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Mendel’s law reveals fatal flaws in Bateman’s 1948 study of mating and fitness

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Bateman’s experimental study of *Drosophila melanogaster* produced conclusions that are now part of the bedrock premises of modern sexual selection. Today it is the most cited experimental study in sexual selection, and famous as the first experimental demonstration of sex differences in the relationship between number of mates and relative reproductive success. We repeated the experimental methodology of the original to evaluate its reliability. The results indicate that Bateman’s methodology of visible mutations to assign parentage and reproductive success to subject adults is significantly biased. When combined in offspring, the mutations decrease offspring survival, so that counts of mate number and reproductive success are mismeasured. Bateman’s method overestimates the number of subjects with no mates and underestimates the number with one or more mates for both sexes. Here we discuss why Bateman's paper is important and present additional analyses of data from our monogamy trials. Monogamy trials can inform inferences about the force of sexual selection in populations because in monogamy trials male-male competition and female choice are absent. Monogamy trials also would have provided Bateman with an a priori test of the fit of his data to Mendel’s laws, an unstated, but vital assumption of his methodology for assigning parentage from which he inferred the number of mates per individual subject and their reproductive success. Even under enforced monogamous mating, offspring frequencies of double mutant, single mutant, and no mutant offspring were significantly different from Mendelian expectations proving that Bateman’s method was inappropriate for answering the questions he posed. Double mutant offspring (those with a mutation from each parent) suffered significant inviability as did single mutant offspring whenever they inherited their mother’s marker but the wild-type allele at their father’s marker locus. These inviability effects produced two important inaccuracies in Bateman’s results and conclusions. (1) Some matings that actually occurred were invisible and (2) reproductive success of some mothers was underestimated. Both observations show that Bateman’s conclusions about sex differences in number of mates and reproductive success were unwarranted, based on biased observations. We speculate about why Bateman’s classic study remained without replication for so long, and we discuss why repetition almost 60 years after the original is still timely, necessary and critical to the scientific enterprise. We highlight overlooked alternative hypotheses to urge that modern tests of Bateman’s conclusions go beyond confirmatory studies to test alternative hypotheses to explain the relationship between mate number and reproductive success.


Keywords: fitness variances, number of mates, number of offspring, reproductive success, Mendel’s rules, *Drosophila melanogaster*, A.J. Bateman, genetic parentage tests

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impact on empirical and theoretical studies in modern sexual selection. Bateman's conclusions are now part of the bedrock premises of modern sexual selection and they are foundational ideas in the evolutionary study of sex differences. Bateman's results were that (1) male number of different mates (NM) was greater than female NM, (2) males had greater variation in number of adult offspring, i.e., “reproductive success” (RS) than females, and (3) because of the number of mates, which Bateman inferred from plots of RS against NM (now known as “Bateman gradients”). Bateman said that his data showed that sex difference in the variance in number of offspring was the sign (italicized in the original) of “intramale sexual selection” while the cause of selection among males was a “stronger correlation” in males between NM and RS. His conclusions were consistent with Darwin's discussion of the evolution of reproductive success (VRS), which is sometimes considered the only measure of sexual selection. Bateman's paper is part of the standard historical trajectory in the controversial paradigm of the origins of sex differences.

Why did We Replicate Bateman (1948)?

Given that replication is a pillar of the scientific method, the previous lack of strict repetition seemed odd to us. A few previous investigators had evaluated and questioned Bateman's methods; his results, the sweep of his conclusions and the implications others took from his study. In particular, we wondered if the obvious failure of data in Bateman's Table 4 (reproduced in Fig. 1) to match Mendel's expectations was a systematic problem associated with all of his populations. We wondered: Would a strict repetition of Bateman's methods calm our concerns and those of earlier critics?

We also wondered if other explanations might explain the key results as well as or better than sexual selection seemed to do, so we planned to test the results against the predictions of sexual selection and stochastic demography (i.e., chance variation in individual survival and/or chance encounters with potential mates). In addition, we were interested in following up the questions about the validity of Bateman's method that arose when we were reading his paper to ensure that our repetition was as faithful as possible to Bateman's methods. The most important results of the PI idea was its prediction that when PI is greater in males, males would be “the choosy sex” and females the indiscriminate sex.

