Males Influence Maternal Effects That Promote Sexual Selection: A Quantitative Genetic Experiment with Dung Beetles *Onthophagus taurus*

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ABSTRACT: Recently, doubt has been cast on studies supporting good genes sexual selection by the suggestion that observed genetic benefits for offspring may be confounded by differential maternal allocation. In traditional analyses, observed genetic sire effects on offspring phenotype may result from females allocating more resources to the offspring of attractive males. However, maternal effects such as differential allocation may represent a mechanism promoting genetic sire effects, rather than an alternative to them. Here we report results from an experiment on the horned dung beetle *Onthophagus taurus*, in which we directly compare genetic sire effects with maternal effects that are dependent on sire phenotype. We found strong evidence that mothers provide more resources to offspring when mated with large-horned males. There were significant heritabilities for both horn length and body size, but when differential maternal effects were controlled, the observed estimates of genetic variance were greatly reduced. Our experiment provides evidence that differential maternal effects may amplify genetic effects on offspring traits that are closely related to fitness. Thus, our results may partly explain the relatively high coefficients of additive genetic variation observed in fitness-related traits and provide empirical support for the theoretical argument that maternal effects can play an important role in evolution.

Keywords: differential allocation, maternal effects, indirect genetic effects, heritability, coefficient of additive genetic variance.

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The differential allocation hypothesis predicts that females can increase their reproductive success by investing in reproduction depending on the attractiveness of their current mate (Burley 1986). The hypothesis has two underlying assumptions. The first assumption is that a basic life-history trade-off exists so that an increased allocation of resources into current reproduction must compromise resource allocation into future reproductive events (Williams 1966; Trivers 1972; Reznick 1985; Burley 1986). The second assumption is that resource allocation into reproduction with an attractive mate must be greater than the average resource allocation across all reproductive events (Burley 1986). Burley's original work examined both of these assumptions (Burley 1985, 1986, 1988). However, there are few other studies that have (Wedell 1996; Reyer et al. 1999), and most of the more recent studies that claim evidence for the differential allocation hypothesis fail to examine the life-history trade-off required by the hypothesis (Petrie and Williams 1993; Gil et al. 1999; Cunningham and Russell 2000; Kolm 2001). If increased resource allocation in current reproduction does not come at a cost to the female, support for the differential allocation hypothesis is equivocal. Effects of male phenotype on female reproductive performance can arise from alternative sources, including male effects such as paternal investment (Wedell 1996; Hunt and Simmons 2000; Qvarnström and Price 2001), which may enhance offspring performance without adversely affecting future female reproductive performance.

Because sire effects on offspring phenotype may result from allocation of more resources to the offspring of attractive mates, it has been suggested that they may confound genetic effects observed in traditional analyses (Gil et al. 1999; Cunningham and Russell 2000). However, rather than confounding genetic sire effects, differences in the allocation of resources to offspring may in fact promote sexual selection via an amplification of the genetic sire

effects (Sheldon 2000). To be able to amplify genetic effects, there must be some underlying genetic variance in phenotypic traits to start with. This cannot be seen as a problem since virtually every trait will show some genetic variation (Houle 1992; Pomiankowski and Møller 1995; Lynch and Walsh 1998). The second requirement for amplifying genetic sire effects is that the traits under study must be directionally affected by resource allocation. This should not be seen as a problem either because most traits are likely to show positive phenotypic condition dependence to some extent (Nur and Hasson 1984; Andersson 1986; Rowe and Houle 1996; Kotiaho 2000; Kotiaho et al. 2001). Thus, any differences in the allocation of resources to offspring that are correlated with sire phenotype have the potential to amplify genetic effects of sires on their offspring (Sheldon 2000). Moreover, if there is genetic variance for these phenotype-dependent patterns of resource allocation, they will act as indirect genetic effects that can amplify evolutionary responses to selection (Wolf et al. 1998).

