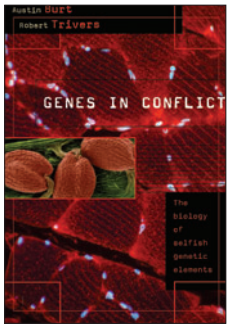
Genes get diabolical



Genes in Conflict: the Biology of Selfish Genetic Elements

By Austin Burt and Robert Trivers

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Reviewed by Harmit S Malik

It has been nearly three decades since Richard Dawkins' *The Selfish Gene* popularized the notion that genes could act in their own self-interest, sometimes at huge cost to the organism encoding them. Now, in a meticulously assembled, thought-provoking and sometimes deliciously speculative textbook, Burt and Trivers' *Genes in Conflict* documents the selfish genetic elements that populate eukaryotic biology. Reading this book from a narrow viewpoint, one could see it as a recurrent tale of genetic conflict, but even from that perspective, the staggering variety of selfish strategies it discusses alone makes it worth reading. A broader view of this compilation is as a recurrent tale of genetic opportunity, revealing the innovative and insidious nature of genes as they vie with each other in complex sociogenetic negotiations for evolutionary survival.

Opportunities to cheat are thought to arise owing to three central biological differences. First and perhaps least controversial is the differential ability to replicate. One would brook no argument with the contention that viruses are selfish entities. By the same token, 'transposable elements' (chapter 7), 'homing endonucleases' (chapter 6) and similar genetic elements belong in this category, because of their ability to increase their copy number within the genome. 'Selfish cell lineages' (chapter 11), such as cancer cells, can disobey an ordered set of rules that ensure that a multicellular organism develops along a canonical pathway. A cancer-predisposing mutation can spontaneously arise within clonal descendants of a single progenitor cell. In addition, nature holds several examples of organisms that are derived from cells from genetically distinct progenitors. These genetically distinct cells also compete for evolutionarily valuable niches in the developing organism, such as the germline that will get passed on to the next generation.

The second opportunity to cheat arises from differential transmission. Selfish elements can act as 'autosomal killers' (chapter 2) to ensure their own evolutionary dominance by eliminating or reducing the fitness of gametes or embryos that do not carry the selfish element. 'Selfish sex chromosomes' (chapter 3) reduce the transmission of the other sex chromosome. Thus, in XY males, genetic elements on the X can prevent the transmission of the Y. To counteract this, suppressors will arise on the Y (to simply survive) or on autosomes (to prevent deleterious skews in sex ratio in the population). A particularly insidious means of subverting transmission is via the process of 'genomic exclusion' (chapter 10), in which the entire complement of either the paternal or maternal chromosomes is eliminated in every single generation. In wasps, a supernumerary chromosome, PSR, is exclusively transmitted through males but eliminates all the male chromosomes that were inherited with it, ensuring its own maximal transmission. Not all drive necessarily involves such large-scale fratricide. In female meiosis, only one of four meiotic products gets selected to become the egg, whereas the others are destined for elimination. Here, chromosomes can 'cheat' by favoring their own position (a process called 'gonotaxis') (chapter 8) such that they are selected to be the egg more often than not, defeating one of the chief rules of mendelian inheritance. The evolution of many chromosomal features—from 'wannabe centromere' knob elements in maize to even *bona fide* centromeres on plant and animal chromosomes—can be explained by this simple process. Intriguingly, the evolutionary origins of asymmetry in female meiosis are themselves shrouded in mystery, with few convincing arguments for why this asymmetry would benefit the organism. Many supernumerary B chromosomes (chapter 9) also exploit gonotaxis in both female meiosis and plant male meiosis. As organellar genomes tend to be uniparentally inherited, typically from the maternal germline, 'selfish mitochondrial mutations' can also exploit differential transmission to benefit females even at considerable cost to males (chapter 5). It would have been especially interesting in this chapter to compare mitochondria with other intracellular bacteria, such as *Wolbachia*, that have a decidedly more nefarious reputation of subverting host physiology for their own benefit.

The third opportunity to cheat arises from 'differential resource investment'. In all mammals, because most of the parental investment in offspring development is contributed maternally, the paternal genome is typically hard wired to gain maximal resources for the developing offspring, whereas the maternal genome allows for control of limited maternal resources. These ideas have allowed a grand synthesis of seemingly disparate observations to a unified, predictable theory of 'genomic imprinting' (chapter 4). It is hard to overstate the contribution of this elegant extension of economic theory to explain the differential expression of homologous genes, depending on which parent contributed these genes.

One of the chief merits of the book is that Burt and Trivers have eschewed the more convenient approach of editing chapters penned by other experts. This allows them to bring the same multidisciplined scrutiny to bear on all the selfish elements discussed here, filling in gaps with (mostly) cautious speculation. It has required tremendous scholarship and decade-long dedication. The result is a powerful resource in evolutionary genetics that will help influence many skeptics who would (still) rather think of selfish genes as simply rare deviants in a largely symbiotic network of genetic interactions. The overwhelming value of the book is the opportunity it highlights, as there are unanswered questions even in the best-studied instances of selfish genes. It both serves as a guidebook and a 'call to arms' for the next generation of inquisitive students, hopefully whetting their appetites and leaving them asking for more.

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COMPETING INTERESTS STATEMENT
The author declares that he has no competing financial interests.