

Why Don't Male Mammals Lactate?

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(Received 7 August 1978, and in revised form 20 November 1978)

Why has male lactation never evolved? This question is discussed with attention to (a) the physiological and ontogenetic modifications that such an evolutionary development would require, and (b) the reproductive strategies of monogamous mammals and the costs and benefits of extant forms of parental investment. Functional male lactation would require changes in sexually differentiated ontogenetic processes at both prepubertal and circumpubertal stages, as well as some male analogue of lactogenic events in late pregnancy. None of these modifications seems impossible, but together they constitute a formidable barrier to the evolution of male lactation. Moreover, it is by no means clear that such an evolutionary development could enhance its bearers' fitness. Other factors than maternal lactational capacity evidently limit the reproductive potential of monogamous mammals. Much more information is needed on the economics of mammalian reproduction.

1. Introduction

As far as is known, male lactation has never evolved in any mammalian species. Why this should be so is an evolutionary problem worthy of consideration (Maynard Smith, 1977).

Only a minority of mammalian species could be considered candidates for such an evolutionary development. As many authors have remarked, the predominant reproductive strategy of mammalian males involves rather little parental investment of any sort. Reproductive effort is instead directed primarily toward male–male competition for the opportunity to fertilize females, who make a substantial parental investment (Eisenberg, 1966; Trivers, 1972; Wilson, 1975; Daly & Wilson, 1978). If the mammalian mode of reproduction, namely internal fertilization followed by gestation and lactation by the female, is taken as given, it is furthermore apparent why male parental behaviour should be rare: there is a long interval between conception and the stage at which the male can contribute usefully to the young. This interval entails prolonged costs in time, energy and fertilization prospects if the male closely attends and sequesters his mate,

and a risk of cuckoldry and misdirected parental care if he does not. This interval and the associated threat to paternity confidence (defined as the probability that the male indeed sired the offspring in question) apparently tip the scales toward low parental investment (Daly & Wilson, 1978; Kurland & Gaulin, 1979).

Despite generally low paternal investment in the class Mammalia, however, there is a widespread minority incidence of stable pair-bonding with resultant high paternity confidence (Kleiman, 1977), and it is in such monogamous species that Maynard Smith (1977) suggests male lactation might be expected to have evolved. In this paper, I discuss its absence, dividing the problem into two main issues. The first concerns the magnitude of the physiological hurdles that would have to be overcome for male lactation to evolve: are requisite preadaptations simply lacking? The second issue concerns the relationship between reproductive success and the forms of mammalian parental investment: would a monogamous male indeed increase his inclusive fitness if he were to relieve his mate of all or part of the lactational burden?

2. The Physiological Hurdles

In order to assess the physiological modifications that would be required for male lactation to evolve, we must analyse the control mechanisms of normal lactation in females, and note what factors are absent or altered in males. In this regard, we must understand not only the proximate control of functional lactation in the mature female, but all relevant sexually differentiated ontogenetic processes, since it is presumably by the suppression of such sexual differentiation that male lactation might evolve.

(A) THE ONTOGENY AND PHYSIOLOGICAL CONTROL OF FUNCTIONAL LACTATION IN MAMMALIAN FEMALES

The mammary gland is diagnostic of the class Mammalia and is of the same basic structure in all species (Cowie, 1972). Supportive tissue, the stroma, is distinct from secretory tissue, the parenchyma. The latter is organized into lobules, each containing a hollow alveolus into which milk is secreted by a single layer of epithelial secretory cells enveloped by myoepithelial cells and capillaries. The alveoli are drained by a system of tributary ducts converging to one or a few final channels through the nipple.

Two processes in the functional mammary gland are usually distinguished: milk secretion and milk removal. Secretion refers to the synthesis of milk within the alveolar cells and its passage through the cell

walls into the alveoli. Removal is the process whereby the secreted milk travels from the alveoli to the nipple.

A number of stages in the development of the lactating mammary gland can be distinguished. The embryonic tissue of origin is ectodermal. Described embryonic stages include the mammary streak, band, crest, hillock, bud and sprouts. In the fetus, the mammary sprouts differentiate into the ductal system and the developing nipple. For present purposes, all embryonic and fetal stages may be subsumed under the label of prenatal development. Elaboration of the mammary system is then arrested until the approach of puberty, when much ductal growth and cellular proliferation occurs in surges associated with early ovarian cycles. The circumpubertal period is thus our second broad developmental stage. Thirdly, lobulo-alveolar development occurs mainly in pregnancy and early lactation, with the precise timing varying considerably among species (Anderson, 1974).

The role of hormones in mammary development and function is exceedingly complex, and varies remarkably among species. Cortisol, corticosterone, cortisone, aldosterone, oestrogen, progesterone, insulin, thyroxine, parathyroid hormone, prolactin, growth hormone, placental hormones and relaxin have all been shown to influence mammary tissue in some mammalian species at one or more stages of development (Anderson, 1974).

