

# On the Evolution of Juvenile Life-Styles in Mammals

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Growing up is not easy. Small size and inexperience guarantee that youngsters will be more vulnerable to predation and less able to compete for critical resources than adults. Yet the costs of not overcoming these inherent problems are high. Since youngsters at some point must strike out on their own and attempt to reproduce for themselves, what transpires while growing up should have a dramatic effect on subsequent reproductive success. Whereas enriched ontogenies might enhance adult opportunities, impoverished circumstances, by limiting growth and experience, might hinder them. Small size will reduce competitive ability and limit fecundity. Moreover, small size could force individuals to mature either very early because of their deficiencies or very late as they delay attempting to compensate for them. If breeding begins too early before necessary skills are mastered or when fecundity is directly limited by size, reproductive success will be reduced. Similarly, if breeding is delayed too long, even if initial deficiencies are completely eliminated, opportunities will have been lost. Consequently, it is not surprising that natural selection has tailored the age of maturity, or the age of first reproduction, so that it maximizes an organism's Darwinian fitness (Charlesworth 1980; Stearns & Koella 1986).

Age of first reproduction is one of many life-history traits that appear to be highly correlated. Many studies on a variety of mammalian groups (e.g., Millar, 1977, 1981; Western 1979;

Harvey & Clutton-Brock 1985; May & Rubenstein 1985; Boyce 1988; Ross 1988; Promislow & Harvey 1990) show that fast breeding species tend to be small, to have short gestation and nursing periods, and to produce large litters of small young, whereas slow breeding species tend to be large, to show prolonged developments, and to produce a few large young. In all instances these traits scale allometrically, not geometrically, with body size (Calder 1984; Reiss 1989). The theory of *r*- and *K*-selection argues that selection has acted on different species to fashion life histories that allow them to succeed in different demographic or ecological conditions. Extensions of the theory (Charnov & Schaffer 1973; Caswell 1983; Horn & Rubenstein 1984) and analysis of empirical data (Harvey & Zammuto 1985; Promislow & Harvey 1990) suggest that once body size is held constant, species with high unavoidable juvenile mortality relative to adult mortality should tend to delay the onset of sexual maturity.

Although the onset of breeding almost always occurs at the end of the juvenile period, the age of maturity and the length of the juvenile period are not synonymous. Ontogeny entails a gradual lessening of dependence on parents. In mammals, weaning begins a phase of the juvenile period when direct nutritional dependence on mother's milk ceases. Yet until the onset of breeding, parental assistance in terms of protection from predators and competitors and aid in developing important motor and social skills

continues. Moreover, since so much of parental investment and care is intended to prevent mortality among young, features of the juvenile period, as well as the forces that shape its duration, must be examined.

It is the aim of this chapter to investigate the features that constitute and determine the length and function of the juvenile period. We will begin by developing a simple model that shows what factors influence the optimal timing of sexual maturity. Then we will examine how mortality patterns in mammals—especially those of rodents, carnivores, primates, and ungulates—help shape juvenile life-styles in order to evaluate the predictions of the model. Finally, we shift from inter- to intraspecific case studies to determine how different types of parenting produce different juvenile life-styles and whether they affect adult reproductive success and longevity.

## THE THEORY

In growing populations the intrinsic rate of growth,  $r$ , also known as the Malthusian parameter, is usually considered the appropriate measure of fitness (Charlesworth 1980). It is related to other life-history features as

$$r = \ln R_0/T_c \quad (1)$$

where  $R_0$  is the average number of female offspring produced over a female's lifetime, and  $T_c$  is the "cohort generation time," which corresponds closely with our intuitive notion of generation length and is roughly proportional to the time it takes to reach sexual maturity (May & Rubenstein 1985). Since the lifetime reproductive success term contributes to fitness logarithmically, hence weakly, fitness in an *expanding* population ( $R_0 > 1$ ) depends mostly on the age of first reproduction. Individuals that mature early have an advantage.

Most extant mammal populations, however, are rarely found increasing in size. Many simply persist at the more or less constant levels they reached after their initial increase following colonization. Slight changes in population density tend to be self-correcting, since increasing numbers slow reproduction and increase mortality and decreasing numbers do just the opposite. In such populations fitness is best characterized by

$R_0$ , the average number of daughters a female produces in her lifetime (Lande 1982; Charnov 1986). If  $l(x)$  is the probability that a newborn female is alive at time  $x$  and  $b(x)$  is the number of daughters she produces at time  $x$ , then

$$R_0 = \sum l(x) \cdot b(x) \quad (2)$$

If we assume that survival can be divided into juvenile and adult components, and that birth and death rates remain fairly constant throughout adulthood, then

$$R_0 = b(a) \cdot S(a)/M(a) \quad (3)$$

Here,  $a$  depicts the age of first reproduction,  $b$  represents a female's birth rate in terms of daughters per year,  $S$  represents the fraction of these daughters that survive to maturity, and  $M$  is the instantaneous adult mortality rate. All are functions of  $a$ , since delaying reproduction could potentially result in more robust, mature, and skilled adults that then could have higher than average birth rates while experiencing lower than average mortality for both themselves throughout their adult lives and for their offspring as they mature. This relationship, although simple, captures the important life-history trade-offs and reveals that the lifetime reproductive success of a female is essentially the product of the number of daughters a female produces during each of  $1/M$  years and the proportion of these that survive to reproduce.\*

Following Charnov (1990) it can be shown that there is an optimal age of first reproduction,  $a^*$ , and that it depends primarily on the magnitude of juvenile mortality and on the way in which changing the timing of first reproduction affects adult mortality and fecundity. If juvenile survival,  $S(a)$ , is composed of an early compo-

\*For  $R_0$  to be an appropriate fitness measure the population must not be growing ( $R_0 = 1$ ). Since the birthrate  $b(\cdot)$  and mortality functions  $S(\cdot)$  and  $M(\cdot)$  are as much functions of population density as they are of first reproduction ( $a$ ), it is important to determine how changes in density affect these functions and, in turn, if they affect the optimal age of maturity. Charnov (1990) has shown that as long as the negative affects of increasing density increase the mortality mostly on younger juveniles, or increase the mortality or decrease the fecundity uniformly for all aged adults, then density dependence is not likely to affect how natural selection affects the optimal age of first reproduction.

ment,  $S_0$ , that is fixed and depends on characteristics of the organism and ecological circumstances that are largely outside of its or the parents' control, and a later component,  $e^{-Q(a)}$ , where  $Q(a)$  is the amount of juvenile mortality between the early phase and maturation, then

$$R_0 = F(a) \cdot S_0 e^{-Q(a)} \quad (4a)$$

where lifetime fecundity is defined as  $F(a) = b(a)/M(a)$ . In this case, lifetime reproductive success is simply the product of the number of offspring born in a lifetime and the likelihood that each survives to independence. Or lifetime reproductive success can be defined as

$$R_0 = b(a) \cdot S_0 e^{-Q(a)}/M(a) \quad (4b)$$

if instantaneous adult mortality and fecundity remain explicitly defined. Maximizing  $R_0$  with respect to  $a$  is the same as maximizing  $\ln R_0$ , which yields

$$\frac{d \ln R_0 / da = d \ln F(a) / da - Q'(a)}{\quad} \quad (5a)$$

or

$$\frac{d \ln R_0 / da = d \ln b(a) / da - Q'(a) - d \ln M(a) / da}{\quad} \quad (5b)$$

Setting Equations (5a) or (5b) equal zero and solving reveals that lifetime reproductive success is maximized in the former case when, for a given change in the age of maturity, the rate of change in lifetime fecundity equals the rate of change in juvenile mortality (see Charnov 1990; Pagel & Harvey, Chapter 3, Fig. 3.1, this volume) and in the latter case when the rate of change in the instantaneous birth rate equals the sum of the rates of change in the juvenile and the logarithm of adult mortality rates. In the first case, this occurs when the difference between  $\ln F(a)$  and  $Q(a)$  is maximized and means that for any given lifetime fecundity curve, as the slope of the juvenile mortality curve increases, the optimal age of maturity decreases. In the second case, it means that the effect of adult mortality will be weaker, because it is logarithmically adjusted, than that of juvenile mortality. In both cases, however, determining the optimal age of maturity will depend on how  $b(\cdot)$ ,  $Q(\cdot)$ , and  $M(\cdot)$  change with  $a$ , the age of maturity. To do this, we need to specify some particular forms of these functions.

If we assume for simplicity that the instantaneous juvenile mortality rate either remains fairly constant during the juvenile period or represents the average of a rate that declines with age, then  $Q(a)$  can be represented by  $q \cdot a$  where  $q$  represents the constant instantaneous rate (after the high initial rate  $S_0$ ), or the average of the declining rate, for the juvenile period, and  $a$  represents the length of the juvenile period. Clearly, for any given level of juvenile mortality, the longer the juvenile period the lower the likelihood that a youngster will survive to maturity. What sets the level of  $q$ , however, is less clear. Perhaps as Janson and van Schaik (Chapter 5, this volume) propose, levels of predation or competition with adults play important roles: Juvenile mortality should be low for large-bodied species, or for those that live in relatively predator-free environments, or in habitats where resources can be acquired with little competition because they are abundant and evenly distributed.

