The lek paradox and the capture of genetic variance by condition dependent traits

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1. SUMMARY

Recent evidence suggests that sexually selected traits have unexpectedly high genetic variance. In this paper, we offer a simple and general mechanism to explain this observation. Our explanation offers a resolution to the lek paradox and rests on only two assumptions; condition dependence of sexually selected traits and high genetic variance in condition. The former assumption is well supported by empirical evidence. We discuss the evidence for the latter assumption. These two assumptions lead inevitably to the capture of genetic variance into sexually selected traits concomitantly with the evolution of condition dependence. We present a simple genetic model to illustrate this view. We then explore some implications of genic capture for the coevolution of female preference and male traits. Our exposition of this problem incidentally leads to new insights into the similarities between sexually selected traits and life history traits, and therefore into the maintenance of high genetic variance in the latter. Finally, we discuss some shortcomings of a recently proposed alternative solution to the lek paradox; selection on variance.

2. INTRODUCTION

There is abundant evidence of persistent female choice of male traits in species where there are no apparent direct benefits of choice to females (Bradbury & Andersson 1987; Kirkpatrick & Ryan 1991; Andersson 1994). The most extreme cases are found in lek mating systems, where males gather in arenas and females choose among them. Here males give no resources to females and no parental care to their offspring, so it is a challenge to understand what could lead to the evolution and maintenance of female preference. One possibility is that females receive 'good genes' for their offspring from preferred males. However, it is a long-held expectation that there will be little genetic variance in fitness (Falconer 1981; Charlesworth 1987), and hence that there would be little potential for benefit to female choice through good genes. This is the origin of the lek paradox (Borgia 1979; Taylor & Williams 1982; Kirkpatrick & Ryan 1991; Andersson 1994).

The expectation of low genetic variance in fitness is based on the fact that fitness is, by definition, always under directional selection, and therefore a single best genotype should become predominant (Falconer 1981; Charlesworth 1987). This view has been bolstered by a number of studies demonstrating that the heritability (h^2, additive genetic variance standardized by phenotypic variance) of life history traits, which are presumably closely related to fitness, is relatively low (Falconer 1981; Gustafsson 1986; Roff & Mousseau 1987; Mousseau & Roff 1987). However, comparisons based on heritability are misleading, as Fisher's fundamental theorem of natural selection shows that the response of fitness to selection depends only on the additive genetic variance, and not on other components of variance. Mean standardized measures of the additive variance, such as coefficients of variation, are thus more appropriate to assess the amount of genetic variation in such traits (Crow 1958; Houle 1992). Comparisons of coefficients of variation show that life history traits have low h^2 because they have relatively high environmental variance (Price & Schluter 1991; Houle 1992), and not because of a lack of genetic variation.

In fact, a review of the literature on the quantitative genetics of outbreeding populations demonstrates that life history traits have on average significantly higher amounts of additive genetic variance on a mean standardized scale than do metric traits (Houle 1992). The average additive genetic coefficient of variation for life history traits is slightly in excess of 10%. A trait with this level of variation is expected to respond to linear directional selection at the rate of 1% per generation. If genetic variance in life history traits implies genetic variance in fitness, then females who are able to discriminate and mate with high fitness males can expect to gain a substantial benefit over those mating randomly. If this is so, we also expect that sexually selected traits should share this abundance of genetic variance. Recently, Pomiankowski & Møller (1995) have found significantly higher additive genetic variation in sexually selected traits than in non-sexually selected traits in similar taxa.

These comparative data from life history and sexually selected traits have in one sense resolved the lek paradox. Both those traits most closely related to fitness and traits which are the proximal targets of

female choice have relatively high genetic variance. However, a complete resolution of the lek paradox requires an explanation for this maintenance of high genetic variation in the face of strong and persistent directional selection for fitness. Pomiankowski & Møller (1995; Turner 1996) favour one hypothesis for the maintenance of greater genetic variation under directional selection. They suggest that fitness may increase at a greater than linear rate with the exaggeration of sexually selected traits. Increased phenotypic variance is favoured in traits subject to fitness functions of this form (Lande 1980). Pomiankowski & Møller (1995) see such changes in terms of changes at modifier loci which act to increase the number of loci with direct effects on the sexually selected trait, or increase the average contribution of each locus to phenotypic variance.

