Instituto Juan March
de Estudios e Investigaciones

132 CENTRO DE REUNIONES
INTERNACIONALES SOBRE BIOLOGÍA

2001
Annual Report

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

Contraria sunt complementa.

(Niels Bohr's coat of arms)

INSTITUTO JUAN MARCH DE ESTUDIOS E INVESTIGACIONES

CENTRE FOR INTERNATIONAL MEETINGS ON BIOLOGY

2001 ANNUAL REPORT

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FOREWORD

This publication covers the activities of the Centre for International Meetings on Biology during the year 2001. 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 2001 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, 255 speakers were invited to the meetings during this year, and 388 participants were chosen from among 587 applications received.

13 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 second consecutive year in 2001, as described in the following pages.

A short notice is given on reviews published during 2001 in scientific journals regarding meetings organized by the Centre.

The schedule of meetings to take place in 2002 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 2001 the Scientific Council of the Centre comprised 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
Göttingen (Germany)

Margarita Salas
Centro de Biología Molecular. "Severo Ochoa"
CSIC – Universidad Autónoma de Madrid (Spain)

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 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 September 2000 to Prof. José López-Barneo (School of Medicine, University of Seville, Spain). In November 2001 it was awarded for the second time to Dr. Jorge Moscat (Centro de Biología Molecular "Severo Ochoa", 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 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.

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.

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.

Pumps, Channels and Transporters: Structure and Function

Organized by
D.R. Madden, W. Kühlbrandt and R. Serrano

(12-14 February)

Co-sponsored by

EMBO **EUROPEAN MOLECULAR BIOLOGY ORGANIZATION**

Membrane proteins are implicated in the most basic physiological functions, such as nerve signalling, learning and memory, nutrient uptake, energy conversion and muscle contraction. Correspondingly, their malfunction has been linked to a wide variety of pathologies. Furthermore, determination of membrane protein structures will be an essential component of efforts to extract meaning from the human genome: approximately 30% of the new proteins discovered are predicted to be membrane bound. Yet despite their fundamental importance, membrane proteins generally remain refractory to structural characterization, and thus to a molecular understanding of their function. The difficulties begin with the production of sufficient quantities of protein for structural studies and continue through crystallization and structure determination, all of which are particularly difficult for membrane proteins. As a result, only a comparatively small number of structures have been determined from this class of proteins, predominantly from those that are naturally abundant and generally involving considerable scientific and financial resources. Nevertheless, a number of recent developments provide hope that membrane protein structure determination will accelerate, including expression, refolding as well as the two-and three-dimensional crystallization of several new membrane proteins. Coupled with molecular biological analysis of the function and regulation of such proteins, the new structural information could stimulate a much deeper understanding of their modes of action. By their nature, membrane proteins are involved in transmitting molecules or signals across lipid bilayers. The theme of transport and transporters thus provides a natural motif running throughout the program.

The goal of this workshop was two-fold: to provide an overview of the state-of-the-art in membrane protein expression, purification and crystallization, and to review the current knowledge of membrane protein structure and function. It thus included both structural presentations and technical talks, protein biochemistry and structure determination, and talks aimed at interpreting structural data in functional terms. Significant advances have been made recently in the analysis and prediction of transmembrane protein structure and function on the basis of sequence information now available from the numerous genome projects. New techniques and systems for the overexpression and two- and three-dimensional crystallization offer the prospect of direct structural and biochemical information for functionally important but previously inaccessible membrane proteins.

Channel proteins permit the passive, but regulated flow of ions and metabolites across membranes. In some cases, such channels are constitutively open, raising questions of selectivity that can be investigated, for example, in the comparison of the bacterial aquaporins and glycerol channels. In others, channels are opened or "gated" by external stimuli, such as transmembrane voltage changes or neurotransmitter binding. The conformational basis for such coupling is being investigated, among others, for the glutamate receptor ion channels responsible for excitatory synaptic signalling in the brain. Finally, both gating and flux of such channels can be modulated by protein modifications or by complex interactions between the channel and its permeant.

Pumps and transporters are involved in the active translocation of substrates across the membrane, building up electrochemical gradients or moving metabolites to the appropriate compartment in the cell. The source of energy for translocation can be provided by light, as in

bacteriorhodopsin or photosynthetic proteins. It can also be chemical, as for the P-type ATPases or drug resistance proteins. The advent of structural data for such systems means that the conformational basis of such reactions can now be investigated. It can also be approached for proteins that exploit electrochemical gradients, such as secondary ion transporters or the ATP synthase.

The number of recent structures presented and the development of several promising technical advances suggest that the coming years will provide many further insights into the molecular mechanisms of active and passive membrane transport.

Dean R. Madden, Werner Kühlbrandt and Ramón Serrano

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Common Molecules in Development and Carcinogenesis

Organized by
M. Takeichi and M.A. Nieto

(26-28 February)

Recent findings emerging from different research fields are reinforcing the idea that the same molecules and mechanisms operate during embryonic development and in the adult, under both physiological and pathological conditions.

In this workshop, held at the Juan March Foundation between February 26-28, 2001, a total of 50 scientists, including speakers, gathered to exchange ideas regarding the molecules and signalling pathways that are common to development and cancer. The link between development and tumorigenesis is stronger than ever, and may open new avenues in cancer research owing to the availability of experimentally amenable systems.

Different topics were covered in this workshop, and particular attention was paid to the molecules and signalling pathways triggering one of the key processes in morphogenesis and tumour progression: the Epithelial-Mesenchymal Transition (EMT). This process involves a dramatic change in cell phenotype by which a well-differentiated and polarised epithelial cell is converted into a mesenchymal cell with a leading edge that facilitates its migration through the extracellular matrix, and thus, the colonisation of different structures. The acquisition of this phenotype is fundamental for the formation of many tissues and organs during embryonic development and constitutes the first step in the metastatic process in tumours of epithelial origin.