If we also assume that the adult mortality rate is a function of the juvenile rate and that delaying the onset of reproduction can reduce the instantaneous rate of adult mortality, then it is reasonable to define the adult mortality rate as  $M(a) = qe^{-ma}$ , where  $m$  is the mortality exponent that describes how much a particular change in  $a$  will change  $M$ . At  $m = 0$ , the mortality rate of adults is unaffected by age at which maturity occurs and is that of juveniles. When  $m > 0$ , delaying puberty is accompanied by decreasing rates of adult mortality, but when  $m < 0$  the opposite occurs. As  $m$  increases, a given delay in the age of maturity will lead to an increasing difference between the juvenile and adult rates. If, in part, levels of adult mortality incorporate "costs of reproduction," then larger values of  $m$  reflect the fact that delaying maturity can lower these costs.

Finally, if we assume that mature parents are better breeders than younger, more naive ones, then birth rates should increase with increases in the age of maturity as  $b(a) = b_0 a^k$  where  $b_0$  is the characteristic birth rate of an average individual, and  $k$  represents the fecundity exponent that describes how much a given change in the age of maturity alters the characteristic, or average, fecundity. If  $k = 0$ , fecundity is unaffected by changes in the age of maturity, and since  $k$  is likely to be proportional to body size or experience, a  $k = 0$  implies that factors other than the

timing of maturity or certain features of the phenotype are shaping birth rate. When  $k > 0$ , however, the larger it gets, the more dramatic will be the effect of a given  $a$  on birth rate.

It should be noted that although these variable and exponents depict species-specific characteristics, they represent averages based on responses of individuals to their environment. And these responses are themselves likely to vary depending on features of personal or parental phenotype as well as social status. Typically large values of the exponents  $k$  and  $m$  indicate that, on average, fecundity and adult survival are extremely sensitive to changes in the age of maturity for a particular class of individuals. If the variation among individuals within a population is not too great and the values of these exponents are large, then negative values of the exponents are unlikely. However, when the values of the exponents lie close to zero, it would not be surprising to observe some individuals exhibiting those that are negative.

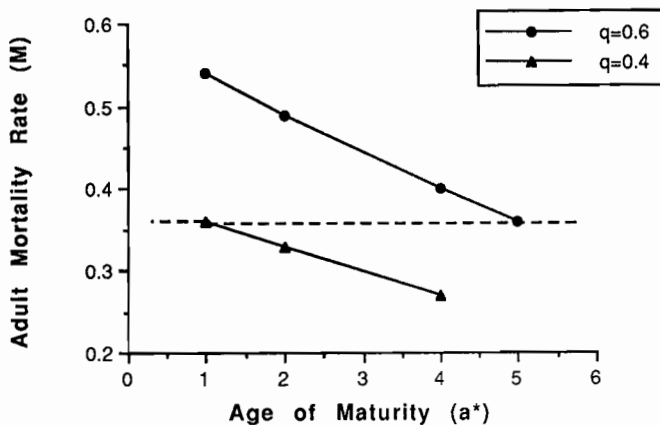
Substituting these functions into Equation (5b), differentiating them with respect to  $a$ , setting the equation equal to zero, and then solving for the optimal age of maturity yields

$$a^* = k/(q - m) \quad (6)$$

From this relationship it is clear that the optimal age of maturity increases as the impact that age

of maturity has on fecundity, as measured by the sensitivity exponent,  $k$ , increases. Even if one of the consequences of this sensitivity is disproportionately diminished reproduction for animals that begin breeding when very young, selection will favor delay as long as the delay ultimately has a marked effect on boosting fecundity. It is also clear from this relationship how the optimal age of maturity is affected by the sensitivity of adult mortality rate to the timing of maturity. Increases in the impact that age of maturity has on adult mortality, as measured by the sensitivity exponent  $m$ , increases the age of maturity as long as the rate of juvenile mortality,  $q$ , is held constant and  $m < q$ . Since increases in the sensitivity of adult mortality,  $m$ , result in lower rates of adult mortality, age of maturity ultimately increases as the rate of adult mortality decreases.

It is less clear, however, how the age of maturity is affected by changes solely in levels of juvenile mortality. At first glance, it appears that for a given  $m$ , any increase in the rate of juvenile mortality will lower the age of first reproduction. Holding  $m$  constant, however, does not hold the adult mortality rate constant since  $M(\cdot)$  is also a function of  $q$  (Fig. 4.1). In fact, for a constant  $m$ , increases in juvenile mortality are accompanied by increases in adult mortality. Thus increases in  $q$  represent increases in mortality over the entire lifetime, and, not surpris-



**Fig. 4.1.** Relationship between instantaneous rates of adult ( $M$ ) and juvenile mortality ( $q$ ). In these hypothetical relationships  $m = 0.1$  and  $k = 0.6$ . Note that although  $m$  is held constant for either level of juvenile mortality, the rate of adult mortality changes. The dashed line depicts a constant rate of adult mortality and shows that as the rate of juvenile mortality increases, so does the optimal age of first reproduction ( $a^*$ ). Conversely, for any level of juvenile mortality, increases in the rate of adult mortality lowers the optimal age of first reproduction.

ingly when this occurs, selection appears to favor accelerating the onset of reproduction. We can examine the sole affect that changing levels of juvenile mortality can have on the optimal age of maturity by dividing juvenile by adult mortality. As Figure 4.1 shows, increases solely in the rate of juvenile mortality tend to delay the onset of reproduction. Thus, overall, longer juvenile periods are favored when juvenile rates of mortality *relative* to those of adults increase, or adult fecundity is markedly enhanced by postponing the onset of sexual maturity.

The structure of the model is built on the premise that delays in maturity lower adult mortality rates, enhance adult fecundity, and lower the chances of juveniles surviving to adulthood, given a fixed juvenile survival rate. Thus it is not too surprising to find that when adult mortality is no different from juvenile mortality ( $m = 0$ ) increases in the levels of juvenile mortality select for shortening the period during which juveniles are at risk by lowering the age of first reproduction. Such an acceleration of puberty will be offset only when shortening the juvenile period markedly diminishes future fecundity. In fact, if maturity is reached at 2 years of age, for example, and is to remain unchanged in the face of environmentally induced age-dependent changes in mortality and birth rate, then an increase in the rate of juvenile mortality by 0.10 would have to be accompanied by a 0.20 increase in  $k$ . This would mean that the species-specific birth rate would have to be increased by 0.15 to offset a 0.10 increase in juvenile mortality. And for the age of maturity to remain unchanged at higher ages of maturity, the fecundity benefit would require an even larger value of  $k$  to counteract a given increase in a species' rate of juvenile mortality.

If the model is expanded to make the instantaneous juvenile mortality rate,  $q$ , a function of the age of maturity rather than a constant, then age of maturity need not automatically decline when  $q$  is increased in the absence of offsetting increases in fecundity. Such a function implies that the length of the juvenile period actually changes the prospects for juvenile survival either by the actions of the juveniles themselves or by those of their parents. The rate of juvenile mortality could be represented as  $q(a) = q_0/a^z$  where  $q_0$  represents a baseline level of juvenile mortality before the effects of delay and  $z$  is the experience exponent ( $z < 1$ ) that deter-

mines how strong the delay effects are. In turn,  $Q(a) = q_0 a^{1-z}$ . For the special case,  $m = 0$ ,

$$a^* = [k/q_0(1-z)]^{1/(1-z)} \quad (7)$$

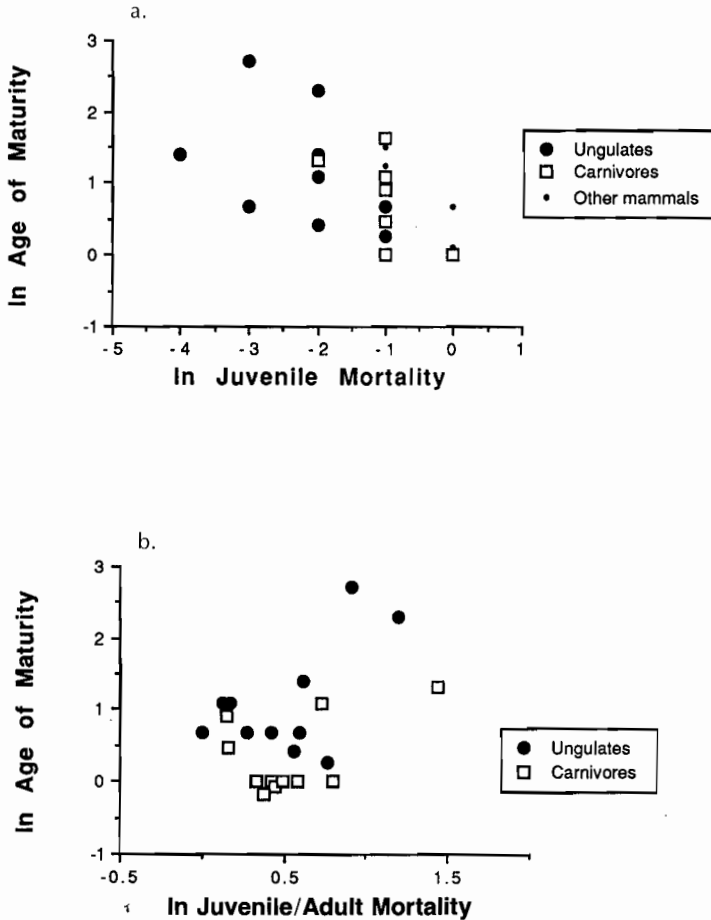
Clearly, when changing the age of maturity has no effect on the rate of juvenile mortality ( $z = 0$ ), this expanded expression for the optimal age of maturity reduces to the original form. But as  $z$  increases, the optimal age of first reproduction increases markedly. Thus even if environmental pressures increase the baseline level of juvenile mortality, as long as the experiential benefits of delay are sufficiently strong, no change in the optimal age of maturity need occur.

## THE PATTERNS

### Interspecific Comparisons

Using data compiled by Promislow and Harvey (1990) for a wide array of mammals, Harvey and Clutton-Brock (1985) and Ross (1988) for primates, and Bekoff et al. (1987) for carnivores, it is possible to test some of the predictions of the models. Figure 4.2a shows, as predicted, that as absolute rates of juvenile mortality increase, age of maturity decreases. But as the model also predicts, and Figure 4.2b shows, when juvenile mortality rates relative to those of adults increase, the opposite occurs [ $r^2 = 0.22$ ;  $F(1, 21) = 5.76$ ;  $p < 0.03$ ]. As with most life-history variables, age of maturity and rates of both juvenile and adult mortality scale allometrically with body size. Whereas age of maturity increases as body size increases (Fig. 4.3; coeff. = +0.22), similar increases in size are associated with lower rates of juvenile and adult mortality [coeff. = -0.24;  $F(1, 38) = 29.3$ ;  $p < .0001$ ]. Even after removing the effect of body size from each of these critical mortality variables, the overall patterns remain the same.\* Thus, regardless of body size, species

\*Since many aspects of life history scale with body size, body size must be removed from the analysis before the unconfounded relationship between the two life history variables can be measured. In this chapter two methods have been employed to do this depending on the form of the published data. In one, residuals from the regressions of each life history variable versus adult body weight were regressed against each other. If the slope of the regression equation was significantly different from zero the relationship between the relative, stan-



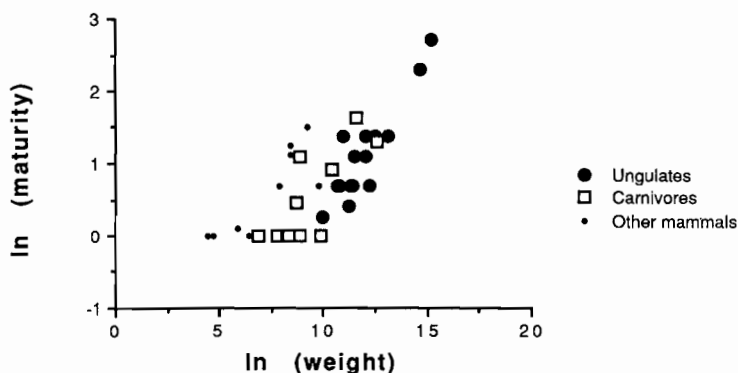
**Fig. 4.2.** Relationship between adult and juvenile mortality rates and age of first reproduction. (a) As the rate of juvenile mortality increases, the age of maturity decreases [ $\ln(a) = \ln(q) + 0.03$ ]. Although ungulates tend to have lower rates of juvenile mortality than carnivores, the relationship is the same. (b) When adult mortality rate is held constant, then as the rate of juvenile mortality increases so does the age of maturity [ $\ln(a/wt) = 0.80 \ln(q/wt/M/wt) - 9.4$ ]. Ungulates show a slight lengthening of puberty in relation to standardized levels of juvenile mortality relative to carnivores.

that have high levels of juvenile mortality tend to breed relatively early in life [ $F(1, 37) = 9.28$ ;  $p < 0.005$ ]. Only after the effect of adult mortality is removed is there a tendency for heightened

standardized, or weight-specific variables (as they are often referred to) were considered significant. In the other method, the ratio of each life history variable when divided by adult body weight was regressed against each other. Again, if the slope of the regression was significantly different from zero, the relationship between the two relative life history variables was considered significant.

levels of juvenile mortality to delay the onset of puberty [ $F(1, 37) = 3.1$ ;  $p < 0.04$  (one-tailed)].

Unfortunately, it is difficult to use interspecific data to examine directly the sensitivity of fecundity to changes in the age of first reproduction (values of  $k$ ). However, an indirect test can be made if we assume that for sexually dimorphic species where sexual competition among males is intense, small increases in size or experience will greatly increase a male's chance of not only breeding, but of acquiring a disproportionate share of mates. Then if in-

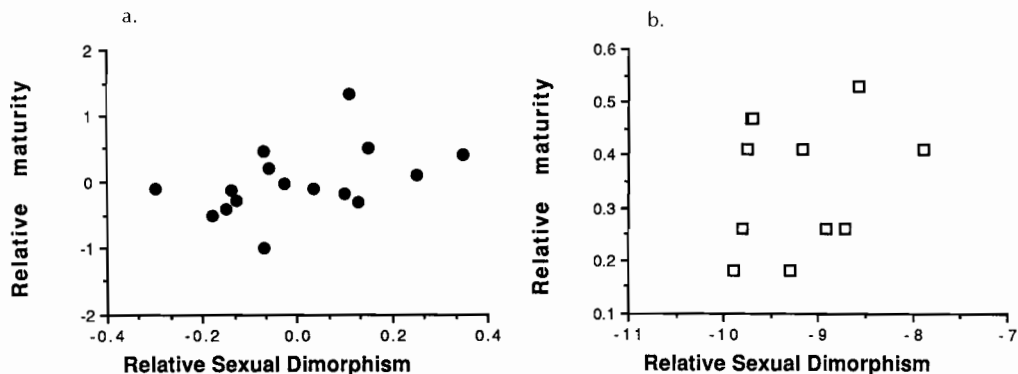


**Fig. 4.3.** Relationship between body size and age of first reproduction. The relationship is allometric, with large-bodied species having longer juvenile periods than smaller species [ $\ln(a) = 0.22 \ln(wt) - 1.4$ ]. On a weight-specific basis, however, the length of the juvenile period is relatively shorter for larger species.

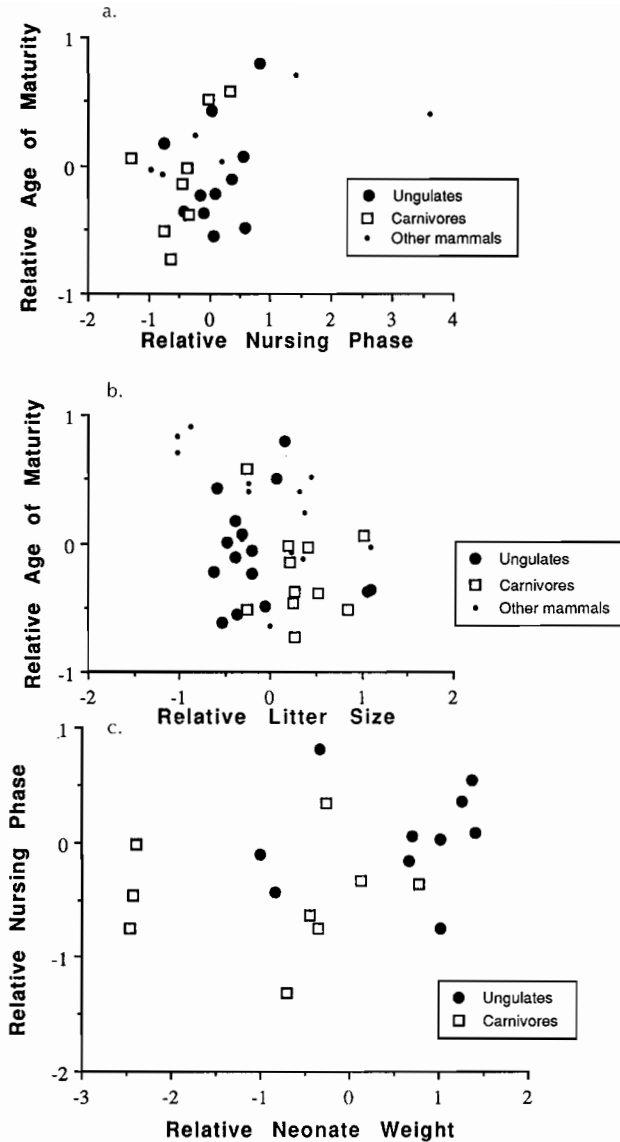
creases in size can be attributed to the lengthening of the juvenile period, more sexually dimorphic species should show stronger sensitivities of age of maturity on fecundity, and hence have higher  $k$  values, than less sexually dimorphic species. And since the optimal age of maturity increases as values of  $k$  increase, the optimal age of maturity for males should also increase as the degree of sexual dimorphism increases across species. As Figure 4.4 shows, this is the case for both primates [ $F(1, 14) = 3.45$ ;  $p = 0.04$  (one-tailed)] and carnivores

[ $F(1, 7) = 3.94$ ;  $p = 0.05$  (one-tailed)]. When size is removed from the analysis, species in which males are relatively larger than females have relatively delayed puberties.

