40th ANNIVERSARY

THE Crafoord PRIZE

Crafoord *Days* 2022

25–27 APRIL IN LUND, SWEDEN

PHOTO: ANNGRI

DANIEL L. KASTNER

The Crafoord *Prize* in Polyarthritis 2021

Abstracts and Programmes

Anna-Greta and Holger Crafoord Fund

THE FUND WAS ESTABLISHED in 1980 by a donation to the Royal Swedish Academy of Sciences from Anna-Greta and Holger Crafoord. The Crafoord Prize was awarded for the first time in 1982. The purpose of the fund is to promote basic scientific research worldwide in the following disciplines:

- Mathematics
- Astronomy
- Geosciences
- Biosciences (with particular emphasis on Ecology)
- Polyarthritis (e.g. rheumatoid arthritis)

Support to research takes the form of an international prize awarded annually to outstanding scientists and of research grants to individuals or institutions in Sweden. Both awards and grants are made according to the following order:

year 1: Mathematics and Astronomy
year 2: Geosciences
year 3: Biosciences (with particular emphasis on Ecology)
year 4: Mathematics and Astronomy
etc.

The Prize in Polyarthritis is awarded only when the Academy's Class for medical sciences has shown that scientific progress in this field has been such that an award is justified.

Part of the fund is reserved for appropriate research projects at the Academy's institutes. The Crafoord Prize presently amounts to 6 million Swedish krona.

The Crafoord Prize is awarded in partnership between the Royal Swedish Academy of Sciences and the Crafoord Foundation in Lund. The Academy is responsible for selecting the Crafoord Laureates.

Content

PROGRAMMES

The Crafoord Laureate in Polyarthritis 2021

PHOTO: NHGRI

DANIEL L. KASTNER

NATIONAL HUMAN GENOME RESEARCH INSTITUTE, NATIONAL INSTITUTES OF HEALTH, BETHESDA, USA

Daniel L. Kastner, National Human Genome Research Institute, National Institutes of Health, Bethesda, USA,
"*for establishing the concept of autoinflammatory diseases*".

The Crafoord *Prize* in Polyarthritis

Crafoord Laureate discovered the explanation for mysterious fevers

Daniel (Dan) L. Kastner, receives the 2021 Crafoord Prize in Polyarthritis "for establishing the concept of autoinflammatory diseases". He has identified the mechanisms responsible for familial Mediterranean fever, TRAPS and other diagnoses within this group. They are genetic diseases that are unusual in most of the world, but may have a higher incidence in some areas. One or two of every thousand people in the eastern Mediterranean have familial Mediterranean fever, while TRAPS was initially discovered among families in Ireland and Scotland.

Even just 20 years ago, researchers could not explain why those afflicted had recurring fevers, abdominal pain, joint inflammation, troublesome rashes and muscle aches. Dan Kastner started by studying patients with familial Mediterranean fever, and discovered that it was caused by a mutation in a single gene. He then identified the cause of TRAPS and established the concept of autoinflammatory diseases.

In autoimmune diseases and autoinflammatory diseases, the body's tissues are attacked by the immune system. However, in autoinflammatory disease the problem is within the immune system itself. Recently, research has broadened the understanding of rare (monogenic) and more common (polygenic) autoinflammatory diseases such as Crohn's disease and gout.

Dan Kastner is currently working on Behçet's disease, which is often found along the old Silk Road and is caused by inflammation in the blood vessels.

