

Pleiotropy and the Genomic Location of Sexually Selected Genes

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ABSTRACT: Sexual selection drives the evolution of traits involved in the competition for mates. Although considerable research has focused on the evolution of sexually selected traits, their underlying genetic architecture is poorly resolved. Here I address the pleiotropic effects and genomic locations of sexually selected genes. These two important characteristics can impose considerable constraints on evolvability and may influence our understanding of the process of sexual selection. Theoretical models are inconsistent regarding the genomic location of sexually selected genes. Models that do not incorporate pleiotropic effects often predict sex linkage. Conversely, sex linkage is not explicitly predicted by the condition-dependent model (which considers pleiotropic effects). Evidence largely based on reciprocal crosses supports the notion of sex linkage. However, although they infer genetic contribution, reciprocal crosses cannot identify the genes or their pleiotropic effects. By surveying the genome of *Drosophila melanogaster*, I provide evidence for the genomic location and pleiotropic effects of 63 putatively sexually selected genes. Interestingly, most are pleiotropic (73%), and they are not preferentially sex linked. Their pleiotropic effects include fertility, development, life span, and viability, which may contribute to condition and/or fitness. My findings may also provide evidence for the capture of genetic variation in condition via the pleiotropic effects of sexually selected genes.

Keywords: sexual selection, pleiotropy, sex linkage, condition dependence, *Drosophila melanogaster*, evolution.

Sexual selection is defined as the competition for mates (Darwin 1871). Elaborate adaptations in males are widely studied as being targets of mate choice by females and/or

used in male-male competition (Andersson 1994). These male traits and female preferences often result in differential fitness effects on the sexes due to factors such as sexual conflict (Chapman et al. 2003). Fisher (1931), and later Rice (1984), proposed that genes having differential fitness effects on males and females (sexually antagonistic) are expected to be found on the sex chromosomes (see also Charlesworth et al. 1987; Hurst and Randerson 1999). On this premise, several recent articles have explored the location of sexually selected genes and the subsequent implications for the operation of sexual selection, all of which found significant sex linkage (Reinhold 1998; Lindholm and Breden 2002; Reeve and Pfennig 2002).

In a meta-analysis of reciprocal crosses from a variety of mammals and insects (including *Drosophila*), Reinhold (1998) implicates the X chromosome in at least one-third of the phenotypic variation in sexually selected traits. In organisms that have recombination between the sex chromosomes (e.g., poeciliid fishes), Lindholm and Breden (2002) predict sexually selected genes to be primarily Y and X linked. The authors use a meta-analysis of reciprocal crosses in poeciliids to support these predictions. Assuming a single gene for male trait and a single gene for female preference, Reeve and Pfennig (2002) develop a model that assumes sexual selection as the sole evolutionary force driving the evolution of sexually selected genes; they do not consider pleiotropic effects. Their model determined that sex chromosomes are the preeminent genomic location of sexually selected genes where they are most protected against chance loss by factors such as drift. In particular, the "Z" sex chromosome in female heterogametic systems (ZW; birds and Lepidoptera) was predicted to be the favored location. With support from a meta-analysis, the authors argue that animals having the ZW sex chromosome system are more conducive to sexual selection (e.g., birds have extremely elaborate sexually selected traits). Recent work using additional mathematical models further shows that sex linkage can influence the correlation between sexually selected traits in males and the preferences for those traits in females (Kirkpatrick and Hall 2004).

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The multiple phenotypes associated with a single genetic locus are called pleiotropic effects (Caspari 1952; Barton 1990). It is now widely acknowledged that most genes have pleiotropic effects (Caspari 1952; Wright 1968; Waxman and Peck 1998 and references 14–25 therein), including genes that influence behavior (Hall 1994). Contrary to initial estimates of approximately 120,000 genes, the human genome has now been reduced to approximately 30,000 genes (Lander et al. 2001; Venter et al. 2001). Pleiotropic effects may in part provide a mechanism to explain how such a reduced number of genes can produce animals as complex as humans.

Pleiotropic effects can significantly alter the predicted path of evolution because of the restrictions they can impose on the adaptability and evolvability of genes and their phenotypes (Hodgkin 1998). For example, Barton (1990, p. 774) points out that “the variation of any one character may be a mere side effect of polymorphisms maintained for quite other reasons.” Therefore, to fully understand the selective forces acting on any given gene, we must identify and consider all of the phenotypes associated with that locus.

