

Banbury Center

COLD SPRING HARBOR LABORATORY

2008



BANBURY CENTER

Banbury Center is a 55-acre estate adjoining the waters of Long Island Sound on the north shore of Long Island, barely 40 miles east of downtown Manhattan and some five miles from Cold Spring Harbor Laboratory. The estate was donated to the Laboratory in 1976 by Charles Sammis Robertson, together with funds for necessary architectural conversions and an endowment to cover upkeep of the grounds and the original estate structures. With the Laboratory's international reputation for research and education, the magnificent Banbury grounds and buildings are an ideal site for small conferences in the areas of molecular biology and genetics, especially as they relate to health, social, and policy issues.

What was once the estate's original seven-car garage is now the Conference Room, containing administrative offices, a small library, and—at its center—a room of an ideal shape and size for workshop-style discussion meetings. Complete with extensive, unobtrusive sound and projection facilities as well as wall-to-wall blackboard space, the room can accommodate as many as 40 participants while remaining equally conducive to either formal presentations or informal give-and-take.

The Robertsons' family house, situated on the final promontory before the grounds descend to the shore of Cold Spring Harbor, now serves as the center for participant accommodations and dining, while the extensive grounds, swimming pool, tennis court, and beach present ample recreational resources. On-site accommodations were supplemented by the opening in 1981 of the Sammis Hall guest house—a modern embodiment of the sixteenth century Palladian villas—designed for the Center by the architectural firm of Moore Grover Harper. In 1997, the Meier House, opposite the Conference Center, was added to provide extra housing so that everyone attending a Banbury Center meeting can stay on the estate.



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BANBURY CENTER

EXECUTIVE DIRECTOR'S REPORT

The year 2008 was unusually quiet at Banbury. Our meetings season was much shorter than usual, finishing at the end of October. It was then that we began a major renovation of Robertson House that necessitated emptying the house and turning it over to the Laboratory's facilities crews and outside contractors. Times have changed and conditions that were once acceptable are no longer so. Most significantly, air-conditioning is being installed—an upgrade that will be much appreciated by all those who will spend the summer months here! We are taking advantage of this disruption to rewire the house, redecorate, and upgrade Internet facilities. The house was built in 1936 so we will be in good time to celebrate its 75th anniversary in 2011.

As a consequence of the shortened season, there were only 18 scientific meetings in 2008 and a correspondingly smaller number of participants: 524. Of these, 81% came from the United States, specifically from 33 states, with New York, California, Massachusetts, and Maryland providing most of these participants. Almost one-fifth of participants came from outside of the United States, from 16 countries. In addition to the meetings, there were six CSHL lecture courses, and two CSHL laboratories held retreats here. We were happy to again welcome local organizations including the Conservation Board of the Village of Lloyd Harbor, the Cold Spring Harbor School District, and the Caumsett Foundation. So, overall, the Center was busy throughout the year.

The year 2008 marked Jim Watson's 40th year at Cold Spring Harbor Laboratory, and to observe the occasion, Banbury Center held three meetings on topics of special interest to Jim. The common thread of the meetings involved topics of interesting scientific research, the results of which have important consequences for society and should help form public policy. The titles of each were cast as questions, in the expectation that this phrasing would help focus minds and provoke debate. Regrettably, but not surprisingly, we did not answer any of the questions. However, the meetings brought together fascinating groups of people for very lively discussions.



Coffee break discussions

The first of the three meetings was entitled *How Will We Prevent Most Forms of Cancer?* We have been performing research on cancer for more than a century and since the early 1970s, we have discovered many of the genetic and biochemical changes that turn a normal cell into a cancer cell. This knowledge is going to increase rapidly with the application of large-scale genomic analysis of tumors. However, despite this intensive research, the number of cancers that can at present be cured effectively and efficiently remains small. The participants explored why this is so, evaluated current and potential therapies, asked what can be done to improve early detection, and faced the issue that some cancers may not be curable.

The second meeting in the series asked the question *To What Age Should We Be Expected to Work?* Here, the scope of the discussions ranged from laboratory research on aging through to the economic consequences of extended life span. Considerable progress has been made in elucidating some of the genetic changes and molecular processes that contribute to aging in experimental organisms. Although our lives are being extended through better lifestyles and improved health care, it seems, however, that our maximum life spans are unchanged. Is extending the human life span an achievable or desirable goal? Should we concentrate instead on maintaining the quality of life for the extra years that we are gaining now? That is, can we live longer and still be healthy?

The third meeting was also of broad scope, ranging from research to social impact to educational policy: *How Can We Improve Our Brains?* It is the hope of every parent that their child will be bright and intelligent. Parents work to help their children's brain work better by providing education and stimulation, and society as a whole makes a tremendous commitment to the education of its young people. Are there data emerging from cognitive neuroscience that such education programs should take into account? Are there learning regimes that might be more effective than those typically found in the classroom? At the other end of life is the hope of all of us that the normal decline in cognitive skills that accompanies aging will be slow. Might "brain exercises" maintain our brains at a higher level of functioning? Is there evidence that such exercises work? Are there effective pharmacological agents? In short, how can we best use the resources of society to help our brains work better throughout our lives?

Meetings dealing with biomedical research that has important social implications seem to have been the norm at Banbury in 2008. *Who Are We? Kinship, Ancestry, and Social Identity* reviewed a field that has been changing rapidly because of technical advances in genomics and rapidly increasing public interest. Participants discussed the issue that although genetics provides a description of human beings that reflects their *biological* ancestry, cultural norms provide a description of *social* ancestry, and these two descriptions need not be, and often are not, the same. This has been highlighted by the increasing use of DNA-based analysis for tracing genealogical ancestry. Here, discrepancies between ancestry revealed by genetic analysis and assumed by cultural descent may profoundly affect individuals' views of themselves. The importance of the relationship between genetic and ethnic identities requires careful, rational, and critical review of what is a controversial subject.

I began my research career working on Duchenne's muscular dystrophy (DMD), studying cells growing in tissue culture. At that time, no human genes, let alone genes involved in genetic disorders, had been isolated, and the cloning in the mid 1980s of the gene involved in DMD was a landmark in human genetics. It was confidently expected, now that the protein was known and the gene was available for gene therapy, that treatments would quickly follow, but that optimism was misplaced. In the succeeding years, we have learned that going from a cloned gene to a therapy is extraordinarily difficult. However, a new therapeutic strategy has been developed that uses oligonucleotides to direct the splicing of an mRNA so that a functional or partially functional protein is produced. This has proved to be successful in experimental models of DMD and spinal muscular atrophy. Participants in the meeting *Oligonucleotide-directed Splicing: Therapeutic Strategies* critically reviewed current progress in the field and discussed new technical advances in the synthesis and properties of oligonucleotides. (A report of the meeting was published in *Science* 322: 1454–1455 [2008].)

The year 2008 was also the first time that a Banbury Center meeting was not held at Banbury! We had planned a discussion meeting on *Epigenetics: Mechanisms and Regulation*, but the Robertson House renovations meant that there was no time to have it at the Banbury estate. The Meetings and Courses office stepped in to help and arranged for the meeting to be in the Plimpton Room on the main campus. We are very grateful to David Stewart and Val Pakaluk for their hard work done on our behalf. The meaning of the term "epigenetics" has come to cover a very wide range of biological processes, from dynamic short-lived chromatin-mediated gene regulation to long-term alteration of chromatin and other extrachromosomal proteins in nonreplicating cells. Participants were encouraged to think beyond details of molecular mechanisms and to consider how "epigenetics" should be defined. As happens whenever scientists come together to define terms, the discussions proved to be very lively!

For the second year, there has been a significant change in Banbury Center staff. In May 1978, Victor McElheny was appointed as director of the Center. He was followed, in September 1978, by the appointment of Beatrice Toliver as administrative assistant, a position that she has held for 30 years, providing all four Banbury directors with essential support. Bea's knowledge of Banbury and its operations was particularly important to me when I became director following the tragic death of Steve Prentis. With her help, we were able to maintain and expand the program. During the 30 years that Bea has been at Banbury, she has become well known to the thousands of scientists who have participated in Banbury meetings. On their behalf, as well as the Laboratory, we wish Bea the happiest of retirements.

As always, the operations of the Banbury Center depend on many people: Ellie Sidorenko at the Conference Room, Basia Polakowski at Robertson House, and Mike Peluso and the grounds crew who look after the Banbury estate. We have many interactions with David Stewart and his staff in the Meetings and Courses office, and, of course, we could not do anything here without the work of the Laboratory's Culinary Services and Housekeeping.



Bea Toliver

Jan Witkowski
Executive Director

BANBURY CENTER MEETINGS

Genes and the Environment: New Strategies for Research on Multiple Sclerosis

February 3–6

FUNDED BY **Stanley Trotman, Jr. Trust**

ARRANGED BY **B. Greenberg**, The Johns Hopkins Hospital
A. Mellor, Accelerated Cure Project for Multiple Sclerosis
H. Schmidt, Accelerated Cure Project for Multiple Sclerosis

The goal of this meeting was to explore what would be the ideal study for identifying gene–environment interactions involved in multiple sclerosis (MS). Although associations with MS have been identified for a few risk factors such as the HLA-DR2 gene haplotype and cigarette smoke, very little progress has been made in explaining the specific biological role of these factors and the manner in which risk factors interact in the development of MS. However, there are reasons to hope for faster progress in the coming years. High-throughput technologies such as genomic microarrays and new DNA sequencers are producing data in a much more cost-effective fashion than ever before and enabling new experimental strategies. Participants in the meeting were drawn from a wide variety of research areas including MS clinical research, genetics, genomics, environmental toxicology, and epidemiology.

Introduction: **J.A. Witkowski**, Banbury Center, Cold Spring Harbor Laboratory
Welcoming Remarks: **B. Greenberg**, The Johns Hopkins Hospital, Baltimore, Maryland: Overview of the agenda/schedule and objectives.

SESSION 1

Chairperson: **B. Greenberg**, The Johns Hopkins Hospital, Baltimore, Maryland

B. Greenberg, The Johns Hopkins Hospital, Baltimore, Maryland: Overview of multiple sclerosis: Knowns and unknowns.	Waltham, Massachusetts: Genetics of multiple sclerosis.
A. Bar-Or, Montreal Neurological Institute, Canada: Immunology of multiple sclerosis.	S. Subramaniam, Vanderbilt Stallworth Rehabilitation Hospital, Nashville, Tennessee: Role of DNA damage pathway in MS.
H. Schmidt, Accelerated Cure Project for Multiple Sclerosis,	W.R. McCombie, Cold Spring Harbor Laboratory: Massively parallel targeted resequencing: Opportunities and challenges.

SESSION 2

Chairperson: **A. Ascherio**, Harvard School of Public Health, Boston, Massachusetts

S. Jacobson, NINDS/National Institutes of Health, Bethesda, Maryland: Virus associations and MS.	D. Mohr, Northwestern University, Chicago, Illinois: Stress and MS.
W.I. Lipkin, Columbia University, New York: Identifying viral pathogens in neurological disease.	S. Schmidt, Duke Center for Human Genetics, Durham, North Carolina: Gene–environment interaction in age-related macu- lar degeneration: A success story.
C. Hayes, University of Wisconsin, Madison: Vitamin D and MS.	B. Banwell, Hospital for Sick Children, Toronto, Canada: Pediatric MS: Insights into the disease.
B. Winstock-Guttman, SUNY University of Buffalo, New York: Gender and MS.	

SESSION 3

Chairperson: **T. Vollmer**, Barrow Neurological Institute, Phoenix, Arizona

B. Greenberg, The Johns Hopkins Hospital, Baltimore, Maryland: Overview of agenda and goals for the day.	Carolina: Empiric delineation of patient heterogeneity to improve diagnosis and intervention.
S.D. Vernon, CFIDS Association of America, Charlotte, North	T. Vollmer, Barrow Neurological Institute, Phoenix, Arizona:

Biorepositories: Challenges and applications.
A. Ascherio, Harvard School of Public Health, Boston,
Massachusetts: Use of prospective cohorts in investigating

causes of disease.
D.E. Ganem, University of California, San Francisco:
Discovering novel pathogens.

SESSION 4

B. Greenberg, The Johns Hopkins Hospital, Baltimore, Maryland

E.W. Daw, Washington University, St. Louis, Missouri:
Oligogenic modeling with environmental covariates for asso-
ciation and linkage studies.

D.W. Bigwood, Diogenix, Inc., Madison, Connecticut: Analysis
of large gene expression data sets: Strategies for identifying

novel pathogenic pathways.

M. Wigler, Cold Spring Harbor Laboratory: How might one
apply copy-number analysis to understand the genetics of
multiple sclerosis?

Developing a Study Protocol

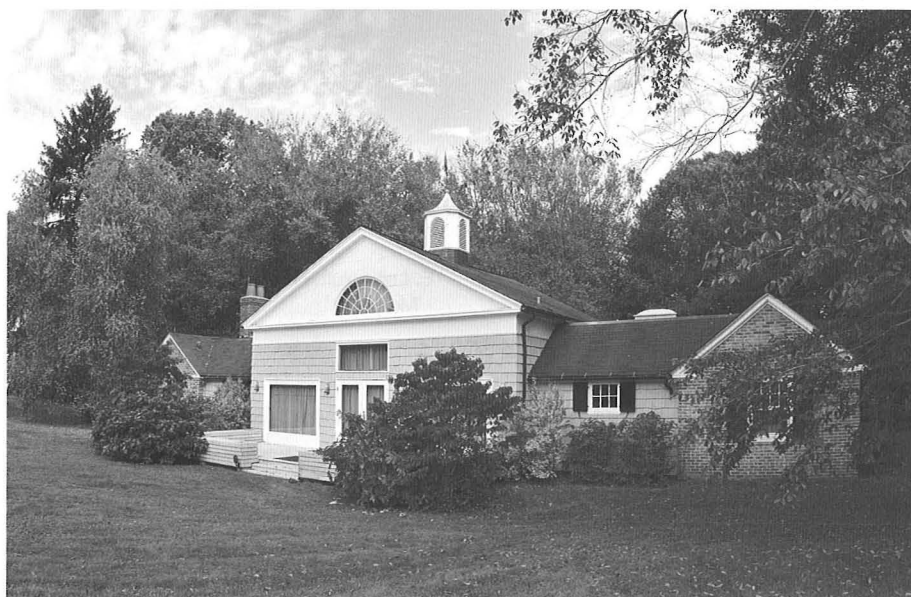
Discussion initiated by B. Greenberg, The Johns Hopkins Hospital, Baltimore, Maryland: Day conclusions.

