Instituto Juan March
de Estudios e Investigaciones

118 CENTRO DE REUNIONES
INTERNACIONALES SOBRE BIOLOGÍA

2000
Annual Report

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

Man is endowed with a relentless curiosity about the world. We cannot be satisfied forever by the guesses of yesterday, however much the charms of tradition and ritual may, for a time, lull our doubts about their validity. We must hammer away until we have forged a clear and valid picture not only of this vast universe in which we live but also of our very selves.

Francis Crick. "The Astonishing Hypothesis" (1993)

INSTITUTO JUAN MARCH DE ESTUDIOS E INVESTIGACIONES

CENTRE FOR INTERNATIONAL MEETINGS ON BIOLOGY

2000 ANNUAL REPORT

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FOREWORD

This publication covers the activities of the Centre for International Meetings on Biology during the year 2000. All of them were, in due time, broadly announced by means of brochures, posters, advertisements in scientific journals and other periodicals.

The core of the Centre's work during 2000 was the organization of thirteen 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, 263 speakers were invited to the meetings during this year, and 394 participants were chosen from among 602 applications received. Four lectures open to the general public were held to coincide with the meetings mentioned above.

13 booklets were published on these meetings, including the abstracts of the contributions presented by the participating scientists. About 400 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 has been established by the Fundación Juan March in 2000. Details of this new iniciative are described in the following pages.

A short notice is given on reviews published during 2000 in scientific journals regarding meetings organized by the Centre.

The schedule of meetings to take place in 2001 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 comprises: Juan March (Chairman), Carlos March (Deputy Chairman), Leonor March, Alfredo Lafita, Antonio Rodríguez Robles, Pablo Vallbona, Enrique Piñel and Jaime Prohens. 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 2000 the Scientific Council of the Centre comprised the following members:

Miguel Beato
Institut für Molekularbiologie und
Tumorforschung. Marburg (Germany)

José A. Campos-Ortega
Institut für Entwicklungsbiologie. Köln (Germany)

Gregory Gasic
Neuron Editorial Office. Cambridge (USA)

César Milstein
Medical Research Council, Cambridge (UK)

Margarita Salas
Centro de Biología Molecular. CSIC - Universidad
Autónoma de Madrid (Spain)

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

Since the beginning of 2001 the Scientific Council will comprise the following members:

César Milstein
Medical Research Council
Cambridge (United Kingdom)

Ginés Morata
Centro de Biología Molecular "Severo Ochoa"
CSIC - Universidad Autónoma de Madrid (Spain)

Erwin Neher
Max-Planck-Institut für Biophysikalische Chemie
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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 has decided to award each year a Grant of 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.

In September 2000 the Grant was awarded, for the first time, to Prof. José López-Barneo (School of Medicine, University of Seville, 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 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.

2000 Meetings Schedule

CENTRE FOR INTERNATIONAL MEETINGS ON BIOLOGY
2000 MEETINGS SCHEDULE

Date	Meeting Subject	Organizers
28 February-1 March	**The Molecules of Pain: Molecular Approaches to Pain Research**	**S. P. Hunt.** University College London. **F. Cervero.** Universidad de Alcalá. Madrid.
13-15 March	**Control of Signalling by Protein Phosphorylation**	**J. Schlessinger.** NYU Medical Center. New York. **G. Thomas.** Friedrich Miescher Institute. Basel. **F. de Pablo.** Centro de Investigaciones Biológicas. Madrid. **J. Moscat.** Centro de Biología Molecular "Severo Ochoa". Madrid.
27-29 March	**Biochemistry and Molecular Biology of Gibberellins**	**P. Hedden.** University of Bristol. **J.L. García-Martínez.** Instituto de Biología Molecular y Celular de Plantas. Valencia.
10-12 April	**Integration of Transcriptional Regulation and Chromatin Structure**	**J.T. Kadonaga.** University of California. San Diego. **J. Ausió.** University of Victoria. **E. Palacián.** Centro de Biología Molecular "Severo Ochoa". Madrid.
8-10 May	**Tumor Suppressor Networks**	**J. Massagué.** Memorial Sloan-Kettering Cancer Center. New York. **M. Serrano.** Centro Nacional de Biotecnología. Madrid.
22-24 May	**Regulated Exocytosis and the Vesicle Cycle**	**R.D. Burgoyne.** University of Liverpool. **G. Alvarez de Toledo.** Facultad de Medicina. Universidad de Sevilla.
* 5- 7 June	**Dendrites**	**S. Siegelbaum.** Columbia University. New York. **R. Yuste.** Columbia University. New York.
19-21 June	**The Myc Network: Regulation of Cell Proliferation, Differentiation and Death**	**R.N. Eisenman.** Fred Hutchinson Cancer Research Center. Seattle. **J. León.** Facultad de Medicina. Universidad de Cantabria. Santander.
2- 4 October	**Regulation of Messenger RNA Processing**	**W. Keller.** University of Basel and Biozentrum. Basel. **J. Ortín.** Centro Nacional de Biotecnología. Madrid. **J. Valcárcel.** European Molecular Biology Laboratory. Heidelberg.
16-18 October	**Genetic Factors that Control Cell Birth, Cell Allocation and Migration in the Developing Forebrain**	**P. Rakic.** Yale University. New Haven. **E. Soriano.** Facultad de Biología. Universidad de Barcelona. **A. Alvarez-Buylla.** Rockefeller University. New York.
6- 8 November	**Chaperonins: Structure and Function**	**W. Baumeister.** Max--Planck-Institute for Biochemistry. Munich. **J.M. Valpuesta.** Centro Nacional de Biotecnología. Madrid. **J.L. Carrascosa.** Centro Nacional de Biotecnología. Madrid.
27-29 November	**Comparison of the Mechanisms of CellularVesicle and Viral Membrane Fusion**	**J.J. Skehel.** National Institute for Medical Research. London. **J.A. Melero.** Centro Nacional de Biología Fundamental. ISCIII. Madrid.
11-13 December	**Molecular Approaches to Tuberculosis**	**B. Gicquel.** Institut Pasteur. Paris. **C. Martín.** Facultad de Medicina. Universidad de Zaragoza.

