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Bateman (1948): pioneer in the measurement of sexual selection

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Darwin (1871) identified sexual selection as the process whereby the members of one sex, generally males, compete with one another for reproductive access to members of the other sex, generally females. He argued that sexual selection should be a much stronger evolutionary force in polygamous than in monogamous mating systems, and that, in the former, such selection would fall primarily on members of the male sex. Nearly 80 years later, in his classic fruit fly study, Bateman (1948) reported the *first* empirical demonstration that the cause of the sex difference in intensity of selection was variation among males in mate numbers. This seminal paper set the stage for much of the modern research into sexual selection.

Bateman's stated goal was to identify (p 352) 'a fundamental cause of intra-masculine selection, independent of mating system and probably inherent in the mechanics of sexual reproduction.' He presumed that this cause would reveal 'why it is a general law that the male is eager for any female, without discrimination, whereas the female chooses the male.' Bateman addressed the matter of sex differences not in terms of behavior, as Darwin did (with 'arduous' males and 'coy' females), but rather in terms of the intensity of intra-sexual selection acting within each sex. Bateman then interpreted his results in terms of sexual differences in morphology, including gamete size and tendencies to provide parental care. Thus, Bateman's contribution simultaneously gave rise to the two leading approaches to sexual selection and mating systems research that exist today: (1) the conceptual and empirical focus on sex differences in parental investment (Williams, 1966; Trivers, 1972; Emlen and Oring, 1977) and (2) the theoretical and empirical focus on measuring sexual selection itself (Wade, 1979; Wade and Arnold, 1980; Shuster and Wade, 2003; Jones, 2009).

In his experiments, Bateman established 64 fly populations organized into nine 'series,' each designed to explore mating success and fitness for each sex and to control for the effects of different genetic markers, mating schedules, female life histories and genetic backgrounds. Whereas modern sexual selection researchers use microsatellites to document paternity and maternity, Bateman used a suite of visible single-gene mutations to uniquely identify the parents of each offspring in his populations. Although Bateman's genetic markers did not have the advantage of neutrality that microsatellites do, this apparent constraint necessitated his extraordinarily detailed set of experimental controls, as well as the elegant statistical methods used in analyzing his results.

For example, to avoid inbreeding, Bateman used different markers in males and in females, guaranteeing that all offspring from which matings per parent were to be inferred would be double heterozygotes, not homozygotes. Bateman also emphasized that he was measuring the actual fertility of individuals rather than their potential contributions to the next generation. He recognized how this limited his inferences of the number of mating attempts: 'the number of inseminations … should, however, be regarded as a minimum, for two reasons: the possibility that some matings might be ineffective, and the inability to distinguish single and multiple inseminations involving the same pair of flies.' (p 353). The same limitations apply to microsatellite estimates of parentage, but are seldom as well recognized. Bateman also noted that uncontrolled viability differences among matings were more likely to *reduce* rather than *increase* the variance in offspring numbers within a series, making his observed results conservative.

Even with these constraints, Bateman's results were consistent and striking. He observed a sex difference in the variance in fertility that was greater for males than for females in every series. Bateman also noted (Table 7; p 360) 'whereas only 4% of females were unrepresented in progeny, 21% of the males were unrepresented.' Males were five times more likely to fail in reproductive competition than females. Importantly, when Bateman subtracted the sum of squares owing to the effect of mate numbers from the total sums of squares, he found the remainder to be equal for the two sexes. He summarized his findings in the often-repeated quotation: 'Variance in number of mates is, therefore, the only important cause of the sex difference in variance of fertility.' These famous words were also presented in his Figure 1 (p 362) as a linear relationship between mate number and offspring number for males and a much smaller, but similar effect for females. Such regressions today are commonplace and are referred to as 'Bateman Gradients' (Arnold and Duvall, 1994), consistent with Bateman's observation that 'a sex difference in the variance in fertility is therefore a measure of the sex difference in intensity of selection.'

Several authors have claimed that Bateman's methods were flawed, including sampling problems that resulted in unequal male and female mean reproductive success, miscalculations of variances, statistical pseudoreplication and selective presentation of data (Snyder and Gowaty, 2007). These authors imply that Bateman was hindered in his conclusions by the limitations of the markers he used, and further that he was careless, even dishonest, in how he conducted his measurements and analyses. A careful reading of Bateman (1948) reveals that nothing could be further from the truth. Bateman's original emphasis was on mean squares to capture the 'gross variability' in fertility among males and among females within each mating series. In every case, even when reciprocal matings were made between males and females from different marker lineages, the variation in male fertility was significantly greater than for females. Far from an attempt to inflate the degrees of freedom used in his analysis, this approach accurately measured the fitness variance for each sex within each series, and his significance tests of these variance ratios showed the relationship he needed to make his points.

Other authors (Sutherland, 1985; Hubbell and Johnson, 1987) have suggested that, because random mating can lead to sex differences in mating success, Bateman's data do not show the actual cause of sexual selection. This

criticism ignores Bateman's partitioning of the separate variance component for mate numbers from the total variation in fertility, as well as the internal consistency of the results across series, and the layers of controls he applied. The authors, as well as other adherents to the notion that chance is causal in studies of sexual selection, neglect to acknowledge that chance is part of any study of selection. Some individuals survive, mate and reproduce by chance, whereas others do not, even if preferred or favored attributes exist within the population. This is why the probability of fixation of a 'good gene' with positive effect on fitness, $s>0$, is only $2s$ instead of 1.0 (Kimura, 1962).

What makes Bateman's results definitive is that certain markers appeared disproportionately among progeny, indicating that some individuals mated and others did not. This result led to the most enduring conclusion of his work, the signature cause of sexual selection: the sex difference in fertility, which causes the positive regression of offspring numbers (fertility) on mate numbers for males. Bateman distilled his findings to a single statement that is as powerful today as it was in 1948 (p 364): 'Variance in number of mates is, therefore, the only important cause of the sex difference in the variance in fertility.'

Conflict of interest

The authors declare no conflict of interest.

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