Male *Onthophagus taurus* exhibit dimorphic horn morphology: minor males have practically no horns, while major males have large, well-developed horns (Hunt and Simmons 1998*a*, 2000). In general, males with large horns are competitively superior (Emlen 1997*a*; Moczek and Emlen 2000) and sire the majority of offspring produced (Hunt and Simmons 2001). After mating, females construct brood masses from dung and lay a single egg into each brood mass. The brood mass constitutes all available resources for the larva during its development and is a major determinant of adult body size (Emlen 1994, 1997*b*; Hunt and Simmons 1997, 2000). In several dung beetle species, major males assist females in brood mass construction (Cook 1988; Sowig 1996; Hunt and Simmons 1998*b*, 2000), and in *O. taurus* this results in the production of brood masses that are 48% larger than those produced by a female working alone (Hunt and Simmons 1998*b*, 2000). In order to directly compare additive genetic sire effects and differential maternal effects on the number and size of offspring, we adopted an experimental design where we prevented direct paternal contribution to brood masses.

Material and Methods

Female Reproductive Performance

To study the effect of sire and dam on the number of brood masses produced, we housed each of 106 field-collected sires with four F1 laboratory-reared virgin dams. We used pronotum width as a measure of body size, and dams did not differ in body size across sires (mean: $F = 0.11$, df $= 106, 321$, $P = 1.000$; variance $F = 0.36$, df $= 106, 321$, $P = 1.000$). After 5 d, dams were established in individual breeding chambers (PVC piping, 30 cm in length and 9 cm in diameter, three-quarters filled with moist sand and topped with 250 mL of fresh cow dung) without the sires to construct brood masses. Brood masses were collected, and the sand and dung were replaced with a fresh supply every 7 d until the death of the dam. This provided us with the longevity of the dams and the lifetime number of offspring produced for each dam and sire. To study the effects of sire and dam on the weight of the brood masses, we randomly selected 50 sires with three dams each from the above set of 106 sires. From these 150 dams, we weighed the brood masses produced during the first 7-d period. Brood masses ($n = 2{,}904$) were weighed individually to the nearest 0.01 g and placed in individual sand-filled chambers. After emergence, offspring were preserved in alcohol for subsequent measurements. Horn length of the sires and male offspring were measured to the nearest 0.03 mm using a binocular microscope, and pronotum width was measured to the nearest 0.02 mm with digital calipers (table 1).

Quantitative Genetic Analysis

In order to examine potentially amplifying effects of maternal allocation on genetic sire effects, we analyzed our data using three different methods. First, we used a standard mean offspring on single-parent regression analysis to estimate the heritability, coefficient of additive genetic variance (CV_A), and coefficient of residual variance (CV_R) of horn length and pronotum width (Falconer and Mackay 1996; Roff 1997; Lynch and Walsh 1998). This analysis will incorporate any differential maternal effects into genetic sire effects. Single-parent regressions were used because females do not possess horns and because the variance in pronotum width is significantly different between males and females (Levene statistic $= 16.34$, df $= 1, 2{,}523$, $P <$

Table 1: Basic statistics about the populations used

Trait	Mean	SD	*n*
Pronotum width of sires	4.461	.815	50
Horn length of sires	1.004	1.194	50
Pronotum width of dams	5.373	.132	150
Pronotum width of sons	5.345	.242	1,211
Horn length of sons	2.615	.975	1,211
Pronotum width of daughters	5.260	.260	1,314
Mean of mean pronotum width of sons for each sire	5.341	.096	50
Mean of mean horn length of sons for each sire	2.602	.406	50
Mean of mean pronotum width of daughters for each dam	5.256	.131	150

.001). We used the mean son horn length and mean son or daughter pronotum widths averaged across the three dams of each sire and regressed these separately on sire horn length or pronotum width. Heritability estimates and their standard errors were derived from the least squares regression coefficients by multiplying both the slope and its standard error by two (Falconer and Mackay 1996; Roff 1997; Lynch and Walsh 1998). Each of the regressions was weighted by the respective number of offspring for each sire to adjust for the variance in error arising from uneven sample sizes. Additive genetic variance for each trait was estimated as twice the covariance between offspring and parent.

