Disentangling genetic and epigenetic components of heritable phenotypic variation in the coevolution of hosts and parasites

Christoph Grunau
University of Perpignan Via Domitia/CNRS, Laboratory “Interactions Hosts-Pathogenes-Environment”, UMR 5244, F-66860, Perpignan, France

Co-evolution in host-parasite systems requires that in both partners the systems that generate heritable phenotypic variants must be synchronized. If variability is too high the organism drives into error catastrophe, if the capacity to produce phenotypic variants is too low, one partner is eliminated. Heritable variability is based on the complex interplay of genetic and epigenetic mutations. Our current research is focused on deciphering the relative importance of both systems in the human parasite *Schistosoma mansoni* and it's intermediate host, *Biomphalaria* spp. *S. mansoni* is a parasitic blood-fluke who's life cycle is characterised by the passage through two hosts: a freshwater snail of the genus *Biomphalaria* for asexual multiplication and man or rodents for sexual reproduction. We have shown that expression of genes that are essential for infection of the snail host is under epigenetic control on the level of histone modifications. Epimutations are parasite strain-specific and are faithfully transmitted to the offspring. Pedigree studies allowed us to trace the heritability of genetic and epigenetic information. Concerning the snail host, our data indicate that DNA methylation is directly linked to the capacity to generate heritable phenotypic variants. In summary, we begin to understand how low-fidelity (epigenetic) and high-fidelity (genetic) inheritance interacts to maintain the fragile equilibrium between too much and to little variability.