BIOGRAPHICAL SKETCH

Tobias Meyer

Mrs. George A. Winzer Professor in Cell Biology
Professor of Chemical and Systems Biology
Stanford University

Education

University of Basel, Basel Switzerland	Vordiplom	1980	Physics, Math, Biology
University of Basel/CERN Geneva, Switzerland	Masters	1983	Experimental Physics
Biocenter of the University of Basel	Ph.D.	1986	Biophysical Chemistry

A. Personal Statement

I seek to understand how mammalian cells process information and make decisions. This is a fundamental open question as cells are controlled by multiple signaling pathways with tens of signaling proteins, second messengers and chromatin modifiers connected to each other on time-scales of seconds to days by positive and negative feedbacks. Understanding how signaling circuits control cell proliferation, migration and other outputs is important for identifying optimal drug targets and to facilitate the development of combination therapies. Much of my work is built on the premise that genetic and biochemical methods can be used to identify and characterize components of signaling circuits, but that single-cell microscopy, live-cell signaling reporters, and rapid perturbations are needed to understand the design principles of signaling circuits. My laboratory has pioneered the development and use of molecular tools and quantitative microscopy methods to understand feedback-connected signaling circuits and made key contributions to our understanding of the spatial and temporal control of calcium, lipid second messenger, small GTPase, and protein kinase signaling processes. Our current research identifies general control principles and specific mechanisms how cells integrate receptor, cell contact and stress inputs to decide between quiescence, proliferation and senescence, how they switch metabolic states, and how they trigger polarization and decide to move. We are investigating these signaling circuits by combining high-resolution live-cell analysis of signal transduction and local chromatin activity with optogenetic perturbations, single-cell RNAseq and computational modeling.

The following papers exemplify my thinking and research strategy:

- Cappell SD, Mark KG, Garbett D, Pack LR, Rape M, Meyer T. 2018. EMI1 switches from being a substrate to an inhibitor of APC/C^{CDH1} to start the cell cycle. **Nature** 558, 313-317.
- Gu B, Swigut T, Spencley A, Bauer MR, Chung M, Meyer T, Wysocka J. 2018. Transcription-coupled changes in nuclear mobility of mammalian cis-regulatory elements. **Science** 359, 1050-1055.
- Yang HW, Chung M, Kudo T, Meyer T. 2017. Competing memories of mitogen and p53 signalling control cell-cycle entry. **Nature** 549, 404-408.
- Yang HW, Collins SR, Meyer T. 2016. Locally excitable Cdc42 signals steer cells during chemotaxis. **Nat Cell Biol.** 18, 191-201.
- Cappell, SD, Chung M, Jaimovich A, Spencer SL, Meyer T. 2016. Irreversible APC^{Cdh1} inactivation underlies the point of no return for cell cycle entry. **Cell** 166, 167-80.
- Tsai FC, Seki A, Yang HW, Hayer A, Carrasco S, Malmersjö S, Meyer T. 2014. A polarized Ca2+, diacylglycerol and STIM1 signalling system regulates directed cell migration. **Nat Cell Biol**. 16, 133-44.
- Spencer SL, Cappell SD, Tsai FC, Overton KW, Wang CL, Meyer T. 2013. The proliferation-quiescence decision is controlled by a bifurcation in CDK2 activity at mitotic exit. **Cell** 155, 369-383.

B. Positions and Honors

1981-1983 Masters (Diploma) thesis research, University of Basel. My thesis work was part of a group effort to collect and analyze data from a particle detector to test whether a novel four-quark state may exist. My main contribution was the development of a Monte Carlo method to simulate particle showers in a new detector at CERN, Geneva.

1983-1986 Ph.D. thesis research with Professor H.G. Schindler, Department of Biophysical Chemistry, Biocenter, Basel. My thesis work was the design, construction, and application of a laser scanning fluorescence correlation fluctuation microscope (FCS) to measure protein-protein binding interactions and vesicle fusion.

