Instituto Juan March
de Estudios e Investigaciones

147 CENTRO DE REUNIONES
INTERNACIONALES SOBRE BIOLOGÍA

2002
Annual Report

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Headquarters of the Fundación Juan March
(Home of the Centre for International Meetings on Biology)

The highlights of tomorrow are the unpredictabilities of today.

(César Milstein, Nobel lecture, 1984)

INSTITUTO JUAN MARCH DE ESTUDIOS E INVESTIGACIONES

CENTRE FOR INTERNATIONAL MEETINGS ON BIOLOGY

2002 ANNUAL REPORT

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FOREWORD

This publication covers the activities of the Centre for International Meetings on Biology during the year 2002. All of them were, in due time, broadly announced by means of brochures, posters, advertisements in scientific journals and other periodicals, and are also described in detail in the Internet page www.march.es/biology.

The core of the Centre's work during 2002 was the organization of fourteen workshops, dealing with very different biological topics. An introduction to each of these meetings is presented here, followed by a list of invited speakers and participants selected from among the applications received. In total, 278 speakers were invited to the meetings during this year, and 429 participants were chosen from among 676 applications received.

14 booklets were published on these meetings, including the abstracts of the contributions presented by the participating scientists. About 450 copies of each booklet were distributed to research groups and laboratories working on problems relating to the subject of each meeting.

A Grant for Basic Research was established by the Fundación Juan March in 2000. It has been awarded for the third consecutive year in 2002, as described in the following pages.

A short notice is given on reviews published during 2002 in scientific journals regarding meetings organized by the Centre.

The schedule of meetings to take place in 2003 is also offered in this report.

Instituto Juan March de Estudios e Investigaciones

THE CENTRE FOR INTERNATIONAL MEETINGS ON BIOLOGY

The Centre for International Meetings on Biology endeavours actively and systematically to promote close cooperation and interaction among Spanish and foreign scientists working in the field of Biology. This scientific field is understood in the widest sense, and emphasis is given to advanced lines of research.

The Centre's activities stem from the Plan for International Meetings on Biology, initiated by the **Fundación Juan March** in January 1989 and ending in December 1991. A wide range of meetings and scientific activities were organized under this Plan. The Fundación Juan March, in addition to its well-known support of the fine arts and culture in general, has devoted particular attention to the biological sciences since its creation in 1955 by the Spanish financier Juan March Ordinas.

The Centre for International Meetings on Biology was established in January 1992 within the **Instituto Juan March de Estudios e Investigaciones**, a private foundation created in October 1986 and recognized by the Spanish Ministry of Education and Culture. This foundation complements the work of the Fundación Juan March, as an entity specializing in scientific activities. The Board of Trustees of the Instituto comprised: Juan March (Chairman), Carlos March (Deputy Chairman), Leonor March, Alfredo Lafita, Antonio Rodríguez Robles, Pablo Vallbona, Enrique Piñel and Jaime Prohens (deceased on December 31st, 2002).

Javier Gomá is the Secretary and José Luis Yuste is Managing Director of the Institute.

The Centre for International Meetings on Biology is located at Calle Castelló 77, Madrid.

SCIENTIFIC COUNCIL AND MANAGEMENT OF THE CENTRE

During the year 2002 the Scientific Council of the Centre comprised the following members:

† **César Milstein**
Medical Research Council
Cambridge (United Kingdom)
(Deceased on March 24th, 2002)

Ginés Morata
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CSIC - Universidad Autónoma de Madrid (Spain)

Erwin Neher
Max-Planck-Institut für Biophysikalische Chemie
Göttingen (Germany)

Margarita Salas
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Ramón Serrano
Instituto de Biología Molecular y Celular de Plantas.
CSIC – Universidad Politécnica de Valencia (Spain)

Sir John E. Walker
Medical Research Council
Cambridge (United Kingdom)

The Scientific Council determines the priorities for the Centre's activities. It may put forward initiatives to be carried out in collaboration with Spanish or foreign laboratories. It will also consider proposals of meetings submitted to the Centre by Spanish or foreign scientists, selecting and approving those it feels deserve support.

In general terms, the Scientific Council advises the Centre for International Meetings on Biology on any scientific subject or issue falling within the scope of the Centre's activities.

The Director of the Centre is **Andrés González**.

MARCH GRANT FOR BASIC RESEARCH

The Fundación Juan March decided in 2000 to award each year a Grant of 901.518 euros (150 million pesetas) to support the work of a Spanish scientist, aged under 50, carrying out original and creative research in Spain. The scientific field selected in principle to receive this award is Biology, thereby maintaining the support given to this science by the Foundation since its creation in 1955. The Grant will be paid out over a period of between 3 and 5 years, in accordance with the needs of the recipient scientist.

The Scientific Council of the Centre for International Meetings on Biology will submit a proposal for the annual award of this grant to the Fundación Juan March.

This Grant for basic research will be awarded without prior submission of proposals. It is neither a prize nor an expression of recognition for a lifetime's achievement, but a means of supporting the work of a scientist leading a team of high-level scientific production, carrying out top quality research and with promising prospects. The March Grant will be incompatible with any other major grants from private institutions, it cannot be prolonged and will not include additional allowances to the scientist's host institution. At the end of the Grant a final report of the research will be published.

The Grant was awarded for the first time in 2000 to Prof. José López-Barneo (School of Medicine, University of Seville, Spain) and in 2001 to Dr. Jorge Moscat (Centro de Biología Molecular "Severo Ochoa", Madrid, Spain). In November 2002 it was awarded for the third time to Prof. Francisco Sánchez-Madrid (Universidad Autónoma de Madrid and Immunology Department of the Hospital de la Princesa, Madrid, Spain).

The terms of this Grant are as follows:

1. Aim. To support the work of a Spanish scientist, aged under 50, carrying out original and creative research in Spain. It is neither a prize nor an expression of recognition for a lifetime's achievement, but a financial award to be used on basic research.

2. Area. The scientific field initially selected is Biology, thereby maintaining the support that the Fundación Juan March has given to research in this area for over 25 years.

3. Endowment. A single Grant of 901.518 euros (150 million pesetas) will be awarded every year.

4. Selection Committee. The Grant for basic research will be awarded without prior submission of proposals. The award will be made on the basis of the recommendation of a Selection Committee chaired by the Managing Director of the Fundación Juan March.

5. Payment. The Grant will be paid out over a period of between 3 and 5 years, in accordance with the needs of the recipient scientist and will be subject to current tax laws.

6. Incompatibilities. The March Grant will be incompatible with any other major grant from a private institution. Its compatibility with any other grant, public or private, Spanish or foreign, shall be decided in consultation with the Fundación Juan March.

7. Use of the Grant. The recipient scientist will use the Grant to advance his/her research in accordance with his/her own criteria. Only the amounts devoted to personnel costs must be agreed with the Foundation beforehand. The Grant cannot be extended and will not include additional allowances for the scientist's host institution.

8. Obligations. The selected scientist will provide the Foundation with a summary of the work to be undertaken during the period covered by the Grant. Expenses charged to the Grant shall be justified to the Foundation once a year, and sent together with a brief report on the results achieved and a list of the scientific papers published during that period. At the end of the Grant, a final report will be submitted and may be published. The Fundación Juan March retains the right to withdraw the Grant on justifiable grounds.

