



Genome Sciences Seminar

Wednesday, 1.11.17 | 3:30 | Foege Auditorium



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"Evolution, dynamics and genetics of protein post-translational regulation"

Beltrao Lab Research Overview:

Our group is interested in understanding how novel cellular functions arise and diverge during evolution. We study the molecular sources of phenotypic novelties, exploring how genetic variability that is introduced at the DNA level is propagated through protein structures and interaction networks to give rise to phenotypic variability. Within the broad scope of this evolutionary problem, we focus on two areas: the function and evolution of post-translational regulatory networks; and the evolution of genetic and chemical-genetic interactions. Looking beyond evolutionary process, we also seek to understand the genomic differences between individuals and improve our capacity to devise therapeutic strategies.

In collaboration with mass-spectrometry groups, we develop a resource of experimentally derived, post-translational modifications (PTMs) for different species in order to study the evolutionary dynamics and functional importance of post-translational regulatory networks. We use these data to create novel computational methods to predict PTM function and regulatory interactions. Our goal is to gain insights into the relationship between genetic variation and changes in PTM interactions and function.

Changes in cellular interaction networks underpin variation in cellular responses and sensitivity to environmental perturbations or small molecules. As we model and study the evolution of cellular interaction networks, we begin to see how different individuals or species diverge in their response to drugs. Understanding this relationship will enable us to develop methods to predict how genetic changes result in specific sensitivity to drug combinations.

Refreshments served outside the Auditorium at 3:20pm

Questions? Contact Brian Giebel at bgiebel@uw.edu or visit the Seminar website at <http://www.gs.washington.edu/news/seminars.htm>

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