The Truthful Signalling Hypothesis: An Economic Approach

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ABSTRACT

In mating competition, the Truthful Signalling Hypothesis (TSH), or handicap principle, asserts that higher-quality (H) males signal while lower-quality (L) males do not (or else emit smaller signals). Also, the signals are "believed" (females mate preferentially with higher-signalling males). Our analysis employs specific functional forms to generate equilibrium solutions that validate the TSH. Numerical simulations illustrate how the equilibrium responds to changes in the exogenous parameters.

"Net" male quality is what remains after deducting signalling costs from intrinsic or "endowed" quality. Following Grafen (1990b) in dealing with endowed quality that is non-heritable, we introduce several crucial analytic innovations: (1) A Mating Success Function (MSF) indicates how female mating choices respond to higher and lower signalling levels. (2) A "congestion" function rules out corner solutions in which females would mate exclusively with H males. (3) In addition, for biological equilibrium a Malthusian condition is employed to balance population size and resources.

Equilibria validating the TSH are achieved over a wide range of parameters, though not universally. Contra some statements in the literature, for TSH equilibria it is not a strictly necessary condition that the high-quality males have an advantage in terms of lower per-unit signalling costs, although any cost difference in favor of the L-type males cannot be too great. TSH equilibria may also fail if the proportion of H-type males in the population is too large.

Signalling can start up from a no-signalling equilibrium only if the trait used for signalling is not initially a handicap, but instead is functionally useful at low levels. Selection for this trait sets in motion a bandwagon whereby the initially useful indicator is pushed by male-male competition into the domain where it does indeed become a handicap.

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This model of mating competition is aimed at determining the conditions needed to support the Truthful Signalling Hypothesis (TSH). (Sometimes known as "the handicap principle".) Dealing for simplicity with just two distinct male types, in a TSH equilibrium the high-quality (H) males signal (exhibit highly visible yet disfunctional traits) while the low-quality (L) males do not signal (or else emit smaller signals). Also, the signals are "believed" by the females, meaning that the females mate preferentially (though in general not exclusively) with the higher-signalling males.[2]

1. Introduction

To explain the evolutionary survival of disfunctional traits such as the peacock's tail, there have been two main analytical approaches. The first, due to R.A. Fisher,[3] proposes that a runaway or disequilibrium process is involved -- rather like a stockmarket bubble. Males are selected to have big tails because females prefer mates with big tails. And females continue to prefer such mates only because, in the next generation, their own big-tailed male offspring will similarly be preferred (for the same reason) by the females of the following generation:

> ... plumage development in the male, and sexual preference for such developments in the female, must thus advance together, and so long as the process is unchecked by severe counterselection, will advance with ever-increasing speed. (Fisher, 1958 [1929], p. 152)

The second approach, the one pursued here, postulates instead an equilibrium process. The disfunctional trait is evolutionarily stable because, as a handicap, it serves a signalling function (Zahavi, 1975). Ability to bear the cost of the disfunctional trait indicates that a male is a superior consort. The handicap trait can survive in equilibrium (under suitable conditions) because it represents a balance of forces: a disadvantage in terms of ordinary natural selection, versus a countervailing advantage on the score of sexual selection.[4] The question is whether an evolutionary equilibrium (ESS)[5] can be attained.

Formalizing the indicated equilibrium has turned out to be no easy task. The best-known model supporting the evolutionary validity of the TSH, Grafen (1990a,b), is at a high level of abstraction. The model here, which we believe complements Grafen's work, is more explicit. By making use of credible

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[2] Signalling occurs in non-mating contexts as well, for example in threat displays aimed at over-awing hostile competitors. The fullest description is Zahavi and Zahavi (1997). In the economics literature, the standard exemplar of signalling is the acquisition of educational credentials, assumed to be costly but non-productive (Spence [1974]).

[3] Fisher (1958 [1929]), pp. 146-156.

[4] Zahavi and Zahavi (1997, p. 40) propose the more genral terminology: utilitarian selection versus singal selection (p. 40).

[5] ESS signifies "evolutionarily stable strategy" (Maynard Smith, 1976). The terminology is imprecise because (apart from the special case of a homogeneous population) a set of strategies may be involved, one for each type of player in a stratified population.

specific functional forms, we produce a variety of exact analytical and/or numerical solutions. In return for the sacrifice of generality, such a successful specification demonstrates that at least the minimal standard of internal consistency has been achieved. In addition, we claim, the particular functional forms serve to illuminate the working of signalling interactions. (In economics, Cobb-Douglas or CES production functions -- although involving special assumptions about the functional relations between inputs and outputs – have proved to be extremely useful for advancing understanding of productive processes.) Instantiating the model may be suggestive in other ways as well, for example we will be exploring ranges of parameter values within which a TSH equilibrium can or cannot be obtained. And finally, the need for logical completeness may turn up issues not hitherto noticed. Thus, to achieve closure our equation system required "congestion" restrictions to limit females' ability to mate preferentially with high-signalling males. And again, equilibrium requirements indicated a need to satisfy a Malthusian condition, that population numbers be in balance with resources.

As outsiders we have found the modelling achievements of biologists to rate very highly on the scores of imagination, analytic power, and sensitivity to real-world implications. Yet even in highly impressive instances such as the Grafen papers referred to, it is difficult to figure out just what is going on. What are the assumptions? What are the constraints? Who is competing against whom, and what are the determinants of success? Which are the dependent variables, and which are parameters subject to exogenous variation? What are the conditions of equilibrium? Here is where the economic modelling tradition has something to offer. Economists have developed a logical procedure, under the heading of general equilibrium analysis, that organizes all the necessary elements in a structured and transparent way. We hope to show that a corresponding general equilibrium analysis of the signalling problem will help resolve a number of unsettled questions.[6][7]

Here is the paradox of signalling equilibrium models, as applied to mating choices. If females are attending to truthful signals, then higher-intrinsic-quality (hence higher-signalling) male contenders must necessarily win disproportionate mating success. So, assuming quality is heritable, in the next generation high-quality high-signalling males must increase in representation in the population. This process, continuing over the generations, leads inexorably to extinction of the low-quality males. But if signalling is to persist in evolutionary equilibrium, both low-quality and high-quality males must remain represented! So on the one hand the high-quality high-signalling males are always gaining ground, yet on the other hand the low-signalling low-quality males do not go extinct. (This seeming contradiction was presumably what led R.A. Fisher to postulate a runaway <u>disequilibrium</u> process instead.)

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[6] But we will <u>not</u> be simply translating economic signalling models into biological terminology. Our presentation, although consistent with the principles of economic equilibrium analysis, is not actually very closely related to current signalling models in the economic literature.

[7] Representing a somewhat different economic approach, the valuable paper by Noldeke and Samuelson (2001) came to our attention only after our work was substantially completed. Several of our key themes appear also in N&S, in particular the distinction between (what we call) endowed versus net male quality. N&S were primarily concerned to find formal conditions upon the distribution of endowed male quality (treated as a continuum) that make a TSH equilibrium possible. Our analysis, in contrast, dichotomized male quality (High versus Low). This simplification allowed us to generate explicit equilibrium solutions and to study detailed conditions on a number of parameters – among them the costs of signalling and the population proportions of the H and L types – required for achievement of a TSH equilibrium.

A way out, following Grafen (1990b), is to postulate that male quality is <u>non-heritable</u>:

In the first stage, males are randomly assigned a quality independently of their genotype ...
– Grafen (1990a), p. 475.

Male quality not being heritable,[8] what is inherited instead is a "signalling rule".[9] I.e., a scaled-response gene that in effect instructs its (male) bearer: "If you find yourself in a (non-heritably) high-quality body or with ample external resources, emit a big signal. If the opposite, emit a small signal (or no signal at all)."[10] The heritable rule specifies the tuning of this gene so as to quantify the males' scaled signalling responses to their higher or lower quality endowments.

The measure of quality, reproductive success (RS), is defined here in terms of female offspring only. A male's quality is the number of viable daughters that, if chosen as a mate, he can father and help rear into the next generation. (Of course the mother's quality is also involved in determining RS, but in this analysis all the females are assumed identical.) The non-heritable variations themselves – bodily features or external resources that contribute to reproductive success – constitute what we will term the male's <u>endowed quality</u>. However, the individual male will be dissipating some or all of his endowment in order to generate signals. So endowed quality represents only potential RS. The male's <u>net quality</u>, which is the trait of interest to females, is what remains after deducting signalling cost.

The analysis that follows employs a number of simplifying assumptions, among them: (i) There are only two male types, high-quality (H) and low-quality (L), constituting the respective fractions π and $1-\pi$ of the population. (ii) Within each quality type, all males are identical. (iii) Also, all females (F) are identical. (iv) The H and L male types summed together equal the females in number.