*** This meeting took place at Columbia University, New York. All others were organized at the Instituto Juan March, Madrid.**

The Molecules of Pain: Molecular Approaches to Pain Research

Organized by
S. P. Hunt and F. Cervero

(28 February-1 March)

Pain research holds a halfway position between the pure and the applied neurosciences. Pain is a distinct sensation that shares properties and mechanisms with other sensations but that preserves peculiarities of its own. Some of these peculiarities, particularly the sensory amplification phenomenon known as hyperalgesia, separate pain from the other sensations and offer an exciting insight into the workings of the brain. However, pain is also the most common symptom of disease and a frequent cause for patients to seek medical attention. The applied aspects of the study of pain mechanisms have become, understandably, an essential concern of the pharmaceutical industry.

Pain researchers are frequently called upon to suggest targets for the development of novel analgesics. There are those who favour the peripheral sensory nerve as the most accessible target for exploration and those, with perhaps more respect for the plastic qualities of pain, who point to the central nervous system as the most likely site for effective long term control of pain. Whichever option is chosen it seems from past experience that certain pain conditions yield to peripheral intervention while others require drugs that act predominantly within the central nervous system. However, the success of these therapeutic approaches tended to be somewhat serendipitous. The arrival of molecular biological approaches to the pain field promises rational approaches to the alleviation of suffering.

The objective of the Workshop was to discuss the cellular and molecular aspects of pain mechanisms, an area of research where considerable progress has been made in the last few years. In essence the approach has been to identify a molecule that is potentially involved in the signalling of pain and then to manipulate the gene by genetic 'knockout' or some other approach. Even allowing that these approaches can be compromised by developmental and other compensations, the results have been very revealing. For the most part gene knockout has given, at best, partial confirmation of our previous hypotheses regarding the function of the encoded protein or polypeptide. More important have been the insights into pain processing that have been gained from these molecular approaches causing us to rethink and modify some of our previously held views.

Our understanding of the physiology of the sensory nerve and sensory transduction has been accelerated by the cloning of a number of sensory receptors and ion channels that are specific to the peripheral sensory nerve. Selective knockout of these genes has begun to reveal modest phenotypes. Deletion of genes, such as those that code for the substance P and opiate receptors that are expressed within the brain and spinal cord and that have previously been assumed to play an important role in pain processing, has proved more complicated. This is because we have had to confront not only changes in sensory processing but also in the emotional and motivational dimensions of the pain response. Ablation of biochemically specific subpopulations of neurons was also discussed in the Workshop and the results have forced us to reconsider the interrelationships between the spinal cord and the brain through ascending and descending pathways.

The Workshop was organised into sessions on nociceptors and sensory transduction, followed by sessions built around substance P, opiate and glutamate receptors. Each topic served as a starting point for the exploration of pain control from both peripheral and central perspectives.

The Workshop underscored the conclusion that ultimately effective pain control, particularly of long term chronic pain, will demand an understanding of pain mechanisms at all levels of the neuraxis. Different pain conditions will require different therapeutic interventions each tailored to the particular biological signature of the condition.

F. Cervero and S. P. Hunt

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Control of Signalling by Protein Phosphorylation

Organized by
J. Schlessinger, G. Thomas, F. de Pablo and J. Moscat

(13-15 March)

The phosphorylation and dephosphorylation of proteins and lipids are critical processes for the execution of the cellular programs leading to proliferation, differentiation and survival. The recent completion of the *D. melanogaster* genome and its comparison with that of other organisms such as *C. elegans and S. cerevisiae*, has revealed that protein kinases are among the top 10 protein families that are common to all three organisms and constitute aproximately 2% of each proteome. In *Drosophila*, the kinase group is constituted by aproximately 75 % serine/threonine kinases and 25% tyrosine or dual specificity kinases. The impressive recent progress in identifying kinases and phosphatases implicated in signal transduction in eukaryots has been paralleled by progress in defining their phosphorylation sites, the effect of phosphorylation on protein-protein interactions and consequences of their mutation or functional inactivation at the level of the cell an the whole organism.

This meeting has served as a lively forum for discussion of recent advances in the field of signal transduction and its regulation by protein phosphorylation in systems ranging from mammals to yeast. The first part of the workshop focused on the role of receptor tyrosine kinases, PI3K and the MAPK/ERK signaling pathways in cell growth, proliferation and survival. Another series of presentations discussed cytokine signaling with special emphasis in the mechanism of NF-κB activation, as a paradigm of cytoplasmic signaling transmitted from membrane to nucleus. The crystal structure of several ligand-receptor complexes, serine-threonine kinases and the tumor suppressor PTEN were presented. The last part of the workshop was devoted to developmental and differentiation models addressing issues such as cell size control, apoptosis in vivo and cell cycle regulation.

Jorge Moscat and Flora de Pablo

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Biochemistry and Molecular Biology of Gibberellins

Organized by
P. Hedden and J. L. García-Martínez

(27-29 March)

The gibberellins (GAs) constitute a large group of diterpenoid compounds, some of which function as regulators of growth and development in plants. They were discovered about 60 years ago in the fungus *Gibberella fujikuroi*, the agent of the "bakanae" rice disease, which is characterized by a very elongated phenotype. GAs were shown later to occur naturally in plants, where they control developmental processes throughout the life cycle, including seed germination, stem elongation, flower induction and development, and fruit growth. They have also been shown to mediate the effects of environmental stimuli, such as photoperiod or low temperatures, on developmental processes, particularly germination, bolting and flowering.

Experiments with mutants and inhibitors of GA biosynthesis have indicated that relatively few of the 125 currently known GA structures function as hormones, many of the others being precursors or catabolites of the active GAs. The complex biosynthetic pathway to the active compounds is well understood. It consists of three stages: in the first, geranylgeranyl diphosphate is converted in two steps to *ent*-kaurene by cyclases (copalyl diphosphate synthase, CPS, and *ent*-kaurene synthase, KS) located in plastids; in the second stage, *ent*-kaurene is oxidised on membranes by cytochrome P450 monooxygenases to GA_{12} and GA_{53}, which, in the third stage, are converted by soluble, 2-oxoglutarate-dependent dioxygenases to the active hormones, GA_4 and GA_1, respectively. Dioxygenases also catabolize the inactivation of GAs by 2β-hydroxylation.

