

# No Evidence for Heritability of Male Mating Latency or Copulation Duration across Social Environments in *Drosophila melanogaster*

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## Abstract

A key assumption underpinning major models of sexual selection is the expectation that male sexual attractiveness is heritable. Surprisingly, however, empirical tests of this assumption are relatively scarce. Here we use a paternal full-sib/half-sib breeding design to examine genetic and environmental variation in male mating latency (a proxy for sexual attractiveness) and copulation duration in a natural population of *Drosophila melanogaster*. As our experimental design also involved the manipulation of the social environment within each full-sibling family, we were able to further test for the presence of genotype-by-environment interactions (GEIs) in these traits, which have the potential to compromise mate choice for genetic benefits. Our experimental manipulation of the social environment revealed plastic expression of both traits; males exposed to a rival male during the sensitive period of adult sexual maturation exhibited shorter mating latencies and longer copulation durations than those who matured in isolation. However, we found no evidence for GEIs, and no significant additive genetic variation underlying these traits in either environment. These results undermine the notion that the evolution of female choice rests on covariance between female preference and male displays, an expectation that underpins indirect benefit models such as the good genes and sexy sons hypotheses. However, our results may also indicate depletion of genetic variance in these traits in the natural population studied, thus supporting the expectation that traits closely aligned with reproductive fitness can exhibit low levels of additive genetic variance.

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## Introduction

Sexual selection is often thought to favour the evolution of elaborate traits that provide honest signals of (usually male) genetic quality [1,2]. According to this idea, secondary sexual traits are expected to convey reliable information about genetic quality, thus generating a correlation between signal attractiveness and the genetic benefits of choice (production of offspring of high genetic quality) which underpin good genes explanations for the evolution of female preferences [1-3]. A correlation between attractiveness and female preferences is also expected, regardless of the relationship between attractiveness and genetic quality, as long as attractive males father attractive sons; choosy females can therefore obtain genetic benefits in the form of sexy sons [4-7]. Furthermore, if attractiveness is understood as the ability of males to

manipulate females into mating, models based on sexual conflict also predict that attractive males should father attractive sons [8]. Thus, a critical assumption of good genes, sexy sons, and sexual conflict models is that attractiveness should be heritable. Accordingly, sexual selection research has focused on estimating the heritability of male traits upon which female choice is based, and there is now ample support for the notion that these traits can harbour significant levels of additive genetic variation [1,9-12]. However, determining whether attractiveness itself (e.g. the probability that a male is chosen as a mate) is heritable has received far less empirical attention [13]. Ultimately we largely lack data to support this key assumption underlying models of sexual selection.

An important complication when assessing the genetic basis of sexual signals is that their expression can depend on the social and/or physical environment in which they are expressed

[14-16], such that the reliability of sexual signals in different mating contexts may be compromised [17,18]. Thus, a given genotype may express different phenotypes when expressed in different environments. Theory [19] and evidence [20-22] suggest that such genotype-by-environment interactions (GEIs) have the potential to disrupt the reliability of sexual signals in heterogeneous environments, thus potentially compromising mate choice for genetic benefits [17,19].

There is compelling evidence for the implication of indirect selection on female choice in *Drosophila* [13,23-28], and in several *Drosophila* species male sexual attractiveness – measured from mating latency – has been shown to be heritable [13,24,29]. However, these studies were conducted under homogeneous environmental conditions and therefore the influence of environmental variation on patterns of genetic variance in male mating latency is presently unknown. Yet phenotypic studies that have manipulated social environments have revealed considerable plasticity in courtship, copulation duration, and ejaculate size according to the presence/absence of rival males, which effectively manipulates the likelihood of both pre- and post-copulatory competition (i.e. mating success and sperm competition) [30-32]. A general pattern found across taxa is that males that encounter rival males either during or prior to mating respond by decreasing the time spent in courtship, increasing the length of time engaged in copulation, and increasing ejaculate size [33]. Nevertheless, it is currently unknown whether there is genetic variation underlying the plasticity of such traits in response to rival males, and thus the extent of GEIs arising from heterogeneity in social environments is presently unresolved.

In this study we determine the level of genetic variation in mating latency and copulation duration in *D. melanogaster* under varying social conditions. Specifically, we used a paternal full-sib/half-sib breeding design to generate pairs of full-sibling males that were exposed to one of two social environments; males were kept in isolation or in the company of another male during the period of early adulthood in which they reach sexual maturity. Our ensuing quantitative genetic analyses enabled us to test for genetic and environmental sources of variance underlying the expression of these traits, and for the presence of GEIs by assessing the interaction between social context and sire genotype.