In vitro, the embryonic differentiation process can proceed to the sprout state without any hormones in the medium. Shortly thereafter, prolactin, oestradiol, progesterone, growth hormone and cortisol each influence particular aspects of further differentiation (Anderson, 1974). Pituitary, ovarian and adrenal hormones have all been shown to be causal agents in mammary ontogeny at each of the three gross developmental stages: prenatal, circumpubertal and pregnancy.

In marsupials, events before parturition are apparently not causally relevant to mammary development or lactogenesis (the initiation of milk secretion): young fostered to virgin females provoke full lactation and grow normally (Sharman, 1976). In eutherians, however, hormonal changes during pregnancy have been implicated in lactogenesis. The effective complex of hormones for lactogenesis and for the maintenance of established milk secretion varies among species. Prolactin is the most universally essential (Schams, 1976), but placental lactogenic hormones, structurally related to prolactin and growth hormone, are sufficient to induce a transitory lactation at parturition in hypophysectomized monkeys (Tucker, 1974). Withdrawal of progesterone near parturition is evidently the stimulus for prolactin release and resultant lactogenesis in some species, but in others, no such withdrawal occurs, and a surge in oestrogen is accorded the same

function (Schams, 1976). Apparently a sharp change in the oestrogen/progesterone ratio is crucial, disinhibiting pituitary release of prolactin (Grosvenor & Mena, 1974). While the ovarian hormones influence lactogenesis principally by their effects on the pituitary, they also influence the mammary gland directly and by their effects on the adrenal cortex (Tucker, 1974). The administration of exogenous oestrogen to intact females may facilitate or inhibit lactation according to the species, but once lactation is established, ovariectomy does not greatly alter it (Tucker, 1974). Adrenal, pancreatic and pituitary hormones are essential to metabolic processes in the synthesis of milk components, but ovarian hormones are not (Baldwin & Yang, 1974).

The maintenance of established lactation depends primarily upon the pituitary hormones. Several of these, including prolactin, ACTH, TSH and growth hormone, are released by suckling stimulation on the nipple, and different ones are causal to milk secretion in different species, but prolactin seems to be of primary importance (Grosvenor & Mena, 1974). Maternal plasma prolactin levels are maintained by nursing, and rapidly drop to non-pregnant levels or even lower when nursing ceases (Amenomori, Chen & Meites, 1970; Tyson, Freedman, Perez, Zacur & Zanartu, 1976), but the capacity to reinstate full lactation persists longer (Grosvenor & Mena, 1974). Other stimuli from nurslings, including odours, can stimulate maternal prolactin release and hence milk secretion. The ejection of milk through the nipple is achieved by a neurohumoral reflex mechanism: stimulation of the nipple induces pituitary secretion of oxytocin, which hormone within seconds provokes contraction of mammary myoepithelial cells resulting in expulsion of milk from the alveoli.

In sum, ovarian, pituitary and adrenal hormones have all been implicated in all three stages of lactational ontogeny, though all may not be involved in every stage in every species (Anderson, 1974). Ovarian function is necessary in the circumpubertal and pregnant stages, but may not be essential in the prenatal stage (e.g. Neumann, Elger & von Berswordt-Wallrabe, 1966) or for the maintenance of established lactation (Tucker, 1974).

(B) SEXUAL DIFFERENTIATION AND MALE RESPONSIVENESS

Sexual differentiation of the mammary gland is similar to other aspects of mammalian sexual differentiation in that development proceeds in a female direction unless acted upon by androgens. If fetal androgen action is blocked in genetic males, full lactational function can develop when a female-simulating hormonal regime, including exogenous oestrogen, progesterone, cortisol and prolactin, is administered in adulthood (Neumann *et al.*, 1966).

The normal degree of mammary development in males varies considerably among species. In the much-studied rat and mouse, male nipple development is completely suppressed by early androgens although internal mammary structure develops. In many other mammals, including primates, canids and others, female-like nipples and ducts develop in males so that there is no gross structural pre-pubertal sex difference at all. However, the male gland remains relatively unresponsive to induction of further ductal and alveolar growth by the action of ovarian and pituitary hormones (Neumann, Elger, Steinbeck & Gräf, 1975).

It was once supposed that the pre-pubertal development of male mammary tissue must be arrested or dwarfed by early androgenic action, but this seems not to be the case. In both 30-day-old rats and pre-pubertal people, the quantity of mammary tissue and its histological appearance are similar in the two sexes. The relative refractoriness of the male gland instead appears to be attributable to alterations in neural sensitivity; a probable differentiating effect of early androgen is a reduction of oestrogen's capacity to block hypothalamic production of PIF (prolactin inhibiting factor; Neumann *et al.*, 1975). The pituitary is evidently undifferentiated: male pituitary extracts studied *in vitro* remain sensitive to the direct stimulation of prolactin release by oestrogen, and are in fact the preferred materials in such studies because female pituitary extracts give more variable results related to recent experience and hormonal buildup or depletion (Grosvenor & Mena, 1974). Differential sensitivity of the mammary tissue itself cannot yet be ruled out (Neumann *et al.*, 1975).