Within the last five years, spectacular progress has been made in our understanding of GA biosynthesis and its regulation though the cloning of genes encoding the biosynthetic enzymes. More than half the genes of the pathway have now been cloned, including the cyclases CPS and KS, the cytochrome P-450 *ent*-kaurene oxidase, and the dioxygenases GA 20-oxidase, GA 3-oxidase and GA 2-oxidase. A GA 3-oxidase in pea was shown to be encoded by the *LE* gene, mutation of which causes dwarfism in pea; difference in stem height between tall (*LE*) and dwarf (*le*) peas was one of the seven genetic traits used by Mendel in his studies on the nature of inheritance. The availability of these clones has enabled progress in several areas. Expression of cDNAs in heterologous systems has provided sufficient amounts of enzymes for characterisation of their function and mechanism. It has also been possible to investigate the regulation of GA biosynthesis in relation to plant development, tissue localisation and environmental stimuli.

Although progress on the mode of action of GAs has not been as rapid as that on GA biosynthesis, impressive advances have also been made in this area, particularly from genetic approaches. GAs are known to modify the expression of genes in relation to several developmental processes. In particular, considerable progress has been made in understanding the regulation of α-amylase gene expression by GA in cereal aleurone cells. There is compelling evidence for the presence of GA receptors on the outside of the plasma membrane of cereal aleurone cells. The involvement of heterotrimeric G-proteins, Ca^{2+}, calmodulin, and a GAMyb transcription factor as signal transduction elements mediating the induction of α-amylase synthesis by GA in such aleurone cells has also been demonstrated. In the model plant species *Arabidopsis thaliana*, three genes (*GAI*, *SPY* and *RGA*) that act as negative regulators of the GA signal transduction pathway have been isolated and their interaction and function are being studied intensively. There have been reports of further genes that modify the GA response so that there should be exciting developments in this area in the next few years.

From a practical standpoint, several laboratories are manipulating GA content in transgenic plants by modifying the expression of GA biosynthesis genes (overexpressing and underexpressing using antisense and ribozyme technologies), or are altering the expression of GA response genes. These approaches are providing the means to modify phenotypic characteristics, such as shoot length or parthenocarpic fruit development, in species of agricultural interest.

The workshop assembled scientists working on molecular aspects of gibberellin biosynthesis and mode of action. It provided an opportunity to review the rapid and exciting progress that is being made in these areas, covering both fundamental and applied aspects of the work. Perhaps more importantly, it brought together workers from these two areas, biosynthesis and action, which have tended to be worked on separately by researchers with different interests and expertise. This was particularly timely since it is now realised that the two areas are closely linked, with GAs regulating the expression of GA-biosynthetic genes in feedback and feedforward regulation of biosynthesis.

Peter Hedden and José L. García-Martínez

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Integration of Transcriptional Regulation and Chromatin Structure

Organized by
J. T. Kadonaga, J. Ausió and E. Palacián

(10-12 April)

The regulation of gene transcription is critical for the proper growth and development of an organism. In eukaryotes, there are tens of thousands of protein-coding genes, each of which has its own unique program of transcription. Someday, we may perhaps be able to decipher the underlying code in the DNA that directs the proper extent of transcription of each gene at the appropriate time and place. This code, in some respects, might be thought of as the transcriptional component of a gene expression code, and would represent a significant achievement in biology.

How might we, however, move forward toward the solution of this gene expression code? One reasonable approach, which is the subject of this Juan March Workshop, is to investigate the basic molecular mechanisms by which transcription is regulated in eukaryotes. The current evidence indicates that the regulation of transcription of protein-coding genes by RNA polymerase II involves the basal transcription machinery, sequence-specific DNA-binding proteins that interact with cis-control elements, numerous co-regulatory factors, and the structure and constitution of the chromatin template. In this Workshop, we have sought to encompass and to integrate all of these factors.

The many stimulating and fascinating talks and discussions at the Workshop have brought forth many current concepts. First, all of the factors that participate in the transcription process play an active role in the regulation of gene expression. Indeed, even the basal transcription factors and the chromatin template participate in gene-selective transcription. Second, the processes by which transcription is regulated are of immense complexity. Factors can alternatively act as activators or as repressors, depending on their context. Moreover, reversible chemical modifications of chromatin such as by methylation (of DNA or histones), acetylation, or ubiquitination can also variably affect gene expression. There are a multitude of pathways and mechanisms by which genes can be activated or repressed. Clearly, we should minimize our expectations and remain completely open-minded with regard to how genes might be regulated. Third, there is the question of how many more regulatory factors remain to be discovered? Have we found most of the relevant factors, or are there many others yet to be identified? Of course, the answers to these questions are, at present, a matter of opinion. Fourth, we can see the emergence of new approaches and tools for the analysis of chromatin structure and transcriptional regulation. Such new assays and techniques will lead to future advances and revolutions in our understanding of gene expression.

The entirety of the Workshop cannot be summarized in a short statement. The Fundación Juan March provided the ideal setting for both talks and discussions. It is our hope that each participant was able to leave with at least a small handful of new knowledge and insight.

Jim Kadonaga, Juan Ausió and Enrique Palacián

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Tumor Suppressor Networks

Organized by
J. Massagué and M. Serrano

(8-10 May)

Tumor suppressor genes (TSGs) are the exact opposites to oncogenes. TSGs are "guardian genes" that watch over the proper proliferation and differentiation of cells, whereas oncogenes could be seen in this metaphor as "delinquents" that sabotage the normality of these processes favoring an unrestrained proliferation. Cancerous cells only arise when the function of these "guardians" is canceled by mutations, thus escaping their vigilance. To understand the normal functions of TSGs and the consequences of their inactivation is a scientific priority and a necessity to improve cancer therapies.

The first tumor suppressor gene (TSG) to be identified was the retinoblastoma susceptibility gene, Rb, in 1986. Since then, approximately 15 TSGs have been isolated. The normal function of all TSGs is to prevent uncontrolled cell proliferation, and they do this by acting on a variety of processes. Some TSGs directly regulate the cell division rate (Rb, NF-1), while others participate in cellular survival (PTEN), cell differentiation (APC, SMAD4), or in preventing proliferation under stressful conditions (ARF, p16, p53). The study of some TSGs has already moved into the stage of defining pathways. Indeed, it has been very gratifying to see that these pathways involve both TSGs and oncogenes in a kind of guardian-delinquent relation, or cat and mouse game, as it has been referred to by other researchers. This may seem obvious in retrospect, but it was not so when TSGs begun to be characterized. There are now several well-established "TSG-oncogene" antagonistic relations, for example (TSGs are underlined, oncogenes are not): p16/CDK4-cycD1/Rb; ARF/MDM2/p53; PTEN/PI3K; Wnt/APC/β-catenin; NF-1/Ras. These are embryonic pathways that are increasing in complexity and subtlety in these very moments thanks to the work of many laboratories and particularly in those of the participants to this workshop. This is a direction of the research that is now in its early days, and that has been the main focus of the Workshop "Tumor Suppressor Networks".

