BANBURY CENTER DIRECTOR'S REPORT

The year 2017 was one of transition for the Banbury Center: It was with exhilaration that I took the baton from Jan Witkowski, who spent 30 remarkable years cultivating critical scientific discourse at the Center. Despite the change in leadership, activities at Banbury continued to be guided by the Center's mission to further scientific knowledge and the well-being of society. The year also marked 40 years since the Banbury Center was officially opened. In his speech, "How Scientists Work," at the 1977 dedication ceremony, Francis Crick pointed to small meetings as the best way for scientists to share and inspire new ideas and strategies (McElheny 2003). Despite technological advances in communication that have allowed a more interconnected world, Crick's sentiment still proves true with each small meeting convened at Banbury. The breadth of Banbury meetings in 2017, spanning discovery and translational science, public health, policy, education, and innovation, reflected the ever-growing need for multisector and multidisciplinary engagement at small meetings across a broad range of issues.

By the Numbers

In 2017, the Banbury Center hosted 46 activities and events, including Banbury meetings, laboratory retreats, and courses directed by the Watson School of Biological Sciences (Immunology; Physical Biology of the Cell) and the Meetings and Courses Program (Workshop on Pancreatic Cancer; Vision: A Platform for Linking Circuits, Behavior, and Perception; Neural Data Science; Autism Spectrum Disorder; Cellular Biology of Addiction; Workshop on Leadership in Bioscience; Scientific Writing Retreat).

A total of 536 individuals took part in Banbury meetings, with 72% marking their first occasion. Participants were drawn from 28 countries, spread across six continents; Antarctica continues to elude representation at Banbury meetings. The 79% of participants from the United States spanned 36 states and territories. The largest portion of Banbury meeting attendees, representing academic organizations (71%), participants from industry (15%), other not-for-profit organizations (9%), governments (4%), and publishing/writing (1%), brought diverse perspectives and new cross-sector relationships. Banbury continues to strive for gender diversity: Women represented 31% of 2017 participants, and we aim to improve this ratio in the coming years.

In 2017 Banbury continued to attract financial support from across sectors, with the largest funding drawn from not-for-profit organizations (59%). For-profit organizations constituted 30% of funding, with the Cold Spring Harbor Laboratory Corporate Sponsor Program (CSP) accounting for more than half of that figure. The CSP funds were absolutely vital to ensuring that Banbury was able to convene cutting-edge meetings in 2017; we continue to be grateful to those member organizations and to Cat Donaldson in recruiting membership and Michelle Corbeaux for coordinating participation in meetings.

Neuropharmacology and Human Stem Cell Models, September

Discovery and Translational Science

Banbury's year began with *Chemiexcitation in Human Disease and Aging*, a meeting that exemplified multidisciplinary engagement, with experts from chemistry, pathology, aging, and neurodegeneration among those brought together to explore the mechanisms and principles underlying chemiexcitation's pathological consequences. In the same way, *NLRs Sans Frontières* brought the plant and animal research communities together to exchange new and unpublished data on a microbial recognition mechanism common to both kingdoms, the NLR proteins. Although the *Enhanceropathies: Enhancer Function Variation in Animal Development, Morphological Variation, and Disease* meeting convened a relatively less diverse group of experts, it likewise sought to identify core principles underlying biological function (or dysfunction), focusing in this case on consequences of changes to noncoding regulatory sequences in DNA.

Two meetings targeted regulated cell death, kicking off in April with *Ferroptosis: A Critical Review* and circling back after Thanksgiving for *Regulated Necrosis: Pathways and Mechanisms*. The former centered on a relatively new addition to the necrotic cell death family, so-named because of its dependence on iron. Highly productive discussions from the meeting shaped a review paper (see publications list at the end of this report), and the burgeoning field of researchers has now outgrown Banbury; a Cold Spring Harbor Asia meeting will pick up the conversation in November 2018. The second meeting, held in autumn, targeted the most well-characterized mode of regulated cell death: necroptosis. This pathway is implicated in viral infections as well as cancer, ischemic injury, and a number of inflammatory conditions. Participants at this meeting were challenged to share their newest research findings to inspire new ideas to move the field forward.

Cancer was in the crosshairs in 2017, with two meetings asking new questions in established research areas. In April, *Better Cancer Therapy from Redox Biology* assessed the complexity of redox regulation in the context of cancer biochemistry and therapy. Later in the year, potential mechanisms underlying anticancer effects of an established drug were explored in *Metformin: Translating Biology into the Clinic*.

Three meetings built on Banbury's strong history in neuroscience. September's *Neuropharmacology and Human Stem Cell Models* meeting centered on the latest advances in generating human stem cell–derived disease models and resources for therapeutic discovery. The ALS Association's meeting in October—*Cell Biology of ALS: Emerging Themes from Human Genetics*—identified

new strategies in the fight against this neurological disease, focusing on new research to uncover detailed pathways involved in vesicular trafficking, autophagy, DNA damage, and neuroinflammation. Finally, the year's meetings concluded with *Post-Traumatic Neuroinflammation: Roles in Pathogenesis of Long-Term Consequences and Repair*, which convened researchers studying the cellular and molecular responses to injury and infection, with physician scientists treating patients affected by stroke, traumatic brain injury, and perinatal brain injury to share data and identify areas for collaboration.

Public Health

Among the earliest Banbury Center meetings were those confronting issues of environmental and public health, including the hazards of cigarette smoking (1979) and concerted efforts in combating AIDS (1983). These are areas in which Banbury-style meetings are especially critical to convening multidisciplinary groups, from experts in pathogenicity and toxicity to epidemiologists, social scientists, community health workers, and policy makers. Banbury's 2017 schedule included three meetings in this vein. May's *Maximizing Impact of New HIV Prevention Technologies in Sub-Saharan Africa,* supported by the Bill & Melinda Gates Foundation, framed a productive strategy session around three broad questions: What do we know?, What do we need to know?, and What do we need to do next? The group's diversity in sector, field, research, and in-country experience was critical, especially during the final sessions during which participants developed a model to integrate major conclusions and recommendations. Key findings were presented at the World STD and HIV Congress.

Although the HIV meeting focused on prevention strategies heavily based on risk and barriers to uptake, two additional meetings paired public health needs with new scientific strategies. The first centered on Lyme disease, well-timed in a year with predictions of escalating outbreaks. *Protective Immunity and Vaccines for Lyme Disease,* made possible through the support of the Steven and Alexandra Cohen Foundation, explored best strategies for vaccine development, with lively discussion despite a forceful autumn storm and subsequent power outage across the estate. Capping Lyme disease activities in 2017, a report on next-generation Lyme disease diagnostics was published, an output of a 2016 meeting (see publications list for detail). The second meeting, July's *Opportunities for Reduction of Aflatoxin Contamination of Food* tackled the fungal contamination of food supplies, a global issue affecting health, agriculture, and trade. Experts at this

M. Levine, B. Ren, A. Rada-Iglesias

Banbury meeting examined opportunities to reduce aflatoxin contamination through production of abiotic stress-resistant crop varieties, biocontrol, and host-induced gene silencing.

Policy, Strategy, and Education

The pastoral setting of the Center and tradition of inspiring discussions in a confidential environment make Banbury an ideal site for important policy discussions, internal strategy development, and intensive training. In addition to the summer courses, we once again welcomed Boehringer Ingelheim Fonds for their North American retreat, *Communicating Science*, during which the foundation's Ph.D. fellows spent nearly 1 week learning and applying tools across the communication spectrum. Two other returning groups were the Integrated Translational Science Center (ITSC) and the Lustgarten Foundation, convening members and external experts to monitor progress, develop strategy, and strengthen collaboration. Funded by the National Cancer Institute, the ITSC works to bridge the gap between bench and bedside, bringing together innovative research at Cold Spring Harbor Laboratory and The Jackson Laboratory with the clinical expertise of SWOG's physician investigators. Representatives of the three groups convened at Banbury to discuss pressing challenges in oncology and to brainstorm collaborative projects to address unmet needs. Similarly working toward a world without cancer, the Lustgarten Foundation returned to the Conference Room for their 2017 Scientific Meeting, which provided an opportunity for the Scientific Advisory Board, as well as Foundation-supported investigators, to discuss research and strategy, identifying the most promising new avenues to bolster progress in the field. Marking their first Banbury visit, *Project Santa Fe* convened leadership from innovative clinical laboratories in March to develop strategies for services that maximize impact, improve patient outcomes, and cut costs.

S. Schutzer, A. Marques, J.W.R. Hovius

The year also found Banbury embracing the surge of innovation and entrepreneurship in the biosciences: Banbury joined efforts with the Keystone for Incubating Innovation in Life Sciences Network (KIILN) for July's *Foundation2017*. Tents were erected on the estate to host nearly 70 bioscience entrepreneurs, investors, translational researchers, and industry leaders for a 1-day retreat that included panel sessions and plenty of informal networking. This nontraditional event was an excellent opportunity for attendees from the tristate area to experience Banbury while discussing practical challenges in bio-entrepreneurship and broadening their networks.

It Takes a Village

Finally, it is with great humility that I acknowledge those who keep the Banbury Center running at such a high level, and who were critical to ensuring continuity of quality during my transition to director. Michelle Corbeaux and Pat Iannotti power the meetings and events with expert coordination and organization. Basia Polakowski oversees our three residence buildings, ensuring our guests are comfortable, while the Culinary Services team keeps them well-fed, and the Audiovisual staff ensures technology supports rather than distracts. Jose Pena Corvera, John Shea, and Paulo Krizanovski look after 55 acres of impeccable grounds, and the entire Facilities team quite literally keep us running. Hakon Heimer has continued to be essential to the development of Banbury's pipeline of mental illness–focused meetings, and our extensive collaboration with the Meetings and Courses Program broadens Banbury's portfolio of activities.

Of course, the Banbury Center's mere existence and international reputation are owed to Charles Robertson and the Robertson family, to Bruce Stillman and James Watson, and especially to my esteemed predecessor, Jan Witkowski.

Rebecca Leshan
Executive Director

PUBLICATIONS

Branda JA, Body BA, Boyle J, Branson BM, Dattwyler RJ, Fikrig E, Gerald NJ, Gomes-Solecki M, Kintrup M, Ledizet M, Levin AE, et al. 2017. Advances in serodiagnostic testing for Lyme disease are at hand. *Clin Infect Dis* doi: 10.1093/cid/cix943.

Jakubowski H. 2017. Homocysteine editing, thioester chemistry, coenzyme a, and the origin of coded peptide synthesis. *Life (Basel)* **7:** E6.

Korf BR, Blitzer MG, Demmer LA, Feldman GR, Watson MS. 2017. Report on the Banbury Summit Meeting on medical genetics training in the genomics era [Commentary]. *Genet Med* **19:** doi: 10.1038/gim.2017.38.

McElhenu VK. 2003. *Watson and DNA: Making a scientific revolution*, p. 170. Perseus Publishing, Cambridge, MA.

NordForsk 2017. *Nordic biobanks and registers: A basis for innovative research on health and welfare.* Policy paper, ISSN 1504–8640. Oslo, Norway.

Sanacora G, Heimer H, Hartman D, Mathew SJ, Frye M, Nemeroff C, Robinson Beale R. 2017. Balancing the promise and risks of ketamine treatment for mood disorders. *Neuropsychopharmacology* **42:** 1179–1181.

Stockwell BR, Friedmann Angeli JP, Bayir H, Bush AI, Conrad M, Dixon SJ, Fulda S, Gascón S, Hatzios SK, Kagan VE, et al. 2017. Ferroptosis: A regulated cell death nexus linking metabolism, redox biology and disease. *Cell* **171:** 273–285.