Following the standard procedures of economic general equilibrium analysis, the various agents <u>optimize</u>, that is, each individual is viewed as maximizing reproductive success subject to the relevant constraints. (A process leading presumably to the same result as evolutionary selection for maximum

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[8] Although Grafen did note that his analytical model dealt with non-heritable quality, that point did not receive much emphasis in his paper. And indeed, some influential biologists seem to believe that Grafen has proved the existence of signalling equilibria for <u>heritable</u> quality variations. Thus Dawkins: "Quality, for Grafen, means that ... females would benefit genetically [sic!] if they mated with good males ..." (Dawkins, 1989, p. 310). And similarly Zahavi and Zahavi (1997), though claiming support from Grafen's demonstration (p. 58), associate handicap signals with a huge range of male quality traits, many of them clearly heritable. Noldeke and Samuelson (2001), however, have noticed the limitation of Grafen's analysis to non-heritable quality variation.

[9] "All the signalling models assumed that the <u>signalling rule</u> is heritable." (Grafen, private communication to one of the authors, emphasis added.)

[10] Thus, consistent with the Zahavi and Zahavi (1997), instead of <u>selection for quality</u> there is <u>selection for signals</u>. Note the analogy with the hypothesis of Trivers and Willard(1973), which deals with the sex ratio of offspring as a scaled-response function of non-heritable parental quality.

fitness.)[11] All the separate individual optimizing choices are brought into consistency with one another in equilibrium, characterized by a balance among the signalling efforts of the two male types and the willingness of the females to allocate mating capacity in response to those signals.

In this analysis we tentatively assume that the Truthful Signalling Hypothesis is valid. Then the high-endowed-quality and low-endowed-quality types H,L are also respectively the bigger-signal and smaller-signal types (indexed as 1,2). Signalling types 1 and 2 will therefore be represented in the same population proportions $\pi, 1-\pi$ as the H and L quality types. In accordance with the TSH, the model must also incorporate the fact that females "believe" the signals. Thus, letting m and $1-m$ denote the proportions of the typical female's mating capacity devoted to the big-signal and small-signal types respectively, it must be that in equilibrium $m > \pi$.

If the analysis of the TSH works out without contradiction, we can at least infer that the hypothesis and the model are internally consistent. In addition, the analytic solutions and numerical simulations that follow will illuminate various aspects of the signalling problem. As one example, it has been said that the validity of the TSH depends upon the higher-quality (and therefore bigger-signalling) male type having lower per-unit signalling cost.[12] We will show that this condition is not actually necessary. As another example, our simulations will indicate that, for a TSH equilibrium to exist, the H-type cannot represent too large a proportion of the male population.

Section 2 that follows sets forth and explains an initial version of the model, and Section 3 provides a baseline numerical instantiation. After noticing that under the baseline condition population size is not in equilibrium, Section 4 extends the analysis to incorporate a Malthusian equilibrium condition. Using this extended model, Section 5 numerically simulates variations in a number of the determining parameters -- among them relative signalling costs and the proportions of the H and L endowment types in the population. Section 6 addresses the question of how, starting from an initial no-signalling situation, a pattern of signalling can ever emerge. Section 7 covers possible extensions of the analysis, and Section 8 concludes.

2. The Model (Initial version)

Male choices

Let us start with the constraints. Subscripts 1 and 2 refer to the male types that emit big and small signals, which by hypothesis are respectively the high and low endowed-quality types H and L. Each male's (non-heritable) endowed quality e_i $(i = 1,2)$ – his available resources, external or internal -- can be employed either to generate signals of magnitude s_i or else to generate net quality n_i as a mate:

$$e_i \equiv n_i + b_i s_i \quad \text{for } i = 1,2 \tag{1}$$

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[11] An approach sometimes called "methodological individualism". In biological terminology, we are eschewing group-selection arguments. An equilibrium is defined by the property that no individual can profitably deviate, regardless of the interests of the group as a whole.

[12] Thus: "... the marginal cost of advertizing must be lower for the better male." (Grafen 1990b, p. 522). Also: " ... signalling of quality can be evolutionarily stable, as long as the costs of signalling are less for better quality individuals." (Godfray 1991, p. 329).

Here the b_i represent the conversion coefficients (assumed constant) between resources and levels of signalling, i.e., the per-unit or marginal costs of signalling, which will in general differ as between the two male types. Since the coefficient of n_i is unity, endowed quality e_i and net quality n_i are denominated in the same "currency". And since all offspring (daughters) are of equivalent quality, only numbers matter. So n_i can be interpreted simply as the number of daughters per mating surviving into the next generation, for each male type.

Let m be the proportion of the females' aggregate mating capacity going to the H male type, the remainder of course going to the L type. When the TSH is valid, these will also be the proportions going respectively to the big-signallers and small-signallers. Since $\pi, 1-\pi$ are the population proportions of the two types, the H and L per-capita mating success rates will be m/π and $(1-m)/(1-\pi)$ respectively.

A male of either type will choose[13] the signalling level that maximizes his reproductive success RS, for which we will usually be using the economic term "utility" U_i – the product of his per-capita mating success, his net quality, and the female's mating capacity K (to be described below):

$$U_1 = K\left(\frac{m}{\pi}\right)n_1$$

$$U_2 = K\left(\frac{1-m}{1-\pi}\right)n_2$$

Male Utilities (2)

If the females "believe" the signal, then $m/\pi > 1$: the H males will have more than one mating per capita and the L males less.

Female choices:

With the aim of maximizing her reproductive success, each female will be choosing the probabilities m and $1-m$ of mating with a high-signalling or low-signalling male. To begin with, her RS will therefore be the average of the net qualities of the two male types, with weights m and $1-m$. However, there must be a constraint upon her range of choice, else she would devote 100% of her mating capacity to the bigger-signalling type (since she "believes" that to be also the male type of higher net quality). By assumption here, this limitation is interpreted as due to congestion. In attempting to mate disproportionately with the high-signalling males, i.e, to raise her chosen m above π, a female must accept some reduction of her mating capacity K.[14]

A specific formulation of the congestion constraint on female mating capacity is:

$$K = 1 - h(m - \pi)^2$$

Female Mating Capacity (3)

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[13] "Males will choose ..." is of course a short-cut expression for "males will be selected as if they were choosing ..."

[14] Females may have to wait in line for the preferred males, or else expend time and effort searching for them.

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where h is a positive constant.

Let M denote the weighted average quality of the typical female's mates:

$$M \equiv mn_1 + (1-m)n_2 \qquad \text{Average Net Quality of Female's Mates (4)}$$

Then the payoff to the female, her reproductive success in terms of number of daughters surviving into the next generation, is:

$$U_F = KM \qquad \text{Utility of Female (5)}$$

Optimization and equilibrium:

Each female will be balancing the marginal cost against the marginal benefit of shifting a unit of her mating capacity from the smaller-signalling type-2 males to the bigger-signalling type-1 males. The marginal benefit is the difference in net qualities $n_1 - n_2$ weighted by her mating capacity K. The marginal cost is the loss of mating capacity due to congestion, multiplied by the average net quality of the males she would otherwise have mated with.

Differentiating utility U_F with respect to m leads by straightforward steps to the female first-order condition:

$$K\left(n_1 - n_2\right) = -DM \qquad \text{Female F.O.C. (6)}$$

where $D \equiv dK/dm$ is the marginal congestion effect:

$$D = -2h(m - \pi) \qquad \text{Congestion Effect (7)}$$

The left-hand side of (6) is the marginal benefit, and the right-hand side the marginal cost.

As a crucial and the most novel step in our development, we now introduce a Mating Success Function (MSF).[15] If type-H and type-L males are signalling at the respective levels s_1 and s_2, the MSF tells us how the signals translate into mating success. Or put another way, the MSF specifies the degree to which a female attends to the signals in choosing her mating fractions m and $1-m$.