In this paper we propose a simpler and more general explanation for the maintenance of high genetic variance in sexually selected traits. Our argument has two premises, both of which are well supported. First, we note that there is abundant empirical evidence that sexually selected traits evolve condition dependent expression. Second, we argue that there is high genetic variance in overall condition. If both of these premises are correct, sexually selected traits will inevitably express the high genetic variance in condition, thus resolving the lek paradox. We then identify some shortcomings of the alternative hypothesis proposed by Pomiankowski & Møller (1995).

3. CONDITION DEPENDENCE AND GENETIC VARIANCE IN CONDITION

We imagine the life history as a process of accumulating resources that are then allocated to the production or maintenance of traits that enhance fitness (figure 1). We will refer to the pool from which resources are allocated as condition, C. Thus, we take a broader view of condition than is often used (e.g. nutritional state) in models of sexual selection (Andersson 1982; Nur & Hasson 1984) and life history evolution (for examples, see Price *et al.* 1988; Rowe *et al.* 1994). Our view of condition is analogous to 'residual reproductive value' in traditional life history models, or 'state' in dynamic life history models, in that it is an internal property of the individual and accounts for a large portion of fitness.

As resources are allocated to one trait, this depletes resources that would otherwise have been allocated to other fitness enhancing traits. For some traits (e.g. foraging structures) this cost may be more than compensated for by subsequent increases in the resource pool. However, such will clearly not be the case for many sexually selected traits; for these we expect a non-recoverable or continuing drain on resources. Allocation to sexually selected traits may be direct when the trait is costly to express, such as in energetically costly calling (see, for example, Ryan 1988) and other displays (see, for example, Vehrencamp *et al.* 1989). More often costs may be indirect, for example, traits that impede foraging behaviour and thereby deplete condition (see, for example, Møller 1989) or those that increase predation risk (Magnhagen 1991) which is expected to lead to reduced condition through behavioural modification (McNamara & Houston 1987; Ludwig & Rowe 1990). Therefore, a key feature of the transition of a trait from stabilizing natural selection to directional sexual selection, is that it will become more costly (see figure 1). The evolutionary exaggeration of the trait will cease when the benefits of exaggeration are balanced by these costs (Partridge & Endler 1987; Price 1987; Kirkpatrick & Ryan 1991; Andersson 1994).

Once a trait becomes costly, we expect that it will evolve condition dependence (see figure 2). There is a great deal of empirical support for the condition dependence of sexually selected traits (reviews in Price *et al.* 1993; Andersson 1994; Johnstone 1995). It is easy to see from the simple life history pictured in figures 1