BANBURY CENTER MEETINGS

Date	*Title*	*Organizer(s)*
February 12–15	Chemiexcitation in Human Disease and Aging	E. Bechara, D. Brash
February 24–March 1	BIF Fellows Retreat: Communicating Science	C. Walther
March 19–22	Enhanceropathies: Enhancer Function Variation in Animal Development, Morphological Variation, and Disease	M. Levine, J. Wysocka
March 28–31	Project Santa Fe	Project Santa Fe Executive Committee
April 2–5	Ferroptosis: A Critical Review	X. Jiang, B.R. Stockwell
April 10–13	Better Cancer Therapy from Redox Biology	C. Chio, D. Tuveson
May 16–19	Maximizing Impact of New HIV Prevention Technologies in Sub-Saharan Africa	D. Pillay, H. Ward
June 12–14	Integrated Translational Science Center Workshop	L. Baker, L. Ellis, E. Liu, A. Schott, D. Tuveson
July 7	Foundation2017: Bio-Entrepeneurship in NYC	D. Brand, N. McKnight
July 9–12	Opportunities for Reduction of Aflatoxin Contamination of Food	J. Harvey, R. Michelmore, R. Nelson
September 10–13	Neuropharmacology and Human Stem Cell Models	N. Brandon, Z. Cader, S. Haggarty
September 17–20	NLRs Sans Frontières	J. Dangl, J. Jones, R. Vance
September 24–27	Metformin: Translating Biology into the Clinic	N. Chandel, V. Stambolic
October 22–24	Cell Biology of ALS: Emerging Themes from Human Genetics	L. Bruijn, A. Gitler, E. Holzbaur
October 29–November 1	Protective Immunity and Vaccines for Lyme Disease	E. Fikrig, S. Schutzer
November 12–14	Lustgarten Foundation Scientific Advisory Board Meeting	D. Tuveson, R. Vizza, A. Whiteley
November 26–29	Regulated Necrosis: Pathways and Mechanisms	D. Green, A. Linkermann
December 6–8	Post-Traumatic Neuroinflammation: Roles in Pathogenesis of Long-Term Consequences and Repair	R. Ransohoff, A. Schaefer, D. Schafer

BANBURY CENTER MEETINGS

Chemiexcitation in Human Disease and Aging

February 12–15

FUNDED BY **The LEO Foundation of Ballerup, Denmark, with additional support from the São Paulo Research Foundation (FAPESP) and L'Oréal**

ARRANGED BY **D. Brash,** Yale University, New Haven, Connecticut
E. Bechara, University of São Paulo and Federal University of São Paulo, Brazil

Chemiexcitation, the chemical excitation of electrons, is the biophysical process underlying bioluminescence. It had not been observed in mammals until a recent report demonstrated that chemiexcitation sends melanocytes down the path to melanoma. The same chemistry can occur in any tissue that contains melanin, suggesting that chemiexcitation may be an unknown step in neurodegenerative disorders such as Parkinson's disease, Alzheimer's disease, macular degeneration, and noise- or drug-induced deafness. The challenges are to identify, for each tissue, the sources of reactive nitrogen and oxygen species that initiate chemiexcitation; the melanin chemistry that creates the excited state; the DNA or protein alterations caused by energy transfer from, or chemical reaction with, the excited molecule; and the contribution of these alterations to pathogenic events. This multidisciplinary meeting brought together experts to build candidate pathways for each disease and a list of principles underlying the fields, suggesting experimental tools to address this previously unrecognized mode of disease.

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

T. Sarna, H. Sies, E. Gaillard, E. Bastos

SESSION 1: Overview: A New Mode of Pathogenesis

Chairperson: D. Brash, Yale School of Medicine, New Haven, Connecticut

D. Brash, Yale School of Medicine, New Haven, Connecticut: Excited electrons in melanoma and beyond.

E. Bechara, University of São Paulo and Federal University of São Paulo, Brazil: Short history of chemiexcitation: Liaisons between dioxygen, carbonyls, and light.

SESSION 2: Excited States and Chemiexcitation

Chairperson: J.-S. Taylor, Washington University, St. Louis, Missouri

B. Kohler, Ohio State University, Columbus: Excited states from light.

L. Blancafort, University of Girona, Spain: Cyclobutane dimers and energy wormholes.

W. Joseph Baader, University of São Paulo, Brazil: Excited states from chemical and biochemical reactions.

SESSION 3: Before the Excited State: The Ingredients

Chairperson: K. George, L'Oréal, Clark, New Jersey

H. Sies, Heinrich Heine University, Dusseldorf, Germany: The redox code and adventures of reactive oxygen and nitrogen.

T. Sarna, Jagiellonian University, Krakow, Poland, and L. Zecca, Italian National Research Council, Milan, Italy: Melanin, neuromelanin, and their reactions.

SESSION 4: After the Excited State: The Weapon

Chairperson: G. Timmins, University of New Mexico, Albuquerque

E. Bastos, University of São Paulo, Brazil: Excited-state reactions: Where the energy goes.

T. Sarna, Jagiellonian University, Krakow, Poland: Singlet oxygen.

G. Wondrak, University of Arizona, Tucson: Ground-state carbonyls are still reactive: Glycation damage and excited states.

SESSION 5: Diseases of Tissues Containing Melanocytes

Chairperson: T. Sarna, Jagiellonian University, Krakow, Poland

J. Shupp, Medstar Washington Hospital Center, Georgetown University, Washington, D.C.: Wound healing and hypertrophic scars.

J. O'Malley, Massachusetts General Hospital, Boston: The cochlea and noise/drug-induced deafness.

E. Gaillard, Northern Illinois University, DeKalb: Retinal pigment epithelium and macular degeneration.

SESSION 6: Diseases of Tissues Containing Neuromelanin

Chairperson: J. Costa, Yale School of Medicine, New Haven, Connecticut

W. Surewicz, Case Western Reserve University, Cleveland, Ohio: Biophysics of prions and amyloid.

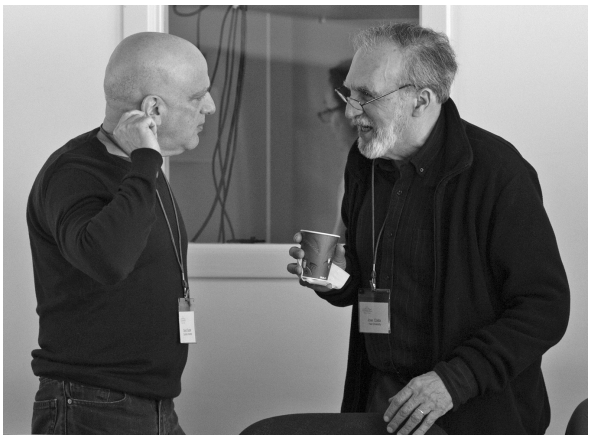
D. Sulzer, Columbia University, New York, and L. Zecca, Italian National Research Council: Parkinson's disease and progressive supranuclear palsy.

A. Vortmeyer, Indiana University, Indianapolis: Alzheimer's disease and late-age Down's syndrome.

SESSION 7: Assembling Pathways Breakout Groups

Chairpersons: D. Brash, Yale School of Medicine, New Haven, Connecticut, **K. George,** L'Oréal Advanced Research, Clark,

D. Brash



D. Sulzer, J. Costa

New Jersey, and **J. Costa,** Yale School of Medicine, New Haven, Connecticut

1. Deafness and Scars
2. Macular Degeneration
3. Parkinson's Disease and Progressive Supranuclear Palsy
4. Alzheimer's Disease and Late-Age Down's Syndrome

SESSION 8: Blocking the Pathways

Chairperson: B. Kohler, Ohio State University, Columbus

H. Sies, Heinrich Heine University, Dusseldorf, Germany: Antioxidant strategies: Enzymes and bioactives.

W. Joseph Baader and E. Bastos, University of Sao Paulo, Brazil: Intercepting and quenching electronically excited states.

L. Blancafort, University of Girona, Spain: Deactivating triplet states rapidly.

SESSION 9: The Précis

Chairperson: D. Sulzer, Columbia University, New York

Participants identified principles, methods, unanswered questions, and future experiments for the following:

1. Excited State Chemistry
2. ROS/RNS Chemistry, Reactive Carbonyls, Antioxidants
3. Pathology

BIF Fellows Retreat: Communicating Science

February 24–March 1

FUNDED BY **Boehringer Ingelheim Fonds**

ARRANGED BY **C. Walther,** Boehringer Ingelheim Fonds, Mainz, Germany

The Boehringer Ingelheim Fonds (BIF) has an international fellowship program supporting outstanding Ph.D. students. Among the opportunities provided to fellows is rigorous training in communication through an annual retreat. It was a great pleasure to have them return in 2017 for interactive instruction in matters such as oral presentations and writing papers. This year's retreat marked the tenth such visit to Banbury.

Opening Remarks: C. Walther, Boehringer Ingelheim Fonds, Mainz, Germany

K. Achenback, Boehringer Ingelheim Fonds, Mainz, Germany: Communication: Why and how?

A. Katsnelson, Freelance Biomedical Writer and Editor, Northampton, Massachusetts: Writing techniques and how to structure papers.

W. Tansey, Vanderbilt University, Nashville, Tennessee: Preparing and delivering a scientific talk.

M. Krzywinski, British Columbia Cancer Agency, Vancouver, British Columbia: Design of scientific concept and data figures with Adobe Illustrator.

C. Walther, Boehringer Ingelheim Fonds, Mainz, Germany: All about BIF.

PowerPoint Presentations, Videotaped with Replay, and Feedback

K. Grace, Weill Cornell Medicine, New York: Image beautification and the slippery slope to misconduct.

Enhanceropathies: Enhancer Function Variation in Animal Development, Morphological Variation, and Disease

March 19–22

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

ARRANGED BY **M. Levine,** Princeton University, New Jersey
J. Wysocka, Stanford School of Medicine, California

Changes to noncoding regulatory DNA sequences can lead to transcriptional variation that, in turn, mediates inter- and intraspecies phenotypic divergence. Genetic alterations in enhancer sequences, or those remodeling their chromosomal context, can result in human disease including congenital malformations, cancer, and neurodegenerative and autoimmune disorders. This meeting convened experts who compared diverse processes and systems to reveal unifying principles underlying functional enhancer variation, such as optimization or loss of activator elements, modulation of repressive inputs, and alterations in long-range enhancer-promoter communication.

Welcoming Remarks: R. Leshan, Banbury Center, Cold Spring Harbor Laboratory

Overview of Meeting Objectives: M. Levine, Princeton University, New Jersey, and **J. Wysocka,** Stanford School of Medicine, California

SESSION I: Enhancer Variation in Development and Evolution

Chairperson: M. Levine, Princeton University, New Jersey

E. Farley, University of California, San Diego: Regulatory principles governing enhancer specificity.

D. Stern, Janelia Research Campus, Ashburn, Virginia: Evolution of transcription through a deep dive into the functional evolution of the shavenbaby enhancers.

E. Furlong, European Molecular Biology Laboratory, Heidelberg, Germany: Functional insights into chromatin topology and gene expression during embryonic development.

C. Danko, Cornell University, Ithaca, New York: Natural selection has shaped coding and noncoding transcription in primate $CD4^+$ T cells.

J. Wysocka, M. Levine, F. Spitz, J. Crocker

A. Stark, C. Rushlow

SESSION II: General Mechanisms of Enhancer Function

Chairperson: C. Rushlow, New York University

J. Wysocka, Stanford School of Medicine, California: On the dangers of mistaking correlation for causation: Histone modifications in enhancer function.

A. Stark, Research Institute of Molecular Pathology, Vienna, Austria: Decoding transcriptional regulation.

M. Levine, Princeton University, New Jersey: Enhancer–enhancer interactions within complex genes.

K. Adelman, Harvard Medical School, Boston, Massachusetts: Making sense of nonsense: A roadmap for deciphering the potential functions of noncoding RNAs.

R. Young, Whitehead Institute for Biomedical Research, Cambridge, Massachusetts: Things we don't understand about enhancers.

SESSION III: Transcription Factors

Chairperson: A. Stark, Research Institute of Molecular Pathology, Vienna, Austria

H. Bussemaker, Columbia University, New York: Accurate and sensitive quantification of protein–DNA recognition.

C. Rushlow, New York University: Zelda-binding sites as quantitative timers of target gene transcription.

J. Taipale, Karolinska Institutet, Stockholm, Sweden: Genome-wide analysis of protein–DNA interactions.

SESSION IV: Topology and Nuclear Architecture

Chairperson: E. Furlong, European Molecular Biology Laboratory, Heidelberg, Germany

F. Spitz, Institut Pasteur, Paris, France: Managing functional interactions between distant enhancers and genes.

B. Ren, Ludwig Institute for Cancer Research, La Jolla, California: An essential role of CTCF in chromatin organization?

A. Rada-Iglesias, Center for Molecular Medicine, Cologne, Germany: Polycomb proteins as topological facilitators of enhancer regulatory function.

G. Blobel, Children's Hospital of Philadelphia, Pennsylvania: Chromatin readers and nuclear architecture.

SESSION V: Enhancer Malfunction in Cancer

Chairperson: K. Adelman, Harvard Medical School, Boston, Massachusetts

B. Bernstein, Massachusetts General Hospital and Harvard, Charlestown: Hypermethylation and insulator dysfunction in cancer.

A. Shilatifard, Northwestern University, Chicago, Illinois: Enhancer biology and enhanceropathies in cancer.

P. Scacheri, Case Western University, Cleveland, Ohio: Aberrant enhancer activation in cancer progression.

SESSION VII: Wrap-Up and Next Steps

Meeting Conclusions

Project Santa Fe

March 28–31

FUNDED BY **Northwell Health**

ARRANGED BY **Project Santa Fe Executive Committee**

Project Santa Fe is a coalition of leadership from innovative clinical laboratories: Northwell Health Laboratories, Geisinger Health System, Henry Ford Health System, Kaiser Permanente Northern California Health Systems, and TriCore Reference Laboratories. We were pleased to host this group at Banbury in March to develop strategies for clinical lab testing services that maximize impact, improve patient outcomes, and cut costs. The outputs of this meeting were subsequently presented at two major conferences.