ABSTRACTS IN POLYARTHRITIS

Crafoord *Days* 2022

Autoinflammatory Disease and the Human Condition

CRAFOORD LAUREATE 2021 **DANIEL L. KASTNER**, NATIONAL HUMAN GENOME RESEARCH INSTITUTE, USA

Autoinflammatory diseases are the consequence of nonlethal natural variation in the innate immune system. Chance and the fallibility of DNA replication provide an endless font of subjects for this science. Here I describe our recent work in 3 areas: (1) new germline inborn errors of innate immunity; (2) the role of pathogen-associated natural selection in perpetuating autoinflammatory disease; and (3) the role of somatic mutation in adult-onset autoinflammatory disease. I will discuss recent work defining germline 'cleavage-resistant RIPK1-induced autoinflammatory (CRIA)' syndrome; studies of a biallelic loss-of-function mutation in SHARPIN, a component of the linear ubiquitin assembly complex; and work on the clinical features, pathophysiology, and treatment of ROSAH (retinal dystrophy, optic nerve edema, splenomegaly, anhidrosis, headache) syndrome, caused by gain-of-function mutations in a sensor of bacterial 7-carbon sugars. Regarding selection, I will discuss the population genetics of familial Mediterranean fever (FMF). Carrier frequencies for multiple FMF mutations are very high in several populations, with the genomic signature of evolutionarily recent selection. Functional studies in leukocytes from FMF patients, heterozygous carriers, and healthy controls, as well as survival studies in knock-in mice, support an IL-1-dependent role for FMF mutations in protection from *Yersinia pestis*, the agent of bubonic plague. Finally, I will describe the discovery of myeloid-restricted somatic mutations in *UBA1*, encoding a key ubiquitylation enzyme, in middle-aged men. This condition, denoted VEXAS (vacuoles, E1 enzyme, X-linked, autoinflammatory, somatic) syndrome, includes patients clinically diagnosed with several illnesses, thus validating genotype-first strategies for refining taxonomy, and raising the possibility that somatic mutation may be a major cause of adult-onset autoinflammation, just as it is an important cause of cancer and other sequelae of aging.

Lalaoui N, Boyden SE, Oda H, *et al.* (2020) Mutations that prevent caspase cleavage of RIPK1 cause autoinflammatory disease. *Nature* **577**:103–108, PMID:31827281.

Park YH, Remmers EF, Lee W, *et al.* (2020) Ancient familial Mediterranean fever mutations in human pyrin and resistance to *Yersinia pestis*. *Nat Immunol* **21**:857–867, PMID:32601469.

Beck DA, Ferrada MA, Sikora KA, *et al.* (2020) Somatic mutations in *UBA1* and severe adult-onset autoinflammatory disease. *N Engl J Med* **383**:2628–2638, PMID:33108101.

Defects in lymphocyte cytotoxicity as a cause of hyperinflammation

YENAN BRYCESON, KAROLINSKA INSTITUTET, SWEDEN

Cytotoxic CD8+ T cells as well as natural killer (NK) cells can recognize and eradicate infected and neoplastic cells. Studies of families with early onset, life-threatening hyperinflammatory syndromes clinically fulfilling the criteria for hemophagocytic lymphohistiocytosis have unraveled a set of genes required for lymphocyte cytotoxicity and highlighted how killing of target cells also is vital for maintenance of immune homeostasis. I will review genetic predisposition to HLH and discuss how molecular insights have provided fundamental knowledge of the immune system as well as detailed pathophysiological understanding of hyperinflammatory diseases, highlighting new treatment strategies.

Autoinflammation and Autoimmunity: Psoriatic Skin and Joint Disease at the Midpoint of the Spectrum

HARALD BURKHARDT, UNIVERSITY HOSPITAL FRANKFURT AM MAIN, GOETHE-UNIVERSITY, FRAUNHOFER INSTITUTE FOR TRANSLATIONAL MEDICINE AND PHARMACOLOGY ITMP, GERMANY

Psoriasis (Pso) is a chronic immune-mediated inflammatory disease of the skin associated with joint manifestations (psoriatic arthritis, PsA) in up to 30% of patients. PsA includes extra-articular involvement and comorbidities that impact the quality of the patient´s life. PsA-susceptibility is determined by polygenic inheritance and environmental risk factors. The multifaceted systemic disorder is driven by a complex pathogenesis combining aspects of autoinflammation and autoimmunity. Features of autoinflammation encompass the impact of biomechanical stress as trigger for disease onset and relapse, the absence of disease specific autoantibodies, and detectability of innate immune response mediators in the skin and synovium. Conversely, a role of autoimmune mechanisms is suggested by genetic associations with class I major histocompatibility complex alleles (Pso: HLA-C*06:02, PsA: HLA-B27), evidence for oligoclonal expansions of $CD8^+$ T cells in psoriatic skin and synovial fluid of PsA-joints. In addition, infiltrates of IL-17 expressing T cells of the $CD4^+$ (Th17) as well as $CD8^+$ (Tc17) lineage detectable in the inflamed synovial tissue could be interpreted as a hint on (auto)antigen evoked immunity in PsA-joints. However, besides conventional T cells responding with cytokine release to MHC-restricted antigen recognition, tissue resident unconventional T cell subsets of innate immunity e.g. innate lymphoid cells (ILC, especially the ILC3 phenotype) or γδ T cells have been demonstrated to produce proinflammatory cytokines like IL-17, IL-22 or TNF locally at predilection sites of PsA, e.g. at the entheses of peripheral joints or the spine. These cells are responsive to myeloid derived IL-23, being also crucial for effector phenotype differentiation of Th17 and Tc17 lymphocytes. Accordingly, the IL-23/IL-17 axis is central to the communication between innate and adaptive immunity in PsA pathogenesis and a preferential target of current therapeutic interventions.