2002 Meetings Schedule

CENTRE FOR INTERNATIONAL MEETINGS ON BIOLOGY
2002 MEETINGS SCHEDULE

Date	Meeting Subject	Organizers
28-30 January	**Stress in Yeast Cell Biology... and Beyond**	**J. Ariño.** Universidad Autónoma. Barcelona.
11-13 February	**Leaf Development**	**S. Hake.** Plant Gene Expresión Center. Albany. **J.L. Micol.** Universidad Miguel Hernández. Alicante.
25-27 February	**Molecular Mechanisms of Immune Modulation: Lessons from Viruses**	**A. Alcami.** Cambridge University. Cambridge. **U.H. Koszinowski.** Max von Pettenkofer-Institut. Munich. **M. del Val.** Instituto de Salud Carlos III. Madrid.
11-13 March	**Channelopathies**	**T.J. Jentsch.** Zentrum für Molekulare Neurobiologie. Hamburg. **A. Ferrer-Montiel.** Universidad Miguel Hernández. Alicante. **J. Lerma.** Instituto Cajal. Madrid.
8-10 April	**Limb Development**	**D. Duboule.** University of Geneva. **M. A. Ros.** Universidad de Cantabria. Santander.
22-24 April	**Regulation of Eukaryotic Genes in their Natural Chromatin Context**	**K.S. Zaret.** Fox Chase Cancer Center. Philadelphia **M. Beato.** Centro de Regulación Genómica. Barcelona
20-22 May	**Lipid Signalling: Cellular Events and their Biophysical Mechanisms**	**E.A. Dennis.** University of California. San Diego. **I. Varela-Nieto.** Instituto de Investigaciones Biomédicas. Madrid **A. Alonso.** Universidad del País Vasco. Bilbao.
3-5 June	**Regulation and Functional Insights in Cellular Polarity**	**A.R. Horwitz.** University of Virginia. **F. Sánchez-Madrid.** Hospital de la Princesa. Madrid.
17-19 June	**The Structure of the Cortical Microcircuit**	**R. Yuste.** Columbia University. New York. **E.M. Callaway.** Salk Institute. La Jolla. **H. Markram.** Weizmann Institute. Rehovot.
7-9 October	**Control of NF-κB Signal Transduction in Inflammation and Innate Immununity**	**M. Karin.** University of California. San Diego. **I. M. Verma.** Salk Institute. La Jolla. **J. Moscat.** Centro de Biología Molecular "Severo Ochoa". Madrid.
21-23 October	**Engineering RNA Virus Genomes as Biosafe Vectors**	**C.M. Rice.** The Rockefeller University. New York. **W.J.M. Spaan.** Leiden University. Leiden. **L. Enjuanes.** Centro Nacional de Biotecnología. Madrid.
4-6 November	**Exchange Factors**	**X.R. Bustelo.** Universidad de Salamanca. **J.S. Gutkind.** National Institutes of Health. Bethesda. **P. Crespo.** Instituto de Investigaciones Biomédicas. Madrid.
18-20 November	**The Ubiquitin-Proteasome System**	**A. Ciechanover.** Technion-Israel Institute of Technology. Haifa. **D. Finley.** Harvard Medical School. Boston. **T. Sommer.** Max-Delbruck-Center for Molecular Medicine. Berlin. **C. Mezquita.** Universidad de Barcelona.
16-18 December	**Manufacturing Bacteria: Design, Production And Assembly of Cell Division Components**	**P. de Boer.** Case Western Reserve University. Cleveland. **J. Errington.** Sir William Dunn School of Pathology. University of Oxford. **M. Vicente.** Centro Nacional de Biotecnología. Madrid.

Stress in Yeast Cell Biology ... and Beyond

Organized by
J. Ariño

(28-30 January)

Living cells must adapt to changes in the environment to survive. In many cases, these changes compromise cell growth or even cell survival, and adaptation requires a fast and complex functional response. By analyzing at the molecular level the characteristics of this response, we can learn about many aspects of the cell biology. Stress responses are particularly relevant in microorganisms, because conditions in their environment are far from constant and they have to face sudden changes in temperature, nutrient availability, osmolarity or exposure to toxic ions. Because of their accessibility to very powerful genetic tools, yeasts (particularly *S. cerevisiae*) represent a very useful model to study stress response mechanisms that are often conserved in more complex organisms.

The aim of this workshop was to analyze, from different points of view, the most recent findings on the response of yeasts to stress, and to identify relevant aspects that could serve as starting points for research in plants and animals. Many different types of stress were examined, such as osmotic and saline, oxidative, temperature, pH, nutritional... It has been pointed out how yeast cells integrate different stress signals to provide a general response. The initial step requires sensing of the stress condition. The sensing machinery has been elucidated in some cases, such as osmotic stress, but the problem still remains unsolved in many others. Transduction of the stress signal often involves elements that are fully conserved among eukaryotes: heat shock factors, cAMP-dependent protein kinase (PKA) or MAP kinases modules. A general feeling among the participants was that more examples of conserved mechanisms should be expected to emerge within the next few years. Finally, the response generates in many cases changes in gene transcription, that have been thoroughly examined by DNA microchip analysis in the last few years, although other mechanisms such as changes in RNA stability or even translational control cannot be excluded.

Yeasts are not only important as a research model, but are also key elements in food production and biotechnology (clearly the case of *S. cerevisiae*). These aspects were not omitted in the workshop, as they were addressed by different talks and poster presentations, pointing out how better understanding of yeast stress response can not only help us to understand how living cells function, but also lead to improvements in very old (and often pleasant) processes, such as wine making.

Joaquín Ariño

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Leaf Development

Organized by
S. Hake and J.L. Micol

(11-13 February)

Questions and answers about the development of plant leaves

The primary pathway for carbon and energy uptake by plants is the leaf, an organ of utmost importance in agriculture. However, little is known about the genetic controls underlying leaf development, in spite of the fact that its biotechnological manipulation offers great potential. Although Bateson realized the existence of inherited leaf shape variants as early as 1913, genetic investigations did not provide major insights into the dissection of leaf initiation and morphogenesis until the last decades of the XX century, when a large number of mutants with abnormally shaped leaves, most of them yet to be characterized, were isolated in model systems such as *Arabidopsis thaliana*, *Antirrhinum majus* and *Zea mays*.

Although most plant leaves are simple structures, many developmental processes are involved in leaf ontogeny. They include, among others, the positioning and initiation of leaf primordia at the flanks of the shoot meristem, the specification of leaf identity as opposed to that of other organs which are assumed to be modified leaves, the establishment of dorsal and ventral identities within the organ, the definition of domains such as ligule, sheath and blade in some monocotyledonous plants, as well as petiole and lamina in dicots, the control of cell division and expansion, the formation of patterns such as those of venation, trichomes or stomata, the mechanisms responsible for the diversity of compound and simple leaves and those that specify heteroblastic differences among different leaves within a plant. A large body of detailed information on what actually happens at a morphological level is available for most, if not all such processes. At the present time, an expanding number of studies on leaf variants including molecular and genetic analyses are being published for several plant species. Thanks to these efforts, answers are beginning to be available to the questions on the nature, action and interactions of the genes driving the sequence of developmental events that contribute to the making of a leaf.

We discussed at this workshop recent progress in the study of genetic mechanisms that control the elaboration of plant leaves. The topics covered were the specification of leaf identity, the definition of axes and polarities in the formation and growth of leaves and different aspects of cell differentiation, pattern formation, phase change and heteroblasty. Answers to many of the above mentioned fundamental questions are coming from the study of specific genes, thanks to the enormous progress that has been made in recent years in the area of leaf development.