## Methods

### Ethics statement

There are no ethical issues for this non-invasive study of an invertebrate species. This species is not an endangered or protected species. Adult flies were collected on private land with the permission of Howard Park and Houghton's wineries and then returned to the laboratory.

### Flies

Adult *Drosophila melanogaster* were collected in the Swan Valley region of Western Australia in May 2009 and maintained in the laboratory in mass populations of over 500 individuals with overlapping generations until the start of our experiment in January 2010. Populations were fed on a *Drosophila* medium

containing water, oats, sugar, agar and baker's yeast, with a sprinkling of dry yeast. Populations were kept on a 12:12hr light: dark cycle at 26°C. To generate the paternal full-sib/half-sib families for the experiment, we collected 100 virgin sires from the stock population and mated each of them to three virgin females at two-day intervals. Virgin flies were collected within 12 hours of eclosing from pupal cases and kept in separate vials for each sex for three days before the first mating. Each dam was removed from her respective laying vial three days after she had mated. From each full-sib family we collected four sons within 12 hours of eclosion and split them into two social environment treatments. These males (hereafter 'focal males') were either housed alone, or with an unrelated sexually mature (5-7 days old) 'rival' male from the stock population. All focal males were maintained in their respective social treatments for seven days during the sensitive period of adult sexual maturation. We then removed the rival male and transferred each focal male to a fresh vial with a 3-4 day old sexually mature virgin female from the stock population. We presented focal males individually to a single female to avoid potentially confounding effects of direct male-male competition on the traits measured (see below) [32]. Mating trials took place over four hours in the morning from first light, and four hours in the evening prior to dark. Siblings from both treatments were tested during the same time period to reduce variation due to time of day. All pairs of focal males were then observed continuously until copulation occurred, up to a maximum of 3 hours. A minimum of 80% of males mated within this time. For each successful copulation, we recorded the mating latency (time taken to initiate copulation) and copulation duration. Within the context of our experiment, we are interested only in the effect of perceived competition from rival males during the sexual maturation phase, rather than the effects of specific individuals on focal male mating success per se (sensu 'interacting phenotypes' approach [34]) and so we did not measure these behaviours in rival males.

Our final analysis comprised data from 91 sires, 194 dams and 663 sons (mean 2.1 dams per sire; mean 3.4 sons per dam). The effects of social environment on genetic variation in male mating behaviour were examined with general linear mixed-effects models using Type III sums of squares in STATISTICA 8.0 [35]. Sire, and dam nested within sire, were included as random factors, and social environment as a fixed factor. Interactions involving random terms were also coded as random effects. We used the interaction between social environment and dam (nested within sire) as the error term for testing the significance of the sire-by-environment (i.e. GEI) interaction [36] and followed Satterthwaite's method of denominator synthesis to account for unequal sample sizes of offspring [37]. Copulation duration was normally distributed but mating latency deviated from normality and was log-transformed prior to evaluating the sire-by-social environment interactions. We removed data for two sons as they alone generated a significant sire-by-social environment interaction for copulation duration. When we re-ran the analysis with these two males excluded from the dataset, the interaction term was no longer significant (see Results).

To estimate genetic parameters (including heritabilities) of the behavioural traits we conducted separate analyses within each social environment treatment group. For hypothesis testing, we used a mixed-model nested ANOVA with Type III sums of squares. Variance component estimation and subsequent analyses of genetic variation were conducted using restricted maximum-likelihood (REML) in a mixed-model fitted with the lmer function (package lme4 [38]; in R 3.0.0 [39]). We used untransformed variables to analyse genetic variation [40]. We calculated narrow-sense heritabilities due to sires, heritabilities due to dams, and the genotypic (mean) estimate of heritability [41]. We calculated evolvability measures ( $I_A$  and the coefficient of additive genetic variation,  $CV_A$ ) [40,42,43] and coefficients of phenotypic and residual variation ( $CV_P$  and  $CV_R$ , respectively) following methods described in [40], where  $CV_A = \sqrt{V_A} / \bar{X}$ . Finally, we calculated standard errors for heritabilities, variance components and evolvability estimates using the jack-knife procedure [41,44].