It should be stressed that the refractoriness of the male mammary gland is relative rather than absolute. Williams & Turner (1961) induced lactation in a steer by prolonged administration of oestrogen and progesterone, though the milk yield was low. External application of oestrogen may induce gynaecomastia (male breast development) in normal adult men; in such cases, the effect is not mediated by prolactin, although some other clinical cases of gynaecomastia are associated with elevated plasma prolactin (Turkington, 1972). Women are more sensitive than men to nipple stimulation (Short, 1976a), and respond with a plasma prolactin surge to stimuli that do not affect men (Noel, Suh & Frantz, 1974), but one study (Kolodny, Jacobs & Daughaday, 1972) demonstrated a substantial prolactin response to manual nipple stimulation in men, although still less than in women.

Moreover, lactation has been reported in human males under radical clinical treatment. Huggins & Dao (1954) induced two men, aged 64 and 62, to lactate after prolonged oestrogen treatment for prostate cancer, by a five-to seven-day treatment with luteotrophin (a prolactin-like hormone). In the

older man, castrated and taken off hormone treatment, lactation persisted for seven years and then abated. I believe it warrants note that I have been unable to find any other modern clinical description of lactation by a human male. Darwin (1871) claimed without citing evidence that "In man and some other male mammals (the mammary glands) have been known occasionally to become so well developed as to yield a fair supply of milk" (p. 186). This claim appears to be a myth. Several casual allusions in recent literature to the existence of such clinical cases cite only similar allusions in older literature, and such chains of citation repeatedly led nowhere.

(C) THE MODIFICATIONS NECESSARY FOR MALE LACTATION TO EVOLVE

What modifications of sex-typical physiology would be required to produce a functional male lactation? First, a reduction in the early androgenic suppression of later mammary development and responsiveness, whether such effects occur in the hypothalamus, the mammary gland or elsewhere. Second, a substitute for the oestrogenic facilitation of circumpubertal mammary development. Third, a substitute for stimulatory processes that occur in pregnancy, provoking further mammary development and lactogenesis. On present evidence, none of these appears to present an insurmountable barrier to the evolution of male lactation.

The first modification is of a type that has occurred frequently in mammalian evolution. Sexual dimorphism in any number of organismic features varies substantially among closely related species. Examples are structural characteristics like body size and canine development and behavioural characteristics like aggressivity; such features commonly vary in the degree and even the direction of sexual dimorphism. The influence of the early androgenic sexual differentiation mechanism (which may control *all* aspects of sexual differentiation; Ohno, 1976) on particular organismic features thus appears to be very labile evolutionarily. More particularly, species differences in the effects of early androgen upon fetal nipple and duct development (see above) argue that mammary differentiation is susceptible to evolutionary modification of the suppressive effects of early androgens. Whether hypothalamic sexual differentiation relevant to lactational function might be inextricably bound up with other essential sex differences cannot be answered.

The second modification, namely a male equivalent of the female's circumpubertal surge of mammary growth, is also mechanistically plausible. The steroids are so alike structurally that a localized conversion of androgens or adrenal hormones to oestrogens in the male mammary gland

could evolve where this was selectively advantageous. Indeed, intracellular conversion (aromatization) of testosterone to estradiol is a necessary step in testosterone's action upon certain cells (Naftolin *et al.*, 1975). Although the enzymes for this aromatization process have not been located in mammary cells, a variety of synthetic and natural steroids, including androgens, have been shown capable of substituting for oestrogen and/or progesterone in stimulating mammary growth, although the effects are quantitatively reduced in comparison with the ovarian hormones (Anderson, 1974). Moreover, gynaecomastia in pubertal boys is a common clinical syndrome unaccompanied by any gross pathology, and familial patterns in the syndrome indicate that genetically based variability in tissue sensitivity exists (Turkington, 1972).

The third requirement for male lactation, namely a mechanism that could substitute for those processes in pregnancy which stimulate further mammary growth and then lactogenesis, again seems to require only a plausible quantitative alteration of male responsiveness rather than any major qualitative changes. The experience of pregnancy *per se* is apparently less critical than might be supposed: it is causally irrelevant to mammary development and lactogenesis in monotremes and marsupials (Sharman, 1976), and in eutherians, a much briefer regimen of hormone treatment can mimic pregnancy's lactogenic effects (Tucker, 1974). Placental lactogens, which are related structurally and functionally to prolactin, are important in some eutherian species, but are evidently completely lacking in others (Schams, 1976). In the pregnant female, a high progesterone/oestrogen ratio inhibits prolactin release until parturition is near; lactogenesis is then induced by a prolactin surge, caused by a sudden change in the ratio of ovarian hormones and a resultant reduction in hypothalamic PIF production.