Genetic and immunological causes of life-threatening COVID-19

JEAN-LAURENT CASANOVA, THE ROCKEFELLER UNIVERSITY, USA AND NECKER HOSPITAL, PARIS, FRANCE

Clinical outcome upon infection with SARS-CoV-2 ranges from silent infection to lethal COVID-19. We have found an enrichment in rare variants predicted to be loss-of-function (LOF) at the 13 human loci known to govern TLR3- and IRF7-dependent type I interferon (IFN) immunity to influenza virus, in 659 patients with life-threatening COVID-19 pneumonia, relative to 534 subjects with asymptomatic or benign infection. By testing these and other rare variants at these 13 loci, we experimentally define LOF variants in 23 patients (3.5%), aged 17 to 77 years, underlying autosomal recessive or dominant deficiencies. We show that human fibroblasts with mutations affecting this pathway are vulnerable to SARS-CoV-2. Inborn errors of TLR3- and IRF7-dependent type I IFN immunity can underlie life-threatening COVID-19 pneumonia in patients with no prior severe infection.

Also, interindividual clinical variability in the course of SARS-CoV-2 infection is immense. We report that at least 101 of 987 patients with life-threatening COVID-19 pneumonia had neutralizing IgG auto-Abs against IFN-ω (13 patients), the 13 types of IFN-a (36), or both (52), at the onset of critical disease; a few also had auto-Abs against the other three type I IFNs. The auto-Abs neutralize the ability of the corresponding type I IFNs to block SARS-CoV-2 infection *in vitro*. These auto-Abs were not found in 663 individuals with asymptomatic or mild SARS-CoV-2 infection and were present in only 4 of 1,227 healthy individuals. Patients with auto- Abs were aged 25 to 87 years and 95 were men. A B cell auto-immune phenocopy of inborn errors of type I IFN immunity underlies life-threatening COVID-19 pneumonia in at least 2.6% of women and 12.5% of men.

The human type I interferonopathies

YANICK J. CROW, UNIVERSITY OF EDINBURGH, UK

As brutally demonstrated by the COVID-19 pandemic, an effective immune system is essential for survival. Developed over evolutionary time, viral nucleic acid detection represents a central pillar in the defensive armamentarium employed to combat foreign microbial invasion. To ensure cellular homeostasis, such a strategy necessitates the efficient discrimination of pathogen-derived DNA and RNA from that of the host. In 2011, it was suggested that an upregulation of type I interferon signalling might serve as a defining feature of a novel set of Mendelian inborn errors of immunity, where anti-viral sensors are triggered by host nucleic acids due to a failure of self versus non-self discrimination. These rare disorders have played a surprisingly significant role in informing our understanding of innate immunity and the relevance of type I interferon signalling for human health and disease. Here I will consider some aspects of what we have learned in this time, and how the field may develop in the future.

Intracellular Complement and Immune Regulation

CLAUDIA KEMPER, NATIONAL HEART, LUNG, AND BLOOD INSTITUTE, USA

The complement system has been acknowledged for over a century as a liver-derived and serum-operative arm of innate immunity. It is a key component of the host's pathogen- and danger-associated molecular pattern (PAMPs and DAMPs) recognition receptor (PPR) repertoire and is at the center of our defense against bloodborne pathogens.

We have recently discovered, however, that this ancient system is not confined to the extracellular space but also operates within cells where it impacts on cell behavior in an intrinsic fashion. The intracellular complement system (the complosome) is present in a broad range of immune (and non-immune) cells and is among the defining features of tissue-resident immune cells. Functionally, the complosome is a central coordinator of cell metabolic pathways: it controls the expression of nutrient transporters allowing nutrient influx, directly modulates the activity of glycolytic enzymes, supports oxidative phosphorylation, and is required for cellular oxygen turn-over and mitochondrial activity – all events underlying normal immune cell functions. Consequently, perturbations in complosome activity contribute to immune cell hypo- or hyperactivity and several human diseases, including recurrent infection, autoimmunity/autoinflammation, and cancer.

