
WELCOME TO THE

THE
Crafoord
PRIZE 

Crafoord *Days* 2009

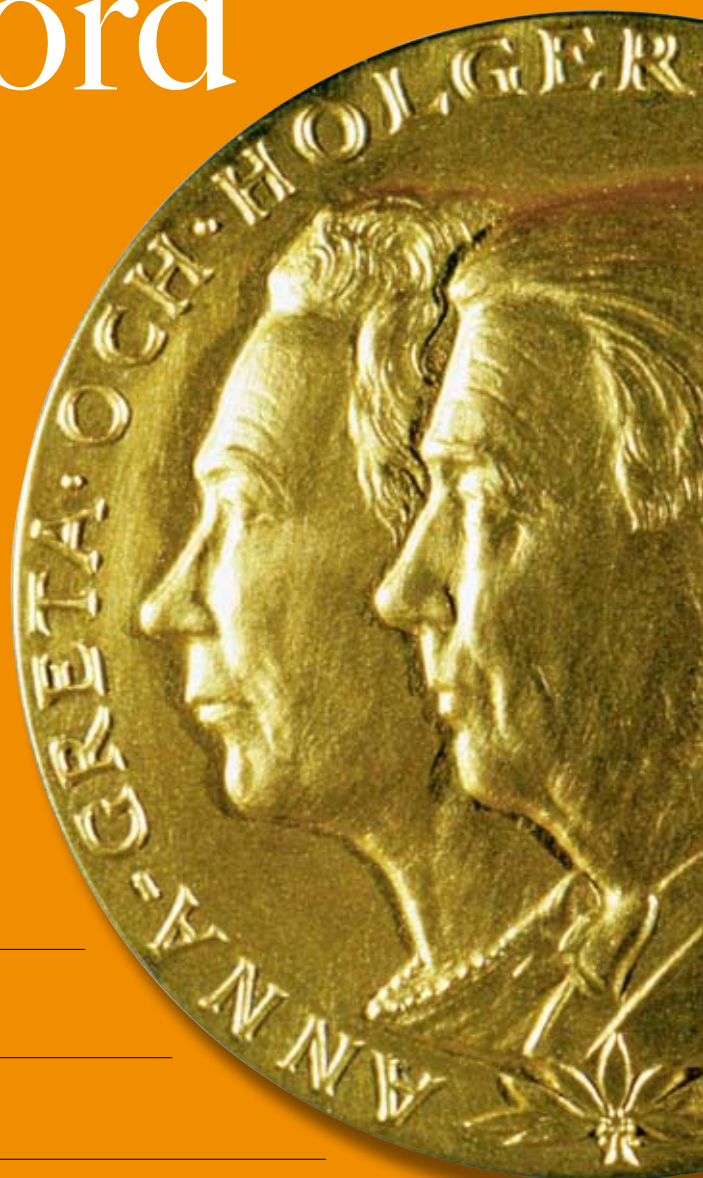
11-13 MAY



Programme

Abstracts

The Crafoord Prize in
Polyarthritis 2009



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:

- Astronomy and Mathematics
- Geosciences
- Biosciences
- Polyarthrititis

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: Astronomy and Mathematics
year 2: Geosciences
year 3: Biosciences
year 4: Astronomy and Mathematics
year 5: Geosciences
year 6: Biosciences
et.c.

The prize in Polyarthrititis is awarded only when a special committee 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 USD 500 000. In addition to the prize, financial support is granted to other researchers in the same field in which the prize is awarded for that year.

The Crafoord Prize is awarded by the Royal Swedish Academy of Sciences.

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Monday

11 MAY, STOCKHOLM

16.20–17.40

Prize award ceremony

Presented by His Majesty the King of Sweden

BEIJER HALL
ROYAL SWEDISH ACADEMY OF SCIENCES
LILLA FRESCATIVÄGEN 4A, STOCKHOLM
No registration

Tuesday

12 MAY, STOCKHOLM

10.00–16.30

Prize Symposium in Polyarthritits

CYTOKINE REGULATION IN INFLAMMATION

Lectures by the Crafoord Laureates 2009: Prof. **CHARLES A. DINARELLO**, Prof. **TADAMITSU KISHIMOTO**, Prof. **TOSHIO HIRANO**, and invited speakers Sir Ravinder N. Maini, Prof. Marc Feldmann, Prof. Cem Gabay and Dr. Helena Erlandsson Harris

