VALLEE FOUNDATION SYMPOSIUM 2016

RAPALLO, ITALY | JUNE 17-20, 2016

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IMPORTANT NOTE:

The abstracts in this meeting book should be treated as personal communications and be cited only with the consent of the author.

ORGANIZERS

Gordon G Hammes, PhD
Duke University

Jesper Z Haeggström, MD, PhD
Karolinska Institutet

Peter M Howley, MD
Harvard Medical School

Ernst-Ludwig Winnacker, PhD
Gene Center Munich, LMU

MEETING STAFF

Alexa M Mason
The Vallee Foundation

Amanda Chin
Strategy Implemented Inc

Daniela Daveri
Soriasco, Italy

Amanda Pullen, PhD
Strategy Implemented Inc

BENVENUTO!

In the spirit of fostering collaboration among scientists in a number of basic biomedical fields, this year's Vallee Foundation Summer Symposium is a multi-disciplinary meeting which will showcase work being done by selected Vallee Visiting Professors and Young Investigators. **The meeting has been divided into five main sessions:** *Young Investigator Talks; Responding to the Environment; Crossing Membranes; Cellular Homeostasis Mechanisms; and Cancer Biology.*

As a tradition, and to give younger scientists the opportunity to participate, ten outstanding trainees have also been invited to attend and will present posters on their work.

The two-day event is being held in Rapallo on the beautiful Italian Riviera. With conversations across a broad spectrum of science against a background of sun, sea, and Italian food, the meeting promises to follow the traditions established by Bert and Kuggie Vallee when they created the Vallee Foundation twenty years ago.

We are delighted to welcome you to the 2016 Summer Symposium.

EXCELSIOR PALACE
HOTEL

Bert L Vallee, *Founder*
1919-2010

N Kuggie Vallee, *Founder*
1921–2011

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Assistant Secretary

THE BERT L & N KUGGIE VALLEE FOUNDATION INC

Bert and Kuggie Vallee established their namesake foundation in 1996 to improve scientific research by promoting dialogue between active and prominent biomedical scientists around the world—and to enjoy themselves while doing so. The means they first chose was by sponsoring visiting professorships among institutions with which Bert had developed close collaborations and by organizing biennial meetings of this group. Twenty years later, their dream to foster originality, creativity, and leadership within biomedical scientific research and medical education has developed into a robust foundation that supports a unique visiting professorship program, provides unrestricted funding to junior faculty, and offers a number of other ways to recognize scientific excellence, help young people at the start of their career, and encourage women in science.

The Vallee Visiting Professorships pair outstanding scientists with premier biomedical research institutes worldwide in an informal arrangement that promotes intellectual exchanges and fosters new partnerships. To date, 56 senior scientists have been able to take their one-month VVP sabbatical at any biomedical research institute worldwide and have returned home reinvigorated, inspired, and with new collaborations in their pockets.

The Young Investigators are junior faculty in tenure-track positions working independently on outstanding projects that could do with a little extra flexible funding. These awards can be spent over a period of five years, during which time the YIs meet each other and the VVPs to share their findings and cement their ties within the Vallee network. The fourth round of Young Investigators will be appointed this summer.

The Bert and Natalie Vallee Award in Biomedical Science recognizes international achievements in the sciences basic to medicine. This year's recipient, the third, is Aziz Sancar, the Sarah Graham Kenan Professor of Biochemistry and Biophysics, University of North Carolina School of Medicine, who also shared the 2015 Nobel Prize in Chemistry (with Tomas Lindahl and Paul Modrich). He received his Vallee Award at the annual American Society for Biochemistry and Molecular Biology meeting this past spring.

The Vallee Lindau Fellowships allow gifted, motivated, and productive postdocs to attend the Lindau Nobel Laureate Meetings in physiology and medicine or chemistry. This week of unparalleled exposure to the brightest of minds is often a turning point in the life of young scientists, inspiring them to continue the scientific path and start making the connections that are so important later in life.

We are delighted to announce a new program to inspire young women to continue a career in science and to honor our co-founder. Bonnie Bassler, Squibb Professor of Molecular Biology at Princeton University, will be the first **N Kuggie Vallee Distinguished Lecturer**.

Bert Vallee's original idea to bring the VVPs together every other year to discuss their science has developed into the **Vallee Summer Symposia**. Meetings are sometimes themed, sometimes focus on the Young Investigators, and sometimes bring together VVPs and YIs in all fields to share their work in an inspiring location that sparks the imagination. In the words of Sir Alan Fersht (VVP 2008): "*It's very important for scientists to have fun together. Bert had a really rather grand vision of what science and scientists should be like…that science should be fun in itself—everyone should enjoy doing it—and scientists should get together and have fun discussing science and doing things together. Bert was definitely a catalyst for that.*"

PROGRAM

The Excelsior Palace Hotel, Rapallo
June 16-20, 2016

THURSDAY, JUNE 16, 2016

Arrivals/Board Members
19.30 Board Dinner *(Yacht Club)*

FRIDAY, JUNE 17, 2016

08.30-10.00	Finance Committee Meeting *(Sala Soraya)*
10.00-10.30	Break
10.30-13.00	Board Meeting *(Sala Soraya)*
13.00-14.00	Board Lunch
Afternoon	Arrival of Symposium Delegates
18.30	Welcome Reception *(terrace outside Sala Simpson)*
19.30	Dinner *(Sala Faruk)*

SATURDAY, JUNE 18, 2016

Session 1 YOUNG INVESTIGATOR TALKS *(Sala Simpson)*

*Chaired by **Gordon Hammes**, Duke University*

09.00 Welcome and Introduction
***Peter Howley**, President, The Vallee Foundation*

09.30 **Imaging and Regulation of the Cellular Events that Shape the Embryo**
***Jérôme Gros**, Institut Pasteur*

10.10 **Human Adipose Tissue and Metabolic Health**
***Kirsty Spalding**, Karolinska Institutet*