Following our methodological program of instantiating the TSH with specific functional forms, we postulate that females will respond to the male signals in accordance with:

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[15] The MSF is analogous to the Contest Success Function (CSF) employed in the economic conflict literature (see Hirshleifer 1989, 1991, Skaperdas, 1996). Such a function is perhaps implicit in some of the biological literature, but we have not seen it used explicitly.

$$\frac{m}{1-m} = \frac{\pi}{1-\pi}\left(\frac{s_1}{s_2}\right)^r$$

Mating Success Function (8)

Thus, the ratio in which females distribute their mating capacity between the two male types reflects the proportionate representation of the latter in the population (the first factor on the RHS) augmented by a signalling factor – expressed as the ratio of the signalling efforts raised to a power $r > 0$.[16] Here r, which scales the extent to which differing signalling intensities translate into mating success, is the "signalling decisiveness parameter". If $r = 0$ there is no effect at all: the females pay no attention to signals and mate at random, in which case $m = \pi$. The higher is r the more <u>decisive</u> are the signals in bringing about mating success.[17] Although r is parametric from the point of view of each and every male and female acting singly, for the analytic system as a whole the magnitude of r is an endogenous variable. More specifically, it is the equilibriating variable that glues together the male and female decisions. As an analogy, in economic models of perfect competition, each and every supplier and demander takes the market price as given -- yet the equilibrium price is endogenous to the system as a whole, determined by the overall balance of market conditions. Similarly the equilibrium level of the signalling decisiveness parameter r reflects the overall "market conditions" in the signalling process – the willingness of the different types of males to emit signals, and of the females to attend to them.[18]

Given the females' choice of m, on the male side there will be a <u>pair</u> of signalling levels s_1 and s_2 for the H and L types respectively. At equilibrium no individual male can advantageously deviate from the specified pattern of scaled responses. For the two types of males, this no-deviation condition together with the Mating Success Function lead to the first-order conditions:[19]

$$b_1 s_1 = r(e_1 - b_1 s_1)$$
$$b_2 s_2 = r(e_2 - b_2 s_2)$$

Male F.O.C.'s (9)

So, apart from the fixed parameters, the male signalling levels depend only upon the intermediating "decisiveness" parameter r that signifies the extent to which the females attend to the signals.

Taking stock at this point, leaving aside the utility variables for the moment, the core equation

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[16] In the economic literature there has been some discussion of alternative versions of the Contest Success Function. For example, proportionate success might respond to the <u>difference</u> rather than the <u>ratio</u> of the inputs. See Hirshleifer (1989), Skaperdas (1996), Neary (1997).

[17] Similarly in the economic conflict literature, r scales the <u>decisiveness</u> of force disparities in determining conflictual outcomes (Hirshleifer, 1991).

[18] The MSF is also somewhat analogous to a "contract" that the female might be regarded as offering to indicate her promised response to different signalling levels. However, the contract terminology is perhaps misleading, because in the model here females are just as constrained as the males. An individual female is not in a position to unilaterally offer contracts, but can only respond to the "market conditions" reflected in the equilibrium value of r.

[19] For the derivation, see Appendix 1.

system has 9 variables: s_1, s_2, n_1, n_2, m, M, K, and D, and r. Condition (1) provides 2 equations, and there are one each for conditions (3) and (4) and (6) through (8). With the two equations of (9), the numbers of variables and equations correspond.[20] An analytic solution of this model is provided in Appendix 2.

The utility variables are easily derived from the core solution. U_1 and U_2 are specified by equations (2) and U_F by equation (5). As a check, note that female utility is the average of the male utilities, weighted of course by the mating fractions m and 1-m.

$$U_F = KM = Kmn_1 + K(1-m)n_2 = \pi U_1 + (1-\pi)U_2 \qquad \text{Female Utility (10)}$$

For purposes of comparison it will also be convenient to define "no-signalling utilities" (signified here by asterisks) – the payoffs that would have been earned in the absence of any signalling at all, so that the females mate at random:

$$U_1^* = e_1$$
$$U_2^* = e_2 \qquad \qquad \text{No-signalling utilities (11)}$$
$$U_F^* = \pi e_1 + (1-\pi)e_2$$

3. Baseline Numerical Results – Initial Model

A numerical solution will be illuminating at this point. For this initial Model 1, the first column of Table 1 shows specified values of the underlying constants and the associated solution values of the variables. There are 8 core variables tabulated (we have omitted D, which is only an intermediate variable):

s_1:	Signalling level of H-type males
s_2:	Ditto for L-type males
n_1:	Net quality of H-type males
n_2:	Ditto for L-type males
m:	Proportion of female mating capacity going to H-type males
M:	Average net quality of mated males
K:	Female mating capacity
r:	Signalling decisiveness parameter

From the solution for these variables, 6 associated utility variables can easily be calculated:

U_1:	RS of H-type males
U_2:	RS of L-type males
U_F:	RS of females
U_1^*:	RS of H-type males under random mating (in absence of signalling)
U_2^*:	Ditto for L-type males
U_F^*:	Ditto for females

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[20] That the numbers of equations and variables match up does not of course demonstrate that the system is internally consistent and complete. But our numerical simulations provide such a test.

The following are the settings of the fixed parameters in the "baseline" numerical solution:

$\pi = 0.5$ Proportion of H-type males

$e_1 = 2.0$ Resource endowment of H-type males

$e_2 = 1.0$ Ditto for L-type males

$b_1 = 0.5$ Signalling costs per unit for H-type males

$b_2 = 1.0$ Ditto for L-type males

One additional parameter will be held constant throughout:

$h = 1.0$ Congestion effect coefficient

(Higher values of this parameter would increase the female's loss of mating capacity in the attempt to mate preferentially with the H-type males.)

[Table 1 about here]

Table 1 shows, in the column labeled "Initial Model 1", the solution values for the eight core variables (omitting D) of the initial system plus the six utility variables. Ability to obtain such a solution confirms the internal consistency of the model. Apart from that, a number of aspects of the numerical baseline solution are worthy of note:

1. At this baseline solution the TSH is validated. The H-type males, characterized by higher endowed quality ($e_1 > e_2$), do indeed signal more heavily than the L-type males ($s_1 > s_2$). And the signal is believed, that is, the higher-quality males capture a disproportionate share of the matings ($m > \pi$).

2. The parameters used here are such that the H type is not only of higher intrinsic quality ($e_1 > e_2$) but also benefits from lower per-unit signalling cost ($b_1 < b_2$). So it is not yet clear whether the observed validation of the TSH depends upon one or the other, or both, of these inequalities holding.

3. All three utility levels – U_1, U_2, and U_F – of the signalling solution are respectively less than the no-signalling utilities U_1^*, U_2^*, and U_F^*. In other words, the signalling process is a losing enterprise for all concerned.

This last point suggests the question of how the process might have gotten started, a topic that will be taken up in Section 5. First, however, we will be addressing a somewhat different problem: to wit, that the baseline solution represents a result inconsistent with a stable population size, hence falls short of being a true equilibrium.

4. Extended Model – Malthusian Condition

In equation (5) female utility U_F was defined as equal to KM, where K is her congestion-constrained mating capacity and M is the mean net quality of her male consorts (weighted by the mating fractions m and $1 - m$). But male net quality was defined in terms of the number of daughters per mating. In a Malthusian equilibrium, on average there must be exactly one surviving daughter per female, else population numbers would be rising or falling. So, for the population to be in balance it must be that:

$$U_F = 1 \qquad\qquad \text{Malthusian stability condition (12)}$$

But Table 1 indicates that, at the baseline solution, $U_F < 1$. Thus, the population would have to be falling. (Different parameter settings could equally well have led to the opposite result, in which population would be rising instead of falling.) To satisfy the conditions of equilibrium, the model has to be extended to provide for stable population numbers.

Let the total population be $2N$, so that the number of females equals N. It is the variation of N that will make condition (12) hold. More explicitly, the endowed qualities e_1 and e_2, previously taken to be constants, now become declining functions of N.

The enlarged equation system has three more variables: e_1, e_2, and N. The conditional equality (12) is one new equation. The other two equations are the relations between the quality endowments and population size, which we specify as taking the simple form:

$$e_1 = E_1 / N^w \quad \text{and} \quad e_2 = E_2 / N^w, \quad w > 0 \qquad\qquad \text{Population and quality (13)}$$

The new constants E_1 and E_2 will be termed the "underlying" quality parameters. The "endowed" qualities e_1 and e_2, previously defined as constants, now become variables responding to population size. Reflecting the effect of crowding, the value of the diminishing-returns parameter w indicates the degree to which the endowed qualities of both male types fall off as N increases.[21]

Appendix 3 provides an analytic solution of the extended system.

5. Parametric Variations of the Extended Model

We will now be using the extended Malthusian model to explore:
(1) Over what ranges of parameters will a TSH equilibrium – a handicap equilibrium involving truthful signalling – exist?
(2) Does a TSH equilibrium depend upon the higher-quality male type having lower unit signalling cost $(b_1 < b_2)$? Or upon differences in the underlying qualities $(E_1 > E_2)$ -- which, for given population size N, always imply corresponding differences in the endowed qualities $(e_1 > e_2)$? Or some combination thereof?
(3) For a TSH equilibrium to exist, must the male population fractions $\pi, 1 - \pi$ fall within certain bounds?

A baseline solution for the extended model incorporating the Malthusian condition is shown in the second column of Table 1. New constants E_1 and E_2 are shown for the underlying male qualities, while the endowed qualities e_1 and e_2 have been shifted from among the constants to the variables. Associated with these changes is the crucial new variable N representing population size . The baseline parameters of the extended system have been selected so that the solution values for e_1 and e_2 as

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[21] At the "nominal" population size $N = 1$, the endowed qualities as variables equal the respective underlying quality parameters. (N can be interpreted as measured in units of hundreds or thousands or whatever.)

variables, 2.250 and 1.125 respectively, end up quite close to the previous constant values $e_1 = 2$ and $e_2 = 1$ of the initial system before the Malthusian condition was imposed. As a result, most of the tabulated solution values are very similar to those of the initial system. As for the new variable N, the equilibrium population size ($N = 0.444$) is the "nominal" $N = 1$.

