Biol. Rev. (2004), **79**, pp. 733–750. © *Cambridge Philosophical Society*
DOI: 10.1017/S1464793103006432 *Printed in the United Kingdom*

Female post-reproductive lifespan: a general mammalian trait

Alan A. Cohen

Department of Biology, *University of Missouri-St. Louis*, 8001 *Natural Bridge Rd.*, *St. Louis*, *MO* 63121-4499, *USA*
(E-mail: aacrg7@*studentmail.umsl.edu*)

(*Received* 23 *April* 2003; *revised* 12 *November* 2003; *accepted* 1 *December* 2003)

ABSTRACT

Traditional explanations for the evolution of menopause and post-reproductive lifespan in human females have been based on the benefits of maternal or grand-maternal care outweighing the cost of lost reproduction. These explanations assume an evolutionary origin of menopause since human divergence with the most recent common ancestor. In this study, I conduct a literature survey of studies of 42 mammal species from eight orders, showing that post-reproductive lifespan appears to be widespread among mammals. I then propose an alternative to traditional hypotheses: following accepted theories of trade-offs and senescence, I suggest that the cost of extending reproductive lifespan might be relatively high in female mammals. Somatic and reproductive senescence appear to follow separate trajectories, so it is not surprising that the two processes should occur on different schedules. The timing of each process is probably determined by maximization of reproductive performance and survival early in adulthood, with consequent trajectories resulting in a post-reproductive lifespan. The early end of reproduction relative to lifespan may be due to the cost of production and/or maintenance of oocytes, which decline exponentially over time. Oocyte number below a threshold may trigger an end to normal hormonal cycling.

Key words: menopause, post-reproductive lifespan, mammal, trade-off, oocyte, reproductive senescence.

CONTENTS

I. Introduction 733
II. Definitions, parameters, and approaches for the analysis of PRLS 734
III. Is female PRLS unique to humans? 735
(1) Humans 735
(2) Non-human primates 737
(3) Non-primates 739
(4) Conclusions on the generality of PRLS in mammals 739
IV. The adaptive hypotheses 740
V. A hypothesis for the origin of PRLS in mammals 742
(1) The broad form of the hypothesis 742
(2) The specific form of the hypothesis 745
VI. Conclusions 747
VII. Acknowledgements 747
VIII. References 747

I. INTRODUCTION

Human female menopause has received considerable attention over the past thirty years, both from the medical community seeking to improve the health of post-reproductive women and from evolutionary anthropologists seeking an explanation for the termination of reproduction (e.g. Lancaster & King, 1985; vom Saal, Finch & Nelson, 1994; Wise, 2001). Although the physiology of menopause is well understood, its evolutionary origins are

still hotly debated (vom Saal *et al.*, 1994; Austad, 1997; Peccei, 2001; Shanley & Kirkwood, 2001). A paradox arises because natural selection should disfavour any lack of reproduction, yet human females often spend more than a third of their lives post-reproductive. Most women, even in hunter-gatherer societies, stop bearing children by their late forties even though they regularly live into their seventies and eighties. Among the Ache of Paraguay, for example, 41 % of those reaching reproductive maturity live to age 70 (Howell, 1979; Hill & Hurtado, 1996). Men, by contrast, are generally fertile until near the end of the lifespan, although there are age-specific declines in fecundity (vom Saal *et al.*, 1994).

Most of the work on the evolutionary origins of post-reproductive lifespan (PRLS) has focused on the intuitively attractive 'grandmother hypothesis', which suggests that the benefits through kin selection of aiding children and grandchildren outweigh the cost of lost reproduction, especially considering the increases with age in risks of death during childbirth and not surviving until offspring independence (Williams, 1957; Alexander, 1974; Hawkes *et al.*, 1998). Now, some researchers distinguish aid to children and grandchildren as separate hypotheses (Peccei, 1995, 2001; Shanley & Kirkwood, 2001), or distinguish menopause as 'stopping early' *versus* PRLS as prolonging lifespan after the end of reproduction (Hawkes *et al.*, 1998). A detailed treatment of these various versions of the grandmother hypothesis is beyond the scope of this review, so for simplicity I will refer to them collectively as 'adaptive hypotheses'. They are adaptive in the strict sense of Gould & Vrba (1982), depicting PRLS as a trait evolved for a current function since the split of humans with their most recent common ancestor.

An often-cited competing idea considers PRLS to be simply an artifact of recent increases in human lifespan (Washburn, 1981; Weiss, 1981), but to my knowledge no one has actively advocated this idea in twenty years. In any case, it does not address why longevity has increased while female reproductive span has not. If longevity increased because of selection, female reproductive span should have been subjected to the same selection and have increased concomitantly. If longevity increased because improved living conditions reduced extrinsic mortality, the discrepancy between age at menopause and age at death still would require explanation. Perhaps because no plausible alternative has been proposed, the adaptive hypotheses, despite some criticism (Hill & Hurtado, 1991; Rogers, 1993), remain the most widely accepted explanations for the evolution of PRLS.

In this review, I critically examine the framework for these adaptive hypotheses. I begin with an assessment of data on reproductive senescence in a wide variety of mammal species. Although differences in the available data and the methods of the studies make the various species difficult to compare directly, I look specifically for indications that the end of reproduction in females tends to occur well before the maximum lifespan. Species are then rated as appearing to have PRLS, not have PRLS, or as ambiguous. This analysis shows that PRLS, although particularly long in humans, is not unique to humans and indeed appears to be widespread among mammals. I then examine adaptive hypotheses in light of this finding and suggest that adaptive hypotheses cannot explain the origin of PRLS, although they may or may not explain variation in length. Lastly, I propose a new hypothesis to explain PRLS in mammals. A broad form of the hypothesis is used to explain how PRLS could occur whenever reproductive senescence and somatic senescence follow distinct trajectories. Maximization of reproduction and body condition in the prime of life may result in trajectories that are asynchronous at their tail ends; PRLS will occur if reproductive senescence occurs faster or earlier than somatic senescence. A specific form of the hypothesis examines how the physiology of reproductive senescence in female mammals (attrition of oocytes): (1) explains demographic data suggesting independence of reproductive and somatic senescence; and (2) suggests possible trade-offs which would lead to fast or early decline in reproduction after the prime of life. I will begin with a discussion of the approaches appropriate for the study of PRLS.

II. DEFINITIONS, PARAMETERS, AND APPROACHES FOR THE ANALYSIS OF PRLS

First, it is important to indicate that PRLS, not menopause, is the appropriate parameter for study. Menopause is a term attached to a primarily human trait, so trying to identify in a comparative analysis which species do or do not have menopause would result more in a linguistic morass than a scientific analysis. Further, it is the period of post-reproductive lifespan, not the event of reproductive cessation, which presents a paradox in terms of hypothetically lost reproduction. I will use the term 'menopause', but I use it in a restricted sense particular to this subject: *the irreversible loss of the physiological capacity to produce offspring due to intrinsic biological factors*. Etymologically, this definition is imprecise, but it pinpoints physiological reproductive decline as a key factor for discussion of PRLS among taxa. It is also distinct from 'reproductive cessation', which is *the last direct reproductive event* (*birth or insemination*) *in the life of an organism*. Caro *et al.* (1995) consider reproductive cessation to have occurred only if more than one inter-birth interval has passed since the last reproductive event. Because all organisms that do not die while giving birth have some PRLS, this corrects for over-estimation of PRLS. I will apply the same principle in reverse: PRLS is significant only if it exceeds one inter-birth interval plus two standard deviations. Note that while some authors have considered reproductive cessation and menopause from the perspective of individual organisms (e.g. Gould, Flint & Graham, 1981; Walker, 1995), I use population averages, since one post-reproductive individual is not evidence of PRLS as a trait of a species. Alternatively, one might model the curve of reproductive senescence for a species and extrapolate age at zero fecundity, but this is not feasible for the data used in this study. 'Reproductive senescence' is *the decline in fecundity after its peak*, and is probably present to some extent in all iteroparous organisms that show gradual senescence (Finch, 1990).

'Post-reproductive lifespan (PRLS)' of a population can now be defined in two ways: *the time between average age at menopause and average age at death for those living beyond menopause*, or *the time between average age at reproductive cessation and average age at death for those living beyond reproductive cessation*. In addition to distinguishing between menopause and reproductive cessation as the onset of PRLS, we can also distinguish two measures of death as the end of PRLS, depending on whether the population is captive or wild. Using menopause in captive or domestic animals and reproductive cessation in wild animals provides two distinct measurements of PRLS. These might be referred to as 'intrinsic PRLS' and 'realized PRLS', respectively, and they will generally differ. The former measures physiological changes with age; the latter measures demography under natural conditions. Both indicate a disjunction between reproductive senescence and somatic senescence; the main distinction is that only realized PRLS could have direct fitness costs or benefits, so any explanation inferring direct fitness benefits of PRLS must be based on realized, not intrinsic, PRLS. At least in mammals, intrinsic PRLS appears to be longer, since the lengthening of the life expectancy observed in captivity is not accompanied by a lengthening of reproductive span (Ricklefs, Scheuerlein & Cohen, 2003).

Austad (1994, 1997) has pointed out that PRLS is well documented in captive mammals, but offers no specific explanation for this because, with a few exceptions, wild animals do not survive long enough to reach PRLS. He explains the presence of PRLS as a result of oocyte depletion in female mammals, timed in accordance with probability of survival. However, the presence of either intrinsic or realized PRLS begs explanation under standard life-history and senescence theory, which suggests that there are trade-offs between longevity and reproduction (Williams, 1957; Rose, 1984; Stearns, 1992). Thus, absent some mitigating selective force, survival beyond reproduction should be curtailed by selection for higher early reproduction. Even intrinsic PRLS demands an explanation, so for the purposes of this review, any indication of a disjunction between reproductive and somatic senescence will be considered PRLS.

Defining PRLS is simple enough; deciding on criteria for determining whether or not it is present in a given species is more problematic. What percentage of a population must live for how long beyond reproductive cessation or menopause for PRLS to be considered significant? Ultimately, as will be shown, the data are a continuum, so there is no objective criterion that can be applied, and any insertion of a cut-off point is arbitrary. However, several parameters may be considered: percentage of reproductively mature individuals reaching age at reproductive cessation or menopause, average length of PRLS for those individuals with some PRLS, and average per cent of lifespan that is post-reproductive. Further, the criteria should be less stringent for realized PRLS than intrinsic PRLS. In the wild, even a small percentage of individuals surviving beyond reproductive cessation demands explanation; in captivity, the null expectation is for curves to reach zero fecundity and zero survivorship simultaneously, but some deviation from that is expected stochastically.

In this study, I have drawn on a wide range of sources to obtain data on reproductive cessation in mammals. Most sources were found through citations in related articles; some were found through database searches on Web of Science or Biological Abstracts. Many of these studies are not themselves related to PRLS, and I have had to cull what data I could from them. I have included any study in Table 1 where the data could have been sufficient to indicate whether or not PRLS appears to be present, even if the result is inconclusive. In the text, I note several cases not included in Table 1 where data were insufficient to analyse. Because the data available are not uniform across studies, I have been unable to present even an arbitrary criterion for PRLS to apply across species. In most cases, however, the data are fairly clear, and I present them for each species, especially where the data are ambiguous. I have tried to present them as uniformly as possible in Table 1, while in the text below I discuss peculiarities of each study.

III. IS FEMALE PRLS UNIQUE TO HUMANS?

Many authors, especially anthropologists, have contended that PRLS is a unique feature of human life history (e.g. Pavelka & Fedigan, 1991; Hawkes, O'Connell & Blurton Jones, 2000). This contention is based on the much longer period of PRLS observed in humans than in other mammals, and comparative data from a few species are often cited to show the difference. However, in order to assess accurately whether or not human PRLS is unique, we need to look at a great number of species. Furthermore, it is not enough to say that PRLS in humans is much longer than in most other species: this is a quantitative, not a qualitative statement. That PRLS is not as obvious in other mammals as it is in humans does not mean that it does not exist, and the sorts of hypotheses needed to account for a quantitative *versus* a qualitative difference are quite distinct. Lastly, there has been debate as to whether PRLS in humans is an artifact of recent lifestyle changes. All these issues are addressed below.