10.50 Break

Session 2 RESPONDING TO THE ENVIRONMENT

*Chaired by **Ernst-Ludwig Winnacker**, Gene Center Munich, LMU*

11.10 **Understanding Mitochondrial Quality Control**
***Wade Harper**, Harvard Medical School*

11.50 **Zooming in on Spatial Control of Receptor-Mediated Cellular Responses**
***Barbara Baird**, Cornell University*

12.30 Lunch *(Sala Faruk)*

13.30 Poster Session & Coffee *(Sala Soraya)*

15.30 Boat Trip

19.30 Dinner *(Ristorante Luca, Porto Turistico Carlo Riva, Rapallo)*

SUNDAY, JUNE 19, 2016

Session 3 CROSSING MEMBRANES

*Chaired by **Jesper Haeggström**, Karolinska Institutet*

09.00 **Dynamic Membrane Organisation**
***Kai Simons**, Max Planck Institute for Molecular Cell Biology and Genetics, Lipotype GmbH*

09.40 **Cell Biology of Virus Entry into Host Cells**
***Ari Helenius**, ETH Zurich*

10.20 **Pumps, Pores and Channels: Out of the Membrane into the Gas Phase**
***Carol Robinson**, The University of Oxford*

10.40 Break

Session 4 CELLULAR HOMEOSTASIS MECHANISMS

*Chaired by **Wade Harper**, Harvard Medical School*

11.20 **Maintaining Expression Homeostasis During DNA Replication**
***Naama Barkai**, Weizmann Institute of Science*

12.00 **From Protein Folding to Cognition: The Serendipitous Path of Discovery**
***Peter Walter**, University of California, San Francisco*

12.40 Lunch *(Sala Faruk)*

Session 5 CANCER BIOLOGY

*Chaired by **Karen Vousden**, Beatson Institute of Cancer Research*

14.00 **The Bcl-2 Family: An Achilles' Heel for Cancer.**
***Suzanne Cory**, Walter and Eliza Hall Institute of Medical Research*

14.40 **How Does the Tumor Suppressor p53 Protect Us From Getting Cancer?**
***Andreas Strasser**, Walter and Eliza Hall Institute of Medical Research*

15.20 **MiRNA Control at the Level of a Single, Ubiquitously Expressed Target Gene**
***Klaus Rajewsky**, The Max Delbrück Center for Molecular Medicine*

16.00 Closing Remarks

16.20 Break

20.00 Reception *(Pool Terrace)*

20.30 Dinner *(Eden Roc Restaurant)*

MONDAY, JUNE 20, 2016

Departures All

SPEAKERS

Jérôme Gros, PhD

Assistant Professor of
Developmental and
Stem Cell Biology,
Institut Pasteur

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Imaging and Regulation of the Cellular Events that Shape the Embryo

Embryonic development requires that the behavior of thousands of cells be finely orchestrated in concert with signaling pathways. While much work has focused on the genetic and transcriptional control of morphogenesis, how cell dynamics and behavior are controlled has remained more elusive. In order to understand how structures and organs form, it is crucial to understand how cells, the building blocks of tissue, assemble in real time, as morphogenesis is taking place. This achievement has proven difficult due to technical hurdles in higher vertebrates, since embryos, optically opaque, are not easily amenable to static live imaging conditions. Taking advantage of recent developments in genome sequencing and editing technologies, we will develop genomic resources and molecular genetic strategies for the quail (*Coturnix Japonica*) as a true genetic animal model to decipher basic rules driving epithelial morphogenesis in amniotes (mammals, birds, and reptiles). By combining such resources and the amenability of quail embryos to live imaging methodologies, we will investigate the dynamic mechanisms underlying epithelial morphogenesis during early embryogenesis. Specifically, we will investigate how cell-cycle progression dynamically modulates adhesive and contractile properties of epithelial cells during early embryogenesis, using genetically modified quail embryos. All together, these studies aim at establishing the quail as the avian genetic model of reference in order to address fundamental mechanisms underlying dynamic morphogenetic mechanisms in higher vertebrates.

Jérôme Gros obtained his PhD in 2006 from the University of Marseille-Luminy in France, in the field of Developmental Biology. He then moved to Boston, USA as a postdoctoral fellow in the laboratory of Cliff Tabin in the Department of Genetics at Harvard Medical School. In 2012, Jérôme became an Assistant Professor of Developmental and Stem Cell Biology at the Institut Pasteur in Paris, France, where his research focuses on the imaging and regulation of the cellular events that shape the early vertebrate embryo.

https://research.pasteur.fr/en/team/morphogenesis-regulation-in-higher-vertebrates/

Human Adipose Tissue and Metabolic Health

Obesity is increasing in an epidemic manner in most countries and constitutes a public health problem by enhancing the risk for diseases such as diabetes, fatty liver disease, and atherosclerosis. Together these diseases form a cluster referred to as the metabolic syndrome. Owing to the increase in obesity, life expectancy may start to decrease in developed countries for the first time in recent history. Despite the importance of the fat mass, very little is known about the maintenance of fat cells (adipocytes) in humans, how different fat depots are regulated, and how, or if, this is altered in obesity. My research group uses recently developed strategies, such as radiocarbon dating and retrospective lineage tracing, to investigate the origin and turnover of adipocytes, their progenitor cells, and lipid stores in lean and obese individuals. Basic physiological functions of adipocytes in lean and obese individuals are also investigated. Understanding adipocyte heterogeneity and the dynamics of adipocyte turnover, may shed new light on potential treatments for obesity.

