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THE HANDICAP PRINCIPLE IN PARENT-OFFSPRING CONFLICT: COMPARISON OF OPTIMALITY AND POPULATION-GENETIC ANALYSES

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Abstract.—The evolution of offspring handicap is studied in two ways. First, the problem is formulated in terms of a population game in which each player, parent and offspring, seeks to increase its own fitness. Second, we study the dynamics of an exact two-locus model in which one locus affects the behavior of offspring and the other affects that of parents. It is shown that the latter approach leads to a more complicated "game structure," in which parents maximize a weighted average number of offspring, with lower weight ascribed to the handicapped type. The weights depend on the rate of recombination. Under the assumption of fitness maximization, used in the game-theoretical approach, it is shown that a handicap always evolves. The two-locus analysis, however, produces a more realistic set of specific conditions for initial success and fixation of the handicap. When linkage is loose, these latter conditions coincide with a verbal prediction of Zahavi. With tight linkage, however, conditions for this evolution of offspring handicap are significantly restricted.

Parent-offspring conflict describes potential differences between the fitness objectives of parents and offspring. The antagonists in this conflict are, on the one hand, parents, whose fitness increases when the limited resources they provide are used more efficiently by offspring and, on the other hand, offspring, whose chance of survival relative to sibs would increase if they were able to sequester a larger fraction of the available resources. Discussions of this potential conflict by Alexander (1974), Trivers (1974), West Eberhard (1975), and Zahavi (1975, 1977, 1981, 1987) have not produced a consistent evolutionary scenario to describe how offspring that acquire a larger fraction of parental resources (to the apparent detriment of parental fitness) might evolve. Offspring of this type are called "handicapped," a terminology originated by Zahavi. In some contexts such offspring might be called "selfish" to distinguish them from less demanding or "altruistic" members of their cohort.

Zahavi's original argument (1975) was couched in terms of characters that are preferred by one sex in their choice of mates but whose possession by the chosen sex is disadvantageous to survival. He suggested that the handicap in survival might serve as a marker of mate quality and might, in the overall accounting of fitness, confer a net advantage to the handicapped individuals. In the theory of sexual selection, this verbal argument is rather controversial (see, e.g., review in

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Vehrencamp and Bradbury 1984). The interpretation of handicap may be broadened, however, to include any conflict in the form of a nonzero-sum game in which a new game structure is established by reducing the options of one player who receives a higher payoff at the Nash solution to the new game (see, e.g., Ben-Porath and Dekel 1988 and references therein). The relevance of this somewhat curious result from game theory to the theory of evolution is that in some cases in which natural selection has led to an optimum, it may be easy to produce natural conditions that lead to departure from this optimum. In the case of parent-offspring conflict (Zahavi 1981, 1987), the argument would be that by reducing its own viability under some circumstances, a handicapped offspring can create a situation in which its parent, in order to increase its own fitness, must invest more in that handicapped offspring. Under some conditions this extra investment by the parent could produce a fitness that is higher for the handicapped offspring than for a normal one. As a consequence, handicaps of this kind might then evolve.

In order to make these arguments more precise, suppose that the probability that a normal offspring survives to maturity is an increasing function $v(x)$ of the amount of resources, x, invested in that offspring by its parents. Here, x is considered a fraction of the total resources available to the brood. If the parent invests a fraction x of its resources in each offspring, then the expected number of offspring for that parent (surviving or not) is x^{-1}, and the expected number of surviving offspring, which is a reasonable representation of the parents' fitness, is (see, e.g., Parker and Macnair 1978)

$$w(x) = v(x)/x. \tag{1}$$

A rather plausible assumption is that $w(x)$ is maximized at some optimal investment x^* with

$$\left.\frac{dw}{dx}\right|_{x=x^*} = \frac{x^*v'(x^*) - v(x^*)}{(x^*)^2} = 0;$$

thus,

$$v'(x^*) = v(x^*)/x^* \tag{2}$$

(Parker and Macnair 1978). In addition, we require that

$$\left.\frac{d^2w}{dx^2}\right|_{x=x^*} = \frac{v''(x^*)}{x^*} - \frac{2v'(x^*)}{(x^*)^2} + \frac{2v(x^*)}{(x^*)^3} < 0,$$

and we have, using equation (2),

$$v''(x^*) < 0. \tag{3}$$

One way to state Zahavi's claim quantitatively is in terms of another function, $u(x)$, the survival function for the handicapped offspring, with $u(x) < v(x)$. Here, $v(x) - u(x)$ could be called the handicap. First, it is easy to show that for any survival function of a normal offspring, $v(x)$, a handicapped survival function $u(x)$

$\leq v(x)$ exists, such that when the parent of handicapped offspring has no normal offspring in its brood, a value y^* may be found, which maximizes $u(y)/y$ and which satisfies $u(y^*) > v(x^*)$, where x^* is defined in equations (2) and (3).

In order to see that this claim is correct, we introduce an infinitesimal local handicap, specified by a handicapped-survival function $u(y)$ defined in the vicinity of x^* by the relations

$$u(x^*) = v(x^*) - \epsilon, \tag{4}$$

$$u'(x^*) = v'(x^*) + \delta, \tag{5}$$

and

$$u''(x^*) = v''(x^*) = -\alpha < 0, \tag{6}$$

where α is a positive number determined by v, $\epsilon > 0$ and small, and $\delta > 0$ and small. For y in the vicinity of x^*, $u(\cdot)$ is a smooth, monotonically increasing function satisfying

$$u(y) = v(y) - \epsilon + (y - x^*)\delta + o(y - x^*); \tag{7}$$

outside this vicinity, we assume only that $u(y) \leq v(y)$. (For example, we might assume that $u(y) = v(y)$ for $y \geq x^* + \epsilon/\delta$ and that $u(y) = v(y) - 2\epsilon$ for $y \leq x^* - \epsilon/\delta$.)

In simple biological terms, $u(\cdot)$ may be interpreted as a survival function of an offspring that tends to beg for food more loudly than a normal offspring, thus increasing by 2ϵ (>0) its chance of attracting predators (see, e.g., Zahavi 1981, p. 320). In this case, the value δ determines the rate of decrease in the offspring's begging activity as the parent increases its share of the resources above x^*.

By a straightforward calculation, it may be verified that as long as

$$\delta = \epsilon^\theta \tag{8}$$

for $\frac{1}{2} < \theta < 1$ (i.e., as long as the offspring reduces its begging activity at an appropriate rate), then

$$y^* = x^* + \delta/\alpha + o(\delta) > x^*. \tag{9}$$

Moreover, even though $v(y^*) > u(y^*)$, we have

$$u(y^*) > v(x^*). \tag{10}$$

In other words, if a parent raises one offspring at a time and if parents estimate that future offspring will be equivalent to the present one, parents' optimal allocation of resources to a handicapped offspring, y^*, would ensure that the survival probability of such an offspring, $u(y^*)$, is higher than that of a normal one, $v(x^*)$.

Result 1.—Suppose that $v(x)$ is a survival function such that x^* maximizes the parents' fitness, $v(x)/x$. Then there is a handicapped-survival function, u, with $u(x) < v(x)$ such that at the maximum of the parents' fitness, y^*, the fitness $u(y^*)$ of the handicapped offspring exceeds that of the normal offspring at x^*, $v(x^*)$.