José Luis Micol and Sarah Hake

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Molecular Mechanisms of Immune Modulation: Lessons from Viruses

Organized by
A. Alcamí, U.H. Koszinowski and M. Del Val

(25-27 February)

Viruses have evolved strategies to evade the powerful host inflammatory and immune responses that can eliminate them. This was the subject of a previous Juan March Institute workshop on 'Viral evasion of host defense mechanisms' organised by M. B. Mathews and M. Esteban in September 1993 (booklet n° 19). The number of viral immune evasion strategies identified has increased dramatically in recent years. Large DNA viruses (herpesviruses and poxviruses) have the capacity to encode many proteins that mimic or target specific components of the host immune system, such as homologues of cytokines and chemokines, and their receptors, or proteins that target specific components of the antigen presentation pathways and prevent immune recognition. The function of these viral molecules is to evade immune responses or to promote viral replication, and they may also contribute to pathology. We have just started to understand the immune evasion strategies at the molecular level, and the list of these strategies forms the 'Who is who' of today's immunology.

This workshop brought together scientists with different views of the interaction of viruses with host defence mechanisms. The aim of the meeting was to discuss strategies of immune modulation encoded by viruses, and focused on large DNA viruses (herpesviruses and poxviruses) and other viruses such as human immunodeficiency virus (HIV)., A number of models for viral pathogenesis were discussed since these are helping us to understand the function of different arms of the immune system in anti-viral defence *in vivo*. The use of animal models of viral infection is starting to uncover the physiological role of viral immunomodulatory molecules and is an area of research that is becoming very active.

The emphasis of the meeting was to discuss how these studies can teach us about the host immune system. These viral proteins have been optimized for millions of years of evolution as effective immunomodulatory molecules and some of them have sequence similarity to human proteins. We can now use the information found in viral genomes to uncover new components of the immune system, to understand cellular processes involved in protein trafficking and antigen presentation, and to identify novel mechanisms of immune modulation. These findings will help us to treat virus-induced pathology and to design safer and more immunogenic vaccines, and will lead to new strategies of therapeutic intervention in human diseases that are caused by an over-reactive immune and inflammatory response.

Antonio Alcami, Ulrich H. Koszinowski and Margarita Del Val

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Channelopathies

Organized by
T.J. Jentsch, A. Ferrer-Montiel and J. Lerma

(11-13 March)

Ion channels play a critical role in the physiology of the nervous system. These molecules are fundamental in the generation of membrane potentials that are the essence of neuronal signalling. Although initially thought to be specific signalling molecules of the nervous system, it is known since long that ion channels are also present on non-neuronal cells such as lymphocytes or sperm cells where they play fundamental roles in cellular proliferation and differentiation. Thus, ion channels are widely distributed in neuronal and non-neuronal tissues. Furthermore, they are present in the plasma membrane, as well as in the membrane of intracellular organelles. The importance of these molecular devices in physiology and pathology is further highlighted by the discovery that they are the targets of a large number of toxins and drugs.

Ion channels are pores in the cell membranes that allow the flow of ions across the lipid bilayer, causing eventually a depolarization or hyperpolarization of the cell. These devices conduct ions down their electrochemical gradient at extremely rapid rates of up to 100,000,000 ions per second. This high permeation rate is often accompanied by an exquisite selectivity to one or more ions. In addition to these two properties, ion channels are controlled by gating mechanisms which involve a conformational change of the protein in response to specific stimuli. Gated channels can open or close rapidly in response to different signals, including voltage, chemical transmitters, heat, and pressure or stretch. It should be noted that non-gated channels also exist and significantly contribute to define the resting potential, as well as to cell-cell communication. Therefore, ion channel are a heterogeneous family of proteins that can be classified attending to their gating mechanism and/or ionic selectivity.

Structurally, ion channels are large integral membrane proteins that have a central pore that spans the entire width of the plasma membrane. They are most often oligomeric proteins formed by identical or different subunits organized around a central axis of symmetry. Channel subunits may be encoded by a single gene (homo-oligomers) or by different genes (hetero-oligomers). This molecular complexity is further expanded by the existence of accessory subunits essential for channel function. The progress of molecular biology along with electrophysiology has allowed the precise characterization of ion channel activity in terms of their underlying protein structure. Recently, the high-resolution X-ray crystallographic structure for a K^+ and a Cl^- channels has been reported, providing information on channel permeation and selectivity at an unprecedented detail.

A central question arises: What happens when ion channels do not work properly? Recent advances in medical genetics, biochemistry, molecular biology and electrophysiology have contributed to unravel the central role played by ion channel dysfunction in several human pathologies. Indeed, malfunction of these molecular machines may result in a variety of neurological and non-neurological disorders that span from myopathies to epilepsy and even bone disease. All these disorders of channel function are called channelopathies. These disorders may be caused by genetic alterations or by toxins or by autoimmunity. Inherited channelopathies may be due to gain-of-function mutations, or loss of function which in some cases requires a dominant negative behaviour of a mutated subunit. A characteristic of some channelopathies is the intermittent or episodic nature of the symptoms, presumably because

significant mutations may have profound consequences in channel function leading to a lethal phenotype.

The increasingly known incidence of channelopathies in humans, along with the emergence of new information on the molecular and cellular causes underlying disease phenotypes, prompted the organization of this specific workshop under the auspices of the Fundación Juan March. The aim of the workshop was to provide an update of our current knowledge on genetic and autoimmune channelopathies. The meeting was organized in specific sessions that covered most aspects related to channel dysfunction and its relationship with human or animal disorders. The session 1 discussed the consequences of glutamate receptor channel dysfunction. The session 2 focused on calcium channels that have been critically involved in diverse human diseases. A discussion on malfunction on excitation (potassium channels and acetylcholine receptors) and inhibition (glycine-gated channels) ensued in session 3. An interesting session 4 was dedicated to describe involvement of non-neuronal channels in the aetiology of an increasing number of disorders including cystic fibrosis, hypertension, Dent's disease and cancer. And last, but not least, in session 5 the underlying mutations and functional alterations underlying connexinopathies were debated. In addition, the role of GABAergic interneurones in network synchrony and oscillatory activity in the brain, and the involvement of vanilloid receptor 1 (TRPV1) in inflammatory pain was presented. An outcome of this meeting was the realization that ion channels are not simple molecular devices that allow the flux of ions. An emerging concept was that these proteins are also important components of supramolecular complexes revealed as essential for cellular signalling.

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Limb Development

Organized by
D. Duboule and M.A. Ros

(8-10 April)

Co-sponsored by

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Investigation in recent years has led to a tremendous advance in our knowledge of the mechanisms and molecular basis underlying development. Quite surprisingly, these studies have also shown that evolutionary distant organisms (e.g. insects and mammals) use similar molecules and genetic interactions to outline pattern. These findings have challenged classical concepts such as analogy and homology and have brought the disciplines of Development and Evolution closer than ever.

Traditionally, the development of the appendages has provided a fruitful system to analyze multiple paradigmatic questions of Developmental Biology. For example, the study of the wing and leg imaginal disk of *Drosophila* has come a long way in identifying the genetic cascades controlling patterning in each axis of the primordium. The studies in vertebrates have benefited from the knowledge obtained in *Drosophila* obtaining a rapid advancement and demonstrating a high degree of conservation not only of the genes but also of their genetic interactions.

This workshop on Limb Development provided an excellent opportunity to bring together and compare different experimental, genetic and molecular approaches in vertebrates and insects. Emphasis was placed in analyzing the establishment of the dorsoventral axis in the developing *Drosophila* wing and whether it relays on a morphogen gradient from the organizer or involves modulatory short-range interactions. The available experimental data were interpreted and discussed accordingly to both models. Latest findings in wingless signaling, at both intra and extracellular levels, were also presented. Further talks presented recent advances in the regulation and function of different gene expressions implicated in *Drosophila* appendage development.

In vertebrates the "progress zone" model has officiated our understanding of the development of the proximodistal axis in the past thirty years. An alternative model is now put forward and all the support in favor and against each model was vividly exposed. Data from mouse knockouts and mutations in several species attracted much attention and challenged the common way in which signaling pathways such as Fgf and Shh, or factors such as Homeobox genes are presently interpreted.