Originally from Perth, Australia, Dr Spalding received her PhD from the University of Western Australia's School of Anatomy and Human Biology, in the field of Neuroscience. In 2002, Kirsty moved to Stockholm, Sweden as an Ambassadorial Academic Scholar. This was followed by a postdoctoral fellowship in the neural stem cell laboratory of Professor Jonas Frisen, at the Department of Cell and Molecular Biology at the Karolinska Institute. Kirsty is currently an Assistant Professor of CMB at KI. Her primary research field is regenerative medicine, with a focus on cell turnover in human fat and brain tissue.

http://ki.se/people/kirspa

Kirsty Spalding, PhD

Assistant Professor,
Department of Cell &
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Karolinska Institutet

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Wade Harper, PhD

Bert and Natalie Vallee Professor of Molecular Pathology

Chairman, Department of Cell Biology

Co-Director, Training Program in Cell and Developmental Biology

Harvard Medical School

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Understanding Mitochondrial Quality Control

Wade Harper's lab employs proteomic and genetic approaches to uncover key signaling systems, ubiquitin ligases, and regulatory circuits that control various biological pathways. Protein turnover through the ubiquitin system is a central means by which the abundance of regulatory proteins is controlled. Many such proteins are involved in signal transduction cascades linked with cell proliferation, checkpoints, and cancer.

Wade Harper joined the faculty in the Department of Pathology at Harvard Medical School in 2003, arriving from Baylor College of Medicine. He earned his PhD at the Georgia Institute of Technology in Chemistry in 1984. Immediately following this, Wade performed his post-doctoral work at HMS before joining the Department of Biochemistry faculty at Baylor College of Medicine in 1988. Wade joined the Department of Cell Biology (HMS) in 2011.

https://harper.hms.harvard.edu/

Zooming in on Spatial Control of Receptor-Mediated Cellular Responses

The Baird/Holowka research laboratory integrates biological, physical, and chemical approaches to investigate basic mechanisms by which cellular receptors mediate transmembrane signals in immune and other physiological responses.

Barbara is the Horace White Professor of Chemistry and Chemical Biology at Cornell University, and she currently serves as Senior Associate Dean in the College of Arts and Sciences. She received her BA in Chemistry from Knox College and her PhD in Chemistry from Cornell University. Her postdoctoral studies were carried out as a Damon Runyon Fellow in the Immunology Branch of the National Cancer Institute at the National Institutes of Health before she joined the Cornell faculty in 1980. Barbara has led a number of programs at Cornell; recent positions include Chair of Chemistry and Chemical Biology, Director of the Nanobiotechnology Center (a Science and Technology Center of the National Science Foundation), Director of Graduate Studies in Chemistry, Director of Cornell's NIH training grant in Molecular Biophysics, and Co-Director of the WM Keck Foundation Program, run jointly with faculty at Weill Cornell Medical College on Molecular and Cellular Biophysics of Signal Transduction. Barbara has served on the Council of the NIH National Institute of Allergy and Infectious Diseases and on scientific advisory/review committees for Los Alamos National Laboratory, Brookhaven National Laboratory, and centers at several universities. She is a Fellow of the American Academy of Arts and Sciences, Fellow of the American Association for the Advancement of Science, and Fellow of the Biophysical Society.

http://chemistry.cornell.edu/faculty/detail.cfm?netid=bab13

Barbara Baird, PhD

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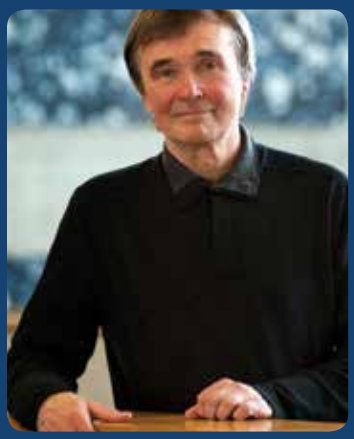

Kai Simons, MD

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Molecular Cell Biology
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Dynamic Membrane Organisation

Kai Simons' recent research has focused on cell membrane organization and function. He has pioneered the concept of lipid rafts as a membrane organizing principle, based on the phase-separating capabilities of sphingolipids and cholesterol in cell membranes.

Kai received his MD degree from the University of Helsinki and his board certification in 1964. He then conducted postdoctoral research with A.G. Bearn at Rockefeller University in New York. In 1967, he accepted a position from the Finnish Medical Research Council at the University of Helsinki with appointments in the Departments of Biochemistry, and Bacteriology and Serology. In 1975, he became a Group Leader at the European Molecular Biology Laboratory (EMBL) in Heidelberg, Germany, and he started the Cell Biology Program, which became the focal point for molecular cell biology in Europe. In 2001, Kai moved to Dresden to build up the new Max Planck Institute for Molecular Cell Biology and Genetics. This Institute is today an internationally recognized center in its area of research.

For his contributions to cell biology, Kai has received numerous accolades, including the Keith Porter Lecturer of the American Society for Cell Biology, a Harvey Society Lecturer, Dunham Lecturer at Harvard Medical School, and Li Lecturer, UC Berkeley. He has received the Anders Jahre Prize for Medical Research, the Runeberg Prize, Finland, the Laurens van Deenen Medal, University of Utrecht, the Schleiden Medal of the Academy Leopoldina and the Äyräpää Prize, Finland. He is Doctor Honoris Causa at the universities of Geneva, Oulu, and Kuopio, Finland and Leuven, Belgium. Kai is a foreign member of the National Academy of Sciences, USA, and was the President of the European Life Scientist Organization. In 2007-2008, he was co-director of the Shanghai Institute for Advanced Studies of the Chinese Academy of Science.

https://www.mpi-cbg.de/research-groups/current-groups/kai-simons/group-leader.html

Cell Biology of Virus Entry Into Host Cells

The common denominator in the research of Ari Helenius has been the use of animal viruses to analyze basic cellular processes, and—in the reverse—the application of cell biological and biochemical methods and concepts to investigate how viruses exploit cellular functions during their replication cycle. The main focus has been on endocytosis, signaling, trafficking, and membrane fusion during cell entry of viruses, as well as on protein folding, quality control, and secretion during propagation of new virus particles. The discoveries include the recognition of a network of acidic prelysosomal organelles (endosomes), the acid-activation and fusion mechanism of viral membrane fusion proteins, the calnexin/calreticulin cycle in glycoprotein folding, and the existence of a quality control system that prevents the deployment of mis-folded and -assembled proteins in the cell. In recent years, a large number of host factors has been identified as important in viral capsid uncoating, nuclear import, endosome maturation, and intrinsic immunity.