That some of the solution values remain exactly the same is due essentially to the fact that the postulated <u>ratio</u> of the underlying quality constants $E_1/E_2 = 2$ in this extended model remains the same as the previously assumed endowed quality ratio $e_1/e_2 = 2$ in the initial model.

We will be selectively varying only a few among the many parameters, in particular those whose variations suggest answers to the questions listed above. And in particular we will be considering variations of: (1) the underlying quality E_2 of the L-type males (relative to the fixed $E_1 = 1$ of the H-type males), (2) unit signalling cost b_2 for the L-type males (relative to a fixed $b_1 = 0.5$ for the H-type males), and (3) the fraction π of the population represented by the H-type males.

<u>(1) Parametric variation of underlying quality</u>
Figure 1 show the effects, in equilibrium, of parametrically varying E_2 – the underlying quality of the L-type males – relative to a fixed $E_1 = 1.0$. All the other parameters are held at their baseline values, while the underlying quality for the L-type males is allowed to vary both upward and downward from its baseline value $E_2 = 0.5$.

<< Figure 1 about here >>

Starting with the bottom panel, note that U_F – female utility (reproductive success) – remains equal to unity throughout. This is of course the feature of the extended model that was imposed to satisfy the Malthusian condition of population stability. In the middle panel the curve for N shows that the <u>level</u> of the population is greatest (reaches its nominal value $N = 1$) at the right edge of the diagram, where E_2 has attained equality with $E_1 = 1.0$.

The most interesting action takes place in the top panel. (For labelling convenience the variables $m, 1-m$ have been re-named m_1, m_2.) Taking the upward variation of E_2 first, as E_2 nears $E_1 = 1.0$ from below, the signalling levels s_1 and s_2 both fall off toward zero. As an intuitive explanation, when the underlying qualities E_2 and E_1 do not differ very much, the females have less need to respond sensitively to the emitted signals. (In the middle panel, the signalling decisiveness parameter r falls off toward zero as E_2 nears $E_1 = 1.0$.) This of course reduces the male incentive to signal. As a further consequence of the decline in signalling levels, the mating fractions m_1 and m_2 in the top panel converge toward equality at 0.5 when $E_2 = E_1 = 1.0$.

Now letting the underlying quality E_2 move <u>downward</u> from its baseline value $E_2 = 0.5$, the associated signalling level s_2 goes to zero as $E_2 \to 0$. (At the limit $E_2 = 0$, the L-type males simply have no resources for paying out signalling costs.) And the decline of s_2 reduces the need for the high-quality type to signal heavily, so s_1 also falls as $E_2 = 0$ is approached. Thus the signalling levels approach zero at either extreme: as the underlying quality of the L-type males, E_2, either moves upward toward equivalence with the quality level of the H-type males or else moves downward toward zero.

TSH equilibria exist here over the entire range of values $0 < E_2 \leq E_1 = 1$. (Except at the lower limit $E_2 = 0$ itself, where the Mating Success Function involves a factor of the form 0/0.)

(2) Parametric variation of signalling cost

Holding the other parameters at their baseline values, and in particular restoring the L-type underlying-quality parameter to $E_2 = 0.5$, the upper panel of Figure 2 shows what happens as the signalling-cost parameter for the L-type male moves away from its baseline value $b_2 = 1.0$ -- holding constant the H-type signalling-cost parameter $b_1 = 0.5$. Figure 2 brings to light a number of remarkable features of TSH solutions that have hardly been appreciated in the literature.

<<Figure 2 about here >>

The interesting range is from $b_2 = 1$ downward, where the L-type males' per-unit signalling cost is parametrically falling toward -- and possibly even beyond! -- equality with $b_1 = 0.5$. First, as unit signalling cost b_2 declines, for evident reasons the signalling level s_2 rises. And then, to maintain their mating representation, the H-type males respond with rising s_1 (though not in the same proportion). Thus, moving toward the left in the upper panel, the curves for s_1 and s_2 both rise sharply, quickly going off-scale in the diagram. Thus, as unit signalling cost for the low-quality males decreases, a "signalling arms race" is occurring.

One might wonder how the males can afford to engage in such extreme levels of signalling. The explanation is that, as shown in the middle panel, moving toward the left population size N is falling, thus raising the endowed qualities e_1 and e_2 relative to the underlying quality parameters E_1 and E_2 -- in accordance with equation (13). As population numbers fall, for Malthusian reasons both male types gain increased per-capita endowment to expend in signalling. And in fact, as b_2 moves downward, e_1 and e_2 rise without limit until the population size N hits zero at around $b_2 = 0.25$. At that point the analytic system breaks down.

That brings us to a second important feature brought out by the simulations: TSH solutions exist only over part of the range of signaling-cost variation. Furthermore, the range of TSH solutions is not defined by the condition $b_2 > b_1$ -- despite assertions to that effect in the literature (see earlier footnote). Perfectly good solutions exist for which the L-type males have lower per-unit signalling costs than the H-type males. And these solutions are still TSH equilibria: the high-signalling males remain of higher net quality (that is, $n_1 > n_2$), and the females continue to (correctly) "believe" the signals (they set $m > \pi$). So, as indicated in our opening discussion, a unit signalling-cost advantage for the high-quality male type is not a necessary condition for the TSH to hold. Without a positive advantage in terms of unit signalling cost, or even at some disadvantage in that respect (if not too great), the superior endowed quality e_1 of the high-type males permits them to profitably generate a positive and "truthful"signalling discrepancy $s_1 > s_2$.[22]

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[22] In economic jargon, an "income effect" and a "substitution effect" are involved. A rich person might purchase more of good X than a poor person, even at a higher price, simply because he can afford to do so. Similarly here, an H-type male can sometimes want to signal more heavily, even when incurring a higher per-unit cost than an L-type male. Owing to this ambiguity Maynard Smith (2001) is committing no error in saying "signals are a reliable indicator ... because they ... would be too expensive for a low-quality individual." That signals are "too expensive" may not be a matter of their per-unit price but simply that the low-quality individual is not well-enough endowed to afford them. On the other hand, Grafen (1990b) refers explicitly to marginal cost, and hence is incorrect when he indicates that for a TSH equilibrium it must be that (in our notation) $b_1 < b_2$.

Another possibly surprising feature in the top panel of Figure 2 is that the equilibrium mating fractions m_1 and m_2 remain unchanged as b_2 varies relative to b_1. This result was shown analytically in Appendices 2 and 3. We know from the MSF equation (8) that m_1/m_2 is a function of the signalling ratio s_1/s_2 raised to the power r (the signalling decisiveness parameter). It follows from equation (E) of Appendix 3 that as b_2 falls the signalling-level ratio declines. But we see in the middle panel of the diagram that the signalling decisiveness parameter r is rising correspondingly. Thus, although the differences in signalling levels are diminishing, the females are attending to these differences more.

Conclusion: It is not a necessary condition for a truthful signalling equilibrium that the higher-quality males have a per-unit signalling cost advantage. TSH equilibria are possible even at a cost disadvantage, although that disadvantage must not be too great.

(3) Parametric variation of the population fraction π:

Figure 3 illustrates variations around the baseline value $\pi = 0.5$ of the population fraction represented by the H-type males. The most notable feature is that the s_1 and s_2 curves are both markedly U-shaped – a result that, so far as we know, has not been remarked in the literature. Signalling efforts are near their minima at the baseline value $\pi = 0.5$ (actually, a little below, at around $\pi = 0.41$). Moving toward either extreme signalling levels rise quite rapidly, the proximate reason being the correspondingly rising signalling decisiveness parameter r. (Whenever females are attending more to signals, other things equal the males will respond with increasing s_1 and s_2.)

<<Figure 3 about here >>

But what determines the U-shape for r? Here the answer is somewhat subtle. Equation (B) of Appendix 2 (repeated in Appendix 3) is:

$$r = \frac{\ln\left[\dfrac{m(1-\pi)}{(1-m)\pi}\right]}{\ln\left[\dfrac{b_2 e_1}{b_1 e_2}\right]} \qquad (14)$$

From this equation the U-shape of the r curve in Figure 3 follows directly. First of all, the denominator is a constant, hence does not affect the shape. In the numerator, from the TSH property ($m > \pi$) it is evident that as $\pi \to 0$ the ratio m/π and therefore the bracketed numerator as a whole must rise without limit. That explains the why the left-hand branch of the curve nearer the vertical axis has negative slope. Moving in the opposite direction, as $\pi \to 1$ we see that m approaches the limit of unity when π is still only around $\pi = 0.75$. Thus once again, the numerator rises without limit.