Some other life-history features that are related to reproduction and help shape species-specific patterns of fecundity appear also to covary with age of maturity. As Figure 4.5 shows, species with relatively long nursing periods and relatively small litters have relatively delayed ages at maturity, whereas those with relatively heavy neonates or relatively heavy litters do not. But if



**Fig. 4.4.** Relationship between relative sexual size dimorphism and relative age of maturity. (a) Species of primates in which males are larger than females have relatively longer juvenile periods than more monomorphic species [ $\ln(y) = 1.4 \ln(x)$ ]. Relative maturity is measured as the deviations, or residuals, of the relationship between  $\ln$  Age of Maturity and  $\ln$  Female Body Weight. (b) The same relationship holds for carnivores. As the degree of sexual dimorphism increases, so does the age of first reproduction. Relative age of maturity is measured as the  $\ln$  of the ratio of age at maturity divided by female body weight and relative size dimorphism is measured as the  $\ln$  of the ratio of male to female body weight [ $\ln(y) = 3.05 \ln(x) - 9.98$ ].



**Fig. 4.5.** Relationship between the relative length of the juvenile period and the relative value of various life-history traits. (a) The relationship between the relative age of maturity and the relative length of the dependent phase, or nursing period, is positive for both carnivores and ungulates [ $\ln(y) = 0.20 \ln(x)$ ;  $F(1, 26) = 6.03$ ;  $p < 0.05$ ]. Thus species with relatively long nursing periods for their body size also have relatively long delays before beginning to reproduce. (b) For species with relatively larger litters, maturity is reached at relatively early age [ $\ln(y) = -0.29 \ln(x) + 0.005$ ;  $F(1, 37) = 5.5$ ;  $p < 0.03$ ]. (c) The relative size of the neonate explains little of the variation in the weight-specific, or relative length of the juvenile period [ $F(1, 23) = 0.22$ ;  $p < 1.0$ ]. Not depicted is the nonsignificant relationship between relative litter weight and relative age of first reproduction [ $F(1, 33) = 0.32$ ;  $p < 0.6$ ]. Relative values are derived from the residuals of the regression of each life-history variable and the  $\ln$  of female body size.

we subtract the length of the nursing period from the time it takes to reach maturity and call this remaining time the "independent juvenile phase," then a somewhat surprising trend emerges. Although it is true that larger species, which have longer nursing periods [ $F(1, 26) = 11.2$ ;  $p < 0.005$ ; coeff. =  $+0.24$ ] and heavier [ $F(1, 37) = 109.7$ ;  $p < 0.0001$ ; coeff. =  $+1.01$ ] but fewer young [ $F(1, 33) = 196.1$ ;  $p < 0.0001$ ; coeff. =  $-0.18$ ], have longer independent juvenile phases [ $F(1, 20) = 30.0$ ;  $p < 0.0001$ ; coeff. =  $+0.20$ ] than smaller species, neither of these *relative*, or weight-specific, life-history variables covaries significantly with the *relative* length of this independent period. In fact, not even rates of juvenile mortality relative to those of adult mortality account for any significant variation in the relative length of the independent juvenile period [ $F(1, 15) = 1.91$ ;  $p < 0.20$ ].

When we examine exactly which species exhibit relatively long independent juvenile periods after controlling for body size, no obvious pattern emerges. The five species with the longest relative juvenile periods are elephant (*Loxodonta africana*), brown bear (*Ursus americanus*), river otter (*Lutra canadensis*), Japanese macaque (*Macaca mulatta*), and mountain goat (*Ovis canadensis*). They represent five different families, range widely in body size, and have markedly different foraging styles and ecological requirements. And although these five species are polygynous, over the entire sample there is no statistically significant relationship between the magnitude of sexual selection, as measured by degree of sexual size dimorphism, and the length of the independent juvenile period [ $F(1, 15) = 1.91$ ;  $p < 0.20$ ]. Among primate species, for example, those in which males are relatively larger than females display relative independent juvenile periods that are somewhat longer than more monomorphic species. The pattern, however, is not statistically significant, as only 0.12 of the variation in the relative length of the juvenile period is explained by the degree of sexual size dimorphism.

These patterns suggest that life-history traits that are physiologically tied to features of reproduction are closely tied to the age at which breeding commences, but not to the length of time that youngsters spend developing after ceasing to rely solely on mother's milk for nour-

ishment. Moreover, even juvenile mortality, the factor most important in determining when adulthood begins has apparently little influence on determining how long the independent phase of the juvenile period should last. It is interesting to note that the dependent (nursing) phase tends to comprise about  $0.16 \pm 0.02$  of the total juvenile period (0.11 for ungulates, 0.21 for carnivores, 0.27 for female primates, and 0.19 for male primates). And although each of these juvenile phases scales allometrically with body size [independent phase: [ $F(1, 25) = 30.0$ ;  $p < 0.0001$ ] and the length of each is positively correlated with the other ( $r^2 = 0.71$ ;  $p < 0.001$ ;  $n = 24$ ), the proportion of either with respect to the whole does not scale with body size [proportion of dependent phase:  $F(1, 18) = 0.97$ ;  $p < 0.40$ ]. In fact, there is only a weak and statistically insignificant positive relationship between the *relative*, or weight-specific, lengths of the dependent and independent phases of the juvenile period [ $F(1, 22) = 3.1$ ;  $p < 0.10$ ]. This lack of a relationship might account for the finding that *relative* length of the independent juvenile period does not covary with relative age of maturity or any other life-history variable, whereas the length of the *relative* dependent period does.

That deviations from the shorter of the two juvenile phases should be most important in accounting for deviations in the onset of sexual maturity is intriguing and suggests that physical aspects of infantile maturation are of primary importance in timing the decision to begin reproducing. This also implies that variation in the independent juvenile phase is much less important in initiating this critical life-history transition to adulthood and raises questions about its ultimate functional significance. Is it really the critical period during which a superior adult is built? Or is it just a period during which a surviving juvenile is made more competent as a juvenile?

### Intraspecific Comparisons

To answer these questions and fully appreciate how juvenility is related to subsequent reproductive success, variation within single species must be examined. It has long been appreciated in mammals that reproductive success increases with increasing parity and age, at least until senescence sets in. In moose (*Alces alces*)

(Saether & Haagenrud 1983), gazelles (*Gazella cuvieri*, *G. dorcas*, and *G. dama*) (Alados & Escos 1991), white-footed mice (*Peromyscus leucopus*) (Fleming & Rausher 1978), and red fox (*Vulpes vulpes*) (Allen 1984), older females tend to produce larger litters. Larger young are produced by heavier mothers in white-footed mice (Myers & Masters 1983), and moose (Saether & Haagenrud 1983), by older mothers in the gazelles (Alados & Escos 1991) and by older, heavier and more dominant females in red deer (*Cervus elaphus*) (Clutton-Brock et al. 1986). And larger young, or those born to older females, tend to have higher survival prospects in each of these species. Prenatal mortality tends to decline in older red fox females (Allen 1984), and adult prospects for survival or breeding in a second consecutive year increase as female condition improves in red deer (Clutton-Brock et al. 1983). Exactly why these age effects occur is unclear, but presumably they are associated with the fact that older individuals are more experienced, are larger, or are in better physiological condition.

With maturity apparently comes enhanced reproductive efficiency. But is the improvement primarily due to benefits gained during the juvenile period, and, if so, is it primarily the result of the juvenile period being lengthened? Unfortunately, answering these questions is difficult because few studies have separated the generalized effects of aging on reproduction from the specific effects of changing the age at which reproduction first begins. A few studies, however, have drawn this distinction and are reviewed below.

**Rodents.** In the Mongolian gerbil (*Meriones unguiculatus*), age of maturity is bimodally distributed. Whereas some females become receptive just prior to weaning, others delay the onset of reproduction until after they have gained independence from their mothers. Clark et al. (1986) have shown that the early-maturing subset of the population matures on average about 20 days earlier and about 20 g lighter than the late-maturing subset. Although first litters born to early-maturing females are larger than those born to late-maturing females, but not significantly so, early-born first litters contain significantly more females and early-maturing daughters than those born to late-maturing females. Over their lifetime, early-maturing females produce significantly more litters and

slightly more than twice as many surviving young than later-maturing females. In part, this is because they begin breeding early. But it is also because early-maturing females continue breeding to later ages, even though late-maturing females live slightly longer. Early-maturing females also invest much less time suckling their pups and are less solicitous in caring for their pups than late-maturing females. That early-maturing females become receptive at lower weights shows that they do not grow faster than late-maturing females and suggests that sexual maturation is independent of morphological development.

Studies on the mechanisms of puberty control in other rodents reveal that variations in timing the onset of sexual maturity as exhibited by the Mongolian gerbils is common in rodent populations and could produce bimodal distributions of age at maturity in strongly seasonal environments. Many chemical compounds secreted by rodents and primates are known to serve as signals. Some attract males to females (Keverne 1976; O'Connell et al. 1981), induce males to mount estrus females (Singer et al. 1980), or advertise reproductive and social status (Preti et al. 1976). Others play a role in priming sexual behavior. Perhaps the best known are those that block pregnancy when a recently mated female detects the scent of a strange reproductively capable male (Bruce 1959) or those that cause dense populations of females to remain anestrous until exposed to a sexually active male (Whitten 1959). More recently, however, Vandenberg and Coppola (1986) have shown that chemical compounds of mice can enhance or retard the onset of female puberty. Juvenile females housed with mature males, or to a lesser extent those exposed to only male scents, reach puberty about 20 days before females reared in isolation or in all female groups. This effect occurs in both laboratory colonies and natural populations and seems to be mediated via androgen levels, since castrated males fail to accelerate the onset of puberty and low-ranking males induce a weaker effect than do dominants. Since exposure to lactating and pregnant females also accelerates puberty, but only by 4–5 days (Dricamer & Hoover 1979), factors other than androgens must be involved. Normally, however, female mice reared in the presence of other nonpregnant or nonlactating females exhibit delays in the onset of sexual maturity (Vandenberg et

al. 1972). Clearly, male and female pheromones can interact, sometimes in opposite ways. When they act simultaneously, Drickamer (1982) has shown that the effect of female pheromones on *prepubertal* females takes precedence over those of males. That these effects also occur in other rodents and primates suggests that the phenomenon of puberty modulation is widespread (Vandenbergh & Coppola 1986).

These studies on both house mice and Mongolian gerbils show that age of maturity can change as social conditions change. But why might flexibility in timing the onset of maturity be adaptive? In other words, what about the environment could be maintaining maturational polymorphisms in the population. Delaying the onset of reproduction has traditionally been viewed as a way in which females can increase their survival prospects when the chances of successfully rearing young are already low (Vandenbergh & Cappola 1986). It is thought that by postponing breeding when environmental conditions are harsh, females can wait in an energetically favorable state for conditions to improve or for dispersal opportunities to arise. However, given that the life expectancy of an adult feral house mouse, for example, is about 1 month, the opportunity costs of delaying would appear to be excessively high. Moreover, given that the likelihood of the environment changing during a delay of only 5–10 days is so small, it seems unlikely that a delay in the onset of breeding would be the result of selection to reduce *adult* mortality. It is much more likely that delayed puberty would be favored by selection because it would increase prospects for *juvenile* survival. That late-maturing Mongolian gerbil females, those that would experience the most crowded and resource depauperate conditions in the wild, do in fact care for their young more vigorously suggests that selection may be operating in this way. Thus it appears that flexibility in timing the onset of reproduction is favored when environments fluctuate and that delay will be favored when longer juvenile periods actually lower juvenile mortality rates.

For rodents in general and Mongolian gerbils in particular, if delaying puberty occurs under harsh conditions and in accord with the predictions of the models, then either late-maturing females should show greater sensitivity to the affects of  $a$  on fecundity or adult mortality (large values of  $k$  and  $m$ ), or (and perhaps more impor-

tantly given the above adaptationist explanation) the offspring of late-maturing females should experience lower levels of juvenile mortality than those of early-maturing females. For the latter to be the case,  $z > 0$  so that  $q(\cdot)$  can be lower for late as opposed to early-maturing females.

From the study by Clark and her co-workers (1986) there is enough intraspecific variation in the life-history data to estimate values of the sensitivity exponents. First, the litter sizes of late-maturing females do not increase as a result of the delay; hence,  $k$  will be very small. Second, given that early-maturing females live only slightly shorter lives than delaying females,  $m$  will be small, if not zero. Since early- and late-maturing classes of females do not differ significantly with respect to these two parameters, according to the models neither factor will have much influence in determining the optimal age of maturity. With respect to juvenile mortality, however, small differences exist, and because they are correlated with significant differences in parenting behavior, they appear to be able to account for the puberty delay that late-breeding females exhibit. Given that  $q(\cdot)$  is quite large, varying from about 0.02/day for the early-maturing females to about 0.01/day for the late-maturing ones, it is not surprising that Mongolian gerbils exhibit short juvenile periods. But more importantly, the slightly smaller  $q(\cdot)$  of late-maturing females is sufficient to account for the delay in their maturity that accompanies breeding during the winter. Under harsh winter conditions, increases in interbirth interval, shifts to the production of more sons than daughters, and devotion of more time to suckling and caring for the young appear to reduce the rate of juvenile mortality, all of which would make  $z > 0$ . According to the models, in a population where size is invariant and adult mortality is similar for both classes of adult females, a reduction solely in juvenile mortality rate such as this should lead to increases in the optimal age of maturity. This is what is seen under these seminatural experimental conditions. If the life histories of Mongolian gerbils and the physiological mechanisms that underlie them are representative of most rodents, then we see that the setting of the age of first reproduction for these kinds of small mammals follows the simple biological trade-offs contained in the models.

*Ungulates.* Some of the best data on mammalian life-history patterns come from long-term studies on ungulates and show that timing the onset of reproduction does have profound effects on fecundity and rates of mortality.

Clutton-Brock and his co-workers have followed the fates of many cohorts of red deer (*Cervus elaphus*) inhabiting Rhum, an island off the west coast of Scotland. Dominance rank is the most important factor affecting lifetime reproductive success of females. High-ranking females breed sooner (3.3 vs. 3.6 years), produce calves at a faster rate (0.8/year vs. 0.7/year), have a greater proportion of them survive to adulthood (0.8 vs. 0.7), and live longer (13 vs. 12 years) than their more subordinate counterparts (Clutton-Brock et al. 1986). Overall, high-ranking females rear more offspring to 1 year of age (6.2 vs. 4.4) over a lifetime than do low-ranking females (Clutton-Brock et al. 1986). Dominance is affected by female body weight, and this is a function of a female's own birth weight, which in turn is influenced by spring temperatures. High temperatures seem to increase fetal growth rates by increasing the amount of available vegetation (Albon et al. 1987).

Although Clutton-Brock and his co-workers have not explicitly determined the effect that altering the age of first reproduction has on either initial or lifetime reproductive success, they have shown that differences in early development have major effects on female status as adults. Even after controlling for differences in birth weight, daughters that are weaned relatively early for their weight attain significantly lower adult ranks than those that are nursed longer (Clutton-Brock et al. 1986). Thus, delaying the age of independence significantly enhances a female's dominance rank and ultimately her reproductive opportunities.

In terms of the models, the red deer is a species in which values of  $k$  and  $z$  will be high, since small delays in puberty enhance a female's dominance and her ability to increase her fecundity as well as the survival rates of her offspring. As a result, the red deer should be a species that exhibit relatively long juvenile periods, and they do (red deer lie above the regression for ungulates shown in Fig. 4.3). Also, given that dominance differences among females affect the survival prospects of their newborn and yearlings more than their daughters (Clutton-Brock

et al. 1986), then in terms of the models  $q(\cdot)$  should be lower and  $z$  should be greater for males than for females. As a result, maturity for males should be reached later than for females. This is indeed the case. Females mature on average at 4 years of age, whereas males often begin breeding as late as 6 years of age.

The age of first reproduction can have dramatic consequences on lifetime reproductive success in feral horses (*Equus caballus*) as well. On a barrier island off the east coast of North America, a population of horses has lived free from human demographic interference for over 100 years. From 1973 to 1986, the population has remained fairly constant, consisting of about 100 animals (Rubenstein 1986). During that time, the reproductive fates of over 70 females and their daughters have been followed. Only a subset of 18, however, has survived to breeding age, then another 3 years, and has lived during a period when population size was stable. As Table 4.1 shows, for this subset of females the onset of reproduction varies, but most females ( $n = 9$ ) reach puberty at 4 years of age, 2 years later than do the youngest females. The timing of maturity has a strong effect on the prospects of survival to 1 year of age of foals conceived at the first breeding attempt [Table 4.1;  $F(2, 14) = 6.8; p < 0.01$ ]. For females, that delay until at least 4 years of age, survival of their young to 1 year of age is 0.89, whereas for those that breed for the first time at 2 years of age, none of their young survives.

When subsequent reproductive episodes—those involving young born during the first, second, and third years after initiating reproduction—are also considered, the significant survival differences of foals born to mothers initiating reproduction at different ages diminish, but remain statistically significant [Table 4.1;  $F(2, 14) = 5.6; p < 0.01$ ]. The survival rate of foals born during a 3-year period subsequent to females initiating breeding at age 2 increases from zero to 0.20, whereas the survival of foals born during the same 3-year period to females waiting until at least age 4 to breed for the first time decreases from 0.89 to 0.80. This survival rate is only slightly greater than the 0.70 for those born during the 3-year period to females beginning to reproduce at age 3. When yearly per capita reproductive success is compared, these same small but statistically significant differences are maintained [Table 4.1;

**Table 4.1.** Different Ages of First Reproduction and Their Effects on Various Measures of Reproductive Success and Survival for Feral Horses Inhabiting Shackleford Banks, N.C.

	Age to maturity (years)				<i>F</i> value	<i>p</i>
	2	3	4	5		
Number of females bearing young during 1 year	3	5	9	1	6.8	< 0.01
Number of young surviving to 1 year of age	0	2	8	1		
Proportion surviving to 1 year of age	0	0.40 (0.24)	0.89 (0.11)	1.0		
Number of young born during 3 years	5	10	19	2	5.6	< 0.01
Per capita yearly birth rate	0.56	0.67	0.70	0.67		
Number of young surviving to 1 year of age	1	7	15	2		
Proportion surviving	0.20 (0.17)	0.70 (0.12)	0.80 (0.08)	1.0		
Per capita reproductive success per year	0.11 (0.09)	0.47 (0.08)	0.56 (0.08)	0.67		
Number of expected breeding opportunities	11	10	9	8	7.3	< 0.01
Expected lifetime reproductive success	1.2	4.7	5.0	5.4		
Number of original females dying within 6 years after beginning to breed	2	2	1	0		
Mean age at death	6.0	7.5	10.0			

Note: Analysis of variance performed only on females beginning to breed at ages 2, 3, and 4. Sample sizes are based on the number of breeding females in the cohort. Standard deviations are in parentheses below the means.

$F(2, 14) = 7.3; p < 0.01$ ]. Given that females live on average for 12 years, we can estimate the expected lifetime per capita reproductive success of females that initiate reproduction at each of these different ages, live to age 12, but lose a breeding episode for every year they delay (Table 4.1). Although the expected lifetime reproductive success of females breeding at very young ages is markedly different from those that delay, the difference between those that delay until 3 years of age (4.7) and those that delay until 4 or greater (5.0) is much smaller.

Delayed breeding enhances expected lifetime reproductive success because postponing puberty beyond the point where females are physiologically able to reproduce lowers juvenile mortality. In terms of the models, whenever  $z > 0$ ,  $q(\cdot)$  declines and so does the optimal age of maturity. But does delay also have an effect on fecundity and adult mortality? With respect to fecundity, Table 4.1 shows that yearly per capita birth rate varies little, ranging from 0.56 for females beginning to breed at age 2 to 0.70 for those delaying until age 4. In terms of the model, the fecundity sensitivity exponent,  $k$ , will be positive but small. If we examine the survivorship of females that actually begin breeding at different ages, we see that 6 years

after the onset of reproduction those that start early have a lower probability of surviving to the end of the 6-year interval than those that delay (Table 4.1). Whereas only 0.1 of those delaying breeding for the first time until at least age 4 perish, of those initiating reproduction at age 2, 0.67 die. As a consequence, female age of death decreases as the age of first reproduction decreases (Table 4.1). In terms of the model adult mortality  $M(\cdot)$  declines with increases in the age of maturity, suggesting that  $m > 0$ . Thus for horses, each of the critical variables is affected by the timing of maturity and in ways that suggest that as a species, the optimal age of reproduction should be delayed well past the physiological age when reproduction becomes possible. Most females do in fact appear to delay 2 years past the first possible date, yet approximately 0.45 of the females breed before this modal age. Why should this variation be maintained if the benefits of delay can be seen in each of the three critical life-history dimensions?

Acceleration of puberty could be favored by natural selection as long as the sensitivity of some females to the effects of age of maturity on fecundity or either juvenile or adult mortality is small. For this to be the case, the exponents  $k$ ,

**Table 4.2.** Number of Females Beginning to Breed Early or Late Depending on Their Dominance Rank

Female dominance	Age of first reproduction (years)	
	2 or 3	4 or 5
High	5	5
Low	3	5

Note:  $\chi^2 = 0.28$ ;  $p < 0.60$ .

$m$ , and  $z$  in the models would have to be close to zero for this class of females, and possibly even negative in species where the average values were not large to begin with. Such small values would tend to accelerate their optimal age of maturity. At least for Shackleford horses, dominant females or females that reside in harems with dominant males tend to exhibit reduced sensitivities of adult mortality to changes in the timing of age of maturity. Although dominant and subordinate females show no significant differences in their propensity to breed early or late in life (Table 4.2;  $\chi^2 = 0.28$ ;  $p < 0.6$ ), four of the five of the females dying within 6 years after beginning to breed are early breeders ( $\leq 3$  years of age) and, of these, three are subordinates. This suggests that the mortality of females of high rank is less affected by the timing of the initial bout of reproduction than that of females of low rank.

In addition, the rank of the male with which a female associates also appears to be an important determinant in timing the initiating reproduction. Table 4.3 shows that significantly more females that begin breeding at ages 2 or 3 live in groups tended by dominant males versus ones tended by subordinates [ $\chi^2 = 2.81$ ;  $p < 0.05$  (one-tailed)]. And here, too, rank of the male influences the sensitivity of adult female mortality to changes in the age of first reproduction. Whereas five of seven (0.71) females of dominant males survive if they breed

**Table 4.3.** Number of Females Beginning to Breed Early or Late Depending on the Rank of the Male with Whom They Associate

Male dominance	Age of first reproduction (years)	
	2 or 3	4 or 5
High	7	5
Low	1	5

Note:  $\chi^2 = 2.81$ ;  $p < 0.05$  (one-tailed).

at 2 or 3 years of age, the one female associating with a subordinate male does not. Yet for the 10 females that delay breeding, all those with dominant males survive the interval ( $n = 5$ ), and 0.6 of the five females with subordinate males also do so.

Clearly, there is a cost to breeding early, but it is much less for those females that are dominant or associate with dominant males. Why should this be so? Since dominant females have more access to limited water supplies than subordinate females (Rubenstein 1993) and those associating with dominant males are harassed less and have more time to graze (Rubenstein 1986), it appears that favorable ecological circumstances can lower the costs of reproduction so that adult mortality rates become unaffected by changes in timing the onset of sexual maturity. For some females, the removal of critical life-history traits from an ordinarily strong maturational sensitivity means that selection no longer opposes their taking advantage of opportunities to begin reproducing early in life because reproduction has become virtually cost-free. Offspring of dominant mothers or of those associating with dominant males derive material benefits, while their mothers avoid costs that are typically harmful when ecological resources are more limiting. At least in this horse population, subordinate females and those associating with low-ranking males show greater sensitivities to changes in age of first reproduction than do dominants, or than those bonded to dominant males. For these subordinate females, selection will favor delaying the onset of reproduction. For those experiencing more favorable ecological circumstances, selection should favor an earlier than average age of first reproduction. In this feral horse population, both tendencies occur and in the ways predicted by the models.

**Pinnipeds.** Lifetime reproductive success has been collected on northern elephant seals (*Mirounga angustirostris*), and although there are strong age effects on breeding and rearing success, sex differences are large and the results of timing the onset of reproduction on these life-history attributes are equivocal. LeBoeuf and Reiter (1988) show that breeding begins in males at age 6, on average, and yearly reproductive output increases until age 11, dropping to zero by age 13. For females, they show the pattern to be different. Although breeding can be

gin at 2 years of age, most females wait until ages 3 or 4, with only a few stragglers delaying until 5 or even 6 years of age. Four- and 5-year-old females are the most prolific breeders, but those between the ages of 4 and 7 wean the most pups. Unlike males, however, female fecundity varies little with age after a female has given birth for the first time.

Only for females does there appear to be a strong relationship between the age at which reproduction is initiated and a variety of factors that affect lifetime reproductive success. Both pup survival to 1 year of age and adult survivorship increases as females delay the onset of breeding (Reiter & LeBoeuf 1991). These relationships result from the fact that although the weight of pups at birth is greater for females breeding for the first time at age 3 as opposed to age 4, the ability of mothers to invest in pups without reducing their own prospects of survival increases as mothers grow older and increase their reserves of blubber. By projecting the growth rate of hypothetical populations composed either all of females commencing breeding at age 3 or all of females beginning at age 4, Reiter and LeBoeuf show that the strategy of delay to age 4 is the superior, since it spreads faster under high-density conditions, irrespective of the level of juvenile survival (40 vs. 80%). It loses its advantage, becoming equivalent to the early-breeding strategy, only when density is low and juvenile survival is high. The authors suggest that the early-breeding strategy is maintained in the population because neighboring populations typically exhibit population cycles that are out of phase, and in recently colonized and expanding populations selection allows early breeders to do well, although in crowded, or declining, ones late breeders have the advantage. As with the red deer and the Shackleford horses, there are ecological conditions where the sensitivity of the life history components, in this case  $m$  and  $z$ , to changes in age of maturity is reduced and with the lowering of the costs of breeding early, female puberty can be accelerated as predicted by the models.

For males, however, there are no significant relationships between the age of first reproduction and the number of young sired in a lifetime [ $F(1, 6) = 1.8; p < 0.7$ ] or the number of these that are weaned [ $F(1, 6) = 1.36; p < 0.7$ ]. Although about 0.12 of the variation in yearly rate of reproductive success is explained by the age

of a male's first breeding attempt, the tendency for males to sire more young per year if they delay the onset of breeding is not statistically significant [ $F(1, 6) = 1.34; p < 0.3$ ]. In this small sample, the male initiating breeding at the youngest age breeds for the longest period, has the highest lifetime reproductive success, and, with respect to yearly offspring production, comes in second to a male delaying breeding until age 8. In terms of the model, the sensitivity exponents should be small and hence breeding should occur early. But empirically this is not the case; on average, males delay breeding 2 more years past the time when females typically begin breeding (LeBoeuf & Reiter 1988). Since dominance and experience play the major role in determining male breeding success (LeBoeuf & Reiter 1988), it appears that knowledge and skills gained once reproduction commences is more important than acquiring these attributes while growing. Perhaps the apparent limited importance of the juvenile period derives from the fact that breeding takes place on land, whereas most of the juvenile period, and its effects on the sensitivity parameters, occurs in the sea.

**Primates.** Perhaps more attention has been focussed on the juvenile lives of primates than on any other group of mammals. Detailed studies range from the physiological to the social and to the reproductive consequences of ontogeny, and they have been performed on laboratory as well as on wild populations.

Baboons (*Papio cynocephalus*) reveal a common pattern in which dominance rank has a profound affect on female reproduction (Altmann et al. 1988). Daughters born to mothers of high rank tend to have accelerated menarche and conceive about 200 days earlier than those born to low-ranking mothers. Over a female's lifetime, this acceleration amounts to an increase of 0.5 infants. Although age affects fertility up to about 6 years of age, the survival prospects of offspring after the first breeding attempt remain constant throughout life. Moreover, the age of first conception has no affect on the survival chances of the first born young. Since 0.79 of the variance in lifetime reproductive success is accounted for by life span and no ecological factors seem to strongly affect it (Altmann et al. 1988), it appears that breeding early is the key to enhancing a female's reproductive success in yellow baboons.

For other primate species, dominance seems to operate in ways similar to the baboons, accelerating puberty and hence enhancing lifetime reproductive success. Drickamer (1974) has shown for rhesus macaques that daughters of high-ranking females breed earlier than those of lower rank, and Gouzoules et al. (1982) have shown for Japanese macaques that age of maturity increases as rank decreases. Yet for other species, age of maturity seems little affected by social or ecological factors, such as for bonnet macaques where all females give birth at the same age (Silk et al. 1981), even though timing of puberty often still influences female lifetime reproductive success. In vervet monkeys (*Cercopithecus aethiops*), for example, age and dominance rank have little effect on female lifetime reproductive success (Cheney et al. 1988). Birth rate does not decline with age, and primiparous females have the same number of surviving offspring as do multiparous ones. Similarly, dominants do not produce more young than do subordinates, nor do their young have higher probabilities of survival. Dominants also do not reproduce at an earlier age than subordinates, nor do they have shorter interbirth intervals. The major determinants of differences among females in lifetime reproductive success appear to be juvenile survival prospects and adult longevity. And juvenile mortality rate, in particular, is correlated with age of first reproduction. For those females delaying reproduction the most, survival to adulthood is least. In troops where reproduction is delayed past five (5.7 and 5.1 years), the chances of infants dying before reaching maturity is on average 0.63 and 0.71, respectively. In the troop where reproduction commences on average at 4.4 years of age, juvenile survivorship is 0.53. Coupled with these changes in rates of juvenile mortality are changes in adult mortality. For troops delaying puberty past 5 years of age, adult yearly mortality is higher (0.17 and 0.22) than it is for females in the troop breeding at the earlier age (0.10).

Although these results are derived from a small number of troops and are not statistically significant, the overall pattern indicates that there is a connection between ecology, as measured in terms of resource availability, and timing the onset of reproductive maturity. Since those troops with the earliest ages of first reproduction have the lowest levels of mortality,

selection apparently favors early reproduction when possible. This suggests that "those that can, do; while those that can't don't," and the "dos" and "don'ts" are determined by the severity of ecological circumstances. Apparently, daughters being raised under harsh conditions have extended juvenile periods and suffer high levels of juvenile mortality. The delay seems to be nonadaptive, since it has little effect in reducing subsequent levels of adult mortality. Since daughters with access to resources are able to accelerate puberty without incurring significant survival costs, selection at least in vervet monkeys is reinforcing traits that enhance competition, ultimately maintaining variation in the timing of puberty.

The tendency for ecological circumstance to influence the onset of reproduction is also seen in at least one group of humans (*Homo sapiens*). In her long-term study on the Kipsigis, an agropastoralist people of southern Kenya, Borgerhoff-Mulder (1988) shows that men marry at around 18 years of age and, if wealthy enough, take additional wives later in life. She also demonstrates that women marry at about 16 years of age after a period of seclusion during which they are fattened. About 0.76 of babies survive to reproductive age, and whereas men in this polygynous society father about 12 offspring in a lifetime, each woman bears about 6. For men, wealth, as measured in terms of the size of herds and plots of land, is the major determinant of reproductive output. Wealthy men marry younger and have more offspring, and more of their sons marry polygynously than do the sons of poorer individuals. For women, wealth is also important but for a different reason: reproductive success is influenced more by the likelihood of children surviving to adulthood than by the length of the reproductive period, even though younger brides have on average about 2.7 more surviving offspring than do their older counterparts. It appears that young brides are better nourished during their early development and simply become better mothers.

Thus, at least for primates, there is one clear generalization about how selection alters the length of the juvenile period. When females are capable of breeding at a young age, their lifetime reproductive success is enhanced because their fertility remains high, while either their own or an offspring's chances of survival also remain high. In yellow baboons and several spe-

cies of macaques, a mother's rank seems to separate those that have access to resources and can begin breeding early without incurring survival costs from those that lack access and cannot. In vervet monkeys, neither age nor dominance affects birth rate or survival, but ecological circumstances, as determined by group and home-range residence, seems to separate the "haves" from the "have-nots." And for at least one group of humans, early breeding also seems to enhance the survival prospects of offspring, despite the fact that adolescents typically show depressed fertility (Jain 1969) and a physiological susceptibility for fetal or infant loss (Leridon 1977; Miller & Stokes 1985). For the Kipsigis, those females who can breed early seem to be superior and, as a result, seem to be able to overcome these inherent physiological problems, thus creating a long reproductive lifetime during which they produce many robust offspring. Although the mechanistic details are less well known for the other primates, the pattern exhibited by human females seems likely to be representative. In terms of the models, all these examples have one feature in common: when sensitivities of fertility and survival to age of maturity are reduced (exponents close to zero) for some classes of females, those females should and do show early ages of maturity.

## DISCUSSION

The theory developed to understand the organization of the juvenile period was built on the premise that fecundity and mortality rates, those of both juveniles and adults, are affected by changes in the age of first reproduction (for a different approach, see Stearns & Koella 1986). Each life-history variable was assumed to be a nonlinear function of age of maturity, and, at least for those depicting mortality, delay could either enhance or diminish the rates. Traditionally, it has been assumed that with delay comes increased size, resources, and experience and that all of these can augment fertility and juvenile survival, while reducing the rate of adult mortality. Only because delayed breeding incurs costs associated with missed opportunities and the fact that death is inevitable, is delay thought to have limits. Thus the existence of this trade-off ensures that for any species an optimal age of maturity will exist.