After Trivers, Arnold seized on Bateman's ideas and called the results “principles”; truths to count on or at least important assumptions to test. In a nutshell, what Bateman did with the help of these authors was anchor within-sex variance in number of mates (V_NM) as the key predictor of variance in reproductive success (V_RS), which is sometimes considered the only measure of sexual selection.
Repetitions” using modern molecular genetic studies, of course, exist. However, these are not repetitions in the strict sense, but rather standalone tests of “Bateman’s principles.” Tang-Martinez16 has recently reviewed these studies and shown that while many studies confirm Bateman’s principles, others do not. Studies reporting results of Bateman’s principles to test the fairness of the markers (Figs. 2 and 3): Bateman did test if half the offspring could be assigned to mothers and half to fathers, and concluded that there was no statistical difference in the representation among offspring of the same family.

### What the Repetition Discovered

Our repetition appears to be unique in that we tried to replicate exactly Bateman’s methodology of parentage assignment as far as we could. We used the same mutant lines Bateman used. In our study, we cultured adult subjects as Bateman had done, so that each expressed a unique genetically-determined marker phenotype (Fig. 3A and B). Table S1 in our original report30 illustrates that each adult subject was genetically and phenotypically distinct; that is, (regardless of their sex) in each of our replicated populations, each adult carried a single allele at its marker locus, while having only wild-type alleles at all other subjects’ marker loci. We included the cultured adults in experimental populations in the same combinations of sexes, markers per sex, age of males and females and duration of the period during which mating could occur that Bateman reported (see Table S1 in ref. 30).

Following Bateman’s method closely, we counted the number of mates using only the offspring with a mutation from each parent, the double mutant offspring, M♂M♀. According to Bateman, M♂M♀ allowed an unbiased count of how many mates the subject adults had. Like Bateman we used the sum of the double mutant offspring plus the single mutant offspring (those with a marker mutation from one parent only) as an estimate of the number of offspring for each subject adult (see Fig. 1).

Like Bateman we did not watch the mating behavior of our subjects, so we had no better estimate of who mated with whom than Bateman did. Thus, the veracity of Bateman’s study and our replication turned on the “fairness” of the parental markers: Were they neutral with respect to Bateman’s goals of inferring number of mates and number of offspring for each subject? This question is one that modern forensic scientists and those studying genetic parentage also must do and do in fact address. If markers fail to fit Mendel’s rules or Hardy-Weinberg expectations or are otherwise non-neutral, modern geneticists exclude particular loci from use in their studies of forensics, kinship, and genetic parentage.31,32 Bateman-era geneticists would have used the simple expectations from a consideration of Mendelian principles to test the fairness of the markers.
Figure 2. For figure legend, see page 32.
Bateman did report that the numbers of offspring from $M^wM^w$ and $w^wM^w$ were about equal and his Table 4 (Fig. 1) data clearly significantly depart from Mendelian frequencies. Such a test of the frequency of adult markers in offspring is the decisive one required for demonstration that estimates of number of mates per individual and the $V_{NM}$ were unbiased, fairly representing who did and did not mate.

