THE Crafoord PRIZE

Crafoord *Days* 2025

5–8 MAY IN LUND AND STOCKHOLM, SWEDEN

The Crafoord *Prize* in Polyarthritis 2025

Abstracts and Programmes

PHOTO: GARVAN INSTITUTE OF MEDICAL RESEARCH

CHRISTOPHER C. GOODNOW

PHOTO: SCRIPPS RESEARCH

DAVID NEMAZEE

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 outstandig scientists, and of research grants to individuals or institutions in Sweden. Both awards and grants are today made according to the following order:

year 1: Mathematics and Astronomy
year 2: Polyarthritis
year 3: Geosciences
year 4: Biosciences
year 5: Mathematics and Astronomy
etc.

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

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.

ÅTTA45, JÄRFÄLLA 2025
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Content

The Crafoord *Prize* Laureates in Polyarthritis 2025

PHOTO: GARVAN INSTITUTE OF MEDICAL RESEARCH

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PHOTO: SCRIPPS RESEARCH

DAVID NEMAZEE
SCRIPPS RESEARCH, USA

Christopher C. Goodnow, born 1959 in Hong Kong. The Bill and Patricia Ritchie Foundation Chair, Garvan Institute of Medical Research and Professor at Cellular Genomics Futures Institute, School of Biomedical Sciences, UNSW Sydney, Australia.

David Nemazee, born 1956 in Shiraz, Iran. Professor at the Department of Immunology and Microbiology, Scripps Research, La Jolla, California, USA.

"for the discovery of fundamental mechanisms for B cell tolerance"

The Crafoord *Prize* in Polyarthritis

Fundamental mechanisms for B cell tolerance

Normally, the body's immune system protects us from viruses, bacteria and foreign substances. However, in autoimmune diseases, the immune system starts attacking tissues in the body instead. Researchers had long tried to discover the cause of autoimmune diseases. But, Christopher Goodnow and David Nemazee, independently of each other, adopted a new approach. They asked why we do not all develop these diseases. Their focus was on B cells which, together with white blood cells and T cells, are part of the complex immune system. They discovered important mechanisms that normally prevent faulty B cells from attacking tissues in the body, explaining why most of us are not affected by autoimmune diseases.

In recent years, physicians have started to experiment by using existing drugs to neutralise B cells for patients with severe autoimmune diseases, including lupus, rheumatoid arthritis and multiple sclerosis. This has proven to be very effective.

Thanks to this year's Crafoord Prize Laureates, we have gained fundamental new knowledge about what is happening in the immune system in autoimmune disease. This may also lead to completely new therapies, perhaps even a cure in the future.

Crafoord *Days* 2025

Monday 5 May | LUX, HELGONAVÄGEN 3, LUND — 10:00

THE CRAFOORD PRIZE LECTURES IN POLYARTHRITIS

Held by the Crafoord *Prize* Laureates **Christopher C. Goodnow** and **David Nemazee**.

Wednesday 7 May | THE BEIJER HALL, THE ROYAL SWEDISH ACADEMY OF SCIENCES, LILLA FRESCATIVÄGEN 4A, STOCKHOLM — 09:30

THE CRAFOORD PRIZE SYMPOSIUM IN POLYARTHRITIS

Registration at www.kva.se

B Cell Biology

Lectures by the Crafoord *Prize* Laureates **Christopher C. Goodnow** and **David Nemazee** and invited speakers.

Thursday 8 May | THE BEIJER HALL, THE ROYAL SWEDISH ACADEMY OF SCIENCES, LILLA FRESCATIVÄGEN 4A, STOCKHOLM — 16:30

THE CRAFOORD PRIZE AWARD CEREMONY

By invitation only.

In the presence of **HM The King**.