1987-1991 Postdoctoral Fellow with Professor Lubert Stryer, Department of Cell Biology, Stanford University, supported through Fellowships from the Swiss National Science Foundation. I discovered in my postdoctoral work a non-linear control of IP3-gated Ca2+ release which allowed me to develop the first mathematical model of Ca2+ oscillations. Using biochemical, spectroscopy and modeling approaches, I further showed how the neuronal "memory protein" CaMKII is regulated by Ca2+-triggered autophosphorylation which dramatically increase its calmodulin binding affinity.

1991-1999 Assistant and Associate Professor, Departments of Cell Biology and of Pharmacology and Cancer Biology, Duke University Medical Center, Durham, NC. My laboratory developed quantitative live-cell microscopy approaches and analysis methods to understand the local induction of Ca2+ and lipid second messenger signals and the resulting activation of CaMKII and protein kinase C.

1992-1997 Packard Fellow for Science and Engineering, David and Lucile Packard Foundation

2000-2005 Associate Professor, Department of Molecular Pharmacology, Stanford University. My work focused on developing and applying quantitative methods to explore the logic of mammalian signaling circuits, particularly by understanding how small GTPases, protein kinases and second messengers regulate the actin cytoskeleton, cell polarization and directed cell migration.

2001-2008 Director, Alliance for Cell Signaling Microscopy laboratory (NIGMS), California Avenue, Palo Alto

2006 - Professor, Department of Chemical and Systems Biology; Stanford University. My work seeks to understand design principles of cell decision processes by focusing on mammalian cell-cycle entry and exit control as well as the decision to polarize and migrate.

2006-2011 Vice-Chair, Department of Chemical and Systems Biology, Stanford University

2009 - Mrs. George A. Winzer Professorship in Cell Biology, Stanford University

2011-2016 Chair, Department of Chemical and Systems Biology, Stanford University

2013-2018 Co-Director, Stanford Center for Systems Biology (NIGMS), Stanford University

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C. Contributions to Science

My laboratory has made notable contributions to the mammalian cell signaling field. I am a primary author or co-author on 156 articles (Google Scholar) with 28 of them being contributions to *Nature*, *Science* or *Cell*. My h-index is 78 (78 papers with over 78 citations). My work has been cited over 24,300 times with over 8,400 citations since 2013. A detailed Bibliography is available through Google Scholar or at my website. Many of the tools that we invented or developed have been critical for the work of other groups (we have made over 1000 shipments of biosensors and other tools either directly or through Addgene). My most significant discoveries have been spatial and temporal control mechanisms of signaling processes driven by calcium, lipid second messengers, small GTPases and protein kinases as well as circuit design principles how cells control oscillations and how they make decisions to enter or exit the cell-cycle entry and how they decide to polarize, migrate and chemotax.

(1) We seek to understand how cells integrate information to decide if and when to transition between the cell cycle, quiescence and senescence.

We are developing live-cell reporters, perturbation technologies and single-cell automated analysis methods to understand critical timing, feedbacks and signaling steps of cell-cycle entry and exit decision processes. Recent discoveries were the identification of APC/C^{Cdh1} inactivation as the point-of-no-return for cell cycle entry (Cappell 2016), a new ATR-regulated checkpoint for the S/G2 transition (Saldivar 2018), and an ATR-CDK2 feedback mechanism that controls the speed of origin firing and progression through S-phase (Daigh 2018). We also co-discovered a Cyclin B nuclear translocation-mediated control mechanism for the rapid triggering of mitosis (Santos, 2012) and a spatial control mechanism based on polarized Wnt signaling for asymmetric cell division (Habib, 2014). Most importantly, we uncovered that G1 phase starts with a stoichiometric nuclear competition between Cyclin D and CDK inhibitors that reflects the memory of competing mitogen and stress signal inputs (Chen 2013; Spencer 2013; Yang 2017), and we discovered a critical decision process whereby an inhibitor of the APC/C^{Cdh1} E3 ligase Emi1 can at the same time be its substrate to generate an irreversible switch mechanism that commits cells to the cell cycle (Cappel 2018). Our ongoing work builds on these findings and focuses on the question how cells decide to trigger contact inhibition, quiescence, senescence, proliferation as well as changes in metabolic state. We are in a unique position to pursue this work in part by using an unpublished reporter for CDK4/6 activity that we recently developed, high resolution microscopy methods that we co-developed to understand dynamic changes of local chromatin mobility and gene expression (Bo, 2018), as well as methods to combine live-cell microscopy with single-cell RNAseq sequencing (Lane 2017).