Several signalling pathways have been implicated in triggering EMT. In particular, the TGF-β superfamily has been shown to induce EMT both during embryonic development, and in tumour invasion and metastasis. Members of this family participate in many different developmental processes including the control of neural induction, dorso-ventral patterning and organogenesis, and they promote the conversion of cells with an epidermoid carcinoma phenotype to a spindle morphology *in vitro*. The majority of these processes were revised at the meeting, where an additional very promising role in wound healing was presented.

Undoubtedly, adhesion molecules deserved to be paid special attention during this workshop as individual cells must adhere either to their neighbours or to the extracellular matrix around them. Amongst the main protein families that mediate these interactions are the cadherins and the integrins, and the correct functioning of these molecules is crucial both for tissue morphogenesis and tissue homeostasis in the adult. This has become even more evident when discussing the number of genes involved in adhesion, since it seems that there has been a particular expansion of these molecules in humans. They are tightly regulated, not least because their malfunctioning produces serious consequences including embryonic lethality and the endowment of neo-plastic properties to tumour cells. Indeed, E-cadherin is thought to be an invasion-suppressor gene since its loss is considered a marker of poor clinical outcome. As the loss of E-cadherin is concomitant with the onset of EMT, it is extremely important to understand the mechanisms that regulate cadherin expression. Recently, members of the Snail family of transcription factors that repress cadherin expression have been implicated in EMT, both in different embryonic regions during development and during tumour progression. Indeed, results from several groups have shown that the direct repression of E-cadherin transcription is sufficient to trigger EMT. During the meeting we had the opportunity to

discuss the role of these transcription factors in different systems and to learn about other transcription factors also involved in the regulation of cadherin expression.

Another molecule that occupies a central position in the regulation of cell adhesion, cell growth and tumorigenesis is β-catenin. Apart from its well-known role in cadherin-mediated adhesion processes, recent studies have established its role in a novel signal transduction pathway initiated by Wnt growth factors. This pathway is crucial for embryonic patterning and cell fate determination both in vertebrates and invertebrates through the control of gene transcription. Moreover, the association of β-catenin with the tumour suppressor gene APC reveals a role for this pathway in tumorigenesis. The study in different systems and the availability of several mouse models is helping to understand the important equilibrium between the diverse functions of β-catenin in a particular cell context.

Finally, a theme common to embryonic cell migration and neo-plastic progression is the production of extracellular matrix hydrolytic enzymes. Among them, the metalloproteinases attracted much attention, since a positive correlation between their expression and the invasive potential of tumours, both *in vitro* and *in vivo*, has been found.

The use of different experimental systems and approaches, including transgenic and mutant animals and sophisticated screening approaches, has uncovered a whole plethora of molecules involved in the cellular signalling pathways that lead to the acquisition of normal migratory behaviour or malignant properties. We hope and believe that this meeting has contributed to enhance the communication between scientists working in these fields, and opens up new avenues of research and novel collaborations aimed at better understanding the mechanisms used by common molecules operating in both physiology and pathology processes. It should also served to emphasise the need for basic researchers and pathologists to work together in the hope of understanding the process of tumour progression, paving the way for the design of specific anti-invasive drugs.

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Structural Genomics and Bioinformatics

Organized by
B. Honig, B. Rost and A. Valencia

(12-14 March)

Large-scale sequencing is filling up the catalogue of natural proteins at a breathtaking speed. Today, we have available not just a large number of sequences, but also glimpses of the genetic inventory of entire organisms. It is widely assumed that this will improve our understanding of cells, in particular, and of life, in general. This may appear to be science-fiction; however, structural genomics – the marriage between protein structure determination and genomics – has already begun. The meeting addressed the challenges for bioinformatics resulting from structural genomics in two ways: (1) How can bioinformatics contribute to structural genomics initiatives? (2) What can bioinformatics profit from the flood of new structures?

Structure determination will be accelerated by, and will profit from genomics. Basing research and technical developments (such as drug design) on all three pillars (sequence, structure, and function) will constitute a major step forward towards a better understanding of life. Structure/function determination will benefit from genomics in two ways. (1) The mass of available sequences will facilitate quick determination of structure for most existing folds. (2) Sequences for entire organisms will help to unravel missing links in functional pathways, to explore alternative pathways, and to broaden our understanding of the principle mechanisms and evolutionary cross-links.

Over the last two years the first serious proposals for carrying out structural genomics have been accepted for funding in various countries (USA, Japan, Germany, England and France). In particular, the National Institutes of Health (NIH, USA) have funded seven pilot projects for 2000-2005. The first conclusions from these pilot projects appear to be as follows: (1) The major problem is to set up the 'machinery' for large-scale protein expression and purification. (2) The second major bottlenecks are automatic crystallisation-robots, assignment time for NMR, and accessible Synchrotron time for X-ray. (3) Target selection by bioinformatics is focused at (i) avoiding to duplicate structures for proteins similar to proteins of an already determined structure, and at (ii) dissecting proteins into structural domains before knowing the structure, to facilitate structure determination and to cover structure space. (4) The most important long-term challenge for bioinformatics is to develop methods that will exploit the wealth of experimental information produced.

The meeting has provided an overview of the status of the experimental techniques for high-throughput structure determination (X-ray crystallography, NMR, combinations of the two with other techniques). We have addressed questions such as: What are the problems and bottlenecks in large-scale protein structure resolution? Have we succeeded in solving structures on a large scale? What are the prospects?

As well as issues related with the development of theoretical methods associated with structural genomics, including protein sequence analysis (target selection; separation into families), protein structure prediction (completing the missing information) and prediction of protein function (finding missing links), the presentations addressed practical issues relating to the size of the problem (can bioinformatics cope with the flood of data?) and the interpretation of data (what are we going to learn from the huge amount of data produced?).

Finally, one of the key points of common interest is the problem relating to the integration of structural data with the other genomic information, e.g. functional experiments, expression data, sequencing, genome comparison and variation data.

During the meeting it was also possible to discuss key issues relating to the goals of structural genomics and the implications for Bioinformatics, addressing questions such as: Will the structural genomics initiatives produce a significant gain in functional information? Will this translate into functional annotation? What will the impact of the enterprise be for the advance of biology/health?