(1) Humans

The first question here is, do humans really have PRLS, and if so how much? We know that in modern societies, women usually live well past menopause, but was this true for our remote ancestors? Two approaches have been taken to answer this question, and they lead to different conclusions.

Demographic analyses of PRLS in the !Kung and Ache, modern hunter-gatherers, show patterns similar to what we observe in modern society (Howell, 1979; Hill & Hurtado, 1996). Many women – as many as 80% of those reaching reproductive maturity – survive to reproductive cessation, and PRLS ranges from 20 to 30 years, depending on which population is surveyed and how PRLS is measured. If these hunter-gatherers are like our ancestors, it is clear that humans have always had a uniquely long PRLS. However, even the most primitive tribes for which we have data have been subject to some modern influences or may have evolved since prehistory. Blurton Jones *et al.* (2002)

Table 1. Data on post-reproductive lifespan (PRLS) in mammal species

Order	Scientific name	Common name	Sample size	Captive/ wild	Max. recorded life span (years)	Age at reproductive cessation (years)	% reaching reproductive cessation	Mean PRLS for those with PRLS (years)	PRLS	Sources
Artiodactyla	*Bos primigenius*	Cattle	NA	Domestic	30	~25	NA	5+	Yes	Smith & Robison (1931); Erickson *et al.* (1976); vom Saal *et al.* (1994)
Artiodactyla	*Cervus elaphus*	Red deer	Tens	Domestic	21+	17–18	Many in captivity	3+	Yes	Fisher *et al.* (1996)
Artiodactyla	*Ovis canadensis*	Bighorn sheep	265	Wild	19	16–18	<5	<2	Maybe	Bérubé *et al.* (1999)
Carnivora	*Canis domesticus*	Dog (beagle)	NA	Domestic	15	9	>50	>3.5	Yes	Anderson (1970*b*)
Carnivora	*Felis catus*	Cat	NA	Domestic	20	by 14	NA	NA	Yes	Marshall (1964)
Carnivora	*Panthera leo*	African lion	Hundreds	Wild	17	14	~15	1.8	Yes	Packer *et al.* (1998)
Carnivora	*Ursus maritimus*	Polar bear	~400	Wild	30	<23	1.50	~4	Yes	Ramsay & Stirling (1988)
Cetacea	*Balaenoptera acutorostrata*	Antarctic minke whale	Thousands	Wild	NA	NA	0	0	No	Marsh & Kasuya (1986)
Cetacea	*Balaenoptera borealis*	Sei whale	476	Wild	NA	NA	0	0	No	Marsh & Kasuya (1986)
Cetacea	*Balaenoptera physalus*	Fin whale	~1500	Wild	91	NA	0	0	No	Mizroch (1981)
Cetacea	*Globicephalus macrorhyncus*	Short-finned pilot whale	245	Wild	63	36	25	14	Yes	Kasuya & Marsh (1984)
Cetacea	*Globicephalus melaena*	Long-finned pilot whale	529	Wild	NA	NA	5	NA	Yes	Marsh & Kasuya (1986)
Cetacea	*Orcinus orcus*	Killer whale	211	Wild	78	40	~25	Decades	Yes	Olesiuk *et al.* (1990)
Cetacea	*Pseudorca crassidens*	False killer whale	30+	Wild	NA	41	NA	NA	Yes	Marsh & Kasuya (1986)
Cetacea	*Stenella attenuata*	Spotted dolphin	Hundreds	Wild	NA	NA	1	NA	Yes	Marsh & Kasuya (1986)
Cetacea	*Stenella longirostris*	Spinner dolphin	Hundreds	Wild	NA	NA	1	NA	Yes	Marsh & Kasuya (1986)
Lagomorpha	*Oryctolagus cuniculus*	Rabbit	NA	Domestic	15	5	NA	Several	Yes	Comfort (1979); vom Saal *et al.* (1994)
Perissodactyla	*Equus caballus*	Horse	NA	Domestic	45	by 42	NA	NA	Yes	Comfort (1979)
Primates	*Callithrix jacchus*	Common marmoset	14	Captive	10	NA	36	2	Yes	Caro *et al.* (1995)
Primates	*Gorilla gorilla*	Gorilla	12	Captive	30	NA	40	4.5	Yes	Caro *et al.* (1995)
Primates	*Homo sapiens*	Humans (Ache)	292	Wild	77	42	0.8	21	Yes	Hill & Hurtado (1996)
Primates	*Homo sapiens*	Humans (!Kung)	~500	Wild	88	35	~80	30	Yes	Howell (1979)
Primates	*Lemur* spp.	Lemurs (five spp.)	30	Captive	24	NA	45	3.5	Yes	Caro *et al.* (1995)
Primates	*Leontopithecus rosalia*	Golden lion tamarin	21	Captive	12	NA	47	4	Yes	Caro *et al.* (1995)
Primates	*Macaca fuscata*	Japanese macaque	Hundreds	Research colonies	~35	23–25	<40	4.5	Yes	Takahata *et al.* (1995); Nozaki *et al.* (1995); Fedigan (1991); Caro *et al.* (1995)
Primates	*Macaca mulatta*	Rhesus macaque	Thousands	Mostly in breeding colonies	35	23–25	~20	2.5	Yes	Dyke *et al.* (1986); Tigges *et al.* (1988); Johnson & Kapsalis (1995, 1998); Walker (1995); Caro *et al.* (1995)
Primates	*Macaca mulatta*	Rhesus macaque	38	Captive	20	NA	13	2.5	Yes	Caro *et al.* (1995)
Primates	*Macaca nemestrina*	Pig-tailed macaque	~3500	Breeding colony	30	<22	>15	~4	Yes	Ha *et al.* (2000)
Primates	*Macaca nemestrina*	Pig-tailed macaque	209	Captive	20	NA	26	4	Yes	Caro *et al.* (1995)
Primates	*Macaca radiata*	Bonnet macaque	26	Captive	20	NA	4	6	Yes	Caro *et al.* (1995)
Primates	*Macaca sylvanus*	Barbary macaque	207	Research colony	28+	21–23	~50	>5	Yes	Paul *et al.* (1993)
Primates	*Microcebus murinus*	Mouse lemur	NA	NA	14	11+	NA	NA	No	Finch & Sapolsky (1999)

Primates	*Pan troglodytes*	Common chimpanzee	Various	Mixed	65	40+	NA	NA	Maybe	Gage (1998); Nishida *et al.* (1990); Sugiyama (1994); Dyke *et al.* (1995)
Primates	*Pan troglodytes*	Common chimpanzee	15	Captive	48	NA	60	9	Yes	Caro *et al.* (1995)
Primates	*Papio cynocephalus*	Olive baboon	Hundreds	Wild	27	20–23	~12	~5	Yes	Packer *et al.* (1998)
Primates	*Papio cynocephalus/ anubis* hybrids	Baboon	13	Captive	16	NA	10	3.5	Yes	Caro *et al.* (1995)
Primates	*Pongo pygmaeus*	Orangutan	53	Captive	40+	NA	32	7	Yes	Caro *et al.* (1995)
Primates	*Presbytis entellus*	Hanuman langur	<100	Provisioned	35+	~28	NA	NA	Yes	Borries *et al.* (1991)
Primates	*Saguinus fuscicollis*	Saddleback tamarin	6	Captive	12	NA	20	4	Yes	Caro *et al.* (1995)
Primates	*Saguinus* spp.	Tamarins (two spp.)	NA	Captive	~20	<17	NA	NA	Yes	Tardif & Ziegler (1992)
Primates	*Saimiri sciureus*	Squirrel monkey	28	Captive	19	NA	32	3.5	Yes	Caro *et al.* (1995)
Proboscidea	*Loxodonta africana*	African elephant	800	Wild	60	~50+	~6	NA	Yes	Laws *et al.* (1975)
Rodentia	*Cricetulus griseus*	Chinese hamster	44	Laboratory	>21 months	~16 months	NA	NA	Yes	Parkening (1982)
Rodentia	*Mus domesticus*	Laboratory mice	NA	Laboratory	NA	NA	NA	NA	Yes	vom Saal *et al.* (1994); Bellamy (1981)
Rodentia	*Spermophilus columbianus*	Columbian ground squirrel	229	Wild	9	~7–8	NA	NA	No	Broussard *et al.* (2003)

NA, Not available.

effectively argue that it is unlikely these modern influences have skewed the data, but our poor knowledge of the history of such groups means that we cannot address more historical concerns. We will never know the extent to which selection has changed hunter-gatherers from the ancestral condition, or if perhaps these groups reverted to hunting and gathering after long years of agriculture.

By contrast, palaeodemographic analyses of ancient populations based on fossil remains show very few individuals surviving past 50 (Weiss, 1973; Lovejoy *et al.*, 1977; Willey & Mann, 1986; Trinkhaus & Thompson, 1987; Austad, 1994). In fact, almost no remains of individuals over 50 have ever been recovered from ancient populations. It is possible that lack of calcification in bones of the elderly resulted in poor preservation, but this appears to be true only in certain soil types (Willey & Mann, 1986; Walker, Johnson & Lambert, 1988; Mensforth, 1990). It is also possible that inaccuracies in ageing techniques are responsible, although these techniques have improved in recent years (Bocquet-Appel & Masset, 1982, 1996; Jackes, 1985; Meindl & Lovejoy, 1985).

How are we to reconcile these different indications of PRLS in our ancestors? The potential problems with each data set are not easily resolvable, but it seems likely that our ancestors had some sort of PRLS. Even if none of our ancestors lived past 50, there may have been intrinsic PRLS if they could have lived beyond 50 under better conditions. Or, menopause may have been earlier. And even 50 might be late enough for significant PRLS: average age at last birth in the !Kung is less than 35 (Howell, 1979). Finally, the fact that modern hunter-gatherers show significant PRLS means that some humans have significant realized PRLS in semi-'natural' conditions, even if we cannot prove this for our distant ancestors. So we can say with confidence that humans have a strikingly long and prevalent PRLS. But how unique is this?

(2) Non-human primates

The last 20 years have witnessed an intensive search for primate models of menopause (e.g. Gould *et al.*, 1981; Walker, 1995). Using both physiological and demographic techniques, reproductive parameters have been recorded for several species, primarily chimpanzees and various macaques. The results have been mixed. No primate has a PRLS approaching that of human females, even as a percentage of total lifespan. However, most species show a tendency towards cessation of reproduction ahead of somatic senescence. From a comparative perspective, primates represent only one mammalian order; however, because many of the best data are from primates, I deal with them first. Again, for simplicity of presentation, I have not used adjusted measures of reproductive cessation. I review the data for each species below.

Judge & Carey (2000) performed regressions of brain and body mass against lifespan for 133 primate species, suggesting that human lifespan falls in the normal primate range. They postulate origins of PRLS in the hominid lineage prior to evolution of *Homo sapiens*, although the absence of direct reproductive data on ancient populations is

problematic. No primate species yet studied shows a PRLS close to that of humans, either in absolute years or as a percentage of lifespan.