NOBEL FORUM, KAROLINSKA INSTITUTET
NOBELS VÄG 1, STOCKHOLM

Registration is required and must be made before 4 May 2009 at www.kva.se (aktuellt/evenemang)

Wednesday

13 MAY, LUND

13.30–16.45

The Crafoord Prize Lectures 2009

Held by the Laureates Prof. **CHARLES A. DINARELLO**, Prof. **TADAMITSU KISHIMOTO**, and Prof. **TOSHIO HIRANO**

FERNSTRÖMSALEN, BMC, LUND UNIVERSITY,
SÖLVEGATAN 19, LUND

No registration

Prize award ceremony

Programme

Rondeau

J. J. Mouret



Pingst

O. Lindberg



Opening remarks

Bo Sundqvist

PRESIDENT OF THE ROYAL SWEDISH ACADEMY OF SCIENCES



the Crafoord Laureates in Polyarthritits

Presented by *Catharina Swanborg*

CHAIRMAN OF THE PRIZE COMMITTEE



I denna ljva sommartid

Trad. B. Ollén



Plenty good room

K. Shaw



His Majesty the King presents
the Crafoord Prize in Polyarthritits 2009 to

CHARLES A. DINARELLO

TADAMITSU KISHIMOTO

and

TOSHIO HIRANO



Bort allt vad oro gör (dryckesvisa)

C.M. Bellman



His Majesty the King presents the Crafoord Prize
research grant diploma 2009 to

Magnus Bäck and Nandakumar Kutty Selva



ABBA-medley

B. Andersson & B. Ulvacus

Karl Erik Ljungholm and Fredrik Henriksson, trumpets
Sofia Vokalensemble, Bengt Ollén, conductor



Targeted ('smart') drugs to save joints

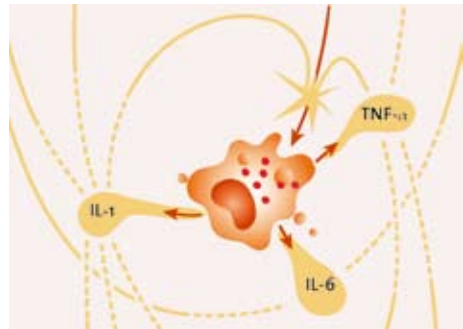
This year's Crafoord Prize Laureates discovered two new key players in the immune system of the human body: the signal substances *interleukin-1* (IL-1) and *interleukin-6* (IL-6).

Child and adult joint-disease sufferers often have high concentrations of these substances in their bodies. Activated by these substances, the immune system then starts to break down cartilage and bone. But new drugs can stop this merciless process.

When the powerful human immune system goes wrong, turning against a person's own body (an 'autoimmune' reaction), the results are painful and sometimes even life-threatening. In polyarthritis the immune system attacks the joints, slowly but surely eroding them. Today, tens of millions live with the commonest form of polyarthritis: rheumatoid arthritis. Juvenile chronic arthritis is also common, as are gout, psoriatic arthritis and Bechterew's disease —all forms of polyarthritis.

The discoveries made in the 1970s and '80s by this year's Crafoord Prize Laureates have opened up new areas of research. It has paved the way for drug development that is now helping polyarthritis patients to avoid the painful course of their disease. **CHARLES A. DINARELLO, TADAMITSU KISHIMOTO and TOSHIO HIRANO** isolated two signal substances in the immune system, IL-1 and IL-6. These substances are released from white blood cells.

In a healthy immune system, IL-1 and IL-6 have a key role in mobilising protection against bacteria, viruses and other microbes.



IL-1, IL-6 and TNF in joint inflammation.

ILLUSTRATION: Airi Iliste & Kungl. Vetenskapsakademien

When white blood cells release IL-1 and IL-6, they signal that an attack is under way. But in people with polyarthritis and other autoimmune diseases, such as type 2 diabetes, the concentrations of IL-1 and IL-6 are constantly far too high. This makes the immune system run amok.

An approved drug that inhibits the effect of IL-1 is now available. To date, this drug has proved effective against rheumatoid arthritis, gout, Still's disease in adults and certain forms of juvenile rheumatism. There are signs that it mitigates type 2 diabetes.