The evolutionary implications of the different issues addressed in the workshop were treated throughout the whole meeting, particularly in the final session. The similarities and differences in developmental mechanisms and gene expressions in different organisms were explored and discussed.

In sum, this workshop on Limb Development was remarkably timely. We believe that the insights gained will have great implications for the scientific community in the field and we look forward seen how the new thoughts and arguments will evolve in the near future.

Marian Ros and Denis Duboule

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Regulation of Eukaryotic Genes in their Natural Chromatin Context

Organized by
K. S. Zaret and M. Beato

(22-24 April)

Long strands of eukaryotic DNA are packaged into the nucleus of the cell by DNA wrapping around octamers of histone proteins, creating nucleosome core particles, and by further packaging of the nucleosome cores into chromatin. The organization of eukaryotic DNA in chromatin influences all processes requiring access to genetic information, including gene transcription, DNA replication, repair, and recombination. These processes are critical for normal cellular physiology and are frequently perturbed in disease states. The significance of chromatin structure for the regulation of gene expression is underscored by the discovery that numerous transcription factor co-regulators, as well as proteins that control DNA metabolism, modify the packaging of DNA in chromatin. Many of these co-regulators, and protein complexes thereof, modify chromatin packaging either by covalently altering the histone proteins or by ATP-dependent, nucleosome remodeling. Furthermore, proteins that modulate DNA-dependent functions are often sensitive to histone modification states. Relevant histone modifications include phosphorylation, acetylation, methylation, and ubiquitylation, each of which are associated with stable states of repression or activation, or the transition from one state to another. The biochemistry of these transcriptional co-regulatory complexes has been studied intensively, but their biological significance in the regulation of individual genes is poorly understood. This workshop focused on the biological context and cellular signficance of chromatin in gene regulation and other DNA-dependent functions.

To date, most studies in this field have focused on the potential role of transcriptional regulatory complexes in artificial contexts, rather than with physiological, regulatory pathways and native DNA sequence contexts. This distinction in approach is understandable, because assessing the biochemical parameters of regulatory proteins in highly controlled reactions is critical to define the proteins' mechanisms of action. On the other hand, evidence is emerging that in some cases, the artificial DNA sequence arrangements and templates studied *in vitro* are inaccurate models of the *in vivo* situation. For example, nucleosomes can be positioned precisely on certain artificial DNA sequences *in vitro*, but the same DNA sequences can exclude a nucleosome when introduced into native chromatin in the cell. Results such as these emphasize the need to understand how DNA sequences function in a native chromatin context. In addition, some parameters of native chromatin context, such as nuclear localization, cannot yet be reconstituted *in vitro*, yet are emerging as a significant influence on gene activity.

Recent technical developments, such as chromatin immunoprecipitation (ChIP) and high resolution *in vivo* genomic analysis, have opened new possibilities for addressing the role of chromatin structure and its modifications in the regulation of genes in their native sequence context in the cell. Results are beginning to accumulate showing that transcription factors and coregulator complexes may strongly depend on the precise organization of the target DNA sequences in nucleosomes and in higher order chromatin structures. In some systems, the packaging of DNA in cellular chromatin determines the nature of the interaction of regulatory proteins with their cognate *cis* regulatory elements and, thus, the outcome of the transcriptional response. Thus, detailed studies that accurately determine native chromatin structures in cells provide a framework for *in vitro* reconstitution of more native-like structures, for subsequent mechanistic analysis.

The aim of this workshop was different from those of previous chromatin meetings. Our intention was to compare the results obtained by groups working with various natural genes in their native chromatin environment, in order to get a more realistic picture of the actual mechanism used by regulatory protein complexes to fulfill their physiological function. To this end, we included genetic and biochemical talks by 25 leading scientists, combined in 5 sessions covering different levels of complexity. The biological systems were from *S. cerevisiae, Drosophila, Tetrahymena*, the mouse and human, and mammalian viruses. Both *in vivo* and *in vitro* systems were described, with the latter based on chromatin structures observed *in vivo*. There was excellent discussion at the meeting, which helped to shed new light on the crucial aspects of chromatin function in physiologically relevant systems.

Miguel Beato and Ken S. Zaret

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Lipid Signalling: Cellular Events and their Biophysical Mechanisms

Organized by
E.A. Dennis, I. Varela-Nieto and A. Alonso

(20-22 May)

The last decade has witnessed the discovery of a large number of lipid molecules that play specific roles in cell signaling. Ceramide, sphingosine phosphate, lysophosphatidic acid and others have joined the previously known phosphatidylinositol derivatives, diglyceride, and phosphatidic acid in their roles as metabolic regulators. Thus, phospholipids and sphingolipids, in addition to their structural involvement in membranes, are now viewed as important reservoirs of lipid second messengers.

Current research in this field includes studies in at least three areas, namely cell biology, biochemistry and biophysics, sometimes without the desirable degree of interaction between them. From the point of view of cell physiology, lipid signals have been found to mediate an amazing variety of processes, from cell activation to apoptosis, including ion channel regulation, intracellular membrane trafficking and membrane adhesion. Simultaneously, metabolic studies of these substances have led to significant advances in understanding the mechanism and regulation of biosynthetic and degradative enzymes, notably phospholipases A2, C and D, sphingomyelinases and CTP:phosphocholine cytydyltransferase. Finally, recent biophysical studies have led to important discoveries in the structure of some of the involved enzymes (e.g. PIP3 kinase, PI-phospholipase C), in the behaviour of lipid signals in bilayers, (e.g. ceramide segregation into domains and rafts), or in the regulation of important enzymes through membrane physical properties (e.g. PI-specific phospholipase C), that may provide the molecular foundation for an understanding of critical lipid-mediated cellular processes.

Moreover the genomics revolution has been impressive and innovative for the biological sciences and as difficult as it was, it has only had to deal with a finite number of genes, estimated to range from 30,000-50,000 for humans. The proteomics revolution is upon us, but the number of discrete proteins is enormous and certainly not finite. Proteins come in many forms; they are acylated, acetylated, phosphorylated, and ubiquinated and exist as preproteins and proproteins, and they can be altered in subtle manners by interaction with other proteins. The metabolomics revolution is next, but the number of distinct metabolites is astronomical. Even if one just thinks of the lipid metabolites, the number of unique structures is difficult to fathom. It is clear that the next decade will enlighten us with numerous novel new lipids and diverse new functions for them. Certainly the last decade moved the interest in lipids from just their traditional roles in energy storage and membrane structure to a central role in all of cell signaling.

Our workshop focused on LIPIDS, both their biophysical and cellular aspects. Our hope was to share the parallel evolution of biophysical approaches to understanding the physical parameters of lipids and the structural parameters of the proteins that make or interact with them. Then, we hoped to integrate this information with our evolving knowledge of the cellular and physiological actions of a variety of lipids as they interact with cellular proteins and with other lipid assemblies. During the course of the workshop, we considered a vast array of proteins that degrade lipids, that transfer lipids, and that synthesize lipids and the cellular responses of activation, proliferation, differentiation, inflammation, and apoptosis. We heard the latest results from both the biophysical and the cellular directions and most importantly, the interplay of both. The meeting succeeded in bringing together lipidologists

working in the often-separate sphingolipid and phospholipid fields and there was much new information to exchange. We all greatly benefited from excellent discussions that were fostered by the intimate atmosphere provided by the Juan March Foundation venue.