Ari received his PhD in the University of Helsinki before moving to the newly founded European Molecular Biology Laboratory (EMBL) in Heidelberg. He was appointed associate professor in 1981 and later full professor of Cell Biology in the Yale School of Medicine. He also served as Chair of the Cell Biology Department until moving to ETH Zurich in 1997, where he chaired the Institute of Biochemistry. He is a co-founder of a biotechnology start-up company, 3V-Biosciences, Inc. Moreover, he is a member of the European Molecular Biology Organization (EMBO), Academia Leopoldina, and the American Academy of Microbiology, as well as a foreign member of the National Academy of Sciences USA. His prizes include the Benoist prize in Switzerland, the Ernst Jung Prize in Medicine, the Schleiden Medal in Cell Biology of the Academia Leopoldina, and the Otto Warburg Medal of the German Society for Biochemistry and Molecular biology.

http://www.bc.biol.ethz.ch/research/helenius.html

Ari Helenius, PhD

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Pumps, Pores and Channels: Out of the Membrane, Into the Gas Phase

Research in the Robinson group is focused on studying proteins and their complexes in the gas phase of the mass spectrometer. Researchers in her laboratory have demonstrated that the overall topology of macromolecular assemblies can be preserved by developing ion mobility methods for use in combination with mass spectrometry. Using this combination of approaches, they have been able to separate two complexes with identical mass to charge, a ring-shaped 11-mer and a collapsed form, separated according to their different packing arrangements. They have since gone on to develop gas phase-unfolding protocols, using this ion-mobility mass spectrometry approach, and to study protein stability as a function of small molecule binding.

Carol pursued her graduate education at the University of Cambridge, where she completed her PhD in two years. Following an eight-year career break to begin raising her three children, she returned to research taking up professorial posts at both the University of Oxford and, later, the University of Cambridge. She returned to Oxford in 2009 to take up the Chair of Doctor Lee's Professor of Chemistry, which she holds today.

Carol's research has attracted international awards and prizes, including the Anfinsen Award from the Protein Society, the Biemann Medal from the American Society of Mass Spectrometry, the Davy Medal and the Rosalind Franklin Award from the Royal Society, the HUPO Award for Distinguished Achievement in Proteomic Science, and the Anatrace Award for Membrane Proteins from the American Biophysical Society. Carol also holds three honorary doctorates and received a DBE in 2013 for her contribution to science.

http://robinsonweb.chem.ox.ac.uk/carol-robinson.aspx

Maintaining Expression Homeostasis During DNA Replication

Naama Barkai is Professor at the Weizmann Institute of Science, where she is using theoretical and computational tools to investigate system-level properties of biological networks. Cells are constantly "making decisions"—monitoring their environment, modulating their metabolism and "deciding" whether to divide, differentiate, or die. For this, they use biochemical circuits composed of interacting genes and proteins. Advances over the past decades have mapped many of these circuits. Still, can we infer the underlying logic from the detailed circuit structure? Can we deduce the selection forces that shaped these circuits during evolution? What are the principles that govern the design and function of these circuits and how similar or different are they from principles that guide the design of man-made machines? The interplay between variability and robustness is a hallmark of biological computation: biological systems are inherently noisy, yet control their behavior precisely. Research projects in our lab quantify biological variability and identify its genetic origins, examine how variability is buffered by molecular circuits, and investigate whether variability can in fact be employed to improve cellular computation. We encourage a multi-disciplinary approach, combining wet-lab experiments, dynamic-system theory, and computational data analysis. This is achieved through fruitful interactions between students with backgrounds in physics, biology, computer science, mathematics, and chemistry.

Naama is a member of the European Molecular Biology Organization (EMBO), the European Bioinformatics Institute (EBI), the ERC Starting Grant evaluation panel, the European Molecular Biology Laboratory (EMBL) and Academia Europaea. She has published extensively and given over 100 seminars and invited talks. Internationally, she has been recognized with the first-ever FEBS/EMBO Women in Science Awards, as well as the Teva Prize for research in system biology, the Michael Bruno Memorial Award (Yan Hanadiv Foundation), the Levinson Award (Weizmann Institute of Science) an EMBO Young Investigator, the Sir Charles Clore prize, among others.

http://barkai-serv.weizmann.ac.il/GroupPage/

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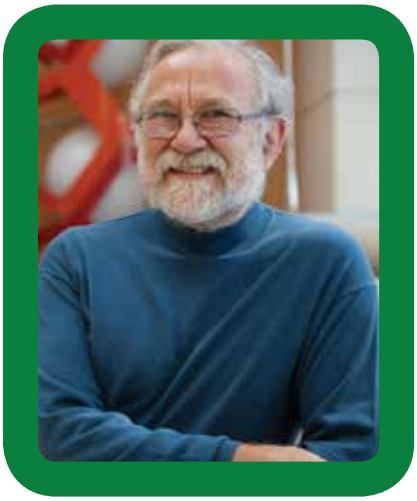

Peter Walter, PhD

Professor,
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HHMI Investigator

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From Protein Folding to Cognition: The Serendipitous Path of Discovery

In his laboratory in the Department of Biochemistry and Biophysics at the University of California at San Francisco (UCSF), Peter Walter turned his attention to deciphering the pathways that cells use to regulate the abundance of their internal organelles. With a particular focus on the endoplasmic reticulum, the organelle in which many newly made proteins are assembled, his lab uncovered the "unfolded protein response," a complex cell-internal signaling network that adjusts the cell's protein-folding capacity to demand. Regulating the abundance of the endoplasmic reticulum is a fundamental process for all eukaryotic cells, and it is a key determinant for any number of diseases, including cancer, diabetes, and neurodegenerative diseases. Most disease connections arise because the cell is programmed to die, rather than putting defective and potentially harmful proteins on its surface. His lab has identified the genes that are centrally involved in the unfolded protein response and deciphered their function in this crucial cell internal-communication pathway.