Probably the best-studied species is the rhesus macaque *Macaca mulatta*, due to the large populations kept for medical research at primate centres and to a provisioned population in the Florida Keys. A few individuals achieve lifespans of 30 to 35 years, female reproductive output drops precipitously after 20 years, and reproduction ceases altogether by 25 to 27 years (Dyke *et al.*, 1986; Tigges *et al.*, 1988; Pavelka & Fedigan, 1991; Johnson & Kapsalis, 1995, 1998; Walker, 1995). Physiological changes that parallel human menopause occur by ages 25 to 27, although a single birth was recorded at age 28 (Dyke *et al.*, 1986; Walker, 1995). Regardless, as many as 10 % of the female population may live to age 25 and cease cycling (Johnson & Kapsalis, 1998). Fertility rate peaks at around 0.4 births/female/year between ages 9 and 16, declines slowly until approximately age 22, and falls sharply thereafter. Out of a total fertility of 6.7 births from age 2 to 28, 1.2 (17 %) occur after age 20, and 0.6 (9 %) after age 22 (Dyke *et al.*, 1986). Many captive populations do not yield straightforward demographic data because individuals are sold for research or selectively removed. The only estimate of PRLS in the rhesus macaque, 2.5 years, is based on a small sample ($N = 38$); overall, 13.2 % of females in this population live past reproductive cessation (Caro *et al.*, 1995). Thus, reproductive senescence in rhesus macaques appears to occur slightly ahead of somatic senescence, resulting in a brief PRLS in most older females. Despite the attention rhesus macaques have received, however, it should be noted that survival past reproductive cessation was the fourth lowest out of 14 primate species analysed by Caro *et al.* (1995). In 10 of the 14 species, all in captive populations, more than 20 % of females survived past reproductive cessation (Caro *et al.*, 1995).

Japanese macaques *Macaca fuscata* show a similar pattern, based on provisioned research populations. One analysis of PRLS found that 41 % of 32 female Japanese macaques lived to age 20, and among these, the average PRLS was six years – 4.5 years after subtracting weaning time for the last offspring. This is 16 % of the total lifespan of these individuals, which lived to 27.3 years on average (Takahata, Koyama & Suzuki, 1995). By contrast, Fedigan (1991) claimed that Japanese macaques exhibit no PRLS. However, Fedigan (1991) used human menopause as a benchmark, and his data indicate that approximately 10 % of females live well past their last parturition. In a different sample of Japanese macaques, Pavelka & Fedigan (1999) found that 20 out of 70 females experienced reproductive cessation (following the definition of Caro *et al.*, 1995) before they died, with an average PRLS of approximately two years, or 9 % of total lifespan. Although authors have differed in their interpretation of the data, all of these studies agree that reproduction generally ceases in Japanese macaques by age 25, although a physiological equivalent of menopause is not reached until approximately age 27 (Nozaki, Mitsunaga & Shimizu, 1995). By this age, many macaques experience profound effects of somatic senescence, even though some may live into their thirties.

Chimpanzees *Pan troglodytes* are the closest living relatives of humans, and are often thought to provide the best estimate of the traits of the ancestor of modern humans (Hill & Hurtado, 1991; Hawkes *et al.*, 1998). Maximum reported lifespans range from 35 to 65 years in the literature (Gould *et al.*, 1981; Sugiyama, 1994; Dyke *et al.*, 1995; Gage, 1998; Hawkes *et al.*, 1998; Alvarez, 2000). Initial reports of menopause-like physiological changes in a few old individuals (Gould *et al.*, 1981) were later called into question, both because of small sample size and because the individuals examined exhibited other manifestations of senescence, so there was no evidence for reproductive senescence occurring ahead of somatic senescence (Pavelka & Fedigan, 1991). One individual in Gombe is 44 years old, pregnant for the ninth time, and looks in fine health. Seven of her eight previous offspring survived to adulthood (Anne Pusey, personal communication). The most reliable data indicating chimpanzee PRLS comes from observations of individuals in a provisioned population in the Mahale Mountains of Tanzania, in which older females regularly lived beyond their reproductive years, with one individual living for nine years after last parturition (Nishida, Takasaki & Takahata, 1990). This trend is contradicted by modeled demographic data, which show a sharp drop in reproduction close to the end of the lifespan (Gage, 1998). Because the inter-birth interval of chimpanzees is long (> 4.5 years) and infant mortality is high (Sugiyama, 1994; Hawkes *et al.*, 1998), chimpanzees may have been under strong selective pressure to evolve a correspondingly long reproductive span (Gage, 1998). Thus, data on chimpanzees produce no consensus as to the presence or absence of PRLS, and they may not be suitable demographic models for other primates.

Many other primate species have been looked at in less detail. Packer, Tatar & Collins (1998) considered data for olive baboons *Papio cynocephalus* in a test of the adaptive hypotheses. Because this study was conducted in the wild, it provides a measure of realized PRLS – in this case, approximately 7 % of the population reached age 21, after which reproduction declined sharply. Life expectancy was an additional five years. Studies on captive or provisioned populations of Barbary macaques *Macaca sylvanus* and pigtailed macaques *Macaca nemestrina* show some PRLS (Paul, Kuester & Podzuweit, 1993; Ha, Robinette & Sackett, 2000) (see Table 1). A provisioned wild population of Hanuman langurs *Presbytis entellus* showed reproductive decline well before maximum lifespan, perhaps due to social dominance traits, but data on length and prevalence of PRLS are not available (Borries, Sommer & Srivastava, 1991). Two species of tamarins show a physiological equivalent to menopause around age 17, and have a maximum lifespan of approximately 20 years, but again there are no data on survivorship to reproductive cessation (Tardif & Ziegler, 1992). Additionally, the study mentioned above by Caro *et al.* (1995) compares PRLS in captive populations of 14 primate species, although mostly with small sample sizes. Only vervet monkeys *Cercopithecus aethiops* show no PRLS (perhaps due to small sample size, $N = 12$); in all other species, there is evidence for at least some PRLS, even after adding one inter-birth interval plus two standard deviations to the age at

last parturition. Ten of the 14 species had at least 20% of individuals survive past this adjusted age at reproductive cessation. Caution is urged with these estimates, however; often the maximum lifespans reported are much lower than other reports, and it is not clear to what extent selective breeding affected these patterns. One mammal that appears to lack a post-reproductive period is the mouse lemur *Microcebus murinus*, which has been observed to maintain fertility even in the last year of life. Curiously, certain prosimians (but not the mouse lemur) are the only mammals known to exhibit *de novo* oogenesis as adults (Finch, 1994; Finch & Sapolsky, 1999).

(3) Non-primates

Female short-finned pilot whales *Globicephalus macrorhyncus* do have a significant PRLS, often living on average 14 years after their last birth, based on histological examinations (Kasuya & Marsh, 1984). In one pod from which 245 females were examined, 24% of the females were post-reproductive. Females cease reproduction by age 40 at the latest (oldest recorded age at birth = 36 years) but live up to 63 years, while the oldest recorded male was 46 years old and reproductively active. Interestingly, although most calves are weaned by age 7 and many by age 3, a few suckle for up to 15 years, hence investment in existing offspring could be important in this species (Kasuya & Marsh, 1984). There are indications that some (but not all) other toothed whales may have a similar life history. The long-finned pilot whale *Globicephalus melaena*, for instance, appears to have only 5% of mature females post-reproductive – a significant proportion in the wild, but nothing like that of the short-finned pilot whale (Marsh & Kasuya, 1986). Olesiuk, Bigg & Ellis (1990) provide strong demographic (but not histological) evidence for a long, prevalent PRLS in killer whales *Orcinus orcus*, on a par with that observed in short-finned pilot whales. Curiously, the only mammals that appear to exhibit no reproductive senescence whatsoever are also cetaceans: the baleen whales. A more-or-less constant percentage of fin whales *Balaenoptera physalus* was pregnant at ages from 10 to nearly 90 years, with no evidence of an age-related decline, and this appears to be typical for baleen whales, although there is sporadic evidence of a slight decline in reproduction in a few species (Mizroch, 1981; Marsh & Kasuya, 1986; Aguilar & Borrell, 1988). However, there is no clear indication that baleen whale lifespan is shorter than 100 years, so it is hard to know how to interpret the lack of a decline in reproduction.

In the study on olive baboons mentioned above, Packer *et al.* (1998) also found that female lions *Panthera leo* exhibit PRLS, and that grandmothers' reproductive status had no effect on survival of young. Roughly 3% of individuals survived to age 14, when fecundity declined sharply, and life expectancy then was an additional 1.8 years. Bighorn ewes *Ovis canadensis* show reproductive senescence after age 14, although somatic senescence may begin as early as 10. Thus, this species does not show accelerated reproductive senescence with respect to somatic senescence. However, several of the oldest individuals in the study (which did not directly address PRLS) were apparently barren; without additional data no conclusion can be drawn (Bérubé, Festa-Bianchet & Jorgenson, 1999). Out of a sample of 800 African elephants *Loxodonta africana* (male and female), reproduction had ceased in 12 of 20 females between 51 and 55 years of age and all eight females 56 and older. In the sample, 62% of females were sexually mature, yielding an estimate of 6% of adult females having ceased reproduction (Laws, Parker & Johnstone, 1975). Out of approximately 400 polar bears *Ursus maritimus*, 30 years was the maximum observed lifespan, and none of the six individuals (1.5% of the sample population) over age 20 years were observed with cubs, having on average four years of PRLS (Ramsay & Stirling, 1988). In Columbian ground squirrels *Spermophilus columbianus*, females can live up to nine years in the wild, but reproduction falls off sharply after age five. However, even some of the oldest individuals were observed to reproduce, so there appears to be early reproductive senescence without clear PRLS (Broussard *et al.*, 2003). One wild post-reproductive ringed seal *Phoca hispida* was observed – anecdotal yet suggestive evidence in this species (McLaren, 1958).

Domesticated and laboratory animals also exhibit PRLS. In recently domesticated red deer *Cervus elaphus* in New Zealand, 19 out of 40 hinds survived to at least age 20, even though reproduction fell dramatically after age 17 and decreased to close to zero within a year or two (Fisher *et al.*, 1996). Similar patterns are observed in cattle, and perhaps in horses, rabbits, and cats (Smith & Robison, 1931; Marshall, 1964; Turner & Dolling, 1964; Erickson, Reynolds & Murphree, 1976; Comfort, 1979; vom Saal *et al.*, 1994). Chinese hamsters *Cricetulus griseus* typically have their last litters at an average age of 16 months, but often live for several more months. During this time, many animals miscarry, but exogenous progesterone supplements can induce successful pregnancies. In contrast to primates and whales, the ovaries of reproductively terminated hamsters have viable oocytes (Parkening, 1982). Many strains of laboratory rats and mice exhibit PRLSs of varying length, as do wild-caught mice (Bellamy, 1981; vom Saal *et al.*, 1994). In laboratory beagles *Canis domesticus*, median lifespan was 12.5 years and maximum lifespan was 15, but reproduction declined after age 5 and was almost zero by age 10 (Anderson, 1965, 1970*a*; Anderson & Rosenblatt, 1965).

(4) Conclusions of the generality of PRLS in mammals

One interesting question that arises from the above patterns of PRLS in female mammals is whether length of PRLS is correlated with lifetime reproduction. For example, if sexually transmitted disease or strain of childbirth is a factor in reproductive senescence, we might expect that those individuals with highest reproduction cease reproduction earlier and thus have longer PRLS. Unfortunately, most of the above data are cross-sectional rather than longitudinal, and do not include information on lifetime reproduction. An anecdotal counter-example is available: the oldest known reproductive female chimpanzee is also the one with the most offspring (Anne Pusey, personal communication).

Males of all mammal species studied show some decline in reproductive output with age, but are generally able to reproduce at a significant fraction of their maximum potential right up to death (vom Saal *et al.*, 1994). In particular, male laboratory rodents and humans may show declines in fecundity, but this appears to be due to ageing of various systems peripheral to reproduction rather than to inescapable ageing of the reproductive system itself; only rarely does the reproductive system fail completely. In both elephants and short-finned pilot whales, it has been confirmed that males are physiologically capable of reproduction at the oldest ages (Kasuya & Marsh, 1984; vom Saal *et al.*, 1994). Most of the data on females presented above are demographic; the literature on mammalian reproductive physiology is in strong agreement that in female mammals reproductive senescence occurs earlier and more predictably than in males, and that it occurs ahead of somatic senescence (e.g. Anderson, 1970*b*; Finch, 1994; vom Saal *et al.*, 1994; Finch & Sapolsky, 1999; Armstrong, 2001).

