QUANTITATIVE AND SYSTEMS BIOLOGY COLLOQUIUM:
From Megabase to Nucleotide Scale: Enhancer Function in Development and Disease

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Date:
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Time:
10:30 AM - 11:45 AM

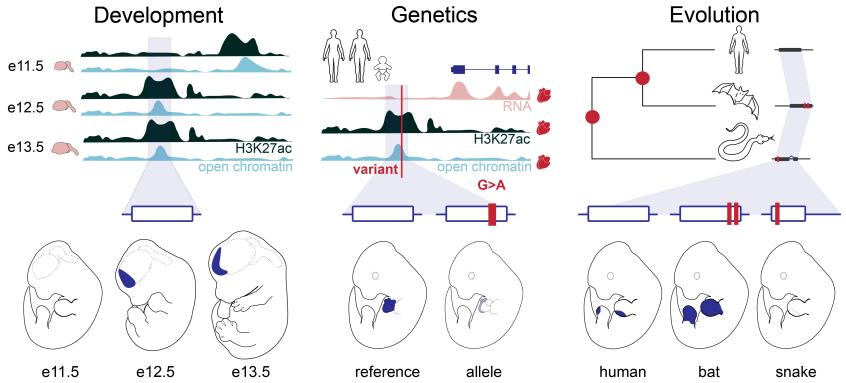
Location:
COB 1 114

About the Speaker:

Dr. Visel received his Ph.D. in 2004 from the Max Planck Institute in Hanover, Germany, where he developed novel tools for large-scale in situ gene expression analysis in mouse embryos and the adult mouse brain. His current research interests cover a wide range of functional genomics approaches aimed at understanding the biological functions encoded in the genomes of animals, plants, and microbes. An area of particular interest that he will be talking about are the functions embedded in the non-coding DNA of the human genome. In particular, his group has been developing methods for the identification and characterization of distant-acting enhancer sequences and has extensively used mouse models to study the role of these enhancers in development, disease, and evolution. Dr. Visel is a Senior Staff Scientist at Lawrence Berkeley National Laboratory and the Deputy Director of Science at the Joint Genome Institute (JGI), a Genome Science User Facility funded by the U.S. Department of Energy. In addition, Dr. Visel holds an appointment as an Adjunct Professor at the School of Natural Sciences at the University of California, Merced.

Abstract:

Understanding the function of enhancers within the context of genome regulation and in vivo biology continues to be a grand challenge. Currently, our limited ability to predict the consequences of sequence changes at the level of an individual enhancer up to larger enhancer architectures is a particular hurdle for the interpretation of whole-genome sequencing data from patients with a wide spectrum of conditions. Our laboratory uses sequence-based molecular approaches including chromatin mapping, synthesis-enabled modification of enhancer sequences, large-scale transgenic mouse studies (http://enhancer.lbl.gov), and CRISPR genome editing in mice to study the in vivo function of enhancers in developmental and disease-related processes. These methods provide insight not only into the in vivo function of enhancers, but also the impact of sequence and structural disruptions on activity and regulatory network architectures. I will illustrate this with data from published and ongoing studies of non-coding functions from the nucleotide to the megabase scale. Examples will include the development of tools for assessing the in vivo significance of clinical enhancer mutations, systematic tiling mutagenesis of enhancers to correlate their inner sequence architecture with in vivo activity patterns, and deletions of megabase-scale gene deserts.



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