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Can Severe Asthma Go Into Remission? Insights From the Largest European Study to Date



PUBLICATION

A large European study of more than 13,000 severe asthma patients across 22 countries shows that many continue to experience significant symptoms and persistent inflammation despite modern biologic therapies. Led by CMM Group Leader Apostolos Bossios, and published in *Lancet Respiratory Medicine*, the research underscores the importance of early identification of patients at risk of more severe disease.

Severe asthma affects around 5–10 percent of all people with asthma and can involve recurrent exacer-

bations, impaired lung function and the need for corticosteroid treatment. In recent years, biologic therapies have improved treatment options for many patients.

In the new study, the researchers analysed data from 13,455 adults with severe asthma in 22 European countries. The patients were included in the European SHARP (Severe Heterogeneous Asthma Research collaboration) registry. The researchers examined factors including lung function, asthma control, exacerbations, use of oral corticosteroids and biological markers of inflammation.

Although 79 percent of the patients were treated with biologic therapies, many still showed signs of active disease. Among patients for whom all four disease domains

studied could be assessed, 89 percent had at least one active domain and more than two-thirds had at least two. Impaired airflow was also common: 62 percent had lung function, measured as FEV1 (forced expiratory volume in 1 second), below 80 percent of the predicted value. Coexisting health issues were also common among many patients.

“Results show that biologic therapies have become an important part of the treatment of severe asthma, but also that many patients still have a significant disease burden. It is therefore not enough to look only at inflammatory markers,” says Apostolos Bossios, CMM Group Leader, associate professor at the Institute of Environmental Medicine, Karolinska Institutet, and Senior Consultant in Pulmonary Medicine at Karolinska University Hospital. ►

"We also need to monitor symptoms from an early stage, exacerbations, lung function and comorbidities, often related to corticosteroid use, which we must aim to eliminate."

The researchers also identified a group of patients who were not yet receiving biologic therapies and whose disease burden had increased rapidly. In 35 percent of these patients, several signs of

active disease had accumulated within less than ten years – an alarming finding.

"This suggests that there may be an important window of opportunity in which we need to identify high-risk patients earlier and initiate the appropriate treatment before more permanent changes in the airways have had time to develop," says Apostolos Bossios.

The study is observational and therefore cannot establish that a particular treatment causes the patterns observed by the researchers. The researchers emphasise that the findings need to be followed up in longitudinal studies in which patients are monitored over time.

This text is based on an article published on the Karolinska Institutet news site.



Evangelia Fouka and Apostolos Bossios. Photos: Private.

PUBLICATION

"Severe asthma and remission prospects in Europe (SHARP): insights from a multicentre observational study based on the European Severe Asthma Registry", Fouka E, Bansal AT, Håkansson KEJ, Jaun F, Louis R, Hanon S, Brusselle G, Ziant S, Paulus V, Popović-Grle S, Pavlisa G, Lampalo M, Vergles M, Kasap EB, Sedlak V, Søndergaard M, Hansen S, Sergejeva S, Perotin JM, Taillé C, Devouassoux G, Giboin C, Klising E, Estellat C, Metdaoui D, Hamelmann E, Samitas K, Porpodis K, Zervas E, Gaga M, Csoma Z, Horvath I, Lúdvíksdóttir D, Sigurdardóttir KD, Canonica GW, Heffler E, Cardini C, Alloni A, Kroiča I, Matisa D, Bieksiene K, Palacionyte J, Christian N, Tching R, Boudewijn I, Sont JK, Hashimoto S, Kuna P, Morawska M, Loureiro CC, Fonseca JA, Canhoto D, Santos H, Mendes A, Belchior I, Lopes C, Mihaltan F, Hromis S, Stjepanovic M, Cupurdija V, Tomic-Spiric V, Skrgat S, Kopac P, Ramos-Barbon D, Pérez de Llano L, Garcia VL, Morenos Martos D, Dahlén B, Yasinska V, Tenselius O, Janson C, Egesten A, Richter J, Emilsson ÖI, Leuppi J, Gemicioglu B, Celik G, Damadoglu E, Goksel O, Heaney L, Busby J, Redmond C, Masoli M, Chaudhuri R, Dennison P, Doe S, Kurukulaaratchy R, Bel E, Djukanovic R, Bourdin A, Ten Brinke A, Schleich F, Porsbjerg C, **Bossios A.** *Lancet Respiratory Medicine*, 14 August 2026, doi: 10.1016/S2213-2600(26)00141-4.

New CMMers



Lucas Zhang is a postdoctoral researcher working on the BIOFUNC project with Associate Professor Cecilia Aulin in Professor Helena Harris's CMM Group. He completed his PhD in Lars Sävendahl's Group at the Department of Women's and Children's Health, Karolinska Institutet, Sweden.

The overall aim of the BIOFUNC project is to identify specific biomarker clusters that correlate with biomechanical assessments of knee function, radiographic signs of knee osteoarthritis (KOA), and patient-reported outcomes. By integrating these data, the project aims to predict the development of KOA in athletes and recreational sports participants undergoing anterior cruciate ligament reconstruction surgery, while also potentially uncovering novel therapeutic targets for osteoarthritis treatment.

In addition to his research background, Lucas holds a medical degree from China, and his long-term goal is to combine scientific research with clinical practice to improve outcomes for patients suffering from osteoarthritis and other bone-related conditions.



Xuemei Liu is a new postdoc in Ning Xu Landén's group. She pursued a PhD in Bioinformatics at the Institute of Genetics and Developmental Biology, Chinese Academy of Sciences, China. She will be studying molecular mechanisms underlying human skin wound healing by integrating single-cell transcriptomics, chromatin accessibility, and spatial transcriptomics data. Her primary research interests include computational biology and regenerative medicine.



Elias Schoenwandt, Neurologist at the Karolinska University Hospital, joined Ingrid Kockum's Group as a PhD Student. The title of his PhD project is: Prevalence and Clinical Impact of Clonal Hematopoiesis in People with Multiple Sclerosis in Sweden.

Martina Tosi is a visiting Post doc from University of Eastern Piedmont, Italy. Martina has been at CMM during the end of spring term and over the summer and will continue to work with Ingrid Kockum's Group during the autumn. She is working both in Kockum and Kiani research groups within the EU funded WISDOM project. She is both working on multiomics integration to understand paediatric multiple sclerosis better and network analysis of disease trajectories leading chronic inflammatory diseases such as Multiple Sclerosis, Rheumatoid Arthritis and Myasthenia Gravis using Italian administrative health care data.

New CMMers



Sofija Glamočlija Jekić is a new postdoctoral researcher in Prof. Susanne Gabrielsson's group at CMM. She obtained her PhD in Immunology from the Institute for the Application of Nuclear Energy, University of Belgrade, Serbia. During her doctoral studies, she investigated the immunomodulatory properties of extracellular vesicles (EVs), enriched from parasite-derived products, and explored their potential for treating respiratory allergies. She is now investigating how components of dendritic cell-derived EVs, specifically the role of leukotriene forming enzymes, modulate the efficacy of EVs as cancer immunotherapy.



Jiyoung Jeong is a new postdoc in Per-Johan Jakobsson's CMM Group. She obtained a PhD in Health Sciences from Dong-A University, Korea, where her research focused on investigating the biological effects and toxicokinetics of environmental toxicants, including microplastics and engineered nanoparticles. At CMM, she will investigate the biological activities and mechanisms of bioactive compounds in inflammatory disease models to identify potential therapeutic targets. Through this work, she hopes to broaden her research scope from environmental toxicology to pharmacology.



Abderrahman Bourahi Taleb is an ERASMUS+ student in Nicolas Ruffin's Team. Abderrahman is a fourth-year Biomedical Sciences student at Universidad Alfonso X el Sabio in Madrid, Spain. His project supervised by Silvia D'Orso, a postdoc in the team, will focus on characterizing EBV-infected B cells in people with multiple sclerosis using in situ hybridization and flow cytometry.



Anna Lagni is a new postdoctoral researcher in Leo Hanke's group at BioClinicum. She earned her PhD in Applied Life and Health Sciences at the University of Verona (Italy), investigating how CFTR dysfunction influences SARS-CoV-2 infection in airway epithelial cells. She now studies the molecular mechanisms of tick-borne encephalitis (TBEV) neuroinvasion using nanobodies. In parallel, she contributes to the development and evaluation of structure-informed vaccine antigens targeting emerging and re-emerging viruses. Her broader interests include viral pathogenesis mechanisms and the development of strategies to prevent viral diseases.

New CMMers

Olof Carlsson is a new PhD student in Ingrid Kockum's Group. He is also a Neurologist at Danderyd Hospital. The title of his PhD project is: Neuromyelitis optica spectrum disease, Myelin oligodendrocyte associated disorder and Multiple Sclerosis – studies on the incidence, and risk factors for disease progression in patients treated with rituximab.



Juahui Li is a new PhD student in Ingrid Kockum's Group. The title of his PhD project is: Deciphering Multiple Sclerosis: A Data-Intensive Approach to Unraveling Clinical and Molecular Complexities through Graph and Language Modeling

Mingcong Zeng is a new lab research assistant in Leo Hanke's CMM Team located at BioClinicum. Mingcong graduated in biological sciences at Lanzhou University, China. He will be working on nanobodies and protein structures and his primary areas of interest are molecular and structural virology.