Taken together, the physiological and demographic data seem to indicate fairly conclusively that most mammals studied show accelerated female reproductive senescence with respect to somatic senescence. Out of 42 species for which there were sufficient data, 35 had at least weak evidence for PRLS, two were inconclusive, and five appear not to have PRLS. In five species, there is strong evidence for at least 5% of individuals having significant post-reproductive lifespan in the wild. Out of 18 extant orders of eutherian mammals, all eight for which I could find data have shown some evidence of PRLS. This is not to suggest that PRLS is uniform among primates or mammals; indeed, there appears to be great variation among taxa in this trait, even if it is present to some extent in most species. Further, while a few species show clear realized PRLS, for many the evidence indicates only intrinsic PRLS, so adaptive explanations may not be relevant. Even within species, there appears to be a great deal of plasticity in reproductive senescence (Finch, 2002), perhaps accounting for the conflicting data on chimpanzees.

IV. THE ADAPTIVE HYPOTHESES

Adaptive hypotheses have been the only viable explanation for the evolution of PRLS to date; however they have not gone unchallenged. Empirical support for such hypotheses comes mainly from evidence of high productivity among grandmothers in one African tribe, the Hadza (Hawkes, O'Connell & Blurton Jones, 1989), and from evidence of increased offspring health in rural Gambia when the maternal (but not paternal) grandmother is post-reproductive (Sear, Mace & McGregor, 2000). Additionally, some models suggest that the allometry of life-history variables makes human data consistent with both general primate patterns and adaptive hypotheses (Hawkes *et al.*, 1998; Alvarez, 2000). Specifically, correlations between annual fecundity and age at maturity, size at weaning and size at maturity, and age at maturity and mortality are established for primates. Human data are shown to fit the correlations only when reproductive output is calculated to include post-menopausal contributions of grandmothers. The problem with such models is that empirical support shows compatibility, but does not address causality. That is, such models do not test adaptive hypotheses directly; they simply indicate whether or not they are consistent with life-history allometries. Failure to conform would not disprove the hypotheses, since such allometries would not necessarily hold if there was strong selective pressure for them to vary; indeed, these allometries are hardly invariant across all species. It is also unclear to what extent other hypotheses might explain patterns of allometry. In the absence of data or models showing that benefits of PRLS outweigh costs, such allometry models provide little insight.

Evidence against adaptive hypotheses is also scanty. Hill & Hurtado (1991) took data from the Ache and applied it to a cost/benefit model of the hypotheses. They found that the benefit of PRLS did not outweigh the cost of lost reproductive opportunity except under the most lenient assumptions, when it did so marginally. Rogers (1993) came to the same conclusion with similar models fitted to a Taiwanese population. In both cases, the cost was lost reproductive opportunity after menopause; the benefit was improved reproductive output of existing offspring. However, such models are limited in their ability to incorporate all relevant variables, most notably an exponential increase in death during childbirth with age (Mace, 2000). Furthermore, Alexander (1990) proposed an adaptive hypothesis that suggests that grandmothers contribute not primarily through 'babysitting' but by using their increased status, influence, and wealth to benefit all kin. The benefits of menopause under such a model would be particularly hard to quantify, but might be substantially higher than those for babysitting alone. Thus, while the models of Hill & Hurtado (1991) and Rogers (1993) notably fail to confirm adaptive hypotheses, they do not falsify them.

A recent model by Shanley & Kirkwood (2001) suggests that combined effects of many benefits of PRLS may be sufficient to justify PRLS between age 50 and 60 in humans. This model incorporates four benefits of PRLS: decreased risk of death during childbirth, increased survival of existing offspring, increased fertility of existing offspring, and increased survival of grandoffspring. Individually, none of these components has sufficient benefit to explain PRLS, but in a combined model, fitness of the population is maximized when menopause occurs between 50 and 60, with a slight decrease in fitness for menopause at later ages. While some aspects of this model are quite convincing, no model yet developed has incorporated an effect of changes in age at menopause on reproduction earlier in life. It is unlikely that selection works on age of menopause without concomitant effects on other life-history traits (see below), so the applicability of these models is not clear. Additionally, it is unclear why there should be a discrepancy between the model of Shanley & Kirkwood (2001) and those of Rogers (1993) and Hill & Hurtado (1991). All portions of the model by Shanley & Kirkwood (2001) are incorporated into the earlier models in one form or another; furthermore, the most important portion of the Shanley & Kirkwood (2001) model, death during childbirth, is explicitly stated as being negligible in each of the other two studies. Hill & Hurtado

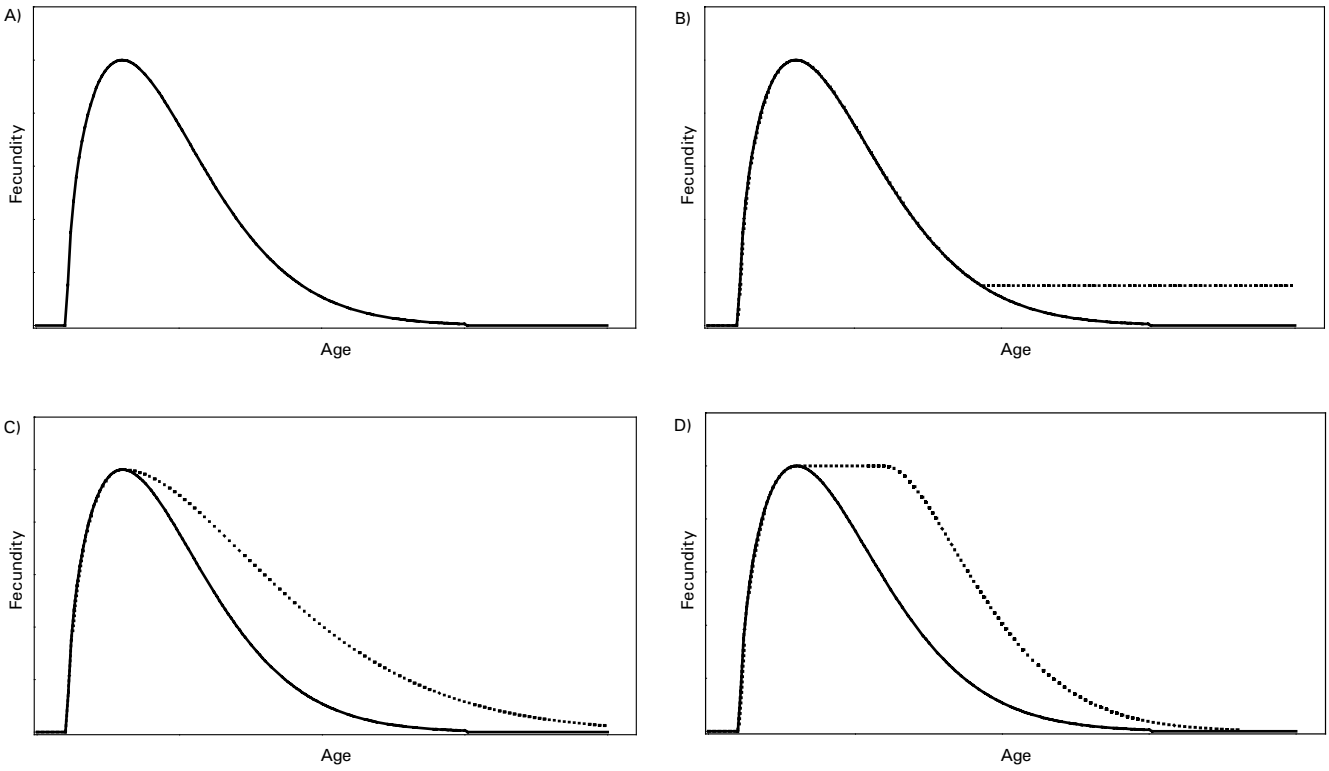

Fig. 1. Alternative models for calculating cost of lost reproduction in an animal with post-reproductive lifespan (PRLS) based on a comparison between fecundity with PRLS and hypothesized fecundity without PRLS. The solid line represents fecundity with PRLS. The dotted line represents alternative hypotheses for fecundity without PRLS. The area under a given curve is lifetime reproductive output. The difference in area between the two curves is the reproductive cost of PRLS. (A) A possible function of fecundity in an animal with PRLS. (B) The cost of PRLS as calculated in most of the literature – continuation past menopause at the level just prior to menopause. (C) Fecundity declines more slowly than with PRLS, but decline starts at the same time. (D) Fecundity declines at the same rate as with PRLS, but starts to decline later. The cost of lost reproduction (i.e. the difference in area between the curves multiplied by the survivorship curve) is much higher in C and D, which are the more realistic hypotheses.

(1991) do not include death during childbirth because its effects are negligible even when estimated at ten times the rate observed among the Ache and five times the highest rates ever observed anywhere (Hill & Hurtado, 1991). Rogers (1993) presents a model explicitly dealing with death during childbirth, and finds that even with lenient assumptions, the effect would have to be approximately 30 times greater than it is to account for PRLS. The unknown parameter here is how much death during childbirth would increase with age after 50 (Mace, 2000). Such mortality may be low enough to have little effect on selection before 50, but depending on the rate of increase assumed, the benefit of PRLS could either be very large or very small.

By contrast, there is strong empirical evidence against adaptive hypotheses in nonhuman mammals. In an elegant study, Packer *et al.* (1998) found no evidence for contributions of grandmothers to grandchild fitness among lions or baboons, two species in which adaptive hypotheses would have predicted such effects based on social system. Both species had individuals with PRLS (although relatively short), and a comparison of the survival of the grand-offspring of these post-reproductive individuals with that of still fecund individuals showed no significant difference. The authors suggested that the observed PRLS might have evolved to allow mothers to wean their last offspring. Fedigan & Pavelka (2001) conducted a similar analysis of data on Japanese macaques. There was no indication of any benefit of PRLS to offspring or grand-offspring, except that the last offspring had a better chance of survival. This did not translate into a lifetime benefit to mothers with PRLS, and is probably due to all post-reproductive mothers surviving long enough to wean their last offspring.

Perhaps the strongest argument against the adaptive hypotheses is that the proposed trade-off between future reproduction and current offspring has been misconstrued. The major cost of PRLS probably is not loss of reproductive time at the end of the reproductive lifespan, but rather the loss of reproduction earlier in life. This is shown graphically in Fig. 1. In order to calculate lost reproduction, a hypothesis of fecundity rates in women without PRLS is necessary. Traditionally, this has simply been calculated as if women reproduced until their death at the level just prior to menopause (Hill & Hurtado, 1991; Rogers, 1993; Shanley & Kirkwood, 2001) (Fig. 1B). More realistically, the curve relating fecundity to age can be extended by having fecundity decline later or more slowly (Fig. 1C, D). As a

consequence, PRLS results in a loss of reproduction during earlier reproductive life at a high cost to individual fitness. The benefit to survival and reproduction of offspring required to offset this cost is high – women must be more productive as grandmothers than they were in their reproductive prime.

More generally, the problem with most thinking on the subject to date is that menopause is viewed as a moment in time when reproduction ceases, and which can be adjusted as necessary to fit selective pressure. However, menopause is apparently a result of physiological processes set in motion far earlier (vom Saal *et al.*, 1994), and selection most likely acts on the entire curve of fecundity with age. Any hypotheses about post-reproductive lifespan must address shifts in the curve, not simply the point of menopause or reproductive cessation. Given the gradual decline in fecundity of human females from 35 to 50 (Hill & Hurtado, 1996), there is no reason to believe that any special process has caused accelerated loss of fecundity or a truncation of reproductive output.