The Crafoord *Prize* Lectures in Polyarthritis

LUX, HELGONAVÄGEN 3, LUND

Monday 5 May

Seating is limited. For registration and further information visit:
www.kva.se/crafoordprizelectures2025

10:00	Presentation of the Crafoord *Prize*	Olle Kämpe, Chair of the Crafoord Prize Committee in Polyarthritis
10:05	Introduction of the Crafoord *Prize* Laureate **Christopher C. Goodnow**	Qiang Pan Hammarström, Member of the Crafoord Prize Committee in Polyarthritis
10:10	*Keeping the peace within our body*	CRAFOORD PRIZE LAUREATE Christopher C. Goodnow, Garvan Institute of Medical Research and UNSW Sydney, Australia
10:45	Questions from the audience	CHAIR: Qiang Pan Hammarström, Member of the Crafoord Prize Committee in Polyarthritis
10:55	COFFEE BREAK	
11:25	Introduction of the Crafoord *Prize* Laureate **David Nemazee**	Rikard Holmdahl, Member of the Crafoord Prize Committee in Polyarthritis
11:30	*Immunological Tolerance in B Lymphocytes*	CRAFOORD PRIZE LAUREATE David Nemazee, Scripps Research, USA
12:05	Questions from the audience	CHAIR: Rikard Holmdahl, Member of the Crafoord Prize Committee in Polyarthritis
12:15	End of the Crafoord *Prize* Lectures	Olle Kämpe, Chair of the Crafoord Prize Committee in Polyarthritis
12:20	LUNCH	Lunch is served outside the lecture hall and included for registered participants.

The Crafoord *Prize* Symposium in Polyarthritis

B Cell Biology

THE BEIJER HALL,
THE ROYAL SWEDISH ACADEMY OF SCIENCES,
LILLA FRESCATIVÄGEN 4A, STOCKHOLM

Seating is limited. For registration and further information visit:
www.kva.se/crafoordprizesymposium2025

Wednesday 7 May

09:30	Opening address	**Hans Ellegren**, Secretary General, The Royal Swedish Academy of Sciences
09:35	Presentation of the Crafoord *Prize* Laureate **Christopher C. Goodnow**	**Qiang Pan Hammarström**, Member of the Crafoord Prize Committee in Polyarthritis
09:45	*B cell tolerance: how it is kept and how it fails*	CRAFOORD PRIZE LAUREATE **Christopher C. Goodnow**, Garvan Institute of Medical Research and UNSW Sydney, Australia
10:35	COFFEE BREAK	
11:05	*Autoreactive B cell responses in rheumatic autoimmune disease; what makes them different?*	**Rene Toes**, Leiden University Medical Center, The Netherlands
11:35	*Glycosylation and selection of the B-cell receptor enable lymphoma and autoimmunity*	**Freda K. Stevenson**, University of Southampton, UK
12:05	*Early-Life Origin B Cells in the Adult Immune System*	**Joan Yuan**, Lund University, Sweden
12:00	LUNCH	(included for registered participants)
13:30	Presentationof the Crafoord *Prize* Laureate **David Nemazee**	**Rikard Holmdahl**, Member of the Crafoord Prize Committee in Polyarthritis
13:40	*Quality control and learning in the antibody system*	CRAFOORD PRIZE LAUREATE **David Nemazee**, Scripps Research, USA
14:30	*Surprising features of the human naïve B cell repertoire*	**Gunilla Karlsson Hedestam**, Karolinska Institutet, Sweden
15:00	COFFEE BREAK	
15:30	*Autoantibodies and autoreactive B cells protecting against rheumatoid arthritis*	**Zhongwei Xu**, Karolinska Institutet, Sweden
16:00	*Allogeneic CAR-T therapy in autoimmune diseases*	**Huji Xu**, Shanghai Changzheng Hospital, China
16:30	*Resetting autoimmune disease by B-cell depletion*	**Georg Schett**, Uniklinikum Erlangen, Germany
17:00	End of the Crafoord Prize Symposium	**Olle Kämpe**, Chair of the Crafoord Prize Committee in Polyarthritis

MODERATORS: William Agace, Anders Bengtsson, Qiang Pan Hammarström, Rikard Holmdahl and Olle Kämpe

ABSTRACTS

Crafoord *Days* 2025

B cell tolerance: how it is kept and how it fails

CRAFOORD PRIZE LAUREATE 2025 **CHRISTOPHER C. GOODNOW**
GARVAN INSTITUTE OF MEDICAL RESEARCH AND UNSW SYDNEY, AUSTRALIA

More than a century ago, blood serum and cell therapies were pioneered by Von Behring and Landsteiner. Achieving benefit, not harm, hinged upon recognizing that horse but not human gammaglobulin provoked antibodies causing serum sickness, and that erythrocytes bearing A, B, or RhD antigens only provoked hemolytic antibodies in people lacking these antigens on their own erythrocytes.

