How our Genes Lie: Honest and Dishonest Genes in Sexual Selection

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Abstract

Natural selection has been understood for over a hundred years, but the mechanisms by which it works have not been identified. One of the forms it takes is sexual selection. Sexual selection is an evolutionary pressure conferred by the opposite sex of the same species. The good genes hypothesis, posed in the 1930s, attempted to reconcile mate choice and the selection for certain traits. The selfish gene hypothesis, first declared in 1976, attempted to explain mate choice as well as our behaviors. With our modern understanding of genetics and DNA that holds the information, these two hypotheses can be applied to identify the honest and dishonest genes that are passed down generation after generation.

Introduction

While the molecular basis is unknown, the role of genes in heredity has been common knowledge since the 1930s. The good genes hypothesis proposed that individuals choose mates on certain phenotypes that pose a genetic advantage for the next generation. To apply this to humans, the attractiveness we prescribe to an individual reflects that individual's genetic superiority. This is an incomplete model given that different people find different individuals attractive. A possible supplement to the model is the selfish gene hypothesis. The selfish gene hypothesis proposes that our mate choice is a result of our interest to pass our genetic code on to the next generation. A human application of this would be that we choose our mates based on that individual's similarity to our own genome, thereby probabilistically increasing the longevity of our genes. Both hypotheses have merit but fail to independently explain the presence of honest and dishonest genes; but, together, honest and dishonest genes are made inevitable.

When discussing honest and dishonest genes, it is important to clarify that sexual selection works via the selection of phenotypes, not genotypes. Phenotypes are observable characteristics of an organism and these traits are influenced by the organism's genes. Since genotypes cannot be seen, phenotypes are used for selection as they are an indirect manifestation of the organism's genes and experiences. An example of this is if a male peacock has a mutation in a gene important in feather development. A result of this mutation is an upregulation of a hormone responsible for feather growth, thereby increasing the relative size of the peacock's plumage. Since plumage size is a sexually selected trait in peacocks, the mutated peacock would be selected to a greater degree by hens than a wild-type peacock. The disparity in the selection of males with varying secondary-sexual traits, affected by variation in genotype, is the basis of sexual selection contributing to the evolution of the organism. Yet, while advantageous mutations account for an evolutionary change over the course of multiple generations, genes do not independently explain why a trait is sexually selected. For that, genes must manifest into phenotypes that suggest an evolutionary fitness of the organism. Unfortunately, the path from gene to trait is not without its own set of variation.

Environment plays a key role in phenotype and the development of a sexually selected trait. Genotype does not determine phenotype. Genes code for proteins. Phenotypes can be anything from horn allometry, as in Onthophagus beetles, all the way to call syllables, as in bush crickets. What links genes to corresponding proteins are, most often, a suite of developmental and cellular mechanisms. It is this developmental and cellular link between genes and phenotypes that explain the plasticity of phenotypes. Phenotypic plasticity is the phenomenon that multiple phenotypes can arise from a single genotype; one example is the case of monozygotic twins. Imagine a pair of monozygotic twins, Jim and Jeff. Jim frequents a gym regularly and ensures he maintains a balanced diet. Jeff, on the other hand, frequents a buffet regularly and ensures his freezer is filled with his favorite midnight snacks. It would not be a surprise to find out that Jim has a lower body mass index (BMI) than Jeff despite having the exact same genotype. There was nothing that predisposed Jeff to a higher BMI than Jim. What ensured his increased insulation was the environment he experienced. In summary, genes lead to phenotype, but the phenotype is also moderated by the environment.

Honest and Dishonest Genes

What determines the honesty of a gene is how accurately it depicts, via a phenotype, the fitness of the organism. From a sexual selection standpoint, the evolution of honest genes would be favorable. In addition, over the course of multiple generations, the scruples of sexually selective pressures would refine the accuracy of the honest genes as it would lead to a sensitive and more prosperous method of selection. This is a case of resolution. Imagine a doe is searching for a buck for mating. Two bucks, Skip and Skippy, appear with similar size and muscle proportion. The only way they differ are their coats and horns. Skip has a relatively dull coat and small horns relative to body size while Skippy has a full shiny coat with a large ornament rack relative to body size. Skippy is favorably selected by the doe for mating. In this situation, genes that synthesize androgenic hormones and genes involved in insulin/insulin-like growth factor signaling (IIS) are honest genes; androgens are positively correlated with hair development and IIS is positively correlated with rack size. This situation is favorable for the doe and Skippy as they both have an increased probability of passing their genes on to the next generation. Skip, on the other hand, draws the short straw in the field of honest genes. He, therefore, favors a dishonest set of cards.

Imagine the doe and two bucks scenario once again with Skippy still being the more sexually favored. Now include a mutant Skip. This Skip has a mutation in genes involved in IIS that increase IIS and, further downstream, upregulate androgenic hormones. Mutant Skip has a glossy coat and large rack relative to body size which catches the doe's eyes to a greater extent than Skippy's features. In turn, mutant Skip is selected instead of Skippy. While the genes involved were originally honest, the mutation in Skip's genome made the environment insubstantial in affecting the final phenotype and thus lead to dishonest phenotypes. In this scenario, the doe and Skip win. However, the doe wins at a probability of smaller magnitude as the offspring may be less fit than the offspring of an honest mate. The disparity in winning magnitude offers logic toward a selective pressure in does to increase their resolution for sexual selection; the better the does are at discerning honest genes, the more likely their genes will survive to the following generations. However, the presence of dishonest genes in species either supports the idea that dishonest genes are inevitable with random mutation or, more poignantly, the disparity in winning magnitude due to potential filial unfitness is not enough to select against dishonest genes.

The Two Theories

The presence of honest and dishonest genes highlights a sexually divergent initiative in sexual selection. The sexual selector prefers honest genes, while the sexual selectee prefers either honest or dishonest genes – whatever offers an advantage in increasing gene longevity. In turn, a theory of sexual selection must reconcile both initiatives.

Together, the good genes hypothesis and the selfish gene hypothesis explain the honest-dishonest genes phenomenon. The good genes hypothesis explains honest genes. In the good genes hypothesis, genes that accurately illustrate the fitness of the organism are preferably selected above inaccurate genes. This theory explains the disparate winning advantage in dishonest selection and offers a selective pressure against dishonest genes. Evidence for this theory can be found in IIS-dependent traits. Almost all animals use IIS for cellular and physiological development. One of the reasons IIS is so conserved is that IIS is upregulated in high nutrition. Therefore, an organism in high nutrition has full or increased development due in part to high IIS. In turn, it makes sense that sexual selection would work on traits that are insulin sensitive, allowing greater selection accuracy of well fed mates. However, the presence of dishonest genes indicates a second manner of sexual selection at work.

The selfish gene hypothesis explains the presence and longevity of dishonest genes despite the selective pressure against them offered by the good genes hypothesis. In the selfish gene hypothesis, animal behavior, including mate choice, is explained to increase the longevity of an individual organism's genes. An example of this would be the mutant described above, Skip. The mutant Skip illustrates the presence of a dishonest gene via a mutation. According to the selfish gene hypothesis, what offers a dishonest gene its longevity, in addition to the phenotypic

advantage, is the fact that organisms with the same gene tend to mate with one another, thus increasing the probable lifetime of the dishonest gene.

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