So as π, the fractional representation of the H-type males, moves toward either limiting value 0 or 1, the signalling decisiveness coefficient r rises sharply. The greater decisiveness of signalling induces higher levels of signalling as either extreme is approached. In the upward direction this "arms race" leads to failure of the equation system at some interior value $\pi < 1$. But there is no such failure toward the low end (apart from the actual limiting value $\pi = 0$), since both the H and the L types remain represented as $\pi \to 0$.

Summing up at this point:
1. For the TSH to be valid it is not necessary that the high-quality males have per-unit lower signalling costs. By definition the H-type males have larger endowed resources, and over some range their resource advantage may permit them to obtain disproportionate mating success even at a unit-cost disadvantage. (However, any signalling-cost advantage favoring the L-type males cannot be too great, else a signalling equilibrium may not exist.)

2. Failure of the equation system typically stems, in our simulations, from an "arms race" -- intensified signalling -- that (so to speak) eventually puts at least one side out of business. This failure occurs when: (i) The L-type males have an excessive unit-signalling-cost advantage. (ii) The H-type males represent too large a fraction of the population.

Time and space do not permit exploration here of other types of parametric variations that might be considerable interest. One example: rising signalling costs for both male types together – higher b_1 and b_2 as well – will surely lead to lower signalling levels s_1 and s_2..

We have not emphasized utility comparisons so far, except for commenting that at the initial parameter baseline values all three utility levels U_1, U_2, and U_F of the signalling solution were respectively less than the no-signalling utilities U_1^*, U_2^*, and U_F^*. And indeed, this condition holds almost throughout the range of all the simulations. The one exception is visible in the bottom panel of Figure 3. When π (the population fraction represented by the H-type males) is sufficiently low the now-scarce H-type males do indeed come out ahead, per capita, as compared with the no-signalling equilibrium. Even then, however, this improvement does not hold for the females (or, a fortiori, for the L-type males).

As has been emphasized, despite the unprofitability in aggregate, no individual male can profitably refrain from signalling, nor can any individual female advantageously refrain from attending to signals. These considerations naturally lead to the question of how the process might have started.

6. A Signalling Start-up Model
If males are not emitting signals, there is no force at work leading females to attend to signals. And if females are not attending to signals, and generating signals is costly, there is no reason for the males to emit them. This "chicken and egg" problem means that the no-signalling solution is an ESS.[23] To break such an equilibrium, the model has to be revised so as to overcome the disadvantage of signalling – at least at initially low levels.

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[23] This is not quite rigorously true. If the females are not heeding signals, it is indeed a strongly superior strategy for males not to emit them (if, as has heretofore been assumed, doing so is always costly). But if the males are not emitting signals, then in our model the females are merely indifferent as to having the capability of attending to them. I.e., no cost has been attached to female development of signal-recognition ability. Thus, holding literally to our assumptions, females might acquire, merely by random drift, enough signal-recognition ability to make it eventually become profitable for males to begin emitting signals. Of course, it is highly unrealistic to say that developing signal-recognition capability involves no cost. Without attempting to explicitly allow for that factor, we will allow ourselves the liberty of presuming that some such cost does exist. If so, the no-signalling outcome would indeed be an ESS.

The disadvantages of signalling are two-fold: (1) the direct costs to the males, and (2) the loss of female mating capacity due to congestion.[24] On the other hand, signalling permits the females to mate disproportionately with higher-quality males. So it is possible in equilibrium to have $U_1 > U_1{}^*$ for the H-type males (as indeed occurs over part of the range in the bottom panel of Figure 3). On the other hand, for the L-type males U_2 can never exceed $U_2{}^*$. Since female utility U_F is always the mean of the males' utilities, when $U_1 > U_1{}^*$ it may or may not be the case that U_F exceeds $U_F{}^*$. But even when the parameters are such that $U_1 > U_1{}^*$ and $U_F > U_F{}^*$ both hold, it does not follow that signalling can ever get started. From an initial no-signalling situation, there would have to be a utility gain to individuals who <u>deviate</u> in the direction of signalling.

Apart from possible group-selection considerations, to escape the no-signalling equilibrium there is always the possibility of a matched pair of simultaneous (or near-simultaneous) mutations -- one dictating signalling on the part of H-type males, the other dictating that females heed the signals.[25] A more attractive explanation is that the trait that constitutes the signal, even though it is on balance costly (i.e., disfunctional) at the eventual signalling equilibrium, might have been functionally advantageous <u>when expressed at low levels</u>. Conceivably, in early evolutionary stages a moderately large tail might well be useful to the peacock for flight or for temperature control. If so, from an initial no-signalling equilibrium the males will be positively selected for that trait, in which case it would pay females to develop an ability to attend and respond to it.

The interesting analytic question is whether the assumptions allowing signalling to start up from a no-signalling equilibrium can set the population on the path to a signalling equilibrium in which the signal ends up as a handicap rather than a benefit.

As usual, we provide a specific instantiation, a revision of equation (1) for the two male types (i = 1,2):

$$n_i = e_i + g_i s_i - a_i s_i^2 \qquad (15)$$

Here the constant coefficient g_i is a "gain parameter", entering linearly with s_i. It is counterbalanced by a "cost parameter" a_i entering quadratically. The equation indicates that, for sufficiently small values of t_i, the signal is indeed functional rather than a handicap.

By straightforward steps, the first-order conditions for the males now can be written:

$$r = \frac{s_1(2a_1 s_1 - g_1)}{e_1 + g_1 s_1 - a_1 s_1^2}$$

$$r = \frac{s_2(2a_2 s_2 - g_2)}{e_2 + g_2 s_2 - a_2 s_2^2}$$

Revised Male F.O.C.'s (16)

These replacements for equations (1) and (9) are the only revisions needed.

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[24] Plus, if the interpretation in the preceding footnote is adopted, the un-modelled cost to females of developing signal-recognition capability.

[25] This would be a "green-beard effect" as described in Dawkins (1976).

A numerical baseline solution for the Start-up Model is shown in Table 1. The parameter values have been chosen to be generally similar to the baseline values used before. The baseline solution turns out to be in the handicap range. That is, despite the fact that the indicator is actually functional at sufficiently low values of s_i, at equilibrium there is a net loss of quality for both types of males: n_1 is less than e_1 for the type-H males, and similarly n_2 is less than e_2 for the type-L males. So, in this case the signalling equilibrium is an improvement for the high-quality males as against the no-signalling outcome $(U_1 > U_1^*)$, although for the females it is not $(U_F < U_F^*)$. For different parameter values, on the other hand, it would be possible to have the H-type males and the females both gain or both lose. Of course, in all signalling equilibria the low-quality males must lose out relative to the no-signalling equilibrium $(U_2 < U_2^*)$.

7. Extensions of the Analysis

Our purpose here was to generate the simplest possible model consistent with the main observed features of a world of where handicaps serve as signals. Methodologically, our analysis employed specific functional forms and plausible ranges of parameter values to instantiate the existence of equilibria. This procedure, although we hope illuminating, has the defect among other things of non-generality. So it is natural to think of directions of generalization, among them:

1. Extending the analysis to multiple levels of quality, or even to a continuum.[26]
2. Allowing for several dimensions of quality.
3. Allowing for heritable quality.
4. Dynamic issues, and in particular what happens if the parameter values are such that no TSH equilibrium exists.

Another kind of extension would be to adapt the model to signalling contexts other than mating competitions, for example to threat displays and their converse -- displays of cooperativeness.

Going somewhat farther afield, it would be of great interest to integrate the analysis of signalling with the analysis of mimicry. In the signalling problem, messages are truthful but costly to emit. In the mimicry problem, there is a trade-off between intrinsic quality and intensity of signalling. In particular, the cheater type invests less in quality but more in signalling effort. Whereas in the signalling problem the issue is the survival of truthful messages, in the mimicry problem it is the survival of untruthful messages.

8: Summary and Conclusions

We sought to determine the conditions needed to support the Truthful Signalling Hypothesis (TSH), sometimes known as the handicap principle. Dealing for simplicity with just two distinct male types, in a TSH equilibrium the high-quality (H) males signal while the low-quality (L) males do not signal (or else emit smaller signals). Also, the signals are "believed" by the females, meaning that the females mate preferentially (but in general not exclusively) with the higher-signalling males.

A seemingly intractable paradox troubles the modelling of signalling equilibrium. If the females are attending to truthful signals, then the higher-intrinsic-quality (hence higher-signalling) males must

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[26] As in Noldeke and Samuelson (2001).

necessarily win disproportionate mating success. But then, if quality is heritable, in the next generation the high-quality high-signalling male type will increase its representation in the population -- a process that must lead inexorably to ultimate fixation. So how can there be an evolutionary equilibrium in which low-quality and high-quality males are both represented, so that a need for signalling persists? Following Grafen (1990b), we postulated that male quality is <u>non-heritable</u>. (Surprisingly, although Grafen's contribution is widely cited, this crucial limitation is hardly ever noticed in the literature.) What is heritable is a signalling rule: a scaled-response gene instructing its (male) bearer: "If you find yourself in a (non-heritably) high-quality body or with ample external resources, emit a big signal; if the opposite, emit a small signal (or no signal at all)."