Edward Dennis, Isabel Varela and Alicia Alonso

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Regulation and Functional Insights in Cellular Polarity

Organized by
A.R. Horwitz and F. Sánchez-Madrid

(3-5 June)

Cell polarization and the establishment of functionally specialized domains plays a pivotal role in many cellular proceses such as vectorial transport of molecules, cell division and differentiation, migration and directional movement of the cells in a chemotactic gradient and activation of the immune response. Polarization may be constitutive, such as in neurons or epithelium, or inducible, such as in mating or budding yeast or during cell migration. This is a complex phenomenon in which the interplay among cell cytoskeletal components, extra- and intracellular signals and organelle and membrane reorganization is crucial to achieve a correct cell shape change.

The aim of this meeting was to compare recent advances in different fields related to cell polarization. Research in yeast provided a basic tool in which to dissect genetics of molecules involved in the generation of specific compartments within the cell and other cycle-related phenomena, such as budding or pheromone mating as well as fission. On the other hand, some of the discussion involved the polarized secretion of molecules and its impact in the development and maintenance of cell polarity, with new data implicating the role of the actin cytoskeleton and the machinery involved in vesicle formation and trafficking. The role of membrane composition and dynamics was also described, with special attention to the role of lipid rafts in the generation of cell compartments and the spatial regulatory role of rafts in activation of signaling components depending on the presence of different molecules in this lipid domains. Cell migration is an active field of study in which the development of polarity is a requisite for cell movement. Recent advances in genes controlling cell adhesion and migration in different cell models, such as *Dictyostelium*, fibroblasts, neutrophils or lymphocytes were reviewed. Finally, the establishment of cognate immune cell-cell interactions and the regulatory molecules was discussed.

Alan R. Horwitz and Francisco Sánchez-Madrid

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The Structure of the Cortical Microcircuit

Organized by
R. Yuste, E. M. Callaway and H. Markram

(17-19 June)

As frequently stated by Ramon y Cajal, understanding how the cortex works is arguably the most important problem in Neuroscience and one of the major challenges of modern Biology. Although some investigators argue that the function of the cortex could be understood without knowing its "wiring diagram", a sufficiently deep understanding of cortical function seems impossible without understanding the structure and basic function of the cortical microcircuit. In spite of over a century of research, the precise microcircuitry of the cerebral cortex, defined as the inter- and intralaminar connections found in any given cortical area, is still basically unknown. Indeed, it is still not even clear how many different classes of neurons exist in the cortical circuits. Widely different views coexist currently among researchers interested in cortical circuits. Some investigators argue that most cortical connections are essentially random, and the cortex is a gigantic neural network whose purpose is to shuffle and mix information in order to enable associations among any possible stimuli. Other investigators argue that the cortex is an ancient machine composed of scores of extremely precise circuits, each implementing a particular function.

While most investigators of cortical function study correlations between the activity of cortical neurons and behavioral or receptive field processing *in vivo*, there is a host of laboratories around the world whose main goal is to describe and reconstruct cortical microcircuits. These laboratories have traditionally used anatomical techniques. In the last decade, however, many new approaches have been introduced, such as dual recordings from synaptically connected neurons in slices, computerized photostimulation of specific cortical locations, optical probing of connectivity, multivariate analysis of morphological data and expression analysis of dozens of different genetic markers. These new techniques are providing exciting new information and have the potential to revolutionize the study of cortical circuits.

This workshop brought together representatives from these novel lines of work with structural researchers that represented and encompassed more traditional approaches. Our goal was to enable the extensive and, at the same time, relaxed, exchange of detailed information in order to facilitate cross-fertilization among a worldwide team of experts in different aspects of cortical microcircuitry. Such a cross fertilization appears necessary to formulate a comprehensive theory of microcircuit function. The workshop did not include the large area of *in vivo* cortical physiology, but remained focused on the architectural principles of the neocortical microcircuit. The meeting was organized into five sessions around three interrelated topics: (1) Excitation, (2) Inhibition and (3) Computation. The recurrent themes, highlighted in many of the presentations and in the discussions that followed, were the description of different cellular types of cortical neurons, particularly among the interneurons, as well as the characterization of what appear to be very precise connections. The more ambitious goal of linking the specific circuits to particular computations was also touched upon by several presentations, although at the same time, recognized as premature at this stage. The overall sense of most participants was one of excitement, since the effort to decipher cortical circuits appears not only fruitful as an experimental program in itself, but also increasingly related to the understanding of the "heart" of the neocortical computation.

Rafa Yuste, Ed Callaway and Henry Markram

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Control of NF-κB Signal Transduction in Inflammation and Innate Immunity

Organized by
M. Karin, I.M. Verma and J. Moscat

(7-9 October)

The mechanisms controlling NF-κB signaling constitute a paradigm of the membrane-nucleus cell communication. NF-κB is central to a number of cellular functions including cell proliferation, apoptosis and cellular differentiation. Also this transcription factor plays essential roles in innate and acquired immunity and its deregulation leads to important human diseases such as inflammation and cancer. NF-κB is formed by members of the Rel family of transcription factors, and are retained in the cytosol by the inhibitory molecule IκB. Upon activation by inflammatory cytokines, IκB is phosphorylated, ubiquitinated and degraded by the proteasome system which leads to the release and nuclear translocation of the classical RelA/p50 NF-κB. A complex formed by IKKβ and IKKγ is responsible for the phosphorylation of IκB in response to TNFα and IL-1. Genetic evidence in mice demonstrates the essentiality of these different components of the classic paradigmatic pathway. However, parallel to this cascade there is another pathway that starts with the activation of IKKα that phosphorylates NF-κB2 (p100) that leads to its cleavage and the release of the p52 subunit which, together with RelB, constitutes a second NF-κB transcriptional complex that presumably targets genes different from those responding to RelA/p50. Again, genetic evidence from knock out mice has demonstrated that both cascades are critical and independent. An important missing piece of this puzzle was the identification of the cytokines that trigger this novel non-canonical IKKα/NF-κB2 pathway. Data presented in this workshop demonstrate that BAFF, for B cells, and LT-β, for fibroblasts, activate this new pathway through an IKKγ-independent mechanism. These exciting evidences explain, at least in part, the immunological phenotype of the IKKα mutant mice and open new avenues for therapeutic intervention in diseases such as rheumatoid arthritis, asthma or some form of diabetes. In addition, the observation that the mutant IKKα mice display impairment in the proliferation of mammary gland epithelial cells, suggests that inhibitors of this kinase could be useful drugs in the treatment of breast cancer. In fact, results were presented in this meeting demonstrating that the incidence of tumors caused by the *neu* oncogene was severely inhibited in IKKα mutant mice. Mutations in the IKKγ gene have been associated with a human disease called Incontinentia Pigmenti. Strikingly, another human genetic disease has been linked to mutations in these pathways. Thus, mutations in IκBα that prevent its degradation are associated with a disease called Anhidrotic Ectoderma Dysplasia that show an impairment in T cell function as well as in the response to activation through the Toll system.

The genetic inactivation of the different NF-κB subunits gives rise to alterations in the function of B and T cells. Interestingly, knock out mice for the PKC isoforms ζPKC and θPKC display defects in B and T cells, respectively. In the case of ζPKC and B cells, the role of this kinase in the control of apoptosis may account for the phenotypic alterations detected in the ζPKC mutant mice. In the case of T cells, a novel pathway involving not only θPKC but also the adapters Bcl10 and MALT-1 was presented. The connection here with cancer is evident. Mutations in both Bcl-10 and MALT-1 genes are associated with lymphomas. On the other hand, the involvement of NF-κB in the acquired as well as the innate immune response is clear. In this regard, much work has been done in the model system of *Drosophila* where both the classical and the non-canonical pathways exist. However, although some components are conserved, others are not, indicating that sometimes findings in flies cannot be automatically translated to mammals and vice versa.

