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Sexual selection: Changing the definition of the fittest

Wouter van der Bijl and Judith E. Mank*
Department of Zoology and Biodiversity Research Centre, University of British Columbia, Vancouver, BC V6T 1Z4, Canada
*Correspondence: mank@zoology.ubc.ca
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Sexual selection has long been known to produce rapid evolution of spectacular traits. A new study reveals how sexual selection can also rapidly reshape the genome.

Sexual selection, the evolutionary forces resulting from mating competition, has produced an array of striking and sometimes beautiful traits (Figure 1). Competition within a sex for access to mates can result in weapons, such as horns in male rhinoceros beetles, which are used to fling rival males away from females[1]. Other sexually selected traits help individuals attract mates, and these can act through a range of sensory systems. Bright colours, such as brightly coloured male butterflies[2], or morphological ornaments, such as wide eye stalks in male stalk-eyed flies[3], are based on visual perception. Mating calls or songs, like the chirping of male field crickets at night[4], are heard. Insects in particular also rely on pheromones, or chemical olfactory cues, for mate choice. For example, in the jewel wasp, the single most highly expressed gene in males is a sex pheromone[5] to attract mates through their sense of smell. New work reported in this issue of *Current Biology* by Wyer *et al.*[6] uses experimental evolution of mosquitoes to demonstrate the pervasive role of sexual selection in shaping genomes.

Sexually selected traits are often energetically costly to produce. The nutritional resources required for a male rhinoceros beetle to grow a horn could be deployed for other things. More importantly, sexually selected traits can reduce survival when they make individuals more conspicuous to predators, as in the case of flashy colours, or less able to escape them, such as the case of the stalk-eyed fly eye stalks, which present a major aerodynamic drag in flight[7]. This results in an apparent conundrum — how can these traits evolve and persist when they would be selected against by natural selection? Darwin[8] conceptualized sexual selection as a distinct evolutionary force to solve this riddle[9]. Indeed, he realized that these traits can evolve despite their inherent toll if they provide an advantage over rivals in producing offspring.

Sexual selection has remained at the heart of evolutionary biology since Darwin's observations, and we have elegant theory and powerful phenotypic models. However, the integration of empirical genomic and population genetic approaches into the study of sexual selection has lagged behind similar approaches in other fields such as adaptation and speciation. Because of this, the genomic basis of sexual selection has remained largely theoretical.

Experimental evolution of sexual selection, where mate competition and choice are manipulated in the laboratory, is a powerful approach to study sexual selection in real time. By placing each female with multiple males, males compete and females choose which to mate with, allowing for sexual selection.