The second way we analyzed our data was by using a multiple linear regression. We entered the brood mass weight (ln transformed) as a second independent variable into the offspring on sire regression analyses to partial out its effects on genetic sire effects. Heritabilities, CV_A's, and CV_R's were calculated as for the simple linear regressions. This analysis should thereby partial out some of the effects of differential maternal allocation to the brood mass from any genetic sire effects. However, the analysis is unable to accurately account for maternal effects because it uses means of offspring trait values and the means of brood mass weights for each family. Nevertheless, we present this analysis because it facilitates a direct comparison of genetic estimates obtained from a single technique.

Finally, since our data come from a traditional half-sib breeding design, it is compatible with a mixed-model nested ANCOVA. This analysis is in some ways superior because all of the maternal effects will appear as dam components, leaving the sire components free of any potential differential allocation effects. We therefore performed such an analysis including a covariate (ln-transformed brood mass weight). However, since the methodology of the analysis differs from regression techniques, comparison of the magnitude of the heritabilities, CV_A's, and CV_R's may be slightly compromised because part of the differences in the estimates may arise due to differences in estimation methodology. This is why the above multiple linear regression analysis was also retained.

Table 2: ANCOVA on number of brood masses

Source	SS	df	MS	*F*	*P*	Eta^2
Longevity[a]	2,657.48	1	2,657.48	43.81	.000	.145
Sire	5,206.69	59	88.24	1.46	.026	.250
Sire × longevity	5,090.28	59	86.27	1.42	.034	.245
Error	15,651.87	258	60.66	…	…	…

Note: Eta^2 refers to the proportion of variance explained (it is the ratio of the between-groups sum of squares to the total sum of squares).

[a] Longevity of the dam is used as a covariate.

Table 3: Nested ANOVA for brood mass weight

Source	SS	df	MS	*F*	*P*	Eta^2
Sire	227.463	49	4.642[a]	2.434	.000	.544
Dam(sire)	191.488	100	1.915	11.879	.000	.301
Error	443.923	2754	.161	…	…	…

Note: Eta^2 refers to the proportion of variance explained.

[a] To account for unequal sample sizes of offspring within sires, the error term for sires was calculated using Satterwaithe's approximation: $0.996\text{MS[dam(sire)]} + (4.394 \times 10^{-3})\text{MS(error)} = 1.908$.

Results

Female Reproductive Performance

The lifetime number of brood masses produced was independent of dam size ($r = -0.01$, $n = 424$, $P = .861$), as found in previous studies (Hunt and Simmons 2000). Dam longevity was also independent of dam size ($r = 0.04$, $n = 424$, $P = .403$). However, we found an effect of sire on dam longevity (ANOVA: $F = 1.30$, df = 105, 318, $P = .045$); sire size was positively related to dam survival (correlation between sire size and mean longevity of his four dams; $r = 0.21$, $n = 106$, $P = .033$). Because dam longevity was also positively related to the lifetime number of brood masses produced ($r = 0.42$, $n = 424$, $P < .001$), we controlled for longevity in all further analyses of the number of brood masses. Interestingly, we found a significant sire by female longevity interaction effect on the lifetime number of brood masses produced by the female, explaining 24.5% of the total variance (table 2). This interaction indicates that the relationship between female longevity and lifetime number of brood masses produced is in fact dependent on the sire with which the female mated. This dependence can at least partly be explained by differences in sire horn length and body size; sire horn length and body size were both positively correlated with the mean lifetime number of brood masses produced by his four dams (partial correlation controlling for mean dam longevity; $r = 0.23$, df = 103, $P = .021$ and $r = 0.24$, df = 103, $P = .015$, respectively).

