

Instituto Juan March
de Estudios e Investigaciones

160 | CENTRO DE REUNIONES
INTERNACIONALES SOBRE BIOLOGÍA

2003
Annual Report

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de Estudios e Investigaciones

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Annual Report



Instituto Juan March (Madrid)

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

Instituto Juan March (Madrid)

Science is a truly international activity which breaks down barriers between the peoples of world, an objective that always has been necessary and never more so than now.

Sir Paul Nurse – From *Les Prix Nobel* 2001.

INSTITUTO JUAN MARCH DE ESTUDIOS E INVESTIGACIONES

CENTRE FOR INTERNATIONAL MEETINGS ON BIOLOGY

2003 ANNUAL REPORT

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FOREWORD

This publication covers the activities of the Centre for International Meetings on Biology during the year 2003. 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 2003 was the organization of twelve 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, 234 speakers were invited to the meetings during this year, and 370 participants were chosen from among 642 applications received.

12 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 short notice is given on reviews published during 2003 in scientific journals regarding meetings organized by the Centre.

The schedule of meetings to take place in 2004 is also offered in this report.

Instituto Juan March de Estudios e Investigaciones

THE CENTRE FOR INTERNATIONAL MEETINGS ON BIOLOGY

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

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

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

Javier Gomá is the Managing Director of the Institute.

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

SCIENTIFIC COUNCIL OF THE CENTRE

During the year 2003 the Scientific Council of the Centre comprised the following members:

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.

2003 Meetings Schedule

CENTRE FOR INTERNATIONAL MEETINGS ON BIOLOGY
2003 MEETINGS SCHEDULE

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

* This meeting took place at the Gulbenkian Foundation, Lisbon. All others were organized at the Juan March Institute, Madrid.

**Membranes, Trafficking and Signalling
during Animal Development**

Organized by
K. Simons, M. Zerial and M. González-Gaitán

(27-29 January)

One of the major questions of development is how equal cells in developing tissues differentiate into different cell types in a position-dependent manner. The generation of positional information involves a particular kind of secreted molecules, termed "morphogens", which emanate from a source, spread across the target fields and form long-range concentration gradients which provide the target cells with positional information. These receiving cells, in turn, interpret the gradient by activating target gene expression at discrete concentration thresholds. This simple model has been proven true during the development of many organs both during invertebrate and vertebrate morphogenesis. This includes dorsal/ventral patterning during early embryogenesis, limb development and neural and mesodermal patterning from flies to mammals.

Much of the success of the positional information model capitalized on the molecular identification of the distinct signaling molecules involved, many of which turned out to belong to the Wnt, Hedgehog, FGF and TGF β families of secreted ligands. In addition, downstream these secreted ligands, the signal transduction pathways, including the receptors and mediators of the cellular responses, have been characterized in much detail at the molecular and biochemical levels. These data have provided a formal picture of signaling during morphogenesis: groups of cells send signals to target cells by generating gradients and the gradients are interpreted by means of rather linear signal transduction pathways which are initiated from a homogeneous plasma membrane.

During the past years a major effort has been taken to provide morphogenetic signaling with a novel cellular dimension beyond the formal molecular pathways and the mere description of inductive mechanisms. In particular, a very intense field of research addressed the role of membranes and their dynamic trafficking during i) the dispersion of the ligands across the target tissues which are thereby distributed as gradients of concentration and ii) the transduction of the signals which occurs not just at the plasma membrane, but at distinct membrane subcompartments within the developing cells. One example is the raft model, which proposes that different molecules mediating consecutive steps along signal transduction pathways cluster in lipid microdomains, rafts, which thereby control morphogenetic signaling. Other is the recent discovery that signal transduction may occur in some cases from an endosomal, intracellular compartment rather than from the plasma membrane. In addition, a picture is emerging whereby the dispersion of ligands may not always occur by free extracellular diffusion, but would involve membrane trafficking through the target cells.

The field is a very young and successful one and implies the mutual confluence of two scientific cultures: molecular developmental genetics and molecular cell biology. The meeting put together major players from both scientific communities that have contributed importantly in recent years to this novel interface research.

During the workshop we had the chance to discuss the molecular mechanisms underlying endocytic trafficking: from endocytosis to endosomal dynamics. We learnt about how a key GTPase Dynamin works to control the fission of endocytic vesicles (Schmid) and,

downstream this event, how small GTPases of the Rab family control the motility of endocytic vesicles and their fusion with endosomes (Zerial). A major focus in trafficking is what are the molecular mechanisms controlling the motility of organelles on cytoskeletal elements: microtubules (Hirokawa) and actin (Small). The Rab proteins may play thereby a key role during development from flies (González-Gaitán) to fish (Campos). During these trafficking events, phosphoinositides play a key role (Thomer).