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Mechanisms of DNA-Bound Proteins in Prokaryotes

Organized by
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(2-4 April)

The replication, conservation, and utilization of the information stored on DNA molecules is performed by proteins, many of which bind to DNA as they carry out their functions. The technologies of both X-ray diffraction and NMR have dramatically improved, and at present, the structure of many DNA-binding proteins, alone and/or complexed with their DNA targets, have been solved. In most cases, however, examination of the structure alone is insufficient to reveal the most important information, that is the protein's mechanism of action. As a consequence, it has become necessary to combine the knowledge gained from structural analysis with genetic, biochemical, and biophysical studies to learn the fundamentals of the mechanisms of action of DNA-bound proteins. New avenues of research in prokaryotic systems are closest to bringing biological understanding to the atomic level. In prokaryotes, not only can biochemical and biophysical experiments readily be done, but genetics allows powerful selections to be performed for rare variants. In combination with knowledge of structure of the molecules involved, the molecular biology of prokaryotic systems is currently well poised to join structure with function and to begin bio-atomic engineering.

The Workshop on "Mechanisms of DNA-bound proteins in prokaryotes" was an important effort to bring together the areas of structural and mechanistic studies on DNA-bound proteins. The main outcome of the Workshop was a thorough discussion on the present knowledge of this field. The final aim was directed towards a comprehensive understanding of the mechanism of action of these proteins, to which a general discussion and final summing-up talk were devoted. Experts from around the world met with their Spanish counterparts for several days of intense interchange under nearly ideal conditions. The meeting topic recognizes the fact that the pursuit of fundamental understandings and basic operating principles in a variety of diverse biological systems is converging on questions of how proteins function. While we all believe that structure determines function, increasingly, as the 14,000 plus structures have been deposited in the protein data bank, it has become clear that for the majority of the structures, the structure does not advertise function, or more properly, activity. In reality, the relationship between protein structure and activity turns out to be very complicated and subtle, similar to the obscurity of an undocumented computer code. Because much of the excitement in biological sciences over the past 30 years has centered around nucleic acids, the focus of the meeting was the mechanisms employed by DNA-bound proteins. The meeting included topics in the areas of general and site-specific recombination, replication, transcription, gene regulation, and structure determination. Thus, we learned of mechanistic analyses on DNA helicases and how they may work. DNA replication in chromosomes or extrachromosomal elements were addressed, as well as the role of proteins involved in recombination acting as molecular motors. Site-specific proteins-mediated recombination is an exciting field in which the role of the so-called "auxiliary" proteins seem to play a regulatory role. Bacteriophages are an excellent example not only for replication studies but also for structure-based studies of polymerases, on which unexpected complexity was shown, although unifying principles affecting DNA and RNA polymerases have been observed. Further, bacteriophages were an excellent example on resolution of structures in which toroidal rings (constituted by protein subunits) are generated and on how these structures may explain the mechanisms by which the phage DNA is packaged. An important and new concept has been developed here and it relates with the flexibility in protein structure and how such a flexibility may allow activity on substrates of diverse structure. Concerning the mechanisms that govern transcription, an important finding was the observation of the remarkable flexibility and adaptability of the interactions between RNA polymerase and the proteins that regulate RNA

polymerase activity. Furthermore, proteins that control transcription initiation play a crucial role of which examples of surprising complexity were reported.

A number of general principles were made ever more apparent at the meeting. One is that Nature is complicated. That is, even for processes that we might expect to be simple, evolutionary pressures for optimization have added multiple refinements that complicate the activity. Examples of this "gratuitous" complexity were found in virtually every area covered. Notable is the variety of ways or mechanisms found in the regulation of different genes. Another principle made apparent was that it is hard to answer mechanistic questions. In a few cases (like toroidal clamps that hold proteins near DNA), the structure of a protein explains its activity, but in most cases, difficult and ingenious experiments must be used to learn even conceptually simple facts about mechanism. Much work was required to demonstrate that a helicase functions as a dimer or to find the residues of RNA polymerase that interact with transcription regulators. A recurring concept in the action of proteins was the role of rigidity or flexibility in providing or preventing energy differences or energy barriers in a process important to a protein´s function. In addition to endonuclease VII, rigidity was seen to be important to the processivity of T7 DNA polymerase, the mechanism of AraC action, the mechanism of lambda phage in protein action, and the behavior of phage 434 repressor protein. In some cases, the biologically relevant energy differences are so small that a significant change in the expression rate of a protein can in principle be achieved by a slight change in the strength of an RNA polymerase-regulator protein. Hence, we may never be able to deduce biological properties without explicit measurement.

A number of tools are now being employed in mechanistic studies. While often difficult to perform, fluorescence studies provided a significant amount of the data presented. Genetics has shifted from a blunt tool often used for the elimination of a protein´s activity to a highly refined and structure-based tool, and a number of mechanistic studies utilized the alteration of a specific amino acid whose choice was determined from the tertiary structure of the protein. Although much of our desire to investigate nature arises from simple curiosity, additional motivation undoubtedly results from our desires to apply our knowledge and understanding to the solution of specific problems. That is, we would like some of what we do to be useful in an engineering sense. Both from the viewpoint of curiosity and from the perspective of biological engineering, it was also notable just how little we actually know and how much more there is to learn. Absent from the meeting because of our current lack of knowledge were titles such as "Predicting DNA Binding Affinities from the Known Structure of a Protein-DNA Complex", or more ambitiously "Determining Ligand Identity and Binding Affinity from a Known Protein Structure", textbooks to the contrary, we do not seem close to "The Mechanism of General Recombination" or "Mechanisms of Site-specific Recombination". We have yet to see work that would justify the title "Mechanism of Allolactose Modulation of Lac Repressor Activity", and clearly much is needed before we are likely to see the general title "Structure-based Calculation of ...".

In summary, the meeting showed that while we know a lot, and work from many different biological areas is heading in the same direction, that of determining the mechanism of a protein´s action and of engineering relatively minor modifications to this activity, much work and creativity and the combination of multiple disciplines will be involved in future advances.