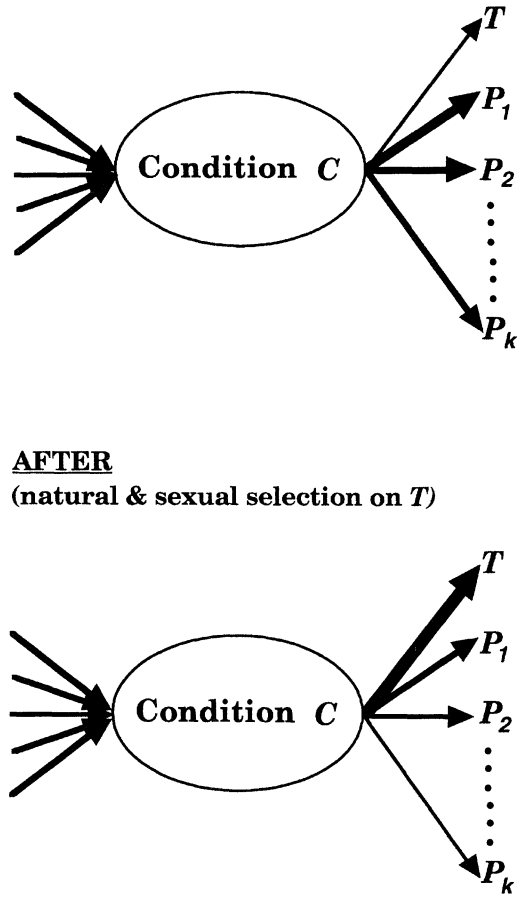

Figure 1. The flow of resources from condition (C). A variety of underlying traits contribute resources to condition. Resources are then parcelled out to a variety of traits (T and P_1-P_k) which contribute to fitness. Although paths are not shown, these traits may also contribute to condition. Thickness of the path indicates amount of resource flow. Figure 1*a* shows a hypothetical case where T is allocated a small amount of resource while under stabilizing natural selection alone. After some period of sexual selection, allocation to T increases dramatically (figure 1*b*). The cost of this increase in allocation to T is a decrease in allocation to one or all of the other traits (P_1-P_k).

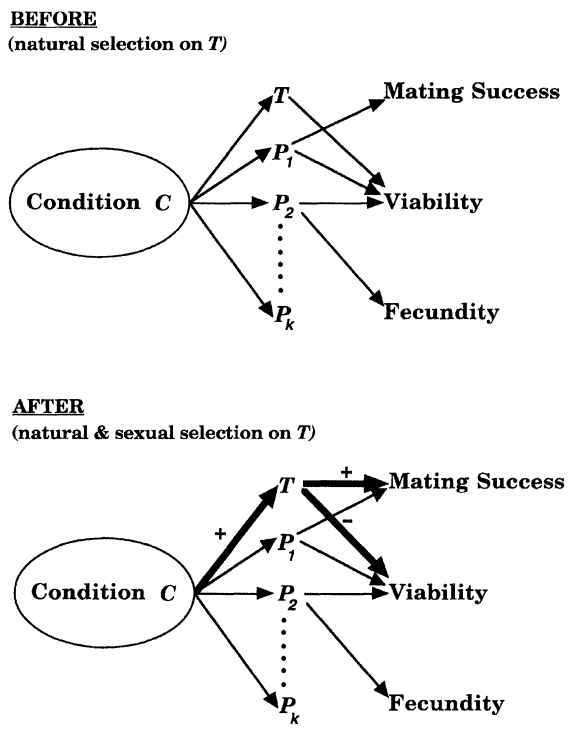

Figure 2. Path diagram of the relationships between condition (C), various traits (T and P_1-P_k), and fitness components. We omit signs on paths not involving T. Before sexual selection on T, we assume stabilizing viability selection on T (figure 2*a*). There is little or no correlation between condition and T, and no correlation between T and mating success. After some period of sexual selection on T, there is conflicting selection on T (paths to mating success and viability of opposite sign), and T becomes correlated with condition (condition dependence) (figure 2*b*).

and 2 that there is a trade-off between allocation to sexually selected traits and the remaining components of fitness (e.g. viability). However, individuals in higher condition have a larger resource pool to allocate among competing demands. Condition dependence is expected to arise because individuals in higher condition are better able to pay higher marginal costs of further exaggeration than those in lower condition (for examples, see Andersson 1982; Parker 1982; Nur & Hasson 1984; Grafen 1990; Pomiankowski *et al.* 1991; Iwasa *et al.* 1991; Price *et al.* 1993; Iwasa & Pomiankowski 1994).

It is crucial to our argument that there is a high genetic variance in condition. We suggest that the observed high genetic variance in sexually selected and life history traits results from their strong dependence on condition. Both types traits are constrained by trade-offs and ultimately limited by condition. We develop two related arguments for high variation in condition that arise from the observation of high genetic variance in life history traits (Houle 1991, 1992).

First, the genetic covariance structure of life history traits suggests that there is a relatively large genetic variance in condition. We expect the variance in life history traits to consist of variance in condition (C), plus some variance because of allocation of C to the various traits (van Noordwijk & de Jong 1986; Houle 1991). Variance in condition is expected to lead to positive covariance between life history traits, while variance in allocation is expected to lead to negative covariance. The relative importance of condition and allocation is indicated by the genetic correlations among life history traits, which are generally near 0, sometimes being slightly negative, and sometimes slightly positive (Houle 1991; Stearns 1992). This suggests that allocation and acquisition are roughly equally important sources of genetic covariance. As only a portion of the overall variance in condition will be reflected in any one trait, the mean standardized variance in life history traits (10% on average) is therefore a conservative estimate of the overall genetic variance in condition.