Geisinger Health System (Danville, Pennsylvania)
M. Wilkerson, M.D.
J. Olson, M.D.
C. Christenson, M.D.
S. Snyder, Ph.D.
D. Wolke, M.D.

Henry Ford Health System (Detroit, Michigan)
G. Sharma, M.D.
R. Zarbo, M.D.
J. Waugh, M.D.
I. Rubinfeld, M.D.
J.M. Tuthill, M.D.

Northwell Health (Lake Success, New York)
J. Crawford, M.D., Ph.D.
D. Breining, M.D.
T. Chang, M.D.
R. Stallone
T. Kothari, M.D., M.P.H.
L. Lomsadze
Y. Jacobs (observer)
S. Roychoudhury, M.D. (observer)
J. Yim
R. Miller
A. Murray

TriCore Laboratories (Albuquerque, New Mexico)
Khosrow Shotorbani
N. Fisher
M. Crossey, M.D.
M. Dodd, PharmD

Special Participants
M. Trusheim (Moderator), MIT Sloan School of Management; President, Co-Bio Consulting, LLC
R. Michel, President and CEO, The Dark Report

Ferroptosis: A Critical Review

April 2–5

FUNDED BY **Burroughs Wellcome Fund, Cold Spring Harbor Laboratory Corporate Sponsor Program, Collaborative Medicinal Development, Memorial Sloan Kettering Cancer Center, and Ono Pharmaceutical Co. Ltd.**

ARRANGED BY **B.R. Stockwell,** Columbia University, New York
X. Jiang, Memorial Sloan Kettering Cancer Center, New York

Ferroptosis is a form of regulated, nonapoptotic cell death that involves overwhelming lipid peroxidation. Originally reported in 2012, ferroptosis has been of increasing interest because of its integration with cellular metabolism and its suggested role in cell death associated with degenerative diseases, carcinogenesis, stroke, intracerebral hemorrhage, traumatic brain injury, ischemia-reperfusion injury, and kidney degeneration in mammals, as well as heat stress in plants. This Banbury meeting brought together, for the first time, leading researchers working on diverse aspects of ferroptosis to explore mechanisms underlying this emerging form of regulated cell death and to suggest tools and guidelines for future studies.

Welcoming Remarks: R. Leshan, Banbury Center, Cold Spring Harbor Laboratory

Overview of Meeting Objectives: B. Stockwell, Columbia University, New York, and
X. Jiang, Memorial Sloan Kettering Cancer Center, New York

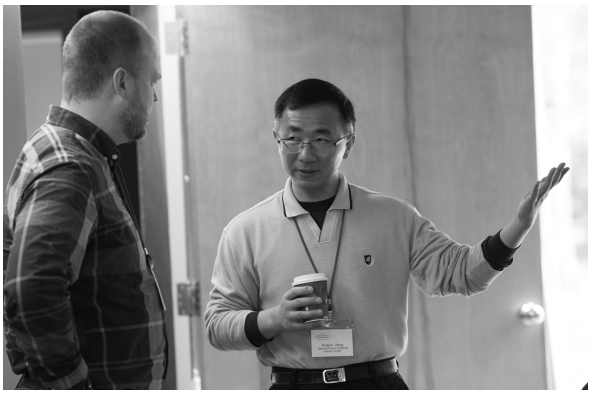
SESSION I: Regulators of Ferroptosis

Chairperson: B. Stockwell, Columbia University, New York

B. Stockwell, Columbia University, New York: Overview and update on ferroptosis.

M. Conrad, Helmholtz Zentrum München, Germany: In vivo mechanisms of ferroptosis control by GPX4.

D. Tang, University of Pittsburgh, Pennsylvania: P53 limits ferroptosis by blocking DPP4 activity.



S. Dixon, X. Jiang

M. Murphy, C. Prives, C. Rosenfeld

SESSION II: Ferroptosis and Metabolism

Chairpersons: X. Jiang, Memorial Sloan Kettering Cancer Center, New York, and **K. Salnikow,** NCI, National Institutes of Health, Bethesda, Maryland

X. Jiang, Memorial Sloan Kettering Cancer Center, New York: Metabolism and ferroptosis.

S. Dixon, Stanford University, California: The regulation of polyunsaturated fatty acid oxidation during ferroptosis.

SESSION III: ROS in Ferroptosis

Chairperson: V.E. Kagan, University of Pittsburgh, Pennsylvania

V.E. Kagan, University of Pittsburgh, Pennsylvania: ROS in ferroptosis.

K. Woerpel, New York University: Development of cyclic peroxides that induce ferroptosis, not apoptosis.

S. Hatzios, Yale University, New Haven, Connecticut: Oxidative cROSstalk at the host–microbe interface: A possible role for ferroptosis in infectious disease.

SESSION IV: Ferroptosis and Degenerative Disease: Part 1

Chairperson: A. Linkermann, Universitätsklinikum Carl Gustav Carus, Germany

A. Linkermann, Universitätsklinikum Carl Gustav Carus, Germany: The in vivo relevance of ferroptosis.

Q. Ran, University of Texas Health Science Center at San Antonio: GPX4 and neurogeneration.

SESSION V: Ferroptosis and Degenerative Diseases: Part 2

Chairperson: A Bush, University of Melbourne, Australia

A. Bush, University of Melbourne, Australia: The role of ferroptosis in Alzheimer's disease.

H. Bayir, University of Pittsburgh, Pennsylvania: What is the role of ferroptosis in acute brain injuries?

J. Pedro Friedmann Angeli, Helmholtz Zentrum München, Germany; Role of Acsl4 as a pro-ferroptotic gene.

SESSION VI: Ferroptosis and Cancer

Chairperson: M. Murphy, The Wistar Institute, Pennyslvania

M. Murphy, The Wistar Institute, Pennsylvania: An African-specific variant of p53 is defective for ferroptosis.

C. Prives, Columbia University, New York: Role of the p53 network in the regulation of ferroptosis.

S. Toyokuni, Nagoya University Graduate School of Medicine, Japan: Role of iron in carcinogenesis and tumor biology.

SESSION VII: Ferroptosis and Other Cell Fates

Chairperson: S. Fulda, Goethe Universitat, Germany

S. Fulda, Goethe Universitat, Germany: RSL3 and Erastin differentially regulate redox signaling to promote Smac mimetic-induced cell death.

S. Gascón, Ludwig-Maximilians University of Munich, Germany: ROS and ferroptosis in cell reprogramming.

B. Stockwell, S. Fulda

SESSION VIII: Ferroptosis in Diverse Contexts

Chairperson: M Overholtzer, Memorial Sloan Kettering Cancer Center, New York

G. Pagnussat, IIB-CONICET-National University of Mar del Plata, Argentina: A conserved cell death pathway across kingdoms: Ferroptosis in plants.

M. Overholtzer, Memorial Sloan Kettering Cancer Center, New York: Nanoparticle-mediated ferroptosis induction in cancer.

SESSION IX: Back to the Starting Point: Iron and Ferroptosis

Chairperson: D. Zhang, University of Arizona, Tucson

F. Torti, University of Connecticut, Storrs: Iron metabolism.

S. Torti, University of Connecticut, Storrs: The "fer" in ferroptosis.

D. Zhang, University of Arizona, Tucson: NRF2: An integrator of cellular iron and redox signaling.

SESSION X: Wrap-Up and Next Steps

Chairpersons: B. Stockwell, Columbia University, New York, and **X. Jiang**, Memorial Sloan Kettering Cancer Center, New York

Better Cancer Therapy from Redox Biology

April 10–13

FUNDED BY **The Oliver Grace Chair Fund**

ARRANGED BY **D. Tuveson,** Cold Spring Harbor Laboratory
C. Chio, Cold Spring Harbor Laboratory

An unanswered question in human health is whether anti-oxidation prevents or promotes cancer. Anti-oxidation has historically been viewed as chemopreventive, but emerging evidence suggests that antioxidants may be supportive of neoplasia. To leverage cellular redox changes toward the development of a safe and effective therapeutic strategy necessitates experimental delineation of specific redox signaling pathways that are uniquely required by cancer cells to grow and to survive. This Banbury meeting focused on the complexity of redox regulation in the context of cancer biochemistry and therapy, exploring ROS genesis and metabolism in cancer cells, as well as the "productive" and "destructive" signal transduction by free radicals through the oxidation of intermediates.

Welcoming Remarks: R. Leshan, Banbury Center, Cold Spring Harbor Laboratory

SESSION I: Free Radicals and Antioxidants in Physiological Functions

Chairperson: A. Holmgren, Karolinska Institute, Stockholm, Sweden

J. Watson, Cold Spring Harbor Laboratory: To overcome chemoresistant cancers, use natural product quinones.

C. Winterbourn, University of Otago, Christchurch, New Zealand: Cellular mechanisms for regulating hydrogen peroxide metabolism and oxidative stress.

U. Jakob, University of Michigan, Ann Arbor: Role of polyphosphate in oxidative stress defense.

N. Tonks, Cold Spring Harbor Laboratory: Redox regulation of protein tyrosine phosphatases for therapeutic development.

C. Winterbourn, T. Dick

T. Mak, I. Chio

N. Chandel, Northwestern University, Chicago, Illinois: Functional genomic screens to uncover redox biology.
P. Schumacker, Northwestern University, Chicago, Illinois: Mitochondrial regulation of cell proliferation.

SESSION II: Free Radicals and Antioxidants in Cancer

Chairpersons: A. Ostman, Karolinska Institute, Stockholm, Sweden, and **K. Liby,** Michigan State University, East Lansing

K. Vousden, Francis Crick Institute, London, United Kingdom: Modulating TIGAR to probe ROS functions in tumor development and metastasis.
T. Mak, University of Toronto, Ontario, Canada: Modulation of oxidative stress as an anticancer strategy.
E. Schmidt, Montana State University, Bozeman: Endogenous oxidants and cellular antioxidant systems in liver cancer.
M. Bergo, Karolinska Institutet, Huddinge, Sweden: Antioxidants cause long-term programming of lung cancer cells into a metastatic phenotype.
N. Hay, University of Illinois, Chicago: Akt, hexokinase 2, ROS, and cancer therapy.

SESSION III: NRF2 in Redox Homeostasis and Metabolism

Chairperson: M. Espey, National Cancer Institute, Rockville, Maryland

J. Hayes, University of Dundee, United Kingdom: The mechanisms of repression of transcription factor Nrf2 and its cross talk with lipid metabolism.
C. Chio, Cold Spring Harbor Laboratory: Nrf2 promotes mRNA translation in pancreatic cancer.
M. Yamamoto, Tohoku University, Sendai, Japan: Molecular basis of Keap1-Nrf2 system and cancer.
T. Papagiannokopolus, New York University Medical School: Pro-tumorigenic NRF2 antioxidant program causes defects in central carbon metabolism.
G. DeNicola, Moffitt Cancer Center, Tampa, Florida: Compartmentalization of ROS production and metabolism.
D. Zhang, University of Arizona, Tucson: NRF2: An integrator of cellular iron and redox signaling.

SESSION IV: Redox Imaging

Chairperson: T. Dick, German Cancer Research Center, Heidelberg, Germany

V. Belousov, Shemyakin-Ovchinnikov Institute of Bioorganic Chemistry: Metabolic engineering tools and fluorescent probes for redox biology.
K. Brindle, University of Cambridge, United Kingdom: Imaging oxidative stress in vivo.
C. Chang, University of California, Berkeley: Chemical imaging and proteomics probes for studying redox biology.
T. Dick, German Cancer Research Center, Heidelberg, Germany: Understanding the "anti-oxidant" *N*-acetyl cysteine.
Y. Yang, East China University of Science and Technology, Shanghai: Genetically encoded sensors for redox biology and their applications in drug screening.
M. Murphy, MRC Mitochondrial Biology Unit Cambridge, United Kingdom: Therapeutic alteration to the mitochondrial redox environment.

SESSION V: Therapeutics

Chairperson: T. Miller, IC-MedTech, Las Vegas, Nevada

D. Boothman, University of Texas Southwestern Medical Center, Dallas: Leveraging NQO1 bioactivatable drugs for tumor-selective ROS production and antitumor activity.
G. Buettner, University of Iowa, Iowa City: Using science to guide clinical trials for cancer treatment where redox biology is at the center.
D. Spitz, University of Iowa, Iowa City: $O_2^{\bullet-}$ and H_2O_2-mediated disruption of Fe metabolism causes the differential susceptibility of NSCLC and GBM cancer cells to pharmacological ascorbate.

S. Morrison, University of Texas Southwestern Medical Center, Dallas: Distant metastasis requires cancer cells to adapt to cope with oxidative stress.