Assuming Mendelian inheritance of alleles when each parent is heterozygous dominant at unique loci (Fig. 1), there should be 25% of offspring with both parental mutations, 25% with the dominant allele at mother’s marker locus but a wild-type allele at father’s marker locus, 25% with the wild-type allele at mother’s marker locus and the dominant allele at father’s marker locus and 25% with the wild-type allele at each of their parents’ marker loci (Figs. 2 and 3). We generalize these types of offspring phenotypically as $M^wM^w$, $M^wM^d$ and $w^wM^d$, respectively. The frequency of $M^wM^d$ offspring was significantly lower than expected overall. 30 The fatal flaw that our repetition revealed is that the method miscounts the number of mates for each sex—key variables in Bateman’s study—to an unknown degree, because in Bateman’s study the only information about who mated with whom was from the phenotypes of the $M^wM^w$ offspring. Using Bateman’s method we coded some subjects as having zero mates, when they in fact had one or more mates, which was clear when we observed “zero mated subjects” whose phenotypes appeared in their single mutant—$M^wM^w$ and $w^wM^d$—offspring (Fig. 4). Thus the method of assigning parentage miscounted the number of subjects with 0, 1, 2, 3, 4 or 5 mates. The error in counts of zero mates was systematically larger for males than for females as Figure 4 shows. Thus counts of NM were inappropriate, and by necessity the estimates of $V_{NM}$ were inaccurate and perhaps misleading to an unknown extent.

The second flaw arose because significantly more single mutant offspring survived when they had their mother’s wild-type allele and their father’s mutant allele (that is, the $w^wM^d$s) than offspring with their mother’s marker allele and their father’s wild-type allele (the $M^wM^w$s), the sex differences in reproductive success were also biased, showing higher RS for fathers than for mothers. Although Bateman did report that the numbers of offspring from $M^wM^w$ and $w^wM^d$ were about equal and his Table 4 (Fig. 1) suggested statistical equality in RS for assigned parents, he did not report the frequency of $M^wM^d$s or the test for parental equality of RS in his entire experiment. Had he reported a test of observed frequencies of $M^wM^w$, $M^wM^d$, $w^wM^d$ and $w^wM^d$ against Mendel’s expectations of $M^wM^w$, $M^wM^d$, $w^wM^d$ and $w^wM^d$, he would have provided the information that
Figure 4. Subjects seemingly without mates had offspring, which is highly unlikely in a sexually reproducing diploid species. Reproductive success counted as the sum of $M^wM^w$ plus $M^wW^+$ for female subjects (A) and as $M^wM^w$ plus $W^+M^+$ for male subjects (B) against the number of mates counted from $M^wM^+$ offspring for females (A) and for males (B) exposes a biological impossibility. Bateman’s method overestimates the number of individuals with zero mates (21 subjects among females and 43 among males), thereby underestimating the number with one or more mates. The magnitude of error among male subjects (43 males out of a total N of 166 males) was greater than among female subjects (21 females out of a total N of 166 females), which would have falsely increased the $V_{NM}$ estimates of males relative to females. We use these plots to illustrate one of the most egregious errors in Bateman’s method: subjects who seemed to have no mates had offspring.

his contemporaries and modern readers needed to evaluate the reliability of his markers given his questions. Although it appeared to be “cutting edge” in its day, the methodology of Bateman’s study appears to have been fatally flawed.

Our replication showed that using Bateman’s method produced two observations that are biologically impossible or at least extremely unlikely. Figure 4 shows the first by examining inferences about NM and RS for the 166 female subjects (A) and 166 male subjects (B) in the replication. The x-axis is the number of mates for subjects counted from $M^wM^+$ offspring, the only offspring providing information about who mated with whom. The y-axis is reproductive success counted as the sum of $M^wM^w + M^wW^+$ (for female subjects, the mothers) or $\Sigma = M^wM^w + W^+M^+$ (for male subjects, the fathers). A single fact exposes the bias in Bateman’s method: Some individuals that we counted using Bateman’s method as having zero mates nevertheless, using Bateman’s method, had offspring (Fig. 4). The mismatch between information in double mutant and single mutant offspring was due in our experiment to significantly lower viability of $M^wM^+$ offspring. Remember that single mutant offspring inform questions about how many offspring each subject had, but they are silent about who mated with whom. Because of the inviability of double mutant offspring, some adults were scored as having zero mates, when in fact that had some unknown number of mates. The resultant miscount incorrectly increased the number of males in the class having zero mates, and the number with $\geq 1$ was consequently underestimated. Using the method Bateman used, Figure 4 shows that Bateman would have incorrectly assigned many more males than females to the zero class of number of mates, so that the effect would be to increase the male $V_{NM}$ relative to the effect on female $V_{NM}$. There is no way to know what effect the incorrect assignment of subjects to the zero mating class has on the under-estimation of the subjects in the classes with $\geq 1$ mate (Fig. 4).

