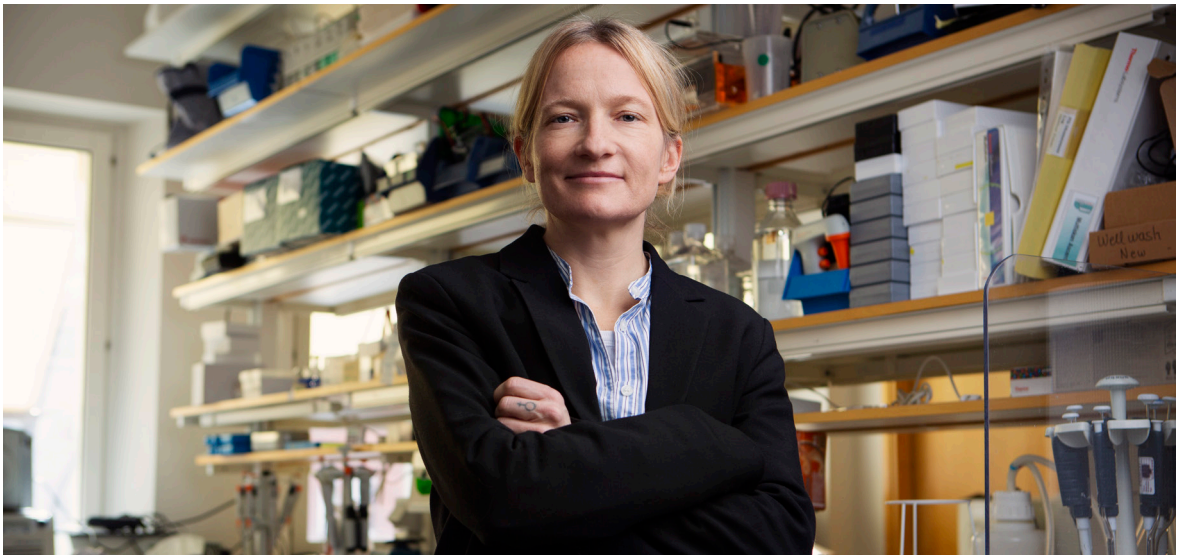
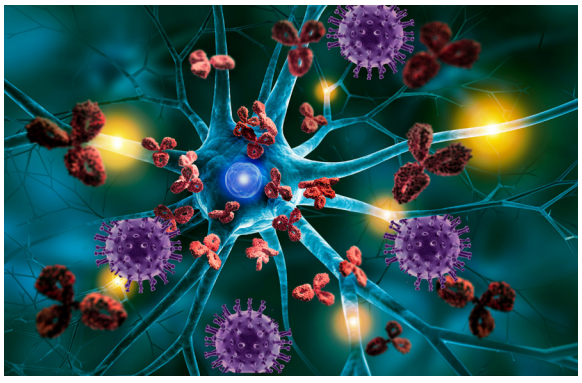
No. 1 / 2026
Center for Molecular Medicine

CMM News



Page 2-3

Birgitta & Sten Westerberg donate to Ida Nilsson's research: 'What happens in the brain when someone has anorexia?'



Page 10

Publication in *Cell*: Anoctamin-2 -specific T cells link EBV to MS

Page 13

CMM Director's update: plans and development in 2026

Download this newsletter here

Editor: Magdalena Lindén
Layout: Edna Fagerstedt

CMM

What happens in the brain when someone has anorexia?

Image: iStock.

FUNDING AND GRANTS

How can we break self-starvation in anorexia nervosa? With the support of a donation from Birgitta & Sten Westerberg, Ida Nilsson and her research group are trying to investigate how normal starvation signals and responses are malfunctioning in the brain of patients with anorexia.

Birgitta & Sten Westerberg have donated to research at Karolinska Institutet on several occasions. In 2021, they wanted to donate money to eating disorder research, and Ida Nilsson's research project was selected. Now, her research support from the couple has been extended by an additional 3.6 million SEK over three years.

Anorexia nervosa (AN) is one of the deadliest psychiatric disorders. The genetic heritability is estimated to be between 50 and 80 percent, meaning that twin studies have shown that if one twin develops AN, the other has a 50 to 80 percent risk of doing so as well. Since there is not enough knowledge about the neurobiological mechanisms behind AN, no adequate pharmacological treatments have been developed for the disorder. The study to which the donation relates aims to understand the molecular mechanisms that underlie and perpetuate self-starvation in AN.

Ida and her research team have developed cellular models that will enable studies of how the communication between cells in the brain is affected in patients with AN. They are working on the hypothesis that there is a malfunction in the communication between nerve cells and microglia. Microglia are a type of immune cell in the brain whose function includes “eating up” nerve connections, known as synapses, that are no longer needed or used. This process is called “synaptic pruning.” In AN, researchers believe that something has gone wrong in the synaptic pruning in the hypothalamus, the region of the brain that is important for regulating appetite