This does not mean that natural selection favors the handicapped offspring type when it is in a state of dynamic coexistence with the normal type. Indeed, since

x^* maximizes $v(x)/x$ and since $u(y^*) < v(y^*)$,

$$u(y^*)/y^* < v(y^*)/y^* < v(x^*)/x^*; \tag{11}$$

thus, in the state of dynamic coexistence, parents with normal broods are fitter (i.e., have larger broods) than those with handicapped offspring.

Parents with mixed broods have alternatives among which to choose in positioning their resources. A crucial question here is whether natural selection, operating on parental allocation of resources, necessarily favors a partition that maximizes a parent's total number of surviving offspring (its "fitness"). It is a tacit but basic assumption in Zahavi's argument that fitness is maximized. Aside from the general problem of the relationship between natural selection and optimization, relation (11) means that if the handicap is heritable (a necessary condition for its evolution by natural selection), then, since handicapped offspring tend to have more handicapped offspring of their own, handicapped offspring should have lower fitness (as parents) than nonhandicapped offspring. Thus, the criterion for maximization of offspring number may be different from the criterion that maximizes grandoffspring number.

If we assume that there is one gene for the handicap and a second that affects parental behavior, then the proportion of nth-generation descendants of a handicapped offspring depends on the recombination fraction between the loci. Indeed, the reproductive value of a handicapped offspring (see, e.g., Eshel 1984, p. 205; Taylor 1988) should depend on the recombination fraction. Nevertheless, it is intuitive that such a measure of fitness should be lower than for a normal offspring. Thus, the tacit assumption that parents' fitness is maximized warrants further examination.

In the next section we address the single-locus problem of initial success and fixation stability of the handicap under the assumption that parents do seek to maximize their own fitness. We then introduce an exact two-locus model in order to determine from the dynamics which sort of maximization criterion, if any, characterizes the system. In a sense this addresses the issue raised by Trivers (1974) and Charnov (1982), namely, the potential genetic disagreement between parent and offspring. Finally, we reexamine the handicap principle in light of our results from the dynamic model.

THE PARENT-OFFSPRING CONFLICT AS A POPULATION GAME FOR FITNESS MAXIMIZATION

One way to approach the possibility that handicap in offspring may evolve is to pose the problem as an asymmetrical population game between parents and offspring. Each is supposed to attempt to maximize some relevant payment function, given the opponent's strategy. In this section we follow Zahavi's postulate that the mother seeks to maximize her expected number of surviving offspring.

Suppose that the handicapped type of offspring appears first as a rare dominant mutation. Then, while rare, it appears almost exclusively in broods of which one parent is heterozygous. Such broods contain half normal and half handicapped offspring, and we assume that mothers of these broods allocate proportions x and

y of their resources to normal and handicapped offspring, respectively. As a result, the fitness of such a mother is

$$w(x,y) = \frac{v(x) + u(y)}{x + y}. \tag{12}$$

This fitness is maximized at $\hat{x},\hat{y}$, such that $\partial w/\partial x = \partial w/\partial y = 0$, which is equivalent to

$$v'(\hat{x}) = u'(\hat{y}) = \frac{v(\hat{x}) + u(\hat{y})}{\hat{x} + \hat{y}}. \tag{13}$$

Recall that the mother's strategy toward offspring in an all-normal brood should be x^*, where x^* satisfies equation (2). Now assume that the mother adopts the strategy $\hat{x},\hat{y}$ that maximizes her fitness with respect to mixed broods and that the total amount of resources available to the parent for offspring production is constant and does not depend on the quality of the offspring. Then, the survival probability of a heterozygous handicapped offspring is $u(\hat{y})$. Since such an offspring will also have a mixed brood, its average expenditure on each of its offspring is $(\hat{x} + \hat{y})/2$, and its brood size (provided that it survives) will therefore be $2/(\hat{x} + \hat{y})$. Thus, the fitness of handicapped individuals may be represented by $2u(\hat{y})/(\hat{x} + \hat{y})$. We determine whether the handicapped type can succeed according to whether

$$\frac{2u(\hat{y})}{\hat{x} + \hat{y}} > \frac{v(x^*)}{x^*}. \tag{14}$$

In the same way, a population of handicapped individuals resists invasion by normals if

$$\frac{2v(\hat{x})}{\hat{x} + \hat{y}} < \frac{u(y^*)}{y^*}, \tag{15}$$

where y^* maximizes $u(y)/y$, and $\hat{x},\hat{y}$ maximizes $w(x,y)$ as in equation (13).

As in the preceding section, we use the perturbed handicap function defined by equations (4)–(8). Using the notation $\hat{x},\hat{y}$ for the solution of equation (13),

$$\hat{x} = x^* + \xi, \qquad \hat{y} = x^* + \eta. \tag{16}$$

From equation (2), neglecting terms $o(\xi)$ and $o(\eta)$, we have

$$v'(\hat{x}) = v'(x^*) - \alpha\xi = v(x^*)/x^* - \alpha\xi, \tag{17}$$

$$u'(\hat{y}) = v'(x^*) - \alpha\eta + \delta = v(x^*)/x^* - \alpha\eta + \delta. \tag{18}$$

Furthermore,

$$v(\hat{x}) = v(x^*) + \xi v'(x^*) = v(x^*) + v(x^*)\,\xi/x^*,$$

and

$$u(\hat{y}) = v(x^*) + [v(x^*)/x^* + \delta]\,\eta - \epsilon.$$

Thus,

$$\frac{v(\hat{x}) + u(\hat{y})}{\hat{x} + \hat{y}} = \frac{2v(x^*) + (\xi + \eta)\, v(x^*)/x^* + \delta\eta - \epsilon}{2x^* + \xi + \eta} \tag{19}$$
$$= v(x^*)/x^* + (\delta\eta - \epsilon)/2x^*,$$

neglecting the terms $o(\xi)$ and $o(\eta)$. Combining equations (13) and (17)–(19), we conclude that

$$-\alpha\xi = -\alpha\eta + \delta = (\delta\eta - \epsilon)/2x^*;$$

thus,

$$\eta = \frac{2\delta x^* + \epsilon}{2\alpha x^* + \delta} = \frac{\delta}{\alpha} + \frac{\epsilon}{2\alpha x^*} + o(\delta), \tag{20}$$

and

$$\xi = \epsilon/2\alpha x^* + o(\delta,\eta). \tag{21}$$

According to our previous assumptions (4)–(8), $\delta^2 < \epsilon < \delta$; thus, $\eta = \delta/\alpha + o(\delta) < \epsilon/\delta$ and $\xi = o(\delta)$. Hence,

$$\begin{aligned} u(\hat{y}) &= v(x^*) + \eta v'(x^*) + \delta\eta - \epsilon + o(\eta) \\ &< v(x^*) + \eta v'(x^*) + o(\eta) \\ &= v(\hat{y}), \end{aligned} \tag{22}$$

which establishes that u is indeed a handicapped-survival function in the relevant region $(x^*,\hat{y})$. Furthermore,

$$\begin{aligned} \frac{2u(\hat{y})}{\hat{x} + \hat{y}} &= \frac{2v(x^*) + 2[v(x^*)/x^* + \delta]\eta - 2\epsilon}{2x^* + \xi + \eta} \\ &= \frac{v(x^*)}{x^*} + \frac{v(x^*)}{2(x^*)^2}\eta + \frac{\delta\eta - \epsilon}{x^*} + o(\delta) \\ &= \frac{v(x^*)}{x^*} + \frac{v(x^*)}{2\alpha(x^*)^2}\delta + o(\delta) \\ &> \frac{v(x^*)}{x^*}. \end{aligned} \tag{23}$$

Thus, condition (14) for the invasion by the handicapped type is satisfied for any values of $v(x^*)$, x^*, and α with the appropriate small perturbation satisfying $\delta > \epsilon > \delta^2$.