Data on non-human mammals presented above requires that we adjust our thinking about the evolution of PRLS in humans. The contributions of grandmothers to the number of their descendants may have made human PRLS as long as it is, but PRLS clearly did not evolve *de novo* during the course of human evolution. The phenomenon arose much earlier in the evolution of mammals or their ancestors. However, PRLS may well have been extended during the course of human evolution by an increase in overall lifespan, consistent with Hawkes *et al.*'s (1998) suggestion. If PRLS predated 'grandmothering', the cost of extending PRLS may have been relatively low, involving an extension of lifespan rather than a reduction in fecundity. Again, evidence for realized (as opposed to intrinsic) PRLS in humans provides some support for such a hypothesis. Until we have a clearer understanding of the origins of PRLS and the costs of extending lifespan in general, it will be hard to assess the extent to which grandmothering or other kin-related behaviours have contributed to the length of human PRLS. In light of the apparent generality of PRLS among mammals, any hypothesis on its origin should either be based on mammalian physiology and developmental constraints, or on shared features of the mammalian selective regime. Below I propose a tentative hypothesis for the evolution of PRLS based on physiological constraints.

V. A HYPOTHESIS FOR THE ORIGIN OF PRLS IN MAMMALS

The data presented above do not imply that PRLS is unique to mammals; in fact, a preliminary analysis of demographic data from zoos shows that it may be present in many birds as well, although it may be most apparent in female mammals (Ricklefs *et al.*, 2003). The hypothesis proposed here has both a broad and a specific form. The broad form, based on trade-offs and life-history theory, could explain the origin of PRLS in almost any organism, while the specific form, based on physiology, is unique to female mammals (and possibly female birds).

(1) The broad form of the hypothesis

Life-history theory suggests that trade-offs between conflicting beneficial traits are a driving force in evolution (Stearns, 1992). In particular, there is a growing body of evidence suggesting that senescence is in large part due to antagonistic pleiotropy, when genes have multiple effects, both positive and negative, on an organism (Williams, 1957; Hamilton, 1966; Rose, 1984; Kirkwood & Austad, 2000). The trade-off generally considered most prominent in ageing is that between reproduction and longevity, with an increase in early reproduction generally resulting in a shortened lifespan, and *vice versa*. This has been well documented in selection experiments on *Drosophila melanogaster* (e.g. Rose, 1984). Apparently, the demands of reproduction result in decreased ability to maintain the soma and/or greater risks. This effect may be even more prominent in long-lived organisms like vertebrates, in which somatic maintenance systems are highly developed (Finch, 1990, 2002). As a hypothetical example, an increase in testosterone levels might increase reproductive behaviour, increasing both exposure to predators and susceptibility to disease through immunosuppression (Raouf *et al.*, 1997). Activity levels would also increase, resulting in higher metabolic expenditure. Diseases contracted might then require a more extreme immune response, resulting in both auto-toxicity and further metabolic expenditure. Metabolic expenditure in turn would result in oxidative damage and accumulation of advanced glycosylation endproducts. All of these factors might result in an accelerated ageing process. Whether or not evolution favoured such an increase in testosterone levels would depend on whether the reproduction gained due to increased reproductive effort outweighed that lost due to a shortened lifespan.

Because of this trade-off between longevity and fecundity, the expectation has generally been that selection should act to synchronize the end of reproduction with the end of the lifespan. This is because there is no reproductive value in one without the other, so any residual amount at the end of life is a waste of energy or resources. Through trade-offs, this residual amount should be convertible into other forms of reproduction. For example, if reproduction finished before the end of the lifespan, the expectation would be that selection would work to shorten the lifespan because, through trade-offs, it would concomitantly increase earlier reproduction. It is because of this expectation of synchronization that PRLS has been perceived as paradoxical.

However, there is not necessarily any reason to expect synchronization between the end of lifespan and the end of fecundity. If reproductive senescence and somatic senescence are subject to independent selection (Fig. 2), it will be possible, depending on the trade-offs involved, for a disjunction between reproductive cessation and the end of the lifespan to evolve. In other words, if there are separate physiological processes that determine the end of reproduction and the end of lifespan, then even if these two physiological processes are linked either through trade-offs or through partial overlap of physiological systems, selection should be able to extend lifespan without extending reproductive span, or *vice versa*. If somatic and reproductive

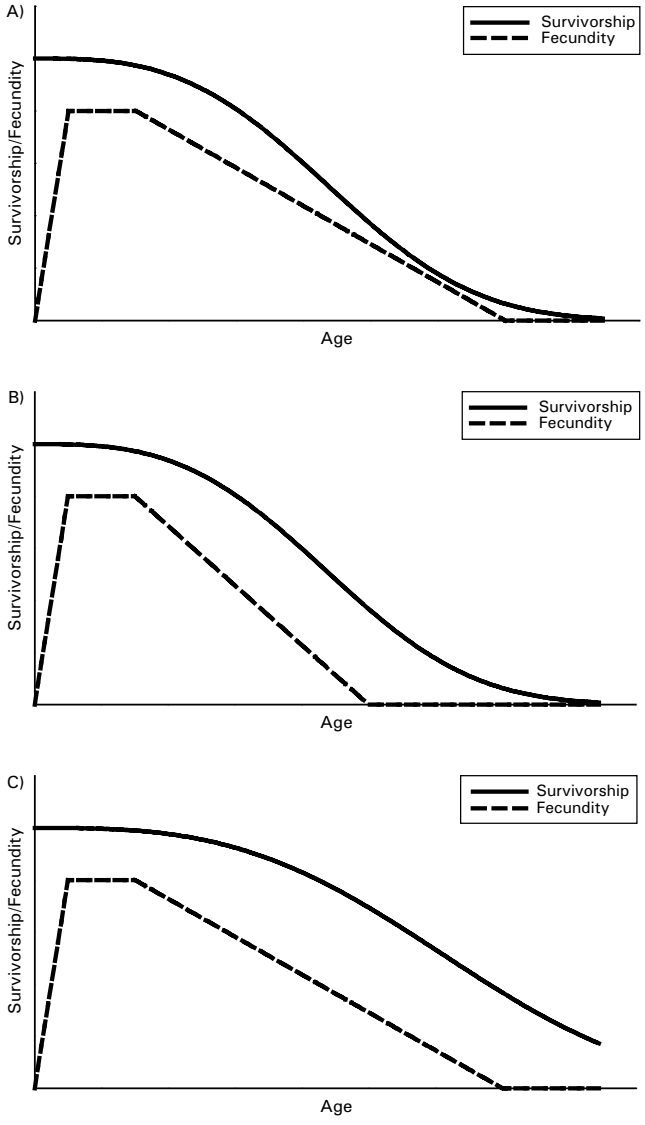

Fig. 2. Independent selection on fecundity and survivorship curves. (A) The end of reproduction (fecundity = 0) and the end of the lifespan (survivorship close to zero) occur at approximately the same time. (B) Because of separate physiological processes determining the senescence of each system, selection can act to hasten reproductive decline with minimal effect on somatic senescence. (C) Survivorship is extended with little effect on reproductive decline. B and C are alternative models for how post-reproductive lifespan (PRLS) might evolve. Not shown but possible are foreshortening of survivorship with constant fecundity and extension of fecundity with constant survivorship relative to A.

senescence respond somewhat independently to selective pressures, selection will still act to synchronize fecundity and survivorship curves earlier in life such that survivorship and fecundity are high during the prime of reproduction, but this will not necessarily result in synchronicity at the end of the curves.

It is helpful to consider these concepts graphically (Figs 1–3). We can plot survivorship on a linear scale (in contrast to the traditional logarithmic scale) and lay it over a plot of individual fecundity. Individual fecundity is the average fecundity of an individual alive at a given age. In Figs 2 and 3, I have used a Weibull model of survivorship and a simple three-stage model of individual fecundity, with a linear increase, a flat plateau, and a linear decrease. The product of these curves gives population fecundity, the individual fecundity at a given age multiplied by the probability of surviving to that age. Population fecundity can also be thought of as age-specific force of selection. The trade-off between longevity and reproduction can thus be conceptualized as changes in the survivorship curve that affect the fecundity curve, or *vice versa*: the extension or heightening of one is accompanied by the foreshortening or lowering of the other, at least to some extent. Selection will maximize the area under this population fecundity curve, which is proportional to the fitness of the population. Since the area under the peak is much greater than the area under the tails, especially the right tail, a small change in fecundity at the peak could have a large effect, while changes in the tail might have less effect.

Fig. 3, in addition to presenting a possible model for non-linear dynamics of trade-offs, shows how maximizing fitness could result in a disjunction between the end of reproduction and the end of the lifespan. The model presents a threshold trade-off model, such that changes in fecundity have minimal effect on survivorship unless they cross a certain threshold. Fig. 3A shows the maximally fit scenario. Since changes in peak fecundity level above or below the threshold have little effect on survivorship, in Fig. 3B, a decrease in fecundity relative to Fig. 3A produces little survivorship gain, and thus reduced fitness. In Fig. 3C, a slight increase in fecundity has exceeded some physiological threshold, and results in a large reduction in survivorship and decreased fitness overall. However, above this threshold, survivorship is once again relatively insensitive to changes in peak fecundity, so fitness can be improved by increasing fecundity, as in Fig. 3D. The population fecundities under each scenario are compared in Fig. 3E, with the area under the curve being proportional to the fitness of the population. Note that the disjunction between the end of reproduction and the end of lifespan is much larger in Fig. 3A than in Fig. 3C, because in order to maintain high enough survivorship during prime reproduction to maximize fitness, the survivorship curve must be extended well beyond the end of reproduction.

This model is not meant to represent a specific example in nature, but merely to illustrate how different cost/benefit trade-off scenarios might create different relationships between survival and fecundity. A threshold model is only one possible model; the trade-offs might be linear, with an increase in fecundity accompanied by a proportional decrease in survivorship; or they might be exponential, with successive increases in fecundity accompanied by increasingly large or small decreases in survivorship. More likely, the actual functions are determined by the complex interactions of many physiological systems, and thus may incorporate elements of many types of functions.