Sexual selection is well documented in *Drosophila melanogaster* (e.g., Greenacre et al. 1993; Andersson 1994; Chapman et al. 1995, 2000, 2001; Chippindale et al. 2001; Iliadi et al. 2001; Bangham et al. 2002; Miller and Pitnick 2002; Friberg and Arnqvist 2003; Mack et al. 2003) and is currently the best model for investigating the genetic basis of sexually selected traits. The vastly annotated genome contains genes underlying traits including male and female courtship behavior, male courtship song, male and female sexual receptivity, and various accessory gland proteins transferred by males during copulation. Moreover, the associated pleiotropic effects of many genes are also known.

This study provides evidence for the location and associated pleiotropic effects of genes that contribute to sexually selected traits in *D. melanogaster* using genome annotations of characterized genes. In contrast, reciprocal crosses cannot identify the genes or their pleiotropic effects. In addition, my survey is not constrained by maternal effects, Y effects, and cytoplasmic effects, all of which are indistinguishable from X effects when measured using reciprocal crosses. Genes were deemed pleiotropic if they contribute to two or more different traits (e.g., the yellow locus underlies male courtship behavior and larval development). If at least one of the traits associated with a gene is under sexual selection, the gene was deemed to be “sexually selected.” This designation was used to differentiate between genes that contribute to sexually selected traits and genes that do not. Sexually selected genes may be influenced by selective forces other than sexual selection.

My results suggest that the genomic location and ability

of sexually selected genes to adaptively respond to sexual selection may be constrained by pleiotropic effects; these genes are not free to evolve in response to sexual selection alone. In addition to their role in sexual selection, many of these genes have pleiotropic effects that could contribute to condition and/or fitness. The important pleiotropic effects of these sexually selected genes may provide evidence for the intralocus capture of genetic variation in condition.

Methods

Baseline Estimation of Pleiotropy within the Drosophila melanogaster Genome

Using FlyBase (<http://www.flybase.net>), I randomly sampled 100 genes to estimate the general extent of pleiotropy throughout the genome. I used the following criteria: the gene must be mapped to a chromosomal region within the genome, and phenotypes must be noted in the “biological process,” “mutants affect,” or “summary” sections of the FlyBase report for each gene. A gene not meeting these criteria was deemed incompletely annotated at the present time and was removed from the analysis, and another gene was sampled randomly. Pleiotropy was conservatively estimated to be present when two or more overtly different structures, behaviors, molecular processes, or combinations of any of these were associated with a single gene. In instances where the various phenotypes were presumably related (e.g., wing venation, wing shape, and flight), the gene was judged to be nonpleiotropic. An example of a pleiotropic gene is *Dorsal switch protein 1 (Dsp1)* located on the X chromosome, with mutations affecting the egg, haltere, viability, fertility, and locomotor behavior. A nonpleiotropic gene is *blisterwing (blis)* located on the second chromosome where mutants affect only the wing. It should also be noted that genes deemed nonpleiotropic in my analysis may actually be pleiotropic because additional phenotypes might be as yet unidentified. This would underestimate the extent of pleiotropy reported here.

Pleiotropy and the Genomic Location of Sexually Selected Genes in Drosophila melanogaster

Sexually selected genes were considered to be those contributing to male or female courtship behavior, courtship song, and receptivity, along with several accessory gland proteins used by males. These genes were identified using two methods. First, I surveyed genes discussed in various primary literature resources (Chapman et al. 1995, 2000, 2001; Sokolowski 2001; Emmons and Lipton 2003). Second, I identified additional genes using the *Drosophila melanogaster* genome database search engine (FlyBase) via the

Table 1: Expected and observed genetic contribution of each chromosome to sexual selection in *Drosophila melanogaster*^a

| Chromosome | Amount of euchromatin (Mb) ^a | Percentage of genome represented ^a | Expected number of sexually selected genes | Number of sexually selected genes surveyed |
|------------|---|---|--|--|
| X | 21.8 | 18.4 | 11.6 | 17 |
| 2 | 44.4 | 37.4 | 23.6 | 25 |
| 3 | 52.4 | 44.2 | 27.8 | 21 |
| Total | 118.6 | 100 | 63 | 63 |

Note: Expected values were calculated based on the portion of euchromatin contained on each chromosome (X, 2, or 3). The genomic location of sexually selected genes does not significantly differ from chance expectations ($\chi^2 = 4.26$, $df = 2$, $P = .1188$).