- Cappell SD, Mark KG, Garbett D, Pack LR, Rape M, Meyer T. 2018. EMI1 switches from being a substrate to an inhibitor of APC/C^{CDH1} to start the cell cycle. **Nature** 558, 313-317.
- Saldivar JC, Hamperl S, Bocek MJ, Chung M, Bass TE, Cisneros-Soberanis F, Samejima K, Xie L, Paulson JR, Earnshaw WC, Cortez D, Meyer T, Cimprich KA. 2018. An intrinsic S/G2 checkpoint enforced by ATR. **Science** 361, 806-810.
- Daigh LH, Liu C, Chung M, Cimprich KA, Meyer T. 2018. Stochastic endogenous replication stress causes ATR-rriggered fluctuations in CDK2 activity that dynamically adjust the global DNA synthesis rate. **Cell Systems** 7, 17-27.
- Gu B, Swigut T, Spencley A, Bauer MR, Chung M, Meyer T, Wysocka J. 2018. Transcription-coupled changes in nuclear mobility of mammalian cis-regulatory elements. **Science** 359, 1050-1055.
- Yang HW, Chung M, Kudo T, Meyer T. 2017. Competing memories of mitogen and p53 signalling control cell-cycle entry. **Nature** 549, 404-408.
- Lane K, Van Valen D, DeFelice MM, Macklin DN, Kudo T, Jaimovich A, Carr A, Meyer T, Pe'er D, Boutet SC, Covert MW. 2017. Measuring signaling and RNA-Seq in the same cell links gene expression to dynamic patterns of NF-κB activation. **Cell Systems** 4, 458-469.e5.
- Cappell, SD, Chung M, Jaimovich A, Spencer SL, Meyer T. 2016. Irreversible APC^{Cdh1} inactivation underlies the point of no return for cell cycle entry. **Cell** 166, 167-80.
- Spencer SL, Cappell SD, Tsai FC, Overton KW, Wang CL, Meyer T. 2013. The proliferation-quiescence decision is controlled by a bifurcation in CDK2 activity at mitotic exit. **Cell** 155, 369-383.
- Habib SJ, Chen BC, Tsai FC, Anastassiadis K, Meyer T, Betzig E, Nusse R. 2013. A localized Wnt signal orients asymmetric stem cell division in vitro. **Science** 339, 1445-8.
- Chen JY, Lin JR, Tsai FC, Meyer T. 2013. Dosage of Dyrk1a shifts cells within a p21-cyclin D1 signaling map to control the decision to enter the cell cycle. **Mol Cell** 52, 87-100.
- Santos SD, Wollman R, Meyer T, Ferrell JE Jr. 2012. Spatial positive feedback at the onset of mitosis. **Cell** 49, 1500-13.

(2) We seek to understand how cells integrate signals to decide to polarize and migrate and how contact inhibition stops cell migration and proliferation.

Our current work builds on our previous findings that PIP2 lipids in the inner leaflet of the plasma membrane mediate cortical actin-membrane adhesion (Raucher 2000) and that PIP2 lipids also selective target many small GTPases and other signaling proteins to the plasma membrane (Heo 2006). In addition to these central roles of PIP2, we also showed that local changes in membrane curvature are critical for regulating membrane retraction and protrusions (Galic 2012, 2014). In earlier work, we co-discovered that cell polarization and directed migration of cells is promoted by gradients in PIP3 lipids (Haugh 2000) and we identified specific roles of local Ca2+ (Tsai 2012), CDC42 signals (Yang 2016), and diacylglycerol signals (Tsai 2014) in controlling directed migration. We also discovered a new engulfed membrane-actin structure that we termed cadherin fingers which we found to be critical for polarization and collective coordination of endothelial cells (Hayer 2016), and we identified a small GTPase and actin-mediated mechanism that mediates symmetry braking during neuron polarization (Fivaz 2008; Winans 2016). Finally, we developed mathematical models demonstrating that cells need at least two positive feedbacks to achieve stable spatial polarity and robust cell fate decisions (Brandman 2008) and we developed the first model showing that stochastic sensing at the front of already polarized cells is sufficient to generate chemotaxis (Arrieumerlou, 2005). In this model, gradients of chemoattractant are read at the front of cells as left-versus right steering decisions that create a biased random walk towards chemoattractant.