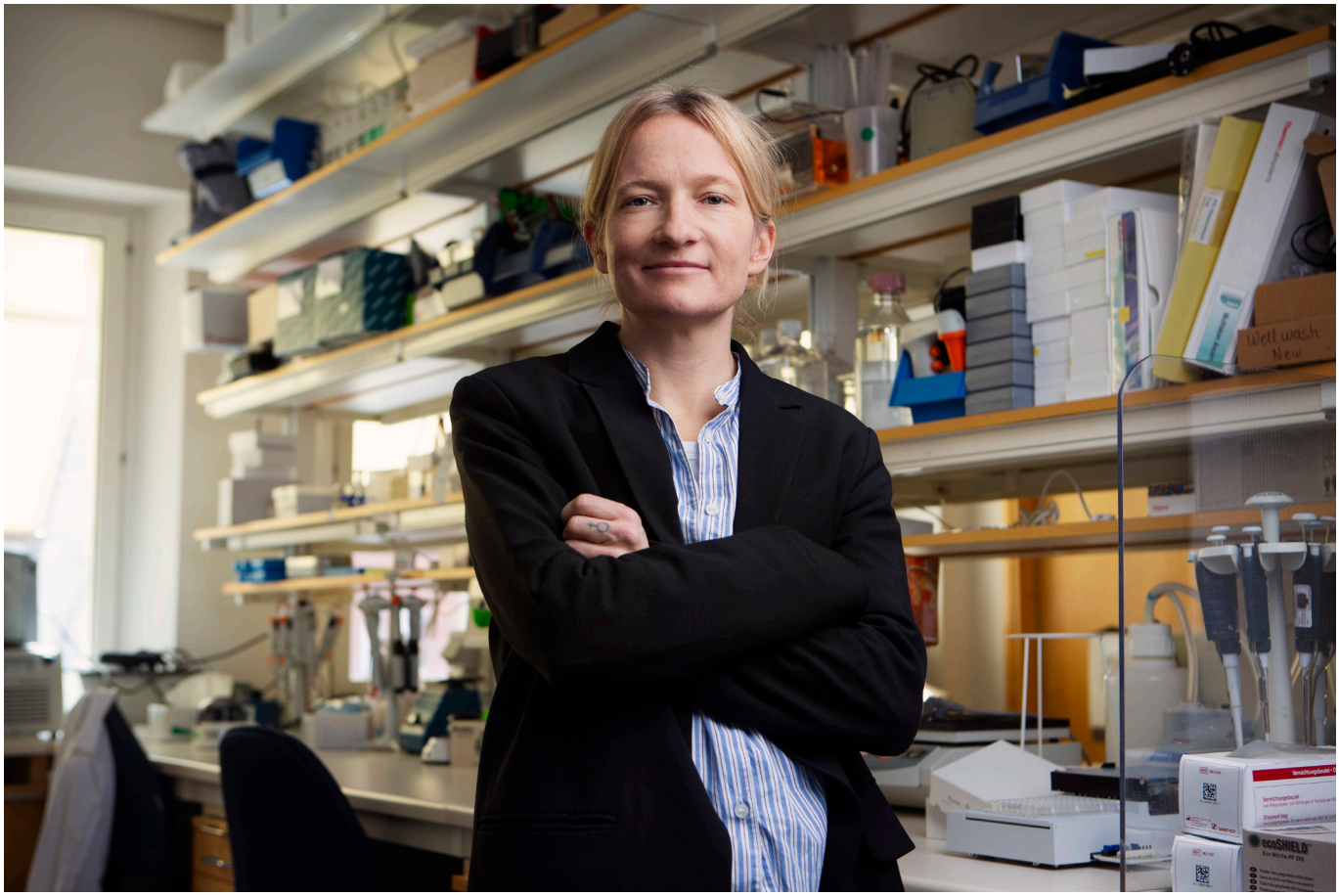
About the donation from Birgitta & Sten Westerberg

Recepient: Ida Nilsson, associate professor, Department of Molecular Medicine and Surgery, Karolinska Institutet, CMM Group Leader.

Project title: 'Hur kan vi bryta självsvält? – studier av neurobiologiska mekanismer bakom anorexia nervosa' (How can we break self-starvation? – studies of the neurobiological mechanisms behind anorexia nervosa).

Amount: SEK 3.600,000

Period: 2026-2029

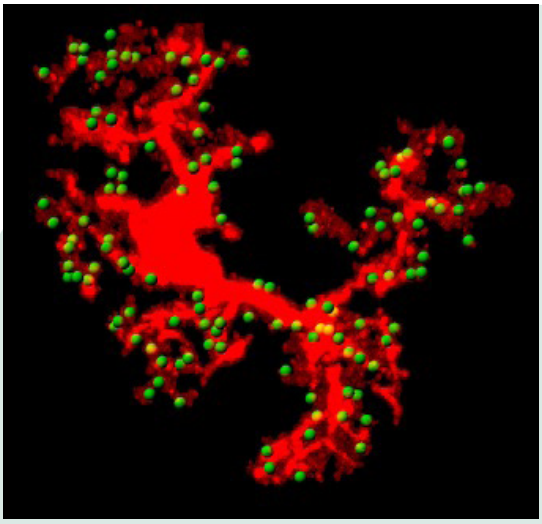


Ida Nilsson. Photo: Karolinska Institutet.

and food intake, which leads to the brain not responding to starvation in the same way as healthy individuals.

The cell model Ida Nilsson is working with consists of patient-derived stem cells (induced pluripotent stem cells, iPS) that have been reprogrammed to become hypothalamic nerve cells and microglia. Since genetics are so important in AN, it is crucial that the cells have the same genetic makeup as the patients, which has been achieved with this model. Through the model, the researchers hope to gain an *in vitro* insight into what the hypothalamus looks like in patients suffering from AN. Among other things, they measure what the cells secrete, what activity they have, how their shape changes under different conditions, and how much synaptic pruning takes place, i.e., how much microglia eat up the hypothalamic synapses.

