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Mutation and Selection in Age-structured Populations

Many features of evolutionary theory depend upon considering the mutational load arising from the slow clearing of slightly deleterious mutations. Kimura, Kingman, Hamilton, and others have developed useful models for this process, and various versions of these models with increasingly sophisticated representations of the genome have been proposed. However, many of these models break down in the presence of epistasis.

This has been a particular challenge for the evolutionary theory of ageing. I will describe a new model, which combines a crude model of the genome with a perfectly general specification of fitness interactions, and describes the evolution in time of the distribution of mutant alleles in the population. The solutions may be derived formally from the Feynman–Kac formula. When we add a (sufficiently strong) recombination term, it turns into a dynamical system on the space of Poisson point processes.

One of the keystones of classic mutation-selection theory is Haldane's Principle, which states that the average mutational load depends only on the rate of mutation generation, and not on the size of the effect of each mutant allele. Our new model implies an age-structured version of this principle: In a stable population, the *aggregate population hazard* is invariant under changes in the strength of mutational effects.

This talk is based on joint work with Steve Evans and Ken Wachter.