Another drug to combat IL-6 will soon be approved in Europe and the US. In Japan it is used, often to very good effect, against rheumatoid arthritis and Castleman's disease. It is probably also effective against some forms of juvenile chronic arthritis. ●



Prize symposium in polyarthritis

CYTOKINE REGULATION IN INFLAMMATION

Open to the public

Tuesday 12 MAY

NOBEL FORUM, KAROLINSKA INSTITUTET,
NOBELS VÄG 1, STOCKHOLM

10:00	Opening of the symposium	<i>Gunnar Öquist</i> , Permanent Secretary, the Royal Swedish Academy of Sciences
10:10	<i>Interleukin-1: The first interleukin</i>	Crafoord Laureate 2009 CHARLES A. DINARELLO , University of Colorado Denver, School of Medicine, USA
11:00	<i>Interleukin-6: from its discovery to medicine and back again</i>	Crafoord Laureate 2009 TADAMITSU KISHIMOTO , Graduate School of Frontier Biosciences, Osaka University, Japan
11:50	Lunch	
13:00	<i>Role of interleukin-6 and IL-6 amplifier in autoimmune disease and chronic inflammatory proliferative diseases</i>	Crafoord Laureate 2009 TOSHIO HIRANO , Graduate School of Medicine, Osaka University, Japan
13:50	<i>The clinical impact of cytokine blockade: a ten years perspective of TNF-antagonists</i>	<i>Sir Ravinder N. Maini</i> , Kennedy Institute of Rheumatology, London, UK, Crafoord Laureate 2000
14:20	<i>The impact of cytokine blockade for the understanding of the molecular pathogenesis of arthritis</i>	<i>Marc Feldmann</i> , Kennedy Institute of Rheumatology, London, UK, Crafoord Laureate 2000
14:50	Break with refreshments	
15:20	<i>Experiences from blockade of IL-1 in arthritis and other inflammatory conditions</i>	<i>Cem Gabay</i> , University Hospitals of Geneva, Geneva, Switzerland
15:50	<i>Young investigator lecture: HMGB1 – the ugly duckling?</i>	<i>Helena Erlandsson-Harris</i> , Karolinska Institutet, Stockholm, Sweden
16:20	End of the symposium	



The Crafoord Prize Laureates in polyarthritis 2009



CHARLES A. DINARELLO

CHARLES A. DINARELLO, American citizen. Born in 1943 in Boston, MA, USA. M.D. 1969 at Boston University, MA, USA. Since 1996 Professor of Medicine, University of Colorado Denver, School of Medicine, USA.



TADAMITSU KISHIMOTO

TADAMITSU KISHIMOTO, Japanese citizen. Born in 1939 in Tondabayashi, Osaka, Japan. M.D. in 1964 and Ph.D. of Medicine in 1969, both at Osaka University Medical School, Japan. Since 2003 Professor and Chair at the Department of Medicine III, Osaka University Medical School, Osaka, Japan.

TOSHIO HIRANO, Japanese citizen. Born in 1947 in Osaka, Japan. M.D. in 1972 and Ph.D. of Medical Science in 1979, both at Osaka University, Japan. Since 2008 Professor and Dean at the Graduate School of Medicine, Osaka University, Japan.



TOSHIO HIRANO

"for their pioneering work to isolate interleukins, determine their properties and explore their role in the onset of inflammatory diseases"



Interleukin-1 β , the first interleukin

CHARLES A. DINARELLO, CRAFOORD LAUREATE 2009
UNIVERSITY OF COLORADO DENVER, SCHOOL OF MEDICINE, USA

Interleukin 1 (IL 1) was originally studied as an endogenous protein produced during infections that caused fever. Following the cloning of IL 1 in 1984, humans were injected with recombinant IL 1 and only 10 ng/kg induced ACTH, fever, neutrophilia and several cytokines such as IL-6. Thus IL 1 is a highly inflammatory molecule. Indeed blocking IL 1 receptors or neutralizing IL 1 β results in a near complete reduction in the severity of systemic and local inflammation, including rheumatoid arthritis. The basis for chronic inflammatory diseases can be “auto-immune” or “auto-inflammatory”. In autoimmune diseases, the lympho-

cyte plays a major role and therapies targeting immune activation are effective. In auto-inflammatory diseases, the monocyte/macrophage is dysfunctional and patients are best treated by reducing IL 1 β activity. The underlying dysfunction in auto-inflammatory diseases is a failure of the monocyte to control the secretion of IL 1 β . The clinical spectrum of auto-inflammatory disease encompasses debilitating systemic inflammation and vasculitis, local inflammation such as gouty and osteoarthritis and silent but progressively destructive inflammation such as type 2 diabetes.