Much of our work on cell polarity and migration is built on tools that we developed to measure dynamic changes in lipid second messenger and receptor signaling in cells. We discovered that GFP conjugated pleckstrin homology (PH)-domain fusion constructs can be used to monitor local changes in PIP2 lipids in cells (PH-PLCdelta) (Stauffer 1998), developed GFP-conjugated PH-Akt domains as tools to monitor local PIP3 lipid signals (Kontos 1998), C1 domain-based constructs to monitor local diacylglycerol signals (Oancea 1998) and GFP-tagged SH2 domains to monitor local receptor activation (Stauffer 1997). We also developed a method to rapidly activate and inactivate small GTPases and change lipid second messenger levels in the plasma membrane using small molecule triggered dimerization and translocation (Suh 2006; Inoue 2005). We further showed that a variant of GFP can be used for local photoactivation that allows one to measure binding affinities, diffusion and movement of proteins in living cells (our study Yokoe 1996 reported light-induced local photo-conversion of a UV-excitable to a blue-excitable GFP). This finding motivated the development by others of engineered photoactivatable GFP that in turn provided a path to super-resolution microscopy. Our current work investigates an unexpected critical role of cortical actin that we discovered in regulation the polarization decisions for cell migration and focuses on the molecular basis for contact inhibition of cell movement and proliferation. Our strategy is in part based on an unpublished reporter that we developed that can monitor changes in the

strength of the local actin cortex as well as on the use of parallel CRISPR knockout and endogenous tagging strategies combined with high-resolution live-cell analysis to understand the local signaling processes that mediate contact inhibition of cell migration and proliferation.