The second biological impossibility that our replication revealed was that there was a systematic bias in counts of offspring with fathers vs. mothers. That is, using Bateman’s method we were able to assign genetic paternity significantly more often than genetic maternity, making it seem as though more offspring had fathers than mothers, verifying that there was something wrong with the methodology, just as we had earlier suspected. More paternal assignments occurred because the number of $w^+w^+$ offspring was greater than the number of $M^wM^+$ offspring. We tested for significance differences with a plot of the difference scores in the apparent number of offspring with fathers minus the apparent number with mothers (see Fig. 1 in ref. 30). Because the bias resulted in assigning more fathers than mothers as parents, the estimate of RS was greater for fathers than mothers, demonstrably because of mismeasurements derived from Bateman’s method: single mutant offspring who inherited the dominant allele from their mother’s marker locus plus a wild-type allele at their fathers had lower viability than offspring who inherited a dominant allele from their father’s marker locus but a wild-type allele from their mother’s marker locus. Systematic, methodological mismeasurement of number of mates and number of offspring leaves open to question Bateman’s conclusion that there is an enhanced effect of RS as a function of NM for males, but not females. Because of the bias in the methodology, our repetition could not address the main questions that Bateman set out to answer. If his
overall data, not just the data in his Table 4 (Fig. 1), were inadequate to his questions as our repetition suggests, his answers to his questions would be unreliable as well.

**M<sup>♂</sup>M<sup>♂</sup> Offspring from Monogamous Pairs also were Inviable**

A control that was available to Bateman, but that he did not report having done was to test the frequencies of M<sup>♂</sup>M<sup>♂</sup>, M<sup>♂</sup>w<sup>♂</sup>, w<sup>♀</sup>M<sup>♂</sup> and w<sup>♀</sup>w<sup>♂</sup> in trials of monogamous pairs representing each possible combination of parents. Such a test a priori would have informed Bateman of the fairness of the markers as unbiased indicators of parentage. Notably because sexual selection is absent in strict monogamy, it would also have provided a strong comparative test of the force of sexual selection in his populations.

We reported monogamy trials in our original report along with the tabled counts and frequencies of M<sup>♂</sup>M<sup>♂</sup>, M<sup>♂</sup>w<sup>♂</sup>, w<sup>♀</sup>M<sup>♂</sup> and w<sup>♀</sup>w<sup>♂</sup> for each combination of heterozygote dominant female and male (see Table S4 in ref. 30). We showed that even under monogamy that only four out of 25 of the pair combinations (each with five trials) had M<sup>♂</sup>M<sup>♂</sup> at or near 25% (see tabled data, Table S14 in ref. 30). The vast majority were under 25%

**Figure 5.** Frequencies of M<sup>♂</sup>M<sup>♂</sup>, M<sup>♂</sup>w<sup>♂</sup>, w<sup>♀</sup>M<sup>♂</sup> and w<sup>♀</sup>w<sup>♂</sup> offspring from monogamy trials (five sets for each parental marker combination). The column on the far left identifies the offspring types: White bars = M<sup>♂</sup>M<sup>♂</sup>, light gray bars = M<sup>♂</sup>w<sup>♂</sup>, dark gray bars = w<sup>♀</sup>M<sup>♂</sup> and black bars = w<sup>♀</sup>w<sup>♂</sup>.