Ehrlich recognized in 1901 that mechanisms prevent antibody formation against our own elements: *"These contrivances are naturally of the highest importance for the existence of the individual"*.

Burnet conceived in 1957 that single lymphocytes with the potential to make antibodies against self-elements would be deleted. However, in the 1960's-80's evidence amassed of B lymphocytes circulating with the potential to make antibodies against self gammaglobulin or self erythrocytes. Most formed the view that B cell tolerance didn't exist: secretion of autoantibodies was secondary to tolerance in helper T cells or the actions of I-J+ CD8+ suppressor T cells.

It took until the end of the 1980's to see Ehrlich's contrivances clearly and directly, acting upon individual B cells bearing self-reactive antibodies. It needed a century of technological advances: antibody structure and diversification mechanisms, monoclonal antibodies, multiparameter flow cytometry, gene cloning, DNA sequencing, and tools to modify the genome of cells and animals.

Here I will focus on how B cell tolerance normally prevents secretion of pathogenic rheumatoid factors against our own gammaglobulin, and prevents secretion of pathogenic autoagglutinins against our erythrocyte Individuality (I) antigens. I will then describe what has been learnt about how secretion of these autoantibodies occurs and reaches the point where "rogue clones" precipitate human autoimmune disease. The phenotype of these rogue clones explains remission of autoimmunity by depleting CD19+ but not CD20+ cells.

Quality control and learning in the antibody system

CRAFOORD PRIZE LAUREATE 2025 **DAVID NEMAZEE**
SCRIPPS RESEARCH, USA

The process of immune tolerance hinders potentially destructive antibody responses against host tissues that are produced by B lymphocytes. Autoantibodies contribute to diseases such as rheumatoid arthritis and systemic lupus erythematosus where tolerance breaks down. Tolerance works in part by reducing the number of B-cells in the body that are self-reactive before they can make antibodies. Our studies focused on tracking and analyzing the fate of autoreactive B cells. B cells are made continually throughout life from precursors in the bone marrow, each equipped with a distinct cell surface receptor able to be secreted as antibody. To visualize rare autoreactive cells, we took advantage of genetic engineering in mice to increase the frequency of B-cells specific for targets of interest. Our findings and those of our colleagues have led to a consensus that tolerance is regulated partly at an immature stage of B cell development by either selective B-cell death or by receptor editing, a process which reprograms the specificity of B cells through secondary recombination of antibody genes. Receptor editing represented a novel, unexpected mechanism of tolerance that was appealing for its efficiency and elegance. The receptor editing model also helped us to understand the counterintuitive features of antibody genes. The first and main part of the lecture will review these studies. The second part of the lecture will describe more recent studies from my laboratory that investigate how mature self-reactive B cells are regulated and how host receptors for sialic acids and nucleic acids can modify tolerance responses.

Surprising features of the human naïve B cell repertoire

GUNILLA KARLSSON HEDESTAM
KAROLINSKA INSTITUTET, SWEDEN

B cells, each expressing a unique B cell receptor (BCR), are produced from hematopoietic stem cells throughout life, providing us with a near limitless capacity to recognize different antigens. The antigen-binding portions of BCRs are encoded by polymorphic immunoglobulin (IG) variable (V), diversity (D) and joining (J) genes that assemble in a combinatorial manner to generate the BCR repertoire. Due to the complexity of the IG loci (highly repetitive and interspersed with pseudogenes), population differences in these genes have not been systematically characterized and most of our current knowledge about the human B cell repertoire comes from studies of European ancestry individuals.

To address this shortcoming, we developed a technique, *ImmuneDiscover*, for multiplex-targeted sequencing of genomic DNA for high-confidence personalized typing of non-rearranged V, D and J genes. By applying *ImmuneDiscover* to over 2500 individuals representing 25 different population groups from around the world, we identified over 500 gene variants that are lacking in currently available reference databases, many of which displayed substantial variation in population frequency. We also found haplotypes with large segmental deletions and copy number variation, adding further diversity to human IG genotypes and illustrating how the IG loci have been shaped by host/pathogen interactions in different geographic regions during human evolution.