## Results and Discussion

### GEIs and sexual selection

Males exposed to a rival male during sexual maturity exhibited significantly shorter mating latencies than those who matured in isolation (mean  $\pm$  SE mating latencies: exposed to a rival male  $46.53 \pm 2.25$  min.,  $N = 341$ ; in isolation =  $62.45 \pm 2.74$  min.,  $N = 322$ ; see Table 1 for supporting statistics and Figure 1). Furthermore, males exposed to a rival male had significantly longer copulation durations than males who matured in isolation (mean  $\pm$  SE copulation duration: exposed to a rival male  $19.97 \pm 0.24$  min.,  $N = 341$ ; in isolation =  $17.69 \pm 0.25$  min.,  $N = 322$ ; Table 1 & Figure 2). Thus, we found evidence for plastic expression of these traits according to the social environment. Despite these treatment effects, we detected no significant sire effects, or additive genetic variation underlying any of the traits examined (discussed below). By contrast, we found significant variation attributable to dams for copulation duration (Table 1), but as this effect likely encompasses maternal and common environmental effects we do not interpret this finding further. There was no evidence for GEIs for either mating latency or copulation duration (see sire-by-social environment interaction; see Table 1).

We found a significant effect of the social environment on both mating latency and copulation duration. Males that were exposed to a rival male during the period between eclosion and sexual maturity were more attractive to females and significantly increased their copulation duration compared to those reared in isolation. This effect was evident despite all males being tested individually in a single male mating arena with a random virgin female. The phenomenon of increased copulation duration in response to a rival male in the rearing or mating arena has been reported across many insect taxa and has been most commonly interpreted as a behavioural response to increased risk of sperm competition [32,33,45,46]. In a recent study, Lize et al. [47] compared mating latency and copulation duration under 'single' or 'competitor' mating conditions in four species of *Drosophila*. They reported that increased copulation duration in response to a competitor was

**Table 1.** Mixed-model nested univariate ANOVAs for male mating latency and copulation duration.

Trait	Source of variance	d.f.	MS	d.f. (denominator)	F	p
Mating latency	Sire	90	0.195	38.78	1.049	.445
	Dam (Sire)	103	0.209	96.15	1.31	.091
	Social environment	1	3.214	101.19	23.23	<b>.000</b>
	Sire x Social environment	88	0.137	95.65	0.86	.759
	Dam (Sire) x Social environment	95	0.159	285	1.108	.261
Error	285	0.144				
Copulation duration	Sire	90	33.1	70.21	1.31	.119
	Dam (Sire)	103	23.6	96.89	1.92	<b>.000</b>
	Social environment	1	850.7	100.52	60.93	<b>.000</b>
	Sire x Social environment	88	13.9	96.07	1.13	.274
	Dam (Sire) x Social environment	95	12.3	285	0.67	.987
Error	285	18.2				

GxEs are tested via the Sire x Social environment interaction (where Dam (Sire) x Social environment term provides the error degrees of freedom). Results significant at alpha < .001 are in bold. Observed power for all tests > .84 (mating latency) and > .93 (copulation duration) (alpha = 0.05).