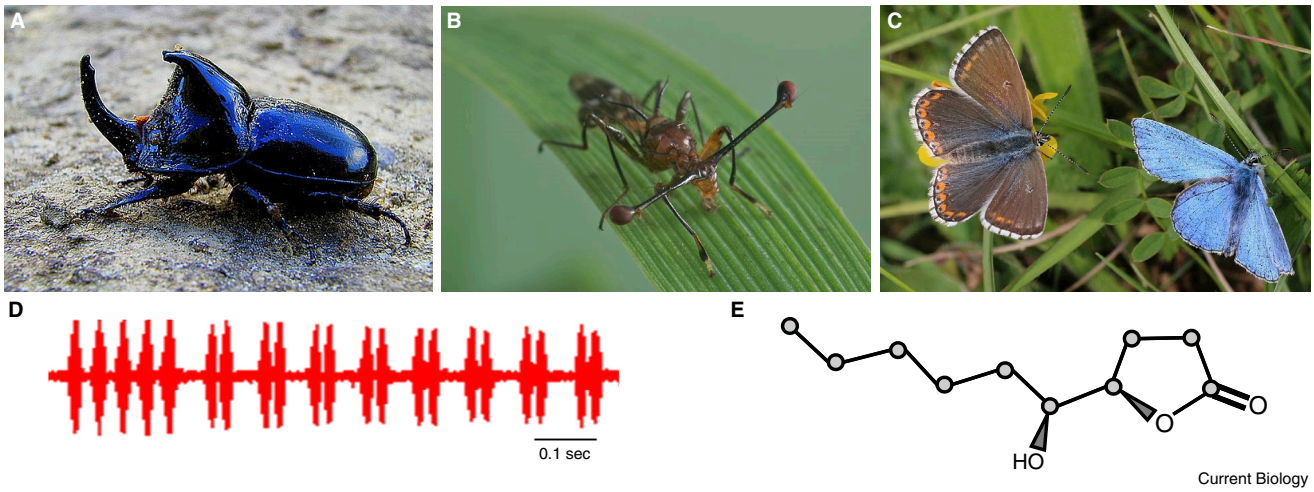

Figure 1. Traits produced by sexual selection.
(A) Male rhinoceros beetles use their horns to compete for access to mates. Photo: Alex Popovkin, Bahia, Brazil/ Flickr (CC BY 2.0). (B) Male stalk-eyed flies with wider eye stalks are preferred by females. Photo: Rob Knell/ Wikicommons (CC BY-SA 2.5). (C) Adonis blue (*Polyommatus bellargus*) males (right) are bright and iridescent while females (left) are more cryptic. Photo: Charles J. Sharp/Sharp Photography via Wikicommons (CC BY-SA 4.0). (D) Male crickets rub the edges of their front wings together to produce sounds, called stridulation, to attract female crickets as mates. Ocellogram of the field cricket stridulation is shown. Image: Rymiduff/Wikicommons (CC BY-SA 3.0). (E) Chemical structure of the male sex pheromone in the Jewel wasp.

Conversely, competition and choice are abolished when females are paired with a single male randomly chosen from the population, and sexual selection is removed altogether. Wyer *et al.*[6] used these approaches, creating separate populations of the yellow fever mosquito (*Aedes aegypti*) where females were placed in a vial with either five males (polyandry) or a single male (monogamy), and maintained these mating systems for over five successive generations.

The evolutionary pressures associated with reproduction are remarkably strong, and traits can change very quickly in response to increased sexual selection. For example, male yellow dung flies evolve larger testes in response to polyandry, and females have larger sexual accessory glands, an organ they use to influence paternity[10]. Experimental polyandry in the fruitfly *Drosophila pseudoobscura* results in both sexes producing more and different pheromones[11].

Abolishing sexual selection experimentally through enforced monogamy can also result in rapid change. For example, dung beetles evolve smaller testes under monogamy, and their ejaculate is less competitive at fertilizing ova compared to males that evolve with sexual selection[12]. Similarly, male mosquitoes evolved under monogamy are less successful in competing for females against males that evolved under sexual selection[13]. The reduced competitive ability of males in response to enforced monogamy reveals the trade offs between sexual and natural selection. Males that invest in these traits are highly competitive and favoured by sexual selection despite the energetic or survival expense. However, the cost of these traits places them at a significant disadvantage when sexual selection is removed and they no longer confer increased paternity.

Wyer *et al.*'s[6] work reveals how sexual selection acts on the genome to produce these rapid changes in response to experimental evolution. When they compared the populations evolved with and without sexual selection, they observed differences distributed throughout the genome. Their results reveal that manipulating the strength of sexual selection acts on many loci at once, underscoring the pervasive influence of sexual selection spanning from one end of the genome to the other[14–16].

Some genomic changes in response to the removal of sexual selection in yellow fever mosquitoes were observed in all the experimental replicates, suggesting that these alleles are consistently favoured under monogamy. Many of these changes were associated with odor perception. Because smell is important in the formation of mating swarms and therefore finding a mate in natural populations of this species, it is easy to see how selection on this sense might change in response to the elimination of sexual selection, when males no longer have to compete to find a receptive female.

Perhaps more surprising, Wyer *et al.*[6] observed that the loss of sexual selection also produced highly variable changes in the genomes of the replicate monogamous populations. While the three polyandrous populations showed relatively little genomic change compared to the ancestral population, which also experiences strong female choice, the monogamous populations each evolved in very different ways from each other and from the ancestral starting population. These key results demonstrate that there are many loci in the mosquito genome that are shaped by sexual selection, and once sexual selection is removed, much of the genome changes more or less randomly in various directions.