^a Based on figure 1 in Adams et al. (2000).

keywords courtship, reproduction, mating, and song. All genes from the primary literature survey arose when searching in FlyBase using the aforementioned keywords. To be included in my analysis, each gene must have met the following criteria: the gene must be mapped to a chromosomal region within the genome, and phenotypes must be noted in the “biological process,” “mutants affect,” or “summary” sections of the FlyBase report for each gene. On the basis of the genes available as of January 2004 and the search criteria outlined above, 63 putatively sexually selected genes were included in my survey. The genomic location and pleiotropic effects of these genes were determined using the same criteria as used for the randomly sampled genes (see above). Genes responsible for primary sex traits such as sex differentiation and, in general, genitalia were not included (but see Eberhard 1985).

Results

Baseline Estimation of Pleiotropy within the Drosophila melanogaster Genome

My baseline estimation of pleiotropy revealed that at least 78% of all genes in *Drosophila melanogaster* are pleiotropic (78 of 100 randomly sampled genes; for a full list, see table A1 in the online edition of the *American Naturalist*). Approximately 73% (46 of 63) of the sexually selected genes have pleiotropic effects. The extent of pleiotropy within the sexually selected genes is not significantly different from my baseline genome estimation (Yates’s corrected $\chi^2 = 0.02$, $df = 1$, $P = .8848$).

Pleiotropy and the Genomic Location of Sexually Selected Genes in Drosophila melanogaster

The 63 putatively sexually selected genes surveyed in this study are not preferentially sex linked; 17 were found on the X chromosome (27%), 25 on the second chromosome (40%), and 21 on the third chromosome (33%). This distribution is not different from that expected based on es-

timates of the relative number of genes on each chromosome type where the X chromosome contributes approximately 18.4%, the second chromosome contributes 37.4%, and the third chromosome contributes 44.2% (values extrapolated from fig. 1 in Adams et al. 2000; $\chi^2 = 4.26$, $df = 2$, $P = .1188$; tables 1, 2). There was a tendency toward a greater number of sexually selected genes on the X chromosome, but this was not significant. Note also the underrepresentation from the third chromosome (27% fewer than expected). Sexually selected genes were not found on the fourth chromosome and Y chromosome. The fourth chromosome of *D. melanogaster* is approximately one-fifth the size of the other chromosomes and contributes approximately 1% of the total number of genes (Adams et al. 2000). As such, the fourth chromosome is routinely disregarded in genetic analyses (Ashburner 1989; Greenspan 1997). The Y chromosome of males, although large in size, consists almost entirely of heterochromatin, which is generally thought to contain incredibly few genes (Adams et al. 2000).

Discussion

Contrary to recent reports (Reinhold 1998; Lindholm and Breden 2002; Reeve and Pfennig 2002), sexually selected genes in *Drosophila melanogaster* are not preferentially sex linked. This distribution was not different from expectations that accounted for the genetic contribution of the chromosomes. Although a tendency toward a greater number of sexually selected genes on the X chromosome was found, this was not significant. This nonsignificant overrepresentation may be an artifact of the intense screening and annotation of X-linked genes in *D. melanogaster* (see Ashburner 1989).

The extent of pleiotropy in sexually selected genes (73%) is not different from that found in my baseline estimation of pleiotropy within the genome (78%). In contrast to the prediction of Reeve and Pfennig’s (2002) recent genetic model, pleiotropic effects may serve to protect these sex-

Table 2: Genes contributing to sexual selection in *Drosophila melanogaster* ($N = 63$)