Prolactin has a variety of functions in males as well as females. It is implicated in prostate and seminal vesicle maintenance, and synergizes with androgen in stimulating spermatogenesis (Nicoll, 1974). Plasma prolactin levels are comparable in males and in non-reproductive females (e.g. Amenomori *et al.*, 1970; Davis, Reichert & Niswender, 1971; Noel *et al.*, 1974). Seasonal prolactin fluctuations (Tucker, 1974) and prolactin responses to various chemical stimuli (Turkington, 1974) are similar in the two sexes. Very high prolactin levels, comparable to those of late pregnant females, can occur "spontaneously" at least occasionally in male samples (Davis *et al.*, 1971). Moreover, breast stimulation can induce a prolactin surge in males (Kolodny *et al.*, 1972). These several facts together suggest that male lactogenesis could occur despite the absence of pregnancy, and could thus become available to enhancement by natural selection, if the first

modification mentioned above, namely a mitigation of earlier sexual differentiation, were achieved.

(D) CONCLUSION

Physiological barriers to the evolution of male lactation do not seem individually insurmountable. However, the rarity of even partial or pathological male lactation indicates that they are formidable. Although experimental manipulations of both mature and immature males can produce lactation, and although there is evidence of heritable variability in the sensitivity of male mammary tissue to the induction of further development, there is no indication that male lactation might ever occur spontaneously in natural mammalian populations.

3. Reproductive Strategies in Monogamous Mammals

Under what circumstances might male lactation evolve? Several authors have discussed the conditions under which males are likely to allocate reproductive effort to parental care rather than merely the pursuit of matings. (Such "parental" investment by a male includes both prenatal activity such as feeding and guarding the female he has impregnated, and postnatal contributions whether made directly to the young or again indirectly via the mother.) Some authors (e.g. Wilson, 1975; Kleiman, 1977) have focused attention on the degree to which biparental care seems to be required by environmental exigencies, while others (e.g. Trivers, 1972; Clutton-Brock & Harvey, 1977; Emlen & Oring, 1977) have stressed the fitness consequences for the male of staying to participate parentally versus deserting to seek other mates.

A strong correlate of male parental investment in mammals is a monogamous mating system. This is to be expected for at least two reasons. In the first place, parental investment and the pursuit of polygynous matings are competing alternatives in a male's reproductive effort budget. Secondly, a reproductive strategy that carries a high risk of misdirected parental investment cannot be evolutionarily stable. Whereas misdirected maternal care is not a serious problem in mammals, since females commonly form individualized bonds with their young at parturition, males can only achieve paternity confidence by persistent association with their mates. (In principle, a male might abandon a fertilized mate to seek others, returning to contribute parentally at a later stage in the reproductive episode. In practice, no male mammals seem to use this strategy, which hazards such mishaps as induced abortion and reinsemination by rivals. Paternal investment appears only where the mates are associated continuously from conception through

pregnancy.) Male parental investment should thus be best developed in species with monogamous mateships or stable harem situations. The latter are relatively rare and oblige considerable male defense, so I will consider only the former as candidates for the evolution of male lactation.

Our limited knowledge of the distribution of monogamy among the mammals has recently been reviewed by Kleiman (1977). Monogamy is rare but widespread, characterizing one or more species in at least nine orders. It is most common in Carnivora (especially Canidae) and Primates (especially Hylobatidae, Callitrichidae and many Cebidae); in each of these two orders there are probably more than fifty living monogamous species.

Kleiman (1977) argues that monogamy "does not imply anything about the degree of paternal investment". She proposes that monogamous species may be categorized as "facultative (type I)" with low paternal investment or "obligate (type II)" with high paternal investment. She further suggests that "if the evolution of Type I Monogamy was due primarily to over-dispersion of adults, one would expect neither a strong bond nor a heavy paternal investment". This argument is not convincing, since a male strategy with neither pursuit of extra matings nor parental investment is unlikely to be evolutionarily stable in competition with strategies involving greater male reproductive effort. Selection favours behaviour with positive fitness consequences in the species-typical selective milieu, regardless of that milieu's origins. Where males are regularly monogamous and have few chances for polygynous matings, selection may be expected to favour their diverting reproductive effort into parental investment (Emlen & Oring, 1977). Where little or no paternal contribution is evident, as in acouchis and dik-diks (Kleiman's exemplary type I monogamists), the adaptive significance of male behaviour patterns needs elucidation. Perhaps monogamy is truly "facultative" in these species, in the sense that opportunities for polygyny occur regularly enough to maintain male readiness to exploit them.

Where stable mateships are the rule, male fitness and the mated pair's reproductive output are the same. Accordingly, the question of the selective advantage of male lactation becomes:

- (1) Could the monogamous pair's reproduction be augmented by reallocation of male parental investment to lactation?

In this form, the question requires examination of the forms, costs and benefits of non-lactational parental, especially paternal, investment. Alternatively, the question of the selective advantage of male lactation might be posed as:

- (2) Is the reproductive capacity of a monogamous pair limited by the female's lactational capacity?

This version of the question suggests that one should examine energetic and other factors limiting a pair's reproductive potential.

With these questions in mind, we will first examine the natural history of some exemplary monogamous mammals and then discuss what limits a pair's reproductive capacity. I have chosen the Canidae and Hylobatidae as examples for several reasons. They have been better studied in the field than other monogamous mammals, and they represent the two orders in which monogamy is most prevalent. Furthermore, they illustrate two hypothetical evolutionary routes by which reproduction might be augmented in evolution: an increase in litter sizes or a shortening of birth intervals.

