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Monographs Editors: J. Alan Diehl, Dale S. Haines, and Serge Y. Fuchs

Drs. J. Alan Diehl (left), Dale S. Haines (middle), and Serge Y. Fuchs (right).

J. Alan Diehl

Dr. Diehl obtained his PhD in biochemistry from the University of Missouri–Columbia, where his research focused on the characterization of avian and viral Rel/NF κ B proteins. Following completion of his PhD in 1995, he moved to St Jude Children's Research Hospital, where he performed postdoctoral research in the laboratory of Dr. Charles Sherr. In the laboratory of Dr. Sherr, he developed an interest in the intrinsic instability of the D-type cyclins. During his postdoctoral training, Dr. Diehl demonstrated that proteolytic turnover of cyclin D1 was coordinately regulated through temporal regulation of subcellular localization access to E3 ubiquitin ligases that in turn marked cyclin D1 for proteolysis. During his postdoctoral training, Dr. Diehl also nurtured an interest in the unfolded protein response pathway, following his discovery that D-type cyclin translation was subject to control during this cellular response.

After completing his postdoctoral training in 1999, Dr. Diehl maintained his interest in the posttranslational regulation of D-type cyclins. Work from the Diehl laboratory has provided direct evidence for loss of cyclin D1 ubiquitin-mediated degradation in human cancers. The intersection of cyclin D1 with human cancer occurs at multiple points, including mutations within cyclin D1 that inhibit phosphorylation-dependent ubiquitination as well as mutations that target the recently identified F-box protein, Fbxo4, that directs cyclin D1 ubiquitination. The ongoing efforts of the Diehl laboratory seek to provide insights into both the normal and pathophysiological role of Fbxo4 and deciphering the molecular mechanisms that underlie the neoplastic functions of D-type cyclin dependent kinases.

Dale S. Haines

Dr. Dale Haines received his PhD in molecular biology in 1992 from Hahnemann University, Philadelphia, under the guidance of Dr. David Gillespie. He

developed an interest in researching ubiquitin pathway components as a postdoctoral fellow in Dr. Donna George's laboratory at the University of Pennsylvania, where he investigated the interaction between the MDM2 oncoprotein/ubiquitin ligase and p53 tumor suppressor. In the early part of his independent career, Dale investigated p53 independent activities of MDM2 and correlated MDM2 expression levels with prognosis in childhood leukemia. Over the past few years his research program has taken a detour from the MDM2 field, and he has been studying mechanisms by which ubiquitination regulates the nuclear mobilization of two endoplasmic reticulum-localized transcription factors in budding yeast. The Haines laboratory has made a number of discoveries in this area, including the elucidation of a proteasome-independent role for ubiquitin and CDC48 in regulating protein complex disassembly. The AAA ATPase CDC48 (called p97 in metazoans) is emerging as an important player in a variety of ubiquitin signaling events and is targeted to specific substrates by a growing number of adaptor proteins. After recently completing a sabbatical in Dr. Raymond Deshaies's laboratory at the California Institute of Technology in the summer of 2010, Dale's future interest is to define the cellular, molecular, and biochemical activities of p97-containing complexes comprised of a subclass of adaptors that do not harbor known ubiquitin association domains. Dale has authored over 15 papers in the past 10 years pertinent to ubiquitin signaling, including those published in *Molecular Cell*, *Current Biology*, *Journal of Biological Chemistry*, and *Molecular and Cellular Biology*. He was a consulting editor for *Oncogene* from 2003–2008.

and has served on numerous NIH study sections and grant review boards for private foundations. He was born in Camden, New Jersey, and has lived in the Philadelphia area his entire life. He is currently a tenured associate professor in the Department of Biochemistry and a member of the Fels Institute for Cancer Biology at Temple University School of Medicine.

Serge Y. Fuchs

Dr. Fuchs obtained his doctor of medicine degree from the Yaroslavl State Medical School in Yaroslavl, USSR in 1987. After working for a few years as a physician in the Soviet Union, he entered a PhD program in experimental oncology at the Institute for Carcinogenesis, All-Russian Cancer Research Center of

Academy of Medical Science in Moscow, Russia. Upon obtaining his PhD degree in 1992, Dr. Fuchs undertook his postdoctoral research in a program directed by Dr. Ze'ev Ronai at the American Health Foundation (Valhalla, NY) and Ruttenberg Cancer Center at the Mount Sinai School of Medicine (New York, NY). His work on the ubiquitination-dependent regulation of cellular stress-responsive transcription factors (c-Jun, ATF2, p53) contributed to the understanding of the interplay between ubiquitination and phosphorylation in the regulation of signal transduction. Further work on the phosphorylation-dependent ubiquitination of I κ B and β -catenin led to identification of an F-box protein, β Trcp2 (then termed HOS) and SCF- β Trcp E3 ubiquitin ligase complex as an important regulator of cell transformation.

In 2000, Dr. Fuchs formed his own group as an assistant professor at the Department of Animal Biology, School of Veterinary Medicine, University of Pennsylvania (Philadelphia). He currently holds a full professorship in this department and leads the Mari Lowe Center for Comparative Oncology. His group has continued to work on the role of SCF- β Trcp E3 ubiquitin ligase in human cancers. Identification of cell surface receptors for interferon alpha/beta and prolactin as novel substrates for this complex has expanded the focus of subsequent studies toward ongoing investigation of the role of signaling receptors ubiquitination in endocytosis and their regulation in normal cells and in disease, as well as the feasibility of targeting these mechanisms for anti-cancer therapy.