Since 1997, Peter has been an investigator of the Howard Hughes Medical Institute. He joined UCSF in 1983 and, as of 2015, is a Scientific Member at the Max Planck Institute for Biophysics. He is the 2016 President-Elect of the American Society of Cell Biology and an elected member of several prestigious scientific societies such as the German Academy of Natural Scientists Leopoldina, the US National Academy of Sciences, the American Association for Arts and Science, and the European Molecular Biology Organization. He is a co-author of the textbooks *Molecular Biology of the Cell* and *Essential Cell Biology*, two of the world's most widely-used standard works in the field of molecular cell biology. Among the many awards he has received are the Eli Lilly Award in Biological Chemistry, the Passano Award, the Wiley Prize in Biomedical Sciences, the Stein & Moore Award from the Protein Society, the Gairdner Award, the EB Wilson Medal from the American Society of Cell Biology, the Otto Warburg Medal from the German Biochemical Society, the Jung Prize, the Paul Ehrlich and Ludwig Darmstaedter Prize, the Shaw Prize, the Lasker Award, and in 2015, the Vilcek Prize.

http://walterlab.ucsf.edu/

The Bcl-2 Family: An Achilles' Heel for Cancer

Suzanne Cory, one of Australia's most distinguished molecular biologists, has had a major impact on the understanding of immunology and the development of cancer. Her current focus is on how the opposing factions of Bcl-2 family arbitrate cellular life or death. This knowledge is leading to the development of more effective therapeutics for cancer and degenerative diseases.

Suzanne was elected a Fellow of the Australian Academy of Science in 1986, a Fellow of the Royal Society in 1992, a Foreign Member of the US National Academy of Sciences in 1997, a Foreign Member of the American Academy of Arts and Sciences in 2001, an Associate Foreign Member of the French Academy of Sciences in 2002, an Academician of the Pontifical Academy of Sciences in 2004, and an Associate Member of the European Molecular Biology Organization in 2007. Her scientific achievements have attracted numerous honours and awards, including the Burnet Medal of the Australian Academy of Science in 1997, the Australia Prize (joint recipient) in 1998, the Charles S. Mott Prize (joint recipient) of the General Motors Cancer Research Foundation in 1998, a L'Oréal-UNESCO Women in Science Award in 2001, the Royal Medal of The Royal Society in 2002, and the Pearl Meister Greengard Prize in 2009. In 1999, she was appointed Companion in the General Division of the Order of Australia, and in 2009, she was awarded the French decoration of Chevalier de l'Ordre de la Légion d'Honneur. In 2010, Suzanne became the first woman to be elected President of the Australian Academy of Science.

http://www.wehi.edu.au/faculty_members/professor_suzanne_cory

Suzanne Cory, PhD

Honorary Distinguished Professorial Fellow in the Molecular Genetics of Cancer Division at The Walter and Eliza Hall Institute

Vice-Chancellor's Fellow of The University of Melbourne

cory@wehi.edu.au

Andreas Strasser, PhD

Alan W Harris Personal Chair
in Experimental Cancer Biology,
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How Does the Tumor Suppressor p53 Protect Us from Getting Cancer?

Andreas Strasser is a world leader in cancer and immunology, with a particular focus on the role of programmed cell death (apoptosis). By exploiting mouse genetics, he was the first to demonstrate that abnormalities in the control of apoptosis can cause autoimmune disease and amongst the first to show that this can cause cancer and render tumor cells refractory to anti-cancer therapy. These discoveries have major biological implications and suggest novel therapeutic strategies for cancer, autoimmunity, and degenerative diseases. Current research interests include identification of the signalling pathways that mediate developmentally programmed cell death in mammals and those that are responsible for chemotherapy-induced killing of cancer cells, with the goal to develop improved strategies for treatment of cancer and autoimmune diseases.

Andreas earned his PhD at Basel Institute for Immunology under Fritz Meichers in 1988 and moved for postdoctoral studies under Suzanne Cory to the Walter and Eliza Hall Institute in 1989, where he has remained ever since. He has been a Senior, Principal, then Senior Principal Research Fellow, then Australia Fellow of the National Health and Medical Research Council. Since 2006, he has been the Joint Division Head of the Molecular Genetics of Cancer Division at Walter and Eliza Hall Institute. He has published more than 240 primary research papers as well as a further 100+ review articles, and has received more than 35,000 citations overall. He has been recognized with the Burnet Prize, the Josef Steiner Cancer Research Prize, the Friedrich Miescher Prize from the Swiss Society for Biochemistry, shared the Glaxo Wellcome Australia Prize with Professor David Vaux, and the Victoria Prize in 2011. In 2003, he was elected to the Australian Academy of Science, and in 2009, became a foreign associate member of the European Molecular Biology Organisation.

http://www.wehi.edu.au/faculty_members/professor_andreas_strasser

MiRNA Control at the Level of a Single, Ubiquitously Expressed Target Gene

Klaus Rajewsky and his collaborators developed a general method of targeted mutagenesis in mouse embryonic stem cells by introducing bacteriophage- and yeast-derived recombination systems, which opened the way for conditional gene targeting. Using this and other novel approaches in their immunological work, they developed, together with NA Mitchison and NK Jerne, the antigen-bridge model of T-B cell cooperation, identified the B-cell antigen receptor as a survival determinant of B cells, and characterized germinal centers as the sites of antibody somatic hypermutation and memory cell formation as well as the major source of human B-cell lymphomas. The latter work included the identification of Hodgkin lymphoma as a germinal center-derived tumor. Over the last few years, the work of his group has focused on mechanisms of microRNA control and the development of mouse models of human B-cell lymphomas.