Endocytic trafficking is at the core of morphogenesis, controlling developmental events from asymmetric cell division (Schweisguth) to morphogenetic signaling. Endocytosis can play different roles during morphogenetic signaling depending on the signaling event considered. It can control the trafficking pathway of receptors, determining its targeting towards degradation (TGF-beta signaling, Wrana). Endocytic trafficking can also control the spreading of ligands, either by promoting its planar transcytosis (Dpp, González-Gaitán), perhaps vehicled by vesicular (argosomes, Eaton) or their degradation (Wingless, Vincent; FGF8, Brand; Hh, Guerrero). The control of the release of Hh ligands by sterol-sensing-domain proteins and trafficking was considered after the work of Ingham and Guerrero. Also, Aarole of endocytosis during the deposition of extracellular elements, such as the heparane sulfate proteoglycans was discussed (Perrimon) and its relevance during complex morphogenetic movements, such as the dorsal closure of the *Drosophila* embryo, is starting to be analysed in Martínez-Arias lab. During dorsal closure, Martínez-Arias discussed the role of the actin cytoskeleton and Wingless, a ligand that play a "catalytic" rather than instructive role.

In order to understand these events at the interphase between morphogenetic signaling and endocytic trafficking, it is key to have access to imaging technics that allow the visualization of signaling events with high temporal and spatial resolution. The work of Fraser and Bastiaens addressed this aspect. Fraser showed how sensitive imaging of espectrally overlapping fluorochromes by using a LSM META confocal microscope allows the simultaneous visualization of many molecules during development. He also proposed Fluorescence Cross-correlation as a powerful method to analyse protein-protein interactions. Bastiaens showed its very powerful and sophisticated FLIM analysis of signaling events and protein-protein interactions *in vivo*. Its work suggests a key role of peroxide species during the spreading of EGF-R signaling within a cell.

Finally we learnt the state of the art in the "raft" field. Simons presented the raft model, Parton showed how he could visualise the rafts at the EM level and Martínez-A. illustrated the role of rafts during polarization of migrating T-cells. In a challenging presentation, Kurzchalia told us about its analysis of the role of a key raft lipid, cholesterol, during development of the worm.

Kai Simons, Marino Zerial and Marcos González-Gaitán

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Synaptic Dysfunction and Schizophrenia

Organized by

P. Levitt, D. A. Lewis and J. DeFelipe

(10-12 February)

The biological substrate of disorders of cognition, emotion and thought remains a central problem of clinical and basic neuroscience. Of the diseases with the greatest negative economic and health impact on the human population, 8 of the top 10 are neuropsychiatric in nature. Indeed, improvements in the treatment and prevention of these disorders are likely to have the greatest impact on reducing disability in the populations of developed countries in the future.

Converging evidence from studies in both humans and animals indicates that dysfunctional communication between specific populations of neurons may be the principal alteration in schizophrenia, a disease that afflicts approximately 1% of the world's population. Our basic understanding of the molecular and cellular mechanisms that mediate synaptic function has never been greater, providing an opportunity to integrate basic biological concepts of synaptic chemistry and physiology with disease-related alterations in the genes and proteins that mediate these functions.

We gathered investigators, who are experts in the fields of neuropsychiatry, synaptic function and neuronal communication, to exchange information that will facilitate a more sophisticated understanding of the biological bases of schizophrenia. While these participants had not before been assembled, we believe their interactions will promote the development of novel, integrative approaches to the study of the etiopathogenesis of schizophrenia and to the identification of new targets for therapeutic interventions.

The meeting was organized around five scientific themes to address the difficult challenge of translating our understanding of the clinical disorder into fundamental biological mechanisms that underlie the etiology and pathogenesis of schizophrenia: 1) *Functional Architecture of Cortical Circuitry and Its Dysfunction in Schizophrenia*. Schizophrenia involves the dysfunction of cortical microcircuits, yet we are still at a very early stage in a detailed understanding of the very cellular and physiological components that altered by the disease. Investigators who focus their studies on the functional attributes of circuitry of the cerebral cortex and thalamus are using technically sophisticated approaches to provide remarkable details of both macro- and microcircuitry. This framework allowed the group to translate the newest concepts towards an understanding of the disrupted circuitry in schizophrenia. 2) *Neuromodulators and Their Alterations in Schizophrenia*. Both basic and clinical research has implicated abnormal functioning of neuromodulatory systems, principally the monoamines, in schizophrenia. To date, the most prevalent and effective pharmacological treatments alter the activity of these neuromodulators. Investigators reported on the use of a variety of technical strategies, to elucidate the roles for these essential neurochemical components in mediating the functions that are abnormal in schizophrenia. Participants were provided with an opportunity to probe the relationship between observations of behavioral and physiological dysfunction in animal models and the functional features of these cortical systems in the context of schizophrenia. 3) *Synaptic Function: Understanding the Pathophysiology of Schizophrenia*. Accumulating evidence suggests that the dysfunction of synapses, the major element through which neurons interact, underlies the pathophysiology that characterizes schizophrenia. Sophisticated molecular and physiological approaches by scientists at the meeting facilitated remarkable progress defining the cellular and molecular

components that are necessary to assemble and maintain mature, functional synapses. The current understanding of the presynaptic elements that control neurotransmitter release, and postsynaptic components that define the responsiveness of neurons in circuits will be placed in the context of new findings that implicate problems of the synaptic machinery in schizophrenia. 4) *Development and Plasticity: Role in the Pathogenesis of Schizophrenia*. The concept of a developmental etiology of schizophrenia was popularized during the last decade, but the theory has been difficult to crystallize from a cellular and molecular perspective of disrupted cortical circuits. In particular, the field wrestles with the polygenic and epigenetic nature of schizophrenia that is characterized by a significant temporal delay in the expression of overt symptoms of the disorder. These issues were addressed by investigators who have made seminal contributions to our understanding of synapse and receptor assembly in cerebral cortex, and the developmental and functional plasticity exhibited by these systems with particular genetic perturbations. This group was challenged with discussing the mechanisms that drive the assembly of dysfunctional circuits and the untapped potential for regulating plasticity as a strategy for intervention and treatment. 5) *Testable Models and Strategies*. The adaptive processes that occur in schizophrenia are likely to be initiated by a combination of genetic susceptibility and environmental perturbation. The issues discussed in the first four sessions provided the framework for the scientists in the last session to elaborate on new concepts of schizophrenia based on disruption of synaptic function. Testable models may provide new approaches to study the pathophysiology and to help define the etiology of the disease.