Methodologically, we attempted to show the usefulness of specifying credible functional forms to fill in the blanks of highly abstract models such as Grafen's. These specifications allowed us to actually solve the models analytically. A next step was to use these solutions for generating what we believe were illuminating numerical simulations of how equilibrium values of the endogenous variables – e.g., the signalling levels of high-quality and low-quality males and their relative mating success– respond to changes in several of the exogenous parameters.

We also tried to show how the framework of economic general equilibrium analysis – in particular, the explicit provision for optimization on the individual choice level and equilibrating balance conditions on the global level -- allows the elements of the signalling problem to be woven together in an understandable way.

Still under the methodological heading, the attempt to generate a complete and internally consistent equilibrium model turned up logical gaps that have hardly been noticed in the literature. Filling those gaps required the specification of somewhat unfamiliar analytic conditions. Among them are what we termed the congestion function, the Mating Success Function, and the Malthusian condition.

To summarize the main lines of the analysis, males of each non-heritable quality type attempt individually to optimize by maximizing reproductive success, the product of number of matings times offspring per mating (for given female mating capacity). Mating success is influenced by the males' chosen levels of signalling, and also by the degree to which females attend to the signals emitted. Offspring per mating is determined by the male's net quality, after expending some of his initial endowed quality by investing in signalling effort. So signalling has both favorable and unfavorable effects upon reproductive success, possibly leading to an interior optimum in which each type of male remains represented.

Females, since by hypothesis signalling is truthful, attempt to tilt their matings toward the high-quality males. Here is where a "congestion" function is needed, a kind of friction that prevents females from mating exclusively with the high-quality males. Loss of mating capacity in the process of disproportionately tilting her mating choices is what guarantees an interior optimum on the female side.

The Mating Success Function (MSF) specifies how the females respond to higher and lower signalling levels. In the specific formulation used here, the MSF is indexed by a single "signalling decisiveness parameter". This parameter, specifying the degree to which the females attend to signals, plays a role somewhat similar to market price in a supply-demand system. It signifies the "market conditions" that guide and link the choices of the females on the one side and of the H and L males on the other side.

And finally in full biological equilibrium population numbers must be constant. This Malthusian effect is achieved by stipulating that the resources constituting male quality erode as population increases.

In our numerical simulations, the parameters allowed to vary were: (i) The endowed quality of the L-type males in comparison with H-type males. (ii) The unit or marginal cost of signalling for the L-type males, again in comparison with H-type males. (iii) The population proportions of the two male types.

Among the substantive results worth noting are:

1. Equilibria validating the TSH can be achieved over a wide range of parameters, though not universally. The equation system fails to generate such an equilibrium (a) when the unit-cost parameter for the L-type males falls to very low levels, and/or (b) when the fraction of the population represented by the H-type males rise to very high levels. (TSH equilibria might well fail for other parameter variations not explored here, for example if signalling costs rise sufficiently for both H and L type males together.)

2. Contra some statements in the literature, within the ranges where equilibria were achieved it was not a strictly necessary condition that the high-quality males have an advantage in terms of lower per-unit signalling costs. By definition H males always retain an initial advantage in terms of higher endowed quality. So even at a unit-cost disadvantage they are (over some ranges of parameter values) able to signal more heavily.

3. Although the equilibria arrived at were always robust with regard to possible _individual_ deviations, it is a separate question whether the process is "profitable" to the (male or female) population as a whole. For the low-quality males the answer is clear: comparing signalling with the non-signalling equilibrium, they are always worse off. The H-type males can sometimes come out ahead, since their gain in terms of mating success may outweigh the net cost of the handicap. And, if so, it _may_ also be the case that the females do better as well – since their RS is necessarily the average of the success of the male types, weighted by the mating proportions. This might plausibly occur, we conjecture, when: (a) the intrinsic quality difference between the male types is large, or (b) the high-quality males represent only a small fraction of the male population. In either of these cases, being able to tilt toward the high-quality males has a big payoff for the females, in comparison with random mating.

We also looked into the problem of how costly signalling can ever get started from an initial no-signalling equilibrium. The answer seems to be that it cannot, if signalling is always disfunctional (a handicap) For signalling to take off, the trait that serves as a signal must initially (i.e., when expressed at very low levels) be functional rather than a handicap. But a mutation generating such a trait can start a bandwagon away from the no-signalling equilibrium, possibly ending up at equilibrium in the hypertrophy range where the signal is on net a handicap rather than a benefit.

Moving to the bottom line, our analysis shows that it is possible to construct an analytical system for the signalling-handicap problem, on the lines of economic general equilibrium analysis, which shows how the optimizing decisions of the various agents are glued together by the overall "market conditions" that determine the success of males' signals in influencing females' mating decisions.

APPENDIX 1: Male First-Order Conditions

Let the equilibrium signalling levels be denoted s_1 and s_2 and the levels chosen by possibly deviant males s_1' and s_2'. The condition for equilibrium is that the optimizing choices of possible type-1 deviants are such that s_1' is equal to s_1, and similarly for possibly deviating type-2 males.

Consider first potentially deviant type-1 males who constitute a small proportion ϵ of the overall male population. Then the utility functions for the deviating and non-deviating type-1 males become:

$$U_1' = K\left(\frac{m'}{\varepsilon}\right)n_1' \quad \text{where} \quad n_1' = e_1 - b_1 s_1'$$

$$U_1 = K\left(\frac{m}{\pi - \varepsilon}\right)n_1 \quad \text{where} \quad n_1 = e_1 - b_1 s_1$$

(App. 1.A)

To find the optimum for the deviating type-1 males, our formula for signalling competition now must allow for three possible signal levels: the original s_1 and s_2 at their candidate equilibrium values and the s_1' representing the optimum for the type-1 deviants. So it is necessary to generalize the MSF equation (8) to allow the female to respond to three levels of signalling.

For convenience let the symbols α, β, γ index any three population fractions summing to unity. Then in a somewhat old-fashioned but self-evident notation we can write:

$$\frac{m_\alpha}{\pi_\alpha} : \frac{m_\beta}{\pi_\beta} : \frac{m_\gamma}{\pi_\gamma} = s_\alpha^r : s_\beta^r : s_\gamma^r$$

(App. 1.B)

where the m_i as well as the π_i sum to unity.

Making the indicated substitutions for the m_i and n_i and carrying out the maximization leads, after some tedious steps, to:

$$(e_1 - b_1 s_1)\frac{r s_1^{r-1}[(\pi - \varepsilon)s_1^r + (1-\pi)s_2^r]}{\pi s_1^r + (1-\pi)s_2^r} = b_1 s_1^r$$

(App. 1.C)

Letting ϵ go to 0 leads to the First-Order Condition in the text for the type-1 males, and of course the development for the type-2 males corresponds exactly.

APPENDIX 2: Analytic Solution of Initial Version

The equations described here solve for 9 "core variables" and then the 6 "utility variables". The solution strategy involves the following steps:

1. m is solved as a function of the exogenous constants only.
2. r is solved in terms of m and exogenous constants.
3. s_1 and s_2 are solved in terms of r and exogenous constants.
4. n_1 and n_2 are solved in terms of r and exogenous constants.
5. D is solved in terms of m and exogenous constants.
6. M is solved in terms of m, n_1, and n_2.
7. K is solved in terms of D, M, $n1$, and $n2$.

(The solutions for the utility variables then follow trivially.)

[In the interests of saving space, the parameter h that entered into the definition of the marginal congestion effect (7) is here taken as numerically equal to 1 and dropped from the equations.]

Detailing the steps:

1. (This is the crucial step in the analysis.) Inserting (1), (3),(4),(7), and (9)) into (6) provides a second-order equation for m, a reduced form expressed in terms of the exogenous constants only:

$$m = \frac{2\pi e_1/e_2 - (2\pi+1) + \sqrt{3 - (1-\pi)^2 - 2(e_1/e_2)(3-(1-\pi)\pi) + (e_1/e_2)^2(3+\pi^2)}}{3(e_1/e_2 - 1)}$$

(App. 2.A)

One of the interesting implications of (A) is that m, the proportion of female matings going to the high-quality high-signalling males, is (in equilibrium) entirely independent of the signalling cost parameters b_1 and b_2. Also, the equilibrium value of m does not depend upon the absolute endowed qualities e_1 and e_2 but only upon their ratio e_1/e_2.