- Winans AM, Collins SR, Meyer T. 2016. Waves of actin and microtubule polymerization drive microtubule-based transport and neurite growth before single axon formation. **Elife**. e12387.
- Yang HW, Collins SR, Meyer T. 2016. Locally excitable Cdc42 signals steer cells during chemotaxis. **Nat Cell Biol.** 18, 191-201.
- Hayer A, Shao L, Chung M, Jouber LM, Yang HW, Tsai FC, Bisaria A, Betzig E, Meyer T. 2016. Engulfed cadherin fingers are polarized junctional structures between collectively migrating endothelial cells. **Nat Cell Biol.** 18, 1311-1323.
- Galic M, Tsai FC, Collins SR, Matis M, Bandara S, Meyer T. 2014. Dynamic recruitment of the curvature-sensitive protein ArhGAP44 to nanoscale membrane deformations limits exploratory filopodia initiation in neurons. **Elife**. 3:e03116.
- Galic M, Jeong S, Tsai FC, Joubert LM, Wu YI, Hahn KM, Cui Y, Meyer T. 2012. External push and internal pull forces recruit curvature-sensing N-BAR domain proteins to the plasma membrane. **Nat Cell Biol.** 14, 874-81.
- Tsai FC, Meyer T. 2012. Ca2+ pulses control local cycles of lamellipodia retraction and adhesion along the front of migrating cells. **Curr Biol.** 2012 May 8;22(9):837-42.
- Brandman O, Meyer T. 2008. Feedback loops shape cellular signals in space and time. **Science**. 322, 390-5.
- Fivaz M, Bandara S, Inoue T, Meyer T. 2008. Robust neuronal symmetry breaking by Ras-triggered local positive feedback. **Curr Biol.** 18, 44-50.
- Heo WD, Inoue T, Park WS, Kim ML, Park BO, Wandless TJ, Meyer T. 2006. PI (3, 4, 5) P3 and PI (4, 5) P2 lipids target proteins with polybasic clusters to the plasma membrane. **Science** 314, 1458-1461.
- Suh BC, Inoue T, Meyer T, Hille B. 2006. Rapid chemically induced changes of PtdIns(4,5)P2 gate KCNQ ion channels. **Science** 314, 1454-7.
- Arrieumerlou C, Meyer T. 2005. A local coupling model and compass parameter for eukaryotic chemotaxis. **Dev Cell** 8, 215-227.
- Heo WD, Meyer T. 2003. Switch-of-function mutants based on morphology classification of Ras superfamily small GTPases. **Cell** 113, 315-28.
- Teruel MN, Meyer T. 2000. Translocation and reversible localization of signaling proteins: a dynamic future for signal transduction. **Cell** 103, 181-4.
- Haugh J, Codazzi F, Teruel M, Meyer T. 2000. Spatial sensing in fibroblasts mediated by 3′ phosphoinositides. **J Cell Biol.** 151, 1269-1280.
- Raucher D, Stauffer T, Chen T, Shen K, Guo S, York JD, Sheetz MP, Meyer T. 2000. Phosphatidylinositol 4, 5-bisphosphate functions as a second messenger that regulates cytoskeleton–plasma membrane adhesion. **Cell** 100, 221-228.
- Stauffer TP, Ahn S, Meyer T. 1998. Receptor-induced transient reduction in plasma membrane PtdIns (4, 5) P2 concentration monitored in living cells. **Curr Biol.** 8, 343-346.
- Kontos CD, Stauffer TP, Yang WP, York JD, Huang L, Blanar MA, Meyer T, Peters KG. 1998. Tyrosine 1101 of Tie2 is the major site of association of p85 and is required for activation of phosphatidylinositol 3-kinase and Akt. **Mol Cell Biol.** 18, 4131-40.
- Oancea E, Meyer T. 1998. Protein kinase C as a molecular machine for decoding calcium and diacylglycerol signals. **Cell** 95, 307-318.
- Stauffer TP, Meyer T. 1997. Compartmentalized IgE receptor-mediated signal transduction in living cells. **J Cell Biol.** 139, 1447-54.
- Yokoe H, Meyer T. 1996. Spatial dynamics of GFP-tagged proteins investigated by local fluorescence enhancement. **Nat Biotechnol**. 14, 1252-6.

(3) We showed how cells generate and use local Ca2+ pulses and global oscillations to regulate cell function.

My most cited paper is in the field of Ca2+ signaling where we performed a human siRNA screen and co-discovered the ER Ca2+ sensors STIM1 and STIM2 that regulate influx of Ca2+ into cells (Liou 2005). We discovered that STIM proteins control Ca2+-influx across the plasma membrane by translocating along the ER membrane to punctate ER-PM junctions in response to a drop of Ca2+ in the lumen of the ER (Liou 2005). We further discovered that the activation of STIM is mediated by rapid oligomerization of STIM proteins in the in the internal ER membrane after Ca2+ dissociates from an EF-hand binding site in the lumen of the ER (Liou 2007), and we demonstrated that the two human STIM genes differ in their activity and sensitivity to Ca2+ which allows STIM2 to function as a feedback regulator that maintains basal Ca2+ levels in the cytoplasm (Brandman 2007).

A second key contribution to the Ca2+ signaling field started with my finding as a postdoc of non-linear control of IP3-gated intracellular Ca2+ release (Meyer 1988a) which allowed me to develop the first mathematical model of Ca2+ oscillations (Meyer 1988b). We concluded that the rapid upstroke of a Ca2+ spike must be a bistable switch that combines a non-linear signaling step with positive feedback, and that frequency-regulation of Ca2+ oscillations requires an additional delayed negative feedback. We later showed that this same feedback architecture can generate local IP3-gated Ca2+ pulses (Horne 1997) and that two or more positive feedbacks are needed to make such a signaling switch robust (Brandman 2005). Finally, we performed live-cell experiments to understand the activation of two of the key targets of Ca2+ signals, protein kinase C and Ca2+/CaM-dependent protein kinase II. Our work revealed the existence of sequential molecular steps of translocation and activation of protein kinase C in response to combined increases of Ca2+ and diacylglycerol (Oancea 1998, Teruel 2002). Using in vitro and in vivo analysis, we further discovered that CaMKII, which is the most abundant neuronal signaling protein, is activated by sequential autophosphorylation, translocation and increased calmodulin binding affinity, which leads to a reduced need of Ca2+ to maintain a persistently activated "memory state" when CaMKII reaches the postsynaptic density (Meyer 1992; Shen 1999). Our main current projects focus on the role of Ca2+ as a co-regulator of cell migration.