Result 2.—For any survival function of the normal offspring, $v(x)$, we can find a slightly perturbed handicapped-survival function $u(x)$, with $u(x) < v(x)$, such that, provided that parents act to maximize their own fitness, the handicapped mutant type characterized by $u(x)$ will initially increase.

In order to examine invasion by normal individuals from fixation of the handi-

capped type, return to equations (7) and (9), where we have, to $o(\epsilon)$,

$$u(y^*)/y^* = v(x^*)/x^* + (\delta^2/\alpha - \epsilon)/x^* . \tag{24}$$

From equations (20) and (21), we also have, to $o(\epsilon)$,

$$\begin{aligned} \frac{2v(\hat{x})}{\hat{x} + \hat{y}} &= \frac{2v(x^*) + v(x^*)\epsilon/\alpha(x^*)^2}{2x^* + \delta/\alpha + \epsilon/\alpha x^*} \\ &= \frac{v(x^*)}{x^*} - \frac{v(x^*)}{2\alpha(x^*)^2}\delta + o(\delta) . \end{aligned} \tag{25}$$

Condition (15) follows from equations (24) and (25), since $\delta^2 < \epsilon < \delta$.

Result 3.—Under the conditions of the perturbation described above, if parents maximize their own fitness, a handicap $u(x)$ permits invasion by the handicapped type when it is rare and prevents invasion by the normal type when the handicapped type is common.

It is important that despite these results 2 and 3, we have

$$\begin{aligned} \frac{v(\hat{x}) + u(\hat{y})}{\hat{x} + \hat{y}} &= \frac{v(x^*)}{x^*} + \frac{\delta^2 - \alpha\epsilon}{2\alpha x^*} + o(\delta) \\ &< v(x^*)/x^* . \end{aligned} \tag{26}$$

The right side of the inequality is the expected number of surviving offspring within a completely normal brood; since the handicapped type is rare, it approximately equals the number of surviving grandoffspring produced by a normal offspring, if it survives. The left side is the expected number of surviving offspring in a mixed brood, which is approximately the number of surviving grandoffspring produced by a handicapped offspring, if it survives. Thus, equation (26) means that the strategy $\hat{x},\hat{y}$, which maximizes the parent's expected number of surviving offspring, does not maximize its expected number of grandoffspring. The latter might be augmented by, for example, increasing the proportion of parental resources allocated to normal offspring. But in this case, the optimal number of grandoffspring would be different again.

A natural solution to this optimization dilemma has been suggested by Taylor (1988; see also Oster et al. 1977). This involves the assumption that the ratio of the reproductive values of handicapped and normal offspring should be some value z. Then, as long as the handicapped type is rare, the total reproductive value of the offspring of a handicapped individual (if it survives) may be written in terms of $\hat{a} = v(\hat{x})/(\hat{x} + \hat{y})$ and $\hat{b} = u(\hat{y})/(\hat{x} + \hat{y})$ as

$$[v(\hat{x}) + zu(\hat{y})]/(\hat{x} + \hat{y}) = \hat{a} + z\hat{b}, \tag{27}$$

which should be compared with the total reproductive value

$$v(x^*)/x^* = 2a^* \tag{28}$$

of the offspring of a surviving normal offspring. Hence, reconciling equations (27) and (28) and the definition of z, we must have $2a^*/(\hat{a} + z\hat{b}) = 1/z$, giving the

solution

$$\hat{z} = \hat{a}/(2a^* - \hat{b}). \tag{29}$$

This suggests that any optimization argument should include different weights for the handicapped and normal offspring, with the ratio $\hat{z}$ between them given by equation (29). Note, however, that the reproductive values themselves depend on the parental strategy $\hat{x},\hat{y}$ that is to be optimized. Furthermore, if parental behavior is under selection, the chance that some descendant carrying a specific allele affecting this behavior also carries an allele for offspring handicap (and therefore, by inequality [26], has fewer surviving offspring) depends on the recombination fraction between the loci that affect parental and offspring behavior. In order to clarify the relationship between the optimality approach described above and the dynamic approach, we introduce a two-locus model that simultaneously incorporates natural selection acting on parental and offspring behavior.

DYNAMIC TREATMENT OF PARENTS' BEHAVIOR: A TWO-LOCUS MODEL

In an earlier study (Feldman and Eshel 1982), we analyzed a formal two-locus genetic model that describes a potential conflict between the fitness objectives of parents and offspring. The model we now describe also includes two loci and involves a substantial extension of our previous work. It is assumed that one locus with alleles A and a determines the dichotomy between handicapped and normal offspring. A second locus with alleles B and b determines the parents' behavior toward both types of offspring. This behavior may be characterized by four categories for the recipient of parental investment: offspring in completely normal broods, offspring in completely handicapped broods, and each type of offspring in mixed broods. The recombination rate between the two genes is r.

We assume first that only one parent—say, the mother—invests in offspring (later the analysis is extended to allow both parents to contribute). Initially, the population is close to fixation of BB and AA, where individuals of genotype AA are normal and have the survival function $v(x)$. A very small fraction of handicapped individuals, of genotype Aa, has the survival function $u(x)$, with $u(x) < v(x)$. The genotype BB determines a maternal strategy $x^*,y^*,\hat{x},\hat{y}$. That is, such a mother invests x^* and y^* in each offspring from completely normal and completely handicapped broods, respectively, and $\hat{x}$ and $\hat{y}$, respectively, in normal and handicapped offspring from mixed broods.

The question is whether, for any handicap function $u(x) < v(x)$, there exists a maternal investment strategy that (1) prevents initial success of the u-type handicap, (2) is unbeatable by any mutation at the B locus (see, e.g., Hamilton 1967), and (3) occurs while the handicap is maintained at low frequency by mutation-selection balance. We therefore begin by assuming that the u-type handicap is at low frequency in such a mutation-selection balance and proceed to demonstrate and characterize the unbeatable maternal strategy $x^*,y^*,\hat{x},\hat{y}$. Such a strategy, determined by the genotype BB, cannot be invaded by any alternative strategy determined by a Bb mutant. Without loss of generality, we may assume that both BB and Bb mothers adopt the same optimal strategies, x^* and y^*, when their

broods are homogeneous. In this case, the alternative maternal strategies may be characterized by just the pair $\hat{x},\hat{y}$ for a *BB* mother and x,y for a *Bb* mother.