E. Parkinson, University of Illinois, Urbana: Deoxynyboquinones as NQO1-targeted anticancer compounds.

SESSION VI: Wrap-Up and Next Steps

Chairpersons: D. Tuveson and J. Watson, Cold Spring Harbor Laboratory

K. Vousden, K. Brindle, U. Jakob

Maximizing Impact of New HIV Prevention Technologies in Sub-Saharan Africa

May 16–19

FUNDED BY **Bill & Melinda Gates Foundation**

ARRANGED BY **H. Ward,** Imperial College London, United Kingdom
D. Pillay, Africa Health Research Institute, Durban, South Africa

Despite the promotion of HIV combination prevention encompassing structural, behavioral, and biomedical interventions, HIV incidence in adolescent girls and young men and women in sub-Saharan Africa remains high. Recent data suggest that "treatment as prevention" approaches alone will be inadequate to limit the epidemic in this setting. A wide-ranging group of participants at this Banbury meeting were challenged to examine current knowledge about who is at risk, what drives that risk, and what facilitates or obstructs the uptake of preventive interventions and to articulate what we need to know to get new and old preventive technologies taken up and used. During the meeting's final sessions, the group applied their discussions toward specific examples of high-risk individuals and concluded with the development of a model to integrate conclusions and recommendations.

Welcoming Remarks: R. Leshan, Banbury Center, Cold Spring Harbor Laboratory

Overview of Meeting Objectives: D. Pillay, Africa Health Research Institute, Durban, South Africa, and **H. Ward,** Imperial College London, United Kingdom

SESSION I: Introductions and Framework for the Meeting

Chairpersons: H. Ward, Imperial College London, United Kingdom, and **D. Pillay,** Africa Health Research Institute, Durban, South Africa

E. Emini and G. Garnett, Bill & Melinda Gates Foundation, Seattle, Washington: The Gates Foundation HIV Prevention Strategy.

H. Ward, Imperial College London, United Kingdom, and D. Pillay, Africa Health Research Institute, Durban, South Africa: Overview of the population (adolescent girls and young men and women in sub-Saharan Africa) and the technologies (new and old) that we will be considering.

R. Barnabas, University of Washington, Seattle: Incidence of HIV in adolescent girls, young women, and young men in sub-Saharan Africa: Who are the highest risk populations, 2016 UNAIDS data?

SESSION II: Who Is at Risk and Why?

Chairperson: H. Ward, Imperial College London, United Kingdom

J. Wamoyi, National Institute for Medical Research, Mwanza, Tanzania: Transactional sex and HIV among AGYW in sub-Saharan Africa.

S. Mojola, University of Colorado, Boulder: How social and ecological contexts shape and complicate individual decision-making about HIV prevention.

SESSION III: User Perspectives on HIV Prevention Technologies

Chairperson: M. Shahmanesh, University College London and Africa Health Research Institute, United Kingdom

J. Seeley, London School of Hygiene & Tropical Medicine, United Kingdom: Why men do and don't use male condoms.

M. Warren, AVAC, New York: Why women do and don't use female condoms.

M. Gafos, MRC, University College London, United Kingdom: Lessons from microbicide research: User perspectives, acceptability, and adherence.

H. Ward, Imperial College London, United Kingdom: Obstacles and facilitators: Summary of evidence on existing technologies.

SESSION IV: Understanding the Market for HIV Prevention Technologies: Products and Population Segmentation

Chairperson: H. McDowell, ViiV Healthcare, Brentford, United Kingdom

A. Gomez, AVAC, New York: Products, platforms, and people: Developing and delivering prevention that works.

K. Hallman, The Population Council, New York: Segmentation of sexual partner types: Results of participatory research.

SESSION V: Understanding HIV Prevention Behaviors: Theoretical Insights

Chairperson: G. Dallabetta, Bill & Melinda Gates Foundation, Washington, D.C.

R. Prasad, Final Mile Consulting, Chicago, Illinois: Using a behavioral economics approach to explain and influence HIV prevention behaviors.

S. Linnemayr, RAND Corporation, Santa Monica, California: Using a behavioral economics approach to explain and influence HIV prevention behaviors.

M. Skovdal, University of Copenhagen, Denmark: Using theories of practice to understand HIV risk: Opportunities and challenges for new HIV prevention technologies in sub-Saharan Africa.

SESSION VI: Translating Technologies into Practice: Lessons from Other Programs

Chairperson: J. Shelton, Johns Hopkins University, Baltimore, Maryland

F. Cowan, A. Pettifor, M. Shahmanesh, D. Pillay, H. Ward

N. Mugo, J. Cleland

V. Chandra-Mouli, World Health Organization, Geneva, Switzerland: Lessons from scaling up reproductive health programs.

J. Cleland, London School of Hygiene & Tropical Medicine, London, United Kingdom: Abstinence, contraception, and condom use among single African women: What can we learn from long-term trends.

R. Ingham, University of Southampton, United Kingdom: Young people, risk, and vulnerability: The success of the English teenage pregnancy strategy and its applicability to other countries.

SESSION VII: Translating Technologies into Practice: Lessons from Three Decades of HIV Prevention

Chairperson: K. Dehne, UNAIDS, Geneva, Switzerland

N. Mugo, Kenya Medical Research Institute, Nairobi, Kenya: Holistic approach to HIV combination prevention with focus on population and geography.

M. Shahmanesh, UCL/Africa Health Research Institute, London, United Kingdom: Delivering HIV prevention: Lessons from history and key populations.

SESSION VIII: Lessons from a Country Case Study: Zimbabwe

Chairperson: G. Garnett, Bill & Melinda Gates Foundation, Seattle, Washington

F. Cowan, Liverpool School of Tropical Medicine, Harare, Zimbabwe: Lessons of HIV prevention program delivery in Zimbabwe.

K. Dehne, UNAIDS, Geneva, Switzerland: Lessons of HIV prevention program delivery in Zimbabwe.

SESSION IX: Design Workshop

Chairpersons: H. Ward, Imperial College London, United Kingdom, and **D. Pillay,** Africa Health Research Institute, Durban, South Africa

This interactive session used specific personae of high-risk individuals to map out the steps necessary for that particular person to benefit from an HIV preventive technology. At the end of the session, the various summaries were reviewed to identify key touch points needing further work.

SESSION X: Reporting and Wrap-Up

Chairpersons: H. Ward, Imperial College London, United Kingdom, and **D. Pillay,** Africa Health Research Institute, Durban, South Africa

Integrated Translational Science Center Workshop

June 12–14

FUNDED BY **National Institutes of Health/National Cancer Institute (grant awarded to D. Tuveson et al.)**

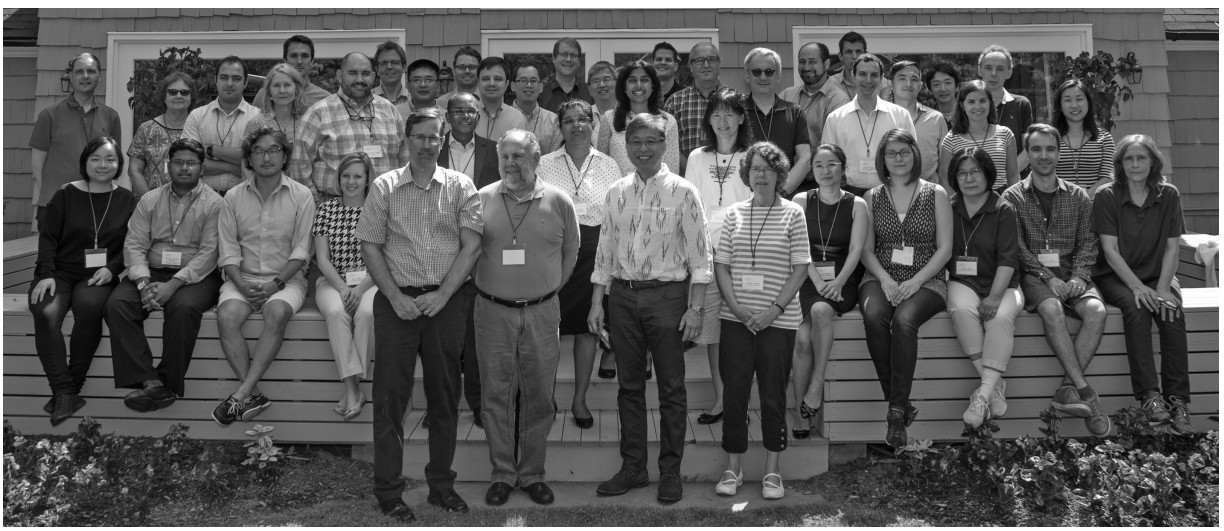
ARRANGED BY **L. Baker,** University of Michigan, Ann Arbor
L. Ellis, University of Texas, Houston
E. Liu, The Jackson Laboratory, Bar Harbor, Maine
A. Schott, University of Michigan, Ann Arbor
D. Tuveson, Cold Spring Harbor Laboratory

The Integrated Translational Science Center (ITSC) formed between SWOG, Cold Spring Harbor Laboratory (CSHL), and The Jackson Laboratory (JAX) to bridge the gap between the laboratory and the clinic by elucidating the key clinical problems and challenges in oncology that can be addressed in the laboratory, discovering new diagnostic and therapeutic approaches that can be integrated into clinical trials and by providing a conduit for clinical trial results to be reinterpreted in the laboratory. In June, the ITSC members convened at Banbury to discuss pressing questions in cancer medicine that are best addressed in partnership with technological and basic/translational science experts at JAX and CSHL. The highly interactive meeting included talks, posters, laboratory demonstrations, and brainstorming sessions to generate ideas for collaborative projects that take advantage of both the clinical expertise of the SWOG participants and innovative science and technology provided by CSHL and JAX scientists.

Welcoming Remarks: R. Leshan, Banbury Center, Cold Spring Harbor Laboratory

Introduction to CSHL/Overview of Meeting Objectives: D. Tuveson, Cold Spring Harbor Laboratory

Introduction to Jackson Laboratory: E. Liu, The Jackson Laboratory, Bar Harbor, Maine



J. Chuang, Z. Mitri

L. Baker, D. Tuveson

Keynote Address

J. Doroshow, National Cancer Institute, Bethesda, Maryland: Evolution of NCI's Clinical Trials Networks in the Era of Precision Oncology.

Overview

E. Liu, The Jackson Lab, Bar Harbor, Maine: SWOG/CSHL/JAX ITSC Discovery Engine.

SCIENTIFIC POSTERS

Technology Presentations I

H. Reddi, The Jackson Laboratory, Bar Harbor, Maine: Clinical genomics at Jax: Technologies and capabilities.

J. Lee, Cold Spring Harbor Laboratory: Approaches to mapping gene expression signatures in space.

SWOG Investigator Presentations I

Moderator: L. Baker, University of Michigan, Ann Arbor

Panel: H. Babiker, P. Chalasani, E. Cobain, A. Danilov, A. Kirschner, J. Leonard, B. Lim, J. Markowitz

Technology Presentations II

P. Robson, The Jackson Laboratory, Bar Harbor, Maine: Cellular phenotyping tumors with single-cell technologies.

H. Tiriac, Cold Spring Harbor Laboratory: Modeling patient response in human pancreas cancer organoids.

SWOG Investigator Presentations II

Moderator: L. Baker, University of Michigan, Ann Arbor

Panel: A. Morikawa, Z. Ibrahim Mitri, A. Scott, C. Speers, A. VanderWalde, J. Carlos Varela, D. Wahl

Developing Projects

Speed Science
Pilot Presentations

Preparing Project Proposals

Moderator: L. Baker, University of Michigan, Ann Arbor

Panel: P. Robson, H. Reddi, H. Tiriac

Foundation2017: Bio-Entrepreneurship in NYC

July 7

FUNDED BY **Keystone for Incubating Innovation in Life Sciences Network (KiiLN), J.P. Morgan, Pfizer, Torreya Advisors, Nixon Peabody, Alston & Bird, LI Bioscience Hub**

ARRANGED BY **D. Brand,** ECHO NYC & Quicksilver Biosystems, New York, New York
N. McKnight, KiiLN & BioLabs, New York, New York

The second annual Foundation conference brought entrepreneurs, investors, industry leaders, and scientists to the Banbury Center for one day of stimulating panel sessions and networking.