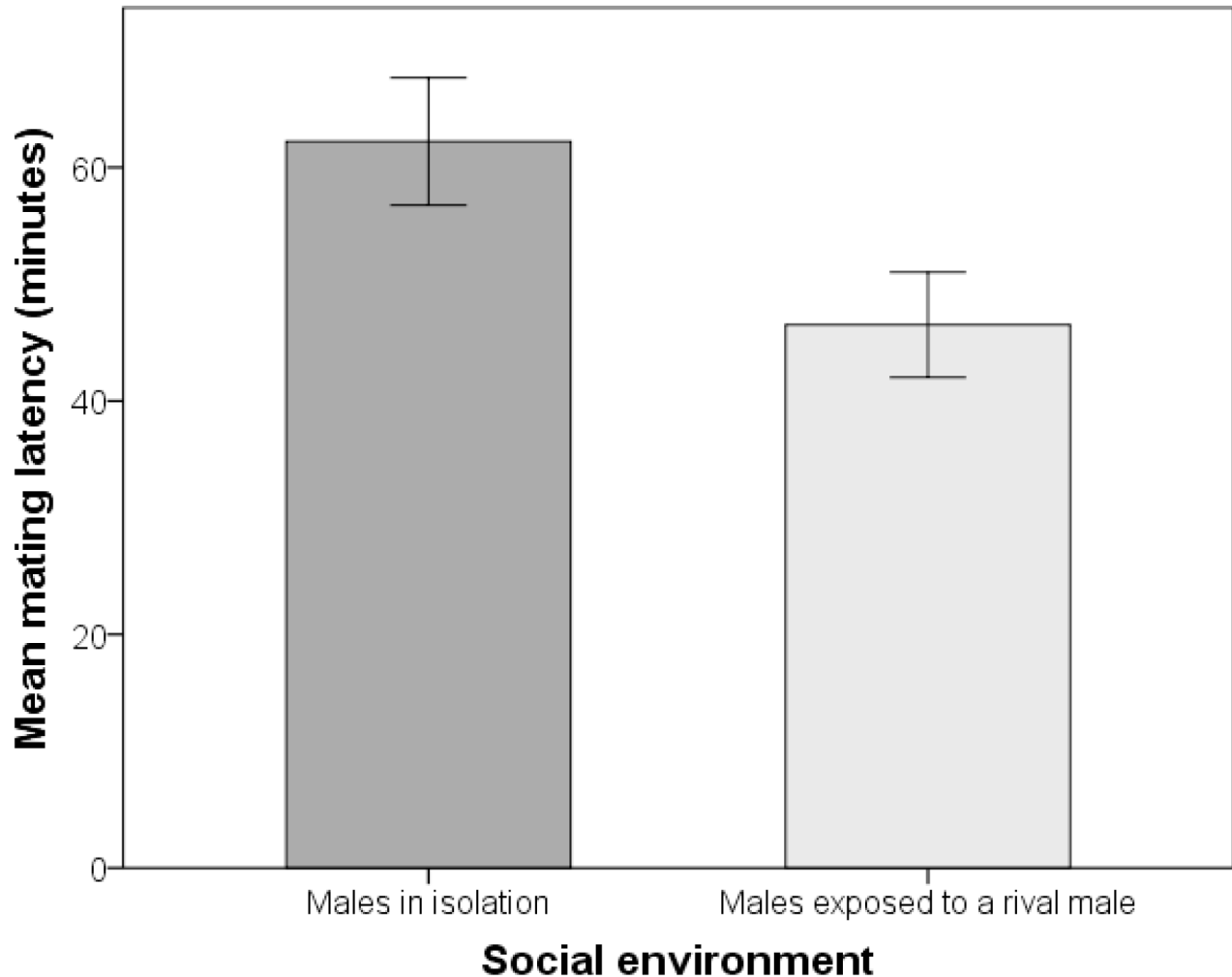
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a consistent response across the four species studied, but ruled out sperm competition as the selective factor underlying this response as two of the four species had extremely low levels of remating.

One way in which males might decrease their latency to mating is to increase the effort and time spent engaged in direct courtship behaviour towards females. Previous work in *D. melanogaster* and its sibling species, *D. simulans*, has shown that males spend considerable amounts of time engaged in courtship, even to the detriment of female survival [48-50]. Another possibility is that males decreased mating latency by increasing their production of cuticular hydrocarbons [51-54]. Consistent with this idea, in Australian field crickets, *Teleogryllus oceanicus*, subordinate males have been shown to compensate for their lower status by up-regulating their investment in short-range cuticular hydrocarbons, which directly increases their mating success [55,56].

### Genetic variation in mating latency and copulation duration

We partitioned total phenotypic variance into its genetic and environmental components and estimated the heritability and evolvability of mating latency and copulation duration. We found no significant additive genetic variation underlying mating latency or copulation duration in either treatment (Tables 2 and 3). The sire and dam estimates of heritability for the two traits analysed were not significantly different from each other, and genotypic estimates were generally moderate in magnitude although not significantly different from zero. These results may seem surprising given that previous studies have found