SESSION 5

Presentation of Proposed Protocol

B. Greenberg, The Johns Hopkins Hospital, Baltimore, Maryland

Discussion and revision of Protocol.



Banbury Conference Center

GEM Meeting

February 13–14

FUNDED BY **The Stanley Foundation**

ARRANGED BY **S. Gary**, Cold Spring Harbor Laboratory

The Genetics of Early-onset Mania (GEM) Study of Bipolar Disorder is a research project organized by Cold Spring Harbor Laboratory and supported by a donation from Ted and Vada Stanley. This collaborative project involves four institutions: CSHL, Zucker Hillside Hospital–North Shore LIJ, NIMH, and Johns Hopkins University. The goal of this project is to identify genes that may be associated with bipolar disorder by (1) performing genome-wide analysis of copy-number variation in bipolar families, (2) assessing the overall contribution of de novo and inherited mutations in sporadic and familial bipolar disorder, and (3) identifying novel candidate genes for further study.

Participants in the GEM project have initiated a new collection focusing on trios (affected child plus both parents) with the goal of collecting samples from 225 patients and their parents. This Banbury meeting brought together the GEM collaborators to provide a project update and to address the following points: (1) increasing the sample size of collection, (2) the GEM database for entering clinical data, (3) an update on CNV analysis of these and related samples, and (4) the next steps for the project.

Welcome, Opening Remarks: **J.D. Watson**, Cold Spring Harbor Laboratory

Overview and Status of GEM Project: **S. Gary**, Cold Spring Harbor Laboratory

Discussion for Increasing Sample Size (Protocol Changes; Identifying Existing Collections with Ability to Collect Parental DNAs: Other Ideas?)

F. McMahon, E. Leibenluft, T. Schulze, NIMH/National Institutes of Health, Bethesda, Maryland

Group Discussion

Phenotype Discussion/Neurocognition Collection

K. Burdick, Zucker Hillside/LIJ, Glen Oaks, New York

E. Leibenluft, NIMH/National Institutes of Health, Bethesda, Maryland

Presentation about GEM Database/Website

J. Pearl, Data Related, NIMH/National Institutes of Health, Bethesda, Maryland

T. Leotta and V. Makarov, Cold Spring Harbor Laboratory

Update on Recent Results

J. Sebat, Cold Spring Harbor Laboratory

Other Recent Findings in Genetics of Bipolar Disorder

Next Steps (Analysis, Data Sharing, etc.)

Group Discussion

Living on Human Beings: Metagenomic Approaches and Challenges

March 2-5

FUNDED BY

Cold Spring Harbor Laboratory Corporate Sponsor Program

ARRANGED BY

E.F. DeLong, Massachusetts Institute of Technology
J.I. Gordon, Washington University School of Medicine
G.M. Weinstock, Washington University School of Medicine

Traditional methods of studying microorganisms begin with the isolation of single cells from all those present in a sample, followed by their culture. This introduces the bias that only those organisms that can be cultured can be studied. In contrast, metagenomics is concerned with the characterization of the entire community of microorganisms in a sample. This can be done because new sequencing strategies can sequence gigabases of DNA and bioinformatics strategies can find sequences of interest in these gigabases of sequence. There are projects under way to examine the entire microbiota of environments such as the oceans, soil, and air. Most excitingly, metagenomics offers the possibility of treating the human body as a set of habitats and examining the microbial communities of each habitat, whether the mouth, skin, or gut. This meeting was designed to define and suggest solutions to some of the conceptual and experimental challenges that this field faces.



G. Weinstock, R. Gunsalus

Introductory Remarks:

J.A. Witkowski, Banbury Center, Cold Spring Harbor Laboratory

Introduction: Why This Meeting?: **G.M. Weinstock**, Washington University School of Medicine, St. Louis, Missouri

SESSION 1: Approaches for Studying Environmental Communities and Their Operations

Chairperson: B. Birren, Broad Institute, Cambridge, Massachusetts

E.F. DeLong, Massachusetts Institute of Technology,
Cambridge: Integrating microbial community genomics and
(eco)systems biology: Problems and prospects.
S.W. Chisholm, Massachusetts Institute of Technology,
Cambridge: Genome-enabled metagenomics:
Prochlorococcus as a case study.
M. Polz, Massachusetts Institute of Technology, Cambridge:

Identifying ecologically differentiated populations.
A.Z. Worden, Monterey Bay Aquarium Research Institute, Moss
Landing, California: Targeted metagenomics of marine eukaryotes.
E.G. Ruby, The University of Wisconsin, Madison, Wisconsin:
Comparative genomic analyses of a beneficial bacterial asso-
ciation: Using natural models to ask how mutualisms are
maintained.



SESSION 2: Approaches for Characterizing Animal Body Habitat-associated Communities and Their Operations

Chairperson: S. Eddy, Howard Hughes Medical Institute, Ashburn, Virginia

R.L. Hettich, Oak Ridge National Laboratory, Tennessee: A proteogenomic approach for characterizing the molecular activities of gut microbiome.

J. Ravel, University of Maryland School of Medicine, Rockville: Genomic tools for studying the ecology of the human vagina.

H. Flint, Rowett Research Institute, Aberdeen, United Kingdom: Molecular and cultural approaches to functional analysis of the human intestinal microbiota.

J. Segre, National Human Genome Research Institute, Bethesda, Maryland: Survey of skin microflora in healthy volunteers.

M.J. Blaser, New York University School of Medicine, New York: Approaches to defining the cutaneous microbiota in health and disease.

S. Dusko Ehrlich, INRA, Jouy-en-Josas, France: MetaHIT: The European project on metagenomics of human intestinal tract.

SESSION 3: Microbial Evolution, Phylogeny, and Diversity

Chairperson: J. Ravel, University of Maryland School of Medicine, Rockville

N.R. Pace, University of Colorado, Boulder: Molecular microbiology of the human environment.

F.E. Dewhirst, Forsyth Institute, Boston, Massachusetts: The human oral microbiome.

R. Knight, University of Colorado, Boulder: Phylogenetically informed community comparisons using metagenomic data.

C. M. Fraser-Liggett, University of Maryland School of Medicine, Baltimore: Surveying human microbiome diversity: How deep do we go?

R. Gunsalus, University of California, Los Angeles: The essential biology of the anaerobic microbial food chains.

SESSION 4: Technology: What's Needed to Move Beyond Present Practices

Chairperson: E.F. DeLong, Massachusetts Institute of Technology, Cambridge

J.K. Nicholson, Imperial College London, United Kingdom: The microbiome-mammalian-metabolic axis in health and disease.

B. Birren, Broad Institute, Cambridge, Massachusetts: Genome sequencing using new technologies.

S.C. Schuster, Pennsylvania State University, University Park: Next-generation sequencing and MEGAN metagenomics analysis.

R. Lasken, J. Craig Venter Institute, San Diego, California: High-throughput single-cell genomics pipeline: Applications to the HMB and the hospital environment.

G.M. Weinstock, Washington University School of Medicine, St. Louis, Missouri: The Human Microbiome Project.

SESSION 5: Informatics/Data Analysis: Attaching the Bottlenecks

Chairperson: S.C. Schuster, Pennsylvania State University, University Park

P. Hugenholtz, DOE Joint Genome Institute, Walnut Creek, California: Resolving genetic gradients using fine-scale metagenomics.

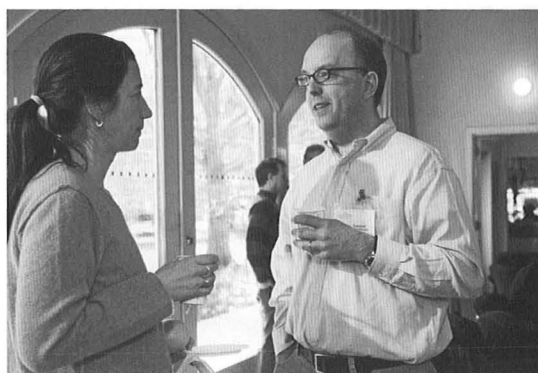
S. Eddy, Howard Hughes Medical Institute, Ashburn, Virginia: Advances in large-scale protein sequence analysis: Pfam and HMMER.

A. Godzik, The Burnham Institute, La Jolla, California: Understanding diversity and divergence in (among others) metagenomics data sets.

S.A. Kravitz, J. Craig Venter Institute, Rockville, Maryland: Challenges of large-scale metagenomics data management.

SESSION 6: Meeting Summary and General Discussion

Chairperson: J.I. Gordon, Washington University School of Medicine, St. Louis, Missouri



J. Segre, A. Gardner

Recent Advances and a Multilevel Analysis From FMRP Biology to Clinical Trials

March 9–12

FUNDED BY

NIMH Grant to the University of Illinois

ARRANGED BY

E. Berry-Kravis, Rush University Medical Center

K. Clapp, FRAXA Research Foundation

W.T. Greenough, University of Illinois

E. Klann, New York University

P.W. Vanderklish, Scripps Research Institute

Significant advances have been made in several areas of fragile X research, particularly those that shed new light on underlying mechanisms. The meeting was designed to review these new findings and to encourage new ideas on the basic relationships between FMRP function, the neurobiological origins of symptoms, and potential treatments. In particular, participants discussed FMRP function and regulation; proteomic and high-specificity FMRP target analyses; alterations in synaptic plasticity, structure, and signaling coupled to mGluR and non-mGluR pathways; mechanistic commonalities between fragile X and other syndromic forms of mental retardation with autism as an endophenotype; systems level approaches to understanding fragile X syndrome and autism; and clinical trials.

Introductory Remarks:

J.A. Witkowski, Banbury Center, Cold Spring Harbor Laboratory

Opening Comments:

K. Clapp, FRAXA Research Foundation, Newburyport, Massachusetts

SESSION 1: Fragile X Phenotypes and Underlying Neural Systems

Chairperson: **M.R. Tranfaglia**, FRAXA Research Foundation, Newburyport, Massachusetts

R.J. Hagerman, M.I.N.D. Institute, Sacramento, California:

Quantitative measures of CNS function for medication trials and comments on ganaxolone study.

D.P. Kennedy, California Technology Institute, Pasadena:

Functional abnormalities of the default network in autism and fragile X syndrome.



R. Zorovic, K. Clapp, M. Bear

- D.L. Nelson, Baylor College of Medicine, Houston, Texas: Conditional mutations in *Fmr1* and *Fxrs* in the mouse: An update on genotypes and phenotypes.
- J.R. Larson, University of Illinois, Chicago: Olfactory learning in the fragile X mouse.
- A. El Idrissi, College of Staten Island, New York: Potential pharmacotherapeutic actions of taurine in a mouse model of fragile X syndrome.
- R.E. Paylor, Baylor College of Medicine, Houston, Texas: Pharmacological modification of *Fmr1* KO behavioral phenotypes.

SESSION 2: Synaptic Morphology Phenotypes and Local Network Abnormalities
Chairperson: W.T. Greenough, University of Illinois, Urbana

- W.T. Greenough, University of Illinois, Urbana: Short tutorial: Neurobiology of fragile X—A review.
- I. Ethell, University of California, Riverside: Minocycline accelerates dendritic spine maturation and alleviates behavioral defects in animal model.
- K. Broadie, Vanderbilt University and Medical School, Nashville, Tennessee: Roles of FMRP in neuronal architecture development and synaptogenesis.



I. Ethell

SESSION 3: Distribution, Functions, and Regulation of FMRP
Chairperson: D.L. Nelson, Baylor College of Medicine, Houston, Texas

- D. Morris, University of Washington, Seattle: *Fmr1* transcript isoforms: Association with polyribosomes; regional and developmental expression in brain.
- M. Ramaswami, Trinity College, Dublin, Ireland: Neuronal FMRP particles and their similarities to P bodies.
- G.J. Bassell, Emory University, Atlanta, Georgia: Stimulating travels and functions of FMRP in dendrites and axons.
- S. Cernan, University of Illinois, Urbana: FMRP expression during learned vocalizations in male zebra finch.

SESSION 4: Proteomics and FMRP mRNA Target Analyses
Chairperson: W.T. Greenough, University of Illinois, Urbana

- J. Darnell, The Rockefeller University, New York: Cross-linking Co-IP (CLIP) identification of novel pre- and postsynaptic RNA targets of FMRP.
- H. Tiedge, State University of New York, Brooklyn: FMRP and small RNAs.
- J.S. Malter, University of Wisconsin, Madison: APP, Abeta, and fragile X syndrome.
- S.J. Tapscott, Fred Hutchinson Cancer Research Center, Seattle, Washington: An antisense transcript spanning the CGG repeat region of FMR1 is up-regulated in premutation carriers but silenced in full mutation individuals.

SESSION 5: Modulation of Synaptic Plasticity and Signaling by FMRP
Chairperson: M.F. Bear, Massachusetts Institute of Technology, Cambridge

- K.M. Huber, University of Texas Southwestern Medical Center, Dallas: Impaired excitatory drive of neocortical inhibitory neurons may contribute to longer persistent activity states in *Fmr1* KO mice.
- J. Lauterborn, University of California, Irvine: Hippocampal LTP deficits in fragile X: Restoration of synaptic plasticity by BDNF.
- A. Bhattacharyya, University of Wisconsin, Madison: cAMP signaling in FX brain.
- M.C. McKenna, University of Maryland School of Medicine, Baltimore: Altered neuronal and astrocytic glutamate metabolism in 18-day-old *Fmr1* knockout mouse brain: Normalization by MPEP.
- R.S. Zukin, Albert Einstein College of Medicine, Bronx, New York: Dysregulation of mTOR signaling in mouse model of fragile X syndrome.
- S.T. Warren, Emory University School of Medicine, Atlanta, Georgia: FMRP signaling pathway mediated by phosphorylation.
- I.J. Weiler, University of Illinois, Urbana-Champaign: Aberrant phosphatase activation in fragile X syndrome.

