accumulating in the central nervous system as the disease progresses. The fact that the disease starts outside the central nervous system enabled Kelly to observe the toxic effects of ATTR's amyloid aggregates more easily than if he had been observing the effect of Alzheimer's aggregates, which is concentrated in the brain.

Kelly's discoveries about amyloid aggregates and his drug to combat them has now motivated the development and regulatory agency approval of nine additional protein aggregation inhibiting and/or modulating drugs. These slow down the progression of other amyloid diseases such as Alzheimer's.

The ability to ameliorate these diseases with medication represents a huge step forward in both our conceptual understanding of and our treatment for amyloid diseases. Thanks to Kelly and his team, ATTR patients are able to increase the quality and the length of their lives, and amyloid disease researchers now have a promising direction to head in when developing new medications for amyloid diseases currently lacking drugs.

Article written by Leanne Cherry

Illustrated by Heather Ng-Cornish

This article has been reviewed by Dr. Jeffery Kelly
and colleagues for accuracy and accessibility.



MEET THE SCIENTIST: Prof. Jeffery W. Kelly

Jeffery W. Kelly is the H. Lutchter Brown Professor of Chemistry at Scripps Research. He earned his Ph.D. in Chemistry from the University of North Carolina and completed postdoctoral training at Rockefeller University. His laboratory studies how proteins fold, misfold and aggregate, processes associated with amyloid and neurodegenerative diseases. Kelly's research on transthyretin amyloidosis led to the development of tafamidis, a medicine that slows disease progression by stabilizing the transthyretin protein and preventing its aggregation. His laboratory continues to investigate new treatments for diseases caused by protein misfolding. Kelly is a member of the U.S. National Academy of Sciences and American Academy of Arts and Sciences.



YOUTH ACCESSIBILITY REVIEW

This article was reviewed by high school students through the Canadian Association for Girls in Science (CAGIS)—Canada's largest and longest-running STEM (science, technology, trades, engineering, and math) club for girls and gender-diverse youth aged 7-17. This award-winning club, founded in 1992, supports interest in STEM with virtual programming and local clubs that visit labs, workshops, and field sites to meet mentors and do fun, hands-on activities. Their thoughtful feedback on clarity, accessibility, and reader engagement helped shape the final version of this article and ensure it is engaging and accessible for youth readers.



Arshia Nair • Grade 10

"I am interested in the science of neurodegenerative diseases, which fuels my desire to pursue a career in healthcare."



Cynthia Rayson • Grade 10

"I hope to study biomedical engineering and develop new dental technologies to enhance patient care."



Valini Parasram • Grade 11

"In the future I hope to study chemistry and its nuclear applications."



June Liu • Grade 11

"I aspire to pursue a career in healthcare or biomedicine with a focus on applied research and potentially business, re-imagining the way we approach real world issues with practical and innovative solutions."

GAIRDNER CONNECTS

Gairdner Connects brings award-winning scientists to communities across Canada to engage, inspire, and spark dialogue. In doing so, it fosters meaningful connections between Gairdner Award winners and the next generation of researchers. **Through interactive events, classroom visits, community discussions, and digital resources, we inspire future innovators and spark dialogue about the role of science in addressing global health challenges.**

This article, developed in partnership with the **Canadian Association for Girls in Science (CAGIS)**, is part of the Gairdner Foundation's student outreach program to bring science to communities across Canada.

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