The love lives of mosquitos do not just teach us about the workings of evolutionary biology, but have real world consequences. Mosquitos are responsible for the spread of many human diseases, including yellow fever, malaria and dengue. Modern control strategies increasingly target the mosquito mating system by large-scale releases of lab-reared males[17]. These males have been made infertile or infected with a pathogen, so that they mate with females but produce no viable offspring[18,19]. Wyer *et al.*[6] show that these lab-reared mosquitoes must 'use it or lose it' — sexual selection must be allowed to act or they will be outcompeted by wild males and the control strategy will fail.

DECLARATION OF INTERESTS

The authors declare no competing interests.

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Metabolism: How removal of damaged cells impacts energy availability in the retina

Connor Lantz[1,2] and Edward B. Thorp[1,2,3,4]
[1]Department of Pathology, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA
[2]Feinberg Cardiovascular and Renal Research Institute, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA
[3]Department of Pediatrics, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA
[4]The Heart Center, Stanley Manne Children's Research Institute, Ann & Robert H. Lurie Children's Hospital of Chicago, Chicago, IL, USA
Correspondence: connor.lantz@northwestern.edu (C.L.), ebthorp@northwestern.edu (E.B.T.)
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Once thought to be a quiescent process, elimination of damaged cells by professional phagocytes is now understood to modulate metabolite availability within tissues. A new study reveals that the retinal pigment epithelium serves as a local source of insulin after engulfment of damaged photoreceptors.

At its core, metabolism is a set of life-sustaining chemical reactions that transforms chemical energy into energy for cellular processes. These chemical reactions allow organisms to grow, reproduce, and respond to their environments. Therefore, it may not be surprising that a critical tissue for responding to environments — the eye — is highly active metabolically. Photoreceptors in the retina rely on aerobic glycolysis to meet their energy demands to convert light into signals that stimulate physiological responses[1,2]. These photoreceptor cells, however, are separated from glucose and other essential metabolites in the underlying layer of blood vessels by the retinal pigment epithelium (RPE), which forms the blood–retina barrier. Consequently, the RPE maintains the health of the sensitive photoreceptors by transporting molecules across this barrier and by removing damaged photoreceptor outer segments (POS). A new study published in *Nature Metabolism* led by the labs of Kodi Ravichandran and Jayakrishna Ambati reports a molecular link between these two functions of the RPE[3]. They discovered that RPE cells provide a phagocytosis-induced source of insulin in the retina, and this source of insulin is necessary for metabolic homeostasis in the retina during starvation (Figure 1). These influential findings add to our understanding of how cell death, removal and recycling modulate tissue metabolism.

Within the vertebrate eye, photoreceptor cells absorb photons through G-protein coupled receptors called opsins that activate a phototransduction cascade in the POS. High levels of light, however, induce photo-oxidative reactions within the photoreceptor cells. To deal with these cytotoxic reactions, photoreceptor cells shed their POS daily, and the POS are subsequently phagocytosed by the RPE (Figure 1B)[4]. The magnitude of this homeostatic process is tremendous; estimates suggest that every phagocyte of the RPE engulfs hundreds of thousands of POS discs over a human lifetime[4]. Thus, POS phagocytosis has been linked to metabolic changes in the retina. Previous insights revealed that engulfment of POS not only fuels the RPE through fatty acid metabolism[5], but also regulates the transport of circulating glucose through the RPE to the retina[6]. The direct molecular pathways that connect POS phagocytosis and metabolic homeostasis in the retina, however, were poorly understood.

Keenly aware of the connection between RPE phagocytosis and metabolism, Iker Etchegaray *et al.*[3] re-analyzed RNA-sequencing data from their previous study examining the effects of the loss of the phagocytic receptor Mer tyrosine kinase (MerTK) on retinal degeneration[7]. The gene *Ins2* (one of two genes that encodes insulin) was significantly downregulated in the RPE of mice lacking MerTK, while other factors related to growth and metabolism were not altered. Furthermore, *Ins2* is distinctly expressed in the RPE and not expressed in the photoreceptors or neurons of mouse and human retinas. Interestingly, *Ins2* mRNA level fluctuated in the retina at different time points during the day, following a similar circadian pattern previously reported for POS phagocytosis. This observation led the investigators to determine whether POS phagocytosis directly induces *Ins2* expression. Inhibition of two phagocytic