| Gene ^a | Location | Sexually selected phenotypes | Additional phenotypes | Pleiotropic |
|--|----------|---|--|-------------|
| <i>Adar</i> | X | Male courtship defective | Wing, photoreceptor cell, adult brain, grooming behavior defective, locomotor behavior defective, recessive flight behavior defective, adult recessive | Yes |
| <i>cacophony</i> | X | Song defect | Phototransduction, optomotor behavior, photophobic | Yes |
| <i>celibate</i> | X | Defect in copulation | | No |
| <i>courtless</i> | X | Failure to court | Male sterile | Yes |
| <i>don giovanni</i> | X | Female courtship behavior | | No |
| <i>dunce</i> | X | Courtship behavior | Learning and memory, olfactory learning, oogenesis, sterility | Yes |
| <i>ether a go-go</i> | X | Courtship behavior | neuromuscular junction, leg | Yes |
| <i>icebox</i> | X | Female courtship behavior | Seminal receptacle | Yes |
| <i>inactive</i> | X | Female courtship behavior | Short life span, chemical sensitive, mating success | Yes |
| <i>no-on-transient A</i> | X | Courtship song | Visual behavior defective | Yes |
| <i>ovarian tumor</i> | X | Female courtship defective | Recessive female sterile, ovariole, ovary, egg chamber, germ-line cyst formation | Yes |
| <i>period</i> | X | Song defective | Circadian rhythm, locomotion rhythm | Yes |
| <i>Shaker</i> | X | Courtship behavior | Potassium ion transport, neurophysiology, gustation, hyperactive, larval muscle fiber, photoreceptor cell, neuromuscular junction | Yes |
| <i>tan</i> | X | Courtship behavior | | No |
| <i>tapered</i> | X | Courtship behavior | Wing, wing vein, male sterility, reduced female fertility | Yes |
| <i>technical knockout</i> | X | Male courtship defective | Female developmental rate defective and chemical sensitive, female lethal | Yes |
| <i>yellow</i> | X | Courtship behavior | Coloration, larval/pupal development | Yes |
| <i>Accessory gland-specific peptide 26Ab</i> | 2 | Oviposition, postmating behavior | | No |
| <i>Accessory gland-specific peptide 29AB</i> | 2 | Postmating behavior | Physiological processes, sperm displacement | Yes |
| <i>Accessory gland-specific peptide 32CD</i> | 2 | Negative regulation of female receptivity, postmating | | No |
| <i>Accessory gland-specific peptide 33A</i> | 2 | Negative regulation of female receptivity, postmating | | No |
| <i>Accessory gland-specific peptide 53Ea</i> | 2 | Postmating behavior | Sperm displacement, physiological processes | Yes |
| <i>aristaless</i> | 2 | Female mating defective | Specific RNA polymerase II transcription factor activity, proximal/distal pattern formation, imaginal disc, eye, leg, pleurum | Yes |
| <i>beethoven</i> | 2 | Male courtship defective | Adult antennal nerve | Yes |
| <i>chaste</i> | 2 | Female receptivity | | No |
| <i>croaker</i> | 2 | Courtship behavior defective, song defective | | No |
| <i>cuckold</i> | 2 | Courtship | Lethal | Yes |

Table 2 (Continued)

| Gene ^a | Location | Sexually selected phenotypes | Additional phenotypes | Pleiotropic |
|--|----------|---|---|-------------|
| <i>dissatisfaction</i> | 2 | Poor sex discrimination, reduced female receptivity | Slow copulation, no voluntary oviposition | Yes |
| <i>flamenco</i> | 2 | Courtship behavior | Accumulate many nuclear viruslike particles, cytoplasmic dense particles, and cisternae filled with fibrous material | Yes |
| <i>freeze</i> | 2 | Male courtship defective | Eye, abdominal tergite | Yes |
| <i>gate</i> | 2 | Male courtship defective | Eclosion rhythm | Yes |
| <i>gomdanji</i> | 2 | Courtship behavior | Learning, calcium ion binding, calcium-mediated signaling | Yes |
| <i>homer</i> | 2 | Courtship behavior | Locomotion, olfaction | Yes |
| <i>lingerer</i> | 2 | Copulation | | No |
| <i>P-element somatic inhibitor</i> | 2 | Male courtship and mating defective | mRNA processing, spermatid, testis, recessive larval lethal, male sterile | Yes |
| <i>Pox neuro</i> | 2 | Male courtship behavior | Antennae, leg, sensory organ, wing, eye | Yes |
| <i>protein ejaculatory bulb</i> | 2 | Postmating behavior | | No |
| <i>quick-to-court</i> | 2 | Male courtship defective | | No |
| <i>spinster</i> | 2 | Courtship behavior, female receptivity | Programmed cell death, abdominal segments, adult central nervous system | Yes |
| <i>tapered</i> | 2 | Courtship behavior | Wing, wing vein, male sterile, poor female fertile | Yes |
| <i>timeless</i> | 2 | Mating behavior | Regulation of sleep, protein binding, Malpighian tubule, posterior adult hindgut, circadian rhythm, locomotor rhythm, reduced fertility | Yes |
| <i>Ubiquitin-conjugating-enzyme-47D</i> | 2 | Male courtship behavior | Ubiquitin cycle, ubiquitin conjugating enzyme activity | Yes |
| <i>à la voile et à la vapeur</i> | 3 | Male courtship defective | Taste, viability | Yes |
| <i>Accessory gland peptide 62F</i> | 3 | Postmating behavior | Determination of adult life span | Yes |
| <i>Accessory gland peptide 63F</i> | 3 | Postmating behavior | Physiological processes | Yes |
| <i>Accessory gland peptide 70A</i> | 3 | Negative regulation of female receptivity, oviposition, postmating behavior | | No |
| <i>Accessory gland-specific peptide 98AB</i> | 3 | Negative regulation of female receptivity, postmating | | No |
| <i>Acetylcholine esterase</i> | 3 | Courtship defective | Acetylcholinesterase activity, central nervous system, embryonic recessive lethal, optomotor behavior, visual behavior | Yes |
| <i>atonal</i> | 3 | Song defective | DNA binding, transcription factor activity, antenna, axon, eye, auditory defective | Yes |
| <i>doublesex</i> | 3 | Song defect | Sterility, abnormal yolk protein production | Yes |
| <i>Ductus ejaculatorius peptide 99B</i> | 3 | Female receptivity | Oviposition | Yes |
| <i>fruitless</i> | 3 | All aspects of male courtship | Lethality, chromosomal lesions, abnormal muscle of Lawrence | Yes |