Anna Frisk graduated from medical school at Karolinska Institutet in January and subsequently joined Professor Marie Wahren-Herlenius' research group as a PhD student. Her research focuses on Kawasaki disease, a pediatric vasculitis of unknown etiology. To advance the understanding of the disease, her PhD project combines epidemiological studies of risk factors and long-term outcomes with experimental studies investigating potential pathogenic mechanisms in a mouse model.



William Purba recently joined Marie Wahren-Herlenius's research group as a PhD student. He holds a master's degree in Molecular Mechanisms of Disease from Radboud University in the Netherlands. His PhD project focuses on understanding how the MRCK-KIMURA network regulates cell motility and immune responses, with the aim of uncovering their role in autoimmune diseases. Outside the lab, he enjoys playing violin and board games.

Welcome all new CMMers!

Using AI and Digital Twins to Redefine MS Management

FUNDING AND GRANTS

CMM Group Leader Ingrid Kockum will head up the Karolinska Institutet-based part of the 5-year NeuroAim project that was recently funded by Horizon Europe JU IHI with € 20 323 463, of which € 172 743 is the share for Karolinska Institutet.

Through the project, the researchers aim at developing a new way to manage multiple sclerosis (MS) by moving from occasional check-ups to continuous, personalized disease monitoring. Using AI-powered digital twins, the project combines clinical records, biological measurements, and digital health data to create a dynamic picture of how each patient's disease evolves over time and responds to treatment.

Today, MS progression can occur silently, even in patients who appear clinically stable, making treatment decisions challenging. NeuroAim aims to address this by transforming diverse health data into actionable insights that help clinicians identify early signs of disease activity, optimize treatments sooner, and reduce reliance on trial-and-error approaches.

By combining real-world patient data, innovative sample collection methods, and close collaboration with patients and healthcare professionals, NeuroAim is building trusted tools that support more proactive, adaptive, and personalized MS care. Ultimately, the project seeks to improve clinical decision-making and long-term outcomes for people living with MS.

The overall NeuroAim project will be coordinated by The European Institute for Biomedical Imaging Research (EIBIR) in Austria.



Ingrid Kockum. Photo: Creo Media Group.

Uncovering Shared and Divergent Pathogenic Pathways in Several Autoimmune Diseases

FUNDING AND GRANTS

Many autoimmune diseases share genetic risk factors and common biological pathways, suggesting that important discoveries in one disease may benefit others. Researchers at CMM have recently received funding to study multiple sclerosis, rheumatoid arthritis, systemic lupus erythematosus, and type 1 diabetes together, seeking to identify common mechanisms of disease, discover new therapeutic targets, and develop tools for earlier detection and prevention.

Autoimmune diseases develop when the immune system mistakenly attacks the body's own tissues. Although diseases such as multiple sclerosis (MS), rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), and type 1 diabetes (T1D) affect different organs, they may share common biological causes. By understanding these shared mechanisms, researchers can identify new ways to prevent, diagnose, and treat autoimmune diseases.

CMMers Ingrid Kockum, Klementy Shchetynsky, Lina Marcela Diaz Gallo Norlén, Leonid Padyukov and Yuan Jiang have received \$ 149,500 during 1 year. It is a joint funding from Breakthrough T1D, LupaResearch Alliance & National Multiple Sclerosis Society and will be used for the project titled "Uncovering Shared and Divergent Pathogenic Pathways in SLE, MS, RA, and T1D".

The project has three goals. First, the researchers will combine genetic and biological data to identify proteins that contribute to disease

development. Some of these proteins may already be targeted by existing drugs, creating opportunities to repurpose treatments for autoimmune diseases.

Second, they will use Sweden's nationwide health registries and advanced machine learning methods to develop tools that can identify people at increased risk of autoimmune disease before symptoms appear. Early detection could enable closer monitoring and preventive interventions.

Third, the researchers will investigate which genetic risk factors are shared between autoimmune diseases and which are unique to specific conditions. This will improve the understanding of why diseases differ between patients and why treatments work better for some individuals than for others.

By uncovering common and disease-specific pathways, this research aims to accelerate the discovery of new treatments, support earlier diagnosis, and enable more personalized care. Ultimately, the goal is to reduce the burden of autoimmune diseases and improve quality of life for affected individuals.



Klementy Shchetynsky, Lina Marcela Diaz Gallo Norlén, Ingrid Kockum and Leonid Padyukov.
Photo: Majid Pahlevan Kakhki.

Novo Nordisk Grant for Innovative Drug Testing



Stephen Malin and Carolina Hagberg. Photo: Martin Stenmark, Stefan Bladh.

FUNDING AND GRANTS

In cardiometabolic diseases such as type 2 diabetes and atherosclerosis, both the cardiovascular system and metabolism are affected. CMM researchers Stephen Malin and Carolina Hagberg have received a record-breaking grant from the Novo Nordisk Foundation to develop, through an international collaboration, a model that allows drugs to be tested across multiple organs simultaneously.

The grant from the Novo Nordisk Foundation amounts to DKK 75 million, corresponding to just over SEK 110 million, over a six-year period. The objective is to develop improved models for testing both existing and novel drugs targeting cardiometabolic diseases such as type 2 diabetes, stroke, and atherosclerosis. The funded project will utilize systems of small, simplified organ models known as organoids.

The principal investigator is CMM Team Leader Stephen Malin, Senior Researcher and Research Group Leader at the Department of Medicine, Solna, Karolinska Institutet. In the international consortium, he and CMM Group Leader Carolina Hagberg, Senior Researcher

and Research Group Leader at the same department, collaborate with researchers from Denmark and the United States (see fact box). About 46 M SEK of the funding will be given to Stephen's and Carolinas particular research.

Aiming to Create a "Global Village" in the Laboratory

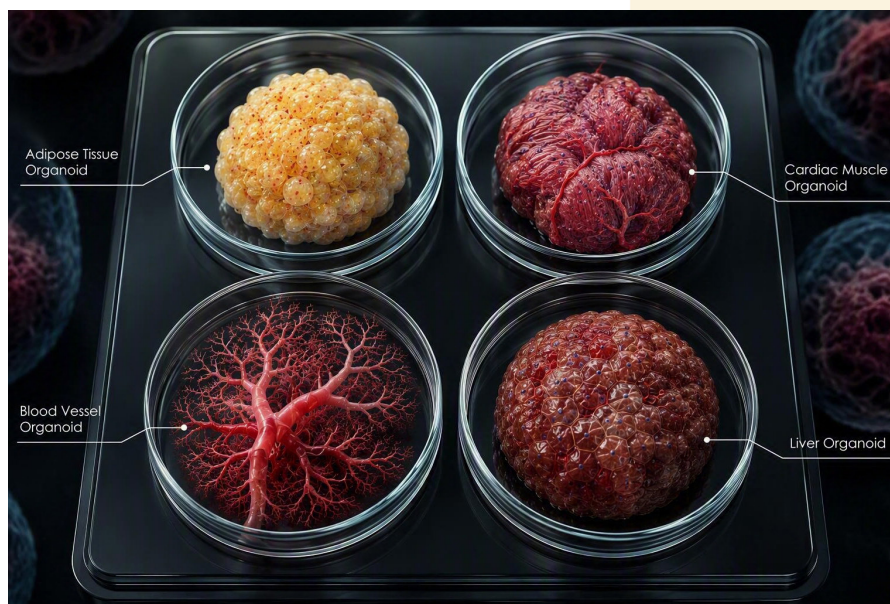
Because cardiometabolic diseases involve both the cardiovascular system and metabolism, the models need to be more complex than those currently available.

"We came up with the idea of using vascularized organoids and creating what we call a 'global village,'" says Stephen Malin.

In simple terms, this means using stem cells from many different individuals with diverse genetic and ethnic backgrounds. From these stem cells, researchers generate various types of organoids in culture dishes and then enable them to communicate with one another.

The researchers primarily aim to replicate four organs: adipose tissue (fat tissue), the liver, the heart, and blood vessels.

"We also want to include immune cells, since the immune system plays a major role in disease develop- ►



AI generated image representing organoids.

ment," he says.

A Project Built on Key Expertise

Disease-inducing factors, including excess lipids, will be introduced into the organoid system to model disease processes.

Karolinska Institutet's leadership of the international project is partly due to the researchers' complementary areas of expertise.

"My research focuses on adipose tissue and insulin resistance, particularly why obesity leads to disease, as well as communication between adipose tissue and blood vessels. Stephen's expertise lies in how atherosclerosis and vascular disease are initiated, as well as immunology," says Carolina Hagberg.

She emphasizes that the project's strength lies in the collaboration among the participating institutions. "None of the groups could accomplish exactly this on their own, but together we form a strong unit," she says.

The Challenge of Getting Organs to Communicate

Initially, the organoids must be differentiated separately. Once they have matured, researchers aim to enable communication between them, for example through fluidic systems where each organoid is housed in a small chamber connected by tubes that transport fluid in and out. Other systems, in which culture media circulate among the organoids, allow for larger-scale experimen-

tation.

"You might think it would be straightforward because our organs communicate with each other seamlessly, but organoids lack many of the barriers that exist in the human body," says Stephen Malin.

Carolina Hagberg agrees:

"It really gives you a profound respect for how complex the body is. Organs are able not only to communicate with one another but also to protect each other from, for example, the excess fat exposure that results from overeating."