The last NF-κB workshop organized in the Juan March Foundation was in 1996. In the last six years our understanding of the pathway has increased spectacularly. New genes have been identified and knock out mice for virtually all the proteins of these cascades have been generated, which allow us to know their function not only in cell cultures but also in a whole organism. In addition, the crystallographic study of the structures of at least some of the proteins of these pathways provides details of the intricate interactions that take place. In summary, much has been accomplished but more questions aroused in the intense and fruitful discussions that took place. Above all, it is important to understand the biology behind these fundamental cellular mechanisms but also important is to take advantage of its powerful therapeutic potential. Examples of this were presented. In the forthcoming years we will witness fabulous changes in the way patients are treated. This will only be possible with an exhaustive understanding of the signaling cascades operating under normal conditions, and subverted under the different pathologies.

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Engineering RNA Virus Genomes as Biosafe Vectors

Organized by
C.M. Rice, W.J.M. Spaan and L. Enjuanes

(21-23 October)

Engineering RNA virus genomes as biosafe vectors for vaccine development and gene therapy was the title of a meeting that took place at the "Instituto Juan March de Estudios e Investigaciones". Progress on human genome knowledge is leading to the identification of gene defects responsible for human diseases. These defective genes can be replaced by using somatic gene therapy, but it is essential to develop vectors that express the healthy gene with a predefined timing and tissue distribution.

Viral and bacterial pathogens are responsible for the major diseases and death worldwide. A recent report of the World Health Organization has shown that AIDS, malaria, tuberculosis, measles and acute respiratory and enteric infections are responsible for 90% death in humans less than 44 year old. To prevent these infectious diseases it is important to improve the current vaccination and gene therapy strategies and design new ones. These strategies have a common element, the administration of drugs that interfere with the replication of the microorganism. Frequently this intervention is mediated by genes encoding proteins that elicite responses inhibiting pathogen replication. The efficient introduction of genetic material into tissues requires to overcome the defenses that the organism has set up to counteract the entrance of foreign materials, to drive these genes to the target organ, and to control their expression. Viruses have evolved through many generations to overcome these barriers and frequently show high tissue specificity, making viruses ideal tools to target expression of heterologous genes. The use of viruses as vectors for the tissue specific expression is essential for both vaccine design and gene therapy.

During the meeting, emphasis was focused on RNA viruses because generally their genomes are not integrated within the host chromosome, except the retroviruses, making RNA virus vectors safer. In addition, vectors based on RNA viruses displaying a variety of tropisms have been emerging during recent years. As reported through the meeting, these viruses are being very helpful both in vaccination and gene therapy since tissue specific expression is essential for the success of the treatment. The engineering of infectious cDNA clones for most viruses, including those with RNA genomes of positive and negative polarity has facilitated studies by reverse genetics with these viruses in order to precisely control their virulence. The construction of replication-competent propagation-deficient viruses, and of non-cytopathic replicons based on viral genomes derived from several virus families, has facilitated the study of virus replication and the use of viruses as vehicles for vaccine development and gene therapy.

The continuous emergence of new viral pathogens was reported. These pathogens frequently cross the species barrier due to their high evolution rates that facilitate the adaptation of viral genomes to new ecological nitches. The existence of viruses as quasispecies poses some uncertainty on the safety of any live virus vector. Therefore it was concluded that strong safety guards must be engineered in these vectors, in order to increase the ratio benefit to risk to acceptable levels. Due to the risk involved in the generation of new virus entities the use of appropriated containment facilities and professional behavior of the scientists involved in the generation of new autoreplicative genomes was considered essential.

It must be expected that in the new millennium the administration of a viral vector to plants, animals or human beings will require that this type of interventions are made in a safe way. Although any intervention in human beings has associated a risk, it is also possible to design strategies that reduce this uncertainty to levels further lower that those associated with common life. Accordingly, biosafety on virus vector design must be a priority.

The meeting was intense, permitted close interaction between scientists working on different aspects of virus replication and, hopefully, will stimulate the opening of new research avenues.

Charly M. Rice, Willy J. M. Spaan and Luis Enjuanes

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Exchange Factors

Organized by
X. R. Bustelo, J.S. Gutkind and P. Crespo

(4-6 November)

Small GTP-binding proteins of the Ras superfamily operate as key molecular switches in signal transduction routes that convey stimuli received in cell surface receptors to the interior of the cell. Today, it is very clear that these proteins play essential roles in the regulation of basic cellular processes such as proliferation, differentiation and apoptosis. Thus, their malfunction can lead to extreme pathologic conditions like cancer.

The hallmark of Ras GTPases function is the transit between an inactive state, in which they are bound to guanosine diphosphate (GDP), and an active state bound to guanosine triphosphates (GTP). This cycle is a strictly regulated process, in which Guanine nucleotide Exchange Factors (GEFs) bring about the activation of the GTPases by catalyzing the nucleotide exchange process. Logic dictates that a fine-tuning of such an essential system requires that GEFs themselves must also be subject to tight regulatory processes. Indeed, it looks as if evolution has taken this matter very seriously and has provided GEFs with a wide array of regulatory domains. The role of many of these domains, in most cases, is still largely unveiled. Even a domain like the Dbl-Homology (DH) domain characteristic of Rho family GEFs, that orthodoxy has long regarded as a *bona fide* catalytic domain, still inspires doubts about whether it also stores other, hitherto unknown, functions. A ripe field in which much efforts are being invested.

Focusing on GEFs for Ras, we have learned that some like Ras-GRF1 and the Cal-DAG family are not only regulated by stimuli that elevate intracellular calcium levels or generate diacylglycerol. These GEFs are also subject to a tight control that is dependent on the cellular location or membrane compartment in which they are acting upon their cognate Ras GTPases. A similar situation is evident on some Rho GEFs like GEF-H1 whose activity is dependent on its interaction with microtubules. Location: a new aspect of GEF regulation is beginning to unfold.

Regardless of whether the regulation of GEFs is achieved through reversible modifications, like phosphorylation or lipidic additions, or by the interaction with other regulatory proteins, GEFs are subject to profound structural changes. In this field, the resolution of the crystal structures of GEFs catalytic domains in complex with their cognate GTPases and of several GEFs regulatory domains, have provided a large body of invaluable information that has enabled profound advances in our understanding of how GEFs work. These techniques, that are under a process of continuous refinement, are providing an essential tool for probing GEFs functions.

The unquestionable importance of GEFs from the biochemical point of view is clearly mirrored when looking at biological readouts. Right from the bottom of the evolution ladder, some bacteria have developed successful weapons on their struggle against the defensive arsenals of their eukaryotic hosts. Proteins that mimic GEF functions and different toxins that can either inhibit or activate GTPases, enable bacteria to orchestrate the cellular machinery to work for their own benefit. We have witnessed how GEFs begin to display their fundamental functional and structural characteristics in yeast and how these evolve in mammalians, to become key players in processes ranging from the regulation of the immune response to the

control of cell proliferation. And how when GEF function is either missing or gone astray, the consequences are dire.

All in a nutshell, this workshop gathered most of the leading scientist on the field of GEFs, intending to provide an insight on the latest trends on the subject, approached from different perspectives. We were enlightened by pouring new data. We were frightened by the speed with which the field evolves and new technologies are absorbed and rendered essential to keep on investigating. And, above all, we were humbled by how much there is still to learn in this fascinating area.