One of the leitmotivs of the workshop has been the use of the mouse as experimentation system. The manipulation of the mouse genome is increasing in sophistication and power, and these technologies are being used very actively for testing the role of TSGs and for developing mouse cancer models. As a good model system, the mouse occasionally reproduces the pathology of human cancers, but this is not always the case and perhaps it is in these occasions when the mouse turns out more informative. These "breaks of equivalence" between human and mouse cancers are indeed the motivation for further research and reveal aspects that otherwise may remain hidden. Also, mutations in two or more TSGs are being combined in the same mouse strains, thus testing TSGs networks directly in a mammalian organism.

This workshop has been privileged in many regards, first of all by the quality of the speakers, but also by other factors such as the opportunity of the topic which is in a very exciting and active moment, and finally by the constructive and collaborative attitude of all the participants. In keeping with the mark of the house, the Juan March Institute provided a professional organization and a warm hospitality.

Manuel Serrano

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Regulated Exocytosis and the Vesicle Cycle

Organized by
R. D. Burgoyne and G. Álvarez de Toledo

(22-24 May)

Neurons, neuroendocrine cells, exocrine cells, mast cells and many other cell types communicate by the regulated release of molecules from a stored vesicle pool. Fusion of the vesicles with the plasma membrane to release the vesicle contents by exocytosis is activated by an intracellular signal, in many cases a rise in calcium concentration. This is followed by rapid endocytosis and vesicle recycling. Over the past few years, intensive studies of the different cellular processes and of the proteins involved in regulated exocytosis and other intracellular steps in vesicular traffic, by several laboratories, has resulted in significant advances and led to the identification of a number of physiological steps and some of the proteins which are key components of a core machinery leading to vesicle fusion. These proteins are conserved in eukaryotic cells throughout evolution and they and their related family members are likely to function in all cell types.

The functional importance of many identified proteins in regulated exocytosis has been investigated and confirmed using a wide range of experimental approaches including the study of mutants in yeast, *Drosophila*, *C.elegans* and mice as well as manipulations of proteins within isolated cells. In addition, a variety of proteins that function in the acute regulation of exocytosis such as protein kinases and their substrates have been characterised. Currently the most exciting challenge is the task of understanding how these proteins interact in a sequential and ordered manner in order to lead to secretory vesicle docking, membrane fusion and vesicle membrane retrieval. The identification and *in vitro* analysis of proteins of the exocytotic machinery has coincided with significant advances in the application of high resolution techniques for the analysis of single fusion events and vesicle recycling in secretory cells by electrophysiological and single cell imaging techniques. These new approaches have not only allowed the demonstration and definition of multiple steps in the exocytotic pathway and characterisation of the endocytotic pathway but are now allowing the investigation of the defined role of identified proteins.

The field of exocytosis and the vesicle cycle is currently very active with many hundreds of papers being published each year. In this workshop many of the top scientists in the world, addressing the different approaches to better understand synaptic transmission at the cellular and molecular level, gathered in Madrid to communicate to the scientific community their new advances in this highly competitive and rapidly changing field of neuroscience.

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Dendrites

Organized by
R. Yuste and S. Siegelbaum

(5-7 June)

Co-sponsored by Columbia University

The role of dendrites in neuronal signal processing has been a topic of research for more than a century, since Cajal's original postulate that dendrites constitute the input side of the neuron. Cajal concluded that dendrites were passive structures. However, studies initiated by Rafael Lorente de Nó, and extended by Eric Kandel and Alden Spencer in the hippocampus and by Rodolfo Llinás in the cerebellum, later found that dendrites could produce action potentials. The workshop on Dendrites, sponsored by the Instituto Juan March and held at Columbia University in New York, on June 5-7, 2000, confirmed that dendrites are, indeed, very active participants in neuronal signaling and plasticity, as well as an extremely exciting and productive area of research.

Recent studies have greatly expanded our view of the functional properties of mammalian dendrites through novel experimental approaches such as two-photon microscopy and dendritic patch recording. These techniques have provided direct evidence for the presence of several types of voltage-gated conductances in dendrites and for the chemical compartmentalization of local biochemical signals in dendritic spines. Although the logic of dendritic processing is still unclear, a host of new findings indicate that mammalian dendrites are richer functionally than previously thought, and suggest that individual neurons are capable of sophisticated information processing.

The meeting was organized into presentations that focused on five main topics: 1. Electrical excitability; 2. Signaling through intracellular calcium; 3. Receptor localization and postsynaptic biochemical signaling; 4. Local protein synthesis and transport; 5. Developmental processes. In addition the meeting contained two special lectures. Rodolfo Llinas lectured on the importance of dendritic active conductances in the context of neuronal circuitry, and in particular of oscillating electrical activity in re-entrant loops in ensembles of neurons. Eric Kandel spoke on the importance of local protein synthesis in tagging active synapses for synapse-specific long-term plasticity at the Aplysia sensory to motor neuron connection.

During the meeting, several recurrent themes appeared. First, the generation of dendritic action potentials and their ability to propagate to the neuronal cell body is a complex process that can be influenced by the specific timing and pattern of synaptic activity. Modulation of dendritic voltage-gated channels by second messenger cascades can further regulate this electrical excitability. Second, there is a dynamic cycling of transmitter receptors in the dendritic postsynaptic membrane. This cycling can be controlled by synaptic activity, giving rise to long-term plasticity. Third, the dendritic spines themselves are plastic and undergo actin-based motility and shape changes. This can lead to formation of dendritic filopodia, totally new spines or a change in the structure of preexisting spines, which in turn can influence spine calcium dynamics.

Studies in the future are needed to determine the physiological function of dendritic excitability, spine calcium dynamics and spine motility.