After postdoctoral work at the Institut Pasteur in Paris, Klaus Rajewsky built an immunology department at the Institute for Genetics at the University of Cologne, where he stayed for 38 years, with a part-time appointment as founding Program Coordinator of the EMBL Mouse Biology Program at Monterotondo near Rome in the later years. He then worked for 10 years at Harvard Medical School in Boston, USA, and is now at The Max Delbrück Center for Molecular Medicine in Berlin, Germany.

https://www.mdc-berlin.de/36143067/en/research/research_teams/immune_regulation_and_cancer

Klaus Rajewsky, MD

Head of the Group,
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Abstract Number 1

Sagar Bhogaraju, PhD

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Studying the Role of Ubiquitin System in the Eukaryotic Cilium

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Cilium is a hair-like structure that is present on most eukaryotic cells. Apart from serving the motility-related functions, the microtubule-based organelle also functions as cell's antenna. Intraflagellar transport (IFT) builds and maintains the cilium. Vesicles positive for IFT proteins transport ciliary-bound axonemal components and membrane proteins from the cytoplasm to the base of the cilium[1]. Microtubule plus end-directed motor Kinesin-II powers IFT particles along with the cargo to the tip of the cilium[2]. Transport of axonemal components and selective membrane proteins from the cilium back into the cytoplasm is critical for regulating the ciliary length and signaling, respectively.[3,4] Here, we hypothesize that ubiquitination might serve as a marker for ciliary components that are bound for removal from the cilium. We isolated wildtype *Chlamydomonas* cilia and performed di-gly proteomics to identify the ubiquitinated proteins in the cilium. Among the ubiquitinated proteins, are axonemal components such as Radial spoke proteins -2, -3, -9, Dynein heavy chain 11, and Central pair associated protein PF16. Surprisingly, ubiquitination sites were also found in the cytoplasmic domains of many ciliary membrane proteins. These include transient receptor potential ion channel 1, an ABC transporter, a Na^+/Ca^{2+} exchanger and Ryanodine inositol triphosphate receptor Ca^{2+} channel. Similar to sorting of plasma membrane proteins into endosomes, we currently hypothesize ubiquitination based internalization and transport of these ciliary membrane proteins into the cytoplasm. Furthermore, we are also characterizing a few candidate ubiquitin ligases and ubiquitin receptor molecules in the cilium that are potentially involved in the transport process.

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Abstract Number 2

Regulatory Control of Cranial Skeletal Muscle Stem Cells: Implications in Development and Disease

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Head muscles are essential for eye movements, mastication, facial expression as well as pharyngeal and laryngeal function. Despite the fact that all skeletal muscles throughout the body are composed of striated fibers, recent reports have uncovered an unexpected heterogeneity between head and trunk muscles with respect to their embryological origins, transcriptional programs, and regenerative capacity. Muscle heterogeneity is highlighted further by the variable sensitivity of specific subsets of muscles to genetic mutations that give rise to muscular dystrophies. In this context, extraocular muscles (EOM) are of particular interest, as they are spared in Duchenne muscular dystrophy.

Here, we exploit a combination of mouse genetic, genome-wide and 3D-imaging approaches to address key unanswered questions in skeletal muscle biology, using EOM/cranial muscle development and adult tissue homeostasis as focal points. From a developmental perspective, it is currently unknown how the correct arrangement of six EOM around the eyeball, which is essential for the coordinated movements of the eyes, is established. From a stem cell biology perspective, it is unclear whether specific traits of EOM myogenic stem cells could determine the preferential sparing of this muscle group in muscular dystrophy. As such, this project will help understand the unexpected biological variation among cranial skeletal muscles groups and their resident stem cells.

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Abstract Number 3

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Analysis of Metabolic Functions of p53 in Vivo

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The p53 protein is essential for the implementation of the cellular stress response during challenging environmental conditions. Although typically conceptualized within the framework of tumour suppression, p53 evolved in simple organisms that did not require tumour surveillance per se, but rather a robust program to help tolerate all manner of stochastic stress and to maintain genome integrity. As part of this general remit, the ability of p53 to regulate metabolism is a powerful feature of p53 biology. During starvation, for example, p53 activity can augment cell survival pathways, inhibit unnecessary growth, and promote efficient nutrient generation, utilisation, and conservation to support cell survival. We are exploring the metabolic functions of p53 during the physiological response to nutrient stress in vivo. We have generated experimental cohorts of genetically engineered mice (GEMs) that harbour tissue-specific deletion of p53 within metabolic compartments. Using these in vivo models, we are characterizing the role of p53 during metabolic stress. This work will help to expand our knowledge of p53 beyond canonical tumour-suppression into the important regime of metabolic syndromes such as obesity and diabetes. In addition, our findings will help to clarify emerging evidence that links polymorphisms within p53 to altered risks of diabetes and other metabolic diseases in humans.