SESSION 6: Mechanistic Parallels between FXS and Other Neuropsychiatric Conditions

Chairperson: E. Klann, New York University, New York

E. Klann, New York University, New York: Short tutorial: Altered translational control in fragile X model mice and other mouse models of MR and ASD.

M.F. Bear, Massachusetts Institute of Technology, Cambridge:

Studies of protein synthesis in hippocampus.

M.A. Smith, Case Western Reserve University, Cleveland, Ohio: Parallels between fragile X and Alzheimer's disease.

SESSION 7: Progress in Clinical Development

Chairperson: S.A. Warren, Emory University School of Medicine, Atlanta, Georgia

E. Berry-Kravis, Rush University Medical Center, Chicago, Illinois: Update on lithium treatment in fragile X syndrome.

B.A. Oostra, Erasmus Universiteit Rotterdam, The Netherlands: The effect of mGluR5 antagonists in vitro and in vivo.

C. Erickson, Riley Hospital for Children, Indianapolis, Indiana:

Aripiprazole in fragile X patients.

F. Gasparini, Novartis Pharma AG, Basel, Switzerland: Targeting Group I and II metabotropic glutamate receptors: Drug discovery and potential therapeutic indications.



Algebraic Statistics, Machine Learning, and Lattice Spin Models

March 16–19

FUNDED BY

Clay Mathematics Institute

ARRANGED BY

J. Carlson, Clay Mathematics Institute
D.A. Ellwood, Clay Mathematics Institute
P.P. Mitra, Cold Spring Harbor Laboratory

Recently, there have been exciting advances in the application of ideas and algorithms from commutative algebra and group theory to problems of data analysis and statistics, particularly in computational genomics. However, these ideas are not yet widely known to other communities of theorists who may benefit from these developments. The goal of this workshop was to bring together mathematicians working in algebraic statistics with researchers in machine learning and statistical physics for mutual pedagogy and for exploration of new research avenues opened up by the application of algebraic techniques to data.

Introductory Remarks:

J.A. Witkowski, Banbury Center, Cold Spring Harbor Laboratory

SESSION 1

Chairperson: C. Myers, Cornell University, Ithaca, New York

P.A. Parrilo, Massachusetts Institute of Technology, Cambridge:
Semigroups and semidefinite programming.

R. Rabadan, Institute for Advanced Study, Princeton, New Jersey: Viruses.

SESSION 2

Chairperson: P.P. Mitra, Cold Spring Harbor Laboratory

L. Pachter, University of California, Berkeley, and S. Sullivan, Harvard University, Cambridge, Massachusetts: Algebraic statistics tutorial.

L. Pachter, University of California, Berkeley, and S. Sullivan, Harvard University, Cambridge, Massachusetts: Algebraic statistics tutorial (cont'd.).

Informal Discussions

SESSION 3

Chairperson: A. Sengupta, Rutgers, The State University of New Jersey

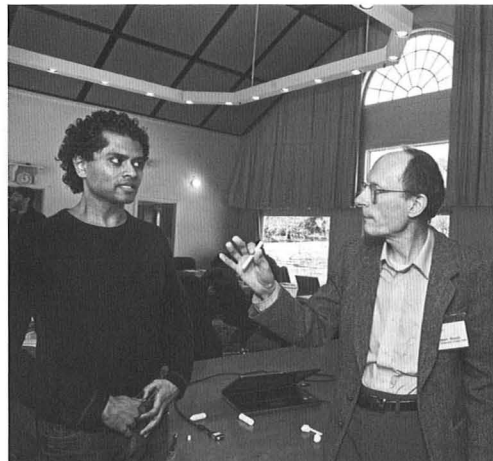
B. Mishra, New York University, New York: Zero-one phenomena in genome sequencing.

SESSION 4

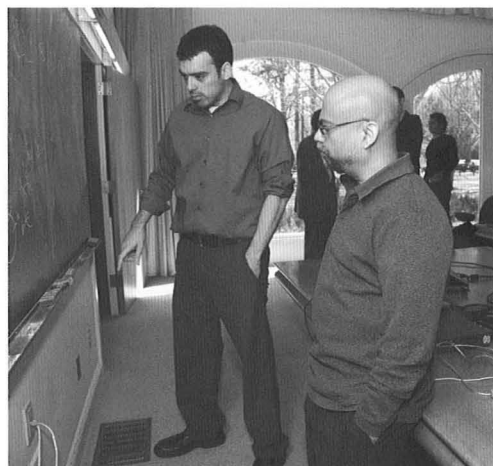
Chairperson: D. Huse, Princeton University, New Jersey

G. Carlsson, Stanford University, California: Algebraic topology for data analysis.

C. Sire, Université Paul Sabatier, Toulouse, France: Poker and statistical physics.



P. Mitra, R. Shrock



P. Parrilo, B. Mishra



How Will We Be Able to Cure Most Cancers?

March 30–April 2

FUNDED BY

OSI Pharmaceuticals, Inc.

ARRANGED BY

A.J. Levine, Institute of Advanced Studies

C.L. Sawyers, Memorial Sloan-Kettering Cancer Center

R.A. Weinberg, Whitehead Institute for Biomedical Research

J.A. Witkowski, Banbury Center, Cold Spring Harbor Laboratory

We have been performing research on cancer for more than a century and, since the early 1970s, we have discovered many of the genetic and biochemical changes that turn a normal cell into a cancer cell. Furthermore, our knowledge of the fundamental biology of cancer is likely to undergo another major increase as genome-based techniques begin to be used on a massive scale to characterize the full set of molecular changes in many tumor types. However, the number of cancers that can at present be cured effectively and efficiently remains small. Why is this? Do cancers vary so much that a treatment specific to one patient's cancer may be ineffective for another patient? What targets are currently available for drug therapy? What can be done to increase the number? What is the potential for combination therapies? What can be done to improve early detection? May we have to face the fact that many cancers may not be curable?

Introductory Remarks:

J.A. Witkowski, Banbury Center, Cold Spring Harbor Laboratory

Remarks:

J.D. Watson, Cold Spring Harbor Laboratory

Introduction: The Problem: M.J. Thun, American Cancer Society, Atlanta, Georgia: Cancer trends.

SESSION 1

Chairperson: R. Weinberg, Whitehead Institute for Biomedical Research, Cambridge, Massachusetts

C. Lengauer, Novartis Institutes for Biomedical Research, Inc., Cambridge, Massachusetts: Oncology target validation—taken seriously.

J. Barsoum, Synta Pharmaceuticals Corporation, Lexington, Massachusetts: Advancing Elesclomol from the lab to phase 3:

Targeting cancer by the selective induction of oxidative stress.

R. Cohen, Genentech, South San Francisco, California:

Targeted therapies: Lessons from Herceptin.

Y. Luo, Tsinghua University, Beijing, China: Molecular cancer therapy of endostatin: The end of the beginning.



SESSION 2

Chairperson: B. Stillman, Cold Spring Harbor Laboratory

- D. Reinberg, New York University School of Medicine, New York: Histone deacetylases, methylases and demethylases: Current status, future potentials.
- J. Schlessinger, Yale University School of Medicine, New Haven, Connecticut: Developing new therapies: The example of PLX4720 and B-RafV600E.
- J. Peto, London School of Hygiene & Tropical Medicine, London,

- United Kingdom: The costs and benefits of HPV vaccination.
- M. Stratton, The Sanger Institute, Cambridge, United Kingdom: Patterns of somatic mutation in human cancer genomes.
- J.B. Hicks, Cold Spring Harbor Laboratory: Breast tumor architecture and progression analyzed by genomic method.
- B. Weber, GlaxoSmithKline, Collegeville, Pennsylvania: Genomic approach to targeted drug development.

SESSION 3

Chairperson: B. Stillman, Cold Spring Harbor Laboratory

- R.A. Weinberg, Whitehead Institute for Biomedical Research, Cambridge, Massachusetts: Mechanisms of malignant progression.
- P.A. Beachy, Stanford University, California: Hedgehog signaling and cancer.
- T. Curran, Children's Hospital of Philadelphia, Pennsylvania: Targeting the Sonic Hedgehog pathway in pediatric brain tumors: Promise and problems.

- L.C. Cantley, Harvard Medical School/Beth Israel Deaconess Medical Center, Boston, Massachusetts: Targeting the PI3K pathway.
- A.J. Levine, Institute for Advanced Study, Princeton, New Jersey: New methods for identifying drug targets.
- C.B. Harley, Geron Corporation, Menlo Park, California: Telomerase-based therapies: Potential to hit a stem cell target.

SESSION 4

Chairperson: B. Clarkson, Memorial Sloan-Kettering Cancer Center, New York

- P.-P. Pandolfi, Beth Israel Deaconess Medical Center, Boston, Massachusetts: Biochemical and genetic pathways as sources of targets.
- G. Evan, University of California, San Francisco: Targeting the untargetable—Modeling anti-Myc therapy in mouse cancer models.
- S. Lowe, Cold Spring Harbor Laboratory: Mouse models in cancer gene discovery and cancer therapy.

- C.J. Sherr, St. Jude Children's Research Hospital, Memphis, Tennessee: Why BCR-ABL-induced acute lymphoblastic leukemia (Ph⁺ ALL) responds poorly to targeted therapy.
- A. Ashworth, Institute of Cancer Research, London, United Kingdom: Synthetic lethal approaches to cancer therapy.
- P. Pharoah, Strangeways Research Laboratory, Cambridge, United Kingdom: Polygenic susceptibility in breast cancer.

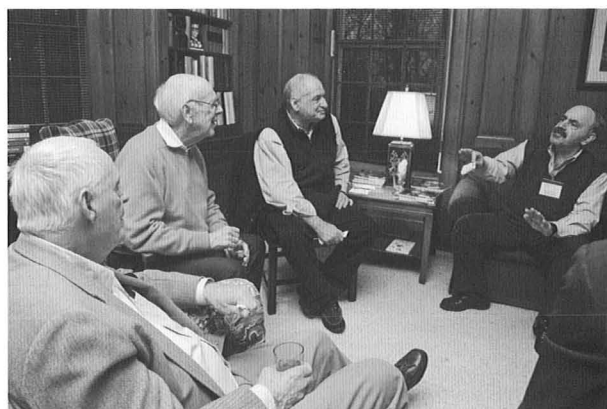
SESSION 5

Chairperson: R.A. Weinberg, Whitehead Institute for Biomedical Research, Cambridge, Massachusetts

- G. Dranoff, Dana-Farber Cancer Institute, Boston, Massachusetts: Balancing tumor immunity and inflammatory pathology.
- D.M. Epstein, OSI Pharmaceuticals, Inc., Farmingdale, New York: Evidence for pathological epithelial to mesenchymal transition and an alternate rationale for multitargeting in cancer.
- R. Kalluri, Harvard Medical School, Boston, Massachusetts: Tumor microenvironment controls the rate of cancer progression and metastasis.
- P. Dirks, Hospital for Sick Children, Toronto, Canada: Cancer stem cells as therapeutic targets.
- G.F. Vande Woude, Van Andel Institute, Grand Rapids, Michigan: Complexities and challenges: Met and malignant progression.

SESSION 6

General Discussion: What Next?



B. Clarkson, J. Watson, J. Schlesinger, R. Weinberg

Molecular Mechanisms Modulating Skeletal Muscle Mass and Function

April 6-9

FUNDED BY

Cold Spring Harbor Laboratory Corporate Sponsor Program

ARRANGED BY

A.L. Goldberg, Harvard Medical School

D.J. Glass, Novartis Institutes for Biomedical Research

Many conferences have focused on the early development of skeletal muscle, the roles of satellite cells, or contractile mechanisms; however, this conference reviewed the mechanisms for muscle homeostasis in the adult animal and human. As anyone who has had a limb immobilized will appreciate, the loss of muscle can be rapid and very hard to replace. Participants in the meeting considered questions such as What are the molecular mechanisms that occur in response to increased exercise that result in hypertrophy and/or fiber-type switching, and how does inactivity lead to fiber atrophy? How are protein synthesis, proteolysis, and gene expression altered in skeletal muscle during the wasting (cachexia) induced by cancer, cardiac failure, sepsis, and renal failure? How do cytokines and hormones influence the properties of muscle in normal and disease states?

Introductory Remarks:

J.A. Witkowski, Banbury Center, Cold Spring Harbor Laboratory

Introduction: Why this meeting?:

A.L. Goldberg, Harvard Medical School, Boston, Massachusetts

SESSION 1: Skeletal Muscle Development and Differentiation

Chairpersons: **S.-J. Lee**, Johns Hopkins University School of Medicine, Baltimore, Maryland, and **G. Pavlath**, Emory University School of Medicine, Atlanta, Georgia

R. Krauss, Mount Sinai School of Medicine, New York: Role of the Ig superfamily receptors CDo and neogenin in myogenesis.

M.A. Rudnicki, Ottawa Health Research Institute, Canada: Molecular mechanisms regulating satellite cell function.

A. Wagers, Harvard Medical School, Boston, Massachusetts: Regenerative potential of skeletal muscle stem cells.

G. Pavlath, Emory University School of Medicine, Atlanta, Georgia: Molecular control of satellite cell function during muscle growth.

K.A. Esser, University of Kentucky College of Medicine, Lexington: Clock genes and adult skeletal muscle structure and function.

SESSION 2: Muscle Disease and Dystrophy

Chairpersons: **N. Rosenthal**, European Molecular Biology Laboratory, Monterotondo, Italy, and **K.P. Campbell**, University of Iowa College of Medicine, Iowa City

M.R. Capecchi, University of Utah, Salt Lake City: Role of muscle lineage in muscle malignancy.

D.C. Guttridge, Ohio State University, Columbus: Muscle wasting in cancer cachexia and lessons learned from muscular dystrophy.

K.P. Campbell, University of Iowa College of Medicine, Iowa City: Muscular dystrophy as a complex disease: Insights from mouse models.