- Tsai FC, Seki A, Yang HW, Hayer A, Carrasco S, Malmersjö S, Meyer T. 2014. A polarized Ca2+, diacylglycerol and STIM1 signalling system regulates directed cell migration. **Nat Cell Biol**. 16, 133-44.
- Wollman R, Meyer T. 2012. Coordinated oscillations in cortical actin and Ca2+ correlate with cycles of vesicle secretion. **Nat Cell Biol.** 14, 1261-9.
- Brandman O, Meyer T. 2008. Feedback loops shape cellular signals in space and time. **Science** 322, 390-5.
- Brandman O, Liou J, Park WS, Meyer T. 2007. STIM2 Is a Feedback Regulator that Stabilizes Basal Cytosolic and Endoplasmic Reticulum Ca2+ Levels. **Cell** 131, 1327-1339.
- Liou J, Fivaz M, Inoue T, Meyer T. 2007. Live-cell imaging reveals sequential oligomerization and local plasma membrane targeting of stromal interaction molecule 1 after Ca2+ store depletion. **Proc Natl Acad Sci U S A.** 104, 9301-6.
- Liou J, Kim ML, Heo WD, Jones JT, Myers JW, Ferrell JE Jr, Meyer T. 2005. STIM is a Ca2+ sensor essential for Ca2+-store-depletion-triggered Ca2+ influx. **Current Biology** 15, 1235-1241.
- Brandman O, Ferrell JE, Li R, Meyer T. 2005. Interlinked fast and slow positive feedback loops drive reliable cell decisions. **Science** 310, 496-498.
- Teruel MN, Meyer T. 2002. Parallel single-cell monitoring of receptor-triggered membrane translocation of a calcium-sensing protein module. **Science** 295, 1910-2.
- Shen K, Meyer T. 1999. Dynamic control of CaMKII translocation and localization in hippocampal neurons by NMDA receptor stimulation. **Science** 284, 162-167.
- Oancea E, Meyer T. 1998. Protein kinase C as a molecular machine for decoding calcium and diacylglycerol signals. **Cell** 95, 307-318.
- Horne JH, Meyer T. 1997. Elementary calcium-release units induced by inositol trisphosphate. **Science** 276, 1690-3.
- Subramanian K, Meyer T. 1997. Calcium-Induced Changes in the Structure of Endoplasmic Reticulum and Nuclear Envelope Calcium Stores. **Cell** 89, 963-971.
- Allbritton NL, Meyer T, Stryer L. 1992. Range of messenger action of calcium ion and inositol 1,4,5-trisphosphate. **Science** 258, 1812-5.
- Meyer T, Hanson PI, Stryer L, Schulman H. 1992. Calmodulin trapping by calcium-calmodulin-dependent protein kinase. **Science** 256, 1199-1202.
- Meyer T. 1991. Cell signaling by second messenger waves. **Cell** 64, 675-8.
- Meyer T, Stryer L. 1988b. Molecular model for receptor-stimulated calcium spiking. **Proc Natl Acad Sci U S A.** 85, 5051-5055.
- Meyer T, Holowka D, Stryer L. 1988a. Highly cooperative opening of calcium channels by inositol 1,4,5-trisphosphate. **Science** 240, 653-6.