"The long-term goal is to develop a drug that affects the neurons and reactivates hunger, so that we can help patients with anorexia regain weight and learn to eat at normal intervals and in normal portions", says Ida Nilsson.



Synaptic pruning occuring in hypothalamic neurons. Image: Ida Nilsson.

New CMMers

Soham Deolankar is a new PhD student in Fredrik Wermeling's Team. He holds a BSc in Biology (Major) and Chemistry (Minor) from the Indian Institute of Science Education and Research (IISER), Pune, India, as well as a MSc in Biology from IISER. He performed his master thesis at UTMB Galveston, Texas, USA. He has also worked as a research assistant at the Translational Biology Department at Aurigene Onclology, 2024-2025.

David Valero recently joined Onur Parlak's Team as a postdoctoral researcher. He holds a PhD in Analytical Chemistry with expertise in nanomaterials and bioanalytical platforms for decentralized diagnostics. His research focuses on developing portable and wearable biosensing devices for point-of-care applications, with the goal of translating innovative technologies into tools for personalized healthcare. Beyond the laboratory, he enjoys long walks with his dogs, practicing sports, traveling, and discovering new restaurants.

Lorenza Di Marsico is a third-year PhD student in Molecular Medicine at the University of Pisa and a visiting PhD student in Sebastian Lewandowski's Team. She will be at CMM for six months to further investigate vascular aspects associated with amyotrophic lateral sclerosis. Her research focuses on the retina as a model to study vascular and neurodegenerative diseases, using experimental models to identify novel therapeutic targets and highlighting the retina as a window into the central nervous system.

Welcome, all new CMM'ers!

New Group Leader

Ida Nilsson has been appointed a new CMM Group Leader as of January 28th 2026. The research focus of her group is the neurobiology of anorexia nervosa.

Associate professor Ida Nilsson's research group investigates the biological mechanisms underlying anorexia nervosa (AN)—a psychiatric disorder with exceptionally high mortality and persistent treatment challenges. Their work focuses on understanding why individuals with AN continue to lose weight despite severe starvation, in contrast to normal physiological responses where weight loss triggers increased appetite and reduced energy expenditure.

Current evidence, including recent genetic findings, suggests that people with or at risk for AN may have a biologically driven lower body-weight set point. The team's overarching hypothesis is that key energy-regulating neurons in the hypothalamus, particularly arcuate nucleus (Arc) neurons, are dysregulated in AN, especially in how they communicate with surrounding microglia. This disrupted neuro-immune interaction may help explain the persistent negative energy balance characteristic of the disorder.

To test this hypothesis, the group uses a multi-model approach, including preclinical models, clinical samples, and patient-derived stem cell models. By defining the molecular pathways that control food intake and weight regulation in anorexia, their goal is to identify new drug targets that could promote sustainable renourishment and healthy weight gain—critical components for both acute treatment and long-term recovery.

Where to find them: Floor 01 @CMM.

Contact: ida.nilsson@ki.se

Current group members:
Jingjing Xu (PhD), Emmy Erskine (PhD student), Barbara Eramo (postdoc), Susanna Parolaro (visiting PhD student).

Marie Skłodowska-Curie grants

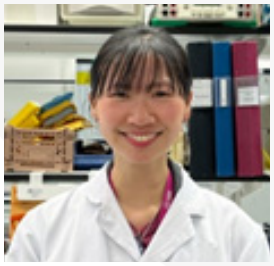
FUNDING AND GRANTS

Two young researchers at CMM have been awarded postdoctoral grants within the EU's Marie Skłodowska-Curie Actions (MSCA). The competition has been particularly intense in this year's call.

The Marie Skłodowska-Curie Actions (MSCA), part of the EU's Horizon Europe programme, support doctoral and postdoctoral training by promoting young researchers' independence, international mobility, and the development of innovative ideas in leading research environments.

MSCA grants typically last two years and provide funding for salary, research expenses, and mobility, offering recipients stability and the resources needed to advance their research projects.

Awarded researchers and projects at CMM:

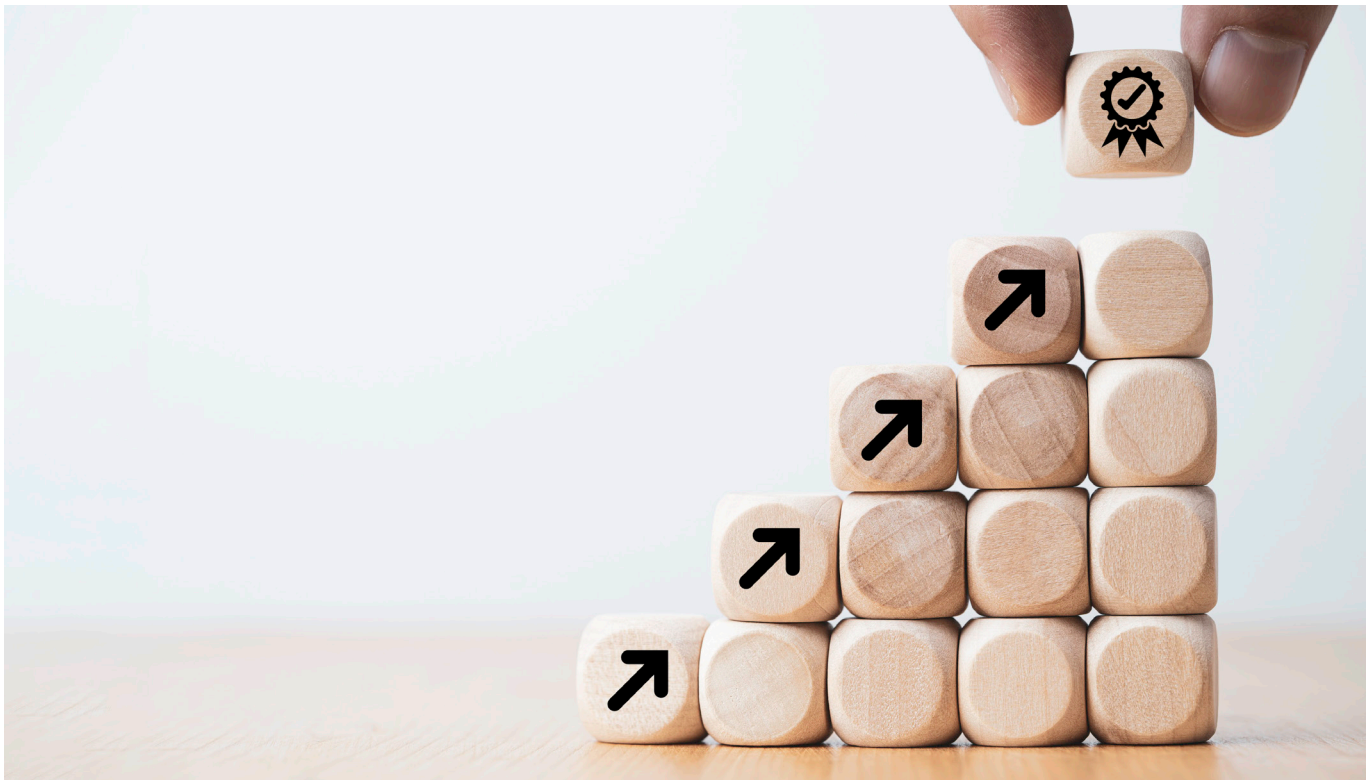


Cheuk Yau (Jane) Luk.
Photo: Private.

Projekt IMAPERFAT: Unmasking the perivascular adipose tissue's role in diabetic vascular disease from molecular clues to clinical impact.
Postdoctoral fellow: Cheuk Yau (Jane) Luk
Principal investigator: Carolina Hagberg, CMM Group Leader.

Mora Massaro.
Photo: Private.

Project PLASMA-HEAL: Exploring plasma-cell role in mucosal healing
Postdoctoral fellow: Mora Massaro
Principal investigator: Eduardo Villablanca, CMM Group Leader.



Some Publications

CMMers IN BOLD

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Di Martino E, Vacondio D, Arroyo-García LE, Bedin M, Balasco L, Schmeh I, **Pieber M,** Kukla A-K, Mukai T, Tsuji S, **Zhang X-M**, Wickström R, Blomgren K, **Harris RA**, Ådén U. Delayed adoptive transfer of bone marrow-derived macrophages modulates post-ischemic inflammation in a model of neonatal hypoxia-ischemia. *Brain Behav Immun* 2026 Jan;131:106179. doi: 10.1016/j.bbi.2025.106179. Epub 2025 Nov 10.

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Niu G, **Geara J, Chen Y, Luo L,** Xiao Y, **Liu Z,** Gaborieau M, Pan L, Loh E, Li D, Sommar P, Wang A, Zheng X, **Landén N**. Mitochondrial CircRNA CircMT-RNR2 safeguards antioxidant defense to support fibroblast functions in wound repair. *Adv Sci,* online 8 February, 2026, doi: 10.1002/advs.202517141

Pahlevan Kakhki M, Rangani F, Ewing E, Starvaggi Cucuzza C, Zheleznyakova G, Kalomoiri M, Kenny L, Raghavan A, Rao Prakash C, van den Hoeven G, Venkata S Badam T, Covacu R, Andreou I, Needhamsen M, Kular L*, Jagodic M*. Comprehensive profiling of CRISPR/dCas9 epigenome editors indicates a complex link between on and off target effects. *Genome Biol.* **2026 Jan 31;27(1):52. doi: 10.1186/s13059-026-03967-6.**