There was a strong dam effect on the weight of brood masses (table 3), with dam size being positively correlated with the mean weight of the brood masses the dam produced ($r = 0.38$, $n = 150$, $P < .001$). What was surprising, however, is that in the absence of direct paternal provisioning, there was also a strong effect of the sire on brood mass weight, explaining 54.4% of the total variation (table 3). Both sire horn length and sire size were positively related to the mean brood mass weight produced by his dams ($r_S = 0.30$, $n = 50$, $P = .036$ and $r = 0.32$, $n = 50$, $P = .025$, respectively). Brood mass weight increased by an average of 10% across the size range of sires used in our experiment (fig. 1). These results indicate that fe-

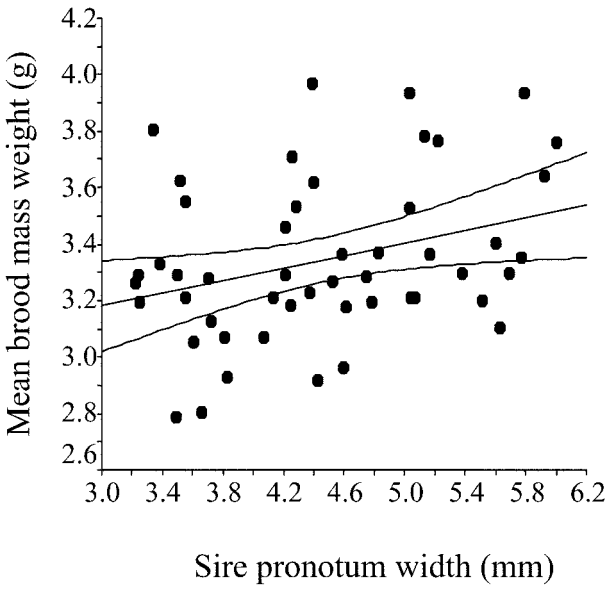

Figure 1: Relationship between mean brood mass weight for each sire and sire pronotum width. The slope is given by the equation $y = 2.85 + 1.12x$. Outer lines represent the 95% confidence intervals for the slope.

males differentially provision larger brood masses for sires that have long horns and large body size.

Size of brood masses had a significant positive effect on the body size of sons and daughters that emerged (correlation between mean brood mass weight and mean offspring body size for each dam; $r = 0.493$, $n = 150$, $P < .001$ and $r = 0.367$, $n = 150$, $P < .001$, respectively). There was also a clear effect of brood mass weight on the horn length of sons ($r = 0.456$, $n = 150$, $P < .001$).

Quantitative Genetic Analysis

In the first stage, we estimated heritabilities for horn length and pronotum width using mean offspring on father regressions (Falconer and Mackay 1996; Roff 1997; Lynch and Walsh 1998). The heritability estimate ($h^2 \pm$ SE) for horn length was significantly greater than 0 ($h^2 = 0.193 \pm 0.089$, $t = 2.16$, df = 48, $P = .036$). Similarly, the estimate of heritability for body size was significantly different from 0 for both sons ($h^2 = 0.094 \pm 0.031$, $t = 3.06$, df = 48, $P = .004$) and daughters ($h^2 = 0.065 \pm 0.022$, $t = 2.88$, df = 48, $P = .006$). In table 4, we have tabulated the heritabilities, CV_A's, and CV_R's. These results suggest that there is significant additive genetic variance for both horn length and body size in *Onthophagus taurus*.