In this section we see that, under the assumption that the normal type is near fixation and a handicapped type is maintained at mutation-selection balance, an unbeatable maternal strategy $\hat{x},\hat{y}$ always exists. Moreover, it is uniquely determined by the handicap function, $u(x)$, given the survival function $v(\cdot)$ of the normal type. Hence, if the fact that the maternal strategy is unbeatable implies that fixation of the normal type is unstable, then it must be true that no combination of maternal and offspring strategies can resist all mutations as long as the resident offspring type is normal. Under these conditions, we may conclude that the handicapped type will eventually succeed. By contrast, if the condition that the mother's strategy is unbeatable implies that fixation of the normal type is stable, then the handicapped type is unlikely to become established in the population.

We begin by assuming that at fixation of the genotype *BB*, which determines the maternal investment strategy $\hat{x},\hat{y}$, there is a mutation-selection balance at which a low frequency of $u(\cdot)$-type handicapped individuals of genotype *Aa* is maintained in a population that is almost fixed on the normal *AA* genotype. We use three relative fitnesses:

$$a^* = v(x^*)/2x^*, \qquad \hat{a} = v(\hat{x})/(\hat{x} + \hat{y}), \qquad \hat{b} = u(\hat{y})/(\hat{x} + \hat{y}), \tag{30}$$

where a^* refers to normal offspring in a completely normal brood, $\hat{a}$ to a normal offspring in a mixed brood, and $\hat{b}$ to a handicapped offspring in a mixed brood, all given the mother's strategy $\hat{x},\hat{y}$ (see also Parker 1984). Accordingly, if the rate of mutation is sufficiently low, then the frequency of the heterozygote *Aa* at the mutation-selection balance is approximately $2p$, where $p = a^*\mu/(a^* - \hat{b})$. For convenience, we use

$$\mu = (a^* - \hat{b})p/a^*. \tag{31}$$

Homozygotes *aa* are sufficiently rare that they may be neglected.

In the neighborhood of this mutation-selection balance, a mutation from *B* to *b* appears, such that the strategy of the mutant mother, as a heterozygote, is x,y. Let

$$a = v(x)/(x + y), \qquad b = u(y)/(x + y), \qquad c^* = u(y^*)/2y^*, \tag{32}$$

and let ϵ_1, ϵ_2, ϵ_3, and ϵ_4 be the frequencies of the mutant heterozygous mothers of genotypes *AB/Ab*, *aB/Ab*, *AB/ab*, and *aB/ab*, respectively. In table 1 are listed all matings in which one party is a mutant heterozygote *Bb* and the other is *BB*, and both are either *AA* or *Aa* at the other locus. In such matings, the probability is 0.5 that the mother is *Bb* and, therefore, employs the strategy x,y. The probability is also 0.5 that the father is *Bb*, in which case the mother employs the resident strategy $\hat{x},\hat{y}$. Denote by

$$\tilde{a} = (a + \hat{a})/2 \qquad \text{and} \qquad \tilde{b} = (b + \hat{b})/2 \tag{33}$$

the average number of surviving *AA* and *Aa* offspring, respectively, from *AA* × *Aa* matings in which one of the parents is *Bb*. Let $c_1/2$, c_2, and $c_3/2$ be the

TABLE 1

MATINGS INVOLVING HETEROZYGOSITY AT ONE LOCUS, *Bb*

MATING TYPE	FREQUENCY	OFFSPRING SURVIVING			
		AB/Ab	*aB/Ab*	*AB/ab*	*aB/ab*
AB/AB × *AB/Ab*	$2(1-2p)\epsilon_1$	a^*	0	0	0
AB/AB × *aB/Ab*	$2(1-2p)\epsilon_2$	$\tilde{a}(1-r)$	0	$\tilde{b}r$	0
AB/AB × *AB/ab*	$2(1-2p)\epsilon_3$	$\tilde{a}r$	0	$\tilde{b}(1-r)$	0
AB/AB × *aB/ab*	$2(1-2p)\epsilon_4$	0	0	c^*	0
AB/aB × *AB/Ab*	$4p\epsilon_1$	$\tilde{a}/2$	$\tilde{b}/2$	0	0
AB/aB × *aB/Ab*	$4p\epsilon_2$	$c_1(1-r)/2$	$c_2(1-r)/2$	$c_2r/2$	$c_3r/2$
AB/aB × *AB/ab*	$4p\epsilon_3$	$c_1r_1/2$	$c_2r/2$	$c_2(1-r)/2$	$c_3(1-r)/2$
AB/aB × *aB/ab*	$4p\epsilon_4$	0	0	$c^*/2$	$c^*/2$

average number of surviving *AA*, *Aa*, and *aa* offspring, respectively, from *Aa* × *Aa* matings in which one of the parents is *Bb*, and let $c^* = u(y^*)/2y^*$ be the average fitness of a completely handicapped brood. No assumptions are necessary for c_1, c_2, c_3, or c^* since they play no role in the analysis. For the time being we ignore the effect of the flux of mutations from *A* to *a* on the distribution of the four *Bb* offspring genotypes.

Sum the columns of the table and divide the results by the average rate of increase of the population, *W*. The proportions $\tilde{\epsilon}_1$, $\tilde{\epsilon}_2$, $\tilde{\epsilon}_3$, and $\tilde{\epsilon}_4$ of the *Bb* genotypes in the next generation are then given by

$$W\tilde{\epsilon}_1 = (2a^*p_1 + \tilde{a}p_2)\epsilon_1 + (1-r)(2\tilde{a}p_1 + c_1p_2)\epsilon_2 + r(2\tilde{a}p_1 + c_1p_2)\epsilon_3, \quad (34)$$

$$W\tilde{\epsilon}_2 = \tilde{b}p_2\epsilon_1 + 2c_2p_2(1-r)\epsilon_2 + 2c_2p_2r\epsilon_3, \quad (35)$$

$$W\tilde{\epsilon}_3 = (2\tilde{b}p_1 + c_2p_2)r\epsilon_2 + (2\tilde{b}p_1 + c_2p_2)(1-r)\epsilon_3 + c^*(2p_1 + p_2)\epsilon_4, \quad (36)$$

$$W\tilde{\epsilon}_4 = 2c_3p_2r\epsilon_2 + 2c_3p_2(1-r)\epsilon_3 + c^*p_2\epsilon_4, \quad (37)$$

where $p_1 = 1 - 2p$ and $p_2 = 2p$. In the resident population, which is *BB*, a fraction $2p$ is *Aa*. Thus, approximately $4p$ of all matings involve *Aa* parents and so produce mixed broods. Thus, we may use the approximation

$$\begin{aligned} W &= (1-4p)v(x^*)/x^* + 4p[v(\hat{x}) + v(\hat{y})]/(\hat{x} + \hat{y}) \\ &= 2a^* + 4(\hat{a} + \hat{b} - 2a^*)p. \end{aligned} \quad (38)$$

To complete the recursion to the next generation we must incorporate the effect of mutation, at rate μ from *A* to *a*, on the frequencies of the four *Bb* genotypes. After mutation we have, ignoring terms $o(\mu)$,

$$\epsilon_1' = (1-2\mu)\tilde{\epsilon}_1, \quad (39)$$

$$\epsilon_2' = (1-\mu)\tilde{\epsilon}_2 + \mu\tilde{\epsilon}_1, \quad (40)$$

$$\epsilon_3' = (1-\mu)\tilde{\epsilon}_3 + \mu\tilde{\epsilon}_1, \quad (41)$$

$$\epsilon_4' = \tilde{\epsilon}_4 + \mu(\tilde{\epsilon}_2 + \tilde{\epsilon}_3). \quad (42)$$

The analysis of the linear transformation (39)–(42) is presented in Appendix A, where we show that the condition for the stability of the strategy $\hat{x},\hat{y}$ against any invading strategy boils down to

$$H = H(\hat{x},\hat{y};\, x,y) < 0\,, \tag{43}$$

where

$$\begin{aligned} H = {} & a^*[a^* - (b + \hat{b})(1 - r)/2](a - \hat{a}) \\ & + (a + \hat{a})[a^* - (b + \hat{b})(1 - 2r)/2](b - \hat{b})/4\,. \end{aligned} \tag{44}$$

Here $\hat{a}$ and $\hat{b}$ are given in equations (30) as functions of $\hat{x}$ and $\hat{y}$, and a and b are given in equations (32) as functions of x and y.