Interleukin 6: From its discovery to medicine and back again

TADAMITSU KISHIMOTO, CRAFOORD LAUREATE 2009
GRADUATE SCHOOL OF FRONTIER BIOSCIENCES, GRADUATE SCHOOL OF MEDICINE AND
WPI IMMUNOLOGY FRONTIER RESEARCH CENTER, OSAKA UNIVERSITY, JAPAN

IL-6 was originally discovered as T cell-derived lymphokine which induces antibody production in B cells in early 1970th. By the collaboration with Dr. T. Hirano, the IL-6 gene was isolated in 1986 and its pleiotropic activities were revealed. Through identification of the molecules involved in IL-6 mediated signal transduction, such as IL-6 receptor, gp130, NF-IL6, STAT3 and SOCS-1, the molecular mechanism by which IL-6 activates immune and inflammatory cells has been elucidated. IL-6 has been shown to be involved in the pathogenesis of cardiac myxoma, multiple

myeloma, Castleman's disease, rheumatoid arthritis (RA), Juvenile idiopathic arthritis (JIA). Anti-IL6R antibody has been prepared and applied for the treatment of these diseases. The antibody showed dramatic therapeutic effect on these inflammatory as well as proliferative diseases, even on anti-TNF-unresponsive RA as well as JIA patients. Recent studies show that IL-6 has an essential role for the induction of a novel CD4 T cell subset, TH17 cells and a transcription factor (Arylhydrocarbon receptor) responsible for autoimmune inflammation.



Role of interleukin 6 and IL-6 amplifier in autoimmune disease and chronic inflammatory proliferative diseases



TOSHIO HIRANO, CRAFOORD LAUREATE 2009

GRADUATE SCHOOL OF FRONTIER BIOSCIENCES, GRADUATE SCHOOL OF MEDICINE AND
WPI IMMUNOLOGY FRONTIER RESEARCH CENTER, OSAKA UNIVERSITY, JAPAN

Thirty years ago, I found pleural effusion cells in patients with tuberculous pleurisy produce an active factor that induces immunoglobulin production in B cells and started isolating it. In 1986, we succeeded to clone IL-6 and thereafter both its receptor and gp130, a signal transducer. IL-6 was found to be a multifunctional cytokine and its involvement in rheumatoid arthritis (RA) was suggested. We then showed IL-6 induces both STAT3 and SHP2/Gab/MAPK signals. To clarify the in vivo roles of each signal, we made several knock-in mice expressing mutated variants of gp130, and showed F759 mice, which have an amino acid substitution (Y759F) and an enhanced STAT3

activation, developed a RA-like arthritis. F759 arthritis was dependent on CD4⁺T cells, IL-6 and IL-17 and it was enhanced by the T cell leukemia virus 1 p40-Tax, suggesting STAT3 and NF-kB involvement. The F759 mutation in non-hematopoietic cells was required for the arthritis development, indicating interaction between non-immune tissues and the immune system plays a key role in the disease. We showed the IL-6 amplifier driven by STAT3 and NF-kB was the consequences of this interaction and it played a role in F759 arthritis. This scenario may be a general etiologic process underlying other autoimmune diseases and inflammatory diseases.



The clinical impact of cytokine blockade: a ten years perspective of TNF-antagonists



RAVINDER N. MAINI

KENNEDY INSTITUTE OF RHEUMATOLOGY DIVISION, IMPERIAL COLLEGE, LONDON, UK

Beginning with the first in rheumatoid arthritis (RA) clinical trial of a monoclonal anti-TNF antibody in 1992, emerged 3 licensed anti-TNF drugs for the treatment of RA, Crohn's disease, ankylosing spondylitis and psoriasis. In the past decade millions of patients worldwide have been exposed to these drugs. In this presentation the focus is on the impact in the clinic, drug development and healthcare of the success of TNF antagonists.