Reflecting Genetic Diversity Is Essential

According to Carolina Hagberg, similar studies have been conducted before, but the time is now right to build more sophisticated systems.

"We have the combination of collaborators and technology needed to use the right stem cells and genetics to build organoids. This is a golden opportunity to create better methods for testing drugs," she says.

The "right" stem cells are those that reflect the diversity of human populations.

"Our goal is to model human diversity, where people of European ancestry represent only a small part of the overall population, so that drugs can be tested preclinically on a genetically broader basis," says Stephen

Malin.

Testing Both Existing and New Drugs

The project will evaluate both novel and established drugs, as well as drug candidates that never reached the market because human trials revealed that they were ineffective.

"I hope to demonstrate that our more advanced system can identify such candidates at an earlier stage. That would save enormous amounts of money as well as time, enabling effective drug candidates to be developed more quickly," says Carolina Hagberg.

Stephen Malin has previously developed drugs within the Drug Discovery and Development (DDD) platform at SciLifeLab.

"Being able to improve the testing of new drugs that ultimately benefit patients with cardiometabolic diseases would be extremely rewarding. If we achieve that, I would feel that I have done something truly useful," says

Stephen Malin.

"The Project Should Serve as a Hub"

The grant is among the largest ever awarded by the Novo Nordisk Foundation. Stephen Malin notes that because Karolinska Institutet is a leading institution in both clinical and basic research, they hope the project will continue to expand.

"Our hope is that the project will serve as a hub around which something larger and more long-term can develop. We want to leverage the expertise and infrastructure that already exist, particularly within precision medicine," he says.

Carolina Hagberg concludes:

"We hope and believe that this project can also create positive ripple effects locally at Karolinska Institutet."

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

Pioneer Innovation Grant

Stephen Malin also received approximately SEK 1.7 million from Novo Nordisk Foundation for a Pioneer Innovation grant to develop new anti-inflammatory therapies together with the Drug Discovery and Development program run by SciLifeLab.

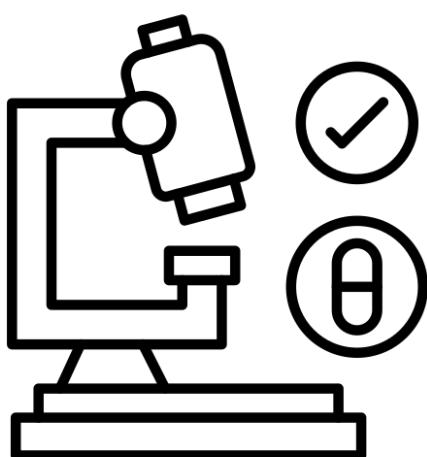
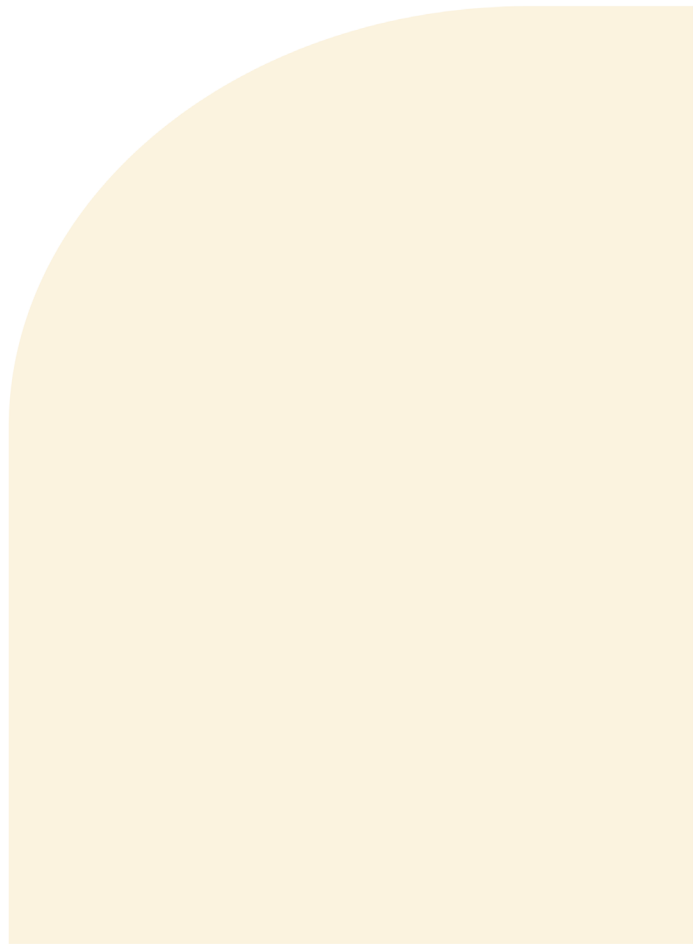


Image: Flaticon



ULTIMA receives Lupus Research Alliance Global Team Science Award

AWARDS

The international research programme ULTIMA (Untangling Lupus Through Integrative Multi-Omics Approaches), coordinated from Karolinska Institutet by CMM Team Leader Ioannis Parodis, has received a Lupus Research Alliance Global Team Science Award (GTSA) of USD 3 million over three years.

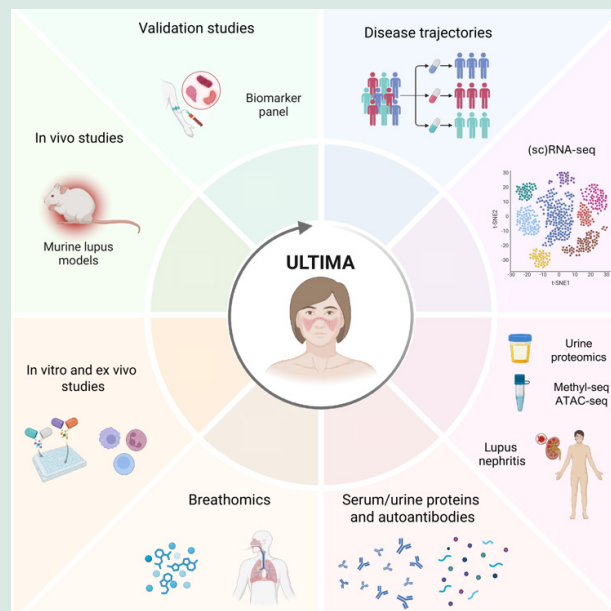
Systemic lupus erythematosus (SLE) is characterised by profound clinical and molecular heterogeneity: patients with apparently similar manifestations can follow very different disease trajectories and respond differently to the same therapy. ULTIMA aims to identify the molecular programmes that underlie this heterogeneity and translate them into biomarkers for biologically informed patient stratification.

Building on the longitudinal SLE cohorts and multi-omics resources established within the 3TR consortium, ULTIMA will focus on two clinically critical transitions: the course from active disease towards treatment response or refractoriness after therapy initiation, and the tran-

sition from quiescent disease to flare. The programme will integrate autoantibody profiling, urine proteomics, epigenomic and chromatin-accessibility analyses, single-cell approaches, microRNA profiling and breathomics with existing molecular datasets and detailed longitudinal clinical information. Candidate mechanisms and biomarkers will subsequently undergo functional and validation studies.

The consortium brings together five academic research groups from Karolinska Institutet, GENYO and the University of Granada, Amsterdam UMC/University of Amsterdam, and Fondazione IRCCS Ca' Granda Ospedale Maggiore Policlinico in Milan. ULTIMA is coordinated by Ioannis Parodis at Karolinska Institutet and CMM, and co-principal investigators are Marta Alarcón-Riquelme, Ronald van Vollenhoven, Lorenzo Beretta and Guillermo Barturen. The long-term goal is to develop clinically actionable biomarker panels and predictive models that improve treatment selection, disease monitoring and flare prediction in SLE.

Text: Ioannis Parodis.



Conceptual overview of ULTIMA: longitudinal disease trajectories are linked to multi-omics profiling, functional studies and biomarker validation with the goal of developing clinically useful predictive tools.

Karolinska receives 2026 EULAR Best Undergraduate Teaching Concept Award for SARI

AWARDS



From left: Ioannis Parodis, Dionysis Nikolopoulos and Alexander Borg representing Karolinska Institutet, with EULAR President Xenofon Baraliakos at the award ceremony.

At the 2026 European Alliance of Associations for Rheumatology (EULAR) congress, Karolinska Institutet received the EULAR Best Undergraduate Teaching Concept Award for SARI, an innovative platform developed for undergraduate medical education in rheumatology.

SARI (Social AI-enhanced Robotic Interface) combines social robotics with large language models to create interactive virtual patient encounters. Medical students can conduct natural, spoken consultations with a socially expressive robotic patient that responds verbally and through facial expressions. After the encounter, the platform provides automated AI-generated feedback on the student's performance, allowing technology-enhanced simulation to be integrated with independent clinical reasoning and reflective learning.

SARI has been implemented in the regular medical curriculum at Karolinska Institutet and has so far been evaluated in studies involving nearly 200 medical students. Published evaluations have shown favourable effects on clinical reasoning, communication skills and empathic approach compared with more conventional computer-based virtual patient platforms.

The work to develop, evaluate and implement SARI is led by **Ioannis Parodis** and **Alexander Borg** at Karolinska Institutet and CMM. The EULAR award recognises the initiative as an innovative teaching concept with the potential to strengthen clinically relevant learning through the combined use of simulation, social robotics and generative AI.