R. Yuste and S. A. Siegelbaum

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The Myc Network: Regulation of Cell Proliferation, Differentiation and Death

Organized by
R. N. Eisenman and J. León

(19-21 June)

Over the last decade there has been a virtual explosion of information on the Myc network. In part this is simply an outgrowth of the longstanding interest in Myc stemming from its involvement in many types of cancer and the fundamental cellular processes of proliferation, differentiation, growth, and apoptosis. However, when Myc was shown to heterodimerize with Max and function as a DNA-binding transcription factor in 1990-1992, the interest of many laboratories became focused on attempting to understand how Myc's role in transcription directly relates to its biological functions in normal and cancer cells. To a large extent this has involved determining the molecular mechanism(s) whereby Myc regulates gene expression and, very importantly, the specific genes that are targets of Myc. The biology of Myc has also come under intense scrutiny as the role of Myc in cell cycle regulation and the importance of Myc-induced apoptosis in oncogenesis have become more apparent. Furthermore targeted deletions of Myc genes in mice and in tissue culture cells as well as genetic analysis of Myc function in Drosophila has highlighted roles for Myc in development, cell cycle, and cell growth.

In addition to the intense interest in Myc itself, the field has also expanded in other, initially unexpected, directions. This has come from the realization that Max, while an obligate dimerization partner for Myc, also interacts with other proteins (Mad, Mnt, Mga) and these proteins, in turn, appear to influence the functions of Myc. For example, much work on the Mad protein family suggests that Mad proteins act at least in part to antagonize Myc function and are likely to be involved in cell cycle exit and differentiation. Furthermore, work on understanding the repression function of Mad has led to identification of novel co-repressors (mSin3) and an analysis of the role of chromatin modification in repression in general. New evidence suggests that Myc interacts with a co-activator and may also influence chromatin structure. Another layer of complexity may arise from the findings that other novel proteins, such as Mlx, may potentially serve to connect specific members of the Myc network to other transcription factor modules.

The idea of the Myc network grew out of the increasing realization that Myc does not function alone but within an immediate context of interacting proteins as well as within a specific cellular environment. The goal of this workshop was to draw together what has become a rather diverse field of research in what is the first international meeting entirely dedicated to the Myc network. The workshop dealt with the most recent advances in this active field of research, from the molecular level to the most complex biological models. The talks and posters included the presentation of the molecular structure of Myc:Max , Mad:Max, and Mad:Sin3 complexes; new Max homologs, the Drosophila Mad orthologs, the interaction of Myc with proteins involved in chromatin remodeling and transcriptional regulation, new Myc target genes which are either positively or negatively regulated by Myc, insights to regulation of Myc expression, Myc interactions in tumorigenesis and, finally, new data on Myc functions in differentiation, apoptosis and cell growth, using Drosophila and mice as model systems.

While many questions on Myc functions and Myc target genes are still to be resolved, the workshop succeeded in enriching our view of the problems and setting the scenario for future developments. Some of the key issues that arose at the workshop were as follows:

1. Mechanisms of Myc activation and repression and the relative importance of activation vs. repression in Myc function.
2. Functional differences between normal and deregulated Myc expression/overexpression.

3. Criteria for defining Myc and Mad target genes and strategies for relating target gene function to Myc and Mad biological effects.
4. Relationship between Myc's roles in cell proliferation, growth, and apoptosis.

We would like to thank the speakers for their presentations and for open and comprehensive discussions. We are also grateful to Juan March Foundation for their excellent support and organization of the workshop.

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Regulation of Messenger RNA Processing

Organized by
W. Keller, J. Ortín and J. Valcárcel

(2-4 October)

Co-sponsored by the European Molecular Biology Organization

Gene expression starts with the synthesis of primary transcripts (pre-mRNAs) from DNA. Pre-mRNAs undergo extensive modifications in the nucleus before they can be transported to the cytoplasm and be translated into proteins. These modifications, collectively known as RNA processing, include addition of a methylated nucleotide at the 5' end (capping), a polyadenylate tail at the 3' end (polyadenylation), removal of internal sequences (splicing) and changes in the nucleotide sequence (editing).

Studies on the regulation of gene expression are essential to understand cell differentiation and development. The extent to which this regulation occurs at the level of messenger RNA processing, however, has only recently become fully recognized. Tissue-specific splicing, polyadenylation and editing are now phenomenologically well established as modes of gene regulation at the basis of key processes, from sex determination in Drosophila to maturation of the immune response, from fertilization to neural development. They are also at the origins of disease, from the control of blood cholesterol transport to viral pathogenesis to metastatic transformation of primary tumors. This wealth of biological implications has attracted the interest of many researchers to the molecular and cellular mechanisms underlying both the basic processes responsible for RNA modification and their key regulatory steps.

The Workshop sponsored by the Fundación Juan March and EMBO brought together leading world experts in the regulation of RNA processing to discuss common denominators between the control mechanisms operating in different processing steps, different organisms and biological phenomena. Many stimulating talks emphasized three main themes. First, the prevalence of mutual influences and intimate connections between different processing events, between these and transcription and nucleo-cytoplasmic transport, and between signal transduction pathways and processing factors. Second, many contributions illustrated how every processing step, even the apparently most simple, is accomplished by complex machines composed of multiple subunits. The roles of the different subunits are often enigmatic, presumably involved in regulation of the function of the complex. As an illustrative example, none of the multiple protein components of the factors responsible for the endonucleolytic cleavage that occurs at the 3' end of most RNA polymerase II transcripts has the sequence features of an endonuclease. Will the catalytic activity be contributed by several subunits ? Will it be contributed by the RNA itself, perhaps conformationally activated by interactions within the complex ?

The third common thread was the realization that the different members of families of proteins, their isoforms and postranslational modifications can provide unique properties to regulatory assemblies, and therefore their precise composition and distribution of cis-acting elements can generate combinatorial complexity able to account for the design of gene expression in space and time.

Hopefully the participants left with renewed appreciation for the complexity and regulatory potential of RNA processing and stimulated to keep deciphering its pervading logic and beauty. There is no doubt that the peaceful and comfortable setting that the Fundación Juan March provided, together the helpfulness of its personnel, significantly contributed to achieve these goals.