In addition, had Bateman performed trials of this sort, he would have known that his methodology—at least with the mutations he used—was also inappropriate for evaluating reproductive success. Consider that counts of M<sup>♂</sup>M<sup>♂</sup> offspring are necessarily equal for mothers and fathers. However, counts of M<sup>♂</sup>w<sup>♂</sup> and w<sup>♀</sup>M<sup>♂</sup> may differ. The question then arises: Are counts of M<sup>♂</sup>M<sup>♂</sup> and w<sup>♀</sup>M<sup>♂</sup> significantly different from 50% of offspring with fathers and 50% with mothers? A simple way to answer this question is in Figure 6 that shows the frequency of difference scores for numbers of M<sup>♂</sup>w<sup>♂</sup> and w<sup>♀</sup>M<sup>♂</sup> in each set of the monogamy trials. Single mutant offspring reveal only one of
offspring were significantly less frequent than expected. If that also were true for Bateman’s original study, Bateman too would have overestimated the number of adults that failed to mate, underestimated the number who mated one or more times and perhaps may also have assigned percentage to more fathers than mothers, a biological impossibility in species in which offspring have both a mother and a father. If Bateman’s method was flawed as our repetition suggests it was, he had no valid evidence for sex differences in numbers of mates or reproductive success, much less for VNM and VRS. In keeping with Bateman’s method, because we did not watch behavior, we have no basis for claiming that males mated indiscriminately or that females were indifferent to mating more than once.

Critics may claim that the mutant lines we used may have undergone significant evolutionary change since Bateman’s day that affected the results. That, of course, is a possibility, yet the hypothesis that guided our research was the evidence in Bateman’s own study (Fig. 1) that double mutant offspring were significantly less frequent than expected. If that also were true for Bateman’s original study, Bateman too would have overestimated the number of adults that failed to mate, underestimated the number who mated one or more times and perhaps may also have assigned percentage to more fathers than mothers, a biological impossibility in species in which offspring have both a mother and a father. If Bateman’s method was flawed as our repetition suggests it was, he had no valid evidence for sex differences in numbers of mates or reproductive success, much less for VNM and VRS. In keeping with Bateman’s method, because we did not watch behavior, we have no basis for claiming that males mated indiscriminately or that females were indifferent to mating more than once.

What can the Replication Say about the Original Study?

What can we make of Bateman’s original study and his claims given our study? First and most important, if throughout his study double mutant offspring occurred in significantly lower frequencies than 25% as they did in the one population for which he displayed all the offspring phenotypes (Fig. 1), his entire study, not just one population, would have been biased. Our replication emphasizes the likelihood of that possibility.

The replicated results show that in all but one of our 46 populations M♂w♂ offspring were significantly less frequent than expected. If that also were true for Bateman’s original study, Bateman too would have overestimated the number of adults that failed to mate, underestimated the number who mated one or more times and perhaps may also have assigned percentage to more fathers than mothers, a biological impossibility in species in which offspring have both a mother and a father. If Bateman’s method was flawed as our repetition suggests it was, he had no valid evidence for sex differences in numbers of mates or reproductive success, much less for VNM and VRS. In keeping with Bateman’s method, because we did not watch behavior, we have no basis for claiming that males mated indiscriminately or that females were indifferent to mating more than once.

Critics may claim that the mutant lines we used may have undergone significant evolutionary change since Bateman’s day that affected the results. That, of course, is a possibility, yet the hypothesis that guided our research was the evidence in Bateman’s own study (Fig. 1) that double mutant offspring were significantly less frequent than Mendel’s expected frequency of ¼. Our
results are very like the results in the onlyit of data in Bateman's study (Fig. 1) that
allowed a test against Mendel's expecta-
tions. And, it remains the case that with-
out a time machine, it would be difficult
for anyone today to replicate Bateman’s
method. We think we did the best that
anyone could do by using the lines of flies
Bateman used that are still available today.

Thus, our repetition raises potentially
unanswerable questions about Bateman’s
original studies. Were the double mutant
offspring in his experiment as likely to die
as in the repetition and as frequently as
offspring in his experiment as likely to die
original studies. Were the double mutant
unanswerable questions about Bateman’s
used that are still available today.

Anyone could do by using the lines of flies
method. We think we did the best that
for anyone today to replicate Bateman’s
repetitions.

