Plenary Abstracts

Günter P. Wagner, *Yale University*

Evolution of Evolvability: adaptive or "arbitrary"

In this talk I want to return to the question how evolvability is determined, or changing in evolution. Evolvability, as I use the term here, is the ability of the genome to produce adaptive genetic variation by mutation. It has long been recognized that evolvability is influenced by the mapping of genetic variation onto phenotypic variation and how the latter then maps onto fitness. Features of the genotype-phenotype map influencing evolvability are epistasis, modularity, mutational effect size, to name a few. The most controversial question is how and whether features of the organism that affect evolvability are evolving and what is determining the outcome of these changes with respect to evolvability. One way of thinking about this is that natural selection is able to shape the genotype-phenotype map in a way to increase evolvability, i.e. adaptive evolution of evolvability. I will discuss a number of objections against this idea, many of which are not supported by a more detailed analysis using mathematical models. However, there is one objection, which terminated my own work on these issues many years ago: it is the finding by Thomas Hansen and Joachim Hermisson that showed that the outcome of natural selection on features of the genotype-phenotype map strongly depends on the statistical structure of epistatic gene interactions. That leads me to the notion of "arbitrary" evolvability, the notion that evolvability evolves by natural selection guided by the structure of the developmental interactions producing the phenotype. I will present a very simple model that illustrates how evolution of "arbitrary" evolvability can shape the genotype-phenotype map and the phenotype of an organism and suggest that the interaction between developmental interactions might be more profound than the latter simply "translating" the genetic mutation into a phenotype variant. These last parts of the presentation are necessarily speculative, and should be viewed as a challenge rather than an attempt to state what is the case.

Chris Jiggins, *University of Cambridge*

The population genomics of adaptation and speciation in tropical butterflies

A major undertaking in evolutionary biology is to link genotype to phenotype and understand the evolutionary changes that lead to adaptation and speciation. An emerging paradigm from this work is the repeated re-use of variants that are far older than the species or populations that harbour them. Furthermore, a pattern of sharing of ancient variants through adaptive introgression seems far more common than was ever envisaged. Here I will give an overview of our work on the brightly coloured Heliconius butterflies. We have studied signatures of selection across wing patterning loci and shown pervasive evidence for selective sweeps, especially at loci with major effects on wing phenotype consistent with strong selection acting on these loci. These loci are a well established example of adaptive introgression which can generate both novelty and convergence in phenotype. Finally, I will focus on the cortex locus which controls yellow and white patterns. CRISPR analysis indicates multiple functional genes at this locus, and provides novel insight into the action of the cortex gene itself. In summary, Heliconius butterflies have provided insight into how a small number of loci with a large array of regulatory alleles can underlie dramatic radiation in ecologically relevant phenotypes.

Tami Lieberman, *MIT*

De novo mutations within human microbiomes

There is an enormous potential for evolution within each of our human microbiomes, with billions of new mutations being created each day. Critically, within-person evolution often leads to diversification of the bacterial population (rather than substitution), enabling the inference of recent within-person evolution without time-series. In this talk, I will highlight the power of tracking within-person evolution for understanding bacterial transmission, identifying genes critical to long-term survival, and for understanding evolutionary principles. I will present examples from infectious diseases, the gut microbiome, and the skin microbiome.

Pleuni Pennings, *San Francisco State University*

How did we stop HIV evolution?

Drug resistance evolution was a huge problem for HIV treatment from the late 80s all the way until a few years ago. It is often suggested that the way to prevent drug resistance evolution is to use at least three drugs at the same time. I will use historical data to show that it is more complicated. The initial three-drug therapies for HIV still allowed for drug resistance evolution to happen. I will propose a hypothesis that can explain why newer triple-drug therapies work much better – we believe that it is related to spatial structure of drug and virus in the human body.