W. Keller, J. Ortín and J. Valcárcel

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Genetic Factors that Control Cell Birth, Cell Allocation and Migration in the Developing Forebrain

Organized by
P. Rakic, E. Soriano and A. Álvarez-Buylla

(16-18 October)

During mammalian evolution the cerebral cortex has undergone a thousand-fold increase in surface area and a remarkable elaboration of cytoarchitectonic areas subserving distinct functions. Execution of these functions range from sensory perception and motor control to cognition and volition, depending on the precise connections with sub-cortical structures within as well as between various areas. Before these connections are established, within each species, an appropriate number of neurons will be generated in the proliferative centers and allotment of postmitotic neurons transported to their proper laminar and areal positions. If these fundamental cellular events are not properly carried out, all subsequent steps, including the formation of connections are jeopardized, resulting in serious brain malformations.

The main goal of this meeting was to present and discuss the latest information on the control of cell production, allocation and migration in the forebrain and how this impacts formation of cortical areas. These early events set up the species-specific framework for subsequent formation of neuronal connections and synaptogenesis in the organ that is the major target of evolutionary expansion and the principle site of human mental capacity and skills. Recent advances in methods of molecular and cell biology have enabled identification of specific genes and signaling molecules that regulate production, differentiation and communication between heterogeneous classes of cells in the embryonic germinal zones of the telencephalon. These genes determine the number, identity and vulnerability of neurons before they arrive at their final destinations. However, it is also important to "climb back" from the whole animal to molecules and genes in order to develop rodent and possibly primate models of cortical dysgenesis that mimic specific genetic or acquired cortical disorders.

The participants of this meeting shared a common interest in the quest for how the proper number and mixing of progenitors dedicated to specific phenotypes and functions is achieved within the proliferative zones and primordial cortical plate. They are engaged in research at different levels of analysis, from anatomical to molecular and from *in vitro* to *in vivo* model systems with a goal of placing the identified genes and transcription factors within a mechanistic perspective.

The meeting was subdivided into 4 topics that focus on the main events: 1) cell proliferation and elimination; 2) migration and settling; 3) allocation; and, 4) deficits of cell production and migration, including human congenital malformations. Since these themes are intimately related and overlapping, many of the participants contributed more than a single topic, which provided a rich environment for discussion.

Several basic principles as well as the large number of genes underlying early cortical development were discussed. Progress has been made in illuminating mechanisms and identifying genes that control the size of the cerebral cortex through regulation of proliferation and cell death. Specific cell types appear to be generated in distinct locations within the telencephalon. Thus, cells from the perventricular zones primarily migrate radially and generate the characteristic laminar structure of the cerebral cortex as well as the positional information for areal specification. On the other hand, tangential migration introduces a subclass of interneurons that are generated in the ganglionic eminence but populate the cerebral cortex; the distinguishing molecules between these classes of neurons are being identified. Several of the participants demonstrated the considerable degree of regionalization of the cerebral cortex before the arrival

of thalamic afferents and suggested the existence of several genes and transcription factors that may be involved in the determination of areal specification. Although input from the thalamus appears to have little influence on the initial regionalization of the cortex, thalamocortical afferents are essential for its proper maturation. The meeting was concluded by discussing the implications of this research for understanding the mechanisms of evolutionary expansion of cortical size as well as pathogenesis of cortical disorders.

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Chaperonins: Structure and Function

Organized by
W. Baumeister, J. L. Carrascosa and J. M. Valpuesta

(6-8 November)

The correct folding of proteins is a cellular process of paramount importance that is far from being completely understood. As first stated by Anfinsen several decades ago, the information necessary for the proper folding of a protein is encoded in its own amino acid sequence. Nevertheless, folding of proteins in the cell is faced with problems associated with the enormous macromolecule concentration, with the intermembrane transport of proteins or with stress situations. To cope with these problems, the organisms have generated a series of proteins, termed molecular chaperones, which assist in the folding of denatured polypeptides. Besides this fundamental role, molecular chaperones are also involved in a variety of cellular processes, ranging from prion-related protection, immunosuppressive function and anti-tumor immunization. Among this ever increasing family of proteins, the best characterised members are the chaperonins, molecular chaperones with a molar mass around 60 kDa. that are assembled into multimeric complexes composed of two rings, each one enclosing a cavity where folding takes place.

The chaperonins have been classified in two groups: the first one contains the chaperonins from eubacteria like GroEL from *E. coli* or those found in eukaryotic organelles (such as the mitochondrial Hsp60 or the Rubisco binding protein from chloroplasts), while Group II encloses all chaperonins from archaeobacteria and the eukaryotic cytosolic chaperonin (CCT). Both types of chaperonins share a similar morphology but they have differences both in structure and function. Some of the differences are related to the degree of oligomerization as well as the presence or absence of a cochaperonin that assists the chaperonin in its functional cycle.

This workshop has assembled scientists working in different aspects in the field of chaperonins, and it has provided a good opportunity to review some of the most exciting aspects of both structure and function of this family of proteins. Reports on the Group I chaperonins have been focused on GroEL. Several talks have dealt with detailed biochemical analysis of the folding activity of GroEL. New kinetic data have allowed to further characterize the allosteric mechanism governing the GroEL folding cycle. The discussion of the detailed structured of GroEL and its complex with GroES, obtained by X-ray diffraction, together with the electron microscopy reconstructions of different conformers, has shed new light on the mechanisms underlying the interaction with the substrate. The fine structural analysis of GroEL mutants, together with the possibility of kinetic simulations, seems to be a very promising avenue for further analysis of the subtle details of the folding process.

The talks presented in the workshop have provided a boost in the field of Group II chaperonins. The great effort invested lately in this type of chaperonins has generated an exciting combination of structural, biochemical and genetic data that have revealed a more complex picture and different function and mechanism than those observed for GroEL. Atomic information obtained for the thermosome and several co-factors of CCT was presented, as well as electron microscopy three-dimensional reconstructions of the thermosome and the cytosolic chaperonin. The three-dimensional structures of substrate-bound CCT, and the structural transitions driven by the binding and hydrolysis of ATP were also discussed. These data, combined with detailed analysis using biochemical and mutational methods, have allowed to outline the basic features of the CCT functional cycle. The discussions during the workshop

highlighted the different roles of every chaperonin type in the folding of either non-specific or specific proteins, and revealed exciting hypothesis on the evolution of these molecular machines, along with their substrates, during the generation of the different phila.