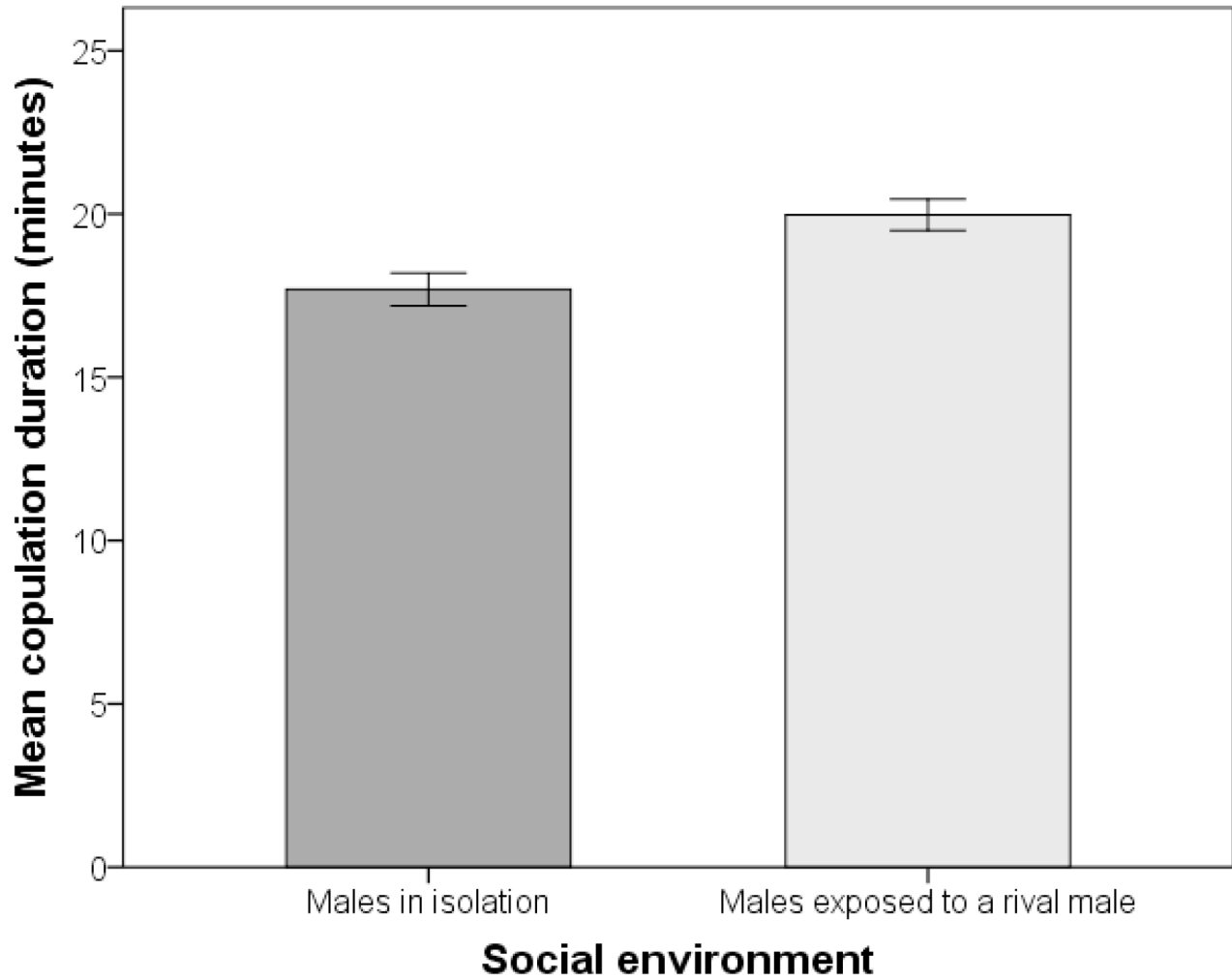
**Figure 1. Males decrease their mating latency after being exposed to a rival male.** Mean  $\pm$  SE of mating latency of males in two different social environments.

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significant levels of genetic variation for these traits in this and in other species of *Drosophila* [13,29]. Nevertheless, Hoffman [29] points out that in *Drosophila*, mating speed and courtship behaviour can exhibit either low or undetectable heritabilities due to “single event testing”, where the traits in focal individuals are measured just once rather than repeatedly. However, in their meta-analysis of repeatability of animal behaviours, Bell et al. [57] report no relationship between repeatability and the number of times behaviour such as courtship was measured.

The problem of a potential underestimation of heritabilities may be further compounded when we consider that in order to measure focal male mating latency and copulation duration we used a random sample of females with which to measure these traits. Thus, any genetic or phenotypic variance in female preferences or female condition as well as sampling error surrounding the choice of females may inflate estimates of phenotypic variance in the traits investigated in the focal males, hence leading to an underestimation of heritability (see [58,59]

for a full exposition of a similar problem in the case of estimating the heritability of sperm competitiveness). Whether this ‘noise’ is strong enough to mask the detection of significant heritability in mating latency and copulation duration in the population studied warrants further investigation, but we found consistently low levels of additive genetic variation, including mean-standardized measures of evolvability, which unlike heritability measures are independent of phenotypic variance. We also note that the sample size in our study (around 90 sire families) is similar to, or even larger than, those employed in other studies using the full-sib/half-sib design that either reported significant additive genetic variance for these traits [13,24] or were calculated as sufficient to detect significant heritability of 0.2 [60]. Finally, as our study characterises patterns of genetic variance in a natural population of *D. melanogaster*, our ensuing estimates of heritability and coefficients of additive genetic variance may reflect both high environmental variance and low additive genetic variance in



**Figure 2. Males increase their copulation duration after being exposed to a rival male.** Mean  $\pm$  SE of copulation duration of males in two different social environments.