Introductory Remarks: D. Brand, ECHO NYC & Quicksilver Biosystems, and **N. McKnight**, KiiLN & BioLabs New York

PANEL 1: Mining Academia

Moderator: K. Neote, Johnson & Johnson Innovation Center, Cambridge, Massachusetts;

J. Mandelbaum, Accelerator Corp, New York, New York
C. Pitt, Versant Ventures, New York, New York
T. Thornton, Northwell Ventures, New York, New York

PANEL 2: Genomics

Moderator: D. Brand, ECHO NYC & Quicksilver Biosystems, New York, New York

J. Crawford, Northwell Health, Lake Success, New York
J. Leslie, Celmatix, New York, New York
J. Pickrell, Gencove, New York, New York

PANEL 3: New Ventures

Moderator: C. Green, Pfizer, New York, New York

S. Bettigole, Quentis Therapeutics, New York, New York
J. Vacca, Highline Therapeutics, New York, New York

PANEL 4: "Dealmakers"

Moderator: A. Tinkelenberg, Torreya Advisors, New York, New York

S. Jacobson, Remedy Pharmaceuticals, New York, New York
J. Magram, Northern Biologics, New York, New York

Fireside Chat

J. Anderson, CTI LifeSciences, New York, New York

E. Schadt, Sema4, New York, New York

Opportunities for Reduction of Aflatoxin Contamination of Food

July 9–12

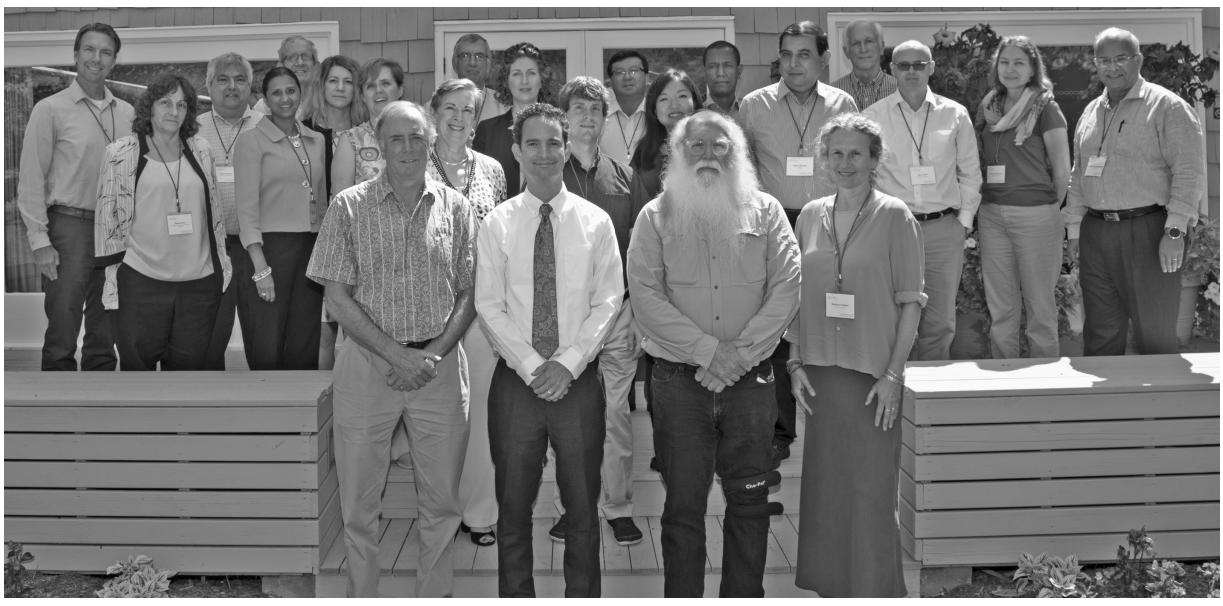
FUNDED BY **Mars, Inc., the U.S. National Science Foundation (support to R.M.), and the Cold Spring Harbor Laboratory Corporate Sponsor Program, with additional support from Kansas State University**

ARRANGED BY **R. Michelmore,** University of California, Davis
J. Harvey, Kansas State University, Manhattan
R. Nelson, Cornell University, New York

Contamination of food by mycotoxins is a worldwide problem: Estimates indicate that at least 25% of the global food supply is contaminated [UN FAO]. Aflatoxins, a type of mycotoxin produced by *Aspergillus* species, can cause liver cancer as well as a variety of ailments related to immunosuppression when adults are subjected to chronic exposure; exposure of children results in stunting. Acute exposure can result in death. Current approaches can help reduce, but not eliminate, contamination. However, recent advances in multiple areas offer new potential. This Banbury meeting reviewed current efforts to date, their limitations, and challenges. Participants assessed the new short- and longer-term opportunities enabled by advances in knowledge and technology: varieties resistant to abiotic stresses, *Aspergillus* species or insects; biocontrol; and transgenic strategies aimed at reducing pathogen growth and aflatoxin production such as host-induced gene silencing. Finally, the group considered the pathways for deployment and adoption of new intervention strategies as well as integration with efforts to reduce contamination with other mycotoxins.

Welcoming Remarks: R. Leshan, Banbury Center, Cold Spring Harbor Laboratory

Overview of Meeting Objectives: R. Michelmore, University of California, Davis



A. Ayalew, R. Michelmore

C. Woteki, A. Records

SESSION I: Current Situation, Limitations, and Challenges

Chairperson: H.-Y. Shapiro, Mars, Incorporated, McLean, Virginia

H.-Y. Shapiro, Mars, Incorporated, McLean, Virginia: Introduction to session themes (efforts, areas most in need, impacts on livestock, and human health).

A. Ayalew, PACA, African Union Commission, Ethiopia: Are country-led approaches the answer for coordinating efforts to win the fight against aflatoxins?

A. Bianchini, University of Nebraska, Lincoln: Mycotoxin assessment on corn production chain by small holders in Guatemala.

M. Manary, Washington University, St. Louis, Missouri: Relationship of serum aflatoxin measurements in pregnant women and newborn length.

F. Wu, Michigan State University, East Lansing: Recent findings in aflatoxin and human health: A liver cancer success story and doubts cast on stunting role.

K. Damann, Jr., Louisiana State University Agricultural Center, Baton Rouge: *Aspergillus flavus* biology and biological control of aflatoxin contamination in corn.

SESSION II: Pre- and Post-Harvest Interventions Possible in the Short Term

Chairperson: R. Michelmore, University of California, Davis

R. Bandyopadhyay, IITA, Nigeria: Biological control and other practices for aflatoxin management in Africa.

P. Cotty, Agricultural Research Service, USDA, School of Plant Sciences, University of Arizona, Tucson: Biological control: One tool reduces aflatoxins throughout the environment.

P. Ojiambo, North Carolina State University, Raleigh: Female fertility and its role in selecting effective biocontrol strains of *Aspergillus flavus.*

J. Harvey, Kansas University/Post-Harvest Loss Innovation Lab, Manhattan: Toward an integrated approach to reducing aflatoxin contamination and exposure.

R. Nelson, Cornell University, Ithaca, New York: Sorting maize at local hammer mills as part of a strategy for reducing mycotoxins in the African food system.

B. Bextine, DARPA/BTO, Arlington, Virginia: DARPA's Biological Technologies Office.

SESSION III: Conventional Breeding and Transgenic Longer-Term Interventions

Chairperson: J. Harvey, Kansas University/Post-Harvest Loss Innovation Lab, Manhattan

M. Warburton, USDA, ARS, CHPRRU, Mississippi: Translational genomics of aflatoxin accumulation resistance in maize: Can I take my lab results to the field?

W. Xu, Texas A&M University, Lubbock: Progress and challenges in breeding aflatoxin-resistant corn.

R. Michelmore, University of California, Davis: Host-induced gene silencing for disease control.

M. Schmidt, University of Arizona, BIO5 Institute, Tucson: The use of host-induced gene silencing (HIGS) to suppress aflatoxin production.

H.-Y. Shapiro, J. Harvey

R. Arias, USDA-ARS-National Peanut Research Laboratory, Dawson, Georgia: RNAi-mediated control of aflatoxins in peanut.

K. Kumar Sharma, ICRISAT, India: Genetic engineering for the control of *Aspergillus flavus* infection and aflatoxin production in peanut.

SESSION IV: Translational Realities for Implementation

Chairperson: R. Nelson, Cornell University, Ithaca, New York

M. Stasiewicz, University of Illinois, Urbana-Champaign: Multispectral sorting to reduce mycotoxin levels in maize.

P. Turner, University of Maryland, College Park: The strength of exposure biomarkers in evaluating intervention strategies for mycotoxins.

C. Woteki, Iowa State University, Ames: Reducing policy barriers to lowering aflatoxin exposures.

T. Herrman, Texas A&M University, College Station: Building a public private partnership to manage aflatoxin risk through a connected and transparent marketplace that delivers aflatoxin-safe food and feed to all Africa.

N. Kazi, Humanitas Global Development, Washington D.C.: Building an enabling environment for wide-scale adoption of proven interventions that reduce mycotoxin contamination.

A. Records, USAID, Washington, D.C.: Mycotoxin mitigation: A global food security priority.

SESSION V: Next Steps

Chairpersons: J. Harvey, Kansas State University, Manhattan, **R. Nelson,** Cornell University, Ithaca, New York, **R. Michelmore,** University of California, Davis.

Neuropharmacology and Human Stem Cell Models

September 10–13

FUNDED BY **The Cold Spring Harbor Corporate Sponsor Program and The Lieber Institute for Brain Development**

ARRANGED BY **N. Brandon,** AstraZeneca, Waltham, Massachusetts
Z. Cader, Oxford University, United Kingdom
S. Haggarty, Harvard Medical School and Massachusetts General Hospital, Boston

Advances in a combination of disciplines—human stem cell biology, chemical biology, and human genetics—are catalyzing new opportunities both to impact our fundamental understanding of human disease biology and to discover next-generation pharmacological agents aimed at targeting the root cause of disease. Perhaps nowhere are these advances more significant and critically needed than the area of neurological and psychiatric diseases. Participants in this Banbury meeting, spanning neurological and psychiatric neuroscience as well as drug discovery, critically reviewed the state of human stem cell modeling as applied to the advancement of neuropharmacology. Specifically, discussion focused on the state of patient-specific bio-banking internationally; approaches to the integration of iPSC models with clinical cohorts for precision medicine, genome sequencing and deep patient phenotyping at the level of neuroimaging and neuropathology; disease-relevant stem cell-based assay development to support large-scale pharmacological and CRISPR/Cas9-based functional genomic screens; and application of stem cell models to systems pharmacology, including use of organoids and organ-on-a-chip technologies.

Welcoming Remarks: R. Leshan, Banbury Center, Cold Spring Harbor Laboratory

Introduction and Meeting Objectives: S. Haggarty, Harvard Medical School and Massachusetts General Hospital, Boston, **N. Brandon,** AstraZeneca, Waltham, Massachusetts, and **Z. Cader,** Oxford University, United Kingdom

SESSION I: Introduction and Overview

Chairperson: D. Panchision, National Institute of Mental Health, Bethesda, Maryland

D. Panchision, National Institute of Mental Health, Bethesda, Maryland: How NIMH is adapting iPSC research to functional genomics, systems neuroscience, and drug discovery.

Z. Cader, Oxford University, United Kingdom: Innovative medicines initiative platforms for iPSC drug discovery research.

A. Kaykas, Novartis Institute for Biomedical Research, Cambridge, Massachusetts: Using stem cell for neuroscience target discovery.

SESSION II: Patient Cohorts and Deep Clinical Phenotyping

Chairperson: R. Perlis, Broad Institute, Boston, Massachusetts

R. Perlis, Broad Institute, Boston, Massachusetts: Applying a large neuropsychiatric biobank to characterize Roy Perlis treatment response.

L. Studer, Memorial Sloan Kettering Cancer Center, New York: Rapid glial fates and the use of a pooled hPSC approach to identify disease phenotypes.

L. Grinberg, University of California, San Francisco: Neuropathological methods and studies on stem cell can complement each other to advance the knowledge on neurodegenerative disease.

SESSION III: Stem Cell Neurotechnology and Advanced Modeling

Chairperson: K. Fabre, AstraZeneca, Waltham, Massachusetts

K. Fabre, AstraZeneca, Waltham, Massachusetts: Microphysiological systems and inducible stem cells for drug development.

O. Brüstle, Institute of Reconstructive Neurobiology, Bonn, Germany: Programming NSCs for disease modeling and drug discovery.

E. Shusta, University of Wisconsin, Madison: Stem cell modeling of the neurovascular unit.

I. Cornella-Taracido, Merck & Company, Boston, Massachusetts: Use of quantitative, high-resolution mass spectrometry–based proteomics to enable phenotype–genotype–proteotype correlation analyses toward target and biomarker discovery in neuroscience.

SESSION IV: Alzheimer's and Related Dementia

Chairperson: F. Livesey, University of Cambridge, United Kingdom

F. Livesey, University of Cambridge, United Kingdom: Small-molecule and genetic screens to identify druggable targets in human stem cell models of dementia.

H. Inoue, Center for iPS Cell Research and Application, Kyoto, Japan: Human pluripotent stem cells in neurological drug discovery.

T. Young-Pearse, BWH and Harvard Medical School, Boston, Massachusetts: Probing heterogeneity of Alzheimer's disease using iPSCs.