To begin to investigate the functional role of this diversity, we combined personalized IG genotyping with studies of expressed antibody repertoires and isolation of antigen-specific monoclonal antibodies. Using functional and structural analyses, we discovered several examples of how IG germline gene variation impacts B cell recognition of foreign antigens. In ongoing work, we are investigating the role of IG gene diversity for the development of autoimmune diseases where genetic factors that predispose to disease remain insufficiently understood.

Glycosylation and selection of the B-cell receptor enable lymphoma and autoimmunity

FREDA K. STEVENSON
UNIVERSITY OF SOUTHAMPTON, UK

The B-cell receptor (BCR) is the key which unlocks the response to antigen, and, in mature B cells, expression of surface immunoglobulin (sIg) of the BCR is required for survival. However, the nature of the response to antigen is influenced by the developmental status of the B cell and is calibrated by the strength of engagement. Most B-cell tumors follow the rules in retaining expression of sIg, but have to find a substitute for antigen to provide optimal levels of stimulation for growth and survival. Follicular lymphomas modify sIg by selecting Ig variable region (IGV) sequences mandated to contain N-glycosylation sites introduced during somatic hypermutation. Addition of sugars to the sites terminates at oligomannoses, a highly unusual spectrum of sugars, facilitated in these lymphomas by steric boundaries conferred by the surrounding IGV sequences. The acquisition of oligomannoses diverts sIg binding away from antigen to a lectin, DC-SIGN, expressed by follicular dendritic cells in the tumor follicle, thereby acquiring continued support for proliferation. Tumor cells of a different origin, chronic lymphocytic leukemia (CLL), have an alternative strategy to modify the BCR. In CLL, the more aggressive form is derived from the natural antibody repertoire, where autoreactivity is common; here IGHV sequences are selected for binding to autoantigens, including oxidized lipoproteins of apoptotic cells. Engagement of autoantigen in tissues is calibrated by reduced expression of sIg, further reversibly downregulated by antigen encounter. The behavior of CLL cells gives insight into the generation and function of non-tumor autoantibodies against modified self-proteins found in patients with rheumatoid arthritis. The balance in B cells between producing antibodies in infection and autoimmunity, and the subversion of sIg by tumors indicates the wide-ranging opportunism used by B cells to survive and grow, and offers defined therapeutic targets.

Autoreactive B cell responses in rheumatic autoimmune disease; what makes them different?

RENE TOES
LEIDEN UNIVERSITY MEDICAL CENTER, THE NETHERLANDS

Many autoimmune diseases respond well to therapies targeting B cells, emphasizing the importance of autoreactive B cells in disease induction and progression. Tolerance mechanisms usually prevent the development of autoantibodies, but when these systems fail, autoimmunity and subsequent autoimmune diseases can arise. Understanding the dynamics of autoreactive B cell responses is crucial for delineation the pathogenic pathways underlying disease.

Rheumatoid arthritis (RA), is a prototypic autoimmune disease featuring autoreactive B cell responses against post-translationally modified proteins. The Anti-Citrullinated Protein Antibody (ACPA) response targeting citrullinated proteins is hallmarking disease. Recently, we have shown that the "ACPA B cell response" behaves differently compared to e.g. anti-viral responses as autoreactive B cells display an activated phenotype similar to virus-specific B cells shortly after infection, but do not become quiescent in time. Interestingly, the activated autoreactive B cells remain active despite clinical disease control across a range of interventions. This persistent activity indicates the absence of immunological remission and might explain why ACPA-positive RA-patients rarely reach sustained drug-free remission and frequently flare upon drug tapering.

The reason why the autoreactive B cells continuously express an activated phenotype is not known, but conceivably relates to continuous antigen-recognition, combined with an unique feature of ACPA/citrullinated antigen-directed BCRs; the expression of N-linked glycans in the variable domain. We estimated that a large proportion of ACPA and citrullinated antigen-directed BCRs on B cells are glycosylated in the variable domains. Combining crystallography, glycobiology, and functional B cell assays, allowed us to dissect how these glycans can affect human B cell biology.

The reasons and consequences of the features displayed by autoreactive antibodies and B cells will be discussed.