Thus, these scientists had the opportunity to integrate increasingly complex data from neuroimaging and circuit analyses, which continue to provide important details of the neural networks that are dysfunctional in schizophrenia, with the fundamental molecular and cellular processes of synaptic communication that form the basis for the disease. Rather than segregate investigators according to their specific technical approaches or broader laboratory interests, we purposely organized the sessions in which data on a key topic, extracted from studies of normal and disease states, was presented and discussed in an integrated fashion. Thus, each had broad representation and expertise. This approach facilitated the interactions among scientists with different backgrounds and perspectives, enhanced the cross-fertilization of ideas from different disciplines, and emphasized the importance and opportunities for translating fundamental discoveries in synaptic function and cortical circuitry into strategies for understanding neuropsychiatric disorders.

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Plasticity in Plant Morphogenesis

Organized by

G. Coupland, C. Fankhauser and M. A. Blázquez

(24-26 February)

A unique feature of plant development is that plants continue to produce and differentiate new organs during their whole life cycle, which contrasts with most other higher organisms. This peculiarity has resulted in two critical characteristics of plant development; first, many developmental decisions taken during post-embryonic life are subject to influence by the environment; and second, since plants cannot willingly change their location, they have developed a highly efficient strategy to adapt their architecture and physiology to changing environments.

Although we have reached a high level of description of signaling pathways, we still know very little about the mechanisms that integrate those external cues with the endogenous developmental status of the plant. This integration is extremely important since the same external stimuli are differentially interpreted in distinct organs or at different stages of development. Thus, to reach the next level of understanding, it is necessary to compare the different physiological mechanisms that allow plasticity in plant development, as well as to start building the connections between these different pathways to hopefully render a more integrated view of modulation of plant growth. This was the main goal of the workshop.

Recent progress in the study of how plants grow and differentiate –much of it presented at this meeting– is beginning to show the molecular mechanisms that underlie the tremendous plasticity in plant development. This plasticity has been achieved mainly through two strategies: first, a very sensitive machinery that perceives environmental conditions –such as light quality and intensity, daylength, temperature, and nutrients–; and second, the interweaving of multiple signaling pathways –light, hormones– that allows fine tuning of developmental programs as they proceed. How these two strategies combine with each other to allow plasticity is probably the next big question in plant development.

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Wnt Genes and Wnt Signalling

Organized by

J. F. de Celis, J. C. Izpisúa Belmonte and R. Nusse

(24-26 March)

Over the past few years, there has been a surge in interest in the function of Wnt molecules in biological processes. Wnt proteins are secreted from cells, act as short range signaling molecules and are now recognized as one of the major families of developmentally important signaling molecules. Mutations in *Wnt* genes display remarkable phenotypes in the mouse, *C. elegans* and in *Drosophila*. Among functions provided by Wnt proteins are such intriguing processes as embryonic induction, the generation of cell polarity, and the specification of cell fate. One example of the role of Wnt signaling in body plan formation is axis formation and head induction in vertebrate embryos. Several inhibitors of Wnt signaling have been discovered and when those inhibitors are over-expressed or deleted, dramatic phenotypes ensue. Wnt signaling in vertebrates also plays a role in limb outgrowth. In addition, several components of Wnt signaling are implicated in the genesis of human cancer.

Until 5-6 years ago, our knowledge of the molecular mechanism of Wnt signaling was very limited, but over the past years, several major gaps have been filled. These include the identification of cell surface receptors and a novel mechanism of relaying the signal to the cell nucleus. These insights have come from different corners of the animal kingdom and have converged on a common pathway. Wnt signal transduction proceeds through a complex series of protein interactions. Signaling is initiated by binding of the Wnt protein to cell surface receptors, which then generate a signal to downstream components. There are two kinds of receptors: Frizzled and LRP; LRP is a general receptor, while the Frizzleds are specific for certain Wnts. After activation of Dishevelled, a key event in signaling is the regulation of the GSK3 protein kinase and its substrate beta-catenin. In the absence of a Wnt signal, GSK3 phosphorylates beta-catenin, which then becomes targeted for degradation. Beta-catenin and GSK are brought together by two scaffolding proteins: APC and Axin. The binding of Wnt to its receptors initiates a cascade of events that inhibits GSK3 and ultimately results in an accumulation of beta-catenin. Together with the DNA binding protein TCF, beta-catenin activates expression of Wnt target genes.