However, because females provisioned brood masses depending on sire phenotype and brood mass influenced offspring phenotype, we suspect that the above estimates may be amplified by differential maternal effects. In an attempt to control for differential maternal effects and facilitate a comparison of heritabilities, CV_A's, and CV_R's, we ran a multiple linear regression partialling out the effect of brood mass weight on offspring phenotype. Heritabilities and their standard errors, CV_A's, and CV_R's are tabulated in table 4. This analysis revealed that heritability estimates from simple linear parent-offspring regression were 20% to 35% amplified, and CV_A's were 10% to 20% amplified due to the differential maternal effects on brood mass weight (table 5). However, we note that the difference between the heritability estimates are not significant (based on overlapping SEs).

Finally, we performed a fully saturated mixed-model nested ANCOVA separately for each of the offspring traits. The highest-level interaction effect, between dam nested within sire and the covariate brood mass weight, on horn length of sons was marginally significant ($F = 1.22$, df = 98, 913, $P = .079$). Procedures for selecting models of covariance recommend that significance of interaction terms be set at a value of α greater than the traditional 0.05; for example, Hendrix et al. (1982) suggest a value as high as 0.20. Therefore, we considered this full model in addition to one in which the interaction was removed. When the interaction was retained, the second interaction between sire and brood mass weight on horn length also indicated a significant interaction ($F = 1.33$, df = 49, 913, $P = .065$). When we removed the least significant dam nested within sire by brood mass weight interaction and ran the model again, the significance of the sire by

Table 4: Heritabilities, coefficients of additive genetic variation, and coefficients of residual variation calculated from parent-offspring regression in parent-offspring multiple regression including brood mass weight and in mixed-model nested ANCOVA

	Simple linear regression			Multiple linear regression			Mixed-model nested ANCOVA		
Trait	$h^2 \pm$ SE	CV_A	CV_R	$h^2 \pm$ SE	CV_A	CV_R	$h^2 \pm$ SE	CV_A	CV_R
Pronotum width$_{\text{sire-son}}$	.094 ± .030	5.61	17.39	.066 ± .030	4.70	17.65	.159 ± .033	.82	4.45
Horn length$_{\text{sire-son}}$	.193 ± .089	52.20	106.81	.125 ± .086	41.98	111.23	.055 ± .012	3.82	37.11
Pronotum width$_{\text{sire-daughter}}$	.065 ± .022	4.67	17.66	.052 ± .024	4.17	17.78	.011 ± .003	.17	4.10

Table 5: Change in heritabilities, coefficients of additive genetic variation, and coefficients of residual variation

Trait	Change in $A - B$ (%)	Change in $A - C$ (%)
Heritability of son's pronotum width	−29.79	+69.15
Heritability of son's horn length	−35.23	−71.50
Heritability of daughter's pronotum width	−20.00	−83.08
CV_A of son's pronotum width	−16.22	−85.38
CV_A of son's horn length	−19.58	−92.68
CV_A of daughter's pronotum width	−10.71	−96.36
CV_R of son's pronotum width	+1.50	−74.41
CV_R of son's horn length	+4.14	−65.26
CV_R of daughter's pronotum width	+.68	−76.78

Note: Changes in heritabilities, coefficients of additive genetic variation, and coefficients of residual variation are calculated as the difference between analysis where maternal effects were not controlled for (table 4 simple linear regression) and when they were controlled with multiple regression (table 4 multiple linear regression; change in $A - B$ [%]) and between analysis where maternal effects were not controlled for and when they were controlled with mixed-model nested ANCOVA (table 4 mixed model nested ANCOVA; change $A - C$ [%]).

