Dominance-recessivity hierarchy arising from evolution of small RNA regulatory elements.

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By definition, an allele that determines the phenotype of the heterozygote is dominant. The earliest attempt to describe that phenomenon revolved around Fisher's theory on the evolution of dominance.

Fisher's theory of the evolution of dominance is based on the fact that heterozygotes for rare deleterious alleles, maintained by recurrent mutation from the wild-type allele at the locus, greatly outnumber homozygotes in a random-mating population at equilibrium. Fisher postulated that mutant alleles were originally semidominant. He explained the fact that most mutations are observed to be recessive to wild-type as the result of selection for modifier genes (elements controlling dominance-recessivity interactions between alleles at another locus). Modifier elements enhance the fitness of the heterozygotes alone. Fiser noticed that dominance interactions are the result of the evolution of these dominance modifiers.

Wright criticised this. The heterozygotes for mutant alleles are very rare. The intensity of selection on a gene which modifies is of the same order as the rate of mutation to deleterious alleles at the locus undergoing dominance modification. He proposed that such a weak selection pressure in unlikely to overcome the effects of random genetic drift. Although he did not fully reject the possibility that such genetic elements might be exist.

The existence of dominance modifier genes has been debated as a theoretical possibility for more than 90 years. A study of the self-incompatibility locus in the *Brassicaceae* plant family provided the evidence of small non-coding RNAs. This sRNAs control dominance-recessivity by mediating methylation on the promoter of the recessive allele. Small non-coding RNAs operate as a trans-modifier of the gene controlling pollen specificity by methylation of the promoter of recessive alleles. This leads to transcriptional silencing of recessive alleles by dominant alleles. Nonetheless, the mechanism in the more complex dominance-recessivity network is unknown.

The mechanisms of genetic dominance have been strongly debated. However, for deleterious mutations this disagreement was nearly resolved. The understanding of physiological properties of metabolic pathways and dominance modifiers explain many dominance-recessivity interactions in genomes.

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