Importantly, the complosome does not operate in isolation but engages with other innate immune sensor systems, including the toll-like receptors, the MAVS protein and the inflammasomes. During this Crafoord Symposium lecture, we discuss the intracellular complement system, specifically its intersection with some of the molecular pathways that have been defined by this year's recipient of the Crafoord Prize in Polyarthritis, Dr. Daniel L. Kastner.

Inflammasome activation in autoinflammatory diseases: from signaling pathways to functional diagnosis

MOHAMED LAMKANFI, GHENT UNIVERSITY, BELGIUM

Inflammasomes are multi-protein complexes that promote production of the inflammatory cytokines interleukin (IL)-1β and IL-18 from innate immune cells. In addition, inflammasomes induce cleavage of Gasdermin D to induce an inflammatory cell death mode termed pyroptosis. Inflammasomes play a key role in host defense against microbial pathogens, but uncontrolled inflammasome activation also drives detrimental autoinflammatory diseases. Here, I will review work from my laboratory describing how altered inflammasome signaling contributes to inflammatory pathology in preclinical models of autoinflammatory diseases, and highlight recent work illustrating how such mechanistic insights can be translated into functional immunoassays to support differential diagnosis of Familial Mediterranean Fever.

Transglutaminases as targets in autoimmune disease

NILS LANDEGREN, UPPSALA UNIVERSITY, SWEDEN

The transglutaminase protein family holds a unique place in autoimmunity with presently six of its nine members implicated as autoantigens in clinically distinct disorders. Tissue transglutaminase (TGM2) is the major autoantigen in celiac disease. Gluten-sensitivity frequently involves also the skin, and in rare cases the nervous system. Dermatitis herpetiformis is associated with autoantibodies against epidermal transglutaminase (TGM3), while gluten-related cerebellar ataxia has been linked with autoantibodies against TGM6. Acquired haemophilia can develop from autoantibodies targeting coagulation factor FXIII, another member of the transglutaminase family, and we recently identified TGM4 as a prostate-specific autoantigen in autoimmune polyendocrine syndrome type 1 with a possible role in the male infertility seen in this condition.

Because of the frequent roles of transglutaminases in autoimmunity, we decided to explore whether remaining members might also constitute autoantigens in as yet unexplained disorders. The TGM1 protein is primarily expressed in squamous epithelia, and we therefore focused on skin diseases as possible manifestations of potential autoimmune reactions. By investigating sera from patients with various forms of acquired skin disorders we could indeed identify TGM1 as a major autoantigen in the severe bullous disease paraneoplastic pemphigus. Our results suggest that TGM1 autoantibodies are 100% specific for paraneoplastic pemphigus and they may therefore be clinically useful as a diagnostic marker that should prompt investigations for an occult malignancy in patients presenting with bullous disease.

Our study of TGM1 illustrates a novel approach to biomarker discovery, starting with a putative autoantigen to search for the corresponding disease. This gene-centric approach leverages the increasing wealth of data available for human genes in public databases, and may prove broadly applicable for biomarker discovery in autoimmune diseases.