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Regulation of Protein Function by Nitric Oxide

Organized by
J.S. Stamler, J.M. Mato and S. Lamas

(7-9 May)

Nitric oxide (NO) plays important roles in the function of every organ system and participates in most complex physiological responses. The history of NO biology is one replete with examples of "one more function for that molecule". But the progress in understanding the mechanism of NO action has lagged behind—until recently. The advent of new tools and reagents, and increasing support for the concept of precise regulation of protein function by NO is providing a revised picture of the biology, one defined less by physiologic response and more by core principles of signal transduction. The theme that has emerged is of NO mediating posttranslational modification of proteins containing critical thiol or transition metal centers. The aim of this workshop was to review new developments in the area of NO and related reactive nitrogen intermediate (RNI)-mediated modification of proteins (NO signaling and host defense) as well as defects in signaling pathways that underlie the basis of disease (nitrosative stress).

S-nitrosylation, or the covalent attachment of NO groups to protein sulfhydryls, has emerged as the best characterized example of RNI-induced protein modification. Its functional relevance has been shown for multiple proteins of different classes including hemoglobin, the ryanodine receptor and caspase-3. While many other intriguing examples of function-regulating protein nitrosothiols were described, their physiological relevance remains to be shown. Interesting examples of S-nitrosylation as a regulatory step were provided by the reversible inactivation of two isoforms of methionine adenosyltransferases (MATI/III), the activation of the olfactory cyclic nucleotide-gated channel, the regulation of thioredoxin and the stimulation of methyl transferase activity.

The effect of NO on proteins governing the process of gene expression was analyzed at several levels. Human iron metabolism is regulated post-transcriptionally by the IRE/IRP protein system which is exquisitively sensitive to oxidative and nitrosative stresses. In the case of IRP-1, endogenous NO is able not only to promote RNA-binding activity but also results in a reduction of IRP-1 protein levels. Models for the regulation of gene expression by oxidative and nitrosative stresses are provided by the bacterial regulator OxyR and the transcription factors AP-1 and NF-κB. Both S-nitrosylation and oxidative modifications such as S-glutathionylation may occur. NO can also interact upstream in signal transduction pathways that couple cell surface signals to gene expression, including responses that control DNA methylation. Approaches using microarray methodology suggest that NO can control cell cycle progression by modifying critical proteins such as p53.

NO plays an important role in host defense both by eliminating infectious agents and regulating immunity. This is well exemplified by studies in Salmonella, where sustained inhibition of bacterial growth is mediated by S-nitrosoglutathione, and in the Coxackie virus model where RNI inhibit viral replication. In both of these cases critical proteins thiols may be modified through nitrosative mechanisms. A GSNO reductase that may serve a conserved function to alleviate nitrosative stress in microbes and mammals was described. NO can also interact with superoxide anion to form peroxynitrite, and the discovery of enzymes capable of metabolizing peroxynitrite and even repairing peroxynitrite-like damage point to a possible role for this species in human disease states. Nitration of tyrosine hydroxylase was described in an animal model of Parkinson disease and attributed to peroxynitrite.

Data presented at this meeting suggest that NO is a powerful regulator of cell respiration and apoptosis. Nitrosylation of mitochondrial protein targets such as cytochrome C oxidase, cytochrome C and caspases were shown to be responsible for these effects. Structural studies of model proteins such as hemoglobin and Ras provided mechanistic insights into NO-regulation of protein function. Connectivity between hemes and thiols as a means of propagating NO signals was described in studies of hemoglobin.

The atmosphere and spirit of the workshop were highly enjoyable and provided a perfect setting for scientific interaction.

Santiago Lamas, José M. Mato and Jonathan S. Stamler

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The Regulation of Chromatin Functions

Organized by
F. Azorín, V. G. Corces, T. Kouzarides and C. L.Peterson

(21-23 May)

The main goal of this workshop was to discuss the recent advances occurring in the field of chromatin structure and function. In eukaryotic nuclei, DNA is found in the form of chromatin, the basic structural matrix of chromosomes, which originates from the tight association of DNA with histones and non-histones proteins. Chromatin constitutes the actual framework for all biological processes involving DNA as a substrate (i.e., transcription, replication, recombination, repair). Learning about the structure and function of chromatin is equivalent to learn how the hereditary information is organized and used by the eukaryotic cell. The basic structural organization of chromatin is by now well understood but how it adapts to changing chromosomal conditions and functions is just beginning to be unravelled. Chromatin is not a uniform static structure. On the contrary, different chromosomal regions show a differential degree of condensation and chromatin structure is remodeled during development, cell cycle progression and in response to its various functions.

The basic building blocks of chromatin are nucleosome core particles. At its simplest level, the chromatin structure of all eukaryotes is organized as long arrays of these histone-DNA complexes. Over the past few years information gleaned from the high resolution crystal structures of the histone octamer and of the nucleosome core particle have revolutionized our understanding of histone-histone interactions and DNA organization within the nucleosome. In addition, biophysical analyses of nucleosomal arrays have provided valuable insights into the dynamic nature of chromatin, as well as leading to the identification of histone domains and histone modifications that govern the folding of nucleosomal arrays into higher order chromatin structures. Even as we learn more about how chromatin is assembled, folded, and stabilized, several new types of enzymes have been identified over the past few years that use the energy of ATP hydrolysis to disrupt chromatin structure. These "chromatin remodeling machines" are all complex, multi-subunit enzymes that appear to play key roles in gene transcription, development, and in the control of cell proliferation. Moreover, many enzymes are known to covalently modify histone tails. These include histone acetyl-transferases, deacetylases and methylases. The identification of so many enzymes raises a number of questions (a) how is their role distinct mechanistically; (b) is there regulation of enzymatic activity by, for example, post-translational modification or protein-protein interaction and (c) what is their biological function in the cell, relative to cell proliferation and differentiation. The workshop provided an up-to-date view into the mechanistic and regulatory properties that govern the in vivo and in vitro activities of several of these "chromatin remodelling and modifying enzymes".