TNF blockade, especially when used as co-therapy with methotrexate, and introduced early in the course of the disease, achieves effective control of all domains of disease, improved quality of life and reduction in co-morbidity.

This has set a new standard of patient care, with access limited by cost. However, a significant proportion does not respond or lose responsiveness and hence other therapeutic interventions are still required. Safety concerns exist and most can be successfully managed by pre-screening and monitoring, and observational studies on large cohorts of patients have differentiated between drug-related and disease-related complications.

Biological drugs have established themselves as a viable therapeutic option and provide unique tools for the investigation of molecular pathogenesis of disease.



The impact of cytokine blockade for the understanding of the molecular pathogenesis of arthritis

 **MARC FELDMANN**
KENNEDY INSTITUTE OF RHEUMATOLOGY, LONDON, UK

Since the discovery that blockade of a single cytokine, tumour necrosis factor (TNF) yielded significant therapeutic benefit in 1992, there have been attempts to build upon that knowledge. How TNF is regulated in disease tissue has been studied, and atypical T cells are important in synovial TNF production. These have been characterized to seek therapeutic target which may thus interfere just with synovial TNF and not in the rest of the body, a potentially safer approach. The molecular triggers of chronic TNF production in arthritis have been investigated, and toll like receptors (TLRs) are involved, their blockade in synovium and mice with arthritis being beneficial. As macrophages make most of the anti-inflammatory cytokines, pathways of downregulation of macrophage function was investigated and CD200 triggering is beneficial.

In recent years IL-17 has emerged as an important pro-inflammatory cytokine and its role in arthritis studied, chiefly in mice. Anti TNF therapy does not interfere with IL-17 and in mice augments it, suggesting a potential therapeutic combination. There is increasing evidence that patients with rheumatoid arthritis have augmented risk of atherosclerosis, suggesting that these two diseases have pathogenic features in common. The role of TNF and TLRs in atherosclerosis will be compared to that in arthritis. The prospects for the future are bright; a deeper understanding of molecular pathogenesis is revealing many approaches which may improve on today's relatively effective and safe TNF and IL-6 blockade.



Experiences from blockade of IL-1 in arthritis and other inflammatory conditions

**CEM GABAY**

UNIVERSITY HOSPITALS OF GENEVA, GENEVA, SWITZERLAND

Interleukin (IL)-1 exerts prototypical pro-inflammatory effects upon binding to IL-1 receptors. IL-1 bioactivities are tightly regulated at the level of production as well as by several natural inhibitors such as IL-1 receptor antagonist (IL-1Ra), type 2 IL-1R and soluble receptors. The production of mature and bioactive IL-1 β is a 2-step process including the production of inactive pro-IL-1 β , and the activation of the inflammasome, a cytosolic complex of proteins, leading to the cleavage of pro-IL-1 β by caspase-1 and the release of IL-1 β outside the cells. Microbial components and endogenous products can activate the NALP3/cryopyrin inflammasome and induce the

release of mature IL-1 β . Some mutations of the NALP3/cryopyrin gene can also lead to IL-1 β overproduction causing recurrent episodes of inflammatory systemic manifestations. IL-1 inhibitors such as recombinant IL-1Ra, soluble receptors, anti-IL-1 β or anti-IL-1R have been shown to be modestly effective in rheumatoid arthritis. In contrast, some of these IL-1 inhibitors have been reported to be markedly effective in a variety of rheumatic and inflammatory conditions, such as systemic-onset juvenile idiopathic arthritis, adult-onset Still's disease, crystal-induced arthritis, and periodic fever syndromes associated with NALP3/cryopyrin mutations.

YOUNG INVESTIGATOR LECTURE:

HMGB1 – the ugly duckling?

**HELENA ERLANDSSON HARRIS**

KAROLINSKA INSTITUTET, STOCKHOLM, SWEDEN

HMGB1, originally described as a nuclear protein, promotes inflammation when present outside cells. HMGB1 can be passively released by dying cells or secreted by activated leukocytes. Extracellular HMGB1 attracts inflammatory cells, induces cytokine production, promotes stem cell proliferation and activates dendritic cells. The mechanisms underlying the proinflammatory functions of HMGB1 are not fully resolved. Evidence suggests that its actions are regulated by complex formation with proinflammatory molecules, by its redox status and through interplay with at least three receptors including RAGE, TLR-2 or TLR-4.