Genetics of Behçet's Disease, a Journey On and Off the Silk Roads

ELAINE REMMERS, NATIONAL HUMAN GENOME RESEARCH INSTITUTE, USA

Behçet's disease (BD) is a complex autoinflammatory disease often recognized by the symptoms the Turkish physician Behçet first used to describe it, a combination of painful oral and genital ulceration, skin erythema and ocular inflammation. It is common in Middle Eastern and Far Eastern populations and has therefore been termed the Silk Road disease. We performed genome-wide association studies (GWAS) in Silk Road populations to identify genetic variants that might provide clues to disease pathogenesis. We identified a strong disease-associated interaction of the MHC class I HLA-B*51 with a haplotype of *ERAP1*, the aminopeptidase responsible for trimming peptides for loading on MHC class I molecules, implicating peptide antigen presentation in BD. Surprisingly, 8 of the 17 loci we identified as susceptibility loci for BD were reported to have genome-wide significance for recurrent aphthous ulcers (RAU). We hypothesized that PFAPA (periodic fever, aphthous stomatitis, pharyngitis, adenitis), a common childhood autoinflammatory disease, might also share genetic susceptibility loci with BD and RAU, and found 4 shared loci, illustrating genetic similarities among this range of Behçet's spectrum disorders. The shared risk alleles likely render individuals more susceptible to abnormal innate-immune cell function and heightened T cell activation. In populations outside of the Silk Roads, BD is too uncommon for GWAS, but on and off the Silk Road we found rare families with apparently dominantly inherited, early onset autoinflammatory disease consistent with BD diagnosis. In several such multi-case families we identified novel loss of function mutations in *TNFAIP3* encoding A20, a negative regulator of the proinflammatory NF-kappa-B complex, and termed their Behçet-like disease HA20 for haploinsufficiency of A20. Loss of function mutations in *TNFAIP3* did not explain Behçet's-like familial disease in other families, suggesting other genetic explanations, are yet to be found.

PROGRAMME

Crafoord *Days* 2022

Overview programme Crafoord *Days* 2022

Monday 25 April | LUX, HELGONAVÄGEN 3, LUND — 09:30

THE CRAFOORD PRIZE LECTURE IN **MATHEMATICS**
Held by the Crafoord Laureate **Enrico Bombieri**.

THE CRAFOORD PRIZE LECTURE IN **ASTRONOMY**
Held by **Nicola Fox,** on behalf of the late Crafoord Laureate **Eugene N. Parker**.

THE CRAFOORD PRIZE LECTURE IN **POLYARTHRITIS**
Held by the Crafoord Laureate **Daniel L. Kastner**.

THE CRAFOORD PRIZE LECTURE IN **GEOSCIENCES**
Held by the Crafoord Laureate **Andrew H. Knoll**.

Registration at www.kva.se

Tuesday 26 April

09:35 CRAFOORD PRIZE SYMPOSIUM IN **MATHEMATICS**
Number Theory
Lectures by the Crafoord Laureate **Enrico Bombieri** and invited speakers.
THE FACULTY OF ENGINEERING, LTH, ANNEXET, MA5, SÖLVEGATAN 20, LUND

09:00 CRAFOORD PRIZE SYMPOSIUM IN **ASTRONOMY**
Solar wind and magnetic fields in space
Lectures by **Boon Chye Low,** on behalf of the late Crafoord Laureate **Eugene N. Parker** and invited speakers.
THE FACULTY OF ENGINEERING, LTH, ANNEXET, MA7, SÖLVEGATAN 20, LUND

09:00 CRAFOORD PRIZE SYMPOSIUM IN **POLYARTHRITIS**
Autoinflammatory diseases
Lectures by the Crafoord Laureate **Daniel L. Kastner** and invited speakers.
KULTUREN IN LUND, TEGNÉRSPLATSEN 6, LUND

09:30 CRAFOORD PRIZE SYMPOSIUM IN **GEOSCIENCES**
The evolution of life on Earth through deep time
Lectures by the Crafoord Laureate **Andrew H. Knoll** and invited speakers.
LUX, HELGONAVÄGEN 3, LUND

Wednesday 27 April | LUND UNIVERSITY ASSEMBLY HALL, UNIVERSITY MAIN BUILDING, PARADISGATAN 2, LUND — 16:15

THE CRAFOORD PRIZE AWARD CEREMONY
In the presence of **HRH Crown Princess Victoria**.

By invitation only.

Detailed programme

The Crafoord *Prize* Lectures in Mathematics, Astronomy, Polyarthritis and Geosciences

LUX,
HELGONAVÄGEN 3,
LUND

Monday 25 April

Seating is limited. For registration and further information visit:
www.kva.se/en/crafoordprizelectures2022