D. Teaching and Training

19 researchers who trained in my laboratory currently hold faculty positions (listed below). I have also trained another 30 researchers who are currently postdocs, hold teaching positions, or are in research and management positions at Biotech companies. My laboratory typically trains 10-12 researchers with similar numbers of students and postdocs. I am also teaching at Stanford a yearly Cell Signaling class as well as Pharmacokinetics, Pharmacodynamics and Drug Metabolism for Medical Students. I also taught Medical Physiology and a Matlab image analysis class. I taught for two years a 2 week laboratory class at the Woods Hole Physiology Course.

Brandman, Onn Assistant Professor, Biochemistry department, Stanford University

Oancea, Elena	Associate Professor, Department of Medical Science, Brown University
Shen, Kang	Professor, HHMI, Biology department, Stanford University
Tsai, Feng-Chiao	Assistant Professor, Institute of Molecular Medicine, National Taiwan University
Arrieumerlou, Cecile	Professor, Cochin Institute, Paris, France
Cappell, Steven	Group Leader, Stadtman Investigator, NIH/NCI
Collins, Sean	Assistant Professor, Dept of Microbiology and Molecular Genetics, UC Davis
Galic, Milos	Group Leader, Medical Physics and Biophysics, University of Munster
Haugh, Jason M.	Professor, Chemical Engineering, NC State University, Raleigh, NC
Hayer, Arnold	Assistant Professor, Department of Biology, McGill University
Inoue, Takanari	Professor, Department of Cell Biology, Johns Hopkins University School of Medicine
Spencer, Sabrina	Assistant Professor, Dept of Chemistry and Biochemistry, UC Boulder
Tengholm, Anders	Professor, Dept. of Medical Cell Biology, Uppsala University, Sweden
Wollman, Roy	Professor, Chemistry and Biochemistry, UCLA
Xuecai Ge	Assistant Professor, School of Natural Sciences, UC Merced
Yang, Hee Won	Assistant Professor, Department of Pathology and Cell Biology, Columbia University
Bayer, Ulli	Professor, Department of Pharmacology, University of Colorado, Denver
Heo, Won Do	Professor, Department of Biological Sciences, KAIST, Korea
Liou, Jen	Associate Professor, Department of Physiology, UT Southwestern Medical Center

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E. Selected external activities and service

- I am a current member of the editorial board of JCB and past member of JBC and Science Signaling.
- I was a Chair and co-chair of the Gordon Research Conference on Directed Cell Migration, 2009, 2011; Chair and co-chair, of the FASEB conference on Calcium and Cell Function, 2000, 2002; as well as Co-Chair of the International Conference for Systems Biology, ICSB, 2009.
- I was a member of the scientific advisory boards at Senomyx, Blue Shift, and Molecular Devices.
- I participated ad hoc in R01, MIRA, SBRI and Instrumentation NIH review panels, on average approximately one review panel per year. I also served yearly on the review panel of the Swiss SystemsX program, 2004-2017, and bi-yearly on the Damon Runyon Cancer Research Foundation Fellowship Award Committee, 2013-2016. I also participated at the evaluation of the Pharmacology and Cancer Biology Department, Duke University, 2014, and of intramural NCI programs, 2018.

F. Recent and ongoing research support

P50 GM107615 (Co-PIs, Ferrell and Meyer) 09/30/2013 – 06/30/2018
National Institutes of Health
Systems Biology of Collective Cell Decisions
The center focused on deciphering the regulation of cell division, migration and differentiation, with applications to cancer biology, cell biology and developmental biology, respectively.

R01 GM063702, Meyer (PI) 07/01/14 - 06/30/19
Chemotactic Signal Transduction
This project investigated signaling processes that control polarization and directed migration

R01 GM030179, Meyer (PI) 09/01/14 - 08/31/19
Intracellular Calcium Signaling
The project investigated cellular roles of the intracellular Ca2+ signaling system.

R01GM118377, Meyer (PI) 04/01/16 - 03/31/20
Decision points to enter and exit the human cell cycle
The project investigated the signaling processes that control cell cycle commitment.

The 3 RO1 grants above got combined into a R35 GM127026 MIRA award:
Cell Signaling and Cell Decisions 07/01/18 - 06/30/23