2. Inserting the ratio s_1/s_2 derived from the Male First-Order Conditions (9) into the Mating Success Function (8), and then solving for r, yields:

$$r = \frac{\ln\left[\dfrac{m(1-\pi)}{(1-m)\pi}\right]}{\ln\left[\dfrac{b_2 e_1}{b_1 e_2}\right]}$$

(App. 2.B)

3. (9) can then be easily solved for the signalling levels s_1 and s_2:

$$s_1 = \frac{e_1 r}{b_1(r+1)} \quad \text{and} \quad s_2 = \frac{e_2 r}{b_2(r+1)}$$

(App. 2.C)

4. Inserting (C) into (1) yields the net qualities of the two male types:

$$n_1 = \frac{e_1}{(r+1)} \quad \text{and} \quad n_2 = \frac{e_2}{(r+1)}$$

(App. 2.D)

Note that the ratios n_1/n_2 and e_1/e_2 are equal, and both also equal s_1b_1/s_2b_2.

5. Entering the solution value of m into equation (7) directly yields the marginal congestion effect D.

6. Similarly, inserting the solution values of $m, n_1,$ and n_2 into (4) yields the average male quality M.

7. And entering the solution values of $n_1, n_2, D,$ and M into (6) yields the female mating capacity K.

These 9 equations taken together express the equilibrium values of the 9 core variables as functions of the exogenous constants only.

APPENDIX 3: Analytic Solution of Extended (Malthusian) Version
In the Malthusian extension the endowed qualities e_1 and e_2 are no longer constants but appear as variables, together with the new variable for population size N. The new equations are the Malthusian stability condition $KM = 1$ plus the definitions of the new variables e_1 and e_2 as functions of E_1, E_2, and N.

Building on the previous Appendix, the solution for the extended model can be spelled out in more cursory fashion. (Only equations with significant changes will be shown.)

1. First, equation (A) is identical with that in the previous Appendix, except for the substitution of the ratio E_1/E_2 for e_1/e_2. (The equivalence of these ratios follows immediately from equations (14) in the text.) As before, the revised equation (A) expresses m as a function of the exogenous parameters only.

2. Equation (B), expressing r in terms of m and the exogenous parameters, is unchanged.

Note: It follows from the above that in numerical simulations for which the assumed ratio E_1/E_2 in the extended model equals the previously assumed ratio e_1/e_2 of the initial model, the solution values for m and r remain unchanged. (As can be seen by inspection of Table 1.)

From here on the equations are different, however.
3. Equation (C) here represents the solutions for the "net" qualities n_1 and n_2. Inserting the Malthusian condition $KM = 1$ into the female F.O.C. (6) yields as an intermediate step:

$$n_1 - n_2 = \frac{-D}{K^2}$$

Making use of the result from the previous Appendix that $n_1/n_2 = e_1/e_2$, which therefore now also equal E_1/E_2, the above can be solved for n_1 and n_2:

$$n_1 = \frac{-DE_1}{K^2(E_1 - E_2)} \quad \text{and} \quad n_2 = \frac{-DE_2}{K^2(E_1 - E_2)} \tag{App.3.C}$$

4. Combining (C) with equation (D) of the previous Appendix yields the solution for the endowed

qualities as variables:

$$e_1 = \frac{-DE_1(r+1)}{K^2(E_1-E_2)} \quad \text{and} \quad e_2 = \frac{-DE_2(r+1)}{K^2(E_1-E_2)} \qquad \text{(App. 3.D)}$$

5. The solutions for the signalling variables can be obtained by inserting these expressions for e_1 and e_2 into equation (C) of the previous Appendix, so that:

$$s_1 = \frac{-DE_1 r}{K^2(E_1-E_2)b_1} \quad \text{and} \quad s_2 = \frac{-DE_2 r}{K^2(E_1-E_2)b_2} \qquad \text{(App. 3.E)}$$

6. Finally, since $E_1/E_2 = e_1/e_2$, we can solve from (D) for the Malthusian population size variable N in equation (13):

$$N^w = \frac{K^2(E_1-E_2)}{-D(r+1)} \qquad \text{(App. 3.F)}$$

As before, the solutions for the utility variables follow straightforwardly.

Table 1 – BASELINE SIMULATION RESULTS, MODEL 1

	Initial Model	Extended Model (Malthusian)	Start-up Model
Constants			
π	0.5	0.5	0.5
e1 or E1	e1 = 2.0	E1 = 1	E1 = 1.0
e2 or E2	e2 = 1.0	E2 = 0.5	E2 = 0.5
b1 or a1	b1 = 0.5	b1 = 0.5	a1 = 0.5
b2 or a2	b2 = 1.0	b2 = 1.0	a2 = 1.0
g1	--	--	0.8
g2	--	--	0.2
Variables			
e1	(constant - see above)	2.250	1.446
e2	(constant - see above)	1.125	0.723
m	0.764	0.764	0.822
s1	1.834	2.063	1.807
s2	0.458	0.516	0.626
n1	1.083	1.219	1.259
n2	0.542	0.609	0.456
K	0.930	0.930	0.896
M	0.955	1.075	1.116
r	0.846	0.846	1.446
N	--	0.444	0.692
U1	1.539	1.732	1.855
U2	0.238	0.268	0.145
UF	0.889	1.0	1.0
U1*	2.0	2.250	1.446
U2*	1.0	1.125	0.723
UF*	1.5	1.689	1.084

