Candidates should have experience with skills of molecular and cell biology. A background in studying gene regulation and/or intracellular signal transduction is desirable.

Michael R Stallcup, PhD

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Michael R. Stallcup, Ph.D. received his B.A. at Yale University, his Ph.D. at the University of California at Berkeley, and did his postdoctoral training at the University of California at San Francisco. He began his career on the faculty at the University of South Carolina, joining USC in 1985 where he is a Professor in the Department of Biochemistry and Molecular Biology. He serves as co-leader with Dr. Peggy Farnham of the Epigenetics and Regulation Program. In his studies on transcriptional regulation by steroid hormone receptions, he is one of the leading researchers in discovering and characterizing transcriptional coregulators. Specifically his research focuses on coregulators that help steroid receptors alter chromatin structure and recruit RNA polymerase to the target genes that are regulated by steroid hormones and their receptors. His lab discovered the first histone methyltransferase and was the first to demonstrate a role for histone methylation in transcriptional regulation. His lab is currently exploring the molecular mechanisms of coregulator action and the physiological roles of specific coregulators in cancer and inflammatory diseases.

Current projects are examining the role of the homologous coregulators G9a (EHMT2) and GLP (EHMT1), which methylate histones and other proteins, in glucocorticoid-regulated transcription. His lab discovered that G9a and GLP can serve as coactivators for some genes and corepressors for other genes. They showed that the coactivator activity is controlled by adjacent methylation and phosphorylation modifications: self-methylation is required for coactivator activity, while phosphorylation by Aurora kinase B inhibits coactivator activity. His lab showed that G9a and GLP are required for glucocorticoid-induced expression of specific genes that lead to cell death in leukemia cells. Glucocorticoids are used as a standard part of therapy for leukemia and other hematologic malignancies, but many patients become resistant to this therapy. Based on the above molecular mechanism, Dr. Stallcup's lab has shown that Aurora kinase B inhibitors and demethylase inhibitors can enhance the coactivator activity of G9a, enhance glucocorticoid activation of the genes that promote cell death, and enhance glucocorticoid-induced cell death in leukemia cells, including cells from leukemia patients that are resistant to the standard therapy. Studies in mouse models are currently underway with an aim to generate preclinical data to support clinical trials conducted by clinical collaborators.

In other projects, Dr. Stallcup's lab is also exploring the roles of G9a and GLP and their post-translational modification in other physiological systems, including energy metabolism in liver, fat, and muscle tissues.

Research Interests: Regulation of transcription by steroid hormones; molecular and physiological roles of transcriptional coregulators

Disease Models: cancer, inflammatory diseases

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