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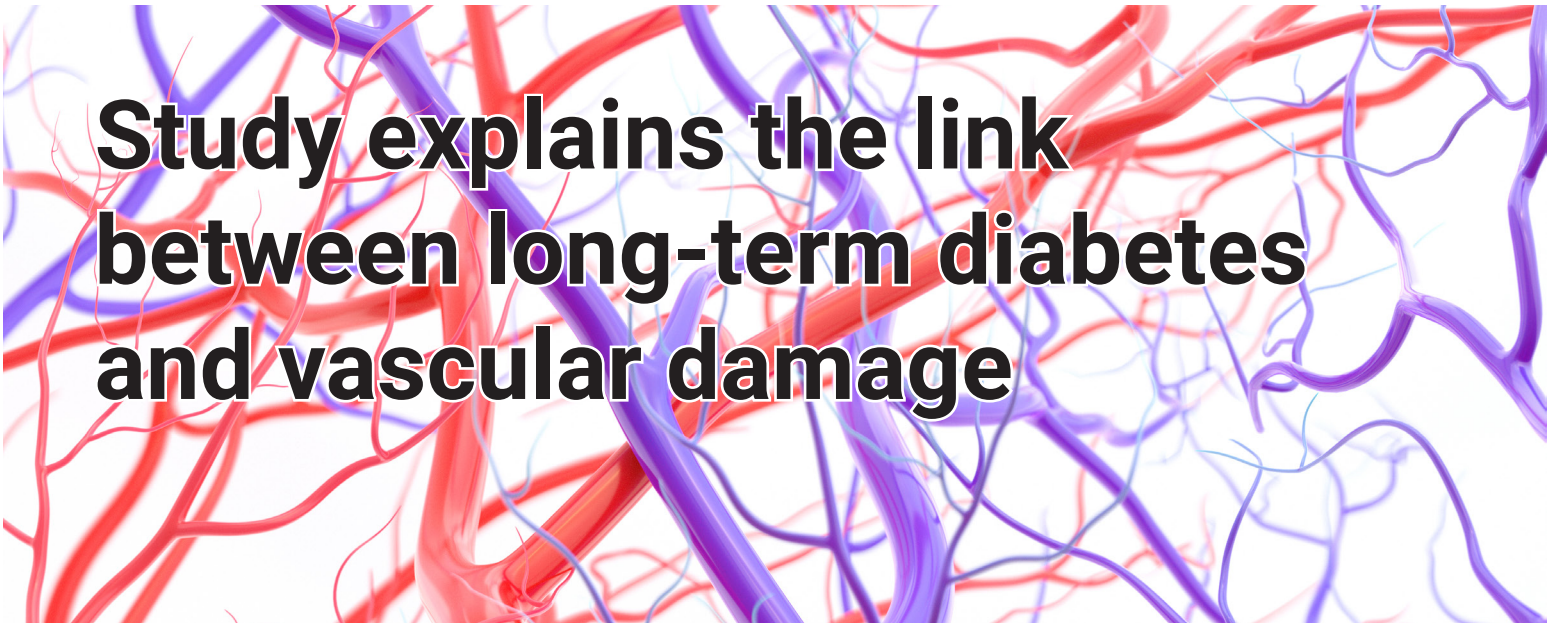


Study explains the link between long-term diabetes and vascular damage

PUBLICATION

The longer a person has type 2 diabetes, the greater the risk of cardiovascular disease. A new study from Zhichao Zhou's CMM Group, published in the journal *Diabetes*, shows that changes in red blood cells may be an important explanation, and identifies a specific molecule as a possible biomarker.

Zhichao Zhou

Eftychia Kontidou

People with type 2 diabetes are at increased risk of heart attack and stroke, and the risk increases the longer they have lived with the disease. Previous research has shown that red blood cells can affect blood vessel function in diabetes. Now, a new study shows that the duration of the disease plays a decisive role in when and how these changes occur—and that long-term type 2 diabetes can make red blood cells directly harmful to blood vessels.

The researchers studied both animals and patients with type 2 diabetes. Red blood cells from mice with long-term diabetes and patients who had had the disease for over seven years had a harmful effect on blood vessel function. No such effect was seen in newly diagnosed individuals, but after seven years of follow-up, their blood cells had developed the same harmful properties. When the researchers restored the levels of microRNA-210 in the red blood cells, vascular function improved.

"What really stands out in our study is that it is not only the presence of type 2 diabetes that matters, but how long you have had the disease. It is only after several years that red blood cells develop a harmful effect on blood vessels," says Zhichao Zhou, CMM Group Leader, associate professor at the Department of Medicine, Solna, Karolinska Institutet, and lead author of the study. The study points to microRNA-210 in red blood cells as a possible biomarker for early detection of the risk of cardiovascular complications. Researchers are now working to investigate whether this can be used in larger population studies.

"If we can identify which patients are at greatest risk before vascular damage has already occurred, we can also become better at preventing complications," says Eftychia Kontidou, doctoral student from the same group and the first author of the study.

The study is funded by, among others, the EFSD/Novo Nordisk Foundation Future Leaders Award, the Swedish Heart-Lung Foundation, and the Swedish Research Council. The researchers report no conflicts of interest.

PUBLICATION: "Long Duration of Type 2 Diabetes Drives Erythrocyte-Induced Vascular Endothelial Dysfunction: A Link to microRNA-210-3p", Kontidou E, Collado A, Humoud R, Manickam K, Tengbom J, Jiao T, Alvarsson M, Yang J, Mellbin L, Mahdi A, Pernow J, Zhou Z. Diabetes, online 9 January 2026, doi: 10.2337/db25-0463.

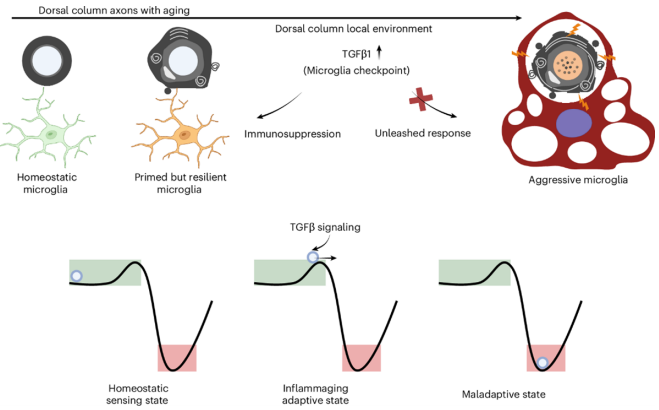
New study reveals how microglia protect the aging spinal cord

Researchers affiliated to CMM have recently uncovered a key mechanism by which immune cells of the central nervous system help safeguard spinal cord integrity during aging. The study, published in *Nature Neuroscience*, shows that as mice age, the myelin sheaths in a specific spinal cord region—the dorsal columns—undergo significant ultrastructural decline. The researchers further found that microglia upregulate a signaling molecule called TGF-β in this region, acting as a "brake" that restrains overactivation and protects myelin from further damage. When TGF-β signaling was experimentally blocked in microglia in older mice, these cells became excessively activated and began attacking myelin in the dorsal column, leading to severe neurological dysfunction.