SESSION V: Neurodevelopmental, Psychotic, and Mood Disorders

Chairperson: S. Haggarty, Harvard Medical School and Massachusetts General Hospital, Boston

L. Ellerby, N. Heintz

I. Cornella-Taracido, R. Livesey, H. Heimer, R. Perlis

S. Haggarty, Harvard Medical School and Massachusetts General Hospital, Boston: Advancing neuropharmacology for rare neurogenetic disorders with patient-derived stem cell models.
B. Maher, Lieber Institute for Brain Development, Baltimore, Maryland: Modeling syndromic autism spectrum disorders with patient-derived induced pluripotent stem cells.
R. Karmacharya, Harvard Medical School and Massachusetts General Hospital, Boston: Ex vivo signature of psychosis and treatment response in patient-derived neurons.

SESSION VI: Movement, Pain, and Other Disorders

Chairperson: Z. Cader, University of Oxford, United Kingdom

Z. Cader, University of Oxford, United Kingdom: Neuropharmacology and drug discovery in pain disorders.
D. Butler, Neural Stem Cell Institute, New York: Disease in a dish modeling of neurodegenerative diseases using induced pluripotent stem cells.
L. Ellerby, Buck Institute for Research on Aging, San Francisco, California: Huntington's disease: Using isogenic human HD models for target identification and drug screening.
B. Ryan, University of Oxford, United Kingdom: Identifying and exploiting phenotypes for drug discovery in Parkinson's using iPSC models.
S. Seo, The Lieber Institute for Brain, Baltimore, Maryland: Spatiotemporal landscape of heterogeneity in hPSCs.

SESSION VII: Opportunities and Challenges for Drug Discovery

Chairperson: N. Brandon, AstraZeneca, Waltham, Massachusetts

D. Butler, D. Hiler

J. Erwin, The Lieber Institute for Brain Development, Baltimore, Maryland: Single-cell-omic approaches to understanding cellular and genomic heterogeneity of the brain.
N. Heintz, The Rockefeller University, New York: Cell-type-specific profiling from postmortem human brain.
S. Finkbeiner, Gladstone Institutes, University of California, San Francisco: Target evaluation and small-molecule development with human cell models of neurodegenerative diseases.

SESSION VIII: Wrap-Up and Next Steps

Chairperson: N. Brandon, AstraZeneca, Waltham, Massachusetts

NLRs Sans Frontières

September 17–20

FUNDED BY **The Cold Spring Harbor Laboratory Corporate Sponsor Program and the Gordon and Betty Moore Foundation, with additional support provided by 2Blades Foundation, Burroughs Wellcome Fund, DuPont Pioneer, and Genentech.**

ARRANGED BY **J. Dangl,** HHMI/University of North Carolina, Chapel Hill
J. Jones, The Sainsbury Laboratory, Norwich, United Kingdom
R. Vance, HHMI/University of California, Berkeley

Plants and animals use intracellular proteins of the nucleotide-binding domain, leucine-rich repeat (NLR) superfamily to detect many types of microbial and viral pathogens. The specific combination of domains that define the NLR architecture likely evolved independently in each Kingdom, and the molecular mechanisms of pathogen detection by plant and animal NLRs have long been considered to be distinct. However, microbial recognition mechanisms overlap, and it is now possible to discern important trans-kingdom principles of NLR-dependent immune function. Participants at this Banbury meeting examined how a common function is achieved by NLA proteins in such diverse Kingdoms to identify features that could be useful in building new pathways through synthetic biology, whether for broadening disease defenses or for constructing new signal-response circuits. New and unpublished data shared at this meeting are expected to disrupt the community's understanding of immunity mechanisms.

SESSION I: Sensing

Chairperson: R. Vance, HHMI/University of California, Berkeley

R. Vance, HHMI/University of California, Berkeley: NLRs in plants and animals: Are there any general principles?

P. Schulze-Lefert, Max-Planck Institute for Plant Breeding Research, Cologne, Germany: Functional diversification and effector recognition mediated by a multi-allelic NLR-type disease resistance gene.

S. Shin, University of Pennsylvania, Philadelphia: Recognition of bacterial ligands by the human NAIP/NLRC4 inflammasome.

J. Jones, R. Vance

J. Dangl, B. Staskawicz

A. Daskalov, University of California, Berkeley: NLR-like proteins in fungi.

J.-M. Zhou, Chinese Academy of Science, Beijing, China: Molecular links between an NLR and a PRR in *Arabidopsis*.

G. Nuñez, University of Michigan, Ann Arbor: Role of NLRs in intestinal inflammation.

E. Lien, University of Massachusetts Medical School, Worcester: Regulation of inflammasome activation by bacterial secretion systems.

SESSION II: Sensing and Signaling 1

Chairperson: J. Parker, Max-Planck Institute for Plant Breeding Research, Cologne, Germany

J. Parker, Max-Planck Institute for Plant Breeding Research, Cologne, Germany: Plant TNL nuclear receptors and defense network reprogramming.

M. Keestra-Gounder, University of Colorado, Aurora: Activation of the NOD1 and NOD2 signaling pathway.

R. Innes, Indiana University, Bloomington: Structure and function of the RPS5 NLR protein from *Arabidopsis.*

V. Hornung, Ludwig-Maximilians-University, Munich, Germany: NLRP3 inflammasome signaling in the human system.

J. Dangl, HHMI/University of North Carolina, Chapel Hill: Sensor and helper NLR function in *Arabidopsis.*

B. Staskawicz, University of California, Berkeley: Specific recognition and activation of plant NLR immune receptors.

X. Li, University of British Columbia, Vancouver, Canada: Distinct E3 ligases regulate the turnover of individual components of paired typical plant NLR immune receptors.

SESSION III: Sensing and Signaling 2

Chairperson: D. Philpott, University of Toronto, Canada

D. Philpott, University of Toronto, Canada: NOD proteins in intestinal inflammation.

J. Jones, The Sainsbury Laboratory, Norwich, United Kingdom: How the *Arabidopsis* RPS4/RRS1 immune receptor complex detects effectors and activates defense.

J. Chai, University of Cologne, Germany: Structural study on ligand recognition by an NLR protein.

F. Sutterwala, Cedars-Sinai Medical Center, Los Angeles, California: Mitochondrial regulation of NLRP3 inflammasome activation.

SESSION IV: Signaling

Chairperson: P. Dodds, CSIRO Agriculture & Food, Canberra, Australia

P. Dodds, CSIRO Agriculture & Food, Canberra, Australia: Mechanisms of plant immune receptor function in resistance to rust fungi.

A. Goverse, Wageningen University & Research, The Netherlands: Distinct roles of surface regions of the CC domain in the modulation of effector-triggered immune responses by the potato resistance protein Rx1.

E. Miao, University of North Carolina, Chapel Hill: NLR-driven pyroptosis defends against intracellular pathogens.

B. Kobe, University of Queensland, Brisbane, Australia: Signaling by cooperative assembly formation by mammalian TIR domains and implications for plant NLRs.

S. Kamoun, The Sainsbury Laboratory, Norwich, United Kingdom: Evolutionary dynamics of plant NLRs: From pairs to networks.

SESSION V: Solutions

Chairperson: J. Jones, The Sainsbury Laboratory, Norwich, United Kingdom

R. Terauchi, Kyoto University, Japan: Molecular interaction and coevolution of rice-paired NLRs and *Magnaporthe oryzae*

AVRs: Similarities and differences in Pik NLR/AvR-Pik and Pii NLR/AVR-Pii interactions.

T. Kroj, INRA Montpellier, France: Unconventional integrated domains in plant NLRs provide novel insight into effector recognition and give new perspectives for the engineering of crop immune receptors.

M. Banfield, John Innes Center, Norwich, United Kingdom: Engineering an integrated domain in a plant NLR immune receptor to extend pathogen effector recognition.

P. van Esse, The 2Blades Foundation, Norwich, United Kingdom: Mining NLRs from crop relatives to establish a diverse pool of disease resistance traits.

N. Krishnamurthy, DuPont Pioneer, Johnston, Iowa: Genome editing of R-genes: Opportunities and challenges.

SESSION VI: Meeting Conclusions

Metformin: Translating Biology into the Clinic

September 24–27

FUNDED BY **The Oliver Grace Chair Fund and IC-Medtech**

ARRANGED BY **N. Chandel,** Northwestern University, Chicago, Illinois
V. Stambolic, University of Toronto, Ontario Cancer Institute, Ontario, Canada

Metformin is widely used to treat patients with type 2 diabetes who exhibit high levels of circulating insulin. Recent retrospective studies have uncovered an association between metformin use and diminished tumor progression in patients suffering from different types of cancers. Given the safety of metformin along with its anticancer, anti-inflammatory, and antidiabetic effects, investigators are considering the use of metformin as the first anti-aging drug in clinical trials. This Banbury meeting convened experts to discuss the mechanisms by which metformin exerts its effects, with emphasis on translating this knowledge into the clinic.

Welcoming Remarks: R. Leshan, Banbury Center, CSHL

Introduction & Meeting Objectives: N. Chandel, Northwestern University, Chicago, Illinois
J. Watson, CSHL, Cold Spring Harbor, New York

SESSION I: AMPK

Chairperson: V. Stambolic, University of Toronto, Ontario, Canada

R. Shaw, The Salk Institute for Biological Studies, La Jolla, California: Molecular dissection of Metformin action: AMPK and beyond

G. Hardie, University of Dundee, Dundee, United Kingdom: AMPK as a target for biguanides in diabetes and in cancer

D. Carling, MRC London Institute of Medical Sciences, London, United Kingdom: AMPK in health and disease: Insights using a gain-of-function mouse model

SESSION II: Mitochondria

Chairperson: S. Burgess, UT Southwestern, Dallas, Texas

N. Chandel, Northwestern University, Chicago, Illinois: Metformin target mitochondrial complex I in cancer

K. Birsoy, Rockefeller University, New York, New York: Systematic approaches to understand **mitochondrial**

M. Pollak, McGill University, Montreal, Quebec, Canada: Tyrosine kinase inhibitors and biguanides

G. Draetta, MD Anderson Cancer Center, Houston, Texas: Metabolic dependencies of glioblastoma stem cells

B. Zheng, MGH, Harvard Medical School, Charleston, South Carolina: Repurposing phenformin for cancer treatment

SESSION III: Metabolic Syndrome

Chairperson: R. Shaw, Salk Institute for Biological Studies, La Jolla, California

M. Schwab, Dr. Margarete Fischer-Bosch-Institute of Clinical Pharmacology, Stuttgart, Germany: Metformin and drug disposition: Update and future perspectives

V. Stambolic, University of Toronto, Toronto, Canada: Obesity and cancer, the insulin connection

P. Darrell Neufer, East Carolina University, Greenville, North Carolina: Viewing metformin from a mitochondrial bioenergetics perspective

S. Burgess, UT Southwestern, Dallas, Texas: Energetics of the liver during insulin resistance and NAFLD

G. Steinberg, McMaster University, Hamilton, Canada: AMPK-independent effects of metformin on metabolism: Role of GDF15 and the microbiome

SESSION IV: Cancer

Chairperson: R. Jones, McGill University, Montreal, Quebec, Canada

K. Struhl, Harvard Medical Center, Boston, Massachusetts: Anticancer effects of metformin in mouse xenografts and inducible mouse models

P. Hwang, NHLBI-NIH, Bethesda, Maryland: Using metformin to regulate aberrant mitochondrial metabolism in Li–Fraumeni syndrome

I. Romero, Universty of Chicago, Chicago, Illinois: Repurposing metformin for ovarian cancer treatment, including targets in the tumor microenvironment

SESSION V: Aging

Chairperson: S. Budinger, Northwestern University, Feinberg School of Medicine, Chicago, Illinois

J. van Deursen, Mayo Clinic, Rochester, Minnesota: Senotherapeutic properties of metformin

A. Soukas, Harvard University, Boston, Massachusetts: Ancient metformin response pathways

SESSION VI: Immunity

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

L. Morel, University of Florida, Gainesville, Florida: Metformin treatment in lupus: Evidence from mouse models and patient T cells

S. Budinger, Northwestern University, Feinberg School of Medicine, Chicago, Illinois: Metformin reduces air pollution–induced thrombosis

R. Jones, McGill University, Montreal, Quebec, Canada: Impacts of biguanides on immune cell function

SESSION VII: New Approaches in Metformin

Chairperson: M. Pollak, McGill University, Montreal Quebec, Canada

R. Kalluri, MD Anderson Cancer Center, Houston, Texas: Perturbing vulnerable metabolic pathways using iExosomes-metformin in pancreatic cancer

I. Romeo, J.D. Watson, S. Apple, M. Schwab

G. Hardie, R. Shaw

D. Campbell, Enlibrium, La Jolla, California: Advancement of the novel biguanide, ENL069, for the treatment of cancer

SESSION VIII: Metformin Trials

Chairperson: N. Chandel, Northwestern University, Chicago, Illinois

R. Whitmer, Kaiser Permanente, UCSF, Oakland, California: Diabetes and dementia

N. Barzilai, Albert Einstein College of Medicine, New York, New York: Targeting aging with metformin (TAME)

SESSION IX: General Discussion/Meeting Wrap-Up

Chairperson: V. Stambolic, University of Toronto. Toronto, Canada

V. Stambolic, University of Toronto, Toronto, Canada: Meeting summary/review, road map for future, opportunities for funding

T. Miller, D. Carling

Cell Biology of ALS: Emerging Themes from Human Genetics

October 22–24

FUNDED BY **The Greater New York Chapter of The ALS Association**

ARRANGED BY **L. Bruijn,** The ALS Association, Washington, D.C.
A. Gitler, Stanford University, California
E. Holzbaur, University of Pennsylvania, Philadelphia

A surge in the discovery of genes associated with amyotrophic lateral sclerosis (ALS) has implicated new pathways in pathogenesis of this neurological disease, including vesicular trafficking, mitochondrial quality control, autophagy, neuroinflammation, and DNA damage. In-depth knowledge of these cellular pathways will be required to target the underlying disease processes and drive therapeutic development forward. This Banbury meeting convened leaders in ALS and related fields, along with experts in the corresponding cell biology areas, to identify gaps in knowledge and to highlight opportunities for further research. Presentations and discussion were used as a springboard to identify new strategies in the fight against ALS.