Matthew Hunt Awarded International Fellowship for Melanoma Research

AWARDS

CMM researcher Matthew Hunt is one of the recipients of the IBSA Foundation Fellowships 2025. He is awarded the fellowship for a research project on new treatment strategies for aggressive skin cancer.

The international research fellowship IBSA Foundation Fellowships is aimed at young and talented researchers and seeks to provide them with the opportunity to conduct outstanding research.

Matthew Hunt, a postdoctoral researcher at the Division of Dermatology and Venereology, Department of Medicine, Solna, and part of Jakob Wikström's CMM Group, was one of six researchers selected following an international review process.

This year's call received 398 applications from 64 countries, the highest number since the fellowship was established 13 years ago.

Seeking new ways to treat aggressive skin cancer Matthew Hunt is awarded the fellowship in dermatology for a project on melanoma, the most aggressive form of skin cancer. His research focuses on mitochondrial dynamics – the processes that regulate the shape and function of the cell's energy-producing organelles – and how these influence tumour development and treatment resistance.

The project specifically investigates melanomas with mutations in the NRAS gene, a patient group for which effective targeted treatments are currently lacking. By combining analyses of patient material with experimental models and gene editing, the research aims to identify new therapeutic targets.

"This fellowship is a great opportunity to deepen our understanding of how melanoma cells adapt and survive treatment. By focusing on mitochondrial processes, we hope to identify new vulnerabilities that could be targeted in future therapies, particularly for patients who currently lack effective treatment options," says Matthew Hunt.



Matthew Hunt with the award. Photo: Private

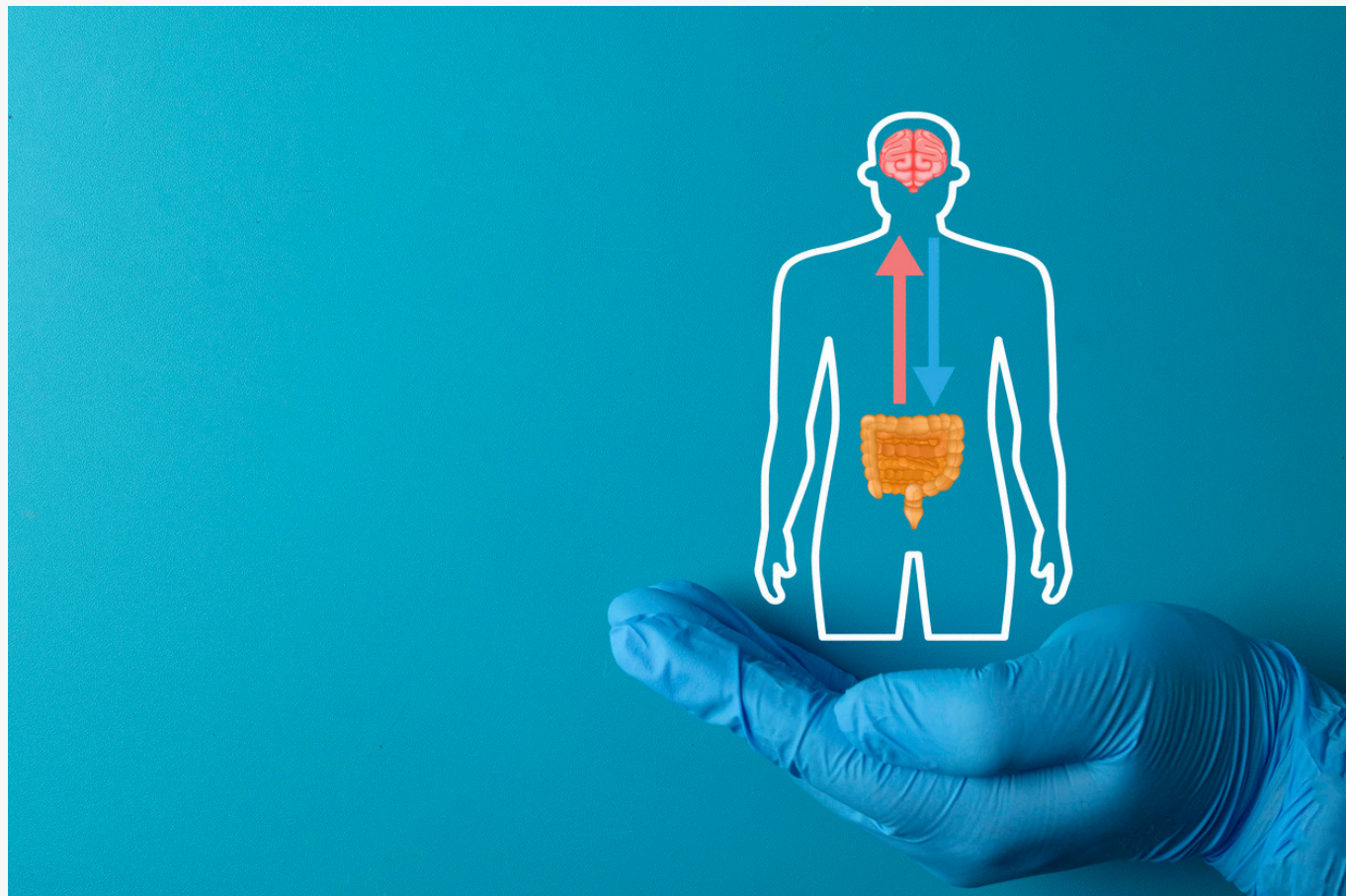
Fellowships promoting global and equitable research

The IBSA Foundation Fellowships are awarded annually to researchers at the beginning of their careers.

The programme includes fellowships of EUR 32,000 each across six therapeutic areas: dermatology, endocrinology, fertility and urology, healthy ageing and regenerative medicine, pain medicine, and rheumatology and orthopaedics.

The awards were presented at a ceremony in Barcelona in May 2026, in the presence of representatives from academia and research organisations.

EMBO Postdoctoral Fellowship



Gut-brain communication. Image: iStock.

GRANTS

Gina Papadopoulou, postdoctoral researcher in Eduardo Villablanca's CMM Group, has been awarded an EMBO Postdoctoral Fellowship (€76,200 per year for two years; €152,400 in total) to investigate how metabolic signals from the gut influence autoimmune responses in the central nervous system. During this project she will explore potential links between intestinal lipid metabolism, T-cell function, and gut-brain communication, with the aim to identify new mechanisms underlying neuroinflammation.



Gina Papadopoulou Photo: Private.

Funding from the Swedish Research Council

GRANTS

CMM Team Leader Elisabeth Nyström is one of three assistant professors at Karolinska Institutet who have been awarded funding as part of the Swedish Research Council's new initiative on strategic recruitment. The grant aims to strengthen the development of future research leaders and create long-term conditions for research of the highest quality.

The initiative on the strategic recruitment of assistant professors is a government mandate to the Swedish Research Council, with the aim of strengthening Swed-

ish research in the long term. The focus is on recruiting internationally competitive researchers early in their careers and giving them the opportunity to establish independent research.

In the first call for applications, 109 applications were received, of which 22 researchers at 11 higher education institutions were awarded funding following fierce competition.



Elisabeth Nyström. Photo: Lisa Thanner

Project: Dynamic regulation of intestinal mucus in gut health and disease.

The project aims to understand how the intestinal mucus barrier is built and regulated, and what goes wrong in inflammatory bowel disease. A key aspect is to connect biological scales that are rarely studied together– from the molecular structure and modification of the giant proteins that form mucus, through specialized mucus-producing cells and tissue-level barrier function, to disease models and patient samples. By connecting these levels, we aim to uncover how molecular and cellular changes translate into altered barrier function and ultimately human disease.

The grant provides funding for five years at assistant senior lecturer level, with the possibility of further support at senior lecturer level following evaluation.

Other Grants

GRANTS

Caroline Wasén in Jon Lampa's CMM Team received a StratNeuro Starting Grant of SEK 1 million for the project "Preventing brain atrophy in rheumatoid arthritis".



Caroline Wasén. Photo: N/A



Julia Lang. Photo: N/A

Dr. **Julia Lang**, post-doc in Keira Melican's Group has received several grants for her research focused on understanding the role of human skin immune cells in the recognition and response to Staphylococcus bacteria.