We seek a maternal strategy $\hat{x},\hat{y}$ that is unbeatable at least under our conditions of a mutation-selection balance near fixation of the normal type AA. In other words, we seek a pair $\hat{x},\hat{y}$ such that, for all $x,y \neq \hat{x},\hat{y}$, inequality (43) is satisfied. But from equation (44) it follows immediately that $H(\hat{x},\hat{y};\, \hat{x},\hat{y}) = 0$.

Result 4.—The maternal strategy $\hat{x},\hat{y}$ resists all invaders determined by mutations at the B locus, when invasion occurs near fixation of the normal AA type, if and only if $H(\hat{x},\hat{y};\, x,y)$ as defined in equation (44) attains its maximum in x and y at $x,y = \hat{x},\hat{y}$.

We may state this result equivalently as follows: $\hat{x},\hat{y}$ resists all mutations (under the same conditions) if and only if it is an evolutionarily stable strategy (ESS; see Maynard Smith and Price 1973) of a formal population game in which the payment function to an individual playing x,y against an opponent playing $\hat{x},\hat{y}$ is H (eq. [44]). (See similar results in Eshel and Sansone 1991.)

It is critical to note, however, that the formal payment function (eq. [44]), deduced from an exact analysis of the two-locus model, is by no means equivalent to the intuitive "fitness" or inclusive-fitness functions that are traditional in ESS analyses, specifically: $a - \hat{a} = v(x)/(x + y) - v(\hat{x})/(\hat{x} + \hat{y})$, the (positive or negative) advantage of the mutant Bb mother over the resident BB population in terms of the total number of surviving normal offspring in a mixed brood. Likewise, $b - \hat{b}$ is the same advantage measured in terms of surviving handicapped offspring in a mixed brood. Since both kinds of mothers behave in the same way toward a homogeneously normal brood, the condition that $H(\hat{x},\hat{y};\, x,y)$ be maximized at $x,y = \hat{x},\hat{y}$ would appear to involve maximization of a weighted average between the normal and handicapped offspring. As can be seen from equation (44), the weights given to $a - \hat{a}$ and to $b - \hat{b}$ are unequal, and parents are not selected just to maximize their total number of surviving offspring. For the case $r = \frac{1}{2}$, we find from equation (44) that

$$H = a^*(a^* - \tilde{b}/2)(a - \hat{a}) + a^*\tilde{a}(b - \hat{b})/2\,;$$

thus, the relative weight of a handicapped offspring turns out to be $\tilde{a}/(2a^* - \tilde{b})$. Taking account of the difference between uniparental and biparental investments, this result is exactly the same as equation (29), above (computed by Taylor's method), for the relative reproductive value of the handicapped offspring. Note, however, that because the "weights" in our treatment are functions of x,y and

$\hat{x},\hat{y}$, maximization of H as a function of x,y is not equivalent to maximization of the sum of offspring reproductive values. Note further that in the general case, these weights depend critically on the recombination fraction between the two loci.

To conclude this section, we briefly study the case in which both parents are equally likely to interfere in the allocation of resources to an offspring. The analysis of this case can be shown to be quite similar to the one just completed. Assume, as before, that the strategy of a couple in which both parties are *BB* is $\hat{x},\hat{y}$, and that, if one is *Bb*, then the strategy is x,y. Again, we seek conditions under which *BB* will not be invaded by any mutation at the *B* locus. Repetition of the preceding analysis produces a local stability matrix that exactly equals matrix (A2) of Appendix A but with the values $\bar{a}$ and $\bar{b}$ replaced by a and b, respectively. (In addition, there are adjustments to $L_1, L_2, \ldots, L_{10}$.) In this way it can be verified that $\hat{x},\hat{y}$ is stable if and only if

$$\begin{aligned} G &= a^*[a^* - b(1 - r)](a - \hat{a}) + a[a^* - b(1 - 2r)](b - \hat{b})/2 \\ &= G(\hat{x},\hat{y}; x,y) \end{aligned} \tag{45}$$

is maximized at $x,y = \hat{x},\hat{y}$.

Result 5.—The parents' strategy $\hat{x},\hat{y}$ will not be invaded by any mutant strategies if and only if $\hat{x},\hat{y}$ is an ESS in a population game where G (defined in eq. [45]) is the payment function of a player choosing strategy x,y against an opponent playing $\hat{x},\hat{y}$.

Again, for $r = \frac{1}{2}$,

$$G = a^*(a^* - b/2)(a - \hat{a}) + aa^*(b - \hat{b})/2\,,$$

and the relative weight of the contribution by a handicapped offspring to that of a normal one is $\hat{a}/(2a^* - \hat{b})$, in agreement with the value from equation (29), calculated by Taylor's method for the relative reproductive value of the handicapped offspring.

As we see below, the functions H and G are maximized at exactly the same values. It follows that the assumption of uniparental or biparental interference does not affect the results.

A REEXAMINATION OF THE DYNAMIC CONDITION FOR INITIAL INCREASE AND FIXATION STABILITY OF THE HANDICAP

Our assumption (to be relaxed below) is that the population is initially close to fixation of the normal type with a small fraction of the handicapped type maintained in a mutation-selection balance. We employ result 4 and equation (44) (which is equivalent to eq. [45]) to compute the mother's (or parents') unbeatable strategy $\hat{x},\hat{y}$. We then check the stability of fixation of the normal type *AA* under the condition of fixation of $\hat{x},\hat{y}$ among mothers or parents; that is, we check the stability of fixation of *BB*.