Abstract Number 4

Targeting Protein Tyrosine Phosphatase 1B in Rett Syndrome

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The X-linked neurological disorder Rett syndrome (RTT) presents with autistic features and is caused primarily by mutations in a transcriptional regulator, methyl CpG-binding protein 2 (MECP2). Current treatment options for RTT are limited to alleviating some neurological symptoms; hence, more effective therapeutic strategies are needed. We identified the protein tyrosine phosphatase PTP1B as a therapeutic candidate for treatment of RTT. We demonstrated that the PTPN1 gene, which encodes PTP1B, was a target of MECP2 and that disruption of MECP2 function was associated with increased levels of PTP1B. Pharmacological inhibition of PTP1B ameliorated the effects of MECP2 disruption in mouse models of RTT, including improved survival in young male (Mecp2-/y) mice and improved behavior in female heterozygous (Mecp2-/+) mice. We demonstrated that PTP1B was a negative regulator of tyrosine phosphorylation of the tyrosine kinase TRKB, the receptor for brain-derived neurotrophic factor (BDNF). Therefore, the elevated PTP1B that accompanies disruption of MECP2 function in RTT represents a barrier to BDNF signaling. Inhibition of PTP1B led to increased BDNF signaling through TRKB. This study presents PTP1B as a mechanism-based therapeutic target for RTT, validating a unique strategy for treating the disease by modifying signal transduction pathways with small-molecule drugs. This study has implications for other neurological disorders that feature obesity and metabolic dysregulation. Furthermore it raises an exciting possibility that there might be other protein tyrosine phosphatases that might regulate critical signaling events in RTT and other neurological disorders, understanding of which could lead to new therapeutic strategies.

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Abstract Number 5

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The Biophysical Characterization of α-Synuclein

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Neuronal death in the brains of patients with Parkinson's disease has been connected with various causes, most of which are associated with the aggregation of α-synuclein (α-syn) into amyloid structures. A small presynaptic protein, α-syn, is one of the major components of Lewy bodies found in the neurons of patients with Parkinson's disease (PD). Oligomeric forms of α-syn appear to be the key factor in the pathogenesis of PD due to their toxicity. The detailed mechanism of α-syn aggregation and the nature of the toxic species, however, remains unknown. In this study, we followed the early events in α-syn aggregation by gel electrophoresis and by fluorescent correlation spectroscopy (FCS). We observed dimers and tetramers of α-syn by sensitive gel imaging based on fluorescence detection and found differences among early species by fluorescence correlation spectroscopy and mass spectrometry. Moreover, we tracked the accumulation of early oligomeric species in time and their further conversion into bigger aggregates, which provided a deeper understanding of the aggregation process. Next to it, we followed the growth of α-syn aggregates using real-time total internal reflection microscopy. Our results show that the morphology of aggregates depends on the conditions applied during the experiments. Extended three-dimensional structures composed of micrometer-long α-syn fibrils are formed on glass surfaces, but on supported lipid bilayers (SLBs) and in solution we found only the growth of linear amyloid fibrils. These two distinct types of aggregate strongly suggest a significant effect of surface properties on the growth and morphology of α-syn aggregates.

Abstract Number 6

Quantifying Early Events on Mitochondria in the PINK1-PARKIN Ubiquitin Ligase Signaling Cascade

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Recent work indicates that a major role for PINK1 and PARKIN, two proteins mutated in Parkinson's disease, is in mitochondrial outer membrane (MOM) ubiquitylation, which promotes mitophagy. We have found that PARKIN can build K6, K11, K48, and K63 ubiquitin (Ub) chains on dozens of MOM proteins in response to mitochondrial depolarization.[1,2] The ability of PARKIN to build these chains is greatly stimulated by PINK1-dependent phosphorylation of not only PARKIN, but also Ub itself on S65. A critical feature of the system is that phosphorylation of Ub chains by PINK1 as they are assembled by PARKIN leads to further recruitment of PARKIN through its phospho-Ub binding site.[1,3] To measure chain assembly activity and kinetics, we previously developed a quantitative mass spectrometry-based assay that measures total Ub, each individual linkage type as well as p-S65-Ub.[3] We are now building assays that include over 100 peptides representative of 21 proteins to examine primary ubiquitylation and phosphorylation sites on MOM proteins. The ability to accurately monitor PARKIN activity at the primary substrate level is critical for multiple purposes, including: a) measuring PINK1-PARKIN pathway activity precisely in cells, b) measuring the effect of small molecule activators of the pathway on mitochondrial ubiquitylation, and c) examining whether there is selectivity in PARKIN substrate targeting in various experimental settings. I will show we can now quantitatively monitor ubiquitylation of primary ubiquitylation sites in a cohort of substrates simultaneously using this novel assay and advance more generally our understanding of ubiquitin signaling cascades.

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Abstract Number 7

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A New Level of Metabolic Enzyme Organization: The Purinosome

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Classical regulatory mechanisms associated with enzyme activity have provided initial insight into the ways metabolism is regulated to promote cellular homeostasis and proliferation. Under high purine demand, the cell employs another regulatory strategy—enzyme clustering. Conditions that impact purine biosynthesis were shown to modulate the spatial organization of enzymes within the *de novo* purine biosynthetic pathway and affect the overall flux of metabolites through the pathway. This new level of enzyme organization within the *de novo* purine biosynthetic pathway is referred to as a purinosome. The biochemical mechanisms of purinosome formation and subcellular localization are starting to be elucidated. Current investigations into two biochemical mechanisms will be presented. First, purinosomes were demonstrated to colocalize with Hsp90 chaperone machinery, and Hsp90 inhibition resulted in a dissociation of purinosomes. Moreover, a synergistic effect was observed between the disruption of the purinosome by Hsp90 inhibition and methotrexate, an anti-folate chemotherapeutic agent. Current studies are exploring how Hsp90 is directly involved in purinosome complex assembly. The second investigation that will be presented looks into the organization of purinosomes within a cell by super-resolution fluorescence microscopy. These studies led to the discovery of purinosome colocalization with mitochondria. This colocalization has been attributed to the mTOR signaling pathway and further supports the influence mTOR has on nucleotide biosynthesis.

Abstract Number 8

From Ancient Lipids to Synthetic Life

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The function of the cell membrane as a barrier and a matrix for biochemical activity relies on the properties imparted by lipids. In eukaryotes, sterols are crucial for modulating the molecular order of membranes. Sterol ordering provides the basis for membrane lateral segregation and promotes a fluid, mechanically robust plasma membrane. How do organisms that lack sterols determine membrane order? Hopanoids are bacterial membrane lipids that have been demonstrated to have sterol-like properties in vitro. We now explore the distribution of hopanoids and their effect on membranes in *Methylobacterium extorquens*. We find that hopanoids determine bacterial outer membrane order in a manner analogous to sterol ordering in the eukaryotic plasma membrane, and that their deletion impairs energy-dependent multidrug efflux.