Based on these findings, the researchers proposed a theoretical framework for maintaining myelin health in the spinal dorsal column during aging. During normal aging, the long-term accumulation of myelin-associated changes triggers low-level activation and immune responses in dorsal column microglia. At this stage, microglia adapt protectively by upregulating TGF-β signaling as an immune checkpoint, helping to restrain excessive immune activation. This represents an adaptive state of "inflammaging" near the threshold of physiological balance. However, when TGF-β signaling can no longer be sustained, spinal cord health crosses an irreversible tipping point, leading to uncontrolled microglial immune activation in the dorsal column and, ultimately, severe pathological myelin damage.

This study reveals the critical role of TGF-β signaling as a microglial immune checkpoint during aging and significantly advances our understanding of microglia–myelin interactions. These findings provide new insights into age-related nervous system decline and may inform future personalized treatment strategies for demyelinating conditions, particularly those with lesions concentrated in the spinal cord.

The study was conducted in collaboration with researchers in China, the United States, and France. Funding was provided by the Swedish Research Council, Alltid Litt Sterkere, Neurofonden and Karolinska Institutet's research initiative StratNeuro, among others. The researchers report no conflicts of interest.



First author: Dr. Keying Zhu (middle); Lead correspondence: Dr. Harald Lund (left), Prof. Robert Harris (right).

PUBLICATION: "TGFβ signaling mediates microglial resilience to spatiotemporally restricted myelin degeneration" Zhu K, Liu Y, Min JH, Joshua V, Lin J, Li Y, Kreutzmann JC, Guo Y, Xia W, Mohammadi E, Pieber M, Suerth V, Xia Y, Andrusivova Z, Hugnot JP, Kanatani S, Uhlén P, Lundeberg J, Li X, Fancy SPJ, Sarlus H, Harris RA, Lund H. *Nat Neurosci,* online 2 January 2026, doi: 10.1038/s41593-025-02161-4.

Mechanism discovered linking Epstein-Barr virus to MS

The immune system's reaction to the common Epstein-Barr virus can ultimately damage the brain and contribute to multiple sclerosis (MS). This is shown by new research mainly conducted from and at CMM and published in *Cell*. The study provides new insight into the long-suspected link between Epstein-Barr virus (EBV) and MS.

Olivia Thomas. Photo: Erik Holmgren.

André Ortlieb. Photo: Private.

Tomas Olsson. Photo: Andreas Andersson.

Multiple sclerosis is a chronic inflammatory disease in which the immune system attacks the central nervous system and causes nerve damage. It has long been known that everyone who develops MS has had an infection with the Epstein-Barr virus (EBV), but exactly how the virus contributes to the disease has been unclear.

The new study shows that when the immune system fights EBV, certain T cells – which normally attack the virus – can also react to a protein in the brain called Anoctamin-2 (ANO2). This phenomenon is called molecular mimicry – immune cells mistaking the body's own proteins for those of the virus. The researchers found that these cross-reactive T cells are significantly more common in people with MS than in healthy controls. The study builds on previous research showing that misdirected antibodies after EBV infection may play a role.

"Our results provide mechanistic evidence that immune responses to EBV can directly damage the brain in MS. It is a complex neurological disease, and it may be that the molecular mechanisms vary between patients," says the study's first author, Olivia Thomas, assistant professor at the Department of Clinical Neuroscience at Karolinska Institutet and CMM Team Leader.

PUBLICATION: "Anoctamin-2-specific T Cells Link Epstein-Barr Virus to Multiple Sclerosis" Thomas OG, Rykaczewska U, Galešić M, van der Burgt R, Hallén N, Ferro F, Bronge M, Marti Z, Li Y, Hill Riqué A, Lin J, Krstic A, Gromadzka A, Levente Szonder A, Sorini C, Reina-Campos M, Sun T, Rubio Rodríguez-Kirby LA, Dumral Ö, Berglund R, Pahlevan Kakhki M, Adzemovic M, Zeitelhofer M, Akpinar B, Tengvall K, Nilsson OB, Holmgren E, Starvaggi Cucuzza C, Asplund Högelin K, Gafvelin G, Fink K, Castelo-Branco G, Needhamsen M, Khademi M, Piehl F, Gräslund T, Alfredsson L, Lund H, Uhlén P, Kockum I, Martin R, Jagodic M, Grönlund H, Ortlieb Guerreiro-Cacais A, Olsson T. Cell, online January 13, 2026, doi: 10.1016/j.cell.2025.12.032

The study is based on analyses of blood samples from people with MS and compared with healthy controls. The researchers were able to isolate T cells that react to both the EBV protein EBNA1 and ANO2 from people with MS. In addition, experiments in a mouse model showed that these cells can exacerbate MS-like symptoms and cause damage to the brain. According to the researchers, the results may help explain why some people develop MS after an EBV infection while others do not.