And, it remains the case that with-
the idea that Bateman’s results and conclu-
sions are so similar to status quo, domi-
inating world-views (competitive males,
dependent females) that pre-existing cul-
tural biases of readers may have damp-
ened skepticism and objectivity. Perhaps
lack of repetition is simply due to lack of
professional incentives such as funding for
repetitions.

Why does the Repetition
Challenge “the Paradigm”?  

It seems that the modern bedrock of sex-
ual selection may have been quicksand.
But does a single replication of an impor-
tant study invalidate an entire field? We
do not think so.

Yet in the company of other challenges
to the competitive male and discriminat-
ing female paradigm, the repetition gives
pause, particularly in light of the “prob-
lems with paradigms.” 7 …paradigms act as a “lingua franca” that facilitates commu-
nication among scientists. On the negative
side, paradigms can lead to simplification,
can blind us to phenomena that do not fit
the accepted world-view, can guide us to
accept hypotheses that are unfounded, and
can prevent us from considering alternative
hypotheses and explanations. In such cases
paradigms become dogma and have detri-
mental effects on the development of sci-
tific ideas” (p. 821). Perhaps we should
routine urge our students to challenge
paradigmatic material. Perhaps we need
new rules such as “if it’s intuitive, test it.”
Are we at a field asking the right ques-
tions? For example, could both males
and females in most species assess oth-
ers’ quality as mates before accepting or
rejecting potential mates,34 as happens in
D. pseudoobscura?:35 Could males and
females in all species be simultaneously
choosy and competitive?34,35 Might the
mechanisms of sexual selection act on
number of mates among males, but on
quality of mates among females?36 Or
more radically, might both females and
males enjoy significant fitness benefits
through mate assessment and adaptively
flexible mate choice based on their con-
stantly updated predictions of the viabil-
ity of potential offspring? 37 What exactly
does a Bateman gradient tell about
among-female competition over the qual-
ity of mates? What if female and male
reproductive competition is mediated in
one sex via within sex variation in qual-
ity of mates and in the other via within
sex variation in number of mates? Would
the metric of sexual selection then be the
same for females and males? If females
and males do compete over different
things, perhaps we need a new metric for
evaluating the effect on $V_{RS}$ of variance
in mate quality ($V_{MQ}$).

Questions about Modern Studies
of Bateman Gradients

A probable rejoinder from some readers
of our recent report is that the repeti-
tion fails to weaken Bateman’s principles,
because other studies have validated them
(see above for examples). Even in the face
of validation from other studies in other
species, the repetition does matter, because
it puts in a different, more interesting
light the published studies with results
inconsistent with Bateman’s conclusions,
and it demands that investigators with
results consistent with Bateman’s conclu-
sions take care of at least two imperative
remaining concerns: (1) Observation bias
and (2) failure to test alternative hypoth-
oses for fitness variances.

Genetic parentage studies allow infer-
ences about mating systems when copu-
lations and identification of individuals
are hard to observe. Some, but far from
most, modern investigators of genetic
parentage watch copulations and there-
fore have an independent estimate of the
possibility that this individual mated
with this and that potential mate. But,
copulations that actually occur may be
invisible to molecular genetic testing
even when genetic markers are neutral,
fit Hardy-Weinberg expectations, or have
low mutation frequencies, because sperm may be inviable or females might not use the sperm. Thus, investigators must watch behavior of individually identified subjects and simultaneously account for “detection bias” in the field\(^\text{[14,19]}\) and in the laboratory. It is rarely possible to identify all potential breeders in a wild-living population, something that challenges all genetic parentage studies in the field, particularly of organisms that fly, flies and birds being notable examples. Laboratory populations have walls around them; wild-living populations do not. But, in truth it is just plain hard to identify individual fruit flies in a jar without intrusive manipulations, which could bias subjects’ mate assessments, or contests among rivals for resources or mate access. Thus, it is a real possibility that observational biases are near ubiquitous in parentage studies on wild and captive organisms as well. We suggest caution as it is likely that estimates of fitness variates are often invisibly biased, with consequent effects on the \(V_{\text{nat}}\) that many consider the key mechanist of sexual selection.

Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

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