In order to estimate the unbeatable maternal strategy $\hat{x},\hat{y}$, we employ equation (44) or (45) to obtain

$$\partial H/\partial a = \partial G/\partial a = a^*[a^* - \hat{b}(1 - r)]\,, \tag{46}$$

$$\partial H/\partial b = \partial G/\partial b = \hat{a}[a^* - \hat{b}(1 - 2r)]/2 . \tag{47}$$

Thus, without loss of generality, we may use the mother's interference function H. We may also compute

$$\left.\frac{\partial a}{\partial x}\right|_{\hat{x},\hat{y}} = \frac{(\hat{x} + \hat{y})v'(\hat{x}) - v(\hat{x})}{(\hat{x} + \hat{y})^2} = \frac{v'(\hat{x}) - \hat{a}}{\hat{x} + \hat{y}}, \tag{48a}$$

$$\left.\frac{\partial b}{\partial x}\right|_{\hat{x},\hat{y}} = \frac{-u(\hat{y})}{(\hat{x} + \hat{y})^2} = \frac{-\hat{b}}{\hat{x} + \hat{y}}, \tag{48b}$$

$$\left.\frac{\partial a}{\partial y}\right|_{\hat{x},\hat{y}} = \frac{-\hat{a}}{\hat{x} + \hat{y}}, \tag{48c}$$

$$\left.\frac{\partial b}{\partial y}\right|_{\hat{x},\hat{y}} = \frac{u'(\hat{y}) - \hat{b}}{\hat{x} + \hat{y}}. \tag{48d}$$

The identities

$$\partial H/\partial x|_{x,y=\hat{x},\hat{y}} = \partial H/\partial y|_{x,y=\hat{x},\hat{y}} = 0$$

are then used to produce

$$2a^*[a^* - \hat{b}(1 - r)]v'(\hat{x}) = \hat{a}[2(a^*)^2 - (a^* + \hat{b})\hat{b}(1 - 2r)] \tag{49}$$

and

$$[a^* - \hat{b}(1 - 2r)]u'(\hat{y}) = 2(a^*)^2 - \hat{b}(a^* + \hat{b})(1 - 2r). \tag{50}$$

We are now in a position to determine the conditions for initial success of a slightly perturbed handicap, characterized by the survival function with the conditions of the infinitesimal local handicap (eqq. [4]–[6]). In Appendix B we show that a sufficient condition for this is

$$-v''(x^*) > a^*/2rx^* . \tag{51}$$

Recall that $2a^* = v(x^*)/x^*$ is the fitness of a completely normal brood. Near fixation of the normal offspring type, AA, we may normalize so that this fitness is 1. Then, $F = 1/x^*$ is the size of a completely normal brood or the average fertility in the population near fixation of AA. Condition (51) can then be written as

$$v''(x^*) < -F/4r . \tag{52}$$

Result 6.—A sufficient condition for the establishment of the handicap, in the long run, is inequality (52), namely, that the relative concavity of $v(x)$ at x^* exceeds $F/4r$. If the relative concavity of $v(x)$ at x^* is less than $F/4r$, there is always a two-locus evolutionarily stable situation in which the handicap cannot enter the population.

In order to evaluate the stability of fixation of the handicapped type, consider a population with offspring survival function $u(x)$ and maternal strategy y^* where, for example, $u'(y^*) = u(y^*)/y^* = 2c^*$. Again assume a mutation-selection bal-

ance at which the heterozygote determined by the dominant mutation is of the normal offspring phenotype and has the survival function $v(\cdot)$, with

$$v(y^*) = u(y^*) + \tilde{\epsilon} \qquad (\tilde{\epsilon} > 0),$$
$$v'(y^*) = u(y^*) - \delta \qquad (\delta > 0),$$
$$v''(y^*) = -\alpha = u''(y^*).$$

Assume first that there is an unbeatable maternal strategy with investments $\hat{x}$ and $\hat{y}$ in the mutant (i.e., normal) and resident (i.e., handicapped) offspring, respectively, in a mixed brood.

Set $\tilde{\eta} = (\hat{y} - y^*)$ and $\tilde{\xi} = (\hat{x} - y^*)$. We now repeat the calculation carried out in the previous sections with $u,v,a^*,\delta,\epsilon,\xi$, and η replaced by $v,u,c^*, -\delta, -\tilde{\epsilon},\tilde{\eta}$, and $\tilde{\xi}$, respectively. (Note that these calculations were independent of the sign of ϵ and δ and depended only on the condition that $\delta^2 < |\epsilon| < |\delta|$.) The condition for initial increase of the new mutant (i.e., normal type) becomes $v(\hat{x})/(\hat{x} + \hat{y}) > c^*$, which entails

$$\delta\tilde{\eta} - \tilde{\epsilon} + c^*(\tilde{\eta} - \tilde{\xi}) < 0. \tag{53}$$

A necessary and sufficient condition is

$$\tilde{\eta} < 0, \tag{54}$$

which becomes equivalent to

$$\alpha\tilde{\xi} + \delta > 0. \tag{55}$$

Under the restrictions that $\delta^2 < |\epsilon| < |\delta|$ with $|\epsilon|$ small, we obtain the approximation

$$(2r\alpha y^* - c^*)(\alpha\tilde{\xi} + \delta) = -(1 - 2r)\delta/2 \tag{56}$$

(see eq. [A17]). Hence, for $r \neq \frac{1}{2}$, the condition for stability of fixation of the handicapped type is

$$\alpha < c^*/2ry^* \simeq a^*/2rx^* + 0(\delta), \tag{57}$$

or, equivalently,

$$v''(x^*) < -F/4r, \tag{58}$$

and the result extends, as above, to $r = \frac{1}{2}$.

Result 7.—(i) If $-v''(x^*) < F/2$—namely, if the relative concavity of the survival function $v(x)$ is less than half the average fertility of the population near fixation of the normal offspring type—then fixation of the handicapped type cannot in the long run resist invasion by nonhandicapped mutations. Fixation of the normal type may, however, be compatible with an unbeatable maternal strategy that precludes invasion by the handicapped type. (ii) If $-v''(x^*) > F/2$, then, provided that the linkage between the *B*/*b* and *A*/*a* loci is sufficiently loose, fixation of the nonhandicapped type cannot in the long run withstand invasion by handicapped offspring. In this case, fixation of the handicapped type can be compatible with an unbeatable maternal strategy that precludes initial increase of

the normal type. The exact condition on the recombination fraction for this to occur is $r > r^*$, with $r^* = -F/4v''(x^*)$, which obviously depends on the relative concavity of v at x^*. (iii) An unbeatable maternal strategy can always preclude both initial increase of the handicapped type and stability of its fixation provided that the linkage between the two loci is sufficiently tight.

Parts i and iii of result 7 stand in sharp contrast to results 2 and 3, which were obtained under the naive assumption that parents should act to maximize their own fitness in terms of the total number of surviving offspring of either kind. In the preceding section, we show that this assumption is false in general, and we now see that its acceptance may produce incorrect predictions.

DISCUSSION

The evolution of a form of handicap to offspring has been studied here in two ways. In the first, the formulation is in terms of a simple parent-offspring population game in which parents seek to increase their expected number of surviving offspring of both handicapped and normal types. In the second, we set up an exact two-locus model with offspring phenotype and parental behavior determined by the distinct loci. When mutation to the handicapped offspring type balances selection against it, maximization of the parents' fitness does not occur. Instead, a weighted sum of contributions from handicapped and nonhandicapped offspring is maximized. The weight ascribed to the nonhandicapped type is higher, which is not surprising since such offspring are in turn likely to have more offspring of their own (see, e.g., Taylor 1988). The relative weights are shown to depend critically on the recombination rate between the two loci. Evolutionary predictions based on the two formulations differ, including those concerned with initial success and final fixation of the handicapped type.