Second, a larger proportion of the genome affects condition and life history traits because they are complex summaries of many processes (Price & Schluter 1991). Every locus in the genome must potentially affect fitness, and therefore traits that summarize a major proportion of fitness will typically be affected by large numbers of loci (Houle 1991). As many of these processes must influence overall health or condition, this explanation favors the existence of very large genetic variance in condition. In fact, it is difficult to imagine metric traits that do not contribute in some way to condition. This view is supported by the observation of a relatively high mutational variance in life history traits (Houle *et al.* 1996). This suggests that a large number of loci contribute to these life history traits, and that this variation is maintained in the spite of persistent directional selection. Moreover, there are high mutational correlations among life history traits (Houle *et al.* 1994). This suggests that the target of most of this mutation pressure is a common trait that underlies all life history traits, rather than the relative levels of allocation to these traits. This target we interpret as condition, C, in our figures 1 and 2.

4. A SIMPLE MODEL

In this section we show why sexually selected traits are expected have high genetic variances if our premises of condition dependence and high genetic variance in condition are met. To do so, we will compare the genetic variance in a metric trait, T, when it is subject to stabilizing natural selection for a constant mean (the NS case), to the genetic variance of T when it is subject to both natural and sexual selection (the SNS case). For convenience we will refer to female preference for a male trait. We only consider cases where T evolves condition dependent expression in the SNS case. Several theoretical studies have established conditions whereby female preference and condition-dependent expression of the male trait can coevolve (see above). In short, condition dependence of exaggerated traits is expected when the marginal costs to an increment of exaggeration are lower for individuals in high than low condition. The emphasis of prior models has been on the evolution of female preference. To model this, the genetically explicit models have assumed that the genetic variance of the traits is constant. Here we do the converse: we assume that female preference evolves,

and investigate the consequences of female preference for the genetic variance in T. Note that we do not assume a specific model for the evolution of female preference.

For simplicity, we assume that T is a linear function of condition

$$T = a + Cb$$

where C is condition, a represents a condition-independent level of expression of T, while b is the rate at which expression increases with condition. Each of the variables C, a, and b are in part genetically determined, and each is affected by a different set of genes. We also assume that the genetic covariance among the three variables is negligible. To justify this, the loci affecting a and b are assumed to be few in number, and unlinked to each other. Variance in condition arises at the wide variety of loci which influence overall health, and rate of energy gain. These are assumed to be scattered throughout the genome, so that only a small proportion of these loci are closely linked to those affecting a and b. By taking the expectation of the variance in T decomposed into genetic and environmental effects, it is easy to show the genetic variance of T is approximately

$$G_T \approx G_a + \bar{b}^2 G_C + \bar{C}^2 G_b$$

where G_i represents the genetic variance of the variable indicated by the subscript i, and the overlines indicate mean values. Terms involving products of the variances are negligible with reasonable parameter assumptions (D. Houle & L. Rowe unpublished data). Both additive and non-additive variance are included in G. Expressions of the same form hold for each component of the genetic variance.

The key term in this equation is $\bar{b}^2 G_C$. It reflects the fact that with condition dependence, a genotype which has a higher than average level of condition will also have a higher than average value of T, while poor genotypes will have lower than average T. While this term does not involve variation at the loci specifically involved in shaping the trait (a and b loci), it nevertheless reflects a source of genetic variance which will be shared by relatives.