The contribution of the structure of the chromatin fiber and the arrangement of the chromatin within the nucleus in the control of gene expression was also reviewed. The association of chromatin with specific proteins that seem to form large multiprotein complexes might alter chromatin structure and therefore transcriptional activity. A similar effect could be the result of the association of chromatin with specific RNAs, or protein-mediated interactions with homologous sites located in a different chromosome. Recently, a combination of molecular and cell biological approaches has allowed the establishment of a correlation between nuclear organization and gene expression. The composition and organization of this structural framework are beginning to emerge. Sequences involved in organizing the DNA within the nucleus have been isolated and proteins that interact with

these sequences are being characterized. A general characteristic of eukaryotic chromatin is the presence of highly condensed heterochromatic chromosomal regions. Heterochromatin regions are usually located near the centromeres and telomeres and they appear to have the same properties in nearly all plant and animal species. Despite extensive data on heterochromatin, its biological significance has remained elusive and it was only until recently that specific heterochromatin elements and functions are beginnig to be understood, mainly through the contributions of studies performed in yeasts (*S. cerevisiae* and *S. pombe*) and in *Drosophila*. Different aspects of heterochromatin structure and function were also addressed during the workshop.

Fernando Azorín

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Left-Right Asymmetry

Organized by
C.J. Tabin and J.C. Izpisúa Belmonte

(4-6 June)

A fundamental aspect of vertebrate embryogenesis are the events responsible for patterning the embryo as it develops from a single-celled fertilized egg into a complex multicellular organism. Understanding the mechanisms responsible for distinguishing the left and right sides of the embryo relative to its anterior-posterior and dorsal-ventral axes has been a main focus of research recently. Although vertebrates are essentially bilaterally symmetrical with respect to their external features, very dramatic internal asymmetries develop about their midline axis during embryogenesis. For instance, the heart, stomach and spleen are invariably on the left, while nearly all of the liver is on the right. The positioning of the internal organs with respect to the midline is highly conserved between species and is referred to as *situs solitus*. Alterations in this classic pattern may occur as a complete mirror image reversal of all of the organs (*situs inversus*), reversal of individual organs along the left-right axis (heterotaxia), or changes in normal symmetry or aberrant bilateral symmetry of a particular organ (isomerism). Except for complete *situs inversus*, the physiological consequence of laterality defects in internal organ positioning is usually severe. The series of embryonic developmental events that distinguish the left and right sides of the embryo along its anterior-posterior and dorsal-ventral axes include the direction of axial rotation, morphogenesis of individual organs, and organ placement.

The findings derived from research aimed at characterizing the molecules responsible for patterning the left-right axis of the embryo formed the basis of a workshop held at the Juan March Foundation in Madrid between June 3-6, 2001.

Current and future challenges in understanding left right asymmetry were discussed. They included the question of how the early bilateral symmetry of the embryo is broken, and how this initial asymmetry is converted into much broader domains of site specific expression that subsequently coordinate the asymmetric development of the various organ primordia. Profound insights emerged regarding the molecular mechanisms by which these broad domain of asymmetric gene expression are confined and regulated to their original side and how, as development proceeds, they subsequently translate into asymmetric organ development. The commonalities, but perhaps more importantly, the differences among animal in the mechanisms that establish left right asymmetry were thoroughly discussed.

One of the most intriguing questions discussed in the workshop concerned the relationship between the left right patterning of the visceral organs and the phenomenon of brain lateralization. Last, but no least, the etiology of human laterality disorders highlighted the ongoing synergism between humans and the various vertebrate model organisms and pointed directions to allow rapid advances in our understanding of the global mechanisms underlying left right pattern formation in vertebrates.

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Neural Prepatterning and Specification

Organized by
K.G. Storey and J. Modolell

(18-20 June)

Understanding the cellular and molecular mechanisms underlying neural specification is an important and fundamental challenge in Modern Biology. While much progress has recently been made in this area following the characterisation of neural inducing molecules, signalling pathways and transcription factors which mediate vertebrate neural specification, our understanding of the mechanisms underlying this process springs in large part from studies conducted in the fruit fly, *Drosophila melanogaster*. It is therefore important and informative to evaluate the similarities and differences manifest at both cellular and molecular levels during the generation of neural tissue in flies and vertebrates.

Recent advances in this field include the identification of multiple steps involved in defining vertebrate neural and neuronal precursors, some of which are homologous to steps identified in the fly. For instance, recent work in the fly has identified novel "pre-patterning" genes whose activity defines cell populations within which neural precursors can arise. Similar genes have now been identified in diverse vertebrates and it is currently being established whether these homologues also prefigure neural specification. Recent research in the fly also indicates that neural specification genes interact with the cell cycle machinery and the extent to which this is a universal mechanism that co-ordinates assignment of neural cell fate with patterns of cell proliferation is an important current issue. Topics of interest to be addressed at the workshop included: neural induction, neural pre-patterns, co-ordination of assignment of neural cell fate and patterns of cell proliferation, neuronal specification, evolutionary conservation of gene pathways and mechanisms.

The meeting brought together researchers investigating neural and neuronal precursor formation and activity in the fly and a variety of vertebrate embryos, including chick, frog, mouse and zebrafish. An initial emphasis was placed on understanding the role of the pre-patterning genes of the Irx family of homeo-domain transcription factors, but the meeting ranged more widely, addressing fundamental issues such as the evolution of proneural/*achaete scute* genes, early steps in vertebrate neural induction and the regulation of distinct spatial and temporal patterns of neuronal differentiation in both flies and vertebrates. Further talks addressed cell type specification within the nervous system particularly with respect to the glial cell lineage and the induction and differentiation of the vertebrate neural crest. Work on the specification and cell fate choices of neuroblasts and their progeny in the fly were presented in the final session, which also addressed the involvement of cell cycle machinery in the generation of asymmetric divisions.

A number of issues were explored during these talks and subsequent discussions. An initial premise that "prepatterning" genes prefigure proneural gene expression in flies and vertebrates was addressed. This concept is well established in the fly and some instances were identified in vertebrates, e.g. the frog neural plate. Comparison of proneural gene function in flies and vertebrates further demonstrated conserved roles in cell type specification and cell identity as well as additionally regulation of cell cycle. Notch signalling in cell fate choice (neural vs glia) emerged as a current topic and new genes regulating this pathway were described in zebra-fish, frog and fly.

