The consequences of genetic drift on polygenic characters under a general model of epistasis

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In general, genotypes at different loci interact non-additively to produce phenotypes. A major difficulty in understanding the consequences of these ubiquitous epistatic interactions is that there has been no simple mathematical formalism to describe them. My collaborator Nick Barton and I have adapted our methods for analyzing general forms of multilocus selection to describe multilocus epistasis. Our formulation leads to relatively simple and intuitive expressions for the population mean and all variance components, namely additive, dominance and all levels of epistatic interaction. To illustrate the power of our description, we have investigated how the population mean and each variance component are expected to change when allele frequencies change because of genetic drift associated with a temporary reduction in population size (a "population bottleneck"). Over the past 15 years, a good deal of theoretical and experimental effort has gone into trying to understand these effects. By assuming that the drift-induced changes at different loci are statistically independent, we show that epistasis always increases the expected additive variance after a bottleneck above the value that would be expected for an additively determined character (with neither dominance nor epistasis). We provide both analytical and numerical support for our independent-loci assumption. This assumption allows us to produce explicit formulas for how genetic drift changes the expected value and variance of the population mean and expected values for all variance components. These results yield general conditions under which population bottlenecks will increase the expected additive genetic variance. I will discuss the biological relevance of these calculations and suggest other possible applications.