José M. Valpuesta, José L. Carrascosa and Wolfgang Baumeister

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Comparison of the Mechanisms of Cellular Vesicle and Viral Membrane Fusion

Organized by
J. J. Skehel and J. A. Melero

(27-29 November)

The fusion of membranes is an event shared by many biological processes, such as oocyte fertilisation, compartmentalisation, endocytosis, secretion synaptic transmission and entry of enveloped virus into cells. Whereas fusion of two lipid bilayers is the common ground in all these processes, the promotion of membrane fusion and the regulation of membrane mixing has specific characteristics In each biological system. In some processes, such as vesicle fusion, there is reversibility of the fusion event and recycling of the membranes. In others, such as virus and cell membrane fusion, disassembly of the membranes does not occur. Despite these differences, understanding the molecular mechanism of membrane fusion in a given process may provide important clues for other biological systems. For this reason, the Workshop sponsored by the Fundación Juan March brought together leading world experts with different backgrounds to discuss the mechanisms of membrane fusion.

Several presentations faced the mechanism of viral and cell membrane fusion at the initial stages of the infectious cycle in different enveloped viruses. In every case, a particular glycoprotein of the viral membrane appears to promote membrane fusion by inserting, through hydrophobic sequences, into the target membranes. This event requires structural reorganisations of the viral glycoproteins that, in some cases, are triggered by a drop in the pH of the vesicles through which the virus particle is endocytosed. In other cases, fusion of the virus and cell membranes occurs at the cell surface and the triggering event for fusion seems to be mediated by the interaction of the attachment proteins of the respective viruses with specific cell receptors.

Concurrently with the rearrangements of viral glycoproteins and their insertion into the cell membranes, there is an apposition of the two membranes that is proposed to favour, first, the interchange of lipid molecules between the outer leaflets (hemifusion) and, later on, mixing of the two lipid bilayers. Completion of membrane fusion may require the formation of multiprotein complexes of the viral glycoproteins, once inserted into the target membranes.

Similar to the process of viral membrane fusion, apposition of vesicle and target membranes is an indispensable intermediate step for membrane fusion within cells. In this process, a complex is assembled between proteins inserted in the vesicle and target membranes. Generally, the formation of such complexes involves refolding of the proteins and formation of intermolecular helical bundles that bring the two membranes into close proximity. The interaction of proteins present in the surface of vesicle and target membranes is highly specific, orchestrating the fusion of membranes within the cell. Other proteins inserted into the vesicle membranes or interacting with them *in vivo* contribute to generate the chemical energy needed for membrane fusion and to regulate the reversibility and specificity of the process.

The lipid composition and the disposition of lipid molecules in the membranes are also important factors that contribute to the fusion process. This was highlighted by the results obtained with simplified model systems such as phospholipid vesicles treated with polyethyleneglycol or phospholipases. It is possible that fusion of natural membranes also requires alteration of the lipid composition at the sites where fusion pores are formed.

The unquestionable success of the meeting was greatly due to the kind hospitality of the Fundación Juan March and the efficient work of its personnel. The informal atmosphere of the meeting, promoted by our hosts, favoured lengthy discussions that crystallised in a few general ideas. Hopefully these ideas will keep all of us busy in our respective places for the next few years.

J. J. Skehel and J. A. Melero

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Molecular Approaches to Tuberculosis

Organized by
B. Gicquel and C. Martín

(11-13 December)

More than one hundred years after the discovery of *Mycobacterium tuberculosis* as the etiological agent of tuberculosis by Robert Koch, tuberculosis is still today one of the leading causes of death worldwide associated in developing countries to AIDS and with alarming increasing levels of drug-resistance.

Differently to other human pathogens, *M. tuberculosis* use a slow growing strategy. A third of the world's population has latent infection. The resurgence of concern about tuberculosis has resulted in a better understanding of parasite-host interactions.

Recent advances in the molecular biology and genetics of mycobacteria have resulted in the development of genetic tools for the manipulation of tubercle bacilli and provided the complete sequence of its genome. Cell biology contributes today to the understanding of the entry of bacteria through specific receptors, survival, phagosome trafficking and activation of signal transduction pathways.

The participants in this remarkably exciting workshop represented an international group of prominent investigators in tuberculosis with diverse interests, backgrounds, research strategies and viewpoints. The workshop has succeeded in updating several topics in tuberculosis fields of genetics and genomics; mycobacterial resistance; *M. tuberculosis* as an intracellular pathogen; tuberculosis immunity and new vaccines and futures perspectives.

The Juan March Institute has provided an excellent forum for the discussion of the recent molecular advances in tuberculosis research that could contribute to the future control of tuberculosis in XXI century.

B. Gicquel and C. Martín

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2000 Fellowships

2000 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 2000, 75 of these fellowships were awarded to participants in 13 different meetings. Among these, 31 fellowships were granted to scientists working in Spain, and 44 to scientists working abroad.

Sessions Open to the Public

In connection with some workshops, prominent invited speakers have given additional lectures in sessions open to the public. In 2000, these were as follows:

During the workshop on **Control of signalling by protein phosphorylation** (13-15 March):

- **ERIC F. WIESCHAUS**
  Howard Hughes Medical Institute
  Princeton University
  Princeton, NJ. (USA)
  1995 Nobel Prize in Medicine

  The genetics of morphological change at the *Drosophila* midblastula transition

Introduced by: **Flora de Pablo**
Centro de Investigaciones Biológicas
Madrid (Spain)

During the workshop on **Integration of transcriptional regulation and chromatin structure** (10-12 April):

- **ROBERT TJIAN**
  Howard Hughes Medical Institute
  University of California
  Berkeley, CA. (USA)

  Intricacies and complexities of the macromolecular machine that decodes the human genome

Introduced by: **James T. Kadonaga**
University of California
San Diego, CA. (USA)

During the workshop on **Tumor suppressor networks** (8-10 May):

- **JOAN MASSAGUÉ**
  Howard Hughes Medical Institute
  Memorial Sloan-Kettering Cancer Center
  New York, NY. (USA)

  How cells read growth factor signals

Introduced by: **Manuel Serrano**
Centro Nacional de Biotecnología
Madrid (Spain)

During the workshop on **Exocytosis and the vesicle cycle** (22-24 May):

ERWIN NEHER
Max- Planck-Institut für biophysikalische Chemie
Department of Membrane Biophysics
Göttingen (Germany)
1991 Nobel Prize in Medicine

Light as a tool to study exocytosis and synaptic plasticity

Introduced by: **Guillermo Álvarez de Toledo**
Facultad de Medicina
Universidad de Sevilla (Spain)

Reviews in Scientific Journals

During 2000, the meetings organized by the Centre have been reviewed in the following articles:

- Waksman, G., Lanka, E. and Carazo, J.-M. (2000). Helicases as nucleic acid unwinding machines. **Nature Structural Biology 7 (1): 20-22.** (On the workshop on *Helicases as molecular motors in nucleic acid strand separation*, held in November 1999).