P. Munoz-Canoves, Centre for Genomic Regulation, Barcelona, Spain: Cytokine-mediated skeletal muscle hypertrophy.

N. Rosenthal, European Molecular Biology Laboratory, Monterotondo, Italy: Enhancing muscle regeneration. Maryland: Funding.



N. Rosenthal

SESSION 3: Ubiquitin-dependent Protein Breakdown in Muscle

Chairperson: A.L. Goldberg, Harvard Medical School, Boston, Massachusetts

D.J. Glass, Novartis Institutes for Biomedical Research, Cambridge, Massachusetts: Signaling pathways that mediate skeletal muscle atrophy and hypertrophy.

D. Attaix, Human Nutrition Research Center of Clermont-Ferrand, Ceyrat, France: Identification of polyubiquitinated substrates of the muscle proteasome.

M. Gautel, King's College, London, United Kingdom:

Sarcomeric links to ubiquitination and autophagy pathways.

S. Wing, McGill University, Montreal, Canada: Role of the USP19 deubiquitinating enzyme in muscle cell proliferation.

C. Patterson, North Carolina University, Chapel Hill: Multiple roles of ubiquitin ligases in muscle biology.

G. Nuckolls, National Institute of Arthritis and Musculoskeletal and Skin Diseases, Bethesda, Maryland: Funding.

SESSION 4: Regulation of Protein Breakdown and Synthesis in Muscle

Chairperson: D.J. Glass, Novartis Institutes for Biomedical Research, Cambridge, Massachusetts

A.L. Goldberg, Harvard Medical School, Boston, Massachusetts: Contributions of the autophagic and ubiquitin proteasome pathways to muscle atrophy.

M.F. Sandri, Dulbecco Telethon Institute at Venetian, Padova, Italy: Regulation of proteolytic systems during muscle wasting.

M. Spencer, University of California Los Angeles: Proteolysis and regulation of muscle mass by calpain 3.

S. Schiaffino, Venetian Institute of Molecular Medicine, Padova, Italy: Activity-dependent signaling pathways controlling muscle fiber size and type.

M.A. Ruegg, University of Basel, Switzerland: Role of the mTOR complex 1 (mTORC1) and mTORC2 in skeletal muscle.

P. Puigserver, Harvard Medical School, Boston, Massachusetts: Transcriptional mechanism modulating mitochondrial oxidative skeletal muscle function.

SESSION 5: Growth Factors and Therapeutic Challenges

Chairpersons: S. Schiaffino, Venetian Institute of Molecular Medicine, Padova, Italy, and **L.A. Leinwand**, University of Colorado, Boulder

S.-J. Lee, Johns Hopkins University School of Medicine, Baltimore, Maryland: Regulation of muscle growth by myostatin.

K. Wagner, The Johns Hopkins Hospital, Baltimore, Maryland: Clinical considerations for modulators of muscle growth.

D. Clemmons, University of North Carolina, Chapel Hill: IGF-1 and muscle cell growth and differentiation.

L.A. Leinwand, University of Colorado, Boulder: Exercise, diet,

and gender effects on skeletal muscle.

W.E. Mitch, Baylor College of Medicine, Houston, Texas: Mechanisms elicited by kidney disease to cause muscle protein losses.

K. Walsh, Boston University School of Medicine, Massachusetts: Akt-mediated growth of type IIb fibers reduces fat mass and improves metabolic parameters in obese mice.

SESSION 6: Meeting Summary and General Discussion

Chairperson: D.J. Glass, Novartis Institutes for Biomedical Research, Cambridge, Massachusetts



M. Capecchi, M. Rudnicki



W. Mitch, D. Attaix

Theoretical and Experimental Approaches to Auditory and Visual Attention

April 20–23

FUNDED BY

The Swartz Foundation

ARRANGED BY

H. Cohen, The Swartz Foundation

J.B. Fritz, University of Maryland

J.H. Reynolds, The Salk Institute For Biological Studies

Research in the human and macaque has provided a wealth of information on the neural mechanisms that mediate visual attention. Recent psychoacoustic and neurophysiological studies of attention in the auditory system, and research on interactions of visual and auditory attention, have added considerably to this picture. These studies have found parallels with visual attention mechanisms but have also raised new questions, such as the role of adaptive plastic changes in spectrotemporal receptive field shape during selective attention and the nature of the coordination of attention-driven changes at multiple processing levels from cochlea to cortex. The inherently temporal nature of auditory stimuli has also led to interesting insights into the temporal dynamics of auditory attention. The purpose of this workshop was to bring together experimentalists and theoreticians working in auditory and visual attention for a vibrant discussion of current research.

Introductory Remarks:

J.A. Witkowski, Banbury Center, Cold Spring Harbor Laboratory

Introductory Remarks:

J.H. Reynolds, The Salk Institute For Biological Studies, La Jolla, California, and

J.B. Fritz, University of Maryland, College Park

SESSION 1: Experimental and Theoretical Perspectives on Attention

Chairperson: **R. Desimone**, Massachusetts Institute of Technology, Cambridge

K. Nakayama, Harvard University, Cambridge, Massachusetts:
Perception, cognition, and action.

S.A. Hillyard, University of California, San Diego, La Jolla:
Attention facilitates multiple features in parallel in human
visual cortex.

J. Duncan, MRC Cognition & Brain Sciences Unit, Cambridge,
United Kingdom: Selective behavior and selective attention in

the human and monkey brain.

R.H. Wurtz, National Eye Institute, National Institutes of Health,
Bethesda, Maryland: Visual gateway to cortex and its
guardian attention in the LGN and TRN.

M. Carrasco, New York University, New York: Effects of spatial
and feature-based attention: Psychophysical and neuroimaging
studies.



SESSION 2: Experimental and Theoretical Perspectives on Attention (cont'd.)

Chairperson: L. Itti, University of Southern California, Los Angeles

L.F. Abbott, Columbia University, New York: Gating of multiple signals through attentional modulation.

D. Heeger, New York University, New York: The normalization model of attention.

SESSION 3: Auditory Attention: Human

Chairperson: S.A. Shamma, University of Maryland, College Park

R.J. Zatorre, McGill University, Montreal, Canada: Functional organization of human auditory cortex: Bottom-up features and top-down processes.

E. Hafer, University of California, Berkeley: A role for memory in shared attention.

R. Carlyon, MRC Cognition & Brain Sciences, Cambridge, United Kingdom: Effects of attention on auditory scene

analysis.

B. Shinn-Cunningham, Boston University, Massachusetts: The costs of switching auditory attention.

C. Alain, Rotman Research Institute of Baycrest Centre, Ontario, Canada: Top-down influences on memory and response-related activity for sound location (dual pathways, parietal cortex, and spatial memory).

SESSION 4: Visual Attention I: Theory and Experiment

Chairperson: R.H. Wurtz, National Eye Institute, National Institutes of Health, Bethesda, Maryland

S. Treue, German Primate Center, Goettingen, Germany: Spatial, feature, and object-based attention in area MT.

L. Itti, University of Southern California, Los Angeles: Quantifying bottom-up and top-down influences on gaze

allocation in humans and monkeys.

L. Chelazzi, University of Verona Medical School, Italy: Mechanisms of feature-selective attention in area V4 of the macaque (task relevance of responses in V4).

SESSION 5: Auditory Attention I: Neurophysiology of Auditory Attention

Chairperson: R. Carlyon, MRC Cognition & Brain Sciences, Cambridge, United Kingdom

T. Zador, Cold Spring Harbor Laboratory: Two components of attentional modulation in rat auditory cortex.

S.A. Shamma, University of Maryland, College Park: Attention

and rapid plasticity in auditory cortex.

J.B. Fritz, University of Maryland, College Park: What is the contribution of frontal cortex to an auditory attentional network?

SESSION 6: Visual Attention II

Chairperson: K. Nakayama, Harvard University, Cambridge, Massachusetts

W.S. Geisler, University of Texas at Austin: Mechanisms of fixation selection evaluated using ideal observer analysis.

J.C. Martinez Trujillo, McGill University, Montreal, Canada: Attentional modulation of sensory inputs at the level of single

neurons in MT.

P. Cavanagh, Harvard University, Cambridge, Massachusetts: Object-based integration and moving attention.

SESSION 7: Cell Type Specificity

Chairperson: L. Chelazzi, University of Verona Medical School, Italy

J.H. Reynolds, The Salk Institute for Biological Studies, La Jolla, California: Mapping the microcircuitry of attention.

J. Mitchell, The Salk Institute, San Diego, California: Attention-dependent response modulation varies between cell classes

in macaque V4.

X.-J. Wang, Yale University School of Medicine, New Haven, Connecticut: Stochastic and synchronous neural circuit dynamics underlying attentional gain modulation.

SESSION 8: Visual Attention III: Attentional Control

Chairperson: J. Duncan, MRC Cognition & Brain Sciences Unit, Cambridge, United Kingdom

J. Gottlieb, Columbia University, New York: Attention, motor planning, and decisions: The perspective from the parietal cortex.

S. Ganguli, University of California, San Francisco: 1-dim

dynamics of attention and decision making in LIP.

J. Mazer, Yale School of Medicine, New Haven, Connecticut: Cortical representations of attention and salience.

SESSION 9: Synchrony and Attention

Chairperson: S. Treue, German Primate Center, Goettingen, Germany

R. Desimone, Massachusetts Institute of Technology, Cambridge: Neural synchrony and selective attention.

E. Neibur, Johns Hopkins University, Baltimore, Maryland: Synchrony and the attentional state.

P. Tiesinga, University of North Carolina, Chapel Hill: Role of interneuron diversity in the cortical circuit for attention.

J. Swartz, The Swartz Foundation, East Setauket, New York: Closing remarks

To What Age Should We Be Expected to Work?

April 27-30

FUNDED BY

Oliver Grace Professorship Fund

ARRANGED BY

R.N. Butler, International Longevity Center

M.D. Hurd, Center for the Study of Aging, RAND Corporation

T.B.L. Kirkwood, Newcastle University

J.A. Witkowski, Banbury Center, Cold Spring Harbor Laboratory

Biomedical research on aging is having an impact on two rather different areas that are not yet closely linked. On the one hand, considerable progress has been made in elucidating some of the genetic changes and molecular processes that contribute to aging in experimental organisms such as *C. elegans*, the fruit fly, and mice. These processes can be manipulated, prolonging the life span of these organisms. On the other hand, our lives are being extended through better lifestyles and improved health care, and thus we are living longer; however, it seems that our maximum life spans are unchanged. Is extending the human life span an achievable or desirable goal? Should we concentrate instead on maintaining the quality of life of the extra years that we are now gaining? That is, can we live longer and still be healthy?

Introductory Remarks:

J.A. Witkowski, Banbury Center, Cold Spring Harbor Laboratory

Why a meeting on this topic?:

J.D. Watson, Cold Spring Harbor Laboratory

SESSION 1

Chairperson: M.D. Hurd, Center for the Study of Aging, RAND, Santa Monica, California

T.B.L. Kirkwood, Newcastle University, Newcastle upon Tyne, United Kingdom: So why does aging occur?

R.N. Butler, International Longevity Center, New York: Changing patterns of morbidity and mortality across the life course.

V.A. Bohr, National Institute on Aging, Baltimore, Maryland: Genome maintenance and DNA repair, changes with aging.

J.W. Shay, University of Texas Southwestern Medical Center, Dallas: Role of telomerase in aging and cancer.



SESSION 2

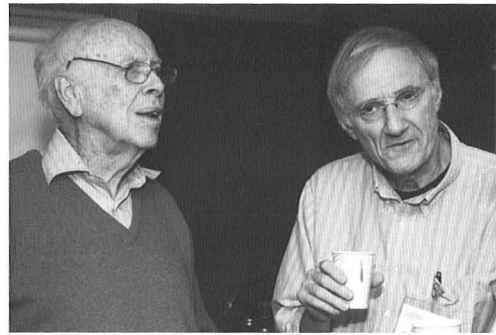
Chairperson: J.W. Shay, University of Texas Southwestern Medical Center, Dallas

D.C. Wallace, University of California, Irvine: Mitochondria and the pathophysiology of aging.

S.N. Austad, University of Texas Health Science Center at San Antonio: Comparative mechanisms of aging: An update.

J.M. Ordovas, Tufts University, Boston, Massachusetts: Gene-environment interactions modulating the risk of age-related disorders.

J.A. Faulkner, University of Michigan, Ann Arbor: Age-related changes in skeletal muscles from whole muscles to single fibers.



J. Watson, M. Raff

SESSION 3

Chairperson: T.B.L. Kirkwood, Newcastle General Hospital, Newcastle upon Tyne, United Kingdom

T.E. Johnson, University of Colorado, Boulder: Role of stress in specifying longevity and rate of aging.

L. Hayflick, University of California, San Francisco, The sea ranch: Manipulating the four aspects of the finitude of life.

B.N. Ames, Children's Hospital, Oakland, California: Delaying (or accelerating) the degenerative diseases.

C.B. Harley, Geron Corporation, Menlo Park, California: Telomerase-based therapies: Potential to hit a stem cell target.

M. Harman, Kronos Longevity Research Institute, Phoenix, Arizona: Hormonal changes and hormone replacement in older persons: Promise, hype, and realities.

SESSION 4

Chairperson: D.C. Wallace, University of California, Irvine

N. Barzilai, Albert Einstein College of Medicine, Bronx, New York: Strategies to prevent age-related diseases through human genetics.

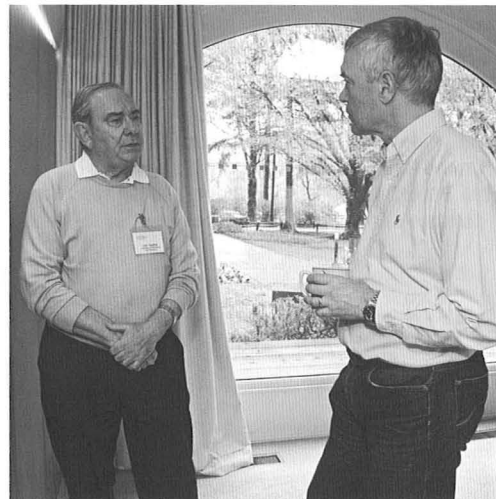
T.A. Salthouse, University of Virginia, Charlottesville: Implications of age differences in cognitive functioning for work.