According to the game formulation, Zahavi's (1975, 1977) argument concerning the evolution of the handicapped type is validated for any survival function of the normal type, $v(x)$, where x is the parents' investment. Specifically, for any $v(x)$, $u(x)$ characterizes the survival of the handicapped offspring, with $u(x) < v(x)$, such that a mutant type with this handicap can initially increase and be stably fixed in the population. In the process, the survival probability of the handicapped type increases, but the overall success of broods with handicapped individuals decreases because of the higher investment in each individual offspring. In contrast, the two-locus dynamics reveal that this result can be true only for functions of resident-offspring survival, $v(x)$, that are sufficiently concave, at least near the optimum of the parents' investment. In fact, for the handicap to succeed, the ratio between the local concavity of the normal offspring's survival function and the parents' fertility must exceed a critical value, which depends on the linkage between the two loci. This critical value is 0.5 for free recombination and increases without bound as the linkage becomes tighter. Thus, as long as the ratio of concavity to fertility exceeds 0.5, a mutation to handicapped-offspring survival ability can always increase when rare, and its fixation in the population is stable provided that its linkage with genes affecting the parents' investment strategy is loose enough. Under the same conditions on concavity and fertility,

however, a mutation that affects parental behavior and prevents the success of the handicap can always succeed if the linkage between the genes affecting parents and offspring is sufficiently tight.

That relatively low fertility and high concavity of $v(x)$ at x^* favor the entry of the handicapped type agrees with Zahavi's verbal argument. Zahavi claimed that the handicap is unlikely to evolve when parents' fertility is high, the optimal investment in any simple offspring is low, or any increase in investment increases the offspring survival function substantially; this would be the case when the local concavity of $v(x)$ near x^* is low.

The key role played by recombination means that ecological information about the conflict between parents and offspring is inherently insufficient to produce deterministic predictions about the direction of evolution. In fact, only in the case of high fertility and low concavity of the offspring survival function can we confidently predict the outcome, namely, that the handicap cannot evolve. Otherwise, the conclusions are rather statistical in nature; in some populations, handicapped offspring are expected, and their frequency may be quite high. The greater the concavity of the offspring survival function and the lower the parental fertility, the more likely is selection to favor handicapped offspring.

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APPENDIX A

Analysis of the Equations for Local Analysis of the Dynamic Model

Using equation (31) in equations (34)–(37) and (39)–(42), the linear transformation has the matrix **A** given by

$$\mathbf{A} = \mathbf{A}(p) = \mathbf{B}(p)/W(p), \tag{A1}$$

where $W(p)$ is given by equation (38), $\mathbf{B}(p)$ can be represented as

$$\mathbf{B}(p)/2 = \begin{Vmatrix} a^* + (\tilde{a} - 2a^*)p - 2(a^* - \hat{b})p & \tilde{a}(1 - r) + L_1 p & \tilde{a}r + L_2 p & 0 \\ (a^* - \hat{b} + \tilde{b})p & L_3 p & L_4 p & 0 \\ (a^* - \hat{b})p & \tilde{b}r + L_5 p & \tilde{b}(1 - r) + L_6 p & L_7 p \\ 0 & L_8 p & L_9 p & L_{10} p \end{Vmatrix}, \tag{A2}$$

and $L_1, L_2, \ldots, L_{10}$ are constants independent of p. Quadratic-order terms in p are neglected.

At $p = 0$, $\mathbf{A}(p)$ reduces to

$$\mathbf{A}_0 = (a^*)^{-1} \begin{Vmatrix} a^* & \tilde{a}(1 - r) & \tilde{a}r & 0 \\ 0 & 0 & 0 & 0 \\ 0 & \tilde{b}r & \tilde{b}(1 - r) & 0 \\ 0 & 0 & 0 & 0 \end{Vmatrix},$$

which has eigenvalues 1, $(1 - r)\bar{b}/a^*$, and 0. With BB fixed, the assumption of mutation-selection balance at the A/a locus entails that the average fitness of Aa, namely,

$$2u(\hat{y})/(\hat{x} + \hat{y}) + O(p) = \hat{b} + O(p),$$

is less than the average fitness of AA, which is a^*. Hence, $\hat{b} < a^*$. For mutations of small effect at the A/a locus, $b < a^*$; and indeed, $\bar{b}(1 - r) = (b + \hat{b})(1 - r)/2 < a^*$. Thus, we have the expected result that, when $p = 0$, the largest eigenvalue of the local stability matrix is 1. (In fact, the mutation $B \to b$ only changes the mother's strategy toward the mutant type Aa, which does not exist at $p = 0$; i.e., the mutation $B \to b$ is neutral.) This means that the characteristic polynomial $\psi_0(x)$ of $\mathbf{A}_0$ intersects the x-axis at $x = 1$, and it must do so from below. We may then use the implicit-function theorem to establish that, for small $p > 0$, the leading eigenvalue of $\mathbf{A}_p$ is less than 1 if and only if

$$\partial\psi_p(1)/\partial p|_{p=0} > 0, \tag{A3}$$

where $\psi_p(x)$ is the characteristic polynomial of $\mathbf{A}_p$. But

$$\psi_p(1) = W(p)^{-4}|\mathbf{B}_p - \mathbf{I}W(p)|,$$

and at $p = 0$, $|\mathbf{B}_0 - \mathbf{I}W(0)| = W(0)\psi_0(1) = 0$. Hence,

$$\left.\frac{\partial\psi_p(1)}{\partial p}\right|_{p=0} = \frac{1}{16(a^*)^4}\left|\frac{\partial}{\partial p}|\mathbf{B}_p - 2a^*\mathbf{I}|\right|_{p=0}. \tag{A4}$$

Straightforward calculation of the right side of equation (A4), differentiating the determinant row by row, produces the result

$$\begin{aligned}\left.\frac{\partial\psi_p(1)}{\partial p}\right|_{p=0} &= 32(a^*)^2[a^* - \bar{b}(1 - r)](\hat{a} - a^*) + 16a^*\bar{a}[a^* - \bar{b}(1 - 2r)](\hat{b} - \bar{b}) \\ &= 16\{(a^*)^2[a^* - (b + \hat{b})(1 - r)/2](\hat{a} - a) \\ &\quad + (a + \hat{a})a^*[a^* - (b + \hat{b})(1 - 2r)/2](\hat{b} - b)/4\},\end{aligned}$$

which gives us the function H in equations (43) and (44).

APPENDIX B

The Conditions for the Initial Success of a Slightly Perturbed Handicap

Specifically, we seek the conditions under which the initial net fitness $2\hat{b} = 2u(\hat{y})/(\hat{x} + \hat{y})$ of the handicapped type exceeds that of the normal type, $2a^* = v(x^*)/x^*$. To this end, write

$$\xi = \hat{x} - x^*, \qquad \eta = \hat{y} - x^*, \qquad \alpha = -v''(x^*). \tag{B1}$$

With the definition of $u(x)$ given in equations (4)–(8), we seek conditions on ϵ and δ with ϵ sufficiently small so that $\hat{x} = x^* + \xi$ and $\hat{y} = x^* + \eta$ solve the equations (49) and (50), with $|\xi|$ and $|\eta|$ less than ϵ/δ, and such that $u(\hat{y}) < v(\hat{y})$ with

$$u(\hat{y})/(\hat{x} + \hat{y}) = \hat{b} > a^* = v(x^*)/x^*.$$

Equation (18) shows that, for small η,

$$u'(\hat{y}) = v'(x^*) - \alpha\eta + \delta = 2a^* - \alpha\eta + \delta. \tag{B2}$$