So, assuming independent selection on survival and fecundity curves, and depending on the nature of the

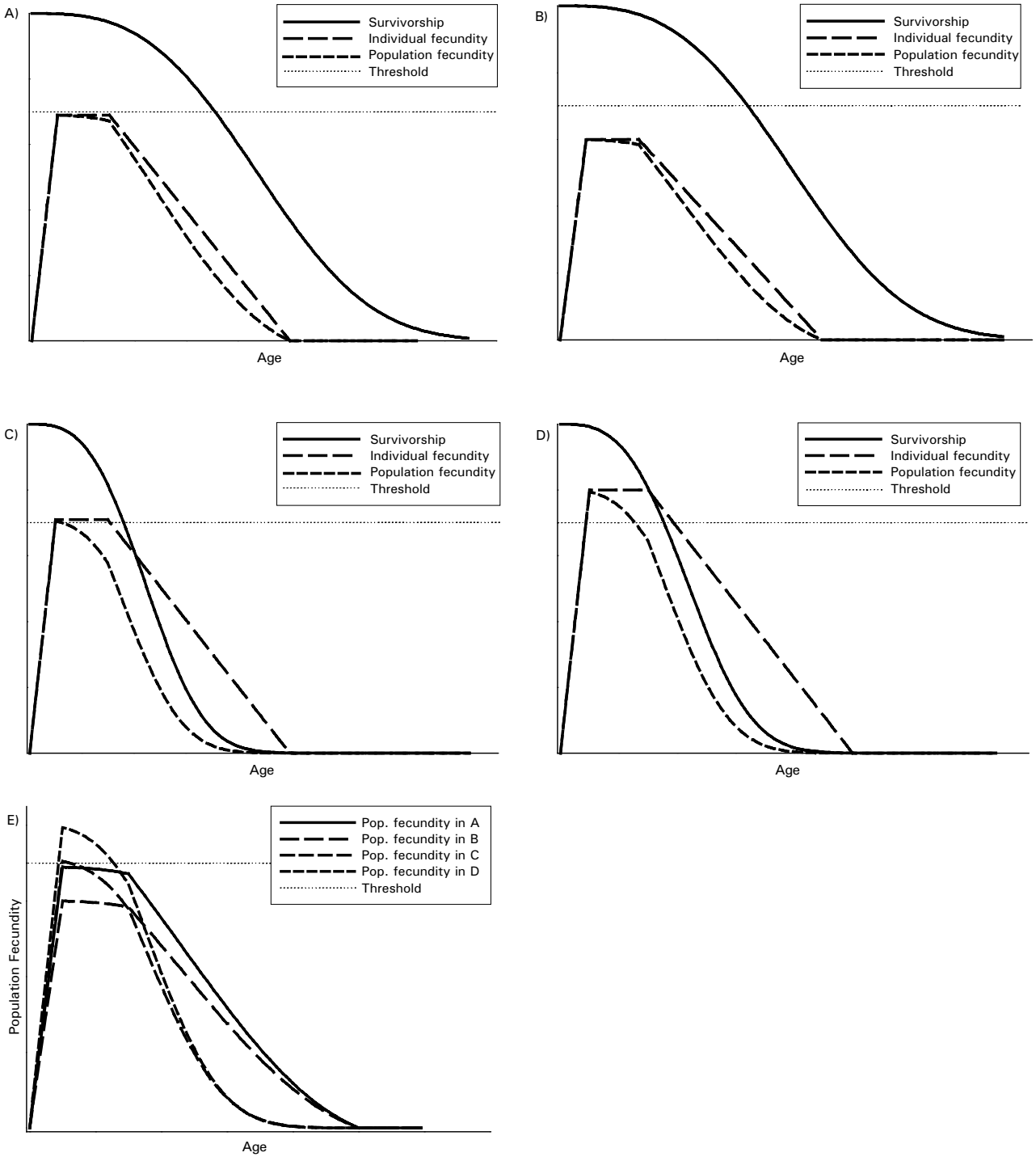

Fig. 3. Possible non-linear dynamics of trade-offs. In A–D, a Weibull model of survivorship is plotted on a linear scale with a three-stage model of individual fecundity: linear increase, flat plateau, and linear decrease. The product of these, population fecundity, is also plotted, and shows force of selection and relative fitness. Each figure shows how a change in maximum fecundity might affect survivorship under a threshold model of trade-offs. (A) The maximally fit scenario, with fecundity just under the threshold. (B) Below the threshold, decreasing fecundity fails to increase survivorship significantly. (C) A slight increase in fecundity relative to A crosses the threshold, resulting in a sharp reduction in survivorship and decreased fitness. (D) Above the threshold, survivorship is again insensitive to changes in fecundity, so fitness can be increased relative to C by increasing fecundity. (E) The population fecundities of the above four scenarios plotted together to show relative fitnesses.

trade-offs involved, we have a likely disjunction between the end of reproduction and the end of the lifespan. It is not difficult to see why this might systematically result in PRLS across a large taxon. For example, if the physiology of the reproductive system were such that reproductive senescence, once started, proceeded quickly, while survivorship, once extended beyond a certain point, tended to decline slowly, then PRLS would be expected. Alternatively, if there was a particularly high cost to extending reproductive span or a low cost to extending lifespan, the same disjunction might result. And if the physiology that determined the trade-offs involved was similar for related taxa, one might see the same pattern throughout an entire class, such as mammals.

There is an analogy here in automotive engineering. It is said that Henry Ford went with his engineers to a junkyard to see what part of his cars lasted longest. It turned out that crankshafts were always in good shape. Henry Ford's response? 'We must invest less money in our crankshafts. It's a waste if they outlast everything else.' This story has been told to illustrate why selection should act to synchronize the end of reproduction with the end of the lifespan. But perhaps Henry Ford was wrong. Different parts wear in different ways, and perhaps the crankshaft, if made only slightly more cheaply, would have consistently failed before the rest of the car. In this case, it might have been best to invest a little more in the crankshaft, even though it ended up sitting useless in the junkyard. As with the different systems in a car, different biological systems may age/wear more or less in concert as the entire organism ages, yet be subject to different specific trade-offs and patterns of wear because of their specific physiologies. Trade-offs need not be linear.

An important question here is whether or not there are age-specific mutations that can affect the curve of somatic senescence alone. If there are age-specific mutations, the shape of the tail can be affected by deleterious mutations that accumulate due to mutation-selection balance under the weak selection at older ages. If there are not age-specific mutations, then any change in the curve must come as a whole – any decrease in longevity would be accompanied by an increase in early vigour, or *vice versa*. Currently, there is debate as to whether there are age-specific mutations in *Drosophila melanogaster* (Charlesworth & Hughes, 1996; Rose *et al.*, 2002). If age-specific mutations are widespread or severe, such mutations should eventually curtail lifespan to the end of reproduction, unless correlated effects between the sexes mitigate this. Potentially, older male mammals may still be reproductively active enough for selection to suppress accumulation of mutations that affect both sexes.

This is the broad form of the hypothesis. For it to appropriately explain the origin of PRLS, several conditions must be met. First, reproductive and somatic senescence must be subject to independent selective pressures. Second, these pressures must result in faster or earlier reproductive senescence relative to somatic senescence. Third, mutation accumulation must not be overly severe. If these criteria are met, PRLS should be observed. Unfortunately, the third criterion may be difficult to assess in most cases, but the first two at least should be testable. How they apply in mammals is the specific form of the hypothesis.

(2) The specific form of the hypothesis

There are two lines of evidence showing that reproductive and somatic senescence appear to be independent in female mammals. By independent, I am not referring to a statistical property but to the ability of selection to act separately on each trait, even if the traits might be correlated in the absence of contrary selective pressures. Physiological evidence shows different mechanisms for somatic and reproductive senescence; inter- and intra-specific variation shows that selection actually has acted separately. Evolutionarily, many of the species for which data is cited above show distinct patterns, even compared to closely related species. For example, the lifespan of humans is much longer than for chimpanzees, even though female chimpanzees and humans both cease reproduction in their forties. Apparently selection was able to act on lifespan without much effect on female reproductive span. Also, PRLS is much longer in short-finned pilot whales than long-finned pilot whales, and lifespan is much longer in female than male short-finned pilot whales (Kasuya & Marsh, 1984; Marsh & Kasuya, 1986). Similarly, a variety of patterns are observed among macaques, among different species of tamarin, and among strains of mice. While the quality of the data assembled here is not high enough to perform a statistical analysis to show independence of reproductive span and lifespan, the presence of even a few species pairs in different orders which demonstrate markedly different patterns of PRLS is sufficient to indicate evolutionary plasticity of reproductive cessation and lifespan relative to each other. Ideally, there would also be data from selection experiments showing such a pattern; however, there is no reason to expect this pattern except in mammals (see below), and to my knowledge the relevant experiments in mice have not been done. Any other mammal species would have prohibitively long generation times.

Physiologically, there appears to be a mechanism for the ageing of the female reproductive system that is largely independent of somatic ageing. Oocyte number is fixed at birth in mammals, and declines exponentially throughout reproductive life (vom Saal *et al.*, 1994; Gosden & Faddy, 1998; Armstrong, 2001). At least in humans, this decline accelerates to a higher rate near the end of reproduction (Fig. 4) (Richardson, Senikas & Nelson, 1987). Reproductive cycling is controlled hormonally by the oocytes; when numbers fall below a threshold, not enough signal is produced to induce the cycle (vom Saal *et al.*, 1994; Armstrong, 2001). Usually, the supply is more or less exhausted by this point anyway, although there is great variation among strains of mice in number of oocytes left at menopause (Bellamy, 1981). In humans, this occurs when there are approximately 1000 oocytes remaining (Gosden & Faddy, 1998). Although individual cells of the ovaries are probably subject to the same cellular ageing as the rest of the soma, it is unlikely this plays a strong role in oocyte attrition, since oocyte numbers have already been halved by puberty, though oocyte senescence may play a role in the

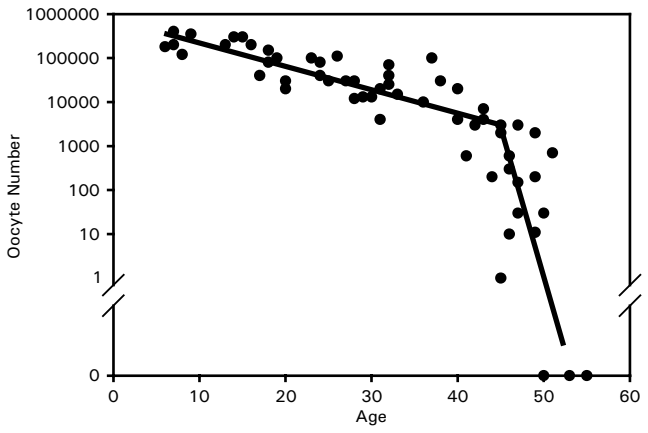

Fig. 4. Accelerated exponential decline in oocyte number of human females with age (Richardson *et al.*, 1987).

acceleration of this decline late in life (vom Saal *et al.*, 1994). Rather, gonadotropin-induced increases in luteinizing hormone appear to accelerate oocyte attrition, although the reasons are poorly understood (Jones & Krohn, 1961; Flaws *et al.*, 1997). Oocyte loss does not, however, appear to explain reproductive senescence completely. For example, melatonin supplements reduced irregular oestrous cycles in older rats without affecting the number of primary ovarian follicles (Meredith *et al.*, 2000). Ageing of individual oocytes also appears to play a role in viability of embryos, among other things, and the endometrium becomes progressively less able to accept implantation (Armstrong, 2001).

Somatic senescence, by contrast, appears to be largely dictated by various cellular ageing processes. Cellular respiration results in oxidative damage to DNA, which eventually leads to the loss of some cells and occasionally to the formation of tumours (Albarrán *et al.*, 2001; Corsolini *et al.*, 2001; Holmes, Fluckiger & Austad, 2001). Accumulation of advanced glycosylation endproducts formed by interactions between glucose and various proteins also can lead to cellular and tissue senescence and phagocytosis by macrophages (Vlassara *et al.*, 1987; Holmes *et al.*, 2001). Shortening of telomeres due to successive cell replications limits lifespan of cell lines (Greider, 1998; Forsyth, Wright & Shay, 2002; Haussmann & Vleck, 2002). Other possible mechanisms for somatic senescence include autoimmune damage and accumulation of toxins. All these processes may affect different organ systems differently, due in part to differential use of protection mechanisms; nevertheless, it may not be completely inaccurate to unite all these different processes under the term 'somatic senescence'. Note that in this analysis I have assumed that lifespan and survivorship curves correlate with somatic senescence. While probably true, this need not be the case.

In any case, it is clear there is a separate physiological mechanism for the senescence of the female reproductive system in mammals, even if there may not be a uniform 'somatic senescence' to contrast it with. This does not necessarily imply a separate mechanism for all reproductive senescence, however. For example, mortality during childbirth increases 10-fold between ages 20 and 40 in humans (Shanley & Kirkwood, 2001). This is probably not due to loss of oocytes, but to a general decline in physical condition attributable to somatic senescence. It is not clear to what extent reproductive decline is due to senescence of the reproductive system itself, except that the endpoint – menopause – is a result of oocyte loss. In some cases, somatic declines may even have a greater effect on reproduction than on survivorship. For example, a forty-year old woman may have one-tenth the ability to withstand the trauma of childbirth that she had at age 20, but she suffers little increased risk of starvation, predation, or disease due to age (Howell, 1979; Hill & Hurtado, 1996).