brood mass weight interaction became stronger ($F = 1.37$, df $= 49, 1{,}011$, $P = .050$). The reduced model is presented in table 6. Both models show that brood mass weight had a significant effect on the genetic sire effect on horn length of sons. That is, the observed differential maternal effects influenced the genetic sire effects. For horn length, the heritability estimate ($h^2 \pm$ SE) was 0.055 ± 0.012, and the coefficient of additive genetic variance was 3.82 (table 4). The estimate of heritability was 71% lower and the estimate of CV_A was over 90% lower than the estimates from simple parent-offspring regression (table 5). For son pronotum width, there were no significant interactions (dam nested within sire by brood mass weight [$F = 1.15$, df $= 92, 848$, $P = .172$] and sire by brood mass weight [$F = 0.78$, df $= 46, 940$, $P = .852$]), and the final analysis was performed without them (table 7). The heritability estimate for son pronotum width was 0.159 ± 0.033, and the coefficient of additive genetic variance was 0.82. Again, both of these estimates were greatly changed in comparison to the analysis where differential maternal effects were not accounted for (table 5). For pronotum width of female offspring, there were significant dam nested within sire by brood mass weight and sire by brood mass weight interactions (table 8), indicating that again differential maternal effects have significant amplifying effects on the genetic components of variance in daughter pronotum width. The heritability estimate was low at 0.011 but, nevertheless, significantly different from 0 (table 4). We note that, although significant, most of the heritabilities are rather low. For body size, this is partly due to relatively low additive genetic variance in sons and daughters (table 4). However, for horn length in sons, there is moderate additive genetic variance (table 4), as expected for a secondary sexual trait (Pomiankowski and Møller 1995), but the heritability estimate is low because of the high residual variance (table 4; Houle 1992).

Table 6: Mixed-model nested ANCOVA for horn length

Source	SS	df	MS	F	P	Eta^2
BMW	74.34	1	74.34	129.49	.000	.114
Sire	41.09	49	.84[a]	1.43	.029	.062
Dam(sire)	166.57	98	1.70	2.96	.000	.223
Sire × BMW	38.41	49	.78	1.37	.050	.062
Error	580.40	1,011	.574	…	…	…

Note: Eta^2 refers to the proportion of variance explained. BMW = brood mass weight (ln transformed).

[a] To account for unequal sample sizes of offspring within sires, the error term for sires was calculated using Satterwaithe's approximation: (1.027×10^{-2})MS[dam(sire)] + 0.990MS(error) = 0.586.

Discussion

The results of our study show that in *Onthophagus taurus*, females differentially provision their offspring depending on the phenotype of their mate. Results such as these have previously been interpreted as supporting the differential maternal allocation hypothesis (Burley 1986, 1988; Petrie and Williams 1993; Wedell 1996; Gil et al. 1999; Cunningham and Russell 2000; Sheldon 2000; Kolm 2001). The differential allocation hypothesis is firmly rooted in life-history theory in predicting that females paired with attractive males should have a reduced future reproduction because of their elevated parental investment (Burley 1986). In contrast to this prediction, our results show that females had an elevated life span and increased lifetime reproductive investment following mating with large, long-horned males. Accordingly, the differential maternal effect observed in our study cannot be taken as support for the differential allocation hypothesis (sensu Burley 1988). Our results illustrate the importance of measuring total lifetime

Table 7: Mixed-model nested ANCOVA for pronotum width of sons

Source	SS	df	MS	*F*	*P*	Eta^2
BMW	5.55	1	5.55	152.28	.000	.134
Sire	7.12	46	.16[a]	1.42	.077	.408
Dam(sire)	10.46	93	.11	3.09	.000	.226
Error	35.91	986	3.64×10^{-2}	…	…	…

Note: Eta^2 refers to the proportion of variance explained. BMW = brood mass weight (ln transformed).

[a] To account for unequal sample sizes of offspring within sires, the error term for sires was calculated using Satterwaithe's approximation: $0.971\text{MS[dam(sire)]} + (2.944 \times 10^{-2})\text{MS(error)} = 0.110$.

reproductive output in studies examining the differential allocation hypothesis.

We have shown elsewhere that, in general, an increase in maternal investment does come at a cost of reduced female life span (Hunt et al. 2002). Therefore, the fact that here large males affected both a greater female investment and increased life span is in accord with some form of phenotype-dependent male paternal contribution that more than alleviates the costs of increased reproductive investment by females. For example, paternal assistance in provisioning has been shown to reduce the longevity costs of female investment for this species (Hunt et al. 2002). In the experiments reported here, females were provisioning alone. Therefore, the most likely explanation for the differential maternal effect is that it represents an indirect sire effect mediated via seminal products.