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**Table 2. Phenotypic and genetic variation in mating latency (minutes) in males exposed to different social environments.**

Social environment	Mean (SE)	V <sub>A</sub> (SE)	V <sub>P</sub> (SE)	h <sup>2</sup> <sub>sire</sub> (SE)	h <sup>2</sup> <sub>dam</sub> (SE)	h <sup>2</sup> <sub>genotypic</sub> (SE)	I <sub>A</sub> (SE)	CV <sub>A</sub> (SE)	CV <sub>P</sub> (SE)	CV <sub>R</sub> (SE)
<i>Isolation</i>	62.45 (2.74)	2.81x10 <sup>-11</sup> (3.72 x10 <sup>-7</sup> )	2414.21 (236.92)	1.16x10 <sup>-14</sup> (1.53 x10 <sup>-10</sup> )	0.63 (0.38)	0.31 (0.19)	7.21x10 <sup>-15</sup> (9.51 x10 <sup>-11</sup> )	8.49x10 <sup>-8</sup> (9.65 x10 <sup>-6</sup> )	0.78 (0.03)	0.78 (0.03)
<i>With rival male</i>	46.53 (2.25)	75.93 (273.30)	1788.42 (234.35)	0.04 (0.16)	0.58 (0.57)	0.31 (0.29)	0.035 (0.126)	0.18 (0.45)	0.90 (0.04)	0.87 (0.08)

Coefficients of additive, phenotypic and residual variation are calculated without a 100 multiplier. Standard errors (SE) of genetic statistics were obtained by jackknifing across sires. Sample sizes for the "isolation" environment: n sires = 90, n dams = 187, n sons = 322; sample sizes for the "with rival male" environment: n sires = 90, n dams = 191, n sons = 341. Significance values are not shown as all  $p > 0.05$ .

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natural populations compared to their lab-adapted counterparts. Conner et al. [61], for example, reported that in wild radish plants, estimates of heritability and additive genetic variance in floral traits were significantly lower in field samples

compared to those coming from the laboratory. A possibility in our case is that genetic variance in male mating behaviour may have been eroded by selection in the natural population, but future studies are needed to further investigate this possibility.

**Table 3.** Phenotypic and genetic variation in copulation duration (minutes) in males exposed to different social environments.

Social environment	Mean (SE)	V <sub>A</sub> (SE)	V <sub>P</sub> (SE)	h <sup>2</sup> <sub>sire</sub> (SE)	h <sup>2</sup> <sub>dam</sub> (SE)	h <sup>2</sup> <sub>genotypic</sub> (SE)	I <sub>A</sub> (SE)	CV <sub>A</sub> (SE)	CV <sub>P</sub> (SE)	CV <sub>R</sub> (SE)
<i>Isolation</i>	17.69 (0.25)	3.91 (5.14)	20.30 (1.99)	0.19 (0.27)	0.38 (0.46)	0.29 (0.16)	0.012 (0.017)	0.11 (0.07)	0.25 (0.01)	0.23 (0.05)
<i>With rival male</i>	19.97 (0.24)	2.41x10 <sup>-9</sup> (1.58 x10 <sup>-8</sup> )	19.58 (2.01)	1.23x10 <sup>-10</sup> (8.15 x10 <sup>-10</sup> )	0.00 (0.82)	6.17x10 <sup>-11</sup> (0.41)	6.06x10 <sup>-12</sup> (3.96 x10 <sup>-11</sup> )	2.46x10 <sup>-6</sup> (7.15 x10 <sup>-6</sup> )	0.22 (0.01)	0.22 (0.01)

Coefficients of additive, phenotypic and residual variation are calculated without a 100 multiplier. Standard errors (SE) of genetic statistics were obtained by jackknifing across sires. Sample sizes for the "isolation" environment: n sires = 90, n dams = 187, n sons = 322; sample sizes for the "with rival male" environment: n sires = 90, n dams = 191, n sons = 341. Significance values are not shown as all  $p > 0.05$ .

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