Why, though, should reproductive senescence precede or outrun somatic senescence in female mammals? The physiology of the mammalian reproductive system may provide an explanation, based on the high cost of oocyte production and maintenance. Because oocyte number is fixed at birth (vom Saal *et al.*, 1994; Gosden & Faddy, 1998), there are only two ways to extend reproductive span: by increasing the initial follicle store, or by decreasing the rate of attrition. Comparatively, it appears both these traits evolve to produce longer reproductive spans in longer-lived animals (Gosden & Faddy, 1998). Since the decline is exponential, increasing the initial store should produce very high costs for very small gains. Across mammalian species, there appears to be a hyperallometric relationship between initial number of oocytes and longevity, but a hypoallometric relationship between initial number of oocytes and size (Gosden & Faddy, 1998). Perhaps, then, as overall size increases the relative proportion of investment represented by a larger oocyte stock decreases, so that even though the benefit of an increase in initial stock is small relative to its cost, it may be worth it in larger, longer-lived, more heavily invested offspring.

In addition to initial stock of oocytes, rate of attrition also appears subject to selection. In humans, some data indicate that most of the inter-individual variation appears to be in initial number of oocytes, not rate of loss (Daniel Promislow, personal communication). In laboratory mice, however, there is variation in rate of loss among strains, and at different ages (Gosden & Faddy, 1998). Furthermore, over large taxonomic gaps rate of loss varies, since mice have many more oocytes initially than humans do two to three years before menopause (vom Saal *et al.*, 1994). Gosden & Faddy (1998) estimated rate of loss based on half-life of follicle store in several species, and found that larger, longer-lived species lose oocytes more slowly, and that humans lose them even more slowly than other larger species. Because of the faster rate of loss later in life for humans, however, approximately 20 years are lost from the reproductive span relative to what it would have been if loss continued indefinitely at the slower rate (Gosden & Faddy, 1998). This could be taken to imply a programmed early end to reproduction, but it could also simply be a result of physiological correlates. For example, the decreasing ability of smaller numbers of oocytes to meet threshold levels of hormones to induce cycling could result in hormonal processes accelerating the rate of loss. Alternatively, if there was a trade-off between early rate of loss and late rate of loss, perhaps slowing the rate of loss early resulted in acceleration later.

If rate of loss accelerates later in life across many mammalian taxa as it does in humans, another implication could be that this acceleration is an unavoidable physiological process that results in a consistently early end to reproduction relative to somatic senescence (Richardson, Senikas & Nelson, 1987). If there was some unavoidable hastening of reproductive senescence towards the end, it would probably result in a disjunction between the end of reproduction and death. Under this scenario, reproduction might be relatively stable and then decline suddenly, as observed in both baboons and lions in the Packer *et al.* (1998) study.

As indicated by the above discussion, there are too many holes in our understanding of mammalian reproductive senescence to evaluate definitively the validity of the hypothesis presented to explain PRLS. However, the uniqueness of the female mammalian reproductive system, characterized by an oocyte stock fixed at birth, an exponential decline in oocyte number, and reproductive cycling dependent on oocytes, strongly suggests a mechanism for the early decline of reproduction in female mammals. The main unanswered question is why oocyte stock should be fixed and decline in such a regular way; but whatever the reason, it does appear that there should be a high cost to extending reproductive span, either through increasing initial stock size or by slowing rate of attrition. Coupled with the possibility that the acceleration of oocyte loss at the end of reproduction observed in humans could be general among mammals, this possibility should at the least provide a clear direction for future research on mammalian reproductive senescence, both physiological and demographic.

How might this hypothesis for the origin of PRLS and the various alternatives within it be addressed? An experiment would be possible but time consuming. In particular, a useful study would be to select strains of mice for late reproduction (regardless of lifespan) and long lifespan (regardless of age at reproduction). A difference between these strains would confirm the selective independence of reproductive and somatic ageing in a mammal species. More importantly, differences in hormone levels, reproductive schedules, and other fitness-related traits could show both endocrinological mechanisms and fitness trade-offs involved in selection for reproductive span in mammals. Given mouse generation times, pilot studies on heritability might be useful. Most of our understanding of these issues, however, must come from the ever-growing body of research that is illuminating the mechanisms and causes of various systems of cellular senescence, trade-offs, and hormone effects. Specifically, it will be exciting to understand the mechanisms and trade-offs involved in oocyte attrition, hopefully leading eventually to an evolutionary understanding of why oocyte number is fixed and declines so predictably. Also, a more rigorous examination of patterns of reproductive decline relative to lifespan is still needed in a wide variety of mammal species. In what taxa does decline in oocyte stock accelerate near the end of reproduction? Is PRLS really ubiquitous among mammals, and how significant is realized PRLS in wild populations? The data presented here are highly suggestive, but not conclusive. A look at other vertebrate groups as well could firmly delimit the taxonomic distribution of PRLS. It appears PRLS is present in chickens *Gallus gallus* and quail *Coturnix coturnix*, but fulmars *Fulmarus glacialis* have been observed to reproduce for 30 years with no decline (vom Saal *et al.*, 1994; Ottinger, Nisbet & Finch, 1995; Ottinger, 2001). And it is unknown whether or not birds exhibit *de novo* oogenesis after hatching – the unique mammalian female reproductive system may not actually be so unique. Intriguingly, nematode worms *Caenorhabditis elegans* show PRLS approximately twice the length of normal reproductive lifespan under laboratory conditions, though in the wild few individuals would ever reach this age (Kerry Kornfeld, personal communication).

VI. CONCLUSIONS

(1) A comparative look at post-reproductive lifespan (PRLS) in females of 42 mammal species from eight orders indicates that this phenomenon is not unique to humans. Thus, traditional adaptive hypotheses based on kin selection are not likely to explain the origin of PRLS adequately in humans, although they may help explain its maintenance and extraordinary length.

(2) An explanation for the origin of PRLS may lie in separate trajectories for reproductive and somatic senescence. Selection would maximize the fitness of each trajectory semi-independently, resulting in a concordant period of maximum vigour and fecundity followed by a disjunction in these types of senescence at older ages.

(3) In female mammals in particular, a potential physiological basis for independent selection on reproductive and somatic senescence is evident. Reproductive senescence appears to be largely determined by the decline in oocyte number. Moreover, the high cost to increasing initial oocyte number suggests a trade-off resulting in earlier reproductive senescence and PRLS.

(4) Further work on the demography and physiology of reproductive senescence in a wide variety of mammalian and avian species is necessary to confirm the patterns observed here and the hypothesis proposed.

VII. ACKNOWLEDGEMENTS

I wish to thank Robert Ricklefs for setting me on the road to this article and for many helpful discussions along the way. Thanks also to Alexander Scheuerlein and the Ricklefs lab. group and to Caleb Finch, Richard Alexander, Craig Packer, Mary Pavelka, Leslie Newman, and two anonymous reviewers for insightful suggestions and references. Financial support was provided by a Predoctoral Fellowship from the Howard Hughes Medical Institute and a University Fellowship from the University of Missouri-St. Louis.

VIII. REFERENCES

Aguilar, A. & Borrell, A. (1988). Age and sex-related changes in organochlorine compound levels in Fin Whales *Balaenoptera physalus* from the eastern North Atlantic. *Marine Environmental Research* **25**, 195–212.

Albarrán, M. T., López-Burillo, S., Pablos, M. I., Reiter, R. J. & Agapito, M. T. (2001). Endogenous rhythms of melatonin, total antioxidant status and superoxide dismutase activity in several tissues of chick and their inhibition by light. *Journal of Pineal Research* **30**, 227–233.

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

Alexander, R. D. (1990). *How did humans evolve? Reflections on the uniquely unique species*. Museum of Zoology, University of Michigan, Ann Arbor.

Alvarez, H. P. (2000). Grandmother hypothesis and primate life histories. *American Journal of Physical Anthropology* **113**, 435–450.

Anderson, A. C. (1965). Reproductive ability of female beagles in relation to advancing age. *Experimental Gerontology* **1**, 189–192.

Anderson, A. C. (1970*a*). *The Beagle as an Experimental Dog*. Iowa State University Press, Ames, IA.

Anderson, A. C. (1970*b*). Reproductive system. In *The Beagle as an Experimental Dog* (ed. A. C. Anderson), pp. 323–324. Iowa State University Press, Ames, IA.

Anderson, A. C. & Rosenblatt, L. S. (1965). Survival of beagles under natural and laboratory conditions. *Experimental Gerontology* **1**, 193–199.

Armstrong, D. T. (2001). Effects of maternal age on oocyte developmental competence. *Theriogenology* **55**, 1303–1322.

Austad, S. N. (1994). Menopause: an evolutionary perspective. *Experimental Gerontology* **29**, 255–263.

Austad, S. N. (1997). Postreproductive survival. In *Between Zeus and the Salmon: The Biodemography of Longevity* (eds. K. W. Wachter and C. E. Finch), pp. 161–174. National Academy Press, Washington DC.

Bellamy, D. (1981). Ageing: with particular reference to the use of the house mouse as a mammalian model. In *Biology of the House Mouse* (ed. R. J. Berry), pp. 267–300. Academic Press, London.

Bérubé, C. H., Festa-Bianchet, M. & Jorgenson, J. T. (1999). Individual differences, longevity, and reproductive senescence in bighorn ewes. *Ecology* **80**, 2555–2565.

Blurton Jones, N. G., Hawkes, K. & O'Connell, J. F. (2002). Antiquity of postreproductive life: are there modern impacts on hunter-gatherer postreproductive life spans? *American Journal of Human Biology* **14**, 184–205.

Bocquet-Appel, J.-P. & Masset, C. (1982). Farewell to paleodemography. *Journal of Human Evolution* **11**, 321–333.

Bocquet-Appel, J.-P. & Masset, C. (1996). Paleodemography: expectancy and false hope. *American Journal of Physical Anthropology* **99**, 571–583.

Borries, C., Sommer, V. & Srivastava, A. (1991). Dominance, age, and reproductive success in female Hanuman langurs (*Presbytis entellus*). *International Journal of Primatology* **12**, 231–257.

Broussard, D. R., Risch, T. S., Dobson, F. S. & Murie, J. O. (2003). Senescence and age-related reproduction of female Columbian ground squirrels. *Journal of Animal Ecology* **72**, 212–219.

Caro, T. M., Sellen, D. W., Parish, A., Frank, R., Brown, D. M., Voland, E. & Borgerhoff Mulder, M. (1995). Termination of reproduction in nonhuman and human female primates. *International Journal of Primatology* **16**, 205–220.

Charlesworth, B. & Hughes, K. A. (1996). Age-specific inbreeding depression and components of genetic variance in relation to the evolution of senescence. *Proceedings of the National Academy of Sciences of the United States of America* **93**, 6140–6145.

Comfort, A. (1979). *The Biology of Senescence*, 3rd Edn. Elsevier, New York.

Corsolini, S., Nigro, M., Olmastroni, S., Focardi, S. & Regoli, F. (2001). Susceptibility to oxidative stress in Adélie and emperor penguin. *Polar Biology* **24**, 365–368.

Dyke, B., Gage, T. B., Alford, P. L., Swenson, B. & Williams-Blangero, S. (1995). Model life table for captive chimpanzees. *American Journal of Primatology* **37**, 25–37.

Dyke, B., Gage, T. B., Mamelka, P. M., Goy, R. W. & Stone, W. H. (1986). A demographic analysis of the Wisconsin USA Regional Primate Center rhesus colony 1962–1982. *American Journal of Primatology* **10**, 257–270.

Erickson, B. H., Reynolds, R. A. & Murphree, R. L. (1976). Ovarian characteristics and reproductive performance of the aged cow. *Biology of Reproduction* **15**, 555–560.