R. Willis, University of Michigan, Ann Arbor: Cognitive capital and the future of work.

H. Fillit, The Alzheimer's Drug Discovery Foundation, New York: Clinical implications of cognitive aging.

T.T. Perls, Boston Medical Center, Massachusetts: The centenarian and supercentenarian looking glass.

D.R. Weir, University of Michigan, Ann Arbor: Does health limit work life?



L. Hayflick, J. Witkowski

SESSION 5

Chairperson: R.N. Butler, International Longevity Center, New York, New York

R. Sutch, University of California, Riverside: Working at advanced ages. Historical evidence and economic perspectives.

K. Christensen, University of Southern Denmark, Odense: Does extreme longevity lead to extreme levels of disability?

K. McGarry, Dartmouth College, Hanover, New Hampshire: Preferences and selection in insurance markets: Evidence from long-term care insurance.

M.D. Hurd, Center for the Study of Aging, RAND Corporation, Santa Monica, California: Demographics of aging around the world.



L. Lederman

General Discussion: Aging in the 21st century.

The Architectural Logic of Mammalian CNS

May 4-7

FUNDED BY

The William M. Keck Foundation, National Science Foundation Grant

ARRANGED BY

P.P. Mitra, Cold Spring Harbor Laboratory
L.W. Swanson, University of Southern California

This meeting was held to assess the progress of the Brain Architecture Project. The goals of the Project are to curate human neuroanatomical connectivity information from the existing literature into a knowledge base and to build suitable user and web interfaces. To complement these literature curation efforts, and to help shape the corresponding knowledge base schemas and geometrical templates for the user interface, it would be of great benefit to have an "outline" version of the connectivity diagram of the mammalian brain as well as a list of "rules" that the circuitry has been observed to follow. Participants were asked to present their overview of the architectural logic of the mammalian CNS. These might take the form of circuit diagrams at a coarse level for the full system, more elaborate circuit diagrams for subsystems or substructures that have sufficient generality across species or across brain regions, or specific rules. In contrast with the more familiar morphological approaches to comparative neuroanatomy, the meeting was concerned with the logic of the "highway map."

Introductory Remarks: **J.A. Witkowski**, Banbury Center, Cold Spring Harbor Laboratory

SESSION 1: Review of Brain Architecture Project

Chairperson: **P.P. Mitra**, Cold Spring Harbor Laboratory

P.P. Mitra, Cold Spring Harbor Laboratory: Introduction to the Brain Architecture Project and to the meeting theme.
J. Bohland, Cold Spring Harbor Laboratory: Progress and future challenges for the Brain Architecture Project.

H. Bokil, Cold Spring Harbor Laboratory: Analysis of spatial gene expression patterns in the Allen Brain Atlas.
J. Lin, Cold Spring Harbor Laboratory: A literature mining and curation system for the Brain Architecture Project.



SESSION 2: Introduction to Architectural Logic Problem

Chairperson: H. Breiter, Massachusetts General Hospital, Charlestown

L.W. Swanson, University of Southern California, Los Angeles:
Understanding the basic wiring diagram of the nervous system.
H.J. Karten, University of California, San Diego, La Jolla:
Conservation of microcircuitry across vertebrate phylogeny.

R. Kotter, Radboud University, Nijmegen, The Netherlands:
CoCoMac database interfaces for integrating primate brain connectivity.

SESSION 3: Architectural Logic Problem (cont'd.)

Chairperson: A. Graybiel, Massachusetts Institute of Technology, Cambridge

C.B. Saper, Beth Israel Deaconess Medical Center, Boston, Massachusetts: The flip-flop switch as a motif in brain architecture.
J.L. Price, Washington University, St. Louis, Missouri:
Hierarchical organization of systems for homeostasis and maintenance of the self.
D. Kleinfeld, University of California, San Diego: (Near) somatic

shunting as a circuit motif: Evidence from vision, somatosensation, and epilepsy.
C.C. Hilgetag, Jacobs University Bremen, Germany: Relating cortical connections to cortical architecture.
H. Barbas, Boston University, Massachusetts: Laminar-specific prefrontal pathways for excitatory and inhibitory control.

SESSION 4: Architectural Logic Problem (cont'd.)

Chairperson: M. Hawrylycz, Allen Institute For Brain Science, Seattle, Washington

S. Haber, University of Rochester, New York: Three-dimensional models of fiber tracts arising from specific cortical areas through the primate brain: Pathways to understanding human functional circuitry.

D.C. Van Essen, Washington University School of Medicine, St. Louis, Missouri: Cortical areas, hierarchies, and networks in monkeys and humans.

Discussion of Architectural Logic Problem

P.P. Mitra, Cold Spring Harbor Laboratory: White paper; strategizing for the large-scale connectivity project.

SESSION 5: Architectural Logic (cont'd.)

Chairperson: P. Freed, Columbia University, New York

J.D. Schmahmann, Massachusetts General Hospital, Boston:
Principles of organization of cerebral white matter pathways: Implications for the architecture and connections of cortical and subcortical nodes of distributed neural circuits.
N. Makris, Massachusetts General Hospital, Charlestown, and H. Breiter, Massachusetts General Hospital, Charlestown:
Methodological and logical challenges in scaling between circuits and function.

SESSION 6: Clinical Perspectives on Brain Architecture

Chairperson: P. Freed, Columbia University, New York

D.G. Herrera, Weill Medical College of Cornell University, New York: Clinical implications of a novel understanding of brain architecture.
J. Safdieh, Weill Medical College of Cornell University, New York: Circuitry in the classroom and the clinic.



J. Schmahmann, H. Breiter



J. Safdieh, P. Freed

Prion Strains: Origins, Mechanisms, and Implications for Disease

May 11-14

FUNDED BY

NIAID and the Medical Research Council, United Kingdom

ARRANGED BY

B. Caughey, NIAID Rocky Mountain Laboratories
J. Collinge, University College London
C. Soto, University of Texas Medical Branch
C. Weissmann, Scripps Florida

Multiple distinct strains of naturally occurring sheep scrapie can be passaged in mice. Such strains are classically distinguished by their biological properties: They produce distinct incubation periods and patterns of neuropathology in inbred lines of laboratory mice. Furthermore, strains can be reisolated in mice after passage in intermediate species with different PrP primary structures. The widely accepted protein-only hypothesis, if correct, must be able to explain how a single polypeptide chain could encode multiple disease phenotypes. Clearly, understanding how a protein-only infectious agent could encode such phenotypic information is of wide biological interest and raises intriguing evolutionary questions. Do other proteins behave in this way? The novel pathogenic mechanisms involved in prion propagation may be of far wider significance and relevant to other neurological and nonneurological illnesses.

Introductory Remarks: **J.A. Witkowski**, Banbury Center, Cold Spring Harbor Laboratory

SESSION 1: Strains: Definition, Concepts

Chairperson: **J. Collinge**, University College London, United Kingdom

J. Collinge, University College London, United Kingdom: Prion strains, transmission barriers, and neurotoxicity.



SESSION 2: Strain Typing: Biochemical

Chairperson: J. Collinge, University College London, United Kingdom

M.H. Groschup, Federal Research Institute for Animal Health, Greifswald-Insel Riems, Germany: Strain typing in animal TSE cases: Criteria for strain definitions.

J. Wadsworth, University College London, London, United Kingdom: Problems in defining human prion strains.

A. Hill, University of Melbourne, Australia: Role of PrP post-translational modifications as markers of prion strain type.

P. Gambetti, Case Western Reserve University, Cleveland, Ohio: Prion strains in human prion diseases.

SESSION 3: Strain Typing In Vivo and in Cell Culture

Chairperson: B. Caughey, NIAID Rocky Mountain Laboratories, Hamilton, Montana

G. Telling, University of Kentucky, Lexington: Transgenic analysis of CWD strains.

U. Agrimi, Istituto Superiore di Sanita, Rome, Italy: Strain typing of animal prions in natural hosts and by transmission to bank voles (*Myodes glareolus*).

H. Laude, Institut National de la Recherche Agronomique,

Jouy-en-Josas, France: Transmission of ruminant TSE to transgenic mice: Further insight about prion strain diversity in naturally infected hosts.

G. Zanusso, University of Verona, Italy: Intraspecies transmission of bovine amyloidotic spongiform encephalopathy.

SESSION 4: Structural Basis of Strain-ness

Chairperson: B. Caughey, NIAID Rocky Mountain Laboratories, Hamilton, Montana

W.K. Surewicz, Case Western Reserve University, Cleveland, Ohio: Recombinant prion protein amyloid: Molecular structure, strains, and infectivity.

I. Baskakov, University of Maryland Biotechnology Institute, Baltimore: Generating multiple strains of amyloid fibrils from a

single polypeptide chain.

D.S. Eisenberg, University of California, Los Angeles: Structural studies of amyloids, prions, and strains.

R.B. Wickner, NIDDK/NIH, Bethesda, Maryland: Yeast prion amyloid structure explains heritability of "strain" information.

SESSION 5: Structural Basis of Strain-ness (cont'd.)

Chairperson: S.B. Prusiner, University of California, San Francisco

B. Caughey, NIAID Rocky Mountain Laboratories, Hamilton, Montana:

Ultrastructure and strain comparison of underglycosylated, anchorless scrapie prion protein fibrils.

SESSION 6: Strain Biology In Vivo

Chairperson: S.B. Prusiner, University of California, San Francisco

B.W. Chesebro, NIAID, Rocky Mountain Laboratories, Hamilton, Montana: Role of anchorless prion protein in pathogenesis induced by different scrapie strains.

I. Vorberg, Technical University of Munich, Germany: Yeast prion aggregation propensities in mammalian cells.

J.C. Bartz, Creighton University, Omaha, Nebraska: Prion

strain interference.

D. Westaway, University of Alberta, Edmonton, Canada: The PrP-like Shadoo protein: Misfolding and in vivo variants.

M. Jeffrey, Veterinary Laboratories Agency, Midlothian, United Kingdom: Species, strain, and cell-associated changes in abnormal PrP processing following prion infection.

SESSION 7: Strain Biology In Vivo (cont'd.)

Chairperson: C. Soto, University of Texas Medical Branch, Galveston

J. Manson, Roslin Institute, Edinburgh, United Kingdom: Prion strains and host susceptibility.

S. Priola, Rocky Mountain Laboratories, NIAID/NIH, Hamilton, Montana: Factors influencing in vitro and in vivo scrapie strain phenotypes.

C. Weissmann, Scripps Florida, Jupiter: Do PrP-linked glycans contribute to prion strain determination?

J.G. Safar, University of California, San Francisco: Conformational intermediates, clearance, and species barrier of natural prion strains.

SESSION 8: Strain Generation and Propagation In Vitro

Chairperson: C. Soto, University of Texas Medical Branch, Galveston

S.B. Prusiner, University of California, San Francisco:
Protease-sensitive and -resistant strains of synthetic prions.

C. Soto, University of Texas Medical Branch, Galveston:
Generation of multiple new prion strains by in vitro PrP replication.

J. Castilla, Scripps Florida, Jupiter: In vitro studies of the transmission barrier.

S.W. Liebman, University of Illinois at Chicago: The birth of a foreign prion in yeast.

SESSION 9: Varia

Chairperson: S.B. Prusiner, University of California, San Francisco

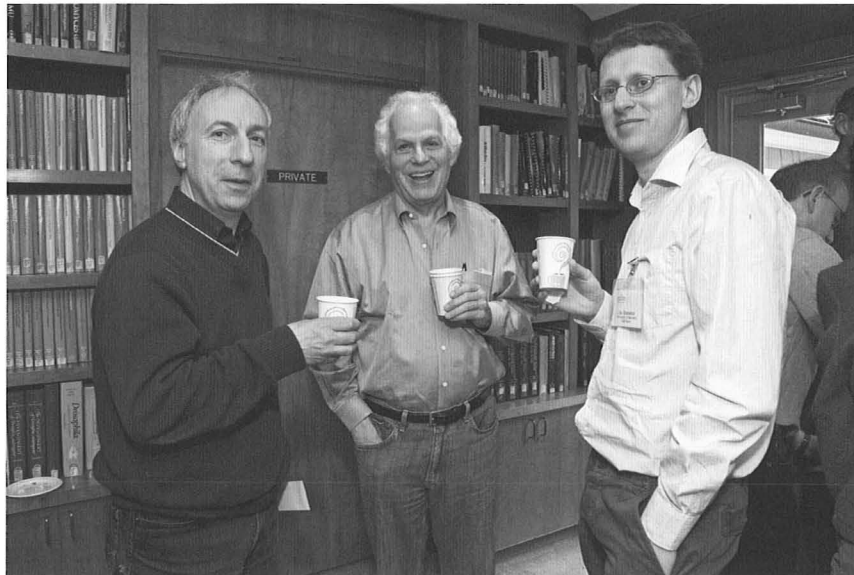
R.A. Bessen, Montana State University, Bozeman: Using lymphoreticular system replication-deficient prion strains to determine routes of prion neuroinvasion.

SESSION 10: General Discussion and Concluding Remarks

Chairperson: S.B. Prusiner, University of California, San Francisco



S. Liebman



D. Westaway, S. Prusiner, I. Baskakov

Identifying KRAS-targeted Therapeutic Approaches for Pancreatic Cancer

June 16-17

FUNDED BY **The Lustgarten Foundation for Pancreatic Cancer Research**

ARRANGED BY **C.J. Der**, University of North Carolina, Chapel Hill
N.K. Tonks, Cold Spring Harbor Laboratory

ADDITIONAL ORGANIZERS **K.A. Johnke**, The Lustgarten Foundation for Pancreatic Cancer Research
R. Hruban, Johns Hopkins Medical Institutions

KRAS mutations occur in 100% of pancreatic cancers, and this meeting focused on targeting the KRAS oncogene for novel therapeutics for pancreatic cancer treatment. Unfortunately, small GTPases such as Ras are not classically considered “druggable” targets, and earlier anti-Ras approaches have not been successful. Nevertheless, there is no doubt that anti-Ras therapeutics have huge potential in the treatment of pancreatic cancer. Two key uncertainties in such efforts are (1) what technologies will be most suitable for functional screens to identify targets for therapeutic intervention and (2) what model of cell/mouse systems should be used to apply these technologies? It is the goal of this meeting to identify the best technologies and systems, so that it will be possible to establish and apply genome-wide screens to identify novel modulators of KRAS-mediated pancreatic cancer growth.