In the NS case, T is only subject to stabilizing natural selection for a constant value. This will minimize condition-dependence of T, and therefore both $\bar{b}$ and G_b would be very near 0, and the variance of T will be approximately G_a. In the SNS case, T becomes the target of directional sexual selection in addition to stabilizing natural selection. The intuition behind the lek paradox is that, in this case, selection on T may become so intense that loci influencing it will become monomorphic. This logic may hold for a and b which could be under weak selection in the NS case. However, C is under directional selection in either case so we expect that G_C will be substantially unaffected by the transition from the NS to the SNS case. As sexual selection is expected to both minimize G_a and G_b and increase $\bar{b}$, the expected result is that the variance in T will be dominated by $\bar{b}^2 G_C$. As outlined above, there seems to be very large genetic variance in condition, G_C, so this term may be quite substantial. If condition dependence becomes strong, this will almost certainly increase the genetic variance of T over the NS case. This transition from the NS case to the SNS case converts variance in overall condition into variance in the male trait. We call this process genic capture.

In addition to raising the issue of the persistence of the variance in sexually selected traits, Pomiankowski & Møller's (1995) review also shows that the genetic coefficient of variation of sexually selected traits is as large as that in other morphological traits. As the means of the sexually selected traits have been increased by sexual selection, the genetic variances must also increase accordingly to explain this observation. Addressing this quantitative question requires more specific models, which we will publish elsewhere. Here we simply note that under our basic model, plausible conditions where the genetic coefficient of variation increases are readily found.

5. DISCUSSION

We argue that a simple resolution of the lek paradox can be found in two robust observations. The first observation is that sexually selected traits are generally condition dependent in their expression (Price *et al.* 1993; Andersson 1994; Johnstone 1995). The second is that there is abundant genetic variance in condition. We argue that much of the observed genetic and environmental variance in fitness reflects underlying variance in condition. Following a widespread tendency to treat condition as environmental in origin (for examples, see Price *et al.* 1988; Price & Liou 1989; Alatalo *et al.* 1990; Schluter *et al.* 1991), it has been argued that to understand the evolution of exaggerated sexual traits we must first control for condition, and then address the partial correlation with fitness. Another school of thought holds that condition is integral to the evolution and maintenance of sexually selected traits (see, for example, Andersson 1982; Nur & Hasson 1984; Grafen 1990; Pomiankowski *et al.* 1991; Iwasa *et al.* 1991; Price *et al.* 1993; Iwasa & Pomiankowski 1994). In our view, it is precisely this condition dependence which is crucial to the maintenance of genetic variance in sexually selected traits, in the face of persistent direction selection. Once developed, the correlation of the trait with condition captures some portion of the abundant genetic variance in condition.

In addition to explaining the existence of substantial variance in sexually selected traits, our hypothesis also has important implications for the dynamics of traits under sexual selection. Previous models have assumed that the genetic variance of the male trait is fixed during the coevolution of female preferences and the male trait. Yet, with the evolution or increase of condition-dependence during coevolution, we expect genetic variance in the male trait to simultaneously increase. Genic capture may enhance the prospect for preference and male trait to increase directly, through an increase in the rate of response of the male trait, and indirectly, by potentially enhancing the important covariance between female preference and the male trait. Efforts to model the coevolution of female

preference and male traits with the inclusion of condition dependence and genic capture would be well spent. Price *et al.* (1993) have developed a relevant model of the evolution of female preference and condition dependence trait expression, assuming that condition is purely environmental and that females derive direct benefits from their mates.

One important prediction of our model is that good genes should always become involved in the expression of sexually selected traits which evolve condition dependence, and in the dynamics of the preference. As our model does not depend on the mechanism by which preference arises, this prediction should apply in the latter stages of coevolution under direct selection on preference, through the Fisher runaway process, or on selection for a revealing handicap. Thus, the existence of condition dependent expression is not sufficient to implicate selection for a handicap, nor is the costliness of the trait a basis for rejecting the Fisher runaway process. This reiterates Kirkpatrick & Ryan's (1991) general point that male traits at equilibrium offer little information about the evolution of female preferences.

More generally, we expect that condition dependent traits will tend to have higher genetic variance than their condition independent counterparts. This suggests an interesting reformulation of the hypothesis that life-history traits have high genetic variance because they are affected by many loci. Presumably part of the reason that life-history traits are affected by many loci is that their optimal expression is condition-dependent. Condition-dependence is an extremely widespread phenomenon in both metric and life history traits. Our understanding of the evolution of these traits will be enhanced by the recognition that with the evolution of condition dependence comes the capture of genetic variation into the trait.