Fedigan, L. (1991). Life span and reproduction in Japanese macaque females. In *The Monkeys of Arashiyama: Thirty-five Years of Research in Japan and the West* (eds. L. M. Fedigan and P. J. Asquith), pp. 140–154. State University of New York Press, New York.

Fedigan, L. M. & Pavelka, M. S. M. (2001). Is there adaptive value to reproductive termination in Japanese macaques? A test of maternal investment hypotheses. *International Journal of Primatology* **22**, 109–125.

Finch, C. E. (1990). *Longevity, Senescence, and the Genome*. University of Chicago Press, Chicago.

Finch, C. E. (1994). The evolution of ovarian oocyte decline with aging and possible relationships to Down syndrome and Alzheimer disease. *Experimental Gerontology* **29**, 299–304.

Finch, C. E. (2002). Evolution and the plasticity of aging in the reproductive schedules in long lived animals: the importance of genetic variation in neuroendocrine mechanisms. *Hormones, Brain, and Behavior* **4**, 799–820.

Finch, C. E. & Sapolsky, R. M. (1999). The evolution of Alzheimer disease, the reproductive schedule, and apoE isoforms. *Neurobiology of Aging* **20**, 407–428.

Fisher, M. W., McLeod, B. J., Mockett, B. G., Moore, G. H. & Drew, K. R. (1996). Reproductive senescence in aged red deer hinds. *Proceedings of the New Zealand Society of Animal Production* **56**, 344–346.

Flaws, J. A., Abbud, R., Mann, R. J., Nilson, J. H. & Hirschfield, A. N. (1997). Chronically elevated luteinizing hormone depletes primordial follicles in the mouse ovary. *Biology of Reproduction* **57**, 1233–1237.

Forsyth, N. R., Wright, W. E. & Shay, J. W. (2002). Telomerase and differentiation in multicellular organisms: turn it off, turn it on, and turn it off again. *Differentiation* **69**, 188–197.

Gage, T. B. (1998). The comparative demography of primates: with some comments on the evolution of life histories. *Annual Review of Anthropology* **27**, 197–221.

Gosden, R. G. & Faddy, M. J. (1998). Biological bases of premature ovarian failure. *Reproduction, Fertility, and Development* **10**, 73–78.

Gould, K. G., Flint, M. & Graham, C. E. (1981). Chimpanzee reproductive senescence: a possible model for evolution of the menopause. *Maturitas* **3**, 157–166.

Gould, S. J. & Vrba, E. S. (1982). Exaptation: a missing term in the science of form. *Paleobiology* **8**, 4–15.

Greider, C. W. (1998). Telomeres and senescence: the history, the experiment, the future. *Current Biology* **8**, R178–R181.

Ha, J. C., Robinette, R. L. & Sackett, G. P. (2000). Demographic analysis of the Washington Regional Primate Research

Center pigtailed macaque colony, 1967–1996. *American Journal of Primatology* **52**, 187–198.

Hamilton, W. D. (1966). The moulding of senescence by natural selection. *Journal of Theoretical Biology* **12**, 12–45.

Haussmann, M. F. & Vleck, C. M. (2002). Telomere length provides a new technique for aging animals. *Oecologia* **130**, 325–328.

Hawkes, K., O'Connell, J. F. & Blurton Jones, N. G. (1989). Hardworking Hadza grandmothers. In *Comparative Socioecology* (eds. V. Standen and R. A. Foley), pp. 341–366. Blackwell Scientific Press, Oxford.

Hawkes, K., O'Connell, J. F. & Blurton Jones, N. G. (2000). Why do women have a mid-life menopause? Grandmothering and the evolution of human longevity. In *Female Reproductive Aging: The Proceedings of the 10th Reinier de Graaf Symposium*, Zeist, The Netherlands, 9–11 September 1999, vol. 9. Studies in Profertility (eds. E. R. Te Velde, P. L. Pearson and F. J. Broekmans), pp. 27–41. Parthenon Publishing Group, New York.

Hawkes, K., O'Connell, J. F., Jones, N. G. B., Alvarez, H. & Charnov, E. L. (1998). Grandmothering, menopause, and the evolution of human life histories. *Proceedings of the National Academy of Sciences of the United States of America* **95**, 1336–1339.

Hill, K. & Hurtado, A. M. (1991). The evolution of premature reproductive senescence and menopause in human females: an evaluation of the 'Grandmother Hypothesis'. *Human Nature* **2**, 313–350.

Hill, K. & Hurtado, A. M. (1996). *Ache Life History: The Ecology and Demography of a Foraging People*. Aldine De Gruyter, New York.

Holmes, D. J., Fluckiger, R. & Austad, S. N. (2001). Comparative biology of aging in birds: an update. *Experimental Gerontology* **36**, 869–883.

Howell, N. (1979). *Demography of the Dobe !Kung*. Academic Press, New York.

Jackes, M. K. (1985). Pubic symphysis age distributions. *American Journal of Physical Anthropology* **68**, 281–300.

Johnson, R. L. & Kapsalis, E. (1995). Ageing, infecundity and reproductive senescence in free-ranging female rhesus monkeys. *Journal of Reproduction & Fertility* **105**, 271–278.

Johnson, R. L. & Kapsalis, E. (1998). Menopause in free-ranging rhesus macaques: estimated incidence, relation to body condition, and adaptive significance. *International Journal of Primatology* **19**, 751–765.

Jones, E. C. & Krohn, P. L. (1961). The effect of hypophysectomy on age changes in the ovaries of mice. *Journal of Endocrinology* **21**, 497–508.

Judge, D. S. & Carey, J. R. (2000). Postreprodctive life predicted by primate patterns. *Journal of Gerontology: Biological Sciences* **55A**, B201–B209.

Kasuya, T. & Marsh, H. (1984). Life history and reproductive biology of the short-finned pilot whale, *Globicephala macrorhyncus*, off the Pacific coast of Japan. *Reports of the International Whaling Commision* **Special Issue 6**, 259–310.

Kirkwood, T. B. L. & Austad, S. N. (2000). Why do we age? *Nature* **408**, 233–238.

Lancaster, J. B. & King, B. J. (1985). An evolutionary perspective on menopause. In *In Her Prime* (eds. J. K. Brown and V. Kerns), pp. 13–20. Bergin and Garvey Publishers Inc., South Hadley, MA.

Laws, R. M., Parker, I. S. C. & Johnstone, R. C. B. (1975). *Elephants and their Habitats: The Ecology of Elephants in North Bunyoro, Uganda*. Clarendon Press, Oxford.

Lovejoy, C. O., Meindl, R. S., Pryzbeck, T. R., Barton, T. S., Heiple, K. G. & Kottting, D. (1977). Paleodemography of the Libben Site, Ottawa County, Ohio. *Science* **198**, 291–293.

Mace, R. (2000). Evolutionary ecology of human life history. *Animal Behaviour* **59**, 1–10.

Marsh, H. & Kasuya, T. (1986). Evidence for reproductive senescence in female cetaceans. *Reports of the International Whaling Commision* **Special Issue 8**, 57–74.

Marshall, F. H. A. (1964). *The Physiology of Reproduction*, 3rd Edn. Longmans, London.

McLaren, A. (1958). The biology of the ringed seal (*Phoca hispida* Schreber) in the eastern Canadian arctic. In Bulletin 118. Fisheries Research Board of Canada, Ottawa.

Meindl, R. S. & Lovejoy, C. O. (1985). Ectocranial suture closure: a revised method for the determination of skeletal age at death based on the lateral-anterior sutures. *American Journal of Physical Anthropology* **68**, 57–66.

Mensforth, R. P. (1990). Paleodemography of the Carlston Annis Bt-5 Late archaic skeletal population. *American Journal of Physical Anthropology* **82**, 81–100.

Meredith, S., Jackson, K., Dudenhoeffer, G., Graham, L. & Epple, J. (2000). Long-term supplementation with melatonin delays reproductive senescence in rats, without an effect on number of primordial follicles. *Experimental Gerontology* **35**, 343–352.

Mizroch, S. A. (1981). Analysis of some biological parameters of the Antarctic fin whale (*Balaenoptera physalus*). *Reports of the International Whaling Commision* **31**, 425–434.

Nishida, T., Takasaki, H. & Takahata, Y. (1990). Demography and reproductive profiles. In *The Chimpanzees of the Mahale Mountains: Sexual and Life History Strategies* (ed. T. Nishida), pp. 63–98. University of Tokyo Press, Tokyo.

Nozaki, M., Mitsunaga, F. & Shimizu, K. (1995). Reproductive senescence in female Japanese monkeys (*Macaca fuscata*): age- and season-related changes in hypothalamic-pituitary-ovarian functions and fecundity rates. *Biology of Reproduction* **52**, 1250–1257.

Olesiuk, P. F., Bigg, M. A. & Ellis, G. M. (1990). Life history and population dynamics of resident killer whales (*Orcinus orca*) in the coast waters of British Columbia and Washington State. *Reports of the International Whaling Commission* **Special Issue 12**, 209–243.

Ottinger, M. A. (2001). Quail and other short-lived birds. *Experimental Gerontology* **36**, 859–868.

Ottinger, M. A., Nisbet, I. C. T. & Finch, C. E. (1995). Aging and reproduction: comparative endocrinology of the Common Tern and Japanese Quail. *American Zoologist* **35**, 299–306.

Packer, C., Tatar, M. & Collins, A. (1998). Reproductive cessation in female mammals. *Nature* (*London*) **392**, 807–811.

Parkening, T. A. (1982). Reproductive senescence in the Chinese hamster (*Cricetulus griseus*). *Journals of Gerontology* **37**, 283–287.

Paul, A., Kuester, J. & Podzuweit, D. (1993). Reproductive senescence and terminal investment in female Barbary macaques (*Macaca sylvanus*) at Salem. *International Journal of Primatology* **14**, 105–124.

Pavelka, M. S. M. & Fedigan, L. M. (1991). Menopause: a comparative life history perspective. *Yearbook of Physical Anthropology* **34**, 13–38.

Pavelka, M. S. M. & Fedigan, L. M. (1999). Reproductive termination in female Japanese monkeys: a comparative life history perspective. *American Journal of Physical Anthropology* **109**, 455–464.

Peccei, J. S. (1995). A hypothesis for the origin and evolution of menopause. *Maturitas* **21**, 83–89.

Peccei, J. S. (2001). A critique of the grandmother hypotheses: old and new. *American Journal of Human Biology* **13**, 434–452.

Ramsay, M. A. & Stirling, I. (1988). Reproductive biology and ecology of female polar bears *Ursus maritimus*. *Journal of Zoology* (*London*) **214**, 601–634.

Raouf, S. A., Parker, P. G., Ketterson, E. D., Nolan, V. J. & Ziegenfus, C. (1997). Testosterone influences reproductive success by increasing extra-pair fertilizations in male dark-eyed juncos, *Junco hyemalis*. *Proceedings of the Royal Society Biological Sciences Series B* **264**, 1599–1603.

Richardson, S. J., Senikas, V. & Nelson, J. F. (1987). Follicular depletion during the menopausal transition: evidence for accelerated loss and ultimate exhaustion. *Journal of Clinical Endocrinology and Metabolism* **65**, 1231–1237.

Ricklefs, R. E., Scheuerlein, A. & Cohen, A. A. (2003). Age-related patterns of fertility in captive populations of birds and mammals. *Experimental Gerontology* **38**, 741–745.

Rogers, A. R. (1993). Why menopause? *Evolutionary Ecology* **7**, 406–420.

Rose, M. R. (1984). Laboratory evolution of postponed senescence in *Drosophila melanogaster*. *Evolution* **38**, 1004–1010.

Rose, M. R., Drapeau, M. D., Yazdi, P. G., Shah, K. H., Moise, D. B., Thakar, R. R., Rauser, C. L. