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LETTER TO THE EDITOR

Are Intragametic Conflicts Common in Nature? Do They Represent an Important Factor in Evolution?

Modern sociobiological approach to Darwinism can be summarized in the statement that natural selection, operating on the individual level, favors, at least in some sense, features which increase the expected number of duplicates of one's gene, passed on to the next generation. In this context, the very use of the term "one's gene" (e.g. Hamilton, 1964) implies the tacit but crucial assumption that any change in the feature of the individual affects the reproductive success of all its genes in exactly the same way. This is indeed the case for all diploid genes, segregating "decently" according to Mendel's law. If two such genes are carried by the same individual, the expected number of duplicates passed on to the next generation by each of them is exactly the survival probability of the carrier, multiplied by half of its fertility, namely its *Fisherian fitness.* In the case of partial selfing, this value must be multiplied by the same factor for all the genes in the genome. The expected number of genes, identical by descent to any Mendelian diploid gene carried by the individual in question, will be equal to the sum of fitnesses of its relatives (including itself), each multiplied by Wright's coefficient of kinship. Any change in the individual's behavior (or phenotype) will, thus, change this expected number in exactly the same way for all genes. This expected change, namely Hamilton's *inclusive fitness* (Hamilton, 1964), can be attributed to the *individual's* behavior rather than to any specific gene.

The fact that the bulk of genes in our genome are fully diploid and apparently segregate according to Mendel's law is the basic justification for the sociobiological attempt to explain the Darwinian association between natural selection and individual adaptation on the basis of selection for the "selfish" gene (e.g. Dawkin, 1982).

In some cases, however, different parts of the genome can reproduce independently or at least according to different probabilistic laws. In such cases, natural selection, operating on parts of the genome can enhance features which are unfavorable for other parts or, maybe, for the individual carrier.

We know, for example, that pieces of DNA can be carried by viruses. They can also replicate and establish themselves as "moving genes" in several loci of the same genom, thereby increasing the chance of their being

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by a fraction α, $0<\alpha<1$, and increasing the survival probability of the carrier's female offspring by $\theta\alpha$, $0<\theta<1$.

A recessive mutation $B \rightarrow b$ on an autosomal locus causes an aggressiveness of a male carrier toward its female offsprings, thereby reducing their survival probability by a fraction $\beta(0<\beta<1)$ and increasing the survival probability of the carrier's male offspring by $\theta'\beta$. For the sake of simplicity assume $\theta'=\theta$. Assume further, that the mutations a and b do not suppress each other's effect (e.g. they may correspond to different aspects of parental care). Instead, they are combined in an additive way. The survival probabilities of male and female offspring born to the six types of father, relative to the survival probability of an offspring of the wild type are given in Table 1.

TABLE 1

Frequencies of male types and survival probability of their offsprings

Father type	Survival probability, male offspring	Survival probability, female offspring	Frequency of father's type in the population
$A\|BB$	1	1	x_{11}
$A\|Bb$	1	1	x_{12}
$A\|bb$	$1+\theta\beta$	$1-\beta$	x_{13}
$a\|BB$	$1-\alpha$	$1+\theta\alpha$	x_{21}
$a\|Bb$	$1-\alpha$	$1+\theta\alpha$	x_{22}
$a\|bb$	$1-\alpha+\beta\theta$	$1+\theta\alpha-\beta$	x_{23}

Finally we assume that neither the mutation b or a affects a female carrier, and that mating occurs at random.

The number of surviving male offspring in the population (relative to the number of surviving males in an equal population of the wild type) is

$$\mu = 1+\theta\beta(x_{13}+x_{23})-\alpha x \tag{1}$$

where

$$x = x_{21}+x_{22}+x_{23} \tag{2}$$

is the proportion of the mutant allele a among males.

The relative number of females will be

$$F = 1-\beta(x_{13}+x_{23})+\alpha\theta\beta. \tag{3}$$

Denote by y_{ij} the frequency of females of the genotype ij where the indexes $i=1, 2, 3$ stand for the combinations AA, Aa and aa of genes on the X chromosome, the indexes $j=1, 2, 3$ stand for the combination of genes BB, Bb and bb respectively. It will be convenient to use the notation:

$$\begin{aligned} y_{11}+\tfrac{1}{2}y_{12}+\tfrac{1}{2}y_{21}+\tfrac{1}{4}y_{22}&=y_{AB}\\ y_{31}+\tfrac{1}{2}y_{32}+\tfrac{1}{2}y_{21}+\tfrac{1}{4}y_{22}&=y_{aB}\\ y_{13}+\tfrac{1}{2}y_{12}+\tfrac{1}{2}y_{23}+\tfrac{1}{4}y_{22}&=y_{Ab}\\ y_{33}+\tfrac{1}{2}y_{32}+\tfrac{1}{2}y_{23}+\tfrac{1}{4}y_{22}&=y_{ab} \end{aligned} \tag{4}$$

y_{AB} is the probability that a random female in the population will pass the alleles A and B to her offspring. y_{aB}, y_{Ab} and y_{ab} are interpreted similarily.

$$y=y_{aB}+y_{ab}=y_{31}+y_{32}+y_{33}+\frac{y_{21}+y_{22}+y_{23}}{2} \tag{5}$$

is the frequency of the allele a among females.

A newborn male will be of genotype A/BB if it obtains the combination of alleles AB from its mother and the allele B from its father. With random mating these two events are independent. The probability of the first event is y_{AB} and the specific mother-type from which the offspring obtains its genes does not affect its survival probability. The probability that a male offspring gets the allele B from its father is

$$x_{11}+\frac{x_{12}}{2}+x_{21}+\frac{x_{22}}{2}.$$

In this case, however, the father-type does affect the offspring's survival probability. Thus the probability that the offspring gets a B allele from either an A/BB or an A/Bb father and survives to maturity is $x_{11}+x_{12}/2$. The probability that an offspring gets a B allele from a father of types a/BB or a/Bb and survives is $(1-\alpha)(x_{21}+x_{22}/2)$. Thus the relative frequency of surviving A/BB male offspring out of all newborn offspring is:

$$y_{AB}\left(x_{11}+\frac{x_{12}}{2}+(1-\alpha)\left(x_{21}+\frac{x_{22}}{2}\right)\right)$$

But the survival probability of a random male offspring in the population is μ. Hence, the relative frequency of the A/BB type among all surviving newborn offspring is:

$$x_{11}=\frac{1}{\mu}\,y_{AB}\left[\left(x_{11}+\frac{x_{12}}{2}+(1-\alpha)\left(x_{21}+\frac{x_{22}}{2}\right)\right)\right]$$

In the same way we get the transformation of the other 14 genotype frequencies from generation to the next:

$$
\begin{aligned}
x'_{12}&=\frac{1}{\mu}\,y_{AB}\left[(1+\theta\beta)x_{13}+\frac{x_{12}}{2}+(1-\alpha-\theta\beta)x_{23}+\frac{1-\alpha}{2}\,x_{22}\right]\\
&\quad+\frac{1}{\mu}\,y_{Ab}\left[x_{11}+\frac{x_{12}}{2}+(1-\alpha)\left(x_{21}+\frac{x_{22}}{2}\right)\right]\\
x'_{13}&=\frac{1}{\mu}\,y_{Ab}\left[(1+\theta\beta)x_{13}+\frac{x_{12}}{2}+(1-\alpha+\theta\beta)x_{23}+\frac{1-\alpha}{2}\,x_{22}\right]\\
x'_{21}&=\frac{1}{\mu}\,y_{aB}\left[x_{11}+\frac{x_{12}}{2}+(1-\alpha)\left(x_{21}+\frac{x_{22}}{2}\right)\right]\\
x'_{22}&=\frac{1}{\mu}\,y_{aB}\left[(1+\theta\beta)x_{13}+\frac{x_{12}}{2}+(1-\alpha+\theta\beta)x_{23}+\frac{1-\alpha}{2}\,x_{22}\right]\\
&\quad+\frac{1}{\mu}\,y_{ab}\left[x_{11}+\frac{x_{12}}{2}+(1-\alpha)\left(x_{21}+\frac{x_{22}}{2}\right)\right]\\
x'_{23}&=\frac{1}{\mu}\,y_{ab}\left[(1+\theta\beta)x_{13}+\frac{x_{12}}{2}+(1-\alpha+\theta\beta)x_{23}+\frac{1-\alpha}{2}\,x_{22}\right]
\end{aligned}
\tag{6}
$$

and by summing

$$
x'=x'_{21}+x'_{22}+x'_{23}=\frac{1+\theta\beta(x_{13}+x_{23})-\alpha x}{\mu}\,(y_{aB}+y_{ab})=y. \tag{7}
$$

As for the female types we get

$$
\begin{aligned}
y'_{11}&=\frac{1}{F}\,y_{AB}\left(x_{11}+\frac{x_{12}}{2}\right)\\
y'_{12}&=\frac{1}{F}\left\{y_{Ab}\left(x_{11}+\frac{x_{12}}{2}\right)+y_{AB}\left[(1-\beta)x_{13}+\frac{x_{12}}{2}\right]\right\}\\
y'_{13}&=\frac{1}{F}\,y_{Ab}\left[(1-\beta)x_{13}+\frac{x_{12}}{2}\right]\\
y'_{21}&=\frac{1}{F}\left\{y_{aB}\left(x_{11}+\frac{x_{12}}{2}\right)+y_{AB}(1+\theta\alpha)\left(x_{21}+\frac{x_{22}}{2}\right)\right\}\\
y'_{22}&=\frac{1}{F}\left\{y_{ab}\left(x_{11}+\frac{x_{12}}{2}\right)+y_{Ab}(1+\theta\alpha)\left(x_{21}+\frac{x_{22}}{2}\right)\right\}\\
&\quad+y_{aB}\left[(1-\beta)x_{13}+\frac{x_{12}}{2}\right]+y_{AB}\left[(1+\alpha\theta-\beta)x_{23}+\frac{1+\theta\alpha}{2}\,x_{22}\right]
\end{aligned}
\tag{8}
$$

$$y'_{23}=\frac{1}{F}\left\{y_{ab}\left[(1-\beta)x_{13}+\frac{x_{12}}{2}\right]+y_{Ab}\left[(1+\alpha\theta-\beta)x_{23}+\frac{1+\theta\alpha}{2}x_{22}\right]\Bigg]\right\}$$

$$y'_{31}=\frac{1}{F}\,y_{aB}(1+\theta\alpha)\left(x_{21}+\frac{x_{22}}{2}\right)$$

$$y'_{32}=\frac{1}{F}\left\{y_{ab}(1+\theta\alpha)\left(x_{21}+\frac{x_{22}}{2}\right)+y_{aB}\left[(1+\theta\alpha-\beta)x_{23}+\frac{1+\theta\alpha}{2}x_{23}\right]\Bigg]\right\}$$

$$y'_{33}=\frac{1}{F}\,y_{ab}\left[(1+\theta\alpha-\beta)x_{23}+\frac{1+\alpha\theta}{2}x_{22}\right]$$

and by summing up

$$y'=\frac{1}{F}\left\{[(1+\theta\alpha)x-\beta x_{23}]\frac{1+y}{2}+[1-x-\beta x_{13}]\frac{y}{2}\right\}. \tag{9}$$

From equations (7), (9) and (3) it follows that in equilibrium $x=y$ and

$$\alpha\theta x(1-x)=\beta[(1-x)x_{23}-xx_{13}]. \tag{10}$$

3. A Necessary and Sufficient Condition for Fixation Stability of an Absolutely Inferior Double Mutant

For local analysis of the double mutant equilibrium $x_{23}=y_{ab}=1$ we check the linear approximation of the transformation

$$T\mathbf{V}=\mathbf{V}' \tag{11}$$

at $\mathbf{V}=\mathbf{0}$ when $x_{23}=1-x_{11}-x_{12}-x_{13}-x_{21}-x_{22}$; $y_{ab}=1-y_{AB}-y_{Ab}-y_{aB}$

$$\mathbf{V}=(x_{11},\ x_{12},\ x_{13},\ x_{21},\ x_{22},\ y_{AB},\ y_{Ab},y_{aB}). \tag{12}$$

Employing equations (6) and (8), with (1), (3) and (4) we have this approximation given by the matrix $\mathbf{A}$:

$$\mathbf{A}=\begin{pmatrix}
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & 0 & 0 & 1 & 0 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & 1 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\
\frac{1}{\mu} & \frac{1}{2\mu} & 0 & \frac{1-\alpha}{\mu} & \frac{1-\alpha}{2\mu} & 0 & 0 & 1\\
\frac{1}{4F} & \frac{1}{8F} & 0 & 0 & 0 & \frac{1}{4} & 0 & 0\\
\frac{1}{4F} & \frac{3}{8F} & \frac{1-\beta}{2F} & 0 & 0 & \frac{1}{4} & \frac{1}{2} & 0\\
\frac{1}{4F} & \frac{1}{8F} & 0 & \frac{1+\theta\alpha}{2F} & \frac{1+\theta\alpha}{4F} & \frac{1}{4} & 0 & \frac{1}{2}
\end{pmatrix} \tag{13}$$

where

$$\mu = 1 - \alpha + \theta\beta \tag{14}$$

$$F = 1 + \theta\alpha - \beta \tag{15}$$

The eigenvalues of the matrix (13) are the solutions of the characteristic equation

$$|\mathbf{A} - \mathbf{I}x| = x^2 \begin{vmatrix} -x & 1 \\ \frac{1}{8F} & \frac{1}{4} - x \end{vmatrix} \begin{vmatrix} -x & 1 \\ \frac{1-\beta}{2F} & \frac{1}{2} - x \end{vmatrix} \begin{vmatrix} \frac{1-\alpha}{2\mu} - x & 1 \\ \frac{1+\theta\alpha}{4F} & \frac{1}{2} - x \end{vmatrix} = 0 \tag{16}$$

which are

$$\lambda_1 = \lambda_2 = 0 \tag{17}$$

$$\lambda_{3,4} = \frac{\sqrt{F} \pm \sqrt{8+F}}{8\sqrt{F}} \tag{18}$$

$$\lambda_{5,6} = \frac{\sqrt{F} \pm \sqrt{8(1-\beta)+F}}{4\sqrt{F}} \tag{19}$$

$$\lambda_{7,8} = \frac{1}{4}\left\{\frac{1-\alpha}{\mu} + 1 \pm \sqrt{\left(1 + \frac{1-\alpha}{\mu}\right)^2 + 4\frac{1+\theta\alpha}{F}}\right\}. \tag{20}$$

One can readily verify

$$|\lambda_6| < \lambda_5 < 1 \tag{21}$$

and also

$$|\lambda_4| < \lambda_3, \qquad |\lambda_8| < \lambda_7. \tag{22}$$

Finally, employing equations (5) and (8) implies that $|\lambda_3| = \lambda_3 < 1$ if and only if

$$1 - \beta + \alpha\theta > \tfrac{1}{6}, \tag{23}$$

Employing equations (14) and (15), equation (20) implies that $|\lambda_7| = \lambda_7 < 1$ if and only if $2\theta\beta < \theta(1+\theta\alpha) - (1-\alpha)$.

This is equivalent to

$$(1+\theta^2)\left(\alpha - \frac{1-\theta}{1+\theta^2}\right) > 2\theta\beta. \tag{24}$$

Since equation (24) implies equation (23), we have our firt corollary.

Corollary 1

If equation (24) holds, the double mutant equilibrium is stable, and if this equilibrium is stable, then $\mu \leq \theta F$. Note that, if equation (24) holds, by considering $\beta \leq \alpha$ and $\beta > \alpha$ separately, it is easy to see that

$$\mu < 1. \tag{25}$$

Hence we get corollary 2.

Corollary 2

A necessary condition for fixation stability of the double mutant is that the survival probability of its male offspring will be less than that of the wild type.

However, if

$$\alpha > \frac{1}{1+\theta} \tag{26}$$

there is a non-degenerate set of parameters for which

$$2\theta^2\alpha < 2\beta\theta < (1+\theta^2)\alpha - (1-\theta).$$

For these parameters the condition 24 is satisfied and also, with fixation of the double mutant

$$F = 1 - \beta + \alpha\theta < 1. \tag{27}$$

Corollary 3

There is a non-degenerate set of parameters for which an absolutely inferior double mutant with a lower survival probability of both male and female offsprings, (i.e., $\mu < 1$, $F < 1$) can become fixed and stable in the population.

4. Instability of the Wild-type Equilibrium

Denote

$$L = \frac{2y+x}{3}. \tag{28}$$

Employing equations (7) and (9), with equations (1) and (3) we get,

$$L' - L = \frac{1}{3F}\{\alpha\theta x(1-x) + \beta[x_{13}x - x_{23}(1-x)]\}. \tag{29}$$

In the interior $x(1-x)>0$, equation (29) can be written as

$$L'-L=\frac{x(1-x)}{3F}\Delta \tag{30}$$

where

$$\Delta=\theta\alpha+\beta\left(\frac{x_{13}}{1-x}-\frac{x_{23}}{x}\right). \tag{31}$$

$x_{13}/(1-x)$ and x_{23}/x are the relative frequencies of the homozygote type *bb* among males carrying the allele A and a respectively. Thus, when the allele b is absent $x_{13}/1-x=x_{23}/x=0$. When the allele B is absent $x_{13}/(1-x)=x_{23}/x=1$. In both cases

$$\Delta=\alpha\theta>0. \tag{32}$$

Corollary 4

The wild-type equilibrium $x_{11}=y_{11}=1$ is never stable.

Proof. At this point $L=0$ but at any interior point at its vicinity $x(1-x)>0$ and $L>0$. Since the condition for equation (32) holds at the equilibrium, we know that $\Delta>0$ at its vicinity and, in interior points it follows from equation (30) that $L'>L>0$. Hence, convergence to the wild-type equilibrium is impossible.

5. A Condition for Global stability of the Double Mutant Equilibrium

Proposition 5

Suppose

$$\beta\leq\alpha\theta \tag{33}$$

then (i) if the condition (24) for local stability of the double mutant equilibrium holds, then this equilibrium is also globally stable. (ii) If

$$2\theta\beta>(1+\theta^2)\left(\alpha-\frac{1-\theta}{1+\theta^2}\right)>0 \tag{34}$$

then we always have fixation of the mutation a with a protected polymorphism of B and b.
(iii) If

$$\alpha-\frac{1-\theta}{1+\theta^2}<0 \tag{35}$$

then the fixation of the wild-type allele B and the mutant allele a is globally stable.

Proof. Since for any point in the interior $x(1-x)>0$, $x_{23}/x-x_{13}/(1-x)>1$, condition (33) implies

$$\Delta=\alpha\theta-\beta\left(\frac{x_{23}}{x}-\frac{x_{13}}{1-x}\right)>0.$$

From equation (30) it then follows that

$$L'-L\geq 0 \tag{36}$$

with a strict inequality at any point for which $0<L<1$. Hence the population converges to the surface $L=1$ on which the X-linked mutant allele a is fixed. But on this surface

$$\mu=1-\alpha+\theta\beta x_{23} \tag{37}$$

$$F=1+\beta\alpha-\beta x_{23} \tag{38}$$

$$x_{23}-1-x_{21}-x_{22},\ y_{ab}=1-y_{aB}$$

and the set of transformations of equations (6) and (8) becomes

$$x'_{21}=\frac{(1-\alpha)}{\mu}y_{aB}\left(x_{21}+\frac{x_{22}}{2}\right) \tag{39}$$

$$x'_{22}=\frac{1-\alpha}{\mu}\left(x_{21}+\frac{x_{22}}{2}\right)(1-2y_{aB})+y_{aB} \tag{40}$$

$$y'_{aB}=\frac{1+\theta\alpha}{2F}\left(x_{21}+\frac{x_{22}}{2}\right)+\frac{y_{aB}}{2}. \tag{41}$$

The equations $x_{21}=x'_{21}$, $x_{22}=x'_{22}$ and $y'_{aB}=y_{aB}$ have three possible solutions. (i) The ab corner $x_{21}=x_{22}=y_{aB}=0$. (ii) The aB corner $x_{21}=1$, $x_{22}=y_{ab}=0$. (iii) A non-corner solution, possibly corresponding to a polymorphic equilibrium.

In order to check the stability of the two corner solutions denote

$$R=\frac{y_{ab}}{2}+\frac{x_{21}}{2}+\frac{x_{22}}{4}. \tag{42}$$

Employing equations (37)–(41) we have, after one generation

$$\begin{aligned}R'&=\frac{1}{2}\left(\frac{1-\alpha}{2\mu}+\frac{1+\theta\alpha}{2F}\right)\left(x_{21}+\frac{x_{22}}{2}\right)+\frac{y_{aB}}{2}\\&=R+\frac{2\theta\beta x_{23}-(1+\theta^2)\alpha+(1-\theta)}{4F\mu}\left(x_{21}+\frac{x_{22}}{2}\right)\\&=R+\frac{(1+\theta^2)\alpha-(1-\theta)-2\theta\beta x_{23}}{4F\mu}\left(x_{21}+\frac{x_{22}}{2}\right).\end{aligned} \tag{43}$$

Hence, if equation (35) holds, $R' \leq R$ with equality at the corners only. R is a Liapunov function on the a-surface obtaining its minimum on the aB corner and convergence to this corner from any inner point on the surface follows.

If equation (24) holds, $R' \geq R$ with equality at the corners only and convergence to the ab corner (on which R obtains its maximum) follows. (In this case, stability can also be proved by local analysis).

Finally, if equation (34) holds none of the corners is stable which proves part (ii) of the propositions.

Remarks

(i) By a change of notation, equations (39)–(41) become equivalent to those corresponding to a special case of a one-locus model, studied by Uyenoyama & Bengson (1980). The reader is referred to their analysis.