(A) CANIDAE

Monogamous social organization and male parental investment are typical in canids, regardless of whether the species-typical hunting unit is a lone animal, a pair or a pack (Kleiman, 1977).

Although canids vary considerably in body size, habitat, and prey characteristics, they are quite similar in their reproductive time tables (Ewer, 1973). While it must be noted that information is far from complete, it appears that most or all wild canids:

- (1) breed but once annually in a well-defined season;
- (2) gestate their litters for approximately two months;
- (3) subsist on an exclusive diet of milk for the first month postpartum;
- (4) are fully weaned by about two months of age; and
- (5) remain dependent upon parental hunting until four or more months of age (Ewer, 1973).

Male participation in provisioning the female and pups has been observed in wolves, hunting dogs, jackals, coyotes, and at least three genera of foxes (Kleiman & Eisenberg, 1973). In the first three of these, other pack members than the breeding pair also feed the young (Moehlman, 1978). Whether the mother's own hunting activity is ever entirely suspended in the early postpartum period is not known.

Could a canid pair increase their reproductive output if the male lactated? There is no reason to suppose that they could, for there is no evidence that it is specifically the mother's lactational capacity that imposes a ceiling on litter size. If that were the case, we might expect pup mortality to decline considerably at weaning. Conclusive evidence on this point does not exist, but that available suggests that pup mortality rates are at least as high after weaning as before, and that, at least in temperate climates, limiting food shortages come into play in winter when pups are several months old and

fully weaned (Jordan, Shelton & Allen, 1967; Ables, 1975; Lloyd, 1975; Nellis & Keith, 1976). More data are needed to assess starvation risk and resource availability over the year, and to assess whether pup condition at weaning *is* limited by maternal lactational capacity and is predictive of later mortality. On present evidence, however, it may be suggested that the factor limiting the number of young raised to maturity is probably not maternal lactational capacity but is instead the family's capacity to extract energy from available (defensible?) hunting grounds in the most arduous season.

(B) HYLOBATIDAE

The Hylobatidae seem a particularly likely candidate family for the evolution of male lactation. All modern species (the siamang, *Symphalangus syndactylus*, and about six species of gibbons, *Hylobates*) are evidently obligate monogamists. Families remain in close association in all seasons. One siamang pair has remained intact on the same home range for at least six years (Chivers, Raemakers & Aldrich-Blake, 1975), and all studies to date are consistent with the suggestion that Hylobatidae are regularly sedentary, territorial, and mated for life.

Limited data indicate birth intervals of three years in siamangs (Chivers, 1974) and two to three years in gibbons (Ellefson, 1974; Tenaza, 1975). Weaned young remain in the family group long after the birth of younger siblings. In several *Hylobates* species, up to four young of different ages may remain in the family (Carpenter, 1940; Ellefson, 1974; Tenaza, 1975). The siamang family studied intensively by Chivers (1974) contained a two-year-old infant, a five-year-old juvenile and a subadult eight or nine years old.

At about eight to ten months postpartum, the siamang father assumes the task of carrying the growing infant, and this he continues to do for over a year, although with declining frequency as the infant's own locomotion improves (Chivers, 1974). Male gibbons exhibit no such conspicuous parental care, but there are several indications of substantial paternal investment in this genus too. Males evidently take a larger role in territorial defense (although adult males and females each defend primarily against their own sex). In *H. lar* observed by Ellefson (1974), mated males spent approximately 6% of their waking time in intergroup conflict and wounds were inflicted upon males in such conflicts. When a family group fed near a border of their territory, the male might remain vigilant or defend a food tree while his mate and offspring fed there, eating nothing himself. In Ellefson's opinion, the energetic cost of such behaviour is evidenced by the fact that the adult male regularly foraged 30 min to an hour longer than the rest of the group in the evening. In *H. klossii*, Tenaza (1975) observed that males took

the main role in defending against human predators, hanging back and circling potential hunters while the females and young retreated. Under conditions of a fully occupied territorial mosaic (a circumstance perhaps typical in hylobatid history but lacking in Chivers's and Ellefson's human-disturbed habitats), Tenaza (1975) furthermore observed an adult male helping his son establish an adjacent territory by co-operative attacks on a neighbour.

Could male lactation augment the reproductive output of a hylobatid pair? The possibility is not so readily dismissed as in the case of the Canidae. What is at issue is the factors responsible for the long birth interval, and we know too little to identify these with confidence. The nursing infant must surely be a proximate cause of the mother's prolonged infertility (otherwise, mothers could not rapidly replace dead infants), probably by the well-known mechanism of suckling inhibiting ovulation via hypothalamic mediation (Short, 1976*b*; Knodel, 1977). This does not necessarily mean that prolonged lactational demands by the infant are the ultimate causes. It would be useful to know the magnitude of the infant's lactational demands over time, and the time-course of maternal depletion and recovery from those demands. (These problems are discussed later.) Meanwhile it remains plausible that two to three year birth spacing is imposed upon these highly *K*-selected (Pianka, 1970) apes by limitations on the family's capacity to harvest energy or other nutrients from a defensible territory, and not by lactational demands *per se*. Parents may also be limited in the frequency at which they can effectively help their offspring establish territories.

(C) LACTATIONAL EFFORT AND MATERNAL DEPLETION

Nobody has yet been able to measure the energetic or other costs of lactation (let alone those of the entire reproductive episode) in any mammal in the wild. In captivity, dietary requirements for lactation have been most extensively studied in dairy cattle, but their history of artificial selection for milk production far beyond the needs of their own young renders them uninformative for our purposes. Results for another domestic species not bred as a milk producer are shown in Table 1.

In the laboratory, the females of several cricetid rodents, namely *Clethrionomys glareolus* (Kaczmarski, 1966), *Microtus arvalis* (Migula, 1969) and *Peromyscus leucopus* (Millar, 1975), have been found to increase energy ingestion at the peak of lactation over that of non-reproductive females by factors of 2.4, 3.3 and 2.6 respectively, besides depleting body fat stores accumulated during pregnancy. All of these are small, *r*-selected (Pianka, 1970) prey species. The relative ingestion increment may be

TABLE 1

Nutrient requirements of horses. (From NAS-NRC subcommittee on horse nutrition, 1973)

	Daily feed (kg)	Digestible energy (Mcal)	Protein (g)	Calcium (g)
Mature horse at rest	5.04	13.9	505	16.0
Mature horse at medium work, 2 h day ⁻¹	6.68	18.4	672	16.0
Last 3 months pregnancy	5.41	14.9	613	19.5
Peak lactation	8.91	24.4	1181	42.0

(All horses at 400 kg body weight.)

considerably less under field conditions, however, since basal metabolism in the field may greatly exceed that in the lab with the absolute incremental cost of reproduction remaining similar; whether this is indeed the case is simply not known.

In our own species, several estimates of the energy cost of lactation have been made. Thomson, Hytten & Billewicz (1970) found lactating British mothers at about two months postpartum to eat 2716 kcal day⁻¹, approximately 28% more than bottle-feeders, while losing weight at a slightly faster rate than the bottle-feeders. Widdowson (1976) estimated that the energy costs of milk production increased from about 450 kcal day⁻¹ in the first month to over 800 kcal day⁻¹ in the sixth month and that about one-third to one-half of the extra energy required for a six-month lactation is stored as fat during pregnancy. On poor diets, Hyderabad village women studied by Ebrahim (1976) maintained lactation of 450–600 ml day⁻¹ over the first six months (adequate to raise their infants with no supplemental feeding) while consuming just 1800 kcal day⁻¹ and losing 2 kg (4.7%) of their postpartum body weight.

So far, I have focused upon energy as the factor limiting a female mammal's reproductive potential, and so have most discussions. This emphasis has been dictated more by the conspicuousness and ease of measurement of maternal body weight than by evidence for energy's primacy. The availability of any of several specific nutrients may often be important in limiting reproduction.

Considerable iron, for example, is required by both the fetus and the pregnant female. In well-fed human mothers, plasma haemoglobin increases

by about 85 g during pregnancy (Hyttén & Thomson, 1968), but poorly nourished human mothers may not recover iron expended in reproduction, and may instead exhibit a cumulative deficiency over successive confinements (Jelliffe, 1976). Another nutrient in which cumulative maternal depletion over successive pregnancies and lactations has been observed is folic acid (Jelliffe, 1976).

Calcium is probably the best studied nutritional element in mammalian reproduction. Lactation generally demands a far greater increment in the mother's calcium intake than in her intake of energy or even protein (e.g. Table 1). Depletion of maternal calcium stores for reproductive purposes is probably typical. In a study of well-fed human mothers by X-ray spectrophotometry, Lamke, Brundin & Moberg (1977) found that maternal skeletal calcium declined through pregnancy and the first three months of lactation, but recovered over the next three months whether or not lactation was continued. Calcium budgeting thus differs from energy budgeting in that maternal stores are being depleted rather than accumulated even in the prepartum period. How calcium availability may constrain reproductive potential under more difficult dietary circumstances is unclear. In Indian women who continue breast feeding during a subsequent pregnancy, milk volume declines rapidly; this is partly compensated by an increase in milk protein concentration, but in the case of calcium, the concentration drops too, so that the nursing infant's total calcium intake falls precipitously (Gopalan & Belavady, 1961). When rats were placed on a low-calcium diet (0.05% Ca) during weeks two to four of lactation, mothers sacrificed fully half of their skeletal calcium "without apparent untoward effects other than slightly stunted growth of the young animals" (de Winter & Steendijk, 1975). This study is particularly interesting in that recovery was also studied. After maternal skeletal calcium was reduced to 46% of its original level during three weeks of lactation on the poor diet, pups were removed and mothers were returned to an adequate diet (1.03% Ca); after three weeks, skeletal calcium had recovered to 78%. On gross external examination, such animals might appear fully recovered while in fact still severely depleted by their reproductive efforts.