Pomiankowski & Møller (1994) (hereafter P&M) suggest that, in traits under sexual selection, fitness may increase with trait expression at a rate greater than linear, thereby leading to selection for increased phenotypic variance (Lande 1980). They further suggest that such selection on variance may lead to the evolution of modifiers which increase the number loci or strength of their effects on the trait. In one sense P&M's argument yields a similar result to our model if our loci affecting condition dependence replaced the 'modifier' loci invoked by P&M. The key difference between the models is that ours does not require any selection on variance in the underlying genetic variables.

The central problem we see with P&M's (1994) explanation for high variance in sexually selected traits is that sexually selected traits are in fact under net stabilizing selection at equilibrium, rather than a concave upwards fitness function, as their argument requires. Although they are under directional sexual selection, the exaggeration of a male trait will eventually stop when the cost of further exaggeration of the trait balances the gains from mating success (Partridge & Endler 1987; Kirkpatrick & Ryan 1991; Andersson 1994). There is abundant empirical evidence of costs to sexually selected traits (Magnhagen 1991; Andersson 1994; Johnstone 1995). Thus, under equilibrium or near-equilibrium conditions, sexually selected traits are expected to be under conflicting, rather than directional selection. Here, there is one trait optimum for any given condition and selection will be for decreased variance. Our model assumes such conflicting selection on the male trait. Persistent net directional selection is probably uncommon for metric traits, and this is what is required at minimum for variance in a trait to be favoured. Direct measures of selection on more than one fitness component confirm that the evolution of metric traits is typically governed by a balance of conflicting fitness advantages (Schluter *et al.* 1991).

As noted above, there is strong evidence for condition dependent expression of sexually selected traits. In these cases, variance in condition among individuals may mask conflicting selection on the trait (Zeh & Zeh 1988; Schluter *et al.* 1991). However, the evolution of condition dependence is not expected to change the form of selection on these traits from conflicting to directional. For any given condition, conflicting selection and an intermediate optimum is expected. Experimental manipulations of condition dependent sexually selected traits, thereby holding condition constant, have revealed such conflicting selection (Møller 1988; Møller 1989; Evans & Hatchwell 1992; Møller & De Lope 1994). For most examples of directional selection used in P&M's (1994) test, directional selection is measured as mating success rather than net selection, which we expect to be stabilizing. As an example, mating success of field crickets increases with duration of calling (Cade 1981), however this appears to come at the cost of increased parasitism by flies that use calls to locate their hosts (Cade 1975). Similarly, mating success appears to increase with the size of various grasping apparatus in psuedoscorpions and water striders (Zeh 1987*a*; Arnqvist 1994), but these come at a cost of increased development time and perhaps mortality (Zeh 1987*b*; Arnqvist 1994).

Turner (1996) has similarly noted that at equilibrium, net selection on sexually selected traits is not expected to be directional, but suggests that the observed genetic variation in such traits may reflect directional selection in the past. This requires that the variance in male traits increase rapidly during episodes of directional selection and decrease slowly after it has ended. This asymmetry is unlikely. Furthermore, as modifier selection is notorious for being extremely weak, we would not expect such a rapid increase in variance.

6. CONCLUSION

We have argued that the evolution of condition dependence in traits under sexual selection will lead directly to an increase in the genetic variance in those traits. This offers a simple and general resolution to the lek paradox. The argument rests on two well supported assumptions; condition dependence evolves and condition is affected by many loci. Traits that evolve condition dependence will capture some of the genetic

variance in condition. This mechanism alone can account for the observed high levels of genetic variance in life history traits and sexually selected traits. We point to three new lines of research. First, at present our understanding of genetic variance in condition is based on indirect evidence. Thus, we need more direct measures of the genetic variance in condition. Second, past models of the evolutionary dynamics of preference and preferred traits typically assume constant genetic variance in male traits. New models should investigate the increases in genetic variance that we predict, and that are implied by the empirical data. Finally, we have argued that 'good genes' will play at least some role in the dynamics of preference, whatever the reason preference originally evolved. The effect of this on the evolution and maintenance of preference and trait will require new modelling efforts.

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