Stiftelsen Lars Hiertas Minne - SEK 40 000

O.E. och Edla Johanssons Vetenskapliga Stiftelse
- SEK 150 000

Petrus och Augusta Hedlunds Stiftelse - SEK 400 000

Hudfonden - SEK 750 000

Publications

CMMers IN BOLD

Andreassen OA, Heimer H, Aarsland D, Dale AM, Gudbjartsson D, Jacobsson B, **Kockum I**, Lappalainen T, Metspalu A, Milani L, Njølstad PR, Palotie A, Ripatti S, Sandvik A, Smeland OB, Stefánsson H, Stefánsson K, Vaudel M, Vilo J, Werge T, Brunak S, Kallioniemi O An AI-Health infrastructure for the Nordic region: technical foundations, data assets, and a roadmap for deployment. *Nat Med*. 2026 Aug 14. doi: 10.1038/s41591-026-04575-4. Online ahead of print. PMID: 42601486 (Comment)

Belevich I, Porcile L, Sola-Carvajal A, Grommisch D, Annusvar K, Heinz P, Das S, Webb AT, Andersson S, Pentimikko N, **Villablanca EJ**, Goldenring JR, Kasper M, Jokitalo E, Coffey RJ, Katajisto P, Scharaw S. Golgi organization regulates stem cell function in the small intestine. *Nat Comm*. 2026, Jul 29, PMID: 42532997

Boccia S, Pastorino R; PROPHET* Consortium†. (Farina S, Masiello M, Osti T, Pezzullo AM, Valz Gris A, Cornel M, Kreeftenberg LL, van El C, Pausan M, Goisau M, Barahona-López C, Perez-Gomez B, Plans Beriso E, Fava V, Quaggia D, Chegkazi M, Merchant A, Scollen S, Metspalu A, Reigo A, Teder-Laving M, Louati C, Zeisl Y, Hein J, Kannan P, Kääriäinen H, Perola M, Alcouffe C, Fadil E, Cervera de la Cruz P, Shabani M, Luis Cardoso M, Costa A, Costa C, Moura Vicente A, Alvarez Ahlgren J, Flodkvist E, **Gyllenberg A**, **Kockum I**, Swartling Peterson S, Sundberg CJ, Borry P, Van Steijvoort E, Blackburn L, Babb de Villiers C.) A Strategic Research and Innovation Agenda for personalized prevention: towards a European implementation Roadmap *Eur J Public Health*. 2026 Jun 10;36(4):ckag111. doi: 10.1093/eurpub/ckag111. PMID: 42406790 (Comment)

Buyukkurt A, Ygonia M, **Manouchehrinia A**, Lefrançois P, **Kockum I**, Harroud A. Shared genetic effects and antagonistic pleiotropy between multiple sclerosis and common cancers. *Ann Clin Transl Neurol*. 2026 Jan 16. doi: 10.1002/acn3.70298. Online ahead of print. PMID: 41540890 JIF 3.9 citations 0

Corona A, Clarelli F, Pääkkönen K, Harroud A, **Shchetynsky K**, Zollo A, Mingione A, Sorosina M, Mascia E, Giordano A, Rocca MA, **Piehl F**, **Stridh P**, Hemmer B, Goris A, Dubois B, Søndergaard HB, Sellebjerg F, D'alfonso S, Harbo HF, **Olsson T**, Saarela J, Priori A, Filippi M, **Kockum I**, Esposito F, Martinelli Boneschi F Pharmacogenomics of response to interferon-beta and glatiramer acetate in Multiple Sclerosis: A multi-centric study. *Mult Scler*. 2026 Mar 3;13524585261417130. doi: 10.1177/13524585261417130. Online ahead of print. PMID: 41776383 JIF 5 citations 0

Díaz-Basabe A, **He N**, **Villablanca EJ**. Promoting intestinal regeneration without tumor risk in immune-mediated inflammatory diseases. *Trends Immunol*. 2026, Jun 9, PMID: 42265014

Ebrahimi A, Wiil UK, Olsson T, Liò P, Kockum I, Manouchehrinia A, Kiani NA. A susceptibility network analysis of disease trajectories leading to multiple sclerosis: A nationwide cohort study. *Mult Scler*. 2026 Aug;32(9):915-926. doi: 10.1177/13524585261421480. Epub 2026 Mar 10. PMID: 41807266; PMCID: PMC13385570.

Ebrahimi A, Wiil UK, **Olsson T**, Liò P, **Kockum I**, **Manouchehrinia A**, **Kiani NA**. A susceptibility network analysis of disease trajectories leading to multiple sclerosis: a nationwide cohort study. *Mult Scler*. 2026

Mar 10;13524585261421480. doi: 10.1177/13524585261421480. Online ahead of print. PMID: 41807266 JIF 5 citations 0

Forsberg D, Steinhoff L, Drevin G, Idborg H, Pejovic NJ, Herlenius E. Sudden Unexpected Postnatal Collapse: A Retrospective Cohort Study Describing Incidence, Risk Factors and Role of Perinatal Transition. *Acta Paediatr*. 2026 Jul 13. doi: 10.1111/apa.70670. Epub ahead of print. PMID: 42444072.

Fransson J, **Sorini C**, **Castillo F**, **Chi Y**, **He N**, **Suarez-Alvarez M**, **Ulloa MA**, **Morales RA**, **Okhovat A**, Sournat H, **Zagami C**, **Cardoso RF**, **Das S**, Giacomello S, Mechling A, **Hedin C**, Smith P, **Villablanca EJ**. Distinct spatiotemporal inflammatory programs underlying experimental chronic colitis *Immunity*. 2026, Jun 9, PMID: 42097141

Giordano A, **Stridh P**, Preziosa P, Pisa M, Lerma-Martin C, Sorosina M, Mascia E, Santoro S, Misra K, Clarelli F, Ferrè L, **Needhamsen M**, **Manouchehrinia A**, Missaglia M, **Moridi T**, **Shchetynsky K**, Ouellette R, Harroud A, de Vries E, Kuttikkatt SB, **Piehl F**, Alfredsson L, Hillert J, **Olsson T**, Fugger L, Attfield K, Granberg T, Schirmer L, Absinta M, **Jagodic M**, DeLuca GC, Rocca MA, Filippi M, **Kockum I**, Esposito F. A HIF1A genetic variant is associated with disability accrual and smoldering inflammation in Multiple Sclerosis. *Acta Neuropathol*. 2026 Feb 4;151(1):12. doi: 10.1007/s00401-026-02984-w. PMID: 41639279 JIF 9.3 citations 1

Hedström AK, **Thomas O**, **Strid P**, Hillert J, **Kockum I**, Waterboer T, Olsson T, Alfredsson L. HLA-dependent association between EBNA1 antibody levels and disability progression in multiple sclerosis *J Neu-*

Publications

CMMers IN BOLD

rol Neurosurg Psychiatry. 2026 Jul 31:jnnp-2026-338964. doi: 10.1136/jnnp-2026-338964. Online ahead of print. PMID: 42538096

Ingvarsson J, Grut V, Biström M, Berg LP, Butt J, Michels B, **Stridh P, Olsson T**, Alfredsson L, **Kockum I**, Waterboer T, Nilsson S, Sundström P Seropositivity against rubella virus envelope protein 1, but not 2, is associated with an increased risk of multiple sclerosis. *Clin Transl Immunology*. 2026 Apr 16;15(4):e70094. doi: 10.1002/cti2.70094. eCollection 2026. PMID: 42007315

Jiang Y, Xiao J, **Kockum I, Stridh P, Liu Q, Olsson T**, Alfredsson L, Jiang X. Multi-omics integration provides biological insight and prioritizes potential drug targets in multiple sclerosis progression. *J Neuroinflammation*. 2026 Jun 2. doi: 10.1186/s12974-026-03895-z. Online ahead of print. PMID: 42231304

Kisten Y, Cîrciumaru A, Dehara M, Grönwall C, Mathsson-Alm L, Hansson M, Lundberg K, Gentline C, Pomiano NV, Hensvold A, Chatzidionysiou K, Geschwind S, Jakobsson PJ, Klint EA, Askling J, Westerlind H, Rezaei H, Klareskog L. Ultrasound-defined presynovitis is associated with progression to clinical arthritis in ACPA-positive individuals at risk of rheumatoid arthritis: longitudinal insights from the Karolinska Risk-RA cohort. *Ann Rheum Dis*. 2026 Jul 18:S0003-4967(26)00370-5. doi: 10.1016/j.ar.d.2026.06.022. Epub ahead of print. PMID: 42471275.

Kowalec K, Manouchehrinia A, Kingwell E, Carruthers R, Marrie RA, Evans C, Rotstein D, Kuan AJ, Luo W, Luo D, Desrochers B, Jokubaitis VG, Lechner-Scott J, Maltby VE, Groen K, Wright GE, Drögemöller BI, Bernatsky S, Levin MC, Knox K, Maruf AA, Zhong M, Alfredsson L, Hillert

J, Kockum I, Olsson T, Stridh P, Shchetynsky K, Dever K, MacIsaac J, Kobor MS, Tremlett H. Lymphocyte count polygenicity underlies dimethyl fumarate-associated lymphopenia in multiple sclerosis. In press *Multiple Sclerosis Journal* PMID: 42608789

Luessi F, **Starvaggi Cucuzza C**, Engel S, Civelek A, Steffen F, Koeck T, Wild PS, **Kockum I, Olsson T**, Bittner S, **Piehl F**, Zipp F CSF Proteins Associated With Neuroaxonal Damage in Early Active Multiple Sclerosis. *Neurol Neuroimmunol. Neuroinflamm*. 2026 May;13(3):e200564. doi: 10.1212/NXI.0000000000200564. Epub 2026 Mar 25 PMID: 41881460

Paslawski W, Doyon D, Ekman CJ, Yngwe H, Mamula D, Zareba-Paslawska J, Beckman M, Xu ZD, Hökfelt T, Tiger M, Lundberg J, Svenningsson P. CSF galanin and noradrenaline downregulation by psilocybin therapy in major depressive disorder. *Neuropsychopharmacology*. 2026 Sep;51(10):1898-1901. doi: 10.1038/s41386-026-02490-3. Epub 2026 Jul 7. PMID: 42414566; PMCID: PMC13487249.

Piehl F, Castelo-Branco G, Jagodic M, Olsson T. Targeting progressive multiple sclerosis: Toward mechanism-informed precision medicine. *J Intern Med*. 2026 Sep;300(3):238-257. doi: 10.1111/joim.70125. Epub 2026 Jul 21. PMID: 42482223; PMCID: PMC13429007.

Suárez-Álvarez M, Ulloa MA, Villablanca EJ. Decoding the complexity of intestinal immunity with spatial transcriptomics. *Curr Opin Immunol*. 2026, Aug, PMID: 42335605

Wen X, Qu H, Benedyk-Machaczka M, Chen D, Sundberg E, Melén E, Altman M, Aulin C, Harris HE. Hippocampal neuroinflammation and altered peripheral neurobiological protein profile in experimental arthritis and systemic juvenile idiopathic arthritis. *EBioMedicine*. 2026 Jul;129:106330. doi: 10.1016/j.ebiom.2026.106330. Epub 2026 Jun 16. PMID: 42302429; PMCID: PMC13292667.

Ye K, Francis N, Dunst J, Borgenstam A, Rocamonde-Lago I, Köhler S, You Y, Dubnovitsky A, Kramer A, Meng F, Zollner V, Vogg L, Ryan TJ, Hanada KI, Regen T, Waisman A, Malmström V, Benson E, Kisielow J, Krey T, Winkler TH, Hanke L, Kreslavsky T. Direct interaction of Vδ7 TCRs with IL17RA drives the differentiation of TH1-like γδT cells. *Science*. 2026 Jul 16;393(6808):eadx9264. doi: 10.1126/science.adx9264. Epub 2026 Jul 16. PMID: 42462026.

Zerebinski J, Broumou I, Kimenyi KM, Aklillu E, Andrade CM, Babiker H, Bejon P, Cherif MK, Crompton PD, Fançon C, Foroogh F, Gil JP, Homann MV, de Jesus Trovoadá M, Kapulu M, Kiwuwa SM, Kweku MA, Liljander A, Lopes D, Marsh K, Mutemi DD, Ngasala B, Normark J, Orikiiriza J, Persson KEM, Portugal S, Ribacke U, Sirima SB, Sonden K, de Sousa TN, Traore B, Tijani MK, Eckerdal N, Villner P, Ochola-Oyier LI, Plaza DF, Färnert A. Widespread variants of the highly polymorphic *Plasmodium falciparum* vaccine candidate antigen MSP2 across Sub-Saharan Africa. *J Infect Dis*. 2026 Jul 22:jia377. doi: 10.1093/infdis/jia377. Epub ahead of print. PMID: 42484022.

Mini Abstracts

Handgrip weakness is associated with motor cortex atrophy in rheumatoid arthritis: a cross-sectional study with a hand exercise intervention

We investigated handgrip strength as a measure of physical function in RA and its relationship with motor-related brain region volume. Atrophy in premotor and supplementary motor area was associated with weaker handgrip, independently of age and joint pathology.

PUBLICATION: Gustafsson, A. L., R. A. Heckemann, M. C. Erlandsson, V. M. Lundgren, Z. Dai, S. T. Silfverswärd, R. Pullerits, M. I. Bokarewa and **C. Wasén** (2026). Handgrip weakness is associated with motor cortex atrophy in rheumatoid arthritis: a cross-sectional study with a hand exercise intervention. BMC Med 24(1)



Ferroptosis promotes aortic stenosis through 5-lipoxygenase

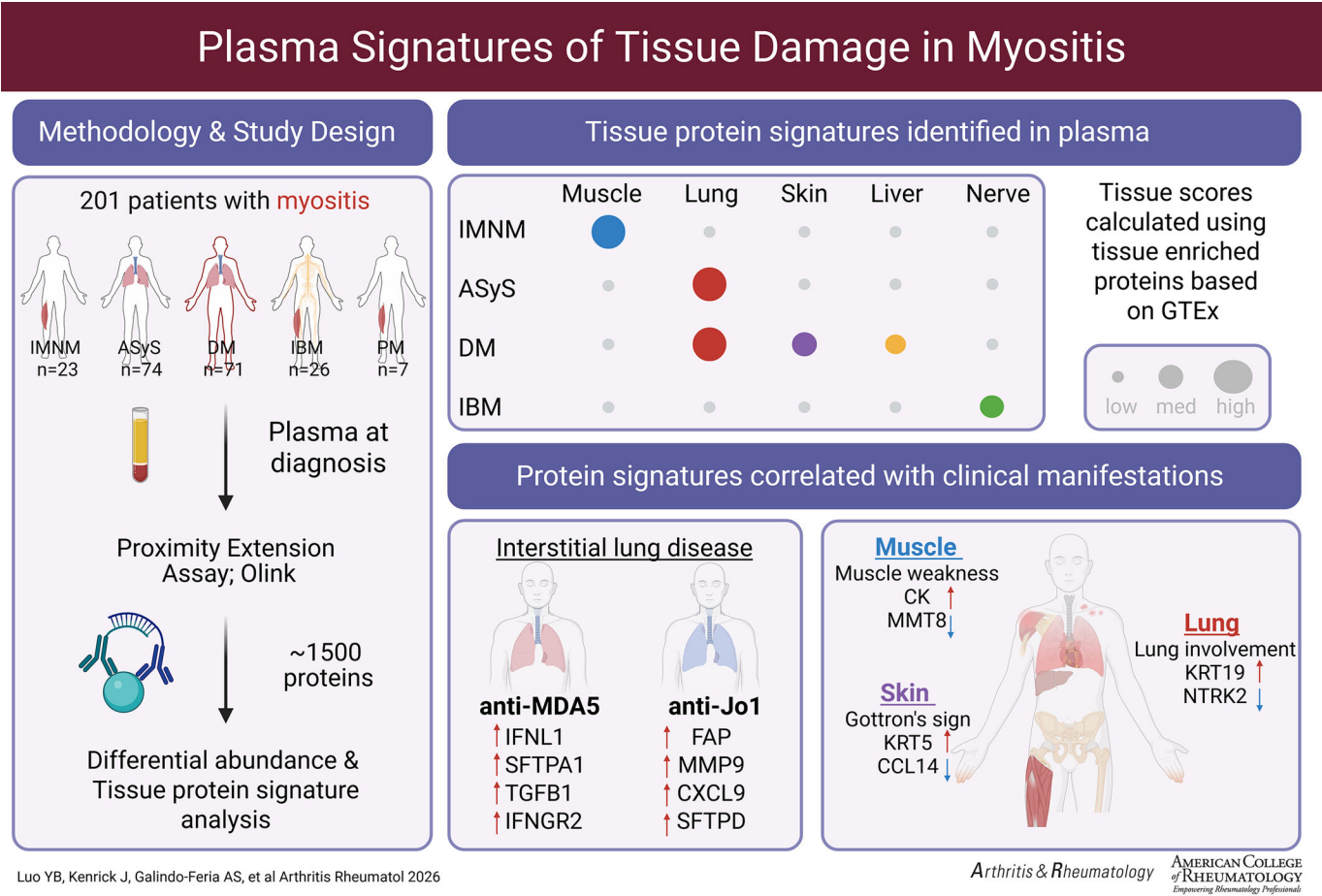
Aortic valve stenosis is common in the elderly and lacks drug treatment. We show that iron from intraleaflet hemorrhage drives cell death called ferroptosis. Blocking this pathway improved valve function in mice and circulating fatty acid levels predicted disease.

PUBLICATION: Qin Z, Haftbaradaran Esfahani P, Pawelzik SC, Shu S, Bosman M, Goovaerts B, De Wilde L, Yang Y, Bergström G, Guns PJ, Franco-Cereceda A, **Bäck M**. Ferroptosis promotes aortic stenosis through 5-lipoxygenase. Eur Heart J. 2026 Jun 2;ehag397. doi: 10.1093/eurheartj/ehag397



Plasma proteome reflects tissue damage and clinical manifestations in patients with inflammatory myopathies

GRAPHIC ABSTRACT



Luo YB, Kenrick J, Galindo-Feria AS, et al Arthritis Rheumatol 2026

PUBLICATION: Luo YB, Kenrick J., Galindo-Feria AS., Notarnicola A., Ulloa Navas AS, Peralta-Garcia I., Demuynck O, Kemp A., Leclair V, Lodin K., Van Gompel E., Dani L., Espinosa-Ortega F, Dastmalchi M., Padyukov L., Preger C., Alvez MB., Uhlen M., Nilsson P, Edfors F., Diaz-Gallo LM., Pin E., Lundberg IE., Horuluoglu B. Plasma proteome reflects tissue damage and clinical manifestations in patients with inflammatory myopathies. *Arthritis Rheumatol.* 2026 Aug 27. doi: 10.1002/art.70311. Epub ahead of print. PMID: 42657660.



New insights into sudden unexpected postnatal collapse

Photo: iStock.

PUBLICATION

Sudden unexpected postnatal collapse during the first week of life is rare but can have deleterious consequences. A new study from researchers at CMM, Karolinska Institutet and Karolinska University Hospital, shows that the condition is more common than previously estimated and highlights measures that may reduce the risk.

Sudden unexpected postnatal collapse (SUPC) occurs when an apparently healthy newborn suddenly stops breathing and collapses during the first week of life. In a new study, published in the journal *Acta Paediatrica*, researchers investigated how common the condition is and when it occurs.