Remarkably, by studying this pathway, researchers have been able to predict which components could play a role in human cancer, and have indeed identified mutations in Wnt signaling components in such important human tumors such as colon carcinomas and melanomas. This component, beta-catenin, is now viewed as a major human oncogene, while negative regulators of Wnt signaling, in particular Axin and APC, are important tumor suppressor genes. At the same time, these molecule serve as an example of the power of the combinatorial approach that researchers have taken in this field: the *Drosophila* counterpart of beta-catenin (called armadillo) was first found in the fly as a component of the Wnt (or wingless) pathway.

The findings on the function of Wnt signaling in various organisms underscore the importance of bringing together researchers working in different areas. It was therefore extremely useful to have a meeting at the Juan March Foundation that was entirely devoted to Wnt signaling. The meeting resulted in extensive dialogues between researchers working on flies or worms and those working on human cancer.

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**Molecular and Genetic Basis of Autoimmune
Diseases: SLE and RA**

Organized by
A. Coutinho, W. Haas and C. Martínez-A.

(7-9 April)

Co-sponsored by
INSTITUTO GULBENKIAN DE CIÊNCIA
EMBO EUROPEAN MOLECULAR BIOLOGY ORGANIZATION

Systemic lupus (SLE) and Rheumatoid Arthritis (RA) are amongst the earliest described autoimmune diseases (AID). They have been extensively studied both at the clinical and basic levels.

AID continue to increase in prevalence in the western world reaching up to 10% of the general population in some countries. For their variety, age of presentation, and chronic/debilitating clinical course, they represent a major socio-economical problem for public health. From the basic point of view, AID raise fundamental questions and offer a wealth of observations on one of the unsolved questions of modern immunology –natural tolerance to body tissues and its relationship to infections. From the medical point of view, we continue to be unable to diagnose AID before lesion of the target organ or function, and have no rational, curative therapies for diseases such as type I diabetes, rheumatoid arthritis, multiple sclerosis and systemic lupus.

In recent years, major progress in understanding the molecular basis of complex processes in cell and tissue biology have been achieved, particularly in the immune system itself and in the physiology of some of the most common target tissues for AID.

Furthermore, the advances in “genomics” have promoted a wealth of studies on the genetic basis of susceptibility/resistance to AID in human populations. Finally, a number of clinical trials have recently been initiated, testing the beneficial effects of a variety of “biology-based” therapies.

The workshop was divided in five sessions, organized from basic immune mechanisms of tolerance and autoimmunity, followed by the genetics of experimental and human SLE and RA, by the analysis of potential therapeutic targets (cytokines, chemokines and their receptors) and by novel therapeutic approaches, that included the analysis of the “biological” therapeutics (monoclonal antibodies and receptor-fusion proteins) as well as the use of stem cell treatment of AID. Therefore in this workshop scientists coming from different disciplines had the opportunity to integrate data from the clinical and therapeutic perspectives with the molecular and cellular processes that form the basis for these diseases.

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**The Dynamics of Morphogenesis: Regulation of
Cell and Tissue Movements in Development**

Organized by
C. D. Stern and M. A. Nieto

(12-14 May)

The cellular events that direct embryonic development have broadly been subdivided into three kinds of processes: fate allocation, pattern formation and morphogenesis. *Fates* are allocated to cells through a combination of their lineage history (through cytoplasmic and nuclear determinants of fate) and cell interactions ("inductions"). *Pattern formation* is the set of processes that shape a more or less uniform field of cells by assigning different fates to cells according to their position with respect to their neighbours. *Morphogenesis* is also a set of processes that generate form, but here the main driving forces are cell movements and tissue reorganizations rather than fate allocation. To illustrate the difference: an example of pattern formation is the generation of the periodic mosaic of ommatidia in the compound eye of *Drosophila*; an example of a morphogenetic process is gastrulation, where cells arrange themselves into three layers which will then behave differently.

In the last few decades considerable effort has been spent in understanding how cells are assigned different fates and how pattern formation occurs in systems like the *Drosophila* eye and the vertebrate limb. Morphogenetic processes, on the other hand, have received comparatively little attention. Genetic and molecular analyses have concentrated on elucidating *pathways*, but very little is known about *how these pathways are integrated in space and time*. Interest is now turning to try to understand the processes that orchestrate these dynamics of cell behaviour. This applies not only to embryonic development, but also to the adult and to pathological processes such as cancer, where invasive behaviour can be considered a form of morphogenesis that profoundly influences the morbidity of the disease.

This meeting aimed to explore the current status of this field, at a time when many laboratories are starting to acknowledge that no solid understanding of development or pathology can be gained until our view can also include an understanding of how gene expression and cell behaviour are integrated in space and time.

Some of the questions are:

- We already understand something about the *mechanics* of cell movement, but what signalling cues control the directed migration of cells?
- Is there directed migration, or just assortments of permissive, repulsive and attractive sites? How general a mechanism is chemotaxis?
- To date, several protein families have been implicated in signalling outside the nervous system (including FGFs, Wnts and HGF/SF), but different signalling proteins have been shown to guide axon navigation in the CNS and PNS (including netrins, semaphorins, collapsins, NOGO, Ephrins/Eph receptors, etc.). Is this a true difference? If so, how/why did different mechanisms evolve to regulate what seems like the same process in different parts of the same organisms?
- As cells migrate, they respond to local inductive/repressive cues from their neighbours which change their gene expression profiles and consequently alter their behaviour. How are the many signals they encounter integrated? For example, as prospective mesoderm cells approach the organizer region of the vertebrate embryo, they start to express organizer-specific genes, and then turn them off as they leave the region.

- his is controlled by combinations of regulatory genetic elements, and how much by the precise positioning and physical range of the inductive/repressive signals?
- How does the signalling machinery result in directional movement? Can cells really sense concentration differences between one of their sides and the other?
- Do other cellular asymmetries (such as asymmetric cell division, epithelial polarity) involve similar directional cues, which direct the migration of intracellular organelles?

How many of the signalling systems involved in development are also implicated in wound healing and cancer invasion/metastasis? Why do embryos heal their wounds without scars, while adults always scar? Why are some tumours invasive and some not?

Of course, a two-and-a-half-day meeting could not possibly answer all of these important questions. However, by focusing on the major current “model” processes (gastrulation, neural crest cell migration, germ cell migration, tubulogenesis and branching morphogenesis, epidermal wound healing and cancer), and “model organisms” (*Drosophila*, *C. elegans*, Sea Urchin, zebrafish, *Xenopus*, chick and mouse), we can obtain an inkling of the great diversity of mechanisms that exist to coordinate complex cell behaviours in time and space. Perhaps one critical conclusion that was achieved during the meeting was that biology itself is extremely diverse and that the whole concept of “model” processes and systems can lead to oversimplifications and to false generalisations. The principles of evolution and development will be best understood by exploiting the advantages of each system, and by opportunities such as this one, to exchange views and experiences between those that have been thinking very deeply about their favourite organism and biological event.

The organisers,
May 2003

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**Developmental Mechanisms in Vertebrate
Organogenesis**

Organized by
G. Oliver and M. Torres

(9-11 June)

Recent advances in cellular and molecular mechanisms underlying organ formation and tissue regeneration in vertebrates have once more positioned developmental biology at the forefront of basic science, and emphasize the relevance of the potential therapeutic application of this knowledge. Advances in stem cell biology and the dissection of molecular networks controlling cell differentiation pathways and pattern formation are the two driving forces behind these new perspectives. In this meeting we have reviewed recent advances in the field by bringing together basic scientists who are contributing significantly to these developments.

Understanding how progenitor cells differentiate and self-organize to build an organ or a tissue requires analysis of the molecular networks that control these processes. Molecular developmental biologists are dedicating considerable effort to the identification and characterization of autonomous and non-autonomous molecules governing differentiation and pattern formation. Molecules that trigger organ formation, specify cell lineages, or control the allocation of different cell phenotypes within organs are the subject of intensive analysis in many leading research groups. The identification of these molecules and mechanisms is crucial to understand normal and pathological aspects of organ development. Research in the last decade provided us with the molecular tools to start dissecting some of these processes and the subsequent functional modifications of many gene products has started to unravel some of the signaling mechanisms and pathways operating in early embryonic development. Further to this, during the last few years, emergence of new powerful technologies has revolutionized this area of research. The use of genomics, microarray analysis, conditional knock-outs and RNA interference, to name some of them, provided the scientific community with extraordinary tools to help dissect and understand vertebrate organ formation. The use of these resources in the most popular vertebrate animal models: e.g., mouse, zebrafish, and chick, produces an ever-increasing body of novel information, often difficult to integrate.

Having the opportunity of bringing together researchers working in different organs in different animal models provides an unique opportunity to compare and exchange approaches and ideas between colleagues working in this exciting field. From the early formation of the three tissue layers, to the formation and pattern of the different organs, extensive discussion over these topics strengthen the idea that although limbs, kidney, pancreas, liver, brain, teeth appear quite different in morphology, many of the genes and mechanisms involved in their development are shared by most of them. Beautiful examples of these exciting studies in normal and pathological conditions are represented here by results obtained in areas such as vascular biology, endodermal organ formation, patterning of ectodermal derivatives, differentiation and proliferation of neural and glial precursors, to name just a few.

The opportunity provided by the Juan March Organization to bring together this workshop was extremely valuable and provided the frame for highly stimulating discussions and exchange of information necessary to integrate knowledge from different fields of vertebrate organ formation.

Guillermo Oliver
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**Neuronal Degeneration and Novel Therapeutic
Approaches in Parkinson's Disease**

Organized by
C. W. Olanow, J. A. Obeso and R. Moratalla

(23-25 June)

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Dendritic Cells: Biology and Therapeutic Applications

Organized by
R. M. Steinman, I. Melero and A. L. Corbí

(6-8 October)

Dendritic cells are bone marrow-derived cells which play a pivotal role in the generation and control of natural and adaptive immunity. As professional antigen presenting cells, dendritic cells display a very high capacity to stimulate naïve T lymphocytes for the generation of primary immune responses, promoting the generation of distinct types of effector T cells which ultimately will lead to the initiation of immune responses or the induction of tolerance. Besides, dendritic cells are now recognized as main players in innate immunity, as they are capable of recognizing numerous pathogen-associated molecular patterns, and secreting cytokines which mobilize and activate macrophages and natural killer cells. Their ability to fulfil both activities probably derives from the existence of various dendritic cell subsets, all of which are included within the "dendritic cell" system that controls immunity. Research on dendritic cells has greatly expanded in the last years because of their potential application to modulate immune responses with therapeutic purposes. In this regard, and because of their functional plasticity, dendritic cells could be seen as "tools" not only to implement cell-based immunotherapies for cancer, but also to down-modulate undesired immune responses in autoimmune diseases. Based on all these ideas, numerous clinical trials are currently underway on a variety of malignancies, and results will be known in the coming years.