The entire Workshop cannot be summarised here. It must suffice to say that the forum created by the Instituto Juan March provided a unique opportunity for researchers working on common questions, but in a wide range of organisms and embryonic neural tissues, to identify new areas of research and to draw parallels and inspiration.

Kate Storey and Juan Modolell

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Signalling at the Growth Cone

Organized by
E. R. Macagno, P. Bovolenta and A. Ferrús

(8-10 October)

The growth cone was first described by Santiago Ramón y Cajal in 1890, who coined the term to suggest a key role for this unique structure in establishing neural connectivity. The growth cone, we now know, is responsible for guiding neuronal projections to their proper targets. The growth cone achieves this goal through its capacity to integrate a large number of extra- and intracellular signals that result, through specific modifications of its cytoskeleton; in its progress through complex environments to its target. For over a century, the growth cone has fascinated many researchers, who have focused their efforts either identifying substrate derived cues and signals or on understanding the internal machinery that controls growth cone behaviour. In the last ten years, however, a number of mechanisms have been described that link substrate interactions with internal processes. In particular, surface receptors and protein complexes that integrate convergent signalling pathways have been identified, beginning to shed light on the network of mechanisms that generate the behaviour of this structure. Also, novel techniques are being applied to study the dynamics of the cytoskeletal components and of the plasma membrane. Finally, structural and functional similarities are being revealed between proteins and processes that occur in the growth cone and in other cell motility events, including mitosis. In other words, the "black box" between signal and response is now opening and revealing a fascinating content. The meeting on growth cone physiology was therefore very timely.

The first two sessions were devoted to external signalling. The optic system was thoroughly used in several organisms to review current data on molecular systems involving Ephrins, Neurotrophins, F-spondin and Sonic hedgehog. The growth cone dynamics was analyzed under novel visualization techniques that served to emphasize the role of microtubuli and their interaction with peripheral actin as molecular events subserving shape changes. Internal mechanisms were also reviewed including membrane traffic, small GTPases and Ca^{2+}. Finally, a renewed interest on the role of regulatory mechanisms of gene transcription, and mRNA translation at the growth cone became evident through the presentations in the last session.

Future studies on the growth cone demand a more quantitative approach of the implicated mechanisms. In addition, present techniques to visualize specific molecules or structures *in situ* would have to incorporate the capacity to detect and track several components in real time. However, these experimental advances require the previous framing of the key questions to be answered. At the meeting, we set up discussions focused on the identification of the major issues to be addressed experimentally. One discussion, for example, assessed whether simpler models would need to be developed to reproduce multi-protein interactions. Developing ways to approach the study of the properties of the plasma membrane *in situ* is another possible challenge. Another possibility is whether we should emulate the comprehensive approach used to elucidate the synaptic terminal, and should aim to identify the full repertoire of proteins localized at the growth cone.

Paola Bovolenta and Alberto Ferrús

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Molecular Basis of Ionic Homeostasis and Salt Tolerance in Plants

Organized by
E. Blumwald and A. Rodríguez-Navarro

(22-24 October)

Salting our agricultural lands is a 6,000 year old problem originated by extensive irrigation in semi-arid regions. This process slowly decreases the surface of productive agricultural lands and works against the increase in food production which must keep pace with population growth. Although famine in the world nowadays is originated by complex problems and not only by an insufficient production of food, there is no doubt that the gains in food production provided by the Green Revolution have reached their ceiling while the world population continues to rise. Therefore, increasing the yield of crop plants in normal soils and in less productive lands, including salinised lands, is an absolute requirement for feeding the world.

The excess of sodium chloride in the soil solution has two physicochemical effects. First, it decreases the water potential and, as a consequence, hinders the water flow from the soil solution to the xylem vessels and to the upper part of the plant. Second, the toxic ions, sodium and chloride, are taken up instead of other nutrients, inhibiting sensitive metabolic processes, and producing deficiencies in essential nutrients, mainly potassium. Plants can overcome the first stress by accumulating solutes both in cells and in the xylem sap. This decreases the osmotic potential, and restores the flow of water and the turgor pressure of the cells. The second stress must be overcome by excluding toxic ions from the cytoplasm of the plant cells, either keeping them outside the cells or confined into the vacuole.

Salt tolerance in halophytes is a complex adaptation which includes, vacuolar confinement, low sensitivity of the normally salt-sensitive metabolic processes, and use of the salt for water potential adjustments. Glycophytes are less adapted to grow in salty environments, and their low tolerance is only accounted for by salt exclusion. Most crop plants are glycophytes, many of them sensitive to sodium chloride at concentrations well below those producing an osmotic stress (ca. 200 mM NaCl), for which traditional plant breeding has produced tolerant cultivars in very few cases. Because ionic tolerance can be explained by simple physiological traits, exclusion, confinement, and intrinsic tolerance of metabolic processes, the improvement of salt tolerance of crop plants may be technically difficult but not impossible. For this improvement, genetic engineering offers the best possibilities, because the processes leading to ionic tolerance can be constructed in crop plants using genes of different species.

A major difference between traditional and genetic-engineered plant breeding is that whereas the first approach is empirical, in the second the processes giving rise to tolerance and the involved genes need to be identified at the molecular level. In other words, the second approach needs a stronger biochemical and physiological background. During the last twenty years the scientific understanding of the processes involved in plant salt tolerance has improved substantially. This includes cloning of many genes encoding sodium and potassium transporters, identification of signalling pathways governing the activity of ion transporters, the expression of other stress tolerance genes, and the development of simpler models to understand salt tolerance. The advance of the knowledge at this moment is very intense and it

is predictable that a breakthrough leading to the construction of salt tolerant glycophytes may occur soon. In fact, transgenic plants with increased salt tolerance have been constructed already.