Welcoming Remarks: R. Leshan, Banbury Center, Cold Spring Harbor Laboratory

Introduction and Meeting Objectives: L. Bruijn, The ALS Association, Washington, D.C.

SESSION I: C9orf72

Chairperson: A. Gitler, Stanford University, California

A. Gitler, Stanford University, California: Session overview and introductory comments.

R. Baloh, Cedars-Sinai Regenerative Medical Center, Los Angeles, California; C9orf72 in neurons and microglia.

J. Ichida, University of Southern California, Los Angeles: Chemical perturbation of vesicle trafficking as a strategy to rescue C9orf72-ALS/FTD neurodegeneration.

M. Sendtner, University of Würzburg, Germany: Function of C9orf72 on actin dynamics in motor neurons and other MND-related molecules on actin dynamics in motor neurons.

R. Baloh, E. Holzbaur, P. Gopal, J. Ichida

L. Gan, V. Mootha, D. Cleveland

J. Wang, Johns Hopkins University, Baltimore, Maryland: RNA and protein homeostasis in C9orf72-linked ALS.

F.-B. Gao, University of Massachusetts, Worcester: Investigating DNA damage as a therapeutic target in C9orf72-related ALS/FTD.

SESSION II: Autophagy

Chairperson: E. Holzbaur, University of Pennsylvania, Philadelphia

E. Holzbaur, University of Pennsylvania, Philadelphia: Session overview and introductory comments.

V. Gerbino, Columbia University, New York: The role of TBK1 and motor neuron autophagy in disease progression in a mouse model of ALS.

T. Lloyd, Johns Hopkins University, Baltimore, Maryland: Nucleocytoplasmic transport and autophagy in *Drosophila* models of ALS.

A. Yamamoto, Columbia University, New York: Selective autophagy and ALS.

C. Behrends, Ludwig-Maximilians-University, Munich, Germany: Roles of autophagy in ALS.

SESSION III: Mitochondria

Chairperson: V. Mootha, Harvard Medical School, Boston, Massachusetts

V. Mootha, Harvard Medical School, Boston, Massachusetts; Session overview and introductory comments.

X. Wang, Stanford University, Palo Alto, California: Regulation of mitochondrial trafficking and quality control: Implication in ALS pathogenesis.

H. McBride, McGill University, Montreal, Canada: Emerging evidence linking mitochondrial antigen presentation and neurodegeneration.

A.P. West, Texas A&M University Health Science Center, College Station: Mitochondrial control of innate immunity and inflammation.

SESSION IV-A: Other Mechanisms

Chairperson: D. Cleveland, University of California, San Diego

D. Cleveland, University of California, San Diego: Session overview and introductory comments.

S. Alberti, Max-Planck Institute of Molecular Cell Biology, Germany: RNP granules: How they form, age, and cause disease.

P. Gopal, University of Pennsylvania, Philadelphia: Dynamic, liquid-like TDP-43 RNP granules in neurons and pathologic transitions in disease.

D. Milovanovic, Yale University, New Haven, Connecticut: Synaptic vesicle clusters at the nerve terminal: An example of a liquid phase?

D. Bosco, University of Massachusetts Medical Center, Worcester: Using patient-derived iPSCs to investigate mechanisms underlying FUS- and PFN1-mediated ALS.

M. Zerial, Max-Planck Institute of Molecular Cell Biology, Germany: A novel Rab5-dependent cytoprotective signaling pathway on mitochondria and its implications for ALS.

SESSION IV-B: Other cell types

Chairperson: D. Cleveland, University of California, San Diego

T. Lloyd, D. Bosco

L. Gan, Gladstone Institutes and University of California, San Francisco: Proteotasis and microglial dysfunction in neurodegenerative diseases.

K. McAvoy, Thomas Jefferson University, Philadelphia, Pennsylvania: Role of ALS astrocytes in pharmacology resistance and beyond.

J. Grutzendler, Yale University, New Haven, Connecticut: Role of glia in neurodegeneration: Evidence from optical imaging.

SESSION V: Other Diseases

Chairperson: L. Bruijin, ALS Association, Washington, D.C.

C. Sumner, Johns Hopkins University, Baltimore, Maryland: Impaired motor neuron development precedes degeneration in SMA.

K. Fischbeck, National Institute of Neurological Disorders and Stroke, Bethesda, Maryland: Therapeutics development for hereditary motor neuron disease.

SESSION VI: Meeting Summary and Roadmap for Future

Protective Immunity and Vaccines for Lyme Disease

October 29–November 1

FUNDED BY **The Steven & Alexandra Cohen Foundation**

ARRANGED BY **E. Fikrig,** Yale University, New Haven, Connecticut
S. Schutzer, Rutgers New Jersey Medical School, Newark

Lyme disease, caused by *Borrelia burgdorferi*, is the number one tick-borne disease in the United States and Eurasia. Although its cause was identified nearly 35 years ago, we still do not have a human vaccine on the market in the United States. In the past few years, our understanding of the immune response to the offending microbe has increased exponentially as have relevant technologies. These advancements now virtually assure that a safe and effective vaccine against Lyme disease can be developed and brought to market, representing a major step in decreasing cases as well as costs to the individual and society at large. Experts in Lyme disease, immunology, vaccine development, and public health convened at Banbury in October to assess the benefits and technical aspects of a Lyme vaccine.

Welcoming Remarks: R. Leshan, Director, Banbury Center, Cold Spring Harbor Laboratory

Introduction and Meeting Goals: S. Schutzer, Rutgers New Jersey Medical School, Newark

SESSION I: Overview

Chairperson: D. Rock, University of Illinois, Urbana-Champaign

R. Dattwyler, New York Medical College, Valhalla: The not-so-brief history of *Borrelia burgdorferi.*

S. Plotkin, University of Pennsylvania, Philadelphia: Historical perspective and why don't we have a Lyme vaccine?

S. Telford, Tufts University, Boston, Massachusetts: Reviving LYMErix: Rationale and strategy.

U. Lundberg, Valneva Austria GmbH, Vienna, Austria: Development of a multivalent OspA-based vaccine for prevention of Lyme borreliosis.

SESSION II: Arthropod Interactions

Chairperson: R. Dattwyler, New York Medical College

E. Fikrig, Yale University, New Haven, Connecticut: Tick immunity.

J. Benach, P. Rosa

E. Fikrig, A. Marques, J. Hovius, P. Arnaboldi

J. Pedra, University of Maryland School of Medicine, Baltimore: *Borrelia*–tick interactions.

J. Hovius, University of Amsterdam, The Netherlands: Different vaccinations strategies to prevent Lyme borreliosis: Targeting *Borrelia burgdorferi* and/or the tick vector.

U. Pal, University of Maryland, College Park: Tick targets.

S. Narasimhan, Yale School of Medicine, New Haven, Connecticut: Tick immunity.

SESSION III: Determining Vaccine Efficacy in Animal Models and Humans

Chairperson: S. Schutzer, Rutgers New Jersey Medical School, Newark

S. Schutzer, Rutgers New Jersey Medical School, Newark: Detection of infection in a vaccinated individual.

P. Molloy, Imugen, Norwood, Massachusetts: The role of PCR in detection of Lyme and related TBDs.

SESSION IV: Animal Vaccines

Chairperson: W. Laegreid, University of Wyoming, Laramie

R. Marconi, Virginia Commonwealth University, Richmond: Chimeric epitope-based vaccines for tick-borne diseases.

M. Gomes-Solecki, University of Tennessee Health Science, Memphis: Oral vaccines for Lyme disease.

J. Benach, Stony Brook University, New York: Antigenic lipids of *Borrelia.*

M. Diuk-Wasser, Columbia University, New York: Part 1: Eco-epidemiological determinants for Lyme disease: Considerations for effective implementation of a Lyme vaccine.

J. Tsao, Michigan State University, East Lansing: Part 2: Eco-epidemiological determinants for Lyme disease: Considerations for effective implementation of a Lyme vaccine.

SESSION V: Bioinformatics

Chairperson: D. Rock, University of Illinois, Urbana-Champaign

S. Mitra, Icahn School of Medicine at Mount Sinai, New York: Bioinformatics applied to selection of targets and host responses.

SESSION VI: Public Health Perspectives

Chairperson: A. Marques, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, Maryland

P. Mead, Centers for Disease Control and Prevention, Ft. Collins, Colorado: A public health perspective on vaccination for tick-borne diseases.

S. Plotkin

B. Backenson, New York State Department of Health, Albany: Human and tick surveillance for Lyme disease in New York: How do we know we are targeting the right culprit?

SESSION VII: Vaccine Development against Other Vectors and Targets

Chairperson: W. Laegried, University of Wyoming, Laramie

P. Arnaboldi, New York Medical College, Valhalla: TMV, a novel delivery platform for vector-borne diseases and beyond.

A. Marques, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, Maryland: New challenges in Lyme disease vaccine clinical studies (STARI).

SESSION VIII: Live Attenuated versus Targeted Recombinants

Chairperson: M. Ilias, National Institute of Allergy and Infectious Diseases, NIH, Rockville, Maryland

F. Yang, Indiana University, Indianapolis: Toward development of a live attenuated Lyme disease vaccine.

P. Rosa, National Institute of Allergy and Infectious Diseases, Hamilton, Montana: Activation or neutralization of tick-borne spirochetes.

C. Cooper, USAMRIID, Ft. Detrick, Maryland: Complexity of vaccine-induced B-cell repertoires and its implications toward rationale vaccine design.

SESSION IX: Meeting Wrap-Up and Next Steps

Chairpersons: E. Fikrig, Yale University, New Haven, Connecticut, and **S. Schutzer,** Rutgers New Jersey Medical School, Newark

Lustgarten Foundation Scientific Advisory Board Meeting

November 12–14

FUNDED BY **The Lustgarten Foundation**

ARRANGED BY **A. Whiteley,** The Lustgarten Foundation, New York
R. Vizza, The Lustgarten Foundation, New York
D. Tuveson, Cold Spring Harbor Laboratory

Banbury was pleased to welcome back the Lustgarten Foundation for their 2017 Scientific Meeting, which provided an opportunity for the Scientific Advisory Board, as well as Foundation-supported investigators, to discuss research and strategy, evaluate performance, provide feedback for improvement, strengthen collaboration, and identify new ideas to bolster progress in the field.

Welcoming Remarks: R. Leshan, Banbury Center, Cold Spring Harbor Laboratory

Introduction and Meeting Objectives: A. Whiteley, The Lustgarten Foundation, New York,
D. Tuveson, Cold Spring Harbor Laboratory, and
R. Vizza, The Lustgarten Foundation, New York

PRESENTATIONS FROM TRANSLATIONAL CLINICAL PROGRAM NOMINEES

Organoid Strategy

D. Tuveson, Cold Spring Harbor Laboratory

B. Wolpin, Harvard University Medical School, Boston, Massachusetts

Project Felix: Progress and Future Aims

B. Vogelstein, Johns Hopkins University, Baltimore, Maryland

REVIEW OF RESEARCH INVESTIGATOR PROPOSALS

Distinguished Scholar Presentations

D. Fearon, Cold Spring Harbor Laboratory
R. Evans, Salk Institute for Biological Studies, La Jolla, California
B. Vogelstein, Johns Hopkins University, Baltimore, Maryland
D. Tuveson, Cold Spring Harbor Laboratory

Stand Up to Cancer Progress

D. Tuveson, Cold Spring Harbor Laboratory

Meeting Summary and Wrap-Up

A. Whiteley, The Lustgarten Foundation, New York

Regulated Necrosis: Pathways and Mechanisms

November 26–29

FUNDED BY **Genentech and the Cold Spring Harbor Laboratory Corporate Sponsor Program**

ARRANGED BY **D. Green,** St. Jude Children's Research Hospital, Memphis, Tennessee
A. Linkermann, Technical University Dresden, Germany

In November, Banbury convened a second meeting on regulated necrosis, with this meeting focusing on necroptosis. After reviewing the signaling pathway, defined by RIPK3-mediated phosphorylation of MLKL and subsequent plasma membrane rupture, participants suggested mechanisms that may lead to therapeutic interference, or induction, for clinical applications. With several small-molecule inhibitors of necroptosis in clinical trials, this field has already entered the clinic. Lively discussions among the expert participants, including sharing of new and unpublished data, inspired ideas to move the field forward on biological and translational levels.