The researchers analysed approximately 483,000 births at seven maternity units in Stockholm between 2002 and 2022 and identified 149 cases of SUPC. This corresponds to 31 cases per 100,000 live births.

"It is important to remember that this is a very rare condition. According to our findings, it affects around

30 infants each year in Sweden, and two to four of these cases lead to death. Most cases occur during the infant's first day of life," says the study's senior author, CMM Group Leader Eric Herlenius, paediatrician at Karolinska University Hospital and Professor of Paediatrics at the Department of Women's and Children's Health, Karolinska Institutet.

The study found that 81 per cent of collapses occurred during the first 24 hours after birth, with half taking place within four hours of delivery. Seven per cent of affected infants died and 26 per cent sustained permanent neurological injuries. Two-thirds of the cases occurred while the infant was sharing a bed with a parent.

"Skin-to-skin contact is important for newborn infants, but parents need to ensure that the baby's airways are always clear and visible. Adults should not fall asleep while holding their baby skin to skin, and infants should not sleep in the same bed as their parents during the first three months of life," says Eric Herlenius

The researchers point out that SUPC still lacks a specific diagnostic code, making the condition difficult to monitor. According to the researchers, this may have contributed to an underestimation of its true incidence. ►

Reviewed medical records

To identify cases, the researchers reviewed electronic medical records of infants born after at least 35 weeks of pregnancy. The records were searched for symptoms suggestive of collapse, such as episodes of apnoea, bluish skin discolouration, or sudden loss of muscle tone. Each suspected case was then assessed according to internationally established criteria for SUPC.

Since 2011, the researchers have also collected urine samples from affected infants and compared them with samples from healthy infants of the same age. They found higher levels of a prostaglandin E2 metabolite in infants who experienced SUPC during the first days of life, the same period during which most collapses occurred

“We believe that clearer guidelines for safe skin-to-skin care and safe sleep environments are needed and could help reduce the number of cases further,” says Eric Herlenius.

At the same time, the researchers aim to improve understanding of the biological mechanisms underlying the condition, including the elevated levels of prostaglandin E2 metabolite observed in some affected infants. By studying the relationship between these levels and the brainstem’s control of breathing, the researchers hope to gain a better understanding of why some newborns develop SUPC and, in some cases, sudden unexpected death.



Eric Herlenius. Photo: Private.

Swedish Research Council, the Swedish Brain Foundation, and Swedish government research funding provided to Region Stockholm (ALF funding) funded the research. The authors declare no conflicts of interest.

This text is based on an article published on the Karolinska Institutet news site.

PUBLICATION:

“Sudden unexpected postnatal collapse: A retrospective cohort study describing incidence, risk factors and role of perinatal transition”, David Forsberg, Louise Steinhoff, Gustaf Drevin, **Helena Idborg**, Nicolas J. Pejovic, **Eric Herlenius**. *Acta Paediatrica*, online 14 July 2026 doi: 10.1111/apa.70670

Can immune therapy restore fertility in premature ovarian insufficiency?

PUBLICATION

A recently published study in *New England Journal of Medicine Evidence*, and led by professor Olle Kämpe at CMM, suggests that immunotherapy could help restore fertility in some women with premature menopause due to autoimmune disease.

Premature ovarian insufficiency (POI) is a condition in which ovarian function declines before the age of 40. It affects around 3.5% of women worldwide, and it is characterized by reduced estrogen production, irregular or absent menstrual periods, and infertility. Women with POI may also experience symptoms commonly associated with menopause, such as hot flashes, and face increased long-term risks of osteoporosis, cardiovascular disease, and cognitive decline. For many patients, the diagnosis can be particularly distressing because fertility is often severely compromised.

In most cases of POI the causes are unknown. However, research suggests that up to one-third of patients have an autoimmune form of the disease, in which the immune system mistakenly attacks ovarian tissue. Current treatment mainly focuses on hormone replacement to relieve symptoms and protect long-term health. While spontaneous recovery of ovarian function may occur, there are currently no established treatments that reliably restore fertility.

A recent proof-of-concept study has raised the possibility that immune-targeted therapy could change this situation. Researchers at CMM and Karolinska Institutet investigated rituximab, a monoclonal antibody that depletes B lymphocytes and is widely used to treat certain autoimmune diseases. Ten women with autoimmune POI underwent ovarian hyperstimulation before and after receiving two rituximab infusions.



Image: iStock.

Before treatment, none of the participants responded with follicular development to ovarian stimulation. Four to six months after rituximab treatment, six of the ten women developed follicles sufficient for egg retrieval. In three of these cases, retrieved eggs were successfully fertilized, embryos were preserved, and pregnancies were later achieved. The children were born healthy. In addition, eight of the ten women experienced at least a temporary return of menstrual cycles, suggesting partial restoration of ovarian activity.

Although the study involved only ten women and requires confirmation in larger clinical trials, the results are highly encouraging. If future studies confirm these findings, treatments such as rituximab could potentially restore ovarian function and improve fertility prospects for women with autoimmune POI.

PUBLICATION: "Immunotherapy for Fertility in Autoimmune Premature Ovarian Insufficiency". Hirschberg, A.L., Björnsdóttir, S., Gunnarsson, I., Sergouniotis, F., Wånggren, K., Sousa Silva, C., Vogt, E., Øksnes, M., Husebye, E.S., Bensing, S. and Kämpe, O. (2026). *NEJM Evidence*, 5(7).



Joint Ectopic Calcification Meeting

September 28-30, 2026

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An Invitation to Communicate Science Beyond Our Labs

CMM OUTREACH

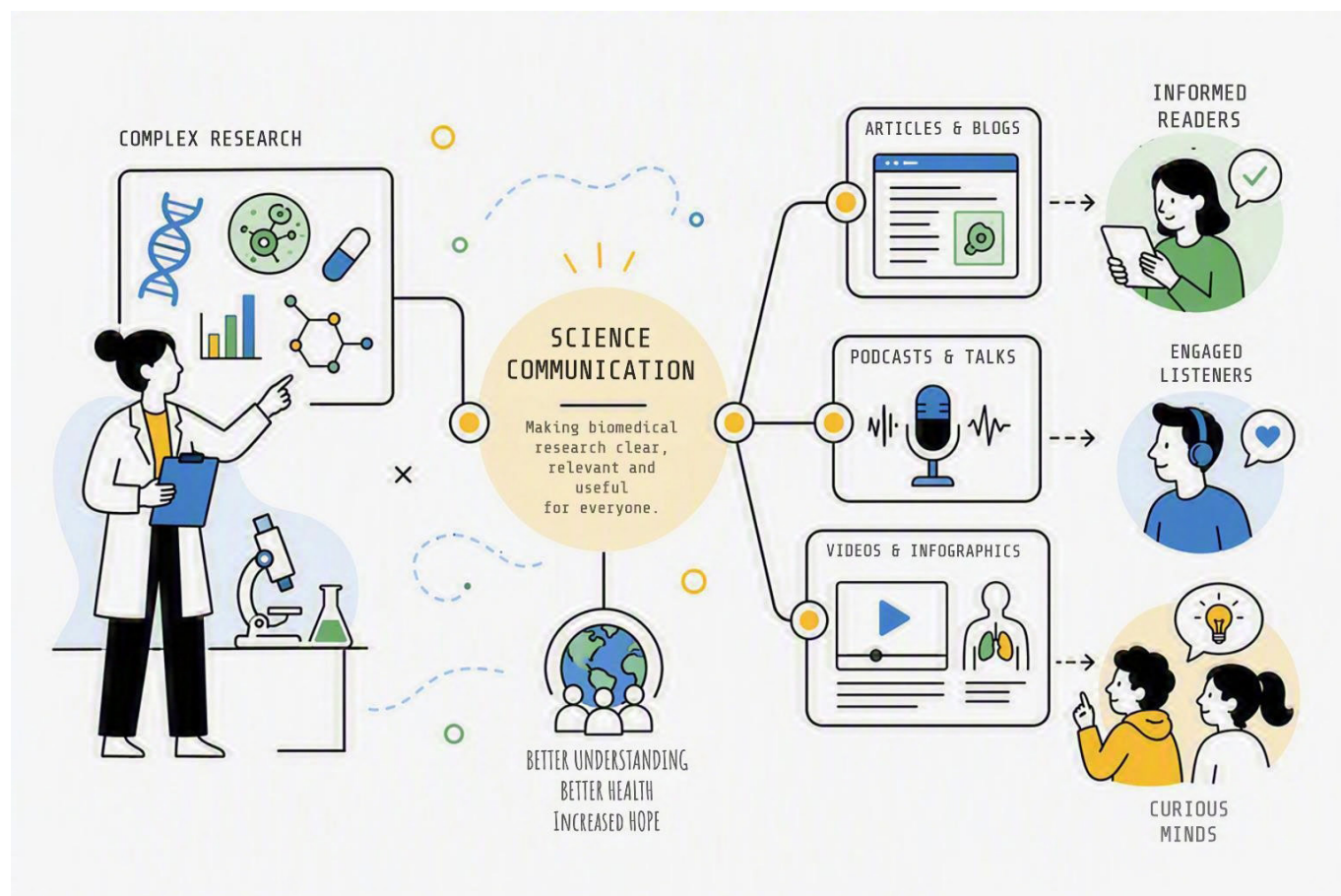


Image: AI-generated.