09:30	Presentation of the Crafoord *Prize*	Nils Dencker, Chair of the Crafoord Prize Committee in Mathematics
09:35	Introduction of the Crafoord Laureate in Mathematics 2020	Per Salberger, Member of the Royal Swedish Academy of Sciences
09:45	*The zeta function: a mystery 283 years old*	CRAFOORD LAUREATE Enrico Bombieri, School of Mathematics, Institute for Advanced Study, Princeton University, USA
10:20	Questions from the audience	CHAIR: Per Salberger, Member of the Royal Swedish Academy of Sciences
10:30	COFFEE BREAK	
10:50	Introduction of the Crafoord Laureate in Astronomy 2020	Dainis Dravins, Member of the Royal Swedish Academy of Sciences
11:00	*The Challenge of Exploring Our Sun: the 60-Year Odyssey to Parker Solar Probe*	Nicola Fox, Science Mission Directorate, NASA Headquarters, Washington, USA, on behalf of the late CRAFOORD LAUREATE Eugene N. Parker, University of Chicago, USA
11:35	Questions from the audience	CHAIR: Dainis Dravins, Member of the Royal Swedish Academy of Sciences
11:45	LUNCH	(Included for registered participants)
12:45	Introduction of the Crafoord Laureate in Polyarthritis 2021	Rikard Holmdahl, Member of the Crafoord Prize Committee in Polyarthritis
12:55	*Cutting the Gordian Knots of Inflammation with the Shears of Genomics*	CRAFOORD LAUREATE Daniel L. Kastner, National Human Genome Research Institute, Bethesda, USA
13:30	Questions from the audience	CHAIR: Rikard Holmdahl, Member of the Crafoord Prize Committee in Polyarthritis
13:40	COFFEE BREAK	
14:00	Introduction of the Crafoord Laureate in Geosciences 2022	Daniel Conley, Member of the Crafoord Prize Committee in Geosciences
14:10	*The Deep History of Life*	CRAFOORD LAUREATE Andrew H. Knoll, Harvard University, Cambridge, USA
14:45	Questions from the audience	CHAIR: Daniel Conley, Member of the Crafoord Prize Committee in Geosciences
14:55	End of the Crafoord *Prize* Lectures	

Detailed programme

THE CRAFOORD SYMPOSIUM IN POLYARTHRITIS 2022

Autoinflammatory diseases

KULTUREN IN LUND,
TEGNÉRSPLATSEN 6,
LUND

Tuesday 26 April

Seating is limited. For registration and further information visit: www.kva.se/en/crafoordpolyarthritis2022

09:00	Opening address	Hans Ellegren, Secretary General, the Royal Swedish Academy of Sciences
09:05	Introduction of the Crafoord Laureate	Olle Kämpe, Chair of the Crafoord Prize Committee in Polyarthritis
09:15	*Autoinflammatory Disease and the Human Condition*[1]	CRAFOORD LAUREATE Daniel L. Kastner, National Human Genome Research Institute, USA
10:05	COFFEE BREAK	(Included for registered participants)
10:35	*Inflammasome activation in autoinflammatory diseases: from signalling pathways to functional diagnosis*[1]	Mohamed Lamkanfi, Ghent University, Belgium
11:10	*Autoinflammation and Autoimmunity: Psoriatic Skin and Joint Disease at the Midpoint of the Spectrum*[2]	Harald Burkhardt, University Hospital Frankfurt am Main, Goethe-University Fraunhofer Institute for Translational Medicine and Pharmacology ITMP, Germany
11:45	LUNCH	(Included for registered participants)
12:45	*The human type I interferonopathies*[3]	Yanick Crow, University of Edinburgh, UK
13:30	*Transglutaminases as targets in autoimmune disease*[3]	Nils Landegren, Uppsala University, Sweden
13:55	*Genetics of Behçet's Disease, a Journey On and Off the Silk Roads*[3]	Elaine Remmers, National Human Genome Research Institute, USA
14:30	COFFEE BREAK	(Included for registered participants)
15:00	*Intracellular Complement and Immune Regulation*[4]	Claudia Kemper, National Heart, Lung, and Blood Institute, USA
15:35	*Defects in lymphocyte cytotoxicity as a cause of hyperinflammation*[5]	Yenan Bryceson, Karolinska Institutet, Sweden
16.00	*Genetic and immunological causes of life-threatening COVID-19*[5]	Jean-Laurent Casanova, The Rockefeller University, USA and Necker Hospital, Paris, France
16.45	END OF SYMPOSIUM	

CHAIRS:

1 Olle Kämpe, Chair of the Crafoord Prize Committee in Polyarthritis.

2 Solbritt Rantapää Dahlqvist, Member of the Crafoord Prize Committee in Polyarthritis.

3 Qiang Pan Hammarström, Karolinska Institutet, Sweden.

4 Anna Blom, Lund University, Sweden.

5 Rikard Holmdahl, Member of the Crafoord Prize Committee in Polyarthritis.

Anna-Greta and Holger Crafoord

Holger Crafoord (1908–1982) was prominent in Swedish industry and commerce. He began his career with AB Åkerlund & Rausing and devoted a larger part of his working life to this company. In 1964, Holger Crafoord founded Gambro AB in Lund, Sweden, where the technique of manufacturing the artificial kidney was developed. This remarkable dialyser soon became world famous. Since then, a series of medical instruments has been introduced on the world market by Gambro.