Table 2 (Continued)

| Gene ^a | Location | Sexually selected phenotypes | Additional phenotypes | Pleiotropic |
|-------------------------------|----------|--------------------------------|---|-------------|
| <i>he's not interested</i> | 3 | Courtship behavior | | No |
| <i>male-specific RNA 57Da</i> | 3 | Postmating behavior | Oviposition | No |
| <i>male-specific RNA 57Db</i> | 3 | Postmating behavior | Oviposition | No |
| <i>male-specific RNA 57Dc</i> | 3 | Postmating behavior | Oviposition | No |
| <i>Monoplane</i> | 3 | Courtship defective | Sterility, poor viability, flightless | Yes |
| <i>nerd</i> | 3 | Mating behavior | Pheromone biosynthesis | Yes |
| <i>NMDA receptor 1</i> | 3 | Mating defective | N-methyl-D-aspartate selective glutamate receptor activity, semilethal | Yes |
| <i>pale</i> | 3 | Courtship behavior | Catecholamine metabolism, dopamine metabolism, adult cuticle, adult spiracle, mouth hooks | Yes |
| <i>slowpoke</i> | 3 | Song defect | Flight defect | Yes |
| <i>takeout</i> | 3 | Male courtship behavior | Circadian rhythm, viability | Yes |
| <i>transformer</i> | 3 | Indiscriminate sexual behavior | RNA splicing, sex determination, germ cell, female sterile, female genitalia, fusome, gonad, ovary, spectrosome | Yes |

Note: Phenotypes affected by mutation along with the known biological processes were used to estimate pleiotropy (see "Methods" in text).

^a References for genes can be obtained from FlyBase (<http://www.flybase.net>).

ually selected genes from being lost, regardless of their genomic location. Sexually selected genes are presumably not free to evolve in response to sexual selection alone because associated pleiotropic effects will also be under selection. As such, the chromosomal location and evolvability of sexually selected genes may be significantly influenced by pleiotropic effects.

Sexually selected traits are now widely acknowledged to be influenced by condition (Rowe and Houle 1996; Wilkinson and Taper 1999; Duckworth et al. 2001; Houle and Kondrashov 2002; Kotiaho 2002; Møller and Petrie 2002). Rowe and Houle (1996) provide a theoretical genetic mechanism for the maintenance of variation in sexually selected traits through the genic capture of condition dependence. The authors postulate that if many loci within the genome each make some contribution to fitness and/or condition, then variation in condition will always exist. Genetic variation in sexually selected traits will be maintained if the expression of these traits is condition dependent. Over time, a fraction of the genetic variation in condition will be captured by the established correlation between sexually selected trait and condition. This model includes pleiotropic effects when this correlation occurs within the same locus. The pleiotropic effects of the sexually selected genes in *D. melanogaster* surveyed in the current analysis include life-history traits that may influence condition and/or fitness. These include fertility (sterility), cell death, vision, development, life span, and viability (lethality; table 3). Interestingly, under Rowe and Houle's (1996) model, the sex chromosomes are not explicitly predicted as a preferred genomic location for the

underlying genes. Because I did not find significant sex linkage, my results support the predictions of Rowe and Houle (1996). Moreover, the important pleiotropic effects of sexually selected genes may provide evidence for the intralocus capture of genetic variation in condition.