The finding that *Drosophila* seminal products can be costly to females (Chapman et al. 1995) has dominated recent literature in this area. However, more taxonomically widespread among insects is a positive influence of male seminal products on female reproduction (Simmons 2001). In beetles, seminal products have been shown to be incorporated into female somatic tissue and developing eggs (Boucher and Huignard 1987; Rooney and Lewis 1999), and they have been found to increase longevity and the number and size of eggs produced during a female's life span (Fox 1993*a*, 1993*b*; Fox et al. 1995; Eady et al. 2000; Drnevich et al. 2001; Nilsson et al. 2002; Rooney and Lewis 2002). The same is true for Lepidoptera and Orthoptera (for review, see Simmons 2001). In general, the positive effects of female multiple mating in insects appear to outweigh any negative impacts on longevity (Arnqvist and Nilsson 2000). The positive effects of seminal products have been shown to vary in a dose-dependent manner (Simmons 2001); for example, large male bruchid beetles provide larger ejaculates with concomitantly greater benefits for females (Fox et al. 1995). Thus, we believe that our data for *O. taurus* are generally consistent with previous studies of insects that show how seminal products have a positive influence on female reproduction; males provide nutrients in their seminal fluids that females utilize in reproduction. In support of this conclusion, we have found significant additive genetic variance for ejaculate size in *O. taurus* (Simmons and Kotiaho 2002) that would account for the intrinsic differences between males in the reproductive performance of their mates. Furthermore, ejaculate size is positively correlated with male size (Simmons et al. 1999) so that larger males may induce greater longevity and lifetime reproductive success of females, as found here, via quantitative variation in the ejaculate.

Whatever the proximate cause of this differential maternal effect, it holds important implications for evolution. Considerable theoretical effort has focused on the role of indirect genetic effects in evolution (Mousseau and Fox 1998; Wolf et al. 1998). Traits that have little or no direct genetic basis themselves can change across generations if the environment that contributes to such traits has some genetic basis. Currently, there is little empirical evidence for such indirect genetic effects in evolution (Wolf et al. 1998; but see Hunt and Simmons 2002; Rauter and Moore 2002). Our results show that differential maternal effects can amplify heritability and levels of additive genetic variation for secondary sexual traits. Differential maternal effects could thus fuel a response to sexual selection on secondary sexual traits even when there is only low levels

Table 8: Mixed-model nested ANCOVA for pronotum width of daughters

Source	SS	df	MS	*F*	*P*	Eta^2
BMW	2.73	1	2.73	94.95	.000	.089
Sire	1.89	46	4.11×10^{-2}[a]	1.06	.398	.289
Dam(sire)	3.84	94	4.08×10^{-2}	1.42	.007	.120
Sire × BMW	1.88	46	4.09×10^{-2}	1.42	.036	.063
Dam(sire) × BMW	3.62	94	3.85×10^{-2}	1.34	.021	.114
Error	28.00	973	2.88×10^{-2}	…	…	…

Note: Eta^2 refers to the proportion of variance explained. BMW = brood mass weight (ln transformed).

[a] To account for unequal sample sizes of offspring within sires, the error term for sires was calculated using Satterwaithe's approximation: $0.847\text{MS[dam(sire)]} + 0.153\text{MS(error)} = 3.896 \times 10^{-2}$.

of additive genetic variance for these traits (Wolf et al. 1998; Qvarnström and Price 2001). Moreover, the finding that secondary sexual traits generally exhibit higher coefficients of additive genetic variation than naturally selected traits (Pomiankowski and Møller 1995) may be at least partly explained by the amplifying effects of differential maternal effects such as those reported here.

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