Allogeneic CAR-T therapy in autoimmune diseases

HUJI XU
SHANGHAI CHANGZHENG HOSPITAL, CHINA

Allogeneic chimeric antigen receptor (CAR)-T cells hold great promise for expanding the accessibility of CAR-T therapy, whereas the risks of allograft rejection have hampered its application. Here, we genetically engineered healthy-donor-derived, CD19-targeting CAR-T cells using CRISPR-Cas9 to address the issue of immune rejection and treated with refractory immune-mediated necrotizing myopathy, diffuse cutaneous systemic sclerosis and systemic lupus erythematosus with these cells. The infused cells persisted for over 3 months, achieving complete B cell depletion within 2 weeks of treatment. During the 6-month follow-up, we observed deep remission without cytokine release syndrome or other serious adverse events in all patients, primarily shown by the significant improvement in the clinical response index scores for the three diseases, respectively, and supported by the observations of reversal of inflammation and fibrosis. Single-cell RNA and BCR/TCR sequencing revealed notable shifts in immune cell composition and function following allogeneic CAR-T cell infusion, with the BCR repertoire reprogramming. Plasma proteomics further revealed substantial changes in the *in vivo* environment, with upregulated pathways linked to immune modulation and tissue repair, while pro-inflammatory pathways were downregulated. Our results demonstrate the high safety and promising immune modulatory effect of the off-the-shelf CAR-T cells in treating severe refractory autoimmune diseases.

Autoantibodies and autoreactive B cells protecting against rheumatoid arthritis

ZHONGWEI XU
KAROLINSKA INSTITUTET, SWEDEN

Rheumatoid arthritis (RA) is a common and debilitating disease that progressively destroys the joints. It is preceded by the presence of autoantibodies, which can appear years before disease onset, and is widely believed to be driven by autoimmune responses. However, our findings suggest a more complex picture, where certain types of autoimmunity may actually protect against disease development. Specifically, we have identified several human autoimmune mediators within B cell immunity that exhibit significant preventive or therapeutic potential in mouse models of arthritis. These protective factors include specific clones of anti-citrullinated protein antibodies, anti-collagen antibodies, and certain antigen-specific B cells. Our results point to the possible existence of a universal protective autoimmunity, at least within B cell immunity. Promoting this protective aspect of autoimmunity could provide a novel therapeutic strategy for RA and other autoimmune diseases.

Early-Life Origin B Cells in the Adult Immune System

JOAN YUAN
LUND UNIVERSITY, SWEDEN

The adult immune system comprises cells with diverse lifespans that emerge at various stages of ontogeny. Over the past decade, our laboratory has focused on elucidating the relationship between developmental origin and composition of the adult B cell pool. Through multiple lineage tracing approaches, we have stratified the murine adult B cell pool based on the timing of output, revealing that a substantial portion originates within a restricted neonatal window. Our findings demonstrate that early-life time-stamped B cells include not only B-1a cells but also IgA plasma cells that are actively maintained by B cell memory within gut chronic germinal centers. Notably, neonatal rotavirus infection recruits recurrent IgA clones distinct from those arising from infection with the same antigen in adults, suggesting that neonatally imprinted B cells confer unique antibody responses later in life.

Further investigation revealed that gut IgA plasma cells and B-1a cells arise from the same hematopoietic progenitors during ontogeny. These progenitors are characterized by the expression of the fetal-specific post-transcriptional regulator Lin28b. Our results demonstrate that Lin28b is both necessary and sufficient to potentiate enhanced positive selection during B cell development, resulting in the positive selection of self-reactive CD5+ immature B cells. Mechanistically, Lin28b amplifies the CD19/PI3K/c-Myc positive feedback loop, and ectopic Lin28b expression restores both positive selection and mature B cell numbers in CD19-/- adult mice. In conclusion, we propose that the temporally restricted expression of Lin28b controls a critical window for the selection of unique BCR specificities into the mature B cell pool that are amenable to antigenic imprinting early in life. These findings provide crucial insights into the impact of early life exposure for the formation of a complex adult B cell repertoire.

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 making Gambro a leading company in this field.

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 University.

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 University.

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 480 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 about seven times each year.

THE ACADEMY´S INSTITUTES offer unique research environments in ecological economics, 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, the Sjöberg Prize, the Göran Gustafsson Prizes, the Söderberg Prize and the Rolf Schock Prizes. The Göran Gustafsson Prizes are awarded to outstanding young researchers and are a combination of a personal prize and research funding. Since 2012, the Royal Swedish Academy of Sciences is involved in the Wallenberg Academy Fellows career programme, which provide 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.

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