Welcoming Remarks; R. Leshan, Banbury Center, Cold Spring Harbor Laboratory

Introduction and Meeting Objectives: D. Green, St. Jude Children's Research Hospital, Memphis, Tennessee
A. Linkermann, Technical University Dresden, Germany

T. Vanden Berghe, F. Chan

H. Walczak, M. Pasparakis, J. Vince, J. Silke, P. Jost

SESSION I: TNF and Necroptosis

Chairperson: A. Oberst, University of Washington, Seattle

F. Chan, University of Massachusetts Medical School, Worcester: Climate change: Environmental control of TNF signaling.

M. Bertrand, VIB-UGent Center for Inflammation Research, Gent, Belgium: Regulation of RIPK1 life/death decisions during TNF signaling.

H. Walczak, UCL Cancer Institute, London, United Kingdom: How linear ubiquitin enables and regulates signaling: Gene activation versus different forms of cell death.

M. MacFarlane, MRC Toxicology Unit, Leicester, United Kingdom: FADD:Caspase-8 signaling complexes: Coordinated control of life/death decisions.

SESSION II: The Immune Response to Necroptosis

Chairperson: D. Vucic, Genentech, South San Francisco, California

J. Blander, Weill Cornell Medicine, New York: The many ways tissue phagocytes respond to dying cells.

M. Albert, Genentech, South San Francisco, California: Cell death and antigen cross-priming.

A. Oberst, University of Washington, Seattle: The immune consequences of necroptotic cell death in normal tissues and in cancer.

J. Vince, Walter and Eliza Hall Institute for Medical Research, Bundoora, Victoria, Australia: Live and let die: Cell death and inflammasomes.

SESSION III: Necroptosis in Viral Defense

Chairperson: E. Mocarski, Emory University School of Medicine, Atlanta, Georgia

E. Mocarski, Emory University School of Medicine, Atlanta, Georgia: Cell death, interferon, and virus-encoded suppressors of apoptosis and necroptosis.

A. Winoto, University of California, Berkeley: Necroptotic signaling pathway during Sendai virus infection.

W. Kaiser, University of Texas, San Antonio: Virus subversion of necroptosis.

J. Upton, University of Texas, Austin: Murine cytomegalovirus IE3-dependent transcription is required for DAI/ZBP1-mediated necroptosis.

SESSION IV: The Therapeutic Potential of Necroptosis

Chairperson: W. Kaiser, University of Texas, San Antonio

A. Degterev, Sackler School of Graduate Biomedical Sciences, Boston, Massachusetts: Small-molecule inhibitors of necroptosis.

A. Linkermann, Technical University Dresden, Germany: Therapeutic intervention of regulated necrosis.

T. Vanden Berghe, VIB-Ghent University, Belgium: Preclinical exploration of ferroptosis: How to induce it or block it?

P. Jost, Technical University of Munich, Germany: Necroptosis in cancer.

SESSION V: Execution Mechanisms of Necroptosis

Chairperson: T. Vanden Berghe, VIB-Ghent University, Belgium

P. Meier, Institute of Cancer Research, London, United Kingdom: Regulation of MLKL.

J. Silke, Walter and Eliza Hall Institute of Medical Research, Bundoora, Victoria, Australia: Chimeric MLKL.

A. Garcia-Saez, Universität Tübingen, Germany: Necroptosis execution by MLKL at the single-molecule level.
S. Oddo, Arizona State University, Tempe: Necroptosis activation in Alzheimer's disease.
R. Buffenstein, Calico Life Sciences LLC, South San Francisco, California: The bare essentials of healthy aging in naked mole rats.

SESSION VI: RIPK1 in Necroptosis

Chairperson: J. Vince, Walter and Eliza Hall Institute for Medical Research, Victoria, Australia

M. Pasparakis, University of Cologne, Germany: RIPK1 signaling in cell death and inflammation.
D. Vucic, Genentech, South San Francisco, California: Regulation of inflammatory cell death signaling by RIP kinases.
D. Green, St. Jude Children's Research Hospital, Memphis, Tennessee: Suppression of necroptosis by RIPK1.

SESSION VII: Meeting Summary and Wrap-Up Chairpersons: **D. Green,** St. Jude Children's Research Hospital, Memphis, Tennessee, and **A. Linkermann,** Technical University Dresden, Germany

R. Buffenstein, S. Oddo

A. Linkermann, J. Blander

Post-Traumatic Neuroinflammation: Roles in Pathogenesis of Long-Term Consequences and Repair

December 6–8

FUNDED BY **Andrea B. and Peter D. Klein**

ARRANGED BY **R. Ransohoff,** Third Rock Ventures, Boston, Massachusetts
A. Schaefer, Icahn School of Medicine at Mount Sinai, New York
D. Schafer, University of Massachusetts Medical School, Worcester

Traumatic injury of the central nervous system (CNS) is a broad term covering diverse conditions, all of which result in an incalculable toll of suffering and loss of function for the patient and a severe burden for family and caregivers. Although all traumatic conditions of the CNS occur suddenly, there is considerable evidence for an ongoing process of tissue injury which extends beyond the time of insult. Particularly relevant are the glial cells, which establish and maintain the optimal environment for neurons; glia respond to tissue injury and to neuronal dysfunction or demise, in a process termed "neuroinflammation." It is becoming apparent that the glial response to neuronal injury may be appropriate or inappropriate, helpful, harmful, or neutral and that complex reaction programs are more the norm than the exception. This Banbury meeting brought together experts from relevant research and clinical fields to examine the effects of the glial reaction to outcomes of CNS injury and to identify promising strategies to manipulate this response to enable improved CNS function, including areas for collaboration and gaps in the overall research agenda.

Welcoming Remarks: R. Leshan, Banbury Center, Cold Spring Harbor Laboratory

Introduction and Meeting Objectives: R. Ransohoff, Third Rock Ventures, Boston, Massachusetts

E. Hughes, G. Lemke

S. Bilbo, P. Greer

SESSION I: Basic Inflammation Biology

Chairperson: A. Schaefer, Icahn School of Medicine, Mount Sinai, New York

G. Lemke, Salk Institute for Biological Studies, La Jolla, California: TAM receptors and neuroinflammation.

R. Ransohoff, Third Rock Ventures, Boston, Massachusetts: Somatic mutations in microglia and neurodegeneration.

R. Klein, Washington University School of Medicine in St. Louis, Missouri: Learning from viruses: Mechanisms of postinfectious cognitive dysfunction.

P. Greer, University of Massachusetts Medical School, Worcester: Detecting internal and external chemical cues using MS4A chemosensors.

SESSION II: Getting to Know the Glia

Chairperson: S. Bilbo, MGH Harvard Medical School, Charlestown, Massachusetts

S. Bilbo, MGH Harvard Medical School, Charlestown, Massachusetts: Brain-immune interactions in neurodevelopment: Implications for health and disease throughout the life span.

X. Piao, Harvard Medical School, Boston, Massachusetts: Glial mechanism of white matter repair.

A. Schaefer, Icahn School of Medicine at Mount Sinai, New York: Epigenetic control of regional microglia clearance activity.

E. Hughes, University of Colorado School of Medicine, Aurora: CNS injury and $NG2^+$ glial cell dynamics.

A. Mishra, Oregon Health & Science University, Portland: Regulation of cerebral blood flow in health and disease.

SESSION III: Let's Get Real: Clinical Problems

Chairperson: M. Chopp, Henry Ford Hospital, Detroit, Michigan

M. Chopp, Henry Ford Hospital, Detroit, Michigan: Stroke: Mechanisms and therapeutic approaches.

P. Gressens, Inserm, Paris, France: Integrative genomics of microglia implicates DLG4 (PSD95) in the white matter development of preterm infants.

A. McKee, Boston University School of Medicine, Massachusetts: Posttraumatic tauopathy.

S. Stukas, University of British Columbia, Vancouver, Canada: TBI in Canada: Assets and opportunities.

J. Ninkovic, Helmholtz Zentrum München & Biomedical Center of LMU Munich, Germany: Features of glial reaction to brain injury in regeneration-competent and regeneration-incompetent vertebrates.

S. Rosi, University of California, San Francisco: The role of infiltrating macrophages and complement initiation after brain injury.

SESSION IV: Injury and Repair: Models and Mechanisms

Chairperson: M. Buckwalter, Stanford University Medical Center, California

M. Buckwalter, Stanford University Medical Center, California: Adaptive immune responses and cognitive impairment after stroke.

SESSION V: Meeting Summary, Wrap-Up, and Next Steps

Chairperson: R. Ransohoff, Third Rock Ventures, Boston, Massachusetts

R. Ransohoff, Third Rock Ventures, Boston, Massachusetts: Prospects for glial-directed therapeutics to enhance outcomes of brain injury.

BANBURY CENTER GRANTS

Grantor	*Program*	*2017 Funding*
FEDERAL SUPPORT		
U.S. National Science Foundation, an award to R. Michelmore, University of California, Davis	Opportunities for Reduction of Aflatoxin Contamination of Food	$14,500
NIH/National Cancer Institute (grant to D. Tuveson, CSHL)	Integrated Translational Science Center Workshop	38,460
NONFEDERAL SUPPORT		
2Blades Foundation	NLRs Sans Frontières	5,000
Andrea B. and Peter D. Klein	Posttraumatic Neuroinflammation: Roles in Pathogenesis of Long-Term Consequences and Repair	40,000
Bill & Melinda Gates Foundation	Maximizing Impact of New HIV Prevention Technologies	60,111
Boehringer Ingelheim Fonds	BIF Fellows Retreat: Communicating Science	69,390
Burroughs Wellcome Fund	Ferroptosis: A Critical Review	15,000
Burroughs Wellcome Fund	NLRs Sans Frontières	5,000
CSHL Corporate Sponsor Program	Enhanceropathies: Enhancer function variation in animal development, morphological variation, and disease	48,335
CSHL Corporate Sponsor Program	Ferroptosis: A Critical Review	17,977
CSHL Corporate Sponsor Program	Neuropharmacology and Human Stem Cell Models	38,969
CSHL Corporate Sponsor Program	NLRs Sans Frontières	38,266
CSHL Corporate Sponsor Program	Opportunities for Reduction of Aflatoxin Contamination of Food	16,442
CSHL Corporate Sponsor Program	Regulated Necrosis, Pathways, and Mechanisms	18,945
Collaborative Medicinal Development, LLC	Ferroptosis: A Critical Review	15,000
DuPont Pioneer	NLRs Sans Frontières	5,000
Genentech	NLRs Sans Frontières	3,000
Genentech	Regulated Necrosis, Pathways, and Mechanisms	30,000
Gordon and Betty Moore Foundation	NLRs Sans Frontières	10,000
IC MedTech	Metformin: Translating Biology into the Clinic	25,000
Kansas State University	Opportunities for Reduction of Aflatoxin Contamination of Food	6,535
Keystone for Incubating Innovation in Life Sciences Network	Foundation2017: Bio-Entrepeneurship in NYC	35,500
L'Orèal USA	Chemiexcitation in Human Disease and Aging	4,535
Lieber Institute for Brain Development	Neuropharmacology and Human Stem Cell Models	13,070
Lustgarten Foundation	Lustgarten Scientific Advisory Board meeting	20,215
MARS, Inc.	Opportunities for Reduction of Aflatoxin Contamination of Food	15,000
Memorial Sloan Kettering Cancer Center	Ferroptosis: A Critical Review	2,550
Northwell Health	Project Santa Fe	35,265
Oliver Grace Chair Fund	Better Cancer Therapy from Redox Biology	74,797
Oliver Grace Chair Fund	Metformin: Translating Biology into the Clinic	40,000
Ono Pharmaceutical Co., Ltd.	Ferroptosis: A Critical Review	5,000
Steven and Alexandra Cohen Foundation	Protective Immunity and Vaccines for Lyme Disease	54,713
The Greater New York Chapter of The ALS Association	Cell Biology of ALS: Emerging Themes from Human Genetics	51,898
The LEO Foundation of Ballerup, Denmark	Chemiexcitation in Human Disease and Aging	41,797