Dear fellow scientists and friends,

I would like to invite all of us to invest a little more of our effort, focus, and energy in science communication. As researchers, we are remarkably good at generating knowledge. However, many of us are less skilled at communicating that knowledge to the general public, and it is crucial that we become better at it. The COVID-19 pandemic provided an important mirror. Social media undoubtedly amplified polarization and misinformation, but part of the problem was also that scientists and research institutions have not consistently dedicated enough time, resources, and energy to systematically communicate our work to society.

This is a friendly invitation for all of us to help change that.

A Recent Experience

Recently, my team, together with many wonderful colleagues and friends, organized an event for families of children living with systemic lupus erythematosus (SLE). The experience was incredibly rewarding. You can find information and a video from the event here: <https://reumatiker.se/blogg/tillsammans-for-framtiden-moten-som-gor-skillnad>

Following the event, I received heartwarming feedback from several participating families. One comment from a mother particularly stayed with me:

“What an absolutely fantastic day you organized last Friday! So many of our friends whom we told about it immediately thought about how important it is—especially given the current global climate—for children and

young people to learn about research and understand how incredibly important it is in everything from saving lives to creating hope.”

This serves as a powerful reminder that science communication is not only about sharing facts. It is about building trust, creating understanding, and inspiring hope.

ForskarFredag, 25 September

ForskarFredag <https://forskarfredag.se> is Sweden's largest science festival and the national contribution to European Researchers' Night <https://marie-sklodowska-curie-actions.ec.europa.eu/european-researchers-night>. Its purpose is to bring researchers and the public together through hands-on activities, experiments, lectures, laboratory visits, workshops, quizzes, citizen science projects, and discussions about research.

This year, together with several colleagues and friends from different associations of Spanish speaking scientists in Sweden (such as RICO <https://www.rico-suecia.org/#home> and ACES <https://aces-sffs.com>, AIChIS <https://www.linkedin.com/in/association-of-chilean-researchers-in-sweden-aichis/>), I will be participating at the Instituto Cervantes from 13:00 to 15:00, sharing science in Spanish with the public <https://forskarfredag.se/festivalprogram/sociedad-celulas-ia/>.

I hope many of you will find opportunities to engage in ForskarFredag activities or other outreach initiatives throughout the year.

Two Simple and Practical Tips

Tip 1: Practice Answering Three Questions

Imagine you are talking to your grandmother, your neighbour, or a child or teenager. Then try to answer these three questions about your research:

1. What do you do?
2. How do you do it?
3. Why is it important?

I know that our research is often complex and highly specialized. The goal of this exercise is not to oversimplify science, but to learn how to translate complex ideas into language that people can understand and relate to.

Tip 2: Always Include a Lay Summary

Whenever you prepare a poster, presentation, or talk, include a short lay summary.

Try to see your research through the eyes of a patient living with the disease you study, or through the eyes of a

family member seeking answers and hope. Be thoughtful about the language you use and strive to communicate clearly. The three questions above can serve as a helpful guide.

A Final Reflection

As we are already full speed in this new academic term, I would like to remind each and every one of you that your work matters. You are talented, capable, and deeply needed by society. The discoveries made in our laboratories today become the treatments, technologies, policies, and hope of tomorrow.

Science communication is not separate from research. It is part of our responsibility as scientists. By sharing what we do, why we do it, and why it matters, we help build a society that understands, trusts, and benefits from science.

I will leave you with two reflections, not to discourage you, but to remind you why we do what we do:

<https://www.instagram.com/reel/DchSc9CxxvY/?stkn=cZ3d3Ntdmhkd2Ny>

The value of research is not measured only by the papers we publish or the grants we get (although they are essential), but also by the lives we touch and the hope we create.

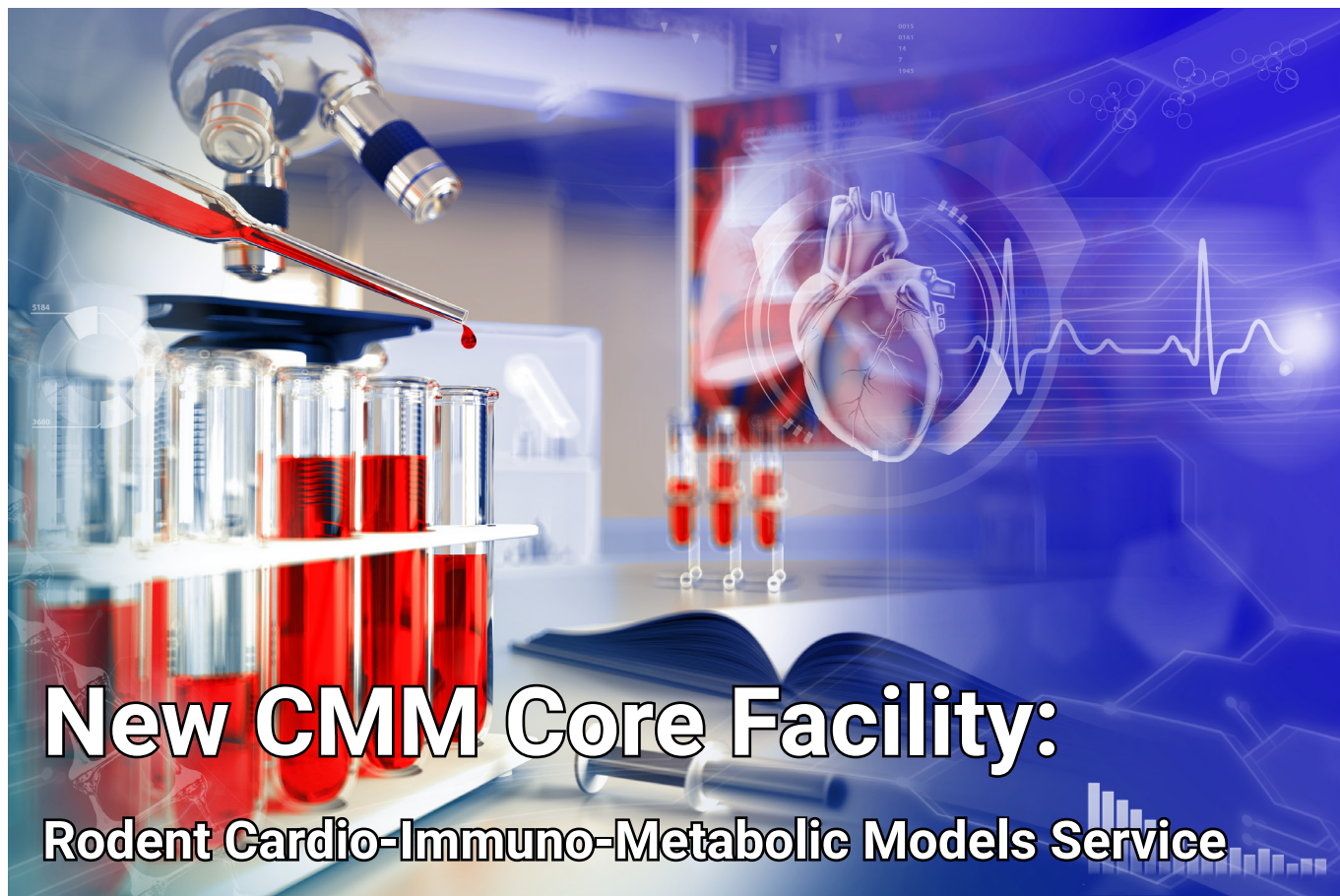
All the best for each of you.

Warm regards,

Lina Marcela Díaz-Gallo



Photo: Gustaf Norlén.



New CMM Core Facility:

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Advanced rodent models for cardiovascular, metabolic, and immunological research

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Interested in discussing your project? Contact us for an introduction to our services and experimental possibilities.

Locations: Biomedicum and Bioclinicum, Karolinska Institutet.

Contacts:

Ljubica Matic, Associate Professor
Ljubica.Matic@ki.se

Hong Jin, Senior Research Specialist in Animal Vascular Surgery & Animal Lab Manager
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Read more about this core facility on the [CMM website](#) (you can also scan the QR code).

Update from the CMM Director

Dear CMMers,

Welcome back from the vacation period. I hope you all had a relaxing and enjoyable summer, for travelling, family and friends – and have gained energy for the coming autumn and winter. Personally, I spent most of my vacation weeks with the grandchildren, which was fun, but also intense.

The major work-related activity during the summer has been the rebuilding of the café area on floor 0, just outside the lecture hall. The construction work has mostly gone according to plan, with some smaller hick-ups, which has caused a couple of weeks delay.

As you can see when entering the building, the area formerly occupied by the (Glada) Restaurant, has been or is in the process of being refurbished. This includes new floors, toilets, a glass wall separation towards the

corridor and a separate kitchen, in which the food 'ATM', a coffee machine and a water fountain soon will be installed. A bit later into September, new furniture will also arrive. I believe this will be a great new area for interaction, networking, receptions and seminars. The new area and the renovated lecture hall will be inaugurated when all has been completed, most likely in mid-October.

At their latest meeting, the CMM Board of Directors approved the required budget for hosting another 'CMM Research Day 2027' – which likely will be arranged about the same time (May) at the same venue as in 2025. More details to follow.

I wish you all a wonderful and successful second half of the year

Michael



Image: iStock



Next deadline for sending in
contributions to CMM News:
1st October.