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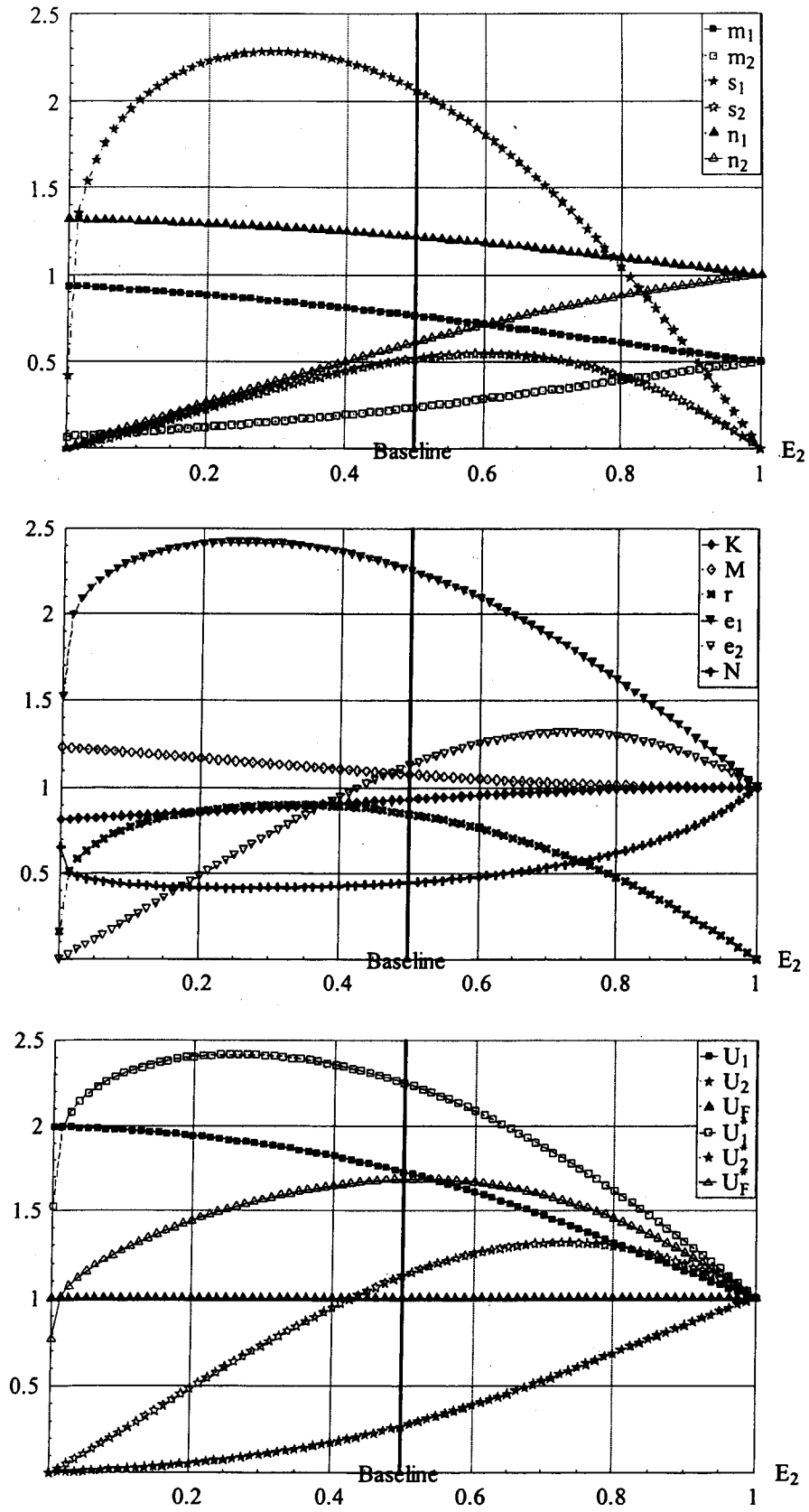
Fig 1. ENDOGENOUS VARIABLES AS FUNCTIONS OF THE L MALE'S ENDOWED QUALITY (E_2), when E_1=1,b_1=0.5,b_2=1,π=0.5,h=1,w=1.
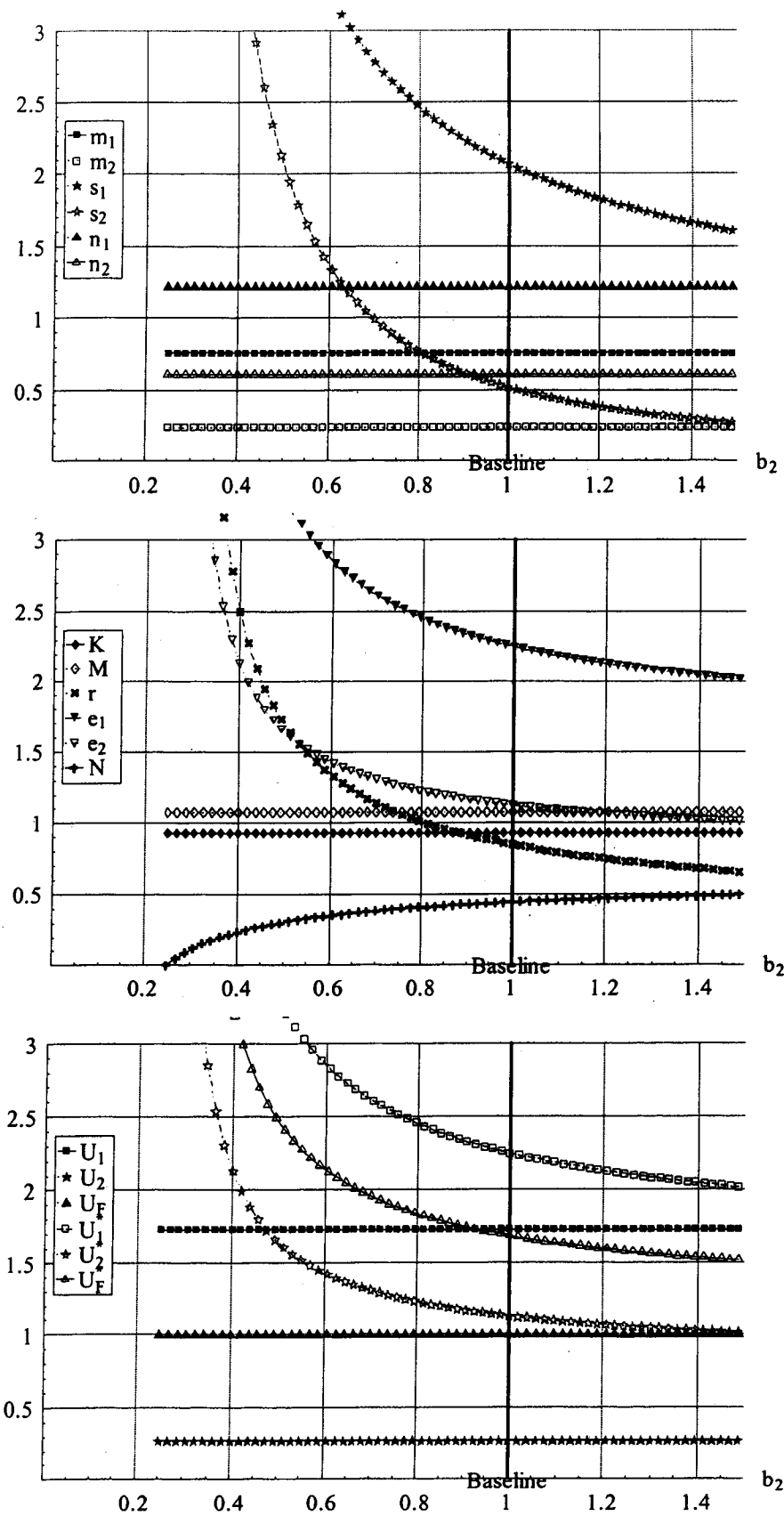
Fig 2. ENDOGENOUS VARIABLES AS FUNCTIONS OF THE L MALE'S UNIT
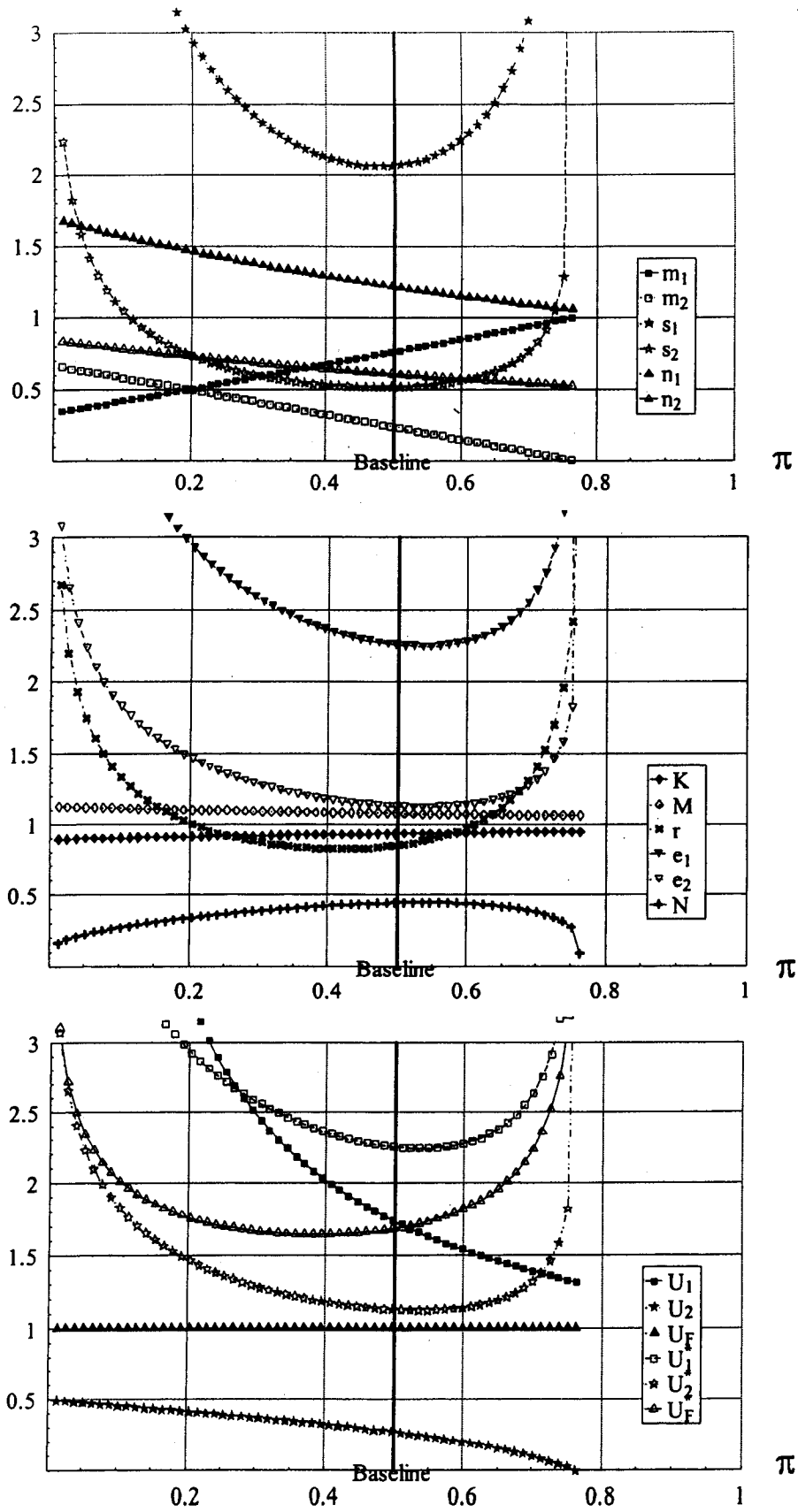SIGNALLING COST (b_2), when $E_1=1, E_2=0.5, b_1=0.5, \pi=0.5, h=1, w=1$.

Fig 3. ENDOGENOUS VARIABLES AS FUNCTIONS OF THE POPULATION PROPORTION (π) OF H MALES, when $E_1=1, E_2=0.5, b_1=0.5, b_2=1, h=1, w=1$.