What emerges from the intraspecific case studies is the fact that on a species-specific basis, those showing life-history variables with strong sensitivities to the timing of sexual maturity tend to delay puberty, whereas those showing weaker sensitivities accelerate it. Rodents, with their normally short life spans, should typically have exponents that are close to zero because delaying puberty would incur large opportunity costs. That they breed early, in accord with the predictions of the model, is only reassuring because the empirical results show that fecundity and mortality in fact show little sensitivity to the timing of age of maturity. The converse applies to the longer lived species. For two species, red deer and horses, where the data are reported in ways so that species-specific tendencies could be evaluated, juvenile mortality shows a strong sensitivity to parental age of maturity. Again, it is reassuring to observe that both species (if we assume that zebras behave like horses, their close kin) show average or greater than average tendencies to delay reproduction for species of their size (Fig. 4.3). It is interesting to note, however, that in addition to these commonalities, adult mortality for horses also shows a strong age sensitivity, whereas for red deer it is fecundity that shows an additional strong maturational sensitivity. This suggests that although the same general rules apply to determining the length of the juvenile period, different ecological or phylogenetic features determine exactly how selection operates and on what life-history components.

Additional insights into how the length of the juvenile period is shaped emerge from examining how life-history consequences of changing the onset of puberty affect the different classes of individuals that compose populations. In every case study analyzed in this chapter, some individuals in a population bred significantly earlier than a modal age of maturity. In each case, these individuals belonged to groups, cohorts, or matriline in which sensitivities of fecundity, but mostly of juvenile mortality, to changes in the age of maturity were reduced. For rodents  $z$  was lower for late breeders than for early breeders; for red deer  $z$  was lower for females than for males; for horses  $z$  was lower for dominant females and females associating with dominant males; for elephant seals  $z$  was lower for cohorts free from competition; and for primates  $z$  was low for females that either had

dominant mothers or were able to achieve access to otherwise limited resources. It is interesting to note that in each case the sensitivity that is most important is the one that measures how much better offspring do as a result of their mother's delay and is the one that is left unconsidered in Charnov's (1990) original model.

Thus although there might be a modal, or typical, age of maturity, one that is initially influenced by body size (Pagel & Harvey, Chapter 3, this volume) and that lies close to the optimal age predicted by the theory, there seems to be alternative reproductive investment strategies that enable some to change the "rules of the game," thus allowing selection to maintain variation, and at times discrete polymorphisms, within populations. Even in the red deer, where rank produces overall sensitivities of fertility and survival to timing of maturation, the sexes show different sensitivities to the effects of pubertal age on juvenile survival. What emerges from these studies is the sense that phenotype, in relation to features of the physical and social environment, does affect investment "decisions," which ultimately alter the levels of juvenile mortality. What is striking is the fact that those females that can reduce the mortality of their offspring seem to be able to do so without increasing their own chances of dying. As Charnov (1990) and Pagel and Harvey (Chapter 3, this volume) suggest, the optimal age of maturity is more a function of mortality than fecundity, and at least for certain species, some "supermoms" seem to be able to develop.

But this conclusion raises two interesting problems. First, what do these within-species comparisons mean for the interspecific ones where the shape parameters cannot be estimated but are assumed to be positive? On a macroevolutionary time scale, the appropriate level of variation is the level that reflects large-scale rearrangements of one life-history variable with another. Thus it is appropriate to assume that levels of mortality are *unavoidable* and that they represent rates that account for all the parents have already attempted to do. Given this assumption, the absolute rates of mortality then assume more prominence in the models and shape the optimal pattern. That this occurs is clearly seen from the results, which indicate that after controlling for body size, species with high levels of juvenile mortality tend to breed relatively early in life, unless the effect of adult

mortality rate is also controlled. Only then do relatively high levels of juvenile mortality lead to delaying the onset of maturity. This striking result was first illustrated by Promislow and Harvey (1990) and shows that the conventional wisdom applies only when many other important effects are controlled. That the age of first reproduction scales more closely with the length of the dependent rather than the independent phase of the juvenile period suggests that physiology places limits on how rapidly any mammal can develop. But at least to a limited extent within a species, if certain classes of individuals can remove themselves from the constraints, then they can speed up the process and breed relatively early without incurring excessive costs.

Second, if selection does indeed favor at least some members of a population advancing the age of first reproduction, then what functions do juvenile periods serve when they are longer than the time it takes to become physiologically competent to reproduce? If individuals that ultimately obtain the highest lifetime reproductive success can do so with juvenile periods that are shorter than average, what does this mean for the notions that the juvenile period performs a valuable role in developing a better adult and that "more is better" because it gives youngsters more time to grow or gain experiences? The importance of this problem is underscored by the interspecific finding that the relative length of the independent juvenile phase, where this enhancement process is supposed to take place, is not correlated with other relative life-history features, including the relative age of maturity.

Answering these questions is not easy. The debate on the function of early development has often been heated and inconclusive (Bateson 1978, 1981; Klopfer 1981; Fagen 1984; Martin 1984). Due to its conspicuousness, play has often been the vehicle that has been used for gaining insights into the adaptive value of juvenile behavior. Although the above demographic and life-history analysis has been able to show that the juvenile period in the abstract has effects on subsequent reproductive success, ethologists have had a hard time demonstrating that particular features of play, or early development in general, are responsible for adult reproductive success (Bekoff & Beyers 1985). In fact, Martin and Caro (1985) argue that it might even be

pointless trying to find such a relationship. They suggest that play may indeed serve to enhance social and cognitive skills and to develop motor proficiency, as has often been assumed. But not because play prepares a juvenile to become a better adult, but because the benefits of play help make a better juvenile, one that is competent presently and will increase its chances of surviving, not necessarily one that will be better able to cope with the unique problems that beset adults. And if play is not very costly as they contend, then it need not provide large benefits to be maintained by selection (cf. Fagen, Chapter 13, this volume).

Evidence supporting this view comes from the detailed studies of play. Cuvier's gazelles (*Gazella cuvieri*), a species we have already identified as one where maturity and experience influence reproductive success, provides one such example. Gomendio (1988) shows that fawns participate in a variety of types of play, but that each type has a typical time course. Whereas locomotor play is frequently engaged in when fawns are very young, it drops off quickly and disappears from the repertoire long before weaning. Play fighting and sexual play, on the other hand, do not appear until about 1 month of age, and although they increase with age, they, too, vanish before weaning. If each of these different types of play evolves to enhance some aspect of later adult existence, then why does each exhibit the particular time course that it does? Play that involves partners, for example, is often thought to develop social and cognitive skills that will certainly be of use during adulthood. Why, then, should these most socially interactive forms of play vanish just prior to weaning when the conditions and peers are likely to be most similar to those that will be encountered during adulthood? While play at any time during the juvenile period can have preparatory effects, it also seems likely, and even more forcibly so, that juveniles engage most vigorously in these types of social play during periods of social uncertainty or whenever new social millieus appear. For gazelles, this is likely to be midway through the juvenile period when reliance on hiding gives way to becoming active and integration into the social group creates many novel social situations. This is precisely when social and sexual play peaks during the life of a gazelle.

The early life-style of the gazelle is just one example that can be counterbalanced to some extent by others, such as horses (Rubenstein 1982) and elephants (Lee 1986), where play does seem to take different directions depending on the juvenile's future role in adult society. But it does highlight the point that the juvenile period seems mostly about producing a competent juvenile that will live to see another day. Thus our finding that the relative length of the independent phase of the juvenile period seems to bear little relation to any other life-history variable when also measured on a relative basis should not be too surprising. And it complements the arguments of Janson and van Schaik (Chapter 5, this volume), who suggest that because juvenile primates are extraordinarily ecologically incompetent, they lengthen the juvenile period more than most mammals to reduce risk of mortality, not primarily to acquire experience since mastery of adult skills is often completed long before maturity is reached. Obviously exceptions exist, and, by highlighting them, the authors conclude that rapid development will be favored when ecological circumstances reduce competition, or niche overlap, between juveniles and adults. Overall, selection seems to be favoring the shortest possible period in which all the necessary developmental hurdles can be crossed without increasing immediate or future costs. Usually, however, only a subset of any population will find itself in such favorable circumstances.

Even the most basic allometric analysis shows that although large mammals have longer juvenile periods than smaller ones, the fact that the exponent describing the power function is less than one indicates that on a weight-specific basis, the larger species are exhibiting relatively shorter times to first breeding. Getting it right is apparently what counts, but deciding how long this should take is variable and, although constrained by some major physiological processes, seems to depend mostly on the sensitivity of an individual's fecundity and mortality to changes in the age of maturity. What, then, shapes these sensitivities? Much appears to depend on what one begins life with, where one's place in society is, and what parents, especially mothers, have to offer.