The aim of this meeting has been to discuss the generation of the distinct dendritic cell subsets and their precursors, their role in the generation of immune responses, T cell polarization and immune tolerance, and the feasibility of the therapeutical applications of dendritic cells. The importance of polarizing cytokines, co-stimulatory molecules and pathogen-recognition receptors on the initiation of immunity by dendritic cells was addressed in depth, and results were presented revealing the existence of functional interactions between dendritic cells with NK and regulatory T cells. The meeting also allowed ample discussion on the different approaches taken for dendritic cell-based cancer immunotherapy, and for comparison of all the currently available information and some preliminary data from ongoing trials. In this regard, some basic consensus concepts were reached which might serve as starting points for future clinical trials. As a whole, the workshop has also proven very fruitful for the exchange of ideas between laboratories involved in basic and clinically-oriented research on dendritic cells.

The organisers,
October 2003

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**Finding the Way Out: Protein Traffic
in Bacteria**

Organized by
A. P. Pugsley and V. de Lorenzo

(20-22 October)

Bacteria manage their limited amount of genetic information in an exquisite manner, exploiting all imaginable devices at the molecular, physiological and cellular levels to survive and proliferate. The containment and compartmentalisation that were essential requirements for the development of life are incompatible with the need to derive nutrient from and communicate with the environment. Containment and compartmentalisation are achieved through the creation of biological membranes based on lipids, hydrophobic molecules that form an essentially impermeable barrier between the cell (or organelle) and its surroundings. This permeability barrier is rendered selectively permeable by the introduction of proteins and protein complexes that form channels and energy-dependent transporters that permit or promote the movement of ions, solutes and even macromolecules across the membrane.

In eukaryotic cells, protein traffic occurs at several levels (into and out of organelles and out of the cell) and by a variety of different mechanisms. Although most prokaryotes do not have internal organelles, they are nevertheless capable of specifically and efficiently localising proteins to different sites, and by a variety of different mechanisms of varying degrees of complexity. Indeed, the number of different protein traffic pathways in bacteria is surprisingly high, reflecting their different origins, parallel evolution and adaptation to specific substrate proteins or to specific functions. For example, bacteria are not only capable of assembling proteins in their cell envelopes but can also assemble protein appendages or coats on their surface (pili, flagella and S-layers), can secrete proteins into the growth medium and can inject proteins into target cells that they infect. Considerable progress has been made over the past ten years in the molecular dissection of these protein traffic mechanisms. While many of them are specific to bacteria but others share many features with major protein traffic pathways in eukaryotes and, in all cases, the study of bacterial protein traffic has revealed invaluable insight into the problems encountered when moving a protein from one side of a membrane to another.

Broadly speaking, protein traffic can be divided into two major processes, depending on whether or not the protein that is transported is in an unfolded or folded configuration before and during its transport through the membrane. Surprisingly, however, the degree of complexity of the transport machine does not always seem to correlate with the extent to which the protein that is transported is folded. Furthermore, the distinction between the two is not always clear because there is no practical way of determining the conformation of a protein *in situ* before it is translocated. However, it is relatively well accepted that proteins that are translocated in an unfolded configuration must be maintained in a translocation competent state, presumably by chaperones with anti-folding activity, prior to translocation, or be translocated in a co-translational manner. In these cases, the proteinaceous channel through which the protein is translocated will have a relatively narrow diameter. In contrast, proteins that are folded prior to translocation might need chaperones to assist their folding and/or to verify that folding has occurred before translocation is permitted, and the channel through which the proteins are transported must have relatively large diameter. In both cases, energy is required to achieve translocation.

Recent structural analyses of membrane protein complexes involved in bacterial protein traffic are beginning to reveal fascinating insights into the molecular mechanisms of protein translocation in bacteria. Supramolecular structures can be visualised by electron microscopy, which reveals information on the overall dimensions of the machines and their potential capacity to handle folded or unfolded proteins. Higher resolution X-ray crystallographic analysis reveals atomic level details of the structure of these machines in which putative translocation channels can be identified. However, these structures do not reveal all that is to be learnt. Instead, they provide a fixed framework on which to design experiments to determine how they function.

Finally, the growing interest in understanding how secretion works is not altogether devoid of practical implications: protein export systems *à la carte* can be developed today not only as cell factories for secreting high-added value polypeptides into the external culture medium. They can also be instrumental for delivering active proteins to the niches where such microorganisms may naturally reside (the animal gut, the skin, polluted soil, the rhizosphere) for protection against pathogens, for remediation of chemical contamination or biological control of pests. Finally, some secreted proteins are at the basis of the virulence of many bacterial pathogens. These features open up new possibilities of screening novel drugs and compounds specifically able to inhibit given secretory mechanisms.

Whether basic or applicable, all these challenges in protein secretion will keep us busy for the next few years!