HOLGER AND ANNA-GRETA CRAFOORD

In 1980, Holger Crafoord founded the Crafoord Foundation, which annually contributes greatly to the Anna-Greta and Holger Crafoord Fund.

Holger Crafoord became an honorary doctor of economics in 1972 and in 1976 an honorary doctor of medicine at Lund Univeristy.

Anna-Greta Crafoord (1914–1994) took, as Holger Crafoord's wife, part in the development of Gambro AB. Through generous donations and a strong commitment in the society around her, she contributed to the scientific and cultural life. In 1986 she founded the Anna-Greta Crafoord foundation for rheumatological research and in 1987 Anna-Greta Crafoord became an honorary doctor of medicine at Lund Univeristy.

Over the years, the Crafoords have furthered both science and culture in many ways and it is noteworthy that research in the natural sciences has received an important measure of support from the Anna-Greta and Holger Crafoord Fund.

THE ROYAL SWEDISH ACADEMY OF SCIENCES was founded in 1739 and is an independent non-governmental organisation, whose overall objective is to promote the sciences and strengthen their influence in society. The Academy has a particular responsibility for natural science and mathematics, but its work strives to increase interaction between different disciplines. The activities of the Royal Swedish Academy of Sciences primarily focus on:

- being a voice of science in society and influencing research policy (policy for science)
- providing a scientific basis for public debate and decision-making (science for policy)
- recognizing outstanding contributions to research
- being a meeting place for science, within and across subject boundaries
- providing support for young researchers
- stimulating interest in mathematics and natural science in school
- disseminating knowledge to the public
- mediating international scientific contacts
- preserving scientific heritage

THE ACADEMY has around 460 Swedish and 175 foreign members who are active in classes, committees and working groups. They initiate enquiries, consultation documents, conferences and seminars. The Academy has General Meetings eight times a year. Open lectures are held in association with these (read more at www.kva.se/kalendarium). They can also be watched via www.kva.se/video.

THE ACADEMY´S institutes offer unique research environments in ecological economics, botany, the history of science and mathematics.

Every year, the Academy awards a number of prizes and rewards. The best known are the Nobel Prizes in Physics and Chemistry and the Sveriges Riksbank Prize in Economic Science in Memory of Alfred Nobel (the Prize in Economic Sciences). Other major prizes are the Crafoord Prize, Sjöberg Prize, Göran Gustafsson Prizes, Söderberg Prize and the Tobias Prize. The Göran Gustafsson Prizes are awarded to outstanding young researchers and are a combination of a personal prize and research funding. Since 2012, the Academy of Sciences has been one of the academies involved in implementing the Wallenberg Academy Fellows career programme, which provides long-term funding to the most promising young researchers. As well as a comprehensive range of scholarships, the Academy is also involved in appointments to research posts in a number of programmes funded by external foundations.

Through its working groups and committees, the Academy also works to promote sustainable, science-based societal development in the area of energy and the environment, among others. Issues relating to education and conditions for teachers are another major interest. The Academy regularly organises lectures and workshops on various scientific topics for teachers and students. In the 1990s, the Academy and the Royal Swedish Academy of Engineering Sciences founded one of Sweden's biggest school development programmes, NTA – Naturvetenskap och teknik för alla (Science and Technology for all).

THE CRAFOORD PRIZE IS AWARDED IN PARTNERSHIP BETWEEN THE ROYAL SWEDISH ACADEMY OF SCIENCES AND THE CRAFOORD FOUNDATION IN LUND. THE ACADEMY IS RESPONSIBLE FOR SELECTING THE CRAFOORD LAUREATES.

WWW.CRAFOORDPRIZE.SE

KUNGL. VETENSKAPS-AKADEMIEN
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