Excessive extracellular expression of HMGB1 occurs during several inflammatory diseases, including polyarthritis. HMGB1 stimulation induces TNF, IL-1 β , IL-6 and MMP production in synovial cells, and intra-articular HMGB1 injections result in synovitis. Therapies based on HMGB1-specific antagonists have beneficial effects in arthritis models. Thus the constitutive nuclear expression of HMGB1, together with its ability to be rapidly released upon stress, indicate that HMGB1 represents a potential therapeutic option distinct from the available cytokine-targeting therapies.



Crafoord Prize lectures

Open to the public

Wednesday 13 MAY

FERNSTRÖMSALEN, BMC, LUND UNIVERSITY
SÖLVEGATAN 19, LUND

13:30	<i>Welcome and practical information</i>	Moderator: <i>Johan Karlsson</i>
13:40	<i>Welcome to the Crafoord Days</i>	<i>Gunnar Öquist</i> , Permanent Secretary, the Royal Swedish Academy of Sciences
13:45	<i>Introduction of the Crafoord Laureates in Polyarthritis 2009:</i> CHARLES A. DINARELLO TADAMITSU KISHIMOTO <i>and TOSHIO HIRANO</i>	<i>Catharina Svanborg</i> , Chairman of the Crafoord Committee in Medicine
14:00	<i>Crafoord Prize lecture</i> From the Study of Fever to the Treatment of Fever Syndromes	CHARLES A. DINARELLO
14:30	<i>Crafoord Prize lecture</i> Interleukin 6: from bench to bedside	TADAMITSU KISHIMOTO
15:00	Break with refreshments	
15:30	<i>Crafoord Prize lecture</i> How extensive a view from the top of the mountain is!	TOSHIO HIRANO
16:00	The audience's questions to the Laureates	
16:45	End	

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.



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 the University of Lund.



HOLGER AND ANNA-GRETA CRAFOORD

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 the University of Lund.

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 is an independent, non-governmental organisation whose aim is to promote the sciences and strengthen their influence in society. Traditionally, the Academy takes a special responsibility for the natural sciences and mathematics, but in its work it strives to increase exchanges between different disciplines.

The activities of the Academy are aimed mainly at:

- *spreading knowledge of discoveries and problems in current research*
- *providing support for young researchers*
- *rewarding outstanding contributions in research*
- *stimulating interest in mathematics and the natural sciences in schools*
- *spreading scientific and popular-scientific information in various forms*
- *offering unique research environments*
- *maintaining contact with foreign academies, learned societies and other international scientific organisations*
- *representing the sciences in society*
- *carrying out independent analyses and evaluations, based on scientific grounds, of issues of importance for society*

The Academy has about 400 Swedish members and 175 foreign members. The Swedish members are active within Classes and Committees. They initiate investigations, responses to government proposals, conferences and seminars. Once a month the Academy holds a General Meeting and in connection with this a public lecture. (Visit www.kva.se for the programme.) The Academy's own institutes offer unique research environments for climate research, botany, ecological economics, the history of science, astrophysics, mathematics and other subjects. Besides the prominent Crafoord Prize, the Academy awards annually a number of prizes, the best known of which are the Nobel Prizes in Physics and Chemistry and the Sveriges Riksbank Prize in Economic Sciences in Memory of Alfred Nobel. Other important prizes are the Söderberg Prize and the Göran Gustafsson Prizes. The latter are awarded to outstanding young researchers and are a unique combination of a personal prize and a research grant. The Academy also supports researchers who have been researching actively for five to ten years after taking their doctorate by providing a salary for five years through the support of external foundations. Through its various Committees the Academy also works for the development of a society based on scientific grounds. Great interest is paid to educational issues and a major school development program, NTA (Natural Sciences and Technology for All), is organised in collaboration with the Royal Swedish Academy of Engineering Sciences.



**KUNGL.
VETENSKAPS-
AKADEMIEN**

THE ROYAL SWEDISH ACADEMY OF SCIENCES