Journal of Bioeconomics 1: 47–72, 1999

Does mother nature punish rotten kids?

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Synopsis: The theory of parent-offspring conflict predicts that mothers and their offspring may not agree about how resources should be allocated among family members. An offspring, for example, may favor a later weaning date than does its mother. Despite a parent's physical superiority, it may be that offspring are able to manipulate their parents' behavior. In this paper, we investigate a two-locus population genetic model of weaning conflict in which offspring can attempt to extort resources from their parents by reducing their own chances of survival if their demands are not met. We find that the frequency of recombination between the genes controlling maternal behavior and those controlling juvenile behavior determines the evolutionary outcome of this genetic conflict. When these genes are tightly linked, the mother will be able to get her way. When they are not, offspring can successfully 'blackmail' their parents into providing additional resources.

Key words: parent-offspring conflict, weaning conflict, parental manipulation, blackmail, parental care, sibling rivalry, kin selection, two-locus model, evolutionary biology, game theory, economics of the family, rotten-kid theorem

JEL Classification:

1. Parent-offspring conflict in economics and biology

1.1. Economic theories of the family

The demand for children and for children's consumption goods have been central themes in the economics of the family.[1] Most economic studies of these topics are based on theory that would apply equally well to the demand for pets and the derived demand for pet food. Children are assumed to have no decision-making authority and hence their preferences are assumed to have no bearing on economic outcomes.

Becker (1974) introduced an economic model of the household in which children are rational decision-making agents with interests distinct from those of their parents. Becker's model allows children to have economic spheres of influence where they can make decisions that influence their own well-being. Becker found a surprising result—which he called the 'rotten-kid theorem.' The rotten-kid theorem assumes that offspring care only about the money value of their consumption and that there is a benevolent 'household head' who is so much wealthier than his children that he chooses to make gifts to each of them. Thus all "marginal" allocational decisions are made by the household head. Although children are entirely selfish and are able to influence the pre-transfer income distribution in the family, it will be in the interest of each to try to maximize total household income. The outcome

is the same as the allocation that would have been selected by a benevolently dictatorial household head. According to Becker (1974, pp. 1077–1078): . . . the head *automatically* internalizes the "external" effects of his actions on other family members. Indeed, because the head maximizes family income, he *fully* internalizes these externalities not only when the income of different members but also when their consumption . . . is directly affected."

Economists (e.g., Bergstrom 1989, Bruce & Waldman 1990, Lindbeck & Weibull 1988) have since demonstrated that the conclusions of the rotten-kid theorem depend critically on special assumptions that are likely to be violated in normal interactions among offspring and parents. In realistic environments, a child who is able to make the 'first move' in interactions with a parent may be able to manipulate the parent to contribute more resources to the child than the parent would if the parent could control the child's actions.

1.2. Biological theories of parent-offspring relations

Biologists, like economists, have found the language of game theory a useful way to study conflict and cooperation between parents and offspring. Evolutionary biologists bring two ideas to the study of the family that are new to economists. First, payoffs in games between family members are usually measured in the currency of reproductive success. Second, in biological models, the strategy that an individual uses in games with its relatives is programmed by its genes, which are passed from parent to offspring by the rules of Mendelian inheritance.

Almost all modern work on familial interaction (surveyed by Clutton-Brock (1991), Godfray (1995), and by Mock & Parker (1997)), has been influenced profoundly by the fundamental contribution of Hamilton (1964) to the theory of *kin selection.* Hamilton demonstrated that evolution will favor siblings who are neither totally altruistic nor totally selfish toward each other. Hamilton stated the following proposition, which has come to be known as 'Hamilton's Rule': 'The social behavior of a species evolves in such a way that in each distinct behavior-evoking situation the individual will seem to value his neighbors' fitness against his own according to the coefficients of relationship[2] appropriate to that situation' (Hamilton, 1964, p. 19).

Trivers (1974) explicitly applied Hamilton's theory of kin selection to parent-offspring conflict. Trivers pointed out that Hamilton's rule predicts that offspring will be more selfish in their dealings with their siblings than their parents would like them to be. For example, since the coefficient of relationship between full siblings is 1/2, a child will value its sibling's fitness at 1/2 of its own. In contrast, Hamilton's rule predicts that parents will value the fitness of all of their offspring equally. Trivers argues that there is abundant evidence in nature of parent-offspring conflict which reflects the lack of coincidence of the genetic interests of children and their parents.[3]

Trivers' view that children often find ways to advance their own genetic interests at the expense of those of their parents was not universally shared by evolutionary biologists. In the same year that Becker introduced the rotten kid theorem, a biologist, Alexander (1974), offered a theory of parent-offspring relations that is more in accord with Becker's ideas. Alexander proposed that evolutionary theory would lead us to expect parents to be able to manipulate their offspring to act in the parental genetic interest.

Alexander supported his view with two arguments; the first of which does not seem to have occurred to economists. Alexander reasoned that a gene that leads a child to act against reproductive interests of its parents will not spread because when a child with such a gene grows up, its own children will act against its reproductive interests, and hence in the long run such a 'rotten kid' will generate fewer descendants.

Dawkins (1976, 1982) disputed Alexander's genetic argument for the primacy of parental interests. Dawkins agreed that Alexander was correct in pointing out that one of the costs of being a selfish child was 'the disadvantage of one's selfishness spreading to one's own children' but argued that this cost is not decisive in the conflict between the reproductive interests of parent and child. Appealing to Hamilton's theory of kin-selection, Dawkins (1976, pp. 137–138) asserts that for sexual diploids, 'a selfish child will still do well to be selfish so long as the net benefit to him is at least half of the cost to close relatives.' Alexander (1979) later agreed to the view expressed by Dawkins and recanted his genetic explanation for parental dominance. But Alexander had in store a second argument for the dominance of parental interests. This argument is more direct and is similar to that made by Becker in the rotten kid theorem. Becker appeals to the economic dominance of the household head, arguing that the head controls the relevant household budget decisions because his wealth is much greater than that of the other family members. Alexander (1974, p. 340) relies on the physical primacy of the parent, asserting that '. . . the parent is bigger and stronger than the offspring, hence in a better position to pose its will.'

The Becker-Alexander appeal to physical superiority and the parental ability to allocate resources has also come under attack by biologists. Dawkins (1976) suggested that offspring may have private information about their own condition which parents can only guess. This puts offspring in a strong position to manipulate their parents by lying about their own condition. Zahavi (1975, 1977) proposed that, by screaming until it is fed, a child might blackmail its parents into giving it more food than the parents would prefer to contribute. Since the screaming is likely to attract predators, the parent must feed it or expect to lose the child. Both of these forms of manipulation of parents by offspring have received attention in the biological literature.

Feldman & Eshel (1982) constructed a model of family behavior that is strikingly similar to Becker's scenario. Feldman and Eshel endowed parents with the power to redistribute wealth away from greedy children, where behavior of parents and of offspring is genetically coded. They found that parental ability to redistribute is not necessarily sufficient to induce offspring to behave as their parents would choose. In a later paper, Eshel & Feldman (1991) present a detailed genetic model that incorporates Zahavi's idea that offspring might blackmail their parents by threatening to bring harm to themselves. They study a two-locus genetic model of interaction between parents and offspring in which individual offspring can advance their own reproductive interests by imposing 'handicaps' on themselves; these handicaps increase the amount of resources needed to reach a given probability of survival. Eshel and Feldman find that under some conditions, this strategy of blackmail can indeed invade and resist invasion by non-blackmailing offspring. Thus, they show that the fact that parents control resource allocation at the margin does not necessarily allow parents to enforce their will.

1.3. The scope of this paper

In the sections that follow, we address the question posed in this paper's title. That is, we ask whether evolutionary forces tend to support the Becker-Alexander position, that parent-offspring conflict will be resolved in favor of the parents' reproductive interests, or the Trivers-Dawkins position, that an individual offspring can manipulate its parents to further its reproductive success at the expense of the parents' own reproductive success. In doing so, we address not only the extent of the genetic conflict between parents and offspring (the *battleground*, in Godfray's (1995) terms), but also the *resolution* of this conflict. This paper is intended to be readily accessible to economists who have little or no background in evolutionary biology. Therefore, we begin with a brief discussion on two-locus genetics. We then launch our attack on the problem of parent-offspring conflict by studying one of the simplest possible non-trivial examples—a model of weaning conflict between a mother and her first-born, in which resources can either be given to the first-born or reserved for a child who is not yet born.[4] Because of its stark simplicity, this example is well-suited for illustrating fundamental principles of parent-offspring conflict that can be obscured in more complicated interactions among parents and their offspring.

In Section 3 we describe the model formally. We determine the weaning age that will be fixed by natural selection if first-born lambs control the weaning age unchallenged by their mothers, and the weaning age that will be fixed if mother have unchallenged control. In Section 4 we consider a set of strategies in which mothers 'offer' a certain weaning age, and offspring can either comply or take drastic and costly action to express their dissatisfaction, bleating until they attract predators. Working within an explicit two-locus genetic framework, we consider the effects of these strategies on the survival probabilities of first-born and second-born offspring. We examine how the genetic conflict between parent and offspring will be resolved in such a system, and explore the characteristics of stable equilibrium.

Our work on this problem has been inspired by the two-locus models of parent-offspring conflict presented by Eshel & Feldman (1991) and many of our results closely parallel findings in their paper. Eshel and Feldman address Zahavi's concept of the non-signalling 'handicap,' examining the evolution of strategies in which offspring actually reduce their own survival probability given any particular amount of resources. By contrast, we consider the evolution of strategies in which offspring reduce their own survival probability (to zero, in our model) if they do not get their way, but do not negatively affect their survival probability if they do get their way. Hence, at the equilibria which we consider, offspring will enjoy the maximum possible survival probability given the amount of resources received. This allows us to separate the persuasive potential of offspring 'threats' from the handicap mechanism itself.

2. Elements of one-locus and two-locus genetics

In sexual diploid species—which include all birds and mammals—each individual has two copies of each 'gene.' More precisely, each individual has two (possibly identical) alleles at each genetic locus; one of these alleles is inherited from its mother and one from its

father.[5] The allele inherited from each parent is a random draw from the parent's two alleles at the same locus. In this paper we will study a two-locus genetic model in which the pair of alleles that an individual carries at one genetic locus determines the strategy that it will use as a child dealing with its parents, while the pair of alleles that it carries at a second locus determines the strategy that it will use as a parent if it survives to adulthood and reproduces.[6]

We introduce a number of definitions that will help us to discuss evolution in populations of individuals with genetically encoded strategies.

Definition 1 (*Homozygous and heterozygous individuals*). An individual is said to be *homozygous* at a given locus if its two allele copies at that locus are the same. An individual is *heterozygous* at a given locus if its two allele copies at that locus are different.

Definition 2 (*Dominant and recessive alleles*). An allele is said to be dominant if a heterozygote with one copy of this allele expresses the same phenotype, strategy, or behavior as does a homozygote with two copies of the allele. An allele is said to be recessive if a heterozygote with one copy of this allele expresses the same phenotype, strategy, or behavior as does a homozygote with two copies of the other allele. That is, a dominant allele has its full effect even if heterozygous, whereas a recessive individual has no effect unless homozygous.

Definition 3 (*Monomorphic populations*). A population is said to be monomorphic at a genetic locus if all individuals in the population—excepting the occasional rare mutant—have the same pair of identical alleles at this locus. A population is said to be monomorphic if it is monomorphic at all loci considered. For example, in the present model, a population will be considered monomorphic if both the locus controlling parental behavior and the locus controlling offspring behavior are monomorphic.

Definition 4 (*Invasion by a dominant mutant allele*). A dominant mutant allele is said to invade a monomorphic population if it can increase in frequency when rare. More formally, a novel allele can invade if, when an arbitrarily small positive proportion of this allele is added to the original population, the average number of copies of each novel allele that are passed on to surviving members of the adult population of the next generation exceeds the average number of copies of each normal allele that are passed on to surviving members of the adult population of the next generation.

Definition 5 (*Stable monomorphic equilibrium*). A *stable monomorphic equilibrium* is a monomorphic population that cannot be invaded by any possible rare dominant allele.[7]

To determine the equilibrium strategies at the loci encoding juvenile and parental behavior, we will need to take into account the details of the transmission process for these alleles. Suppose an individual receives alleles A_1 and B_1, encoding parental and juvenile behavior respectively, from her mother, and alleles A_2 and B_2 from her father. What combinations will she transmit to her offspring? She might transmit the allele combinations received from a single parent (A_1B_1 or A_2B_2), or she might transmit a combination involving one allele

from her mother and one from her father (A_1B_2 or A_2B_1). The probability that the combination transmitted includes one allele from each of her parents depends on the physical arrangement of the A and B alleles on the chromosomes.

Definition 6 (*Recombination fraction*). The *recombination fraction* between two loci A and B is defined as the probability that an individual receiving alleles A_1B_1 from one parent and A_2B_2 from the other will transmit a *recombinant* allele combination A_1B_2 or A_2B_1 to any given offspring.

Definition 7 (*Linkage*). If the alleles at two loci are transmitted only in the combinations received from an individual's parents, the loci are said to *perfectly linked*, with a recombination fraction $r = 0$. When alleles at the A and B loci assort independently—i.e., when an individual is equally likely to transmit all possible combinations A_1B_1, A_1B_2, A_2B_1, and A_2B_2—the A and B loci are said to be *unlinked*, with a recombination fraction of $r = 1/2$. When the alleles are more likely to be transmitted in the combinations received from the parents, but are not necessary transmitted only in those combinations, the loci are said to be *partially linked* with $0 < r < 1/2$.[8]

The coefficient of relationship between siblings. In general, natural selection will not result in a population of individuals who simply maximize their own survival probability without regard to the survival probabilities of their relatives. The key to understanding the evolution of behavior in games between relatives is to notice the following: a rare allele that affects the behavior of one individual is more likely to be found in close kin than it is to be found in an average member of the population. In games played among siblings, or between parents and offspring, the expected payoff to an individual with a rare allele will be influenced not only by the way that this allele changes its *own* behavior, but also by the probability that its relatives carry the same allele and behave accordingly.

Definition 8 (*Coefficient of relationship*). The *coefficient of relationship* between two relatives, which we will denote by k, is the probability that a rare allele carried by one of them will also be carried by the other.[9]

It is instructive to work through a calculation of the coefficient of relationship between two children born to the same mother. Assuming that this allele is not sex-linked and does not have differential effects on survival of the two sexes, copies of the rare allele in the population are equally likely to be present in males and females. When this allele is rare and mating is random, carriers of the rare allele will almost always mate with an individual who is homozygous for the normal allele. Since children receive one allele from each parent, almost all carriers of the rare allele will be heterozygotes with one copy of the rare allele and one copy of the normal allele; moreover, the rare allele is equally likely to be inherited from the child's father or its mother.

Consider a child who carries the rare allele. Suppose that this child inherited the rare allele from its mother. Then, whether or not its maternal sibling shares the same father, the sibling will almost certainly have a homozygous normal father and thus its paternally-inherited allele will almost certainly be normal. The allele that the sibling inherits from its

mother, however, is equally likely to be a copy of her rare allele or of her normal allele. Therefore, if the child inherits the rare allele from its mother, the probability is 1/2 that its maternal sibling also carries a copy of the rare allele. Now suppose that the child inherits the rare allele from its father. If the child's maternal sibling does not share the same father, then since the allele is rare, the maternal sibling's mother and the father will almost certainly both be homozygous normal and in this case the sibling will certainly not have the rare allele. If the child inherited the rare allele from its father, and its maternal sibling also shares the same father, then with probability 1/2, the sibling will also inherit the rare allele from their father. Therefore, if s is the probability that two offspring of the same mother also have the same father, and if a child has inherited the rare allele from its father, then the probability that its maternal sibling also has the rare allele is $s/2$. Since a child who carries the rare allele is equally likely to have inherited it from its father or from its mother, it follows that for two offspring of the same mother, the coefficient of relationship is $k = 1/2(1/2 + s/2) = (1 + s)/4$. If mating is perfectly monogamous, then $s = 1$ and $k = 1/2$. If females never mate twice with the same individual, then $s = 0$ and $k = 1/4$.

2.1. A simplifying assumption about fertility

In the long run, the alleles that are found in the population will be those that mandate strategies that lead to success in reproduction. In general, the long run reproductive success of a gene may depend on more than the expected number of copies that it produces in the next generation. For example, an individual may be able to produce a greater number of surviving grandchildren by having fewer, but healthier and/or more cooperative children. Similarly, in the case of primogeniture, a parent may maximize the number of descendants by treating some offspring differently from others. In the models considered in this paper, we will avoid these complications by assuming the following:

Assumption 1. The probability that an individual reaches adulthood depends only on its own actions and the actions of its parents and siblings. All individuals who survive to adulthood have the same expected number of offspring.

The reproduction rate of an allele can be measured as the expected number of copies of each allele of its kind that are passed from a surviving adult in one generation to a surviving adult in the next generation. Since we have assumed that all individuals that survive to adulthood are equally fertile, the only variation in the reproduction rates of alleles comes from variation in the probabilities that offspring who carry these alleles will survive to adulthood. Assumption 1 allows us to determine the reproductive success of a rare allele simply by comparing the average survival probabilities of offspring that carry the rare allele with the average survival probabilities of homozygous normal offspring.

Remark 1. Given Assumption 1, a rare dominant allele can invade a monomorphic population if and only if the average survival probability of offspring born with a single copy of the rare allele exceeds the average survival probability of offspring born with two copies of the normal allele.

3. The case of the bleating lamb

Imagine a breed of sheep in which adult females have one lamb per year and survive as adults for at most two years. A more realistic model of ovine reproduction would permit ewes to have more than two fertile seasons, in which case the analysis here would apply to the lambs born in the last two years of their mother's life.[10] These simple two-year sheep will, however, be adequate for illustrating the ideas to be discussed here.

As the first-born lamb matures, it is able to forage for itself, but it still benefits from feeding on its mother's milk. The longer that it continues to nurse, the stronger it will be when winter arrives and the more likely it will be to survive to adulthood. But a long period of nursing is costly to the mother. The sooner the first-born lamb is weaned, the healthier its mother will be when she gives birth to her second lamb, and the more likely the second-born will survive. In evolutionary terms, this conflict of interest between mother and lamb takes the form of a tradeoff between the survival probability of the first-born and that of the second-born lamb.[11]

Let x denote the weaning age of the first-born lamb, where possible weaning ages range from a minimum of $\underline{x}$ to a maximum of $\bar{x}$. We assume that where x is the weaning age of the first-born lamb, the probabilities that the first-born and second-born lambs survive to adulthood are both determined by x, where these probabilities are given by $\Pi_1(x)$ and $\Pi_2(x)$, respectively. We make the following assumptions about the functions $\Pi_1(\cdot)$ and $\Pi_2(\cdot)$.

Assumption 2. Where x is the weaning age and $\Pi_1(x)$ and $\Pi_2(x)$ are the survival probabilities of the first-born and second-born lambs:

(i) $\Pi_1'(x) > 0$ and $\Pi_2'(x) < 0$ for all $x \in [\underline{x}, \bar{x}]$.
(ii) $\Pi_1''(x) < 0$ and $\Pi_2''(x) < 0$ for all $x \in [\underline{x}, \bar{x}]$.
(iii) $\Pi_1'(\underline{x}) > -\Pi_2'(\underline{x})$.
(iv) $\Pi_2(\underline{x}) < \Pi_1(x) + \Pi_2(x)$ for all $x \in [\underline{x}, \bar{x}]$.

Part (i) of Assumption 2 requires that survival probability of the first-born is an increasing function of x while the survival probability of the second-born is a decreasing function of x. Part (ii) assumes that as x increases, the first-born's marginal gain from a longer nursing period diminishes while the second-born's marginal cost from delayed weaning of the first-born increases. Part (iii) assumes that for a first-born who is weaned at the earliest possible age, $\underline{x}$, the marginal gain in survival probability from increasing the nursing period exceeds the marginal cost of this extended weaning to its younger sibling. Part (iv) assumes that the expected number of surviving offspring will be lower if the mother does not nurse her first-born at all, letting it die, than if she nurses it for any positive length of time.

It is instructive to look at a graph that traces out the possible combinations of survival probability for the two offspring that can be attained by varying the weaning age of the first-born. As x is varied from $\underline{x}$ to $\bar{x}$, the survival probability of the first-born increases and the survival probability of the second-born decreases continuously. If survival probability of the first-born is measured on one axis and survival probability of the second-born is

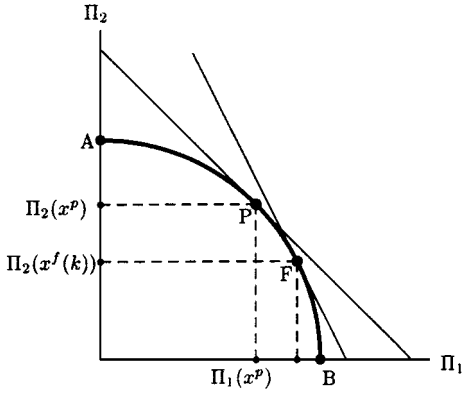

Figure 1. Survival probability frontier.

measured on the other access, the possible combinations of survival probabilities that can be obtained by varying x will trace out a continuous curve which we will call the *survival probability frontier*.[12]

Definition 9 (*Survival probability frontier*). The *survival probability frontier* is the locus of points $(\Pi_1(x), \Pi_2(x))$ where x is between $\underline{x}$ and $\bar{x}$.

The curve *AB* in figure 1 is an example of a survival probability frontier. Taking derivatives, we find that the survival probability has the following properties.

Remark 2. The slope of the survival probability frontier at the point $(\Pi_1(x), \Pi_2(x))$ is $\Pi_2'(x)/\Pi_1'(x)$. Assumption 2 implies that $\Pi_2'(x)/\Pi_1'(x)$ is a decreasing function of x and hence that the survival probability frontier bulges away from the origin, as in figure 1.

3.1. What would the first-born choose?

What age of weaning would we expect to find in a population where first-born lambs could freely dictate the age at which they will stop nursing? We will assume that the age at which a first-born lamb chooses to be weaned is controlled by a single genetic locus and that natural selection operates on the alleles that determine the demands of first-born lambs.

Hamilton (1964) defined the *inclusive* fitness of an individual to be a weighted sum of its own fitness and the fitnesses of its relatives, where the weights are the appropriate coefficients of relatedness. Hamilton proposed that in stable monomorphic equilibrium animals will act toward their relatives in such a way as so as to maximize their own inclusive fitnesses. This proposition has come to be known as 'Hamilton's rule.'

Hamilton's inclusive fitness as applied to first-born lambs in our weaning model is defined as follows.

Definition 10 (*Inclusive fitness of first-born lamb*). For all $x \in [\underline{x}, \bar{x}]$ and $k \in [0, 1]$, the inclusive fitness of a first-born lamb that is weaned at age x is

$$H(x, k) = \Pi_1(x) + k\Pi_2(x). \tag{1}$$

Hamilton's original model focused on a special class of interactions between relatives in which the effect that one's own actions have on a relative's fitness does not depend on the relative's actions and vice versa. There are many plausible examples of game-theoretic interactions between siblings that lack this independence and in which Hamilton's rule does not correctly predict equilibrium behavior.[13]

In the simple weaning model considered in this paper, however, the action of the first-born affects the survival probability of the second-born, but the second-born can not affect the survival probability of the either offspring. In this environment, the independence property assumed by Hamilton is satisfied. We will show that for this model, if first-born lambs could control their age at weaning, then in monomorphic equilibrium they would act as if they were attempting to maximize their inclusive fitness, just as Hamilton's rule would predict.

Lemma 1. *If first-born lambs are able to dictate whether they are weaned at age x or at age x' and if a single genetic locus determines the lambs' demands, then a monomorphic population in which first-born lambs demand to be weaned at x will be invaded by a rare dominant allele which causes lambs to demand to be weaned at x', if and only if $H(x', k) > H(x, k)$ where k is the coefficient of relationship between the two offspring.*

Proof: Consider a monomorphic population in which homozygous normal first-born lambs demand to be nursed until age x. Now suppose that a rare dominant allele appears, such that first-born lambs who carry a copy of the rare allele always demand to be nursed until age $x' \neq x$. Almost every lamb born with the rare allele will have one parent who is heterozygous for this allele and one who is homozygous normal.

Half of the offspring who carry the rare allele will be first-born and half will be second-born lambs. All first-born lambs who carry the rare allele will demand to be nursed until age x' and will have survival probability $\Pi_1(x')$.

The survival probability of a second-born lamb who carries the rare allele will be $\Pi_2(x')$ if its older sibling also carries this allele and $\Pi_2(x)$ if its sibling is homozygous for the normal allele. The probability that a second-born who carries the rare allele has an older sibling who also carries this allele is (by definition) k. The survival probability of a second-born lamb who carries the rare allele is therefore $k\Pi_2(x') + (1 - k)\Pi_2(x)$.

Since half of the carriers of the rare allele are first-born and half are second-born, the average survival probability of offspring who carry the rare allele will be

$$\frac{1}{2}\Pi_1(x') + \frac{1}{2}(k\Pi_2(x') + (1 - k)\Pi_2(x)). \tag{2}$$

Since the alternative allele is rare, almost all copies of the normal allele are carried by homozygous normal individuals who have homozygous normal siblings. Half of them are

older siblings and half are younger siblings. Therefore the average survival probability of carriers of the normal allele is just

$$\frac{1}{2}\Pi_1(x) + \frac{1}{2}\Pi_2(x). \tag{3}$$

It follows that the alternative allele can invade the population only if

$$\frac{1}{2}\Pi_1(x') + \frac{1}{2}(k\Pi_2(x') + (1-k)\Pi_2(x)) > \frac{1}{2}\Pi_1(x) + \frac{1}{2}\Pi_2(x). \tag{4}$$

The expression in Eq. (4) is equivalent to

$$\Pi_1(x') + k\Pi_2(x') > \Pi_1(x) + k\Pi_2(x). \tag{5}$$

which in turn is equivalent to $H(x', k) > H(x, k)$.

Lemma 1 informs us that a population in which lambs choose to be weaned at age x can not be a stable monomorphic population unless the weaning age x maximizes $H(\cdot, k)$ on the interval $[\underline{x}, \bar{x}]$. Lemma 2 will establish that there is one and only one weaning age that satisfies this condition. This allows us to define the first-born's preferred weaning age as a function of the degree of relatedness k between siblings.

Definition 11 (*First-born's preferred weaning age*). For $k \in [0, 1]$, define the first-born's preferred weaning age to be $x^f(k)$ such that for all x in the interval $[\underline{x}, \bar{x}]$, $H(x^f(k), k) \geq H(x, k)$.

Using Assumption 2, it is a matter of straightforward calculus to verify that for all $v \in [0, 1]$ the derivative $H_x(0, v)$ is positive and for all $x \in [\underline{x}, \bar{x}]$, the second derivative $H_{xx}(x, v)$ is negative. It follows that there is a unique x that maximizes $H(\cdot, v)$ on the interval $[\underline{x}, \bar{x}]$. Therefore we have the following result.

Lemma 2. *For all $v \in [0, 1]$, the function $H(\cdot, v)$ is "single-peaked" in x with its peak at $x^f(v)$. That is, $H(x, v)$ is maximized at $x = x^f(v)$ and $H(x, v)$ is strictly increasing in x for $x < x^f(v)$ and strictly decreasing in x for $x > x^f(v)$.*

From Lemmas 1 and 2 it follows that if $x \neq x^f(k)$, a monomorphic population of lambs who demand a weaning age of x can be invaded by a rare dominant allele that causes lambs to demand a weaning age of $x^f(k)$. Thus we conclude the following.

Proposition 1. *If first-born lambs are able to dictate the age at which they are weaned and if a lamb's choice of weaning age is determined by a single genetic locus, then in a stable monomorphic equilibrium it must be that first-born lambs demand a weaning age of $x^f(k)$ where k is the coefficient of relationship between two lambs born to the same mother.*

We can also demonstrate that if first-born lambs can dictate the age of weaning, then in equilibrium the higher the coefficient of relationship between a mother's two lambs, the earlier the first-born will choose to be weaned.

Remark 3. The first-born's preferred weaning age $x^f(k)$ is a decreasing function of the coefficient of relatedness k between maternal siblings.

Proof: The first-order calculus condition for finding $x^f(k)$ is

$$H_x(x, k) = \Pi_1'(x^f(k)) + k\Pi_2'(x^f(k)) = 0 \tag{6}$$

Differentiating both sides of Eq. (6) with respect to k and rearranging terms, we find that

$$\frac{d}{dk}x^f(k) = -\frac{\Pi_2'(x^f(k))}{\Pi_1''(x^f(k)) + k\Pi_2''(x^f(k))} < 0 \tag{7}$$

where the inequality follows from Assumption 2 which requires that $\Pi_2'(x) < 0$, $\Pi_1''(x^f(k)) < 0$ and $\Pi_2''(x^f(k)) < 0$.

It is instructive to look at a geometric representation of these results. Since the point $x^f(k)$ maximizes $H(x, k) = \Pi_1(x) + k\Pi_2(x)$ on the survival possibility frontier, the first-order conditions for maximization require that the slope of the survival possibility frontier, which is

$$\frac{\Pi_2'(x^f(k))}{\Pi_1'(x^f(k))}$$

is equal to $-1/k$. This means that in figure 1 we can find the point $F = (\Pi_1(x^f(k)), \Pi_2(x^f(k)))$, by finding the point at which the survival possibility frontier is tangent to a line with slope $-1/k$. In figure 1, we have drawn a tangent line through the point F with slope -2. This depicts the case of a monogamous species where $k = 1/2$ and $-1/k = -2$. For higher values of k, it must be that $-1/k$ is smaller in absolute value and the tangency will lie further to the left, which corresponds to lower values of x.

3.2. What would mothers choose?

What age of weaning would we expect to find in a population where tough-minded ewes are able to dictate the age of weaning to their pliant lambs?

The answer to this question is simple and not surprising. The only stable monomorphic equilibrium would be one in which each mother weaned her first-born at the age that maximizes the expected total number of her surviving offspring.

Despite the simplicity of this answer, we believe it is important to understand exactly why it is true. Thus, in the Appendix we prove Lemma 3 in what may seem to be excruciating detail. The importance of proving, rather than simply asserting this result will become more

apparent later when we show that if the loci for juvenile and parental behavior are tightly linked, monomorphic populations can sometimes be invaded by alleles that mandate weaning at an age that gives them a smaller expected number of surviving offspring than that enjoyed by normal mothers.

Lemma 3. *If ewes are able to dictate whether their lambs are weaned at age x or at age x' and if a single genetic locus determines a ewe's weaning strategy, then a monomorphic population in which first-born lambs are weaned at x can be invaded by a rare dominant allele for weaning at age x' if and only if* $\Pi_1(x') + \Pi_2(x') > \Pi_1(x) + \Pi_2(x)$.

From Lemma 3, the following result is immediate.

Proposition 2. *If ewes are able to dictate the age at which they wean their first-born and if this action is determined by a single genetic locus, then in a stable monomorphic equilibrium it must be that mothers will wean their first-born at the age x^p where x^p maximizes $\Pi_1(x) + \Pi_2(x)$ on the interval $[\underline{x}, \bar{x}]$.*

This allows us to determine the mother's preferred weaning age.

Definition 12 (*Maternal optimal weaning age*). The maternal optimal weaning age is defined to be x^p where $\Pi_1(x^p) + \Pi_2(x^p) \geq \Pi_1(x) + \Pi_2(x)$ for all x in the interval $[\underline{x}, \bar{x}]$.

The distribution of survival probability between first-born and second-born that corresponds to the maternal optimal weaning age x^p is shown on figure 1. This is the point $P = (\Pi_1(x^p), \Pi_2(x^p))$, where the slope of the survival probability frontier at P is -1.

From the definition of $H(x, v)$, we see that $\Pi_1(x) + \Pi_2(x) = H(x, 1)$. Therefore $x^p = x^f(1)$, and since $k < 1$, it is immediate from Remark 3 that $x^p < x^f(k)$. This implies the following.

Proposition 3. *If mothers are able to dictate the age at which their first-born is weaned, then in stable monomorphic equilibrium, the first-born will be weaned at an earlier age than would be the case in stable monomorphic equilibrium if the first-born were able to dictate the weaning age.*

4. Resolving the genetic conflict

4.1. The lamb who would call wolves

We have shown that in an equilibrium where first-born lambs could choose their age of weaning, they would choose a later date than that which would maximize the number of surviving offspring produced by their mothers. Like Alexander, we may ask, 'So what? Mother sheep are bigger, stronger, and can run faster than their lambs. Surely the mother has the physical ability to enforce her own choice of weaning age.' But, as Eshel and Feldman pointed out, the lamb is not limited to physical coercion as a means of enforcing its will and the resolution of parent-offspring conflict may not always coincide with the parent's will.

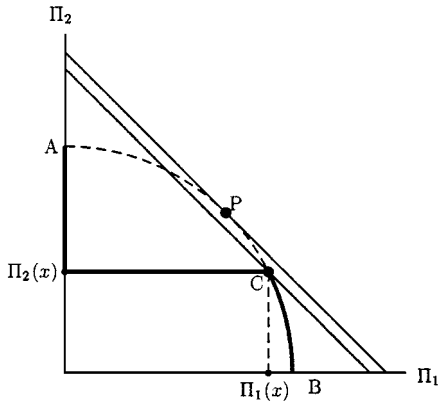

Figure 2. Survival probability frontier with a first-born extortionist.

Let us explore one strategy by which first-born lambs may be able to blackmail their mothers into letting them nurse to an age $x > x^p$ where x^p is the age of weaning that mothers would dictate if they had full control of the behavior of their lambs. Suppose that first-born lambs are genetically programmed to use the following decision rule: 'Demand to be nursed until you reach age x. If you are younger than x and your mother does not let you nurse, then bleat so loudly that you will attract predators.'[14]

If a first-born lamb uses this strategy and if its mother weans it before age x, the lamb will make an awful ruckus, attract a predator, and get eaten; the mother will lose her first-born. The lamb's extortionary strategy changes the shape of the survival probability frontier in such a way that with the altered survival probability frontier, the mother will maximize her number of descendants by yielding to her first-born's demand. In figure 2, we sketch the survival probability frontier *ABCD* for the offspring of a sheep whose first-born lamb adopts the extortionary strategy demanding x. If the mother allows the lamb to nurse until it reaches age x (the lamb's preferred age) it will behave normally, but if the mother attempts to wean it before age x, it will bleat suicidally. The point C represents the distribution of survival probabilities $(\Pi_1(x), \Pi_2(x))$ between her two offspring if the mother accedes to the lamb's demand to be nursed until age x.

When the first-born pursues this extortionary strategy, the distribution of survival probability the mother would have chosen to maximize her expected number of surviving offspring (point P in figure 2) is no longer accessible to her. Given the restricted survival probability frontier imposed by the first-born's threat, the expected number of surviving offspring is maximized at the point $C = (\Pi_1(x), \Pi_2(x))$.

4.2. Equilibrating behavior of parents and offspring

Readers familiar with the notion of subgame perfection in game theory are likely to be skeptical that a first-born lamb's threat 'Feed me or I will bleat until a wolf eats me,' would be respected in equilibrium. Certainly this threat is not credible in the sense that if a rational

lamb who made this threat was ignored by its mother, it would not find it in its interest to commit suicide by bleating. Rational mother sheep, in appraising this situation would realize that their rational progeny would not carry out this incredible threat and would ignore it.[15] But even economists are likely to quail at attributing such powers of ratiocination to a sheep. Instead of making a priori assumptions on the mental abilities of sheep, we will posit that they are genetically programmed to use strategies as lambs and adults that lead to successful reproduction of the genes that program their behaviors and we will investigate possible equilibrium outcomes. In fact, we will see that genetically programmed strategies allow players to commit to playing subgame-imperfect strategies in a manner which would not be possible for rational players.

An evolutionary model of the resolution of conflicting reproductive interests of parents and offspring requires that we specify the genetic basis of childhood behavior, and its relation to adult behavior. In this discussion, we will assume that the alleles at one genetic locus control an individual's behavior towards its parents and siblings when it is a child, and that the alleles at a second locus control its behavior as a mother. As we will show, the extent of linkage between these two loci will be crucial in determining the resolution of this conflict.

It will be useful to spell out more detailed 'rules of the game' that apply in encounters between a first-born lamb and its mother. Let us assume that each day after the lamb reaches the first possible age of weaning, its mother either offers to nurse the lamb or she refuses to nurse it. If the mother offers to nurse the lamb, the lamb can either accept nursing or refuse to be nursed. If the mother refuses to nurse the lamb, it can either submit to its mother's refusal or loudly demand to be fed despite its mother's refusal.

We will simplify our task by confining our attention to a restricted class of strategies. We assume that mothers must use a strategy from a class of strategies that we call *x-offer strategies* and that lambs must use a strategy from a class of strategies that we call *x-demand strategies*.

A ewe who follows an *x-offer strategy* will act according to the rule: 'Offer to nurse your first-born if it is younger than x. Refuse to allow it to nurse if it is older than x.'

We consider two kinds of x-demand strategies, which differ in the action that a lamb takes if its mother offers to let it nurse beyond the age x. A lamb that uses either type of x-demand strategy will bleat and demand to be fed if its mother refuses to nurse it before it reaches age x. A lamb who follows the *greedy x-demand strategy* will continue to nurse beyond age x if its mother permits it to do so. A lamb who follows the *temperate x-demand strategy* will reject nursing after it reaches age x even if its mother would permit it to nurse.

4.3. Invasion one locus at a time

A necessary condition for a population to be a stable monomorphic equilibrium in a two-locus model is that this population can not be invaded by a rare dominant allele at either of the two loci. In this section, we will show that for our weaning model, this necessary condition does nothing to narrow the range of possible resolutions of the conflict between parent and offspring. In fact the maternal optimum x^p as well the preferred age of first-born $x^f(k)$ and all ages in between can be maintained by monomorphic populations that can

not be invaded by rare alleles at a single locus. This is stated formally by the following proposition, which we prove in the Appendix.

Proposition 4. *For any x such that $x^p \leq x \leq x^f(k)$, a monomorphic population in which all first-born lambs use x-demand strategies and all mothers use x-offer strategies cannot be invaded by a dominant allele that mandates that first-borns use an x'-demand strategy for $x' \neq x$, nor can it be invaded by a dominant allele that mandates that mothers use x'-offer strategies for $x' \neq x$.*

Proposition 4 establishes conditions under which a monomorphic population cannot be invaded by a rare allele at a single locus. As we will show in later discussion, this result is not sufficient to establish that these outcomes are stable against invasion by pairs of rare alleles at the two loci, even when these loci are *unlinked*.

4.4. Perfectly linked loci

In a two-locus model, a monomorphic population that is stable against independent invasions at each of the two loci might still be invaded simultaneously by small populations of two dominant mutant alleles, one at each locus, where these alternative alleles have complementary effects. It is particularly easy to see how this works in the case where the two loci are perfectly linked. A two-locus model with perfect linkage is equivalent to a single-locus model, where the characteristics controlled by the two loci are both determined by the contents of a single locus. For example, a two-locus parent-offspring model with perfect linkage is equivalent to one in which a single locus controls behavior of individuals both when they are children and when they are parents. It might be that a monomorphic *can* be invaded by an allele that changed the behavior of *both* parents and children although the original population cannot be invaded either by a novel allele that changes behavior of children and not of parents, or by a novel allele that changes behavior of parents but not children.

In our simple weaning model, if there is perfect linkage between the loci that determine the behavior of first-born lambs and the behavior of mother sheep, we have a striking result. *The only stable monomorphic equilibrium is one in which all lambs are weaned at the maternal optimal age x^p.*

In order to show this result, we find it useful to consider two particular alleles, one found at the locus that controls juvenile behavior and the other found at the locus that controls maternal behavior. We will call these the *Alexandrian alleles*. If a mother and her lamb both have these alleles the lamb and mother will both act to optimize the mother's reproductive interest. The Alexandrian allele for juvenile behavior causes first-born lambs to adopt the greedy $\underline{x}$ strategy. The Alexandrian allele for maternal behavior causes a mother to nurse her first-born until it reaches the maternal optimal weaning age x^p and no longer. We will call a first-born lamb who carries the Alexandrian allele for juvenile behavior an *Alexandrian lamb*, and a mother who carries the Alexandrian allele for maternal behavior an *Alexandrian ewe*, and we will call a sheep who carries both Alexandrian alleles a *fully Alexandrian sheep*. Since $\underline{x}$ is the minimum possible weaning age, we see that an Alexandrian lamb will never

demand to nurse unless its mother offers, but will always accept nursing if offered. In a monomorphic population of fully Alexandrian sheep, every mother would offer to nurse her first-born offspring until it reached age x^p, and every first-born would nurse until this age and then stop nursing without complaint. In the appendix we prove the following.

Proposition 5. *With perfect linkage between the loci controlling the behavior of first-born lambs and the behavior of mother sheep, any monomorphic population where lambs use x-demand strategies and ewes use x-offer strategies and where $x \neq x^p$ can be invaded by the pair of Alexandrian alleles. Conversely, a monomorphic population of Alexandrian sheep cannot be invaded by rare alleles for using any alternative combination of strategies by lambs and mothers.*

4.5. Partially linked loci

If two loci are perfectly linked and if individuals carrying a novel allele at each locus reproduce more rapidly than normal individuals, then the novel allele combination will be able to invade the population. But if the loci are less than perfectly linked, this is not necessarily the case. In a population where the alternative alleles are rare and where mating is random, almost all individuals who carry both alternative alleles will be heterozygous at both loci. Only the fraction $1-r$ of these double heterozygotes will 'breed true' in the sense of transmitting the two rare alleles together rather than separately. Given that individuals who carry only one of the two rare alleles reproduce less rapidly than normal individuals, the pair of rare alleles will be able to invade the population only if the reproduction rate of double heterozygotes is greater than $1-r$ times as large as that of the normals. We sketch a proof of this lemma in the Appendix.

Lemma 4. *In a two-locus model that satisfies Assumption* 1*, consider a monomorphic population that cannot be independently invaded by a mutant allele at either of the two loci. Let the recombination fraction between the two loci be r where $0 \leq r \leq 1$. Suppose that the offspring of individuals with two normal alleles at each locus have a survival probability of S, and that double heterozygote offspring with one normal parent and one double heterozygote parent have a survival probability of S'. The pair of novel alleles will be able to invade the initial monomorphic population if and only if $(1-r)S' > S$.*

The following result is an immediate consequence of Lemma 4.

Proposition 6. *Suppose that the nursing demand strategies of first-born lambs and the nursing offer strategies of ewes are controlled by two genetic loci which have a recombination fraction r. A monomorphic population where first-borns use x-demand strategies and mothers use x-offer strategies can be invaded jointly by a pair of alleles that mandate x'-demand strategies for offspring and x'-offer strategies for first-born if and only if*

$$(1-r)(\Pi_1(x') + \Pi_2(x')) > \Pi_1(x) + \Pi_2(x). \tag{8}$$

In the Appendix we prove a pair of corollaries to Proposition 6 that directly address the question of whether the conflict of genetic interest between parents and offspring will be resolved in favor of parents or of children. The first of these shows that with a mild additional assumption on payoffs, if the loci that control juvenile and maternal behavior are unlinked, then any weaning age between the maternal optimum and the first-born's preferred weaning age can be sustained as a monomorphic equilibrium.

Corollary 1. *If the loci that determine behavior of first-born lambs and of mothers are unlinked (so that $r = 1/2$) and if $\Pi_1(x^p) \geq \Pi_2(x^p)$, then for all x such that $x^p \leq x \leq x^f(k)$, there is a stable monomorphic equilibrium in which lambs are weaned at age x.*

The second result shows that as the degree of linkage increases, the range of possible weaning ages in stable monomorphic equilibria converges toward the maternal optimal age.

Corollary 2. *Where the recombination fraction is r, the set of weaning ages that are consistent with a stable monomorphic equilibrium is an interval with lower bound x^p and with an upper bound that decreases as r decreases and approaches x^p as r approaches zero.*

4.6. Mothers don't always seek to maximize expected number of surviving offspring

Earlier, we promised to show that where there is linkage between the loci controlling maternal and juvenile behavior, selection may not favor maternal behavior that maximizes the number surviving offspring. Here is an example.

Let us suppose that the loci that control the behavior of first-born lambs and of mother sheep are perfectly linked. Consider a monomorphic population in which normal first-born lambs pursue x-demand strategies and normal mothers pursue x-offer strategies, where $x > x^p$. The expected number of surviving offspring that a normal mother will have is $\Pi_1(x) + \Pi_2(x)$. Now suppose that a small proportion of paired Alexandrian alleles is introduced into this monomorphic population. The first-born Alexandrian lambs will use the greedy $\underline{x}$-demand strategy and the Alexandrian ewes will use the x^p-offer strategy.

When the Alexandrian alleles are rare, a mother sheep who carries both Alexandrian alleles will almost certainly be a heterozygote at each locus. With probability 1/2, an Alexandrian ewe's first-born will inherit her Alexandrian alleles. In this case it nurses until it reaches the age x^p at which time its mother stops offering to nurse it and it then stops nursing without complaint. Then its survival probability will be $\Pi_1(x^p)$. With probability 1/2, the first-born of an Alexandrian ewe will not inherit her Alexandrian alleles. In this case it uses the x-demand strategy. Since its mother refuses to nurse her first-born beyond age x^p, the lamb will not survive. Whether or not her first-born has Alexandrian alleles, the mother will wean it at age x^p and hence the survival probability of the second-born will be $\Pi_2(x^p)$. It follows that the expected number of surviving offspring that the mother produces is

$$\frac{\Pi_1(x^p)}{2} + \Pi_2(x^p).$$

As we know from Proposition 5, the pair of Alexandrian alleles will invade the original monomorphic population. But while they are rare, Alexandrian ewes actually have a smaller expected number of surviving offspring than normal ewes (at least if x is sufficiently close to x^p). We can see this as follows. The difference between the expected number of surviving offspring of a normal ewe and an Alexandrian ewe is

$$\Pi_1(x) + \Pi_2(x) - \left(\frac{\Pi_1(x^p)}{2} + \Pi_2(x^p)\right) \tag{9}$$

Since $x > x^p$, the Expression 9 will have the same sign as

$$\frac{\Pi_1(x) - \Pi_1(x^p)}{x - x^p} + \frac{\Pi_2(x) - \Pi_2(x^p)}{x - x^p} + \frac{\Pi_1(x^p)}{2(x - x^p)} \tag{10}$$

In the limit as $x \to x^p$, the first two terms of Expression 10 approach respectively $\Pi_1'(x^p)$ and $\Pi_2'(x^p)$, both of which are finite, while the third term approaches infinity. This implies that for x sufficiently close to x^p, the expected number of surviving offspring of Alexandrian ewes is lower than that of normal ewes.

How can a rare Alexandrian allele invade, even though mothers who carry this allele have fewer expected surviving offspring than normal mothers? The answer is simple and instructive. Although Alexandrian ewes, unlike normal ewes, lose half of their first-born to wolves, the offspring that are lost to wolves do not carry the Alexandrian allele pair. In fact for an Alexandrian ewe, the number of surviving offspring who carry the Alexandrian gene pair is $(\Pi_1(x^p) + \Pi_2(x^p))/2$. Hence the expected number of copies of the Alexandrian alleles that are passed to surviving adults in the next generation is also $(\Pi_1(x^p) + \Pi_2(x^p))/2$. This exceeds the number of copies $(\Pi_1(x) + \Pi_2(x))/2$ of a normal allele that are passed from one generation to the next.

5. Conclusions

There is much to be learned from our simple pastoral fable of weaning conflict. We began by posing a pair of hypothetical questions. At one extreme, at what age will first-born lambs be weaned if mothers have no control of the weaning age and natural selection operates exclusively on the age at which first-borns choose to wean themselves? At the other, at what age will first-born lambs be weaned if mothers have unchallenged control over weaning age and natural selection acts exclusively on the weaning age that mothers choose?

In this model, the answers to both questions turn out to be answers that would be predicted by users of Hamilton's rule. If first-born lambs could choose, they would pick the age of weaning that maximizes a weighted average of their own survival probability and that of their younger siblings. Here, the relative weight placed on the younger sibling is simply the coefficient of relationship between a mother's two lambs. If mothers could choose, they would pick the age of weaning that maximizes an equally weighted average of survival probabilities of first and second born. The weaning age $x^f(k)$ that the first-born would choose is always greater than the age x^p that ewes would choose.

The answers to these questions define the extent of the genetic conflict over weaning age. How will this conflict be resolved? We find that the answer to *this* question depends on the strategies available to mother and offspring, and on the details of the process by which these strategies are inherited. We first considered the possibility of unilateral change in offspring strategies or maternal strategies. We found that there is a large class of monomorphic equilibria that cannot be invaded unilaterally by mutant alleles at the locus that controls the behavior of lambs, or by mutants at the locus controlling the behavior of mothers. Such equilibria support any age of weaning x between the parental optimum x^p and the offspring optimum $x^f(k)$. Thus if single-locus mutations were the only kind observed, there would be little theoretical support for the Alexander view that natural selection would inevitably result in the outcome x^p that maximizes the reproductive interests of the mother.

Two-locus genetic models, however, allow the possibility that novel pairs of maternal and offspring strategies can invade in association with one another. We found that this possibility gives more support to the Alexander-Becker viewpoint. If the genetic loci that control behavior of first-born and behavior of mothers are perfectly linked, then the Alexander-Becker view is dramatically vindicated. The *only* outcome that can be a monomorphic equilibrium is the one in which mothers are able to enforce their will, i.e., in which first-born are weaned at their mothers' preferred outcome x^p. In intermediate cases, where linkage is not perfect, we have intermediate results. The greater the probability that genetic recombination will break up allele combinations at maternal and offspring strategy loci, the further the equilibria can stray from the mothers' preferred outcome. For sufficiently loose linkage, the Alexander-Becker position again collapses and we find that every weaning age between the maternal optimum x^p and the first-born's preferred weaning age $x^f(k)$ can be an equilibrium.

Appendix

Proof of Lemma 3: Suppose that the ewe is able to dictate the age of weaning absolutely, without resistance from her offspring. Then we need to concern ourselves only with a one-locus model in which the pair of alleles at a single locus controls the age at which mothers wean their first-born. Consider a monomorphic population in which mothers wean their offspring at age x. Suppose that to this population is added a small proportion of an alternative dominant allele, such that mothers heterozygous for this alternative allele wean their offspring at age x'.

As we noted in Remark 1, Assumption 1 enables us to determine whether the alternative allele can invade the population by comparing the average survival probability of lambs born with a single copy of the rare allele to that of lambs born with two copies of the normal allele.

Since mating is random and the alternative allele is rare, almost all individuals with this allele will be heterozygotes, with one copy of the alternative allele and one copy of the normal allele. A lamb who inherits the rare allele is equally likely to be a first-born or a second-born and (independently of whether it is first-born or second-born) is equally likely to inherit the allele from its mother or from its father. Thus 1/4 of all lambs fall into each of these four categories.

Since mothers who carry the alternative allele wean their offspring at age x', a first-born who inherits this allele from its mother is weaned at age x' and has survival probability of $\Pi_1(x')$. If a second-born inherits this allele from its mother, the mother will have weaned the first-born at age x' and the survival probability of the second-born must be $\Pi_2(x')$.

If a lamb inherits the alternative allele from its father , then since mating is random and the alternative allele is rare, the lamb will almost certainly have a homozygous normal mother, who will wean her first-born at age x. Therefore a first-born who inherits the alternative allele from its father will have survival probability $\Pi_1(x)$ and a second-born who inherits the alternative allele from its father will have survival probability $\Pi_2(x)$.

It follows that the average survival probability of lambs born with the mutant allele is

$$\frac{1}{4}(\Pi_1(x') + \Pi_2(x') + \Pi_1(x) + \Pi_2(x)). \tag{11}$$

Since the alternative allele is rare, almost all homozygous normal offspring have homozygous normal mothers who wean their first-born at age x. These offspring are equally likely to be first-born or second-born, so their average survival probability is

$$(\Pi_1(x) + \Pi_2(x))/2 \tag{12}$$

The alternative allele for weaning at age x' will be able to invade a monomorphic population of mothers who wean at age x if and only if Expression 11 exceeds Expression 12. Subtracting Expression 12 from Expression 11 and multiplying the result by 4, we see that the rare allele will invade if and only if

$$\Pi_1(x') + \Pi_2(x') > \Pi_1(x) + \Pi_2(x). \tag{13}$$

Proof of Proposition 4: Let us first show that the normal population cannot be invaded by an allele that causes lambs to demand nursing until an age greater than x. Suppose that a rare allele at the locus controlling juvenile behavior causes first-born lambs to use an x'-demand strategy, where $x' > x$. Since mothers all use the x-offer strategy, a first-born lamb that uses x'-strategy will be confronted with a mother who refuses to let it nurse after age x. A first-born carrying this rare allele will not be able to nurse any longer than a normal lamb, but will reduce its survival probability by bleating when its demand is refused. Whether or not its older sibling carries the rare allele, a second-born lamb who carries the rare allele will have survival probability $\Pi_2(x)$ since its mother will nurse the first-born only until age x. Thus first-born carriers of the rare allele have lower survival probability than first-borns with two normal alleles, and second-born carriers of the rare allele have the same survival probability as normals. It follows that on average, carriers of the rare allele will have lower survival probability than normals and hence this allele cannot invade.

Next we show that the normal population cannot be invaded by an allele that causes first-born lambs to accept a weaning age $x' < x$, regardless of whether they use a greedy x'-demand strategy or a temperate x'-demand strategy. Since its mother would offer to nurse it until age x, a first-born lamb carrying a dominant allele for the greedy x'-demand

strategy would be offered—and would accept—the opportunity to nurse until the normal age x. Therefore both first-born and second-born carriers of this allele would have exactly the same survival probability as first-borns and second-borns in the normal population and could not invade.[16] A first-born lamb with a dominant allele for the temperate x'-demand strategy would be offered the chance to nurse until age x but would stop nursing at age x'. Since $x' < x < x^f(k)$, it follows from Remark 2 that $H(x', k) < H(x, k)$ and therefore Proposition 1 implies that the allele for a temperate x'-demand strategy cannot invade the original population.

It remains to be demonstrated that where $x^p \leq x \leq x^f(k)$, a normal population of x-demander first-borns and x-offerer mothers can not be invaded by an allele that alters maternal behavior. Where $x' < x$, a mother who carries a rare dominant allele for an x'-offer strategy will lose her offspring to wolves. If a mother uses an x'-offer strategy where $x' > x$, then those of her offspring who are greedy x-demand strategists will nurse until age x' and those who are temperate x-demand strategists will continue to nurse only until age x. Since $\Pi_1(x) + \Pi_2(x)$ is a decreasing function of x for $x > x^p$, it follows that the expected number of surviving offspring of the rare x'-offer strategists will be no higher than that of normal parents (and strictly lower if some of the offspring are greedy x-demand strategists.)

Proof of Proposition 5: Suppose that a small proportion of perfectly linked mutant Alexandrian alleles for first-born behavior and the behavior of mothers appears in a monomorphic population in which lambs use x-demand strategies and ewes use x-offer strategies.

If a first-born lamb inherits the Alexandrian alleles from its mother, then the mother must be using an x^p-offer strategy and so the lamb will be weaned at age x^p and have survival probability $\Pi_1(x^p)$. If a second-born lamb inherits the Alexandrian allele from its mother, then it must be that the mother weans its older sibling no later than x^p [17] and so the survival probability of the younger sibling will be at least $\Pi_2(x^p)$. Since a lamb inheriting the rare allele pair from its mother is equally likely to be first-born or second-born, the average survival probability of lambs that inherit the rare Alexandrian allele pairs from their mothers is at least $(\Pi_1(x^p) + \Pi_2(x^p))/2$.

If a first-born lamb inherits the rare Alexandrian alleles from its father, then its mother almost certainly will be homozygous normal and will use an x-offer strategy. A first-born lamb who inherits the Alexandrian alleles from its father will be offered nursing until age x by its normal mother. Since an Alexandrian lamb accepts exactly what its mother offers, such a first-born will be weaned at age x and have survival probability $\Pi_1(x)$. Since the first-born is weaned at age x, regardless of whether it carries an Alexandrian allele or two normal alleles, the second-born will have survival probability $\Pi_2(x)$. Therefore the average survival probability of lambs that inherit the rare Alexandrian alleles from their fathers is $(\Pi_1(x) + \Pi_2(x))/2$.

A lamb that is born with the Alexandrian alleles is equally likely to have inherited this allele from its mother or from its father. Therefore the average survival probability of lambs that carry this allele pair is:

$$\frac{1}{4}((\Pi_1(x^p) + \Pi_2(x^p) + \Pi_1(x) + \Pi_2(x)) \tag{14}$$

Since the average survival probability of normal first-born lambs is

$$(\Pi_1(x) + \Pi_2(x))/2, \tag{15}$$

the Alexandrian alleles will be able to invade the normal population if Expression 14 exceeds Expression 15. Subtracting the latter expression from the former and multiplying by 4, we see that this is equivalent to

$$\Pi_1(x^p) + \Pi_2(x^p) > \Pi_1(x) + \Pi_2(x) \tag{16}$$

which is always true since x^p strictly maximizes $\Pi_1(x^p) + \Pi_2(x^p)$. It follows that the Alexandrian alleles can always invade the original population.

Verifying the converse statement is straightforward.

Proof of Lemma 4: Consider a population that is orginally monomorphic at two loci, A and B, where the normal alleles at these loci are denoted A_1 and B_1 respectively. Suppose that a small proportion of alternative alleles arises, where the alternative allele at locus A is denoted A_2 and the alternative allele at B is denoted B_2. Since mating is random, almost all individuals carrying the rare alternative alleles will mate with individuals who are homozygous for the normal alleles at both loci. Therefore almost all carriers of the alternative alleles will be single or double heterozygotes, having inherited either one or two of the rare alleles from a single parent. Individuals who are heterozygous at the A locus but homozygous for the normal allele at the B locus (i.e., $A_1A_2B_1B_1$ individuals) will be called 'Type 1' individuals. Those who are heterozygous at the B locus but homozygous at the A locus will be called 'Type 2' individuals. Individuals who are heterozygous at both loci will be called 'Type 3' individuals. For $i = 1, 2, 3$, let $\epsilon_i(t)$ denote the fraction of the entire population that is type i at time t.

Let S_i be the ratio of the expected number of surviving offspring produced by a Type i individual relative to the expected number of surviving offspring of normal individuals. Where r is the recombination fraction, we see that Type 1 individuals can be produced either as the offspring of another Type 1 individual with a normal mate, or as the offspring of a Type 3 in the event of recombination. Similarly for Type 2 individuals. Therefore when the alleles A_2 and B_2 are rare, we have

$$\epsilon_1(t+1) = S_1\epsilon_1(t) + \frac{1}{2}rS_3(t)\epsilon_3(t) \text{ and} \tag{17}$$

$$\epsilon_2(t+1) = S_2(t)\epsilon_2(t) + \frac{1}{2}rS_3(t)\epsilon_3(t). \tag{18}$$

Type 3 individuals can be produced either as offspring of double heterozygotes when recombination does not occur, or they can occur as offspring of a mating between a Type 1 and a Type 2 individual in the event that recombination *does* occur. Since mating is random and Types 1 and 2 are rare, the proportion of matches between Types 1 and 2 to the number of

these types is becomes arbitrarily small as ϵ_1 and ϵ_2 become small. Therefore the equation for $e_3(t+1)$ can be written as

$$\epsilon_3(t+1) = (1-r)\epsilon_3(t) + O(\epsilon) \tag{19}$$

where the term $O(\epsilon)$ can be made arbitrarily small as the invading proportions ϵ_i are made small. It is easily verified that the eigenvalues of the system of equations are arbitrarily close to S_1, S_2, and $(1-r)S_3$ when the rare alleles are sufficiently rare. By assumption $0 < S_1 < 1$ and $0 < S_2 < 1$. Therefore, when the proportion of invaders is small, all of the eigenvalues of the system will be between 0 and 1 if and only if $(1-r)S_3 < 1$.

Proof of Corollary 1: Since $\Pi_1(\cdot)$ is an increasing function, since by assumption $\Pi_1(x^p) \geq \Pi_2(x^p)$, and since (by definition of x^p) $\Pi_1(x^p) + \Pi_2(x^p) \geq \Pi_1(x') + \Pi_2(x')$ for all $x' \in \{\underline{x}, \bar{x}\}$, it must be that for all $x > x^p$,

$$\begin{aligned} \Pi_1(x) + \Pi_2(x) &> \Pi_1(x^p) \\ &= \frac{1}{2}(\Pi_1(x^p) + \Pi_1(x^p)) \\ &\geq \frac{1}{2}(\Pi_1(x^p) + \Pi_2(x^p)) \\ &\geq \frac{1}{2}(\Pi_1(x') + \Pi_2(x'))) \end{aligned} \tag{20}$$

for all $x' \in \{\underline{x}, \bar{x}\}$. But from Proposition 6 it follows that when $r = 1/2$ a monomorphic population in which all offspring use x-demand strategies and all mothers use x-offer strategies can be invaded by a pair of alleles that mandate x'-demand strategies for offspring and x'-offer strategies for mothers only if

$$\Pi_1(x) + \Pi_2(x) < \frac{1}{2}(\Pi_1(x') + \Pi_2(x')). \tag{21}$$

Therefore the initial monomorphic equilibrium cannot be invaded by any pair of alleles that mandate an x'-demand strategy in lambs and an x'-supply strategy in mothers. It easy to see that if no pair of "coordinated" strategies of this type can invade, then no other pair of strategies can invade the initial population.

Proof of Corollary 2: From Proposition 4 it follows that a monomorphic population in which lambs use x-demand strategies and mother sheep use x-offer strategies can be invaded by Alexandrian alleles if and only if

$$(1-r)(\Pi_1(\cdot) + \Pi_2(\cdot)) > \Pi_1(\cdot) + \Pi_2(\cdot). \tag{22}$$

According to Lemma 2 the function $\Pi_1(\cdot) + \Pi_2(\cdot)$ is a continuous single-peaked function of x that is maximized at x^p. Therefore the set of x's in the interval $[x^p, x^f(k)]$ for which Inequality 22 is true must be an interval with greatest lower bound x^p and with a least upper bound $x(r)$ such that $x(r)$ is an decreasing function of r and where $\lim_{r\to 0} x(r) = x^p$.

Acknowledgements

C. Bergstrom was supported by a Howard Hughes Medical Institute Predoctoral Fellowship and by National Institutes of Health grant GM 28016. The authors thank two anonymous referees.

Notes

1. This literature is ably surveyed by Browning (1992).
2. A definition of the *coefficient of relationship* between two individuals appears later in this paper. (Definition 8)
3. Trivers' (1985) textbook *Social Evolution* devotes a chapter to parent-offspring conflict, with several engaging descriptions of efforts by bird and mammal offspring to manipulate their parents and vice versa.
4. By this device, we sidestep many interesting issues of sibling rivalry that arise when siblings are alive concurrently and compete directly with each other. We hope to address some of these matters in a sequel to this paper. An excellent source of information on this topic is Mock & Parker's (1997) book, which contains thorough accounts both of theory and field observation of sibling rivalry in birds, mammals, insects, and plants.
5. In this paper we consider only autosomal (non-sex-linked) genes, which obey the simple rules of Mendelian inheritance. Genes located on the sex chromosomes (the X and Y chromosomes in mammals) follow somewhat more complicated rules of inheritance.
6. For economists who want to read more about two-locus models, we recommend chapter 8 of Roughgarden's (1996) textbook as a clear introduction to this topic. More detailed and general discussions of conditions under which a single mutant allele can invade a population in a multilocus model can be found in Eshel & Feldman (1984) and Liberman (1988).
7. Because we define invasion to occur only when invading gene reproduces *faster* than the normals, this is weaker notion of a stable equilibrium than one which excludes 'drift' among alleles that reproduce equally rapidly. It is also a weaker concept than Maynard Smith's notion of evolutionarily stable strategies since we do not impose restrictions on the case in which a mutant gene reproduces exactly as rapidly as the normal genes. Thus the equilibria studied here may not be resistant to the possibility of novel alleles reaching high frequency by drift.
8. Recombination fractions greater than 1/2 are almost never observed in nature.
9. A more commonly-used definition (as applied to sexual diploids) is 'The coefficient of relationship between two individuals is the proportion of genes in one that are "identical by descent" to genes present in the other.' For the study of invasion of a monomorphic population by rare mutant alleles, our definition operationalizes the standard definition.
10. Unless female sheep have been secretly cloning themselves for some time, we would not expect to find surviving real breeds in which ewes on average have fewer than two surviving lambs per lifetime. Economists, of course, are accustomed to dealing with abstract sheep—the traditional economists' sheep, unlike a real sheep, produces wool and mutton in fixed, unalterable proportions.
11. Haig (1992, p. 153) describes the situation with a colorful analogy: 'Suppose that a mother buys a milkshake to be shared among her children, but the milkshake has only a single straw. If the first child takes a drink and passes the remainder on to the second, and so on down the line, then the greater the consumption of each child, the fewer children receive a drink.'
12. This curve is conceptually the same as the 'utility possibility frontier' that is commonly used in the theory of welfare economics.
13. This was observed by Grafen (1979) and by Cavalli-Sforza & Feldman (1978, 1981). Mock & Parker (1997, pp. 19–28) offer some particularly compelling examples of sibling interactions for which Hamilton's rule is violated. Hines & Maynard Smith (1979) and Bergstrom (1995) offer extensions of Hamilton's rule that apply to a broad class of game-theoretic interactions for which the original formulation of Hamilton's rule is not appropriate.
14. This is by no means the only sort of strategy an offspring might use to influence its mother (for example, Eshel & Feldman (1991) consider a different class of strategies that serve a similar purpose).
15. Arthur Robson relates that as a child, he once threatened to hold his breath until his mother bought him an ice cream cone. His mother, ever sensible, refused to capitulate. Given that Arthur himself now tells this story, we can infer that his threat was not credible.

16. Recall that by our definition, a rare allele can invade only if it reproduces *faster* than normal alleles.
17. Possibly earlier, if in the original population the older sibling is a temperate x demander and $x < x^p$.

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