The sex chromosomes have been recently implicated as a hot spot for intralocus conflict (Chippindale and Rice 2001; Gibson et al. 2002), a term often used to define when a gene has differential effects on the sexes (Rice and Chippindale 2001). For example, a gene that contributes to the elaboration of a male secondary sex trait (positive effects) can have negative effects, manifested through reduced fitness, when expressed in females. However, it is unclear whether all genes underlying sexually selected traits are under intralocus conflict. Therefore, results from studies of intralocus conflict may not represent a general trend for all sexually selected genes.

Using cDNA microarrays, genes with higher expression in males than in females were recently found to be underrepresented on the X chromosome whereas genes with higher expression in females were overrepresented on the X chromosome of *D. melanogaster* (Parisi et al. 2003). Ranz et al. (2003), in addition to confirming these results, found that approximately 50% of the genes on their microarray were differentially expressed between *D. melanogaster* and *Drosophila simulans*, suggesting evolutionary changes in gene expression between these related species. Although theory predicts that genes under sexual selection will be differentially expressed between the sexes and will be sex linked (Rice 1984), the results of Parisi et al. (2003) and Ranz et al. (2003) cannot separate the effects of sexual

Table 3: Sexually selected genes with pleiotropic effects influencing important life-history traits

| Pleiotropic effect | Gene/genes |
|-----------------------|---|
| Fertility (sterility) | <i>courtless, doublesex, dunce, Monoplane, NMDA receptor 1, ovarian tumor, P-element somatic inhibitor, takeout, tapered, technical knockout, timeless, transformer</i> |
| Development | <i>yellow, technical knockout, gate, transformer, Adar, ether a go-go, tapered, freeze, pale</i> |
| Life span | <i>Accessory gland peptide 62F, inactive</i> |
| Viability (lethality) | <i>Acetylcholine esterase, à la voile et à la vapeur, cuckold, fruitless, Monoplane, P-element somatic inhibitor</i> |
| Oviposition | <i>Accessory gland peptide 70A, dissatisfaction, Ductus ejaculatorius peptide 99B, male-specific RNA 57Da, male-specific RNA 57Db, male-specific RNA 57Dc</i> |
| Cell death | <i>spinster</i> |
| Vision | <i>no-on-transient A</i> |

selection from factors such as dosage compensation (which are known to result in higher expression of X loci in females than in males; see Baker et al. 1994). Moreover, these microarrays contain thousands of genes where presumably very few are under sexual selection. By contrast, the genes included in the current survey were chosen a priori based on known function (and presumed role in sexual selection) and were subsequently assessed for genomic location and pleiotropic effects.

Genomic data from *D. melanogaster* offer the best current database for the present analysis because sexual selection is well studied in this species and numerous genes have been characterized. However, a possible caveat must be raised. The majority of genes surveyed here were identified by mutagenesis; therefore, the contribution of each to the natural variation in sexually selected traits is unknown. It can then be argued that the genes surveyed here do not accurately represent the pattern of naturally varying sexually selected genes in nature. It is expected that some of these genes will be under purifying selection (i.e., no natural variation) and that others will harbor natural genetic variation. Interestingly, several quantitative trait locus mapping studies measuring the genomic location of natural genetic variation in sexually selected traits such as courtship song, competitive fitness, and reproductive success in *D. melanogaster* also found no bias for the sex chromosomes (Fry et al. 1998; Wayne et al. 2001; Gleason et al. 2002).

In contrast to previous work, this study provides evidence that sexually selected genes are not preferentially sex linked. Along with their role in sexual selection, most of

these genes were found to underlie additional traits (pleiotropic effects). Theoretical models predicting sex linkage of sexually selected genes do not include pleiotropic effects, thereby assuming sexual selection to be the sole evolutionary force acting on these genes. However, results from the current study suggest that these pleiotropic effects may have played a major role in shaping the evolution of these genes whereby sex linkage is not necessarily essential for sexually selected genes.

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