Building on this work, I will be starting a new group for synthetic membrane biology in Dresden at the B Cube Center for Biotechnology. The group will be broadly focused on understanding how lipids contribute to membrane function and organismal fitness from synthetic protocells to microorganisms. We are interested in topics ranging from how membranes contributed to the origin of cellular life, to identifying membrane-based targets for antibiotic resistance. One of our central aims will be to mine a repertoire of biological innovations from the simplest microorganisms to elucidate the minimal set of principles and components required to assemble a robust and responsive synthetic cell membrane.

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Abstract Number 9

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HPV Oncoproteins Target Cancer-Associated Cellular Pathways

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Human papillomaviruses (HPV) reprogram host cells to promote virus replication and limit virus detection by the host immune response. Infection with a few of the approximately 200 HPVs can result in cellular transformation and cancer, but most HPV infection is benign. To understand the mechanisms by which some HPVs cause cancer, we have performed systematic analyses of HPV–host interactions. We have defined interactions between virus-encoded proteins and host cellular proteins and profiled gene expression in cells that produce one or more HPV proteins.

We find diverse mechanisms by which HPVs alter host cellular processes. Many of the cellular targets of different HPV E7 oncoproteins are conserved across virus types, while fewer targets interact with specific E7 proteins. In contrast, HPV E6 oncoproteins engage in virus genus-, species-, and type-specific interactions with cellular proteins. Cellular gene expression patterns change upon introduction of different E6 proteins into cells in ways that are consistent with these protein–protein interactions.

Based on these systematic studies, we have chosen novel cellular pathways targeted by cancer-associated HPVs for further analysis. Oncogenic HPV E7 proteins target the cellular protein PTPN14 for proteasome-mediated degradation. PTPN14 is a putative tumor suppressor and negative regulator of YAP in the Hippo signaling pathway. Its degradation may account for the retinoblastoma-independent transforming activity of oncogenic HPV E7, an activity that has long been proposed by the HPV field but whose mechanism has not been defined. Further study of PTPN14 and other HPV targets will identify mechanisms of cellular transformation and virus replication.

Abstract Number 10

Physiological and Pathological Functions of Alpha-Synuclein in a Mammalian Cell Model

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The intrinsically disordered protein alpha-synuclein is genetically and pathologically linked to Parkinson's disease. It is the major constituent of Lewy bodies, the pathological hallmark of Parkinson's, and single-point mutations or changes in expression levels of alpha-synuclein often lead to early onset or sporadic forms of the disease. In patients with Parkinson's disease, it is predicted that the physiological role of alpha-synuclein is disrupted. However, despite intensive research, the normal function of alpha-synuclein in healthy neurons remains elusive. Utilizing RBL-2H3 mast cells as a mammalian model system, we investigated the capacity of alpha-synuclein to independently regulate stimulated exocytosis and endocytosis. Exocytosis was stimulated by the multivalent antigen DNP-BSA or the SERCA inhibitor thapsigargin and monitored using the recycling endosomal marker VAMP8-pHluorin. Here we demonstrate that low expression levels of alpha-synuclein inhibit exocytosis. Inhibition occurs with both WT and mutant alpha-synuclein, and with stimulation by either DNP-BSA or thapsigargin, revealing inhibition downstream of Ca^{2+} mobilization. Alpha-synuclein loses the capacity to inhibit exocytosis when expressed at higher levels, suggesting a potential role for an aggregation-mediated loss of function. Conversely, we find that stimulated endocytosis of fluorescently labeled immunoglobulin E is inhibited when alpha-synuclein is expressed at the higher levels (but not at low levels) in RBL-2H3 cells. Expression of alpha-synuclein at low levels in PC-12 cells, a dopamine-releasing cell line, also inhibits stimulated exocytosis. Together, these findings implicate alpha-synuclein as a regulator of stimulated endosomal trafficking that depends on expression levels within cells.

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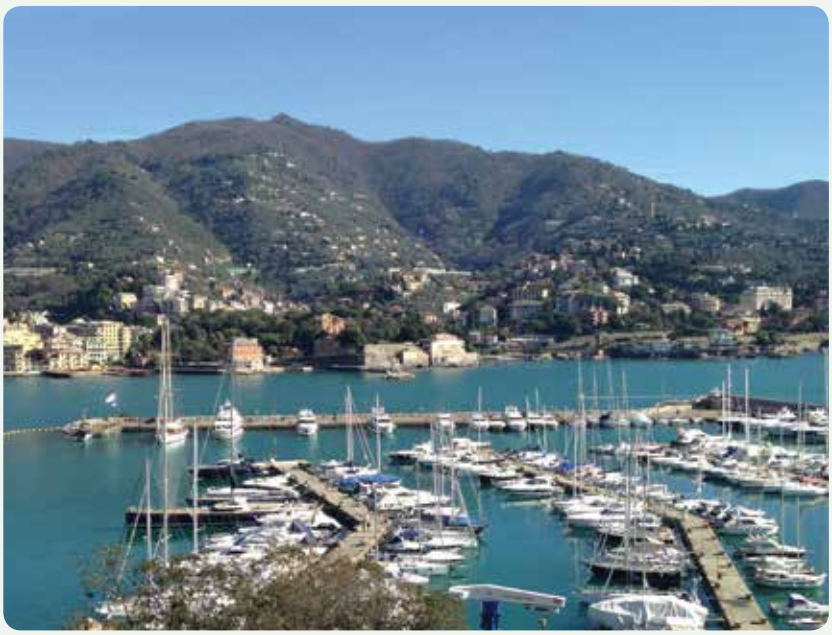

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