Alonso Rodríguez-Navarro and Eduardo Blumwald

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Cross Talk Between Cell Division Cycle and Development in Plants

Organized by
V. Sundaresan and C. Gutiérrez

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Co-sponsored by

INSTITUTE OF MOLECULAR AGROBIOLOGY (Singapore)

Cell proliferation is a highly regulated process that plays a crucial role in generating the cells that make up an organism. At the cellular level, progression through the cell cycle occurs as a temporally coordinated series of events that involve the concerted action of both positive and negative signals. At the organismal level, in turn, maintenance of cell number in the body organs also requires cell renewal and, therefore production of new cells. Terminal differentiation and initiation of a particular developmental pattern implies that cells leave the cell cycle and restrict their proliferative capacity. This is frequently accompanied by activation of tissue-specific sets of genes and by repression and/or modulation of cell cycle regulatory functions.

Plants possess unique growth characteristics, developmental patterns and body architecture. These are the consequences of a number of plant-specific features: the plasticity of plant cells, which contribute to their capacity to dedifferentiate and regenerate new organs, the continuous post-embryonic body remodelling, which needs continuous proliferative potential and the lack of cell migration, among others. The past decade, and in particular the last three or four years, has witnessed a significant progress in the identification of plant cell cycle regulators and many aspects of their interactions are now becoming to be understood at the molecular level. Furthermore, the impact of cell division control on developmental pathways has concentrated the effort of many laboratories. Therefore, it is now the time to ask what are the roles played by cell cycle regulators and by the process of cell division in plant growth, morphogenesis and development. In other words, plant development and body architecture can be manipulated through the targeted action of cell cycle regulators and, conversely, developmental trends have a direct effect on the cell division process.

The topics outlined above were the subject of excellent presentations, containing a significant amount of unpublished data, as well as of active discussions throughout this workshop. The mainstream conclusion was that the interplay between the core cell cycle machinery and the developmental regulators is complex and finely regulated. Consistent with this, the initial structure of talks covering studies of cell cycle transitions, DNA replication and endoreplication, mitosis, exit to cell differentiation, hormonal effects and organ development was clearly surpassed. It was revealed that current approaches involve studies integrating studies at the molecular, cellular, physiological, genetic and developmental levels. We are facing an extremely attractive time where exciting ideas are very likely to contribute to a deeper understanding of the interplay between plant cell proliferation and development. In addition, comparison with eukaryotes where other growth and developmental strategies have evolved should prove very enlightening for a global understanding of cell proliferation and differentiation, cellular plasticity and, perhaps, neoplastic transformation.

Crisanto Gutiérrez

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Molecular Basis of Human Congenital Lymphocyte Disorders

Organized by
H. D. Ochs and J. R. Regueiro

(3-5 December)

A meeting on "Immunodeficiencies of genetic origin" was organized by Drs A Arnaiz and A Fischer on March 1995 with the support of the Juan March Foundation (booklet nº 38). Since then, an enormous progress has taken place in the field: first, the genes responsible for most congenital immunodeficiencies and for several lymphoproliferative disorders have been identified; second, a wealth of new information has accumulated on the biological role of the proteins involved, and on their particular localization within a limited number of biochemical pathways; and third, the first successful gene therapy trial in humans has been reported in immunodeficient children, raising hope for those suffering other genetic diseases including cancer.

The present meeting has reviewed this progress, concentrating on human congenital lymphocyte disorders caused by defects in the biochemical pathways involved in DNA rearrangement, regulation and repair (including Ig isotype switch and somatic hypermutation), cytokine recognition, antigen presentation and recognition, cytolysis, apoptosis, and cytoskeleton control (see table below). The defects may cripple signal transduction, DNA methylation or transcription, vesicle sorting or secretion. The molecules dedicated to those biochemical pathways (mostly proteins, but also RNA in one instance) are thus crucial either for producing immunocytes, or for initiating, maintaining or terminating immune responses, and sometimes for apparently unrelated biological functions in other cell types, such as melanocytes. These "experiments of nature", no matter how rare, have opened unexpected and exciting avenues for future research which will further increase our understanding of the immune system and, in turn, should give way for improved diagnostic and therapeutic procedures.

Hans D. Ochs and José R. Regueiro

HUMAN CONGENITAL LYMPHOCYTE DISORDERS

PROTEIN	SYNDROME
• Defects of DNA modification and repair pathways	
RAG, Artemis	T^-B^- SCID, Omenn
RNAse MRP	Cartilage-hair hypoplasia
DNA methyltransferase DNMT3B	ICF (Immune/Centromeric/Facial)
CD40L/CD40/ NEMO (IKKγ) /AID	Hyper IgM
ATM, hMRE 11, Nibrin	Ataxia Telangiectasia and AT-Like

• Defects of cytokine recognition pathways	
γc / IL-7Rα / Jak 3	Severe Combined ID (SCID)
IL-2R α	SCID
IL-12 / IFN-γ / STAT-1 network	Mycobacterial susceptibility
• Defects of antigen receptor recognition and signalling pathways	
λ5 / μ/ Igα/ BLNK (non X-linked)	Agammaglobulinemia
Btk (X-linked)	Agammaglobulinemia
CD3γ, ε, ζ, CD8α	CID (Combined immunodeficiency)
CD45	SCID
Zap 70, Lck	SCID
• Defects of antigen presentation pathways	
MHC-class I (TAP1,TAP2)	Granulomatosis, CID
MHC-class II (RFX5,AP,B, CIITA)	SCID
• Defects of cytolysis pathways	
Perforin	Hemophagocytic lymphohistiocytosis
Lyst	Chediak-Higashi
Myosin-5a / RAB27A	Griscelli
SAP or SH2D1A (X-linked)	Lymphoproliferative disease, Duncan
• Defects of apoptosis pathways	
TNFR1, cryopyrin, melvalonate kinase	Autoinflammatory/Recurrent fevers
CD95L/CD95/Caspase 10/Foxp3	Autoimmune lymphoproliferation
• Defects of cell mobility and adhesion	
WASP	Wiskott Aldrich / XLT / neutropenia
CD18/GDP-Fucose Transporter	Leukocyte adhesion deficiency

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Genomic vs Non-Genomic Steroid Actions: Encountered or Unified Views

Organized by
M.G. Parker and M.A. Valverde

(17-19 December)

Steroid hormones have been known for decades to be involved in various physiological responses. Their interaction with receptors of the steroid/thyroid family, best known as transcriptional activators, have captivated the attention of investigators since the early days of the study of their mechanism of action. More recently, over the last few years, we have witnessed a scientific revolution that has led to the identification of many novel mechanisms of action for these molecules.