Piero Crespo and Xosé R. Bustelo

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The Ubiquitin-Proteasome System

Organized by
A. Ciechanover, D. Finley, T. Sommer and C. Mezquita

(18-20 November)

Protein degradation, originally thought to function primarily in the elimination of defective proteins, is now understood to be a central regulatory mechanism in eukaryotes. In targeting proteins for degradation by the proteasome, ubiquitination controls the activity of regulatory proteins in response to extracellular signals, stress, developmental transitions, checkpoint activation, and cell cycle transitions. The same modification can also confer nonproteolytic fates on its target proteins. Among these alternative functions of ubiquitination, the best understood—and most extensively covered in the workshop—are in the sorting of membrane proteins. Ubiquitination can signal endocytic events, sorting of proteins into the interior vesicles of the multivesicular body (MVB), sorting of proteins exiting the golgi body, and the budding of many viruses. Within the nucleus, ubiquitination plays nonproteolytic roles in DNA repair and transcription. This type of function was best illustrated in the workshop by studies linking ubiquitination of the DNA polymerase-associated processivity factor PCNA to DNA repair.

It is largely unclear how the same protein modification can have such a variety of functions. At least four principles that may underlie the triaging of ubiquitin-conjugates to alternative fates were discussed. First, ubiquitin is often added to proteins in the form of a multiubiquitin chain, and such chains are thought to be required for recognition of substrates of the proteasome. Other functions of ubiquitination, such as in endocytosis and sorting into the MVB, are driven by monoubiquitination. Second, the multiubiquitin chain can be polymerized using alternative lysine residues in ubiquitin, to form topologically distinct chain configurations. For example, lysine-48-linked chains are critical for targeting proteins to the proteasome, while lysine-63-linked chains serve nonproteolytic roles in DNA repair, protein kinase activation, and translational control. Third, conjugated ubiquitin can be recognized together with additional signals, such as specific phosphoinositides. Finally, it is likely that the localization of proteins, especially whether they are soluble or membrane-bound, helps to determine the way in which ubiquitination affects their fates. This principle has emerged most clearly from studies of the ERAD pathway for the degradation of endoplasmic reticulum proteins, as discussed by several speakers.

The scope of the ubiquitin system is still poorly defined, however, mammalian genomes contain over 600 genes encoding probable components of the ubiquitin-proteasome pathway. Most of these genes encode putative ubiquitin-protein ligases, or E3 enzymes, the primary specificity factors of the system, but a surprising number encode deubiquitinating enzymes. Obviously, the bulk of enzymes in this system remain completely uncharacterized.

Recent studies have also described a family of ubiquitin-like molecules. Although only partially characterized as a group, these molecules have so far been implicated in the regulation of nucleo-cytoplasmic transport, DNA repair, photomorphogenesis, the cytoskeleton, topoisomerase activity, centriole duplication, cell cycle checkpoint control, inflammation, autophagy, and other processes. These aspects of the ubiquitin family were best represented at the workshop by the elegant recent work on autophagy. Ubiquitin-like proteins also serve as positive and negative regulators of the ubiquitin pathway itself. Negative regulation is achieved by competing with ubiquitin for modification of specific sites within

substrate proteins, while positive regulation is achieved either by covalent modification of ubiquitin-ligase subunits or by noncovalent association with the proteasome.

If the success of a meeting is best gauged by its open discussions, this was a splendid few days. The intimate size of the workshop, its schedule, perhaps even the unique venue elicited friendly interaction and constructive thinking. It was the most skilfully but also the most creatively organized conference in my experience, and we are most grateful to the people at the Juan March Foundation for hosting us.

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Manufacturing Bacteria: Design, Production and Assembly of Cell Division Components

Organized by
P. de Boer, J. Errington and M. Vicente

(16-18 December)

The process of division (cytokinesis) is vital to all living cells. In prokaryotes, the process initiates with the assembly of a key division protein, FtsZ, into a polymeric ring (Z-ring) just underneath the cytoplasmic membrane at a precisely selected site to ensure that partition satisfies the physiological requirements of either cell division or differentiation. Other essential division proteins are recruited to this ring in a specific order, resulting in a mature division apparatus (septal ring, septator or divisome) which mediates cell constriction (Margolin. 2000. *FEMS Microbiol. Rev.* 24: 531-548).

Significant progress in the molecular description of prokaryotic cytokinesis has taken place recently and includes:

i) Division proteins FtsZ and FtsA share biochemical properties and tertiary structures with the major eukaryotic cytoskeletal proteins (tubulin and actin, respectively), indicating common ancestries (Löwe and Amos. 1998. *Nature* 391: 203-206; van den Ent and Löwe. 2000. *EMBO J.*19:5300-5307).

ii) Advances in genomics have revealed an almost universal presence of FtsZ in a wide variety of prokaryotic organisms (eubacteria and archaea), as well as in eukaryotic organelles (plastids and some mitochondria). In addition, many of the other division genes, as well as their chromosomal arrangement, have been conserved among dissimilar prokaryotic phyla (Tamames *et al*, 2001. *TIG* 17: 124-126).

iii) A combination of genetic, biochemical, and microscopic techniques has provided a stream of new information on the spatial and temporal regulation of the key initiating event (Z-ring assembly), the composition and order of assembly of the mature apparatus, and on the coupling of cell constriction with synthesis of the peptidoglycan portion of the cell wall (Hale and de Boer. 1999. *J. Bacteriol.* 181: 167-176; Meinhardt and de Boer. 2001. *Proc. Natl. Acad. Sci. USA* 98: 14202-14207; Erickson, 2001. *Curr. Opinion Cell Biol.* 13:55–60).

iv) Activities of the division apparatus have been shown to be intimately coupled with sister chromatid separation, as well as with cell cycle progression and cell differentiation in developmental model organisms (Edwards and Errington. 1997. *Mol. Microbiol.* 24:5 905-5915).

These developments are not only crucial to our understanding of bacterial physiology, but will have far-reaching consequences on the future management of important social issues as diverse as the conservation of the environment, and public health.

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2002 Fellowships

2002 FELLOWSHIPS

In meetings organized by the Centre a limited number of fellowships is normally offered to participants, in order to help them cover at least part of their travel and accommodation expenses.

These fellowships are usually awarded to the younger scientists selected for participation, or to scientists coming from countries where availability of funds is particularly scarce.

During 2002, 52 of these fellowships were awarded to participants in 14 different meetings. Among these, 13 fellowships were granted to scientists working in Spain, and 39 to scientists working abroad.

Reviews in Scientific Journals

During 2002, the meetings organized by the Centre have been reviewed in the following articles:

- Serrano, R. and Rodríguez, P. L. (2002). Plants, genes and ions. **EMBO Reports 3 (2): 116-119.** (On the workshop on *Molecular Basis of Ionic homeostasis and Salt Tolerance in Plants*, held in October 2001).

- Murray, J. A. H. (2002). Plant Development Meets Cell Proliferation in Madrid. **Developmental Cell 2: 21-27.** (On the workshop on *Cross Talk between Cell Division and Development in Plants*, held in November 2001).

- Boniotti, M. A. and Griffith, M. E. (2002). "Cross-Talk" between Cell Division Cycle and Development in Plants. **The Plant Cell 14: 11-16**. (On the workshop on *Cross Talk between Cell Division and Development in Plants*, held in November 2001).

- Smith, C. I. E. (2002). Disease Models for Every Field. Molecular basis of human congenital lymphocyte disorders. **EMBO Reports 3 (6): 516-520.** (On the workshop on *Molecular Basis of Human Congenital Lymphocyte Disorders*, held in December 2001).

- Valverde, M. A. and Parker, M. G. (2002). Classical and novel steroid actions: a unified but complex view. **Trends in Biochemical Sciences 27 (4): 172-173.** (On the workshop on *Genomics vs Non-Genomic Steroid Actions: Encountered or Unified Views*, held in December 2001).

- Alcamí, A., Ghazal, P. and Yewdell, J. W. (2002). Viruses in control of the immune system. **EMBO Reports 3 (10): 927-932.** (On the workshop on *Molecular Mechanisms of Immune Modulation: Lesson from Virus*, held in February 2002).