Whether such covert maternal depletion occurs in natural situations and influences the quantity or quality of subsequent progeny deserves study. In fact, almost nothing is known of the time-course of recovery from maternal depletion of various substances, nor of the effects of variations in maternal nutrition and in the intensity and duration of lactation, nor of the precise effects of maternal condition upon the young's prospects for survival and reproduction. These are huge gaps in the information needed to satisfactorily describe and explain maternal strategies for partitioning reproductive effort.

(E) LACTATIONAL EFFORT AND LIFE HISTORY STRATEGIES

Further correlates of monogamy in mammals are low reproductive rates and slow development of the young (Eisenberg, 1966; Kleiman, 1977). There are several reasons why mammalian monogamy might be especially likely in conjunction with the set of life history characteristics usually referred to as "K-selected": high adult survivorship makes more salient the long-term advantages of commitment to a familiar territory and mate; a no-vacancies breeding population selects for delayed maturity and prolonged parental investment by both sexes; infrequently breeding females can afford to demand male investment as a condition for mating; and impregnable females are not continuously available to tempt male desertion of mates. But whatever the ultimate reasons, monogamy and a relatively slow reproductive pace tend to occur together. (I refer to alternative constellations of life-history characteristics as "r-selected" and "K-selected" as a shorthand, without intending that these terms must correctly describe the process of evolution of the strategies, cf. Stearns, 1977.)

One important consequence of this correlation is that physiological reproductive effort tends to be relatively low in monogamous species, and relatively high in the non-monogamous. Rapid breeders, such as most rodents, may therefore at the same time be (1) the mammals in which maternal lactational capacity most directly limits reproduction (the condition that would favour male lactation), and (2) the mammals in which opportunities for polygyny, brief pairing and low paternity confidence select most strongly *against* male parental investment of any sort. The data in Table 2 support this possibility by illustrating that lactational demands, measured as relative increments in maternal ingestion, are much larger in small r-selected species than in larger, relatively K-selected ones. (Relative ingestion increment may or may not be a good measure of the costs of lactation in time, predation risk, and the like.)

The phenomenon of variable implantation delay affords another bit of evidence that maternal lactational capacity may limit reproduction in rapid breeders to a greater extent than in slower breeding species. Conception during a postpartum oestrus occurs in many rodents, but the durations of lactational pregnancies are highly variable. This is because of a variable latency to implant the new blastocysts, and a major determinant of that latency is the size of the nursing litter (e.g. Enzmann, Saphir & Pincus, 1932). Variable implantation delay represents a flexible maternal capacity to distribute resources among successive litters, and the very existence of postpartum oestrus and this variable implantation delay implies that the mother's lactational capacity indeed limits her reproductive potential.

TABLE 2

The increment in maternal ingestion at peak lactation in a few mammalian species with diverse reproductive strategies

Species	Approximate body weight (kg)	(1) Non-lactational ingestion (kcal day ⁻¹)	(2) Peak lactational ingestion (kcal day ⁻¹)	% increment $\frac{(2)-(1)}{(1)} \times 100$	Reproductive strategy
Bank vole <i>Clethrionomys glareolus</i> (Kaczmarski, 1966)	0.02	17.5	47	169	Highly "r-selected". Continuous litter production in season
Common vole <i>Microtus arvalis</i> (Migula, 1969)	0.03	17.0	38	124	Highly "r-selected". Continuous litter production in season
Horse <i>Equus caballus</i> (NAS-NRC, 1973)	400	13,900	24,400	76	"K-selected". Single offspring annually
Human <i>Homo sapiens</i> (Thomson <i>et al.</i> , 1970)	60	2,125	2,716	28	Highly "K-selected". Single offspring with birth interval of several years

Under conditions of *K*-selection, reproductive possibilities tend to be limited by environmental scarcities. As suggested above for hylobatids and canids, monogamous mammals under such conditions are constrained by their capacity to harvest energy (or other scarce resources) rather than by lactational capacity *per se*. Conditions of *r*-selection are, by contrast, those in which environmental resource constraints are relaxed and reproduction is limited primarily by the intrinsic physiological capacities of the reproducers; such conditions also offer males opportunities for polygyny and hence do not favour the evolution of male parental investment.

(F) THE COLUMBID ANALOGY

Pigeons and doves feed their newly-hatched squabs "crop milk", a substance composed of sloughed epithelial cells. This phenomenon is analogous to mammalian lactation in several respects, notably in aspects of the fluid's composition and in the role of prolactin in inducing and maintaining lactation (Fisher, 1972). Both sexes produce crop milk.

Why do male pigeons lactate when male mammals do not? Kurland &

Gaulin (1979) pose this question and suggest as an answer that the long internal gestation of mammals threatens paternity confidence. This is true, but it is more germane to explaining the relative rarity of male parental investment of any sort in mammals as opposed to birds. There are, after all, mammalian species, like the siamang, in which paternity confidence approaches one (perhaps higher than in most birds, who are vulnerable to inter- and intra-specific brood parasitism) and paternal investment is extensive.