"This discovery opens up new treatments that target these cross-reactive immune cells. Since several EBV vaccines are now being tested in clinical trials, the results may be of great importance for future preventive efforts," says Professor Tomas Olsson, who led the study together with Associate Professor Andre Ortlieb Guerreiro-Cacais at the same institution and who are CMM Group and Team Leaders, respectively.

The study is a collaboration between several research groups at Karolinska Institutet and CMM and has been funded by, among others, the Swedish Research Council, the EU's Horizon program, the Swedish Brain Foundation, and the Swedish Neurological Association. Several of the article's authors have links to pharmaceutical companies; see the scientific article for a complete list of conflicts of interest.

This text is based on an article published on the Karolinska Institutet news site. The study was also covered in an article in Dagens Nyheter.

Molecular patterns behind unstable atherosclerosis identified

PUBLICATION

A large-scale multi-omics study from two CMM Groups, published in *Genome Medicine*, provides new insight into the molecular mechanisms underlying unstable atherosclerosis, one of the most important causes of ischemic stroke and myocardial infarction.

The Vascular Surgery research group under the leadership of CMM Group Leaders Associate Professor Ljubica Matic and Professor Ulf Hedin at the Department of Molecular Medicine and Surgery, has published a large-scale multi-omics study that sheds new light on the molecular mechanisms underlying unstable atherosclerosis, a major cause of ischemic stroke and myocardial infarction. The study has been performed together with researchers at Novo Nordisk and is the result of a collaborative strategic partnership between Karolinska Institutet and the company since 2019. The study is also part of a large EU Horizon Tools project NextGen, an international consortium of 20 partners set to advance AI and multi-modal data integration in personalised cardiovascular medicine.

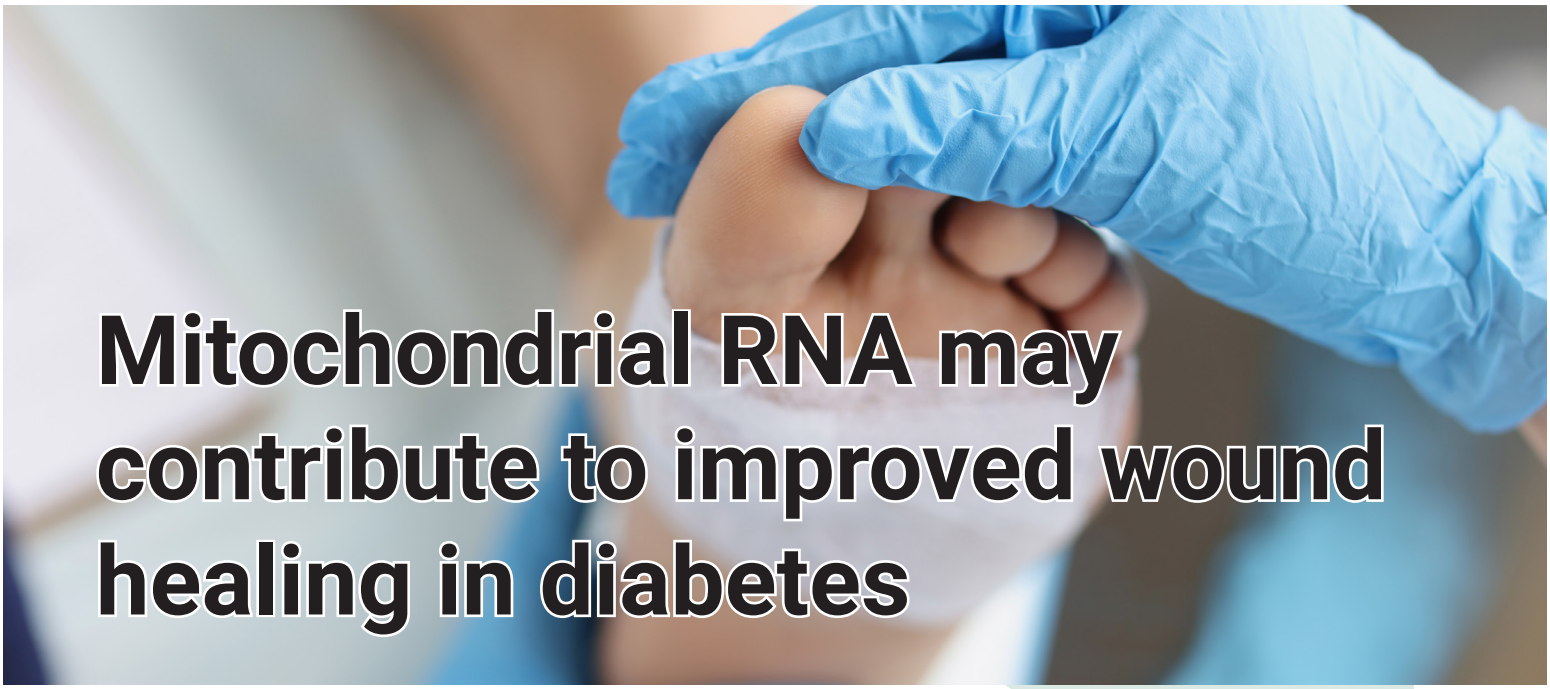
Using samples from the Biobank of Karolinska Endarterectomies (BiKE), the team analysed matched atherosclerotic plaques, circulating immune cells and blood plasma from more than 700 patients undergoing carotid endarterectomy. Patients with symptomatic (unstable) disease were compared with those with asymptomatic (stable) disease to identify molecular patterns associated with plaque instability.