Under the same conditions, equation (25) shows that

$$\hat{a} = v(\hat{x})/(\hat{x} + \hat{y}) = a^* + a^*(\xi - \eta)2x^*, \tag{B3}$$

and from equation (23),

$$\hat{b} = u(\hat{y})/(\hat{x} + \hat{y}) = a^* - a^*(\xi - \eta)2x^* + (\delta\eta - \epsilon)/2x^*. \tag{B4}$$

Insert equations (B2)–(B4) into (49) and (50) to obtain

$$2r\alpha\xi + (1 + 2r)[a^*(\eta - \epsilon) + \delta\eta - \epsilon]/2x^* = 0 \tag{B5}$$

and

$$2ra^*(\alpha\eta - \delta) + (1 - 2r)(a^* - \delta)[a^*(\xi - \eta) - \delta\eta + \epsilon]/2x^* = 0. \tag{B6}$$

From equation (B4), the condition $\hat{b} > a^*$ for initial success of the handicapped type is equivalent to

$$a^*(\eta - \xi) + \delta\eta - \epsilon > 0. \tag{B7}$$

From equation (B5), this means that

$$\xi < 0. \tag{B8}$$

Simple manipulations on equations (B5) and (B6) produce

$$(a^* - \delta)(1 - 2r)\alpha\xi = a^*(-\alpha\eta + \delta)(1 + 2r). \tag{B9}$$

Thus, since δ is small, the condition that $\xi < 0$ for the initial success of the handicap is equivalent to

$$\alpha\eta - \delta > 0. \tag{B10}$$

Insert equation (B9) into (B6) to obtain

$$\left(2a^*r - \frac{1 + 2r}{2\alpha x^*}(a^*)^2\right)(\alpha\eta - \delta) = \frac{1 - 2r}{2x^*}(a^* - \delta)[(a^* + \delta)\eta - \epsilon],$$

which, after reorganization, becomes

$$[4a^*r\,\alpha x^* - 2(a^*)^2 + (1 - 2r)\delta^2]\eta = [4r\alpha x^* - a^*(1 + 2r)]a^*\delta/\alpha - (1 - 2r)(a^* - \delta)\epsilon. \tag{B11}$$

It is not difficult to verify that, except for the degenerate case in which $2r\alpha x^* = a^*$, the condition $\delta = \epsilon^\theta$ with $\frac{1}{2} < \theta < 1$ and ϵ small (see eqq. [4]–[8]) guarantees that $\eta < \epsilon/\delta$ and $u(x) < v(x)$. Thus, $u(x)$ is a handicap for all x in the interval $x^*,\hat{y}$. Also from equation (B11), it follows directly that

$$[4a^*r\,\alpha x^* - 2(a^*)^2 + (1 - 2r)\delta^2](\alpha\eta - \delta) = (1 - 2r)\{[(a^*)^2 - \delta^2]\delta - \alpha(a^* - \delta)\epsilon\}, \tag{B12}$$

which, ignoring terms $O(\epsilon)$ and smaller, becomes

$$(2r\alpha x^* - a^*)(\alpha\eta - \delta) = a^*(1 - 2r)\delta/2. \tag{B13}$$

Provided that $r < \frac{1}{2}$, equations (B10) and (B13) together produce the sufficient condition for initial success of the handicap:

$$\alpha > a^*/2rx^*. \tag{B14}$$

In the case $r = \frac{1}{2}$, this line of reasoning needs modification since equation (B6) then entails $\alpha\eta = \delta$, and thus, $\hat{b} = a^* + o(\delta)$. That is, with free recombination, the intensity of selection for or against the handicap is weak (at least in our example with an infinitesimal local handicap).

In equation (B5), however, if we set $r = \frac{1}{2}$ and $\eta = \delta/\alpha$, we have

$$\xi(\alpha x^* - a^*) = -\delta^2/\alpha - a^*\delta/\alpha + \epsilon, \tag{B15}$$

which under our assumptions on ϵ and δ entails that

$$\alpha > a^*/x^*,$$

since the right side of equation (B15) is negative and so is ξ; hence, equation (B14) is proved for $0 < r \leq \frac{1}{2}$.

LITERATURE CITED

Alexander, R. D. 1974. The evolution of social behavior. Annual Review of Ecology and Systematics 5:325–383.

Ben-Porath, E., and E. Dekel. 1988. Coordination and the potential for self sacrifice. Research Paper 984. Graduate School of Business, Stanford University, Stanford, Calif.

Charnov, E. L. 1982. Parent-offspring conflict over reproductive effort. American Naturalist 119:736–737.

Eshel, I. 1984. On the survival probability of a slightly advantageous mutant gene in a multitype population: a multidimensional branching process model. Journal of Mathematical Biology 19:201–209.

———. In press. Sexual-reproduction population dynamics and optimization in natural populations. *In* R. Selten, ed. Proceedings of the Zentrum für Interdisziplinäre Forschung research group on gene theory in the behavioral sciences.

Eshel, I., and E. Sansone. 1991. Parent-offspring conflict over the sex ratio in a diploid population with different investment in male and in female offspring. American Naturalist (in press).

Feldman, M. W., and I. Eshel. 1982. On the theory of parent-offspring conflict: a two-locus genetic model. American Naturalist 119:285–292.

Hamilton, W. D. 1967. Extraordinary sex ratio. Science (Washington, D.C.) 156:477–488.

Maynard Smith, J., and G. R. Price. 1973. The logic of animal conflict. Nature (London) 246:15–18.

Oster, G., I. Eshel, and D. Cohen. 1977. Worker-queen conflict and the evolution of social conflicts. Theoretical Population Biology 12:49–85.

Parker, G. A. 1984. Evolutionary stable strategies. Pages 30–61 *in* J. R. Krebs and N. B. Davies, eds. Behavioural ecology: an evolutionary approach. 2d ed. Sinauer, Sunderland, Mass.

Parker, G. A., and M. R. Macnair. 1978. Models of parent-offspring conflict. I. Monogamy. Animal Behaviour 26:97–110.

Taylor, P. D. 1988. Inclusive fitness models with two sexes. Theoretical Population Biology 34:145–168.

Trivers, R. L. 1974. Parent-offspring conflict. American Zoologist 14:249–264.

Vehrencamp, S. L., and J. W. Bradbury. 1984. Mating systems and ecology. Pages 251–278 *in* J. R. Krebs and N. B. Davies, eds. Behavioural ecology: an evolutionary approach. 2d ed. Sinauer, Sunderland, Mass.

West Eberhard, M. J. 1975. The evolution of social behavior by kin selection. Quarterly Review of Biology 50:1–33.

Zahavi, A. 1975. Mate selection—a selection for a handicap. Journal of Theoretical Biology 53:205–214.

———. 1977. The cost of honesty (further remarks on the handicap principle). Journal of Theoretical Biology 67:603–605.

———. 1981. Natural selection, sexual selection and the selection of signals. Pages 133–138 *in* G. G. E. Scudder and J. L. Reveal, eds. Evolution today: Proceedings of the Second International Congress of Systematic and Evolutionary Biology. Carnegie-Mellon University, Pittsburgh, Pa.

———. 1987. The theory of signal selection and some of its implications. Pages 305–327 *in* V. P. Delfino, ed. Proceedings of the International Symposium on Biology and Evolution.