Anthony P. Pugsley
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**The Calcium/Calcineurin/NFAT Pathway:
Regulation and Function**

Organized by
E. N. Olson and J. M. Redondo

(3-5 November)

The calcium-regulated phosphatase Calcineurin is implicated in many programs of gene activation, differentiation and development in eukaryotes. The discovery that this protein is a principal target of the immunosuppressive drugs Cyclosporin A and FK-506 has accelerated our understanding of Ca^{2+} signal transduction pathways. In particular, NFAT transcription factors are major substrates of Calcineurin and have been extensively analysed. NFAT proteins couple extracellular signals to the transcriptional activation of genes induced in response to stimuli that mobilise extracellular Ca^{2+} . In the last few years, there has been a substantial expansion in our understanding of the molecular mechanisms that underlie the regulation and function of the Calcineurin/NFAT pathway. This pathway plays critical roles in a diverse array of important biological processes that include the activation and development of the immune system, the development and function of the nervous system, patterning of the vasculature, morphogenesis of the heart valves, and muscle development. Two further important areas where this signalling pathway has been implicated are cardiac and skeletal muscle hypertrophy, and angiogenesis. Indeed, the effectiveness of immunosuppressive drugs in blocking cardiac hypertrophy and angiogenesis in several animal models, together with the major role that NFAT proteins play in immune regulation, provide strong evidence of the critical role of these transcription factors in pathological conditions.

This workshop was held so that scientists working on different aspects of the Calcium/Calcineurin/NFAT pathway could exchange information about the state of the field in different biological systems, including yeast, *Drosophila*, mouse, and humans. Presentations and discussions covered the regulation and function of calcineurins, the signal transduction components and regulation of the pathway in different cell systems, and the distinct roles of the different NFAT members during development. There were also a number of contributions concerning the role of Calcineurin and NFAT in disease, and the identification of components of the pathway as molecular targets in cardiac growth and in muscle regeneration, size and function. Each presentation at the meeting stimulated intense and excellent discussion: creating a unique forum for the exchange and integration of recent findings, and clarifying the critical issues to be addressed in the regulation and function of the Calcium/Calcineurin/NFAT pathway.

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**Telomeres and Telomerase: Therapeutical
Targets for Cancer and Aging**

Organized by
S. Neidle, J. W. Shay and M. A. Blasco

(17-19 November)

Mortal and immortal stories

On one hand, telomere dysfunction has been proposed to be causal of the aging process as indicated by several human premature aging syndromes that are characterized by a faster rate of telomere loss, as well as, by the study of several mouse models with dysfunctional telomeres. Restoring the functionality of telomeres (ie., by re-introduction of telomerase) in cells with compromised viability due to telomere dysfunction is envisioned as a putative Gene Therapy of age-related diseases.

On the other hand, it is now generally accepted that the ability of tumor cells to grow indefinitely is sustained by activation of telomerase. Indeed, detection of telomerase activity can be used as a marker for tumor growth. In addition, both telomerase activity and telomeres are envisioned as potential targets for new tumor therapies.

There has been a significant amount of research on the role of telomerase in aging and cancer over the past five years. The Juan March meeting has brought together some of the leading scientists in the telomere and telomerase field, who have presented their most recent discoveries. The meeting has been also focused on how to advance the most promising areas into translational research.

Therapeutical approaches: relevance for Biomedical research

Cancer

Telomerase is involved in telomere maintenance in 80-85% of human tumors, whereas it is inactive in somatic cells. It is thus a potential target for therapeutic interventions. Proof-of-principle experiments have shown that selective inhibition of telomerase leads to progressive telomere shortening and ultimately to cancer cell apoptosis, and have provided strong support for the concept of telomerase as an anticancer target.

There are a number of distinct approaches to telomerase inhibition that are currently being studied, notably:

- direct inhibition of the catalytic active site
- antisense inhibition of the telomerase RNA template
- induction of higher-order structure in the telomerase substrate/primer
- mutation of the RNA domain.
- telomerase immunotherapy and hTert-promoter oncolytic viruses

The meeting has provided a forum for evaluating the progress in achieving therapeutic outcomes from these different approaches.

Aging

Telomere loss with normal aging, as well as, in the context of premature aging diseases, is the primary cause of pathological states characterized by a decrease in the proliferative and renewal potential of tissues. Re-introduction of telomerase activity in the affected tissues has been envisioned as a putative Gene Therapy of age-related diseases.

Several speakers at the Juan March meeting have addressed the impact of telomerase deficiency and telomere shortening on aging in the human organism, which opens the possibility of using telomerase in Gene Therapy of age-related diseases and premature aging syndromes.

María A. Blasco

LIST OF INVITED SPEAKERS

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2003 Fellowships

2003 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 2003, 41 of these fellowships were awarded to participants in 12 different meetings. Among these, 10 fellowships were granted to scientists working in Spain, and 31 to scientists working abroad.

Reviews in Scientific Journals

During 2003, the meetings organized by the Centre have been reviewed in the following articles:

- Micol, J.L. and Hake S. (2003). The development of plant leaves. **Plant Physiology** **131(389-394)** (On the workshop on *Leaf Development*, held in February 2002).

- Vicente, M. and Löwe, J. (2003). Ring, helix, sphere and cylinder: the basic geometry of prokaryotic cell division. **EMBO Reports** **4 (655-660)** (On the workshop on *Manufacturing Bacteria: Design, Production and Assembly of Cell Division Components*, held in December 2002).