Introductory Remarks: J.A. Witkowski, Banbury Center, Cold Spring Harbor Laboratory

SESSION 1

- I. **R.F. Vizza**, The Lustgarten Foundation, Bethpage, New York: Welcome and introduction
II. **R. Hruban**, Johns Hopkins Medical Institutions, Baltimore, Maryland: Goals and desired outcome of meeting
III. **R. Hruban**, Johns Hopkins Medical Institutions, Baltimore, Maryland: Overview of ongoing Lustgarten initiatives

IV. Model Systems

Moderator: C.J. Der, University of North Carolina, Chapel Hill

- Human model cell systems (hTERT-immortalized normal cells)
- Established pancreatic tumor cell lines
- Primary pancreatic tumor isolates
- Pancreatic tumor stem cells
- Mouse models of pancreatic cancer
- Mouse-model-derived cell cultures
- Transient KRAS activation and induction of cell senescence
- Invertebrate genetic model lethality screens

V. Wrap-up and Summary

Moderator: C.J. Der, University of North Carolina, Chapel Hill

SESSION 2

- I. **C.J. Der**, University of North Carolina, Chapel Hill: Welcome and day 1 review

II. Technical Approaches/Issues in RNAi

Moderator: B. Stillman, Cold Spring Harbor Laboratory, New York

- Targeting therapies
- Drug development and delivery
- RNAi library screening
- microRNA
- Gene arrays
- Proteomics
- Chemical libraries
- Phosphorylated proteins
- Secreted plasma proteins
- Genetic screens

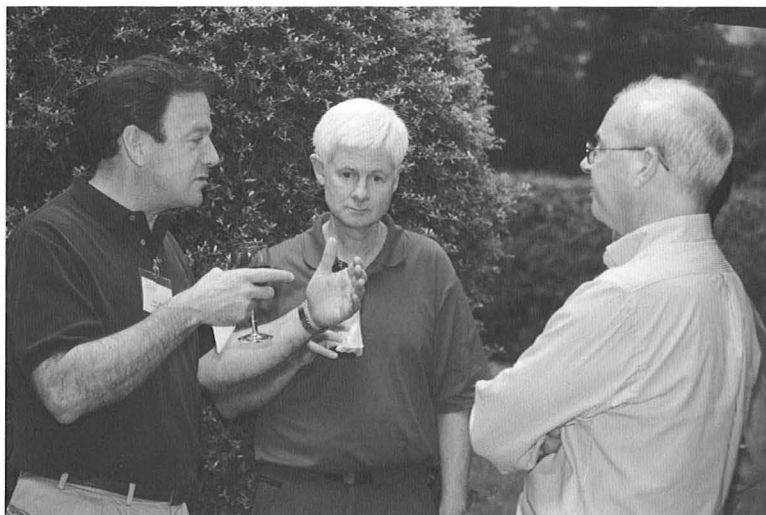
- III. **N. Tonks**, Cold Spring Harbor Laboratory: Wrap-up and summary

IV. Boxed Lunch—Continued Wrap-up

- V. **C.J. Der**, University of North Carolina, Chapel Hill, **N. Tonks**, Cold Spring Harbor Laboratory, **R. Hruban**, John Hopkins Medical Institutions, Baltimore, Maryland: Summary/finalize research plan
VI. **R.F. Vizza**, The Lustgarten Foundation, Bethpage, New York: Closing remarks



D. Bar-Sagi



A. Saltiel, K. Shannon, J. Gibbs

Plant Genetics and Gene Regulation

September 7–10

FUNDED BY **Cold Spring Harbor–Pioneer Collaborative Research Program**

ARRANGED BY **R. Martienssen**, Cold Spring Harbor Laboratory
S. Tingey, DuPont Experimental Station

Welcome: **R. Martienssen**, Cold Spring Harbor Laboratory, and **S. Tingey**, DuPont Experimental Station, Wilmington, Delaware

Minisymposium on Plant Genetics and Development I

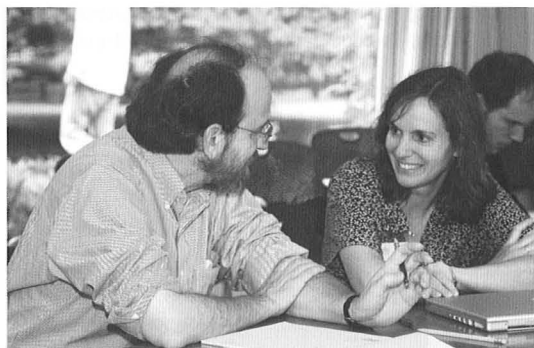
- R.S. Poethig, University of Pennsylvania, Philadelphia: The regulation of leaf shape in *Arabidopsis thaliana*.
R. Simon, Institut für Genetik, Düsseldorf, Germany: Regulation of plant stem cell fate by intercellular signaling.
J. Fletcher, USDA–University of California, Berkeley, Albany, California: Polycomb and trithorax activity in *Arabidopsis* organ formation: Role of the *ULT1* gene.
D. Jackson, Cold Spring Harbor Laboratory: Inflorescence architecture in maize.



S. Tingey, R. Martienssen

Minisymposium on Plant Genetics and Development II

- X.-W. Deng, Yale University, New Haven, Connecticut: An initial analysis of maize epigenomes and their relationship with transcriptional activity.
P. Green, University of Delaware, Newark: Global analysis of miRNAs and miRNA targets.
B.C. Meyers, University of Delaware, Newark: Small RNAs of maize and beyond.
R. Williams, DuPont experimental Station, Wilmington, Delaware: Gene expression profiling of miRNAs.
R. Martienssen, Cold Spring Harbor Laboratory: Inheritance and reprogramming of heterochromatin with RNAi.



R.S. Poethig, M. Timmermans



How Can We Improve Our Brains?

September 14–17

FUNDED BY

The Charles A. Dana Foundation

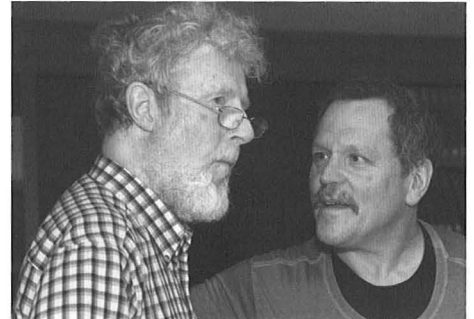
ARRANGED BY

E.R. Kandel, Columbia University

W.T. Dickens, Russell Sage Foundation

J.A. Witkowski, Banbury Center, Cold Spring Harbor Laboratory

It is the hope of every parent that their child will be bright and intelligent. Parents work to help their children's brain work better through providing education and stimulation, and society as a whole makes a tremendous commitment to the education of its young people. Are there data emerging from cognitive neuroscience that such education programs should take into account? Are there learning regimes that might be more effective than those typically found in the classroom? At the other end of life, is the hope of all of us that the normal decline in cognitive skills that accompanies aging will be slow? Might "brain exercises" maintain our brains at a higher level of functioning? Is there evidence that such exercises work? Are there effective pharmacological agents? In short, how can we best use the resources of society to help our brains work better throughout our lives?



J. Flynn, J. Schwartz

Welcome:

J.A. Witkowski, Banbury Center, Cold Spring Harbor Laboratory

Introductory Remarks:

J.D. Watson, Cold Spring Harbor Laboratory



SESSION 1: Overviews

Chairperson: W.T. Dickens, Russell Sage Foundation, New York

E.R. Kandel, Columbia University, New York: We are what we remember: Memory and the biological basis of individuality.

D.K. Detterman, Case Western Reserve University, Cleveland,

Ohio: General intelligence, achievement, and environmental effects.

R.E. Nisbett, University of Michigan, Ann Arbor: Intelligence and how to get it: Why schools and cultures count.

SESSION 2: Measurement

Chairperson: J.R. Flynn, University of Otago, Dunedin, New Zealand

C. Blair, New York University, New York: Improving fluid intelligence.

J.J. McArdle, University of Southern California, Los Angeles: Contemporary measurement issues in the evaluation of adult cognition.

W.T. Dickens, Russell Sage Foundation, New York: What is g?

R. Colom, Universidad Autonoma de Madrid, Spain: 4 + 2 ways to improve our brains.

SESSION 3: Memory and Plasticity

Chairperson: E.R. Kandel, Columbia University, New York

M.C. Potter, Massachusetts Institute of Technology, Cambridge: Conceptual short-term memory and attention.

M. Merzenich, University of California, San Francisco: Brain plasticity-based therapeutics.

F.P. de Lange, Radboud University, Nijmegen, The Netherlands: Structural brain changes following psychotherapy.

J.M. Schwartz, University of California School of Medicine, Los Angeles: Nonreductionist approaches to neuroscience: Neuroplasticity for the coming immaterialist era.



D. Halpern, E. Kandel

SESSION 4: Differences

Chairperson: D.K. Detterman, Case Western Reserve University, Cleveland, Ohio

R.J. Haier, UCI Medical Center, Irvine, California: Neuroimaging studies of learning: Do all brains work the same way?

D.F. Halpern, Claremont McKenna College, California: Sex differences in intelligence and their implications for national/state educational policies.

T.A. Salthouse, University of Virginia, Charlottesville: Mental exercise and mental aging.

L.S. Gottfredson, University of Delaware, Newark: The fragility of maximal performance.

E. Turkheimer, University of Virginia, Charlottesville: The relationship between poverty and the heritability of intelligence.



L. Gottfredson, D. Detterman

SESSION 5: Early Education

Chairperson: C. Blair, New York University, New York

W. Steven Barnett, Rutgers, The State University of New Jersey: New Brunswick: Early education's effect on IQ and achievement.

C.T. Ramey, Georgetown University, Washington, D.C.:

Preschool randomized controlled trials and population interventions to facilitate human competence.

L. Schweinhart, High/Scope Education Research Foundation, Ypsilanti, Michigan: The High/Scope Perry Preschool Study

through age 40.

M. Rosario Rueda, University of Granada, Spain: Enhancing brain function through cognitive training in young children.

J. Brooks-Gunn, National Center for Children and Families, New York: Long-term efficacy of early childhood education programs for poor children.

SESSION 6: Ability and Achievement

Chairperson: D.F. Halpern, Claremont McKenna College, Claremont, California

J.R. Flynn, University of Otago, Dunedin, New Zealand: Critical acumen: Step child of IQ tests and American education.

P.K. Kuhl, University of Washington, Seattle: How children learn: Can (should) we try to improve it?

D. Lubinski, Vanderbilt University, Nashville, Tennessee: Intellectually precocious youth with exceptional potential for scientific creativity: What we currently know about maximiz-

ing their development.

E. Hunt, University of Washington, Seattle: The workplace demands for cognition.

P.D. Zelazo, University of Minnesota, Minneapolis: Promoting the development of executive function prefrontal cortical function in children.

Where do we go from here?

Discussion Leaders:

E.R. Kandel, Columbia University, New York

W.T. Dickens, Russell Sage Foundation, New York



J. Watson, W. Dickens, M. Potter, R. Haier

Nutrient Sensing in Plants: What Can Other Model Organisms Tell Us?

September 21-24

FUNDED BY **Cold Spring Harbor Laboratory Corporate Sponsor Program**

ARRANGED BY **A.M. Jones**, University of North Carolina, Chapel Hill
D.P. Schachtman, Donald Danforth Plant Science Center

Nutrient sensing in response to mineral or carbon deficiency and enrichment is an important area of biological research in multicellular eukaryotes. However, the sensing mechanisms and the components of the signal transduction pathways that connect sensing to response are poorly elucidated. Recent progress has been made using different model organisms, but this is still an emerging and somewhat fragmented field of research. Therefore, the aim of this meeting was to gather together experts in the plant field with experts using other model systems to identify parallels among eukaryotes that will help to advance nutrient-sensing research across organisms.

Introductory Remarks: **J.A. Witkowski**, Banbury Center, Cold Spring Harbor Laboratory

SESSION 1: Nutrient Sensing/Sensors

Chairperson: W.B. Frommer, Carnegie Institute for Science, Stanford

J. Thevelein, Catholic University of Louvain, Belgium:

Transceptor-mediated nutrient sensing in yeast.

M. Kielland-Brandt, Technical University of Denmark, Kgs

Lynby: Model for transporter-like nutrient sensors: Sensing a chemical potential difference over a membrane.

W.B. Frommer, Carnegie Institute for Science, Stanford, California:

Making sense of nutrient sensing with the help of FRET sensors.

F. Tamanoi, University of California, Los Angeles: The TSC/Rheb/TOR signaling pathway in fission yeast.

C. Meyer, Institut Jean-Pierre Bourgin (JPB), Versailles, France: Role in nutrient signaling of the target of rapamycin (TOR) pathway in plants.



SESSION 2: Metal Sensing**Chairperson: M.L. Guerinot**, Dartmouth College

S. Puig, University of Valencia, Spain: The yeast *Saccharomyces cerevisiae* as a model organism to study copper and iron deficiencies.

M.L. Guerinot, Dartmouth College, Hanover, New Hampshire: Metal homeostasis in *Arabidopsis*.

SESSION 3: Nitrogen Sensing**Chairperson: N. von Wiren**, University of Hohenheim, Germany

G. Coruzzi, New York University, New York: A systems approach to nitrogen-regulatory networks and the virtual plant.
N. von Wiren, University of Hohenheim, Germany: Ammonium sensing in *Arabidopsis* roots.

B. Andre, Free University of Brussels, Belgium: Role of membrane transporters in amino acid signaling in yeast.
N. Crawford, University of California, San Diego: Signaling by inorganic nitrogen.

A. Gojon, Institut National de la Recherche Agronomique, Montpellier, France: Nitrogen sensing by NRT1.1 and its role in the regulation of root development in *Arabidopsis*.