The classical view of steroid action proposes that steroids modulate gene expression via their nuclear receptors. These receptors act as transcription factors regulating transcription following the recognition of hormone response elements at the DNA. The ligand-dependent modulation of transcription results in changes in protein synthesis with a time delay, typically, in the range of hours.

On the other hand, the novel mechanisms of action of steroids result in biological responses with typical characteristics as follows: i) rapid time-course (from seconds to minutes) so that the primary effect is too fast to be compatible with either RNA synthesis or protein translation; ii) they can be either dependent or independent to the presence of classical steroid receptors; iii) the extracellular membrane-delimited primary effect might be achieved by steroid conjugated to membrane-impermeable molecules and iv) the mechanism of action generally employs the generation of intracellular signals.

Interestingly, the novel findings (termed "non-genomic", or "alternative pathways") do not integrate nicely into the well established field of the genomic action of steroids (also known as "classic pathway"). The reasons for this lack of communication are difficult to understand and beyond the aim of this short introductory document. However, the fact is that as a result of this posture, no exchange of information, no constructive discussion between these 'encountered' worlds has taken place for far too long.

When planning the organization of the meeting we thought that the main interest would reside in bringing together views from both parties, "genomic *vs* non-genomic". With this purpose in mind we faced the difficult task of selecting specific areas of interest in which both classic and alternative views could be represented. Five areas were chosen: the functional localization of steroid receptors within the cell, intracellular signalling generated in response to steroids as well as the cross-talk between different signalling cascades involved in steroid function, the regulation of membrane excitability by controlling either the expression or activity of plasma membrane ion channels and analysis of the complex phenotype of different steroid receptor knock-out models.

The experiment offered a very positive response. The amount of novel information presented in the talks and the posters as well as the discussion they inspired was amazing. A constructive exchange of information between both parties took place and it was the general view that the existence of one mechanism in a particular cell is not exclusive. In this way, steroids can elicit diverse cellular effects that might depend on their concentration and their primary target. Hopefully, this unified view of steroid actions will soon be feasible and will integrate their different mechanisms of action. In the meantime, we are all expectant about

the advances taking place in the steroid actions' field, especially those related to the molccular identification of new targets, the cross-talk between different signalling cascades and the unravelling of the very complex interaction of nuclear receptors with coactivators and repressors within the cell nucleus.

Malcolm G. Parker and Miguel A. Valverde

LIST OF INVITED SPEAKERS

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2001 Fellowships

2001 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 2001, 62 of these fellowships were awarded to participants in 13 different meetings. Among these, 16 fellowships were granted to scientists working in Spain, and 46 to scientists working abroad.

Reviews in Scientific Journals

During 2001, the meetings organized by the Centre have been reviewed in the following articles:

- Special issue of **The International Journal of Developmental Biology 45 (1)** (2001), with 26 articles signed by invited speakers and participants of the meeting on *Molecular Nature of the Gastrula Organizing Center: 75 years after Spemann and Mangold*, held in May 1999.

- Special issue of the **Journal of Structural Biology 135 : 83** (2001). with 14 articles signed by invited speakers and participants of the meeting on *Chaperonins: Structure and Function,* held in November 2000.

- Aínsa, J. A., Martín, C. and Gicquel, B. (2001). Molecular approaches to tuberculosis. **Molecular Microbiology 42 (2): 561-570.** (On the workshop of the same title, held in December 2000).

- Tate, C. G. (2001). A feast of membrane protein structures in Madrid. **EMBO Reports 2 (6): 476-480.** (On the workshop on *Pumps, Channels and Transporters: Structure and Function*, held in February 2001).

- Martínez-Arias, A. (2001). Epithelial Mesenchymal Interactions in Cancer and Development. **Cell 105: 425-431**. (On the workshop on *Common Molecules in Development and Carcinogenesis*, held in February 2001).

- Stamler, J. S., Lamas, S. and Fang, F. C. (2001). Nitrosylation: The Prototypic Redox-Based Signaling Mechanism. **Cell 106: 675-683**. (On the workshop on *Regulation of Protein Function by Nitric Oxide*, held in May 2001).

- Berger, S. L. and Felsenfeld, G. (2001). Chromatin Goes Global. **Molecular Cell 8: 263-268**. (On the workshop on *The Regulation of Chromatin Functions*, held in May 2001).

- Wright, C. V. E. (2001). Mechanisms of Left-Right Asymmetry: What's Right and What's Left?. **Developmental Cell 1: 179-186**. (On the workshop on *Left-Right Asymmetry*, held in June 2001).

- Baas, P. W. and Luo, L. (2001). Signaling at the Growth Cone: The Scientific Progeny of Cajal Meet in Madrid. **Neuron 32: 981-984**. (On the workshop on *Signalling at the Growth Cone*, 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).

Editors of the following major scientific journals have participated in different meetings of the Centre during 2001: **Cell** (two meetings); **Neuron** (three meetings); **Molecular Cell** (one meeting); **Developmental Cell** (two meetings); **Nature Reviews Neuroscience** (two meetings); **Nature Reviews Cancer** (one meeting); **Science** (one meeting); **EMBO Reports** (one meeting).

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. Alcamí.** Cambridge University. Cambridge. **U.H. Koszinowski.** Max von Pettenkofer-Institut. Munich. **M. del Val.** Instituto de Salud Carlos III. Madrid.
11-13 April	**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. Ros.** Universidad de Cantabria. Santander.
22-24 April	**Regulation of Eukaryotic Genes in their Natural Chromatin Context**	**K. 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.F. 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. 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. 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. Rice.** The Rockefeller University. New York. **W. Spaan.** Leiden University. Leiden. **L. Enjuanes.** Centro Nacional de Biotecnología. Madrid.
4-6 November	**Exchange Factors Lymphocyte Disorders**	**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.

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

It has organized more than 400 *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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