- Wise, R. A. (2000). Addiction becomes a brain disease. **Neuron 26: 27-33.** (On the workshop on *The neural mechanisms of addiction*, held in December 1999).

- Thomas, G., de Pablo, F., Schlessinger, J. and Moscat, J. (2000). The ins and outs of protein phosphorylation. **EMBO Reports 1 (1): 11-15.** (On the workshop on *Control of signalling by protein phosphorylation*, held in March 2000).

- Jones, R., Harberd, N. and Kamiya, Y. (2000). Giberellins 2000. **Trends in Plant Science 5 (8): 320-1.** (On the workshop on *Biochemistry and molecular biology of gibberellins*, held in March 2000).

- Jones, K. A. and Kadonaga, J. T. (2000). Exploring the transcription-chromatin interface. **Genes and Development 14: 1992-1996.** (On the workshop on *Integration of transcriptional regulation and chromatin structure*, held in April 2000).

- Serrano, M. and Massagué, J. (2000). Networks of tumor suppressors. **EMBO Reports 1 (2): 115-119.** (On the workshop on *Tumor suppressor networks*, held in May 2000).

- Burgoyne, R. D. and Alvarez de Toledo, G. (2000). Fusion proteins and fusion pores. **EMBO Reports 1 (4): 304-307.** (On the workshop on *Regulated exocytosis and the vesicle cycle*, held in May 2000).

- Matus, A. and Shepherd, G. M (2000). The millennium of the dendrite? **Neuron 27: 431-434.** (On the workshop on *Dendrites*, held in June 2000).

- Tollervey, D. and Caceres, J. F. (2000). RNA processing marches on. **Cell 103: 703-709.** (On the workshop on *Regulation of messenger RNA processing*, held in October 2000).

Editors of the following major scientific journals have participated in different meetings of the Centre during 2000: **Cell** (seven meetings); **Science** (four meetings); **Neuron** (three meetings); **Nature** (two meetings);**Nature Cell Biology** (two meetings); **Nature Reviews Neuroscience** (two meetings); **Nature Reviews Molecular Cell Biology**; **Nature Neuroscience**; **Genes and Development**.

2001 Meetings Schedule

CENTRE FOR INTERNATIONAL MEETINGS ON BIOLOGY
2001 MEETINGS SCHEDULE

Date	Meeting Subject	Organizers
12-14 February	**Pumps, Channels and Transporters: Structure and Function**	**D.R. Madden.** Max-Planck-Institute for Medical Research. Heidelberg **W. Kühlbrandt.** Max-Plank-Institute for Biophysics. Frankfurt. **R. Serrano.** Instituto de Biología Molecular y Celular de Plantas. Valencia.
26-28 February	**Common Molecules in Development and Carcinogenesis**	**M. Takeichi.** Kyoto University. **M.A. Nieto.** Instituto Cajal. Madrid.
12-14 March	**Structural Genomics and Bioinformatics**	**B. Honig.** Columbia University. New York. **B. Rost.** Columbia University. New York. **A. Valencia.** Centro Nacional de Biotecnología. Madrid.
2-4 April	**Mechanisms of DNA-Bound Proteins in Prokaryotes**	**R. Schleif.** Johns Hopkins University. Baltimore. **M. Coll.** Centro de Investigación y Desarrollo. Barcelona. **G. del Solar.** Centro de Investigaciones Biológicas. Madrid.
7-9 May	**Regulation of Protein Function by Nitric Oxide**	**J.S. Stamler.** Duke University Medical Center. Durham. **J.M. Mato.** Facultad de Medicina. Universidad de Navarra. Pamplona. **S. Lamas.** Centro de Investigaciones Biológicas. Madrid.
21-23 May	**The Regulation of Chromatin Functions**	**V. Corces.** Johns Hopkins University. Baltimore. **T. Kouzarides.** Wellcome/CRC Institute. Cambridge **C. Peterson.** University of Massachusetts. Worcester. **F. Azorín.** Instituto de Biología Molecular. Barcelona.
4-6 June	**Left-Right Asymmetry**	**C.J. Tabin.** Harvard Medical School. Boston. **J.C. Izpisúa Belmonte.** The Salk Institute for Biological Studies. La Jolla.
18-20 June	**Neural Prepatterning and Specification**	**K.G. Storey.** University of Dundee. **J. Modolell.** Centro de Biología Molecular "Severo Ochoa". Madrid.
8-10 October	**Signalling at the Growth Cone**	**E. Macagno.** Columbia University. New York. **P. Bovolenta.** Instituto Cajal. Madrid. **A. Ferrús.** Instituto Cajal. Madrid
22-24 October	**Molecular Basis of Ionic Homeostasis and Salt Tolerance in Plants**	**E. Blumwald.** University of Toronto. **A. Rodríguez-Navarro.** E.T.S. de Ingenieros Agrónomos. Madrid.
12-14 November	**Cross Talk Between Cell Division Cycle and Development in Plants**	**V. Sundaresan.** Institute of Molecular Agrobiology. Singapore. **C. Gutiérrez.** Centro de Biología Molecular "Severo Ochoa". Madrid.
3-5 December	**Molecular Basis of Human Congenital Lymphocyte Disorders**	**H.D. Ochs.** University of Washington. Seattle. **J.R. Regueiro.** Facultad de Medicina. Universidad Complutense. Madrid.
17-19 December	**Genomic vs Non-Genomic Steroid Actions: Encountered or Unified Views**	**M.G. Parker.** Imperial Cancer Research Fund. London. **M.A. Valverde.** Universitat Pompeu Fabra. Barcelona.

Index of Personal Names

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