B.G. Forde, Lancaster University, United Kingdom: Nitrate and glutamate sensing in *Arabidopsis* roots.
D.R. Bush, Colorado State University, Fort Collins: Anion regulation of root architecture.

SESSION 4: Sugar Sensing**Chairperson: A.M. Jones**, University of North Carolina, Chapel Hill

M. Johnston, Washington University Medical School, St. Louis, Missouri: A glucose-sensing reticulum in *S. cerevisiae*.
J. Sheen, Massachusetts General Hospital, Boston: Glucose and energy signaling networks.
J. Rutter, University of Utah School of Medicine, Salt Lake City: Regulation of glucose partitioning by pas kinase.
A.M. Jones, University of North Carolina, Chapel Hill: Glucose sensing through a novel receptor GAP.

P. Leon, Instituto de Biotechnologia, UNAM, Cuernavaca, Morelos, Mexico: Role of AB14 during sugar signaling in *Arabidopsis* early seedling development.

S.C. Smeekeens, University of Utrecht, The Netherlands: Sugar signaling and reprogramming of metabolism in plants.
C. Hoffman, Boston College, Chestnut Hill, Massachusetts: An Hsp90-Git7 requirement for glucose/cAMP signaling in *Schizosaccharomyces pombe*.

SESSION 5: Phosphate Sensing**Chairperson: S. Abel**, University of California, Davis

J. Paz Ares, Centro Nacional de Biotechnologia-CSIC, Madrid, Spain: Phosphate starvation signaling in *Arabidopsis*.
S. Abel, University of California, Davis: Phosphate sensing in root development.
T. Desnos, Centre National de la Recherche Scientifique, St. Paul-lez-Durance, France: Sensing low phosphate at the root tip.
L. Herrera-Estrella, Centro de Investigaciones y Estudios Avanzados, Irapuato, Guanajuato, Mexico: Phosphate availability alters lateral root development in *Arabidopsis* seedlings by modulating auxin sensitivity via a TIR1-dependent mechanism.
C. Xue, Duke University Medical Center, Durham, North

Carolina: The human fungal pathogen *Cryptococcus neoformans* senses plant signals to complete its sexual cycle.

M. Bucher, University of Cologne, Germany: Signaling pathway cross-talk in mycorrhizal phosphate uptake.
T.-J. Chiou, Agricultural Biotechnology Research Center, Taipei, Taiwan, Republic of China: microRNAs in sensing phosphate availability.
W.-R. Scheible, Max-Planck Institute for Molecular Plant Physiology, Potsdam, Germany: Impact of small RNAs and long-distance signaling in the regulation of macronutrient responses.

SESSION 6: Potassium Sensing**Chairperson: D.P. Schachtman**, Donald Danforth Plant Science Center, St. Louis, Missouri

A. Amtmann, University of Glasgow, Scotland: Linking K nutrition to metabolism and defense.
S. Luan, University of California, Berkeley: A calcium signaling pathway for low-potassium response in *Arabidopsis*.
D.P. Schachtman, Donald Danforth Plant Science Center, St. Louis, Missouri: Potassium signaling pathways in *Arabidopsis* roots.

SESSION 7: Conclusions and General Discussion**Chairpersons: A.M. Jones**, University of North Carolina, Chapel Hill, and **D.P. Schachtman**, Donald Danforth Plant Science Center, St. Louis, Missouri

G. Coruzzi, D. Schachtman

Who Are We?: Kinship, Ancestry, and Social Identity

October 6–9

FUNDED BY

The Richard Lounsbery Foundation

ARRANGED BY

A. Chakravarti, Johns Hopkins University School of Medicine

M.W. Foster, University of Oklahoma

J.A. Witkowski, Banbury Center, Cold Spring Harbor Laboratory

Genetics provides, on the one hand, a description of human beings that reflects their *biological* ancestry. On the other hand, cultural norms provide a description (often by self-identification) of *social* ancestry. These two descriptions need not be, and often are not, the same. However, clinical geneticists identify populations that are likely to be genetically more homogeneous by grouping individuals according to their ethnic characteristics. And in recent years, there has been a proliferation of companies offering DNA-based genealogies, established by examining a set of DNA markers, but discrepancies among ancestries revealed by genetic analysis and assumed by cultural descent may profoundly affect individuals' views of themselves. The importance of the relationship between genetic and ethnic identities requires careful, rational, and critical review. The advent of ever cheaper high-throughput genomic techniques together with the proliferation of companies offering DNA-based ancestry analysis highlights the need to begin discussions of this topic.



M. Foster, J. Witkowski



Welcome: **J.A. Witkowski**, Banbury Center, Cold Spring Harbor Laboratory

SESSION 1: Patterns of Global Human Variation

Chairperson: **M.W. Foster**, University of Oklahoma, Norman

L. Jorde, University of Utah, Salt Lake City: Genetics "race" and medicine.

S. Tishkoff, University of Pennsylvania, Philadelphia: Genetic variation in Africa.

J. Bertranpetit, Universitat Pompeu Fabra, Barcelona, Spain:

The Basques in the European genetic landscape: Changing views from changing genetic data and with sociological factors.

H. Ostrer, New York University, New York: Who are the Jews? A 4000-year genetic perspective.

SESSION 2: Kinship, Relationship, and Ancestry: Definitions, Measurements, and Meaning

Chairperson: **K.M. Weiss**, Pennsylvania State University, University Park

M.A. Stoneking, Max-Planck Institute for Evolutionary Anthropology, Leipzig, Germany: Genetic variation in worldwide human populations based on 1 million SNPs.

B.S. Weir, University of Washington, Seattle: F statistics and principal components for ancestry and kinship inference.

D.E. Reich, Harvard Medical School, Boston, Massachusetts: The genetic structure of 25 ethnolinguistically diverse Indian populations and their relationship to worldwide variation.

SESSION 3: Who Is Related to Whom? Population Features

Chairperson: **M.-C. King**, University of Washington, Seattle

M.V. Olson, University of Washington, Seattle: The right way to view kinship is one genome segment at a time.

E. Thompson, University of Washington, Seattle: Inferring identity by descent from genomic SNP data in the absence of pedigree structure information.

J. Sinsheimer, University of California, Los Angeles: Determining ethnic admixture using the Mendel Software Package.

SESSION 4: Who Is Related To Whom? Individual Features

Chairperson: **R. Cook-Deegan**, Duke University, Durham, North Carolina

I. Pe'er, Columbia University, New York: Whole genome, whole population mapping of hidden relatedness.

M.D. Shriver, Pennsylvania State University, University Park: Facial features, biogeographical ancestry, and admixture mapping.

D.B. Goldstein, Duke University, Durham, North Carolina: Rare and common variants in genetic history and genetic medicine.

SESSION 5: Ancestry, Race, and Complex Traits

Chairperson: **M.W. Foster**, University of Oklahoma, Norman

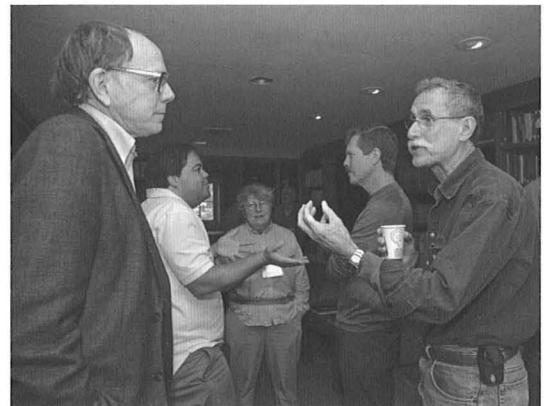
R.S. Cooper, Loyola University, Maywood, Illinois: Chronic disease has social causes.

C. Rotimi, National Human Genome Research Institute, Bethesda, Maryland: Health disparities: Is genomics a piece of the puzzle?

General Discussion

Introduction: **M.C. King**, University of Washington, Seattle

Moderator: **M. Foster**, University of Oklahoma, Norman



M. Olson, C. Bustamante, E. Thompson, L. Jorde, K. Weiss

D. Serre, The Cleveland Clinic, Ohio: Clines, clades, and why it matters.

M.F. Seldin, University of California, Davis: Ascertaining and applying markers informative for population genetic structure and substructure for studies of complex traits.

C.D. Bustamante, Cornell University, Ithaca, New York: Global distribution of genomic diversity underscores rich complex history of continental human populations.

K.M. Weiss, Pennsylvania State University, University Park: How true is fiction?



J. Bertranpetit, B. Weir

Oligonucleotide-directed Splicing: Therapeutic Strategies

October 14–17

FUNDED BY

Foundation to AVI, BioPharma, Inc., Cure Duchenne Foundation, Eradicate Duchenne, and Prosensa

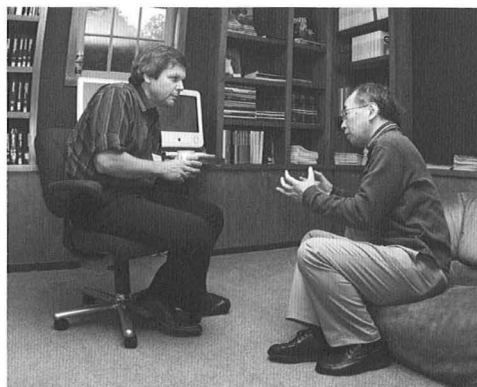
ARRANGED BY

E.P. Hoffman, Children's National Medical Center

A. Krainer, Cold Spring Harbor Laboratory

T.A. Partridge, Children's National Medical Center

Although research and development of small sequence-specific oligonucleotides as small-molecule drugs have been pursued for 25 years, it has been rather disappointing. However, very recent studies have shown that oligonucleotides can be used to modify the splicing patterns of pre-mRNAs in genetic disorders to produce a functional mRNA. Such a strategy might be widely applicable. In recent years, there have also been advances in nucleotide chemistry that have led to oligonucleotides that retain sequence-specific antisense activity while showing little or no protein binding or associated off-target effects, whereas other developments have improved intracellular delivery in a larger variety of tissues and cells. In light of these recent advances, this meeting was held to critically review progress on oligonucleotides as therapeutic agents. The goal was to end the meeting with a clearer understanding of the hurdles that remain in using oligonucleotides as therapeutic agents and to highlight research strategies that are likely to be fruitful.



E. Hoffman, S. Takeda



Welcome: J.A. Witkowski, Banbury Center, Cold Spring Harbor Laboratory
Introduction: E.P. Hoffman, Children's National Medical Center, Washington, D.C.

SESSION 1: Background

Chairperson: G.-J.B. van Ommen, Leiden University Medical Center, The Netherlands

G.-J.B. van Ommen, Leiden University Medical Center, The Netherlands: Cell animal and biomarker studies to improve exon skipping for DMD and extend the approach to other genes.
A. Krainer, Cold Spring Harbor Laboratory: Splicing correction as a therapeutic approach for spinal muscular atrophy.
R.T. Moxley, University of Rochester, New York: Myotonic dystrophy and exon skipping.
S.D. Wilton, University of Western Australia, Perth: Oligo design and evaluation: What can be expected?
L. Cartegni, Memorial Sloan-Kettering, New York: Modulation of alternative splicing in cancer.

SESSION 2: Preclinical

Chairperson: T.A. Partridge, Children's National Medical Center, Washington, D.C.

T.A. Partridge, Children's National Medical Center, Washington, D.C.: Exon choice, issues in detecting efficacy.
R. Kole, AVI BioPharma, Inc., Corvallis, Oregon: Splice switching oligomers: Technology progress and application to treatment of Duchenne muscular dystrophy.
S. Takeda, National Institute of Neuroscience, Tokyo, Japan: The significance of multiexon skipping of the dystrophin gene



P. Furlong, C. Miceli

by morpholino treatment.
E.P. Hoffman, Children's National Medical Center, Washington, D.C.: Local and systemic delivery in dogs.
H. Chao, Mount Sinai School of Medicine, New York: Spliceosome-mediated RNA *trans*-splicing for gene repair: From hemophilia to muscular dystrophy.

SESSION 3: Clinical

Chairperson: F. Muntoni, UCL Institute of Child Health, London, United Kingdom

G. Platenburg, Prosensa, Leiden, The Netherlands: Prosensa Program.
F. Muntoni, UCL Institute of Child Health, London, United Kingdom: Current efforts on morpholino antisense studies in patients with Duchenne and deletions of exon 51.

P. O'Hanley, AVI BioPharma, Inc., Portland, Oregon: AVI's clinical strategy for developing PMO-based dystrophin exon-skipping drugs for DMD.
C.F. Bennett, Isis Pharmaceuticals, Inc., Carlsbad, California: Therapeutic opportunities for oligonucleotides that modulate splicing.

SESSION 4: Strategies to Increase Potency

Chairperson: F. Muntoni, UCL Institute of Child Health, London, United Kingdom

Q.L. Lu, Carolinas Medical Center, Charlotte, North Carolina: Is PPMO safe to use in clinical trial and what regime should be used?
S. Jiang, Gene Tools, LLC, Philmath, Oregon: Vivo-morpholino oligomers induce potent exon skipping of dystrophin in cardiac and skeletal muscles of mice.
T. Yokota, Children's National Medical Center, Washington

D.C.: Exon skipping for dystrophic dogs.
S.F. Nelson, University of California Medical Center, Los Angeles, and M. Carrie Miceli, University of California, Los Angeles: HTS for enhancing exon skipping.
L. Garcia, Institute de Myologie, Paris, France: Dystrophin rescue by using exon skipping and/or *trans*-splicing approaches.

SESSION 5: Promoting the Pipeline in Novel Arenas

Chairperson: G.J. Vella, Charley's Fund, South Egremont, Massachusetts

B. Wentworth, Genzyme, Framingham, Massachusetts, and C. Nelson, Genzyme, Framingham, Massachusetts: Pathways to clinical trials.
J. Larkindale, Muscular Dystrophy Association, Tucson, Arizona: MDA.
B.D. Seckler, Charley's Fund, South Egremont, Massachusetts: Charley's Fund.