- Frankle, W.G., Lerma, J. and Laruelle, M. (2003). The Synaptic Hypothesis of Schizophrenia. **Neuron** **39 (205-216)** (On the workshop on *Synaptic Dysfunction and Schizophrenia* held in February 2003).

- He, X. (2003). A Wnt-Wnt Situation. **Developmental Cell** **4**: 791-797. (On the workshop on *Wnt Genes and Wnt Signalling*, held in March 2003).

- Stainier, D. and Pourquie O. (2003). Entrails, heart, brain, limbs, and lymphatics- a recipe for success?. **Dev Cell** **5(2): 193-6** (On the workshop on *Developmental Mechanisms in Vertebrate Organogenesis* held in June 2003).

- Demengeot, J. and Hori S. (2003). Keeping hopes high. **EMBO reports** **4(11): 1033-7** (On the workshop on *Molecular and Genetic Basis of Autoimmune Diseases: SLE and RA* held in April 2003).

- Solnica-Krezel, L. and Eaton S. (2003). Embryo morphogenesis: getting down to cells and molecules. **Development** **130, 4229-4233** (On the workshop on *The Dynamics of Morphogenesis: Regulation of Cell and Tissue Movements in Development* held in May 2003).

- Vincent, J. P. (2003). Membranes, Trafficking, and Signaling during Animal Development. **Cell** **112: 745-749** (On the workshop on *Membranes, Trafficking and Signalling during Animal Development*, held in May 2003).

Editors of the following major scientific journals have participated in different meetings of the Centre during 2003: **Cell** (four meetings); **Neuron** (two meetings); **Nature Reviews Molecular Cell Biology** (one meeting); **Nature Reviews Neuroscience** (one meeting); **Nature Cell Biology** (one meeting); **Nature Neuroscience** (one meeting); **Nature** (one meeting); **Development** (two meetings); **Science** (two meetings).

2004 Meetings Schedule

CENTRE FOR INTERNATIONAL MEETINGS ON BIOLOGY
2004 MEETINGS SCHEDULE

Date	Meeting Subject	Organizers
19-21 January	Stimulatory and Inhibitory Receptors of the Innate Immune System	D.H. Raulet. University of California. Berkeley. M. López-Botet. Universidad Pompeu Fabra. Barcelona.
2-4 February	Molecular Cross Talk Among Chromosome Fragility Syndromes	H. Joenje. VU University Medical Centre. Amsterdam. J. Surrallés. Universidad Autónoma. Barcelona.
1-3 March	The Making of a Fruit: from Genes to Molecules to Phenotype	M. Bouzayen. UMR INRA-INP/ENSAT. Castanet-Tolosan. J.J. Giovannoni. Cornell University. Ithaca. A. Granell. Universidad Politécnica. CSIC. Valencia.
29-31 March	Molecular Mechanisms of Vesicle Selectivity	B.M.F. Pearse. Medical Research Council. Cambridge. I.V. Sandoval. Centro de Biología Molecular "Severo Ochoa". Madrid.
19-21 April	The p75 Neurotrophin Receptor: Signalling and Function	Y.-A. Barde. University of Basel. P.A. Barker. McGill University. Montreal. J.M. Frade. Instituto Cajal. Madrid.
10-12 May	The Genetic Control of Eye Development and its Evolutionary Implications	W.J. Gehring. University of Basel. E. Saló. Universidad de Barcelona.
24-26 May	The Proteins Controlling Cell Growth and their Role in Tumour Formation: mTOR, TSC and PTEN	G. Thomas. Friedrich Miescher Institute. Basel. A. Carrera. Centro Nacional de Biotecnología. Madrid. I. Mérida. Centro Nacional de Biotecnología. Madrid.
7-9 June	Immunodominance: The Key to Understanding and Manipulating CD8⁺ T Cell Responses to Viruses	P.C. Doherty. The University of Melbourne. Victoria. J.W. Yewdell. National Institute of Allergy and Infectious Diseases. Bethesda. M. Del Val. Instituto de Salud Carlos III. Madrid.
4-6 October	Disease Resistance and Related Signalling Mechanisms in Plants	J.L. Dangl. University of North Carolina. Chapel Hill. P. Schulze-Lefert. Max-Planck-Institut für Züchtungsforschung. Köln. C. Castresana. Centro Nacional de Biotecnología. Madrid.
25-27 October	Interpreting Hedgehog-Gli Signalling	A. Ruiz i Altaba. The Skirball Institute for Biomolecular Medicine. New York. I. Guerrero. Centro de Biología Molecular "Severo Ochoa". Madrid.
15-17 November	Molecular Advances in DAG Signalling	M.G. Kazanietz. University of Pennsylvania. Philadelphia. P.J. Parker. Cancer Research UK. London. T. Iglesias. Instituto de Investigaciones Biomédicas "Alberto Sols". Madrid.
13-15 December	Recombinational DNA Repair and its Links with DNA Replication and Chromosome Maintenance	S. Kowalczykowski. University of California. Davis. S. West. Cancer Research UK. London. A. Aguilera. Universidad de Sevilla. Sevilla. J.C. Alonso. Centro Nacional de Biotecnología. Madrid.

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