A better answer may come from consideration of the different evolutionary pathways leading to the present parental practices of columbids and mammals. The universality of lactation in mammals indicates that this form of maternal care is of great antiquity and presumably antedates the evolution of male parental investment in the various mammalian lines in which the latter has appeared. Thus, from a foundation of sexually dimorphic physiology and behaviour, various forms of male parental investment have evolved independently. Rather than simply shouldering a share of maternal behaviour, mammalian males commonly evolved novel contributions like feeding and guarding the mate. In the case of birds, however, the prevalence of monogamy, shared incubation, and shared feeding of nestlings indicates that these developments antedated the evolution of crop milk. Thus, from a foundation of sexually monomorphic parental behaviour, evolutionary change in the crop's function occurred in both sexes.

(G) HAS MALE LACTATION HAD ENOUGH TIME TO EVOLVE?

The physiological barriers to male lactation appear large but not insurmountable. Perhaps male lactation would enjoy a selective advantage in monogamous mammals if it ever got off the ground. A rare and substantial mutation might be required to produce the phenomenon at a magnitude sufficient for positive selection to occur. Speculation along these lines leads one to wonder how much opportunity there has been for such a mutation to occur. Are monogamous mammals so few and so recent that male lactation has only by chance not yet evolved?

This idea appears to be tenable only if the hypothetical requisite mutation were very improbable indeed. Monogamy in mammals has a substantial history, as can be illustrated by further consideration of our exemplary families Canidae and Hylobatidae.

Kleiman & Eisenberg (1973) suggest that monogamous social organization is primitive in the Canidae. If so, the family's appearance in the early Oligocene (Dawson, 1967) implies some 30 million years of monogamous

evolution. Much more conservatively, we can assume at least two million generations of monogamous evolution in the genus *Canis*, which arose in the late Tertiary.

The fossil record also permits an estimate of the duration of hylobatid monogamy. In body size and dentition, modern hylobatids are essentially monomorphic, a state of affairs which is strongly correlated with monogamous social organization in primates (Clutton-Brock & Harvey, 1977; Harvey, Kavanagh & Clutton-Brock, 1978). In *Dendropithecus* (Andrews & Simons, 1977), a possible hylobatid ancestor of 17 to 22 million years B.P., dental dimorphism greatly exceeds that of modern hylobatids and approximates that of modern chimpanzees and gorillas (Andrews, 1973, fig. 73). By 14 to 15 million years B.P., similar ape species exhibited considerably reduced dimorphism (Andrews, personal communication); this may represent a date for the emergence of monogamous social organization in the hylobatid line. A more conservative estimate for that date is 10 million years, based on biochemical evidence on the duration of separation of modern hylobatids (Andrews, personal communication). These considerations suggest that a monogamous mating system has prevailed in the Hylobatidae for roughly one million generations.

Such considerations as these cannot disprove the hypothesis that male lactation would be favoured selectively but has yet to arise. However, they reinforce my preference for an alternative position, developed in the preceding sections, which holds that:

(1) Male lactation is a non-starter because sexually dimorphic lactational physiology had already evolved considerably before the advent of male parental investment in mammalian evolution.

(2) Those species in which maternal lactational capacity limits reproductive potential so that females might benefit from lactational assistance are precisely those *r*-selected mammals in which the evolution of male parental investment of any sort is unlikely.

(3) Monogamous pairs are limited reproductively by their capacity to extract resources from their environment. Males have evolved non-lactational forms of parental investment, and the addition of male lactation to the parental repertoire would not necessarily be useful.

The validity of this position will not be assessed until we know a great deal more about the economics of reproduction in long-lived mammals in their natural habitats.

4. Conclusions

(1) Physiological impediments to the evolution of male lactation do not seem individually insurmountable. Rarity of even partial or pathological

male lactation, however, indicates that they are formidable. It remains plausible that male lactation has never yet occurred spontaneously in natural mammalian populations at a magnitude adequate to provide any significant alleviation of female lactational depletion and hence be favourably selected. However, I do not favour this as an explanation for the absence of male lactation.

(2) While lactational anovulation is the principal proximate control mechanism of long birth intervals in monogamous mammals, it is to be doubted that maternal lactational depletion *per se* is the ultimate factor proscribing more frequent reproduction. Neither does female lactational capacity necessarily limit litter sizes, which may instead be constrained by resource availability in postweaning stages. Controlled laboratory studies of females on *ad libitum* or restricted diets are not very informative in these matters, since reproductive strategies are adapted to natural ecologies. We know almost nothing of the magnitude of lactation over time, nor of the depletion and repletion of maternal energy, calcium or other stores, for any wild mammal.

(3) Male behaviour is a product of selection for inclusive fitness maximization. Where monogamous mateship and paternity confidence are regular elements in the selective milieu of a species, paternal investment in the welfare of offspring may be expected to evolve, regardless of the original circumstances leading to monogamy. Paternal investment takes many forms, and subtle indirect contributions are difficult to establish and measure. The evolution of male lactation would not necessarily involve increased paternal investment and a concomitant reduction in required maternal investment, nor would it necessarily improve the reproductive performance of monogamous pairs.

I thank Michael Leon and Margo Wilson for critical comments.

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