D. Miller, CureDuchenne, Corona del Mar, California: CureDuchenne.
W. Quirk, Foundation to Eradicate Duchenne, Inc., Alexandria, Virginia: FED.
P. Furlong, Parent Project Muscular Dystrophy, Middletown, Ohio: Parent Project.

Taking on New Complexities in SMA Biology

October 19–22

FUNDED BY

Spinal Muscular Atrophy Foundation

ARRANGED BY

K. Chen, SMA Foundation
C.E. Henderson, Columbia University
T.M. Jessell, Columbia University
C. Joyce, SMA Foundation
M. Winberg, SMA Foundation

The goal of all those working on human genetic disorders is to develop therapies that will alleviate, if not cure, the disorder. This requires identifying therapeutic targets at different organizational levels and determining how best to reach those targets with current or new tools. To this end, the working sessions of this fourth SMA meeting were organized around three themes: Cellular targets (nerve muscle, glia, and their contacts); molecular targets (SMN2 and downstream molecules); and functional targets (SMA phenotypes in models and humans). The expectation was that the meeting would help to identify and promote collaborative experiments, as well as stimulate rapid translation of research ideas into therapeutics development and clinical research



K. Klinger, T. Jessell

Welcome and Introductions:

Introductions and Welcome:

Introduction of Keynote Speaker:

Keynote Talk and Discussion:

J.A. Witkowski, Banbury Center, Cold Spring Harbor Laboratory

D. Singh, SMA Foundation, New York and Organizing Committee

D.C. DeVivo, Columbia University Medical Center, New York

K.P. Campbell, University of Iowa College of Medicine, Iowa City

SESSION 1: Cellular Targets

Chairpersons: **M. Sendtner**, Universitat Wuerzburg, Germany, and **G.J. Bassell**, Emory University, Atlanta, Georgia

G.D. Fischbach, Simons Foundation, New York: Reduced Ach release from type II SMA motor axons.

W. Thompson, University of Texas, Austin: Abnormal muscle development in a mouse model of SMA.

B.D. McCabe, Columbia University Medical Center, New York:

SMN in *Drosophila*: Roles in NMJ Physiology.

J.M. Shefner, SUNY Upstate Medical University, Syracuse:

Motor unit number estimation in a mouse model of SMA.

M. Sahin, Children's Hospital Boston, Massachusetts: RNA targets of SMN in axons.

SESSION 2: Cellular Targets

Chairpersons: **W. Thompson**, University of Texas, Austin, and **B.D. McCabe**, Columbia University Medical Center, New York

C.-P. Ko, University of Southern California, Los Angeles: Synapse loss in the SMN 7 mouse model of spinal muscular atrophy.

S.J. Burden, New York University Medical School, New York: Role of SMN1 in skeletal muscle.

G.Z. Mentis, NINDS/NIH, Bethesda, Maryland: Significant motor neuronal loss and altered synaptic input and excitability of lumbar motor neurons in SMA mice.

R.S. Finkel, Children's Hospital of Philadelphia, Pennsylvania:

Electrophysiological evidence for impaired neuromuscular transmission in children with SMA.

E. Tizzano, Hospital Saint Pau, Barcelona, Spain: SMA during human development: Different pathogenic responses of muscle and motor neurons.

SESSION 3: Cellular Targets

Chairpersons: **S.R. Jaffrey**, Cornell University, New York, and **C.L. Lorson**, University of Missouri, Columbia

G.J. Bassell, Emory University, Atlanta, Georgia: Interactions of SMN with β -actin mRNA-binding proteins important to axonal growth.

C.E. Beattie, The Ohio State University, Columbus: Generating a genetic model of SMA in zebrafish.

M. Sendtner, Universität Würzburg, Germany: Valproic acid, a drug candidate for spinal muscular atrophy, blocks axon

growth and excitability in motor neurons.

H. Wichterle, Columbia University, New York: Characterization of embryonic stem-cell-derived SMA motor neurons.

K. Eggan, Harvard University, Cambridge, Massachusetts: An in vitro model for SMA based on the differentiation of disease-specific human embryonic stem cell lines.

SESSION 4: Molecular Targets

Chairpersons: **S. Paushkin**, PTC Therapeutics, Inc., South Plainfield, New Jersey, and **K.H. Fischbeck**, NINDS/NIH, Bethesda, Maryland

L. Pellizzoni, Columbia University Medical Center, New York: SMN and pre-mRNA splicing.

C.L. Lorson, University of Missouri, Columbia: Readthrough-inducing compounds in SMA: SMN readthrough increases functionality compared to SMN₇.

S. Artavanis-Tsakonas, Harvard Medical School, Boston, Massachusetts: Modeling spinal muscular atrophy in invertebrates.

G.J. Lutz, Drexel University College of Medicine, Philadelphia, Pennsylvania: Preclinical studies of oligonucleotide-mediated SMN expression in mice with spinal muscular atrophy.

L.L. Rubin, Harvard University, Cambridge, Massachusetts: A high-content screen to identify molecules and pathways that regulate survival of motor neuron levels in motor neurons.

SESSION 5: Functional Targets

Chairpersons: **K. Chen**, SMA Foundation, New York, and **J.D. Porter**, NINDS/NIH, Bethesda, Maryland

C.J. Sumner, Johns Hopkins University, Baltimore, Maryland: Development of the motor unit in SMA mice and effects of HDAC inhibition.

P. Aebischer, Swiss Federal Institute of Technology, Lausanne, Switzerland: Gene therapy for SMA: Proof of principle in transgenic mice and scale-up issues.

K.W. Klinger, Genzyme Corporation, Framingham,

Massachusetts: Possibilities for gene therapy in SMA.

P. Kaufmann, Columbia University, New York: The natural history of spinal muscular atrophy: Preliminary findings from the PNCr study.

M. Winberg, SMA Foundation, New York: Developments in mice: An update on Foundation preclinical activities.

Closing Remarks

Moderator: **T.M. Jessell**, Howard Hughes Medical Institute, Columbia University, New York



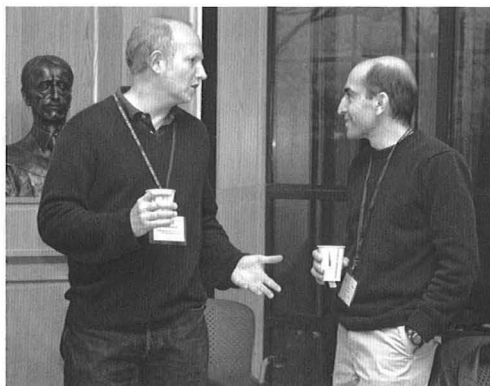
Epigenetics: Mechanisms and Regulation

December 7–10

FUNDED BY **Cold Spring Harbor Laboratory Corporate Sponsor Program**

ARRANGED BY **S.L. Berger**, Wistar Institute
R. Shiekhattar, Center for Genomic Regulation
A. Shilatifard, Stowers Institute for Medical Research

The term “epigenetics” has been used very loosely to cover states ranging from dynamic, short-lived, chromatin-mediated regulation to long-term alteration of chromatin and other extrachromosomal proteins in nonreplicating cells. Given that epigenetics now encompasses such a diversity of phenomena, it was felt that a discussion meeting was needed to reassess what phenomena are epigenetic. Themes of the meeting included How shall epigenetics be defined—narrowly or broadly? What are the phenotypes associated with epigenetics? How are epigenetic states regulated? What are the links between epigenetics and human diseases? What are the understudied areas within epigenetics?



R. Martienssen, D. Moazed

Welcome: **S. Berger**, Wistar Institute, Philadelphia, Pennsylvania,
R. Shiekhattar, Center for Genomic Regulation, Barcelona, Spain
A. Shilatifard, Stowers Institute for Medical Research, Kansas City, Missouri

SESSION 1: Definition of “Epigenetics” and Potential Mechanisms

Chairperson: **R. Shiekhattar**, Center for Genomic Regulation, Barcelona, Spain

Speakers

M. Grunstein, University of California, Los Angeles School of Medicine
S. Henikoff, Fred Hutchinson Cancer Research Center, Seattle, Washington

Discussion

Speakers

R. Kingston, MGH, Harvard Medical School, Boston, Massachusetts
M. Ptashne, Memorial Sloan-Kettering Cancer Center, New York
B. Stillman, Cold Spring Harbor Laboratory

Discussion

SESSION 2: DNA-binding Factors

Chairperson: **K. Struhl**, Harvard Medical School, Boston, Massachusetts

Speakers

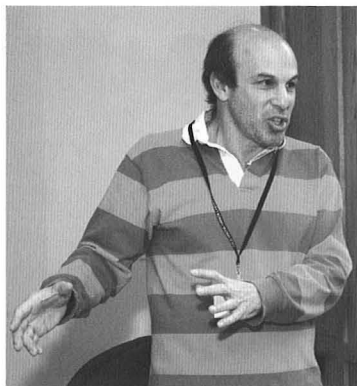
G. Felsenfeld, NIDDK/NIH, Bethesda, Maryland
V. Pirrotta, Rutgers University, Piscataway, New Jersey
K. Struhl, Harvard Medical School, Boston, Massachusetts

Discussion

Speakers

J. Widom, Northwestern University, Evanston, Illinois
R. Young, Whitehead Institute for Biomedical Research, Cambridge, Massachusetts
K. Zaret, Fox Chase Cancer Center, Philadelphia, Pennsylvania

Discussion



G. Struhl

SESSION 3: DNA Methylation

Chairperson: M. Bartolomei, University of Pennsylvania, Philadelphia

Speakers

S. Baylin, Johns Hopkins University School of Medicine, Baltimore, Maryland

M. Bartolomei, University of Pennsylvania, Philadelphia

A. Bird, University of Edinburgh, United Kingdom

Discussion

Speakers

K. Helin, Copenhagen Biocenter, Denmark

R. Martienssen, Cold Spring Harbor Laboratory

P. Jones, University of Southern California, Los Angeles

Discussion

SESSION 4: Histone Modifications

Chairpersons: A. Shilatifard, Stowers Institute for Medical Research, Kansas City, Missouri, and **S. Berger**, Wistar Institute, Philadelphia, Pennsylvania

Speakers

S. Berger, Wistar Institute, Philadelphia, Pennsylvania

B. Bernstein, Broad Institute Pathology, Charlestown, Massachusetts

T. Kouzarides, University of Cambridge, United Kingdom

Discussion

Speakers

D. Reinberg, New York University School of Medicine, New York

A. Shilatifard, Stowers Institute for Medical Research, Kansas City, Missouri

Y. Zhang, Howard Hughes Medical Institute, University of North Carolina, Chapel Hill

Discussion

SESSION 5: RNA AND RNAi

Chairperson: T. Kouzarides, University of Cambridge, United Kingdom

Speakers

S. Grewel, National Cancer Institute, National Institutes of Health, Bethesda, Maryland

G. Hannon, Cold Spring Harbor Laboratory

M. Kuroda, Harvard University, Boston, Massachusetts

Discussion

Speakers

J. Lee, Massachusetts General Hospital, Boston

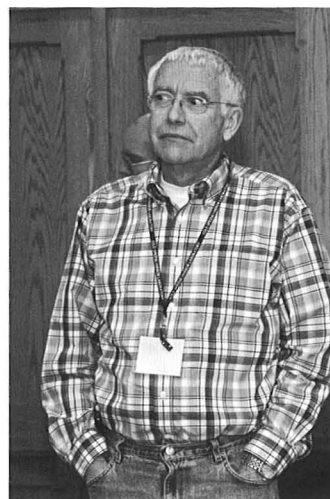
D. Moazed, Harvard Medical School, Boston, Massachusetts

R. Shiekhattar, Center for Regulation of Genome, Barcelona, Spain

Discussion



R. Kingston, M. Ptashne, A. Bird



P. Jones

BANBURY CENTER GRANTS

<i>Grantor</i>	<i>Program</i>	<i>Duration of Grant</i>	<i>2008 Funding</i>
FEDERAL SUPPORT			
NIH–National Institute of Allergy and Infectious Diseases; Office of Rare Diseases	Prion Strains: Origins, Mechanisms, and Implications for Disease	2008	25,000*
NIH–National Institute of Mental Health (through a grant to University of Illinois)	Recent Advance and a Multilevel Analysis from FMRP Biology to Clinical Trials	2008	53,914*
NONFEDERAL SUPPORT			
<i>Meeting Support</i>			
AVI BioPharma, Inc.	Oligonucleotide-directed Splicing: Therapeutic Strategies	2008	10,800*
Clay Mathematics Institute	Algebraic Statistics, Machine Learning, and Lattice Spin Models	2008	13,273*
Cold Spring Harbor–Pioneer Collaborative Research Program	Plant Genetics and Gene Regulation	2008	43,526
Cure Duchenne Foundation	Oligonucleotide-directed Splicing: Therapeutic Strategies	2008	10,800*
The Dana Foundation	How Can We Improve Our Brains?	2008	48,600*
Foundation to Eradicate Duchenne	Oligonucleotide-directed Splicing: Therapeutic Strategies	2008	10,800*
Oliver Grace Professorship Fund	To What Age Should We Work?	2008	43,810*
The W.M. Keck Foundation	The Architectural Logic of Mammalian CNS	2008	33,155
Richard Lounsbery Foundation	Who Are We? Kinship, Ancestry, and Social Identity	2008	46,767*
The Lustgarten Foundation for Pancreatic Cancer Research	Identifying KRAS-targeted Therapeutic Approaches for Pancreatic Cancer	2008	15,181*
Medical Research Council, UK	Prion Strains: Origins, Mechanisms, and Implications for Disease	2008	29,576*
OSI Pharmaceuticals, Inc.	How Will We Be Able to Cure Most Cancers?	2008	56,689*
Prosenza	Oligonucleotide-directed Splicing: Therapeutic Strategies	2008	10,800*
Spinal Muscular Atrophy Foundation	Taking on New Complexities in SMA Biology	2008	54,538*
The Swartz Foundation	Theoretical and Experimental Approaches to Auditory and Visual Attention	2008	51,497*
Stanley Trotman, Jr. Trust	Genes and the Environment: New Strategies for Research on Multiple Sclerosis	2008	22,554*

* New grants awarded in 2008

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