(ii) The increase of the frequency L of the allele a in the population seems obvious from arguments of inclusive fitness. The allele a induces its male carrier to increase the number of daughters (carrying the father's a allele) at the expense of sons (who do not carry the father's a allele). Hence, its effect on the father's behaviour is to increase the gene's number of copies in the next generation.

Yet it is easy to show that the value L (or, for this argument any weighted average of x and y) does not necessarily increase from one generation to the next if $\beta > \alpha\theta$. It can decrease if $x_{23}/x - x_{13}/(1-x)$ is large (close to 1), i.e. if the association between the mutant allele a and b is strong. In this case (with $\beta > \alpha\theta$) the allele b, associated with the allele a, may cause a larger drop in the survival probability of male offsprings.

Note that the argument of inclusive fitness, when applied to multilocus problems, ignores the effects of linkage disequilibrium which may be intrinsic to the evolution of a combination of interacting features. It is the intuitive argument of inclusive fitness, however, that have motivated this work, and specifically the analysis suggested in this section. Moreover, most predictions made on the basis of (modified, locus dependent) inclusive fitness arguments are shown to be virtually true, at least for the model analyzed in this paper (see the discussion).

6. Discussion: Locus Dependent Inclusive Fitness and the Evolution of Inner Conflict

The evolution of a sex ratio 1 : 1 in diploid populations has been explained by Fisher (1938) in the following way: Since in a sexual population the total number of offsprings born to male parents is always equal to the total number of offsprings born to female parents, the expected number of offsprings born to a single male at any moment is larger than the expected number of offsprings born to a single female if there are more females in the population at that moment and vice versa if there are more males. Thus, there is always an advantage to parents who give birth to offsprings of the sex which is rare at the moment, the advantage being measured in terms of the expected number of grand-offsprings, or, using the concept of *inclusive fitness* (Hamilton, 1964) in terms of the expected number of one's gene, being carried by its grand-offsprings.

In reality, however, the sex ratio is not always exactly 1:1. Moreover, an analysis of exact genetic models validates Fisher's argument of selection for a sex ratio 1:1 only when concerning alleles for which the parent in question is diploid, i.e. autosomal alleles or X-linked alleles, affecting the mother's effect on the sex of the offspring. (For the first counter-example to Fisher's argument known to me see Hamilton (1967). For many later references and an analysis of the general situation see Eshel & Feldman (1982*a*)).

Indeed, as regards father-affecting sex-linked alleles, Fisher's argument becomes false when being repeated in terms of inclusive fitness. Although Fisher is right in claiming that giving birth to an offspring of the rare sex is advantageous in terms of the expected number of grand offsprings, it is not necessarily so in terms of the expected number of genes carried by grand-offsprings, for the simple reason that a father is not related to his male offspring (and therefore to their offsprings) in the X-linked alleles. They carry none of his X-linked genes in the same way neither a daughter nor her offsprings carry any of the father's Y-linked genes. It is therefore to be expected that an X-linked mutation, causing a male parent to prefer his female offsprings (even at the expense of the total progeny size) will be selected for. Yet, once such a mutation is established in the population, thus making the sex ratio biased in favor of females, any autosomal mutation rendering the sex ratio closer to 1:1 (even at some further expense of the total progeny) will also be selected for in the population. As a result we can expect selection in favor of an absolutely inferior double mutant, characterized by contradicting actions, detrimental to its own fitness.

Note, however, that the argument of inclusive fitness, when applied to a two locus model, ignores the effects of linkage disequilibrium. Thus, we see that the X-linked allele, causing a father to prefer his daughters may be selected against when it strongly associated with the autosomal allele, causing a larger drop in the daughter's survival probability. In the model described above, this is the case when

$$\Delta = \theta\alpha - \beta\left(\frac{x_{23}}{x} - \frac{x_{13}}{1-x}\right) < 0.$$

Yet, employing the simplest possible two locus model, the surprising prediction based on the inclusive fitness argument is shown to be true for a non-degenerate set of parameters.

The model suggested here is aimed at demonstrating the theoretical possibility that, contrary to common belief, contradicting features, harming the general fitness of the individual, may not only be a result of misadaptation to a new environment, but can evolve in a stable environment due to different rules of selection operating at different loci.

It is not the intention of this paper to claim that the evolution of inner conflicts is typical to sexual x-y populations. It maintains only that this is possible and can be fully explained on the basis of natural selection.

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