- Aizenman, E. and Sanguinetti, M. C. (2002). Channels Gone Bad: Reflections from a Tapas Bar. **Neuron 34: 679-683.** (On the workshop on *Channelopathies*, held in March 2002).

- Torres, M., Couso, J. P. and Ros M. A. (2002). Building limb buds. **EMBO Reports 3 (10): 933-937.** (On the workshop on *Limb Development*, held in April 2002).

- Orlando, V. and Jones, K. A. (2002). Wild chromatin: regulation of eukaryotic genes in their natural chromatin context. **Genes & Development 16: 2039-2044.** (On the workshop on *Regulation of Eukaryotic Genes in Their Natural Chromatin Context*, held in April 2002).

- Special issue of **FEBS Letters 531 (1)** (2002), with 20 articles signed by invited speakers and participants of the meeting on *Lipid Signalling: Cellular Events and Their Biophysical Mechanisms*, held in May 2002.

- Mellman, I. and Ridley, A. (2002). Regulation and Functional Insights in Cellular Polarity. **The Journal of Cell Biology 158 (1): 12-16.** (On the workshop on *Regulation and Functional Insights in Cellular Polarity*, held in June 2002).

- Dustin, M. L. (2002). Shmoos, Rafts, and Uropods- The Many Facets of Cell Polarity. **Cell 110: 13-18.** (On the workshop on *Regulation and Functional Insights in Cellular Polarity*, held in June 2002).

- Nelson, S. B. (2002). Cortical Microcircuits: Diverse or Canonical?. **Neuron 36: 19-27**. (On the workshop on *The Structure of the Cortical Microcircuit*, held in June 2002).

- Dixit, V. and Mak, T. W. (2002). NF-kB Signaling: Many Roads lead to Madrid. **Cell 111: 615-619**. (On the workshop on *Control of NF-kB Signal Transduction in Inflammation and Innate Immunity*, held in October 2002).

Editors of the following major scientific journals have participated in different meetings of the Centre during 2001: **Cell** (six meetings); **Developmental Cell** (one meeting); **Neuron** (two meetings); **Nature Reviews Immunology** (one meeting); **Nature Reviews Neuroscience** (two meetings); **Nature Immunology** (one meeting); **Nature Cell Biology** (one meeting); **EMBO Journal** (one meeting); **Genes & Development** (one meeting); **FEBS Letters** (one meeting).

2003 Meetings Schedule

CENTRE FOR INTERNATIONAL MEETINGS ON BIOLOGY
2003 MEETINGS SCHEDULE

Date	Meeting Subject	Organizers
27-29 January	**Membranes, Trafficking and Signalling during Animal Development**	**K. Simons.** Max-Planck-Institut für Molekulare Zellbiologie und Genetik. Dresden. **M. Zerial.** Max-Planck-Institut für Molekulare Zellbiologie und Genetik Dresden. **M. González-Gaitán.** Max-Planck-Institut für Molekulare Zellbiologie und Genetik. Dresden.
10-12 February	**Synaptic Dysfunction and Schizophrenia**	**P. Levitt.** Vanderbilt University. Nashville. **D.A. Lewis.** University of Pittsburgh Medical School. Pittsburgh. **J. DeFelipe.** Instituto Cajal. Madrid.
24-26 February	**Plasticity in Plant Morphogenesis**	**G. Coupland.** Max-Planck-Institut für Züchtungsforchung. Köln. **C. Fankhauser.** Université de Genève. Genève. **M.A. Blázquez.** Instituto de Biología Molecular y Celular de Plantas. Valencia.
24-26 March	**Wnt Genes and Wnt Signalling**	**R. Nusse.** Howard Hughes Medical Institute. Stanford. **J.F. de Celis.** Centro de Biología Molecular "Severo Ochoa". Madrid. **J.C. Izpisúa Belmonte.** The Salk Institute for Biological Studies. La Jolla.
*7-9 April	**Molecular and Genetic Basis of Autoimmune Diseases: SLE and RA**	**A. Coutinho.** Instituto Gulbenkian de Ciência. Oeiras. **W. Haas.** Instituto Gulbenkian de Ciência. Oeiras. **C. Martínez-A.** Centro Nacional de Biotecnología. Madrid.
12-14 May	**The Dynamics of Morphogenesis: Regulation of Cell and Tissue Movements in Development**	**C. Stern.** University College London. **M.A. Nieto.** Instituto Cajal. Madrid.
9-11 June	**Developmental Mechanisms in Vertebrate Organogenesis**	**G. Oliver.** St. Jude Children's Research Hospital. Memphis. **M. Torres.** Centro Nacional de Biotecnología. Madrid.
23-25 June	**Neuronal Degeneration and Novel Therapeutic Approaches in Parkinson´s Disease**	**C.W. Olanow.** Mount Sinai School of Medicine. New York. **J.A. Obeso.** Universidad de Navarra. Pamplona. **R. Moratalla.** Instituto Cajal. Madrid.
6-8 October	**Dendritic Cells: Biology and Therapeutic Applications**	**R.M. Steinman.** Rockefeller University. New York. **I. Melero.** Universidad de Navarra. Pamplona. **A.L. Corbí.** Centro de Investigaciones Biológicas. Madrid.
20-22 October	**Finding the Way Out: Protein Traffic in Bacteria**	**A.P. Pugsley.** Institut Pasteur. Paris. **V. de Lorenzo.** Centro Nacional de Biotecnología. Madrid.
3-5 November	**The Calcium/Calcineurin/NFAT Pathway: Regulation and Function**	**E.N. Olson.** University of Texas Southwestern Medical Center. Dallas. **J.M. Redondo.** Centro de Biología Molecular "Severo Ochoa". Madrid.
17-19 November	**Telomeres and Telomerase: Therapeutical Targets for Cancer and Aging**	**S. Neidle.** Institute of Cancer Research. London. **J.W. Shay.** University of Texas Southwestern Medical Center. Dallas. **M.A. Blasco.** Centro Nacional de Biotecnología. Madrid.

* This meeting will take place at the Gulbenkian Foundation, Lisbon. All others will be organized at the Juan March Institute, Madrid.

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The **Fundación Juan March** is a private, non-profit making institution established in 1955.

It has organized more than 520 *art exhibitions* in Spain and abroad. Some 500 artists and researchers have received grants from the Foundation for creative or research projects in the fine arts. The Foundation's art collections are exhibited in the Museo de Arte Abstracto Español, in Cuenca; in the Museu d'Art Espanyol Contemporani, in Palma de Mallorca; in the Foundation's headquarters, in Madrid, and also in travelling exhibitions.

In the field of *music*, the Foundation regularly organizes series of monographic concerts, didactic concerts for young people (attended each year by approximately 25,000 students), commemorative concerts in honour of major musical figures, as well as concerts of a variety of other types. In total, more than 200 concerts are organized each year.

Two *libraries*, with specialized collections in Spanish Contemporary Theatre and Spanish Contemporary Music, are housed in the Foundation's headquarters. More than 50 lectures, seminars and courses are organized there each year, on a wide range of subjects.

The Foundation publishes a monthly Bulletin as well as "SABER/*Leer*", an illustrated book review. Annual reports, catalogues, leaflets and other publications are issued on a non-periodical basis.

The **Instituto Juan March de Estudios e Investigaciones** was established in 1986 as a private Foundation to support research and post-graduate studies in scientific fields, by means of specialised Centres of Advanced Study.

In 1987 the *Centre for Advanced Study in the Social Sciences* was created within the Juan March Institute to contribute to the extension of social scientific knowledge through the promotion of research, post-graduate teaching, and exchanges of researchers.

In 1992 the *Centre for International Meetings on Biology* was established to promote close cooperation and interaction among Spanish and foreign scientists working in the field of Biology, through workshops, lectures courses, seminars and symposia.

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