By integrating transcriptomic, proteomic and metabolomic data per patient with a supervised machine-learning method, the researchers identified coordinated molecular signatures that distinguished symptomatic from asymptomatic patients across both the local disease site and peripheral circulation. The analyses confirm the importance of inflammation, coagulation, necroptosis and lipid metabolism in unstable disease, while also highlighting less explored pathways, including sphingomyelin metabolism and bilirubin-related processes. Several molecules consistently emerged as key markers, including IL6, FABP4, ANGPTL3, ICOSLG, F11, and multiple sphingomyelins. Some of these markers were associated with commonly used cardiovascular medications, while others – most notably ANGPTL3 – were not influenced by current therapies and are already under clinical trials. Importantly, identified molecular signals were also linked to long-term risk of major cardiovascular and cerebrovascular events after surgery.

The study was performed as part of the strategic research partnership between Karolinska Institutet and Novo Nordisk, Denmark. The work was also funded by a research grant from the European Union's HORIZON-HLTH-2023-TOOL-05 program with grant agreement NextGen, the Swedish Research Council, Swedish Heart-Lung Foundation and Karolinska Institutet Consolidator program.

This text is based on an article from Karolinska Institutet's new site.

PUBLICATION: "Multi-omics data integration from patients with carotid stenosis illuminates key molecular signatures of atherosclerotic instability." Das V, Narayanan S, Zhang X, Bergman O, Djordjevic D, Kronqvist M, Chemaly M, Karadimou G, Sundman S, Prasad I, Buckler AJ, Knape KC, Michaelsen NB, Hedin U, Matic L. *Genome Med* 2026 Feb 6;18(1):25. doi: 10.1186/s13073-026-01601-5.



Mitochondrial RNA may contribute to improved wound healing in diabetes

PUBLICATION

Researchers from CMM have identified a circular RNA in mitochondria that plays an important role in the healing of chronic wounds. The study is published in *Advanced Science*.

Diabetic foot ulcers are slow healing and increase the risk of infections and amputation. In the new study, researchers from CMM show that a mitochondrial circular RNA, circMTRNR2, is reduced in tissue from patients with long standing diabetic foot ulcers. The molecule appears to support the skin's reparative capacity by protecting the cells' energy metabolism against harmful stress.

The researchers analysed skin samples from patients with non healing wounds as well as from healthy donors. They also used an experimental wound model system in both human skin and mice. The results showed that when circMTRNR2 decreased, fibroblasts' ability to grow, move, and build new tissue was impaired. Fibroblasts are a type of skin cell that are central to wound healing.

"Our results suggest that this RNA molecule acts as a protective factor for the cells' mitochondria by stabilising an antioxidant that counteracts harmful stress," says Jennifer Geara, doctoral student in Ning Xu Landén's research group at CMM.

When circMTRNR2 is absent, cellular stress increases and the mitochondria are damaged, which slows down the healing of the wound.

In both models of human wound tissue and animal models, the researchers observed that the healing process was slower when the amount of circMTRNR2 was reduced and faster when it was increased. According to the researchers, the molecule could therefore become a future target for treating chronic wounds, but more research is needed.

Jennifer Geara. Photo: Private.

"The next step is to investigate how circMTRNR2 can be delivered directly to wound tissue and whether this can improve healing in patients with long-standing wounds," says Jennifer Geara.

The study was conducted in collaboration with Karolinska University Hospital. It was funded by, among others, the Swedish Research Council, Ragnar Söderberg Foundation, the Swedish Skin Foundation (Hudfonden), Ming Wai Lau Centre for Reparative Medicine, LEO Foundation, the Swedish Cancer Society, and the Åke Wiberg Foundation. Another important contribution was the funding of Guanglin Niu's post-doctoral fellowship from the Strategic Research Programme in Diabetes.

PUBLICATION: "Mitochondrial CircRNA CircMT-RNR2 Safeguards Antioxidant Defense to Support Fibroblast Functions in Wound Repair", Niu G, Geara J, Chen Y, Luo L, Xiao Y, Liu Z, Gaborieau M, Pan L, Loh E, Li D, Sommar P, Wang A, Zheng X, Landén N. *Adv Sci,* online 8 February, 2026, doi: 10.1002/advs.202517141

Director's Update

Dear CMM'ers,

We are now well into the new year and research work is in full swing. I would like to take this opportunity to look back at the latest initiatives from the CMM Foundation and update you on a few of the upcoming developments planned by CMM.

We had a bit more relaxed year throughout 2025, compared to 2024 where a large number of scientists and groups joined us from Bioclinicum.

Since my appointment as Director, the CMM Foundation has made significant contributions to our important research activities, in particular donations to Core Facilities as well as a major upgrade of both the wired and wireless network in the CMM building. I expect the Foundation to be able to further support the work carried out by excellent scientists based at CMM also during 2026.

We are now looking forward to further improvements of our work environment, as well as continued success in securing research grants, generating impactful scientific results and publications, as well as seeing additional excellent scientists joining CMM during the year.

You will likely (to some extent) be affected by the renovation project on floor 00, where the Café part outside the lecture hall will be fully refurbished. We anticipate that the work will commence during the first quarter of 2026 – and we will attempt to ensure that the construction work will disturb your work as little as possible.

Patrik Nylund, our property manager, has unfortunately decided to leave CMM. He will have his last working days in April. We have commenced the work and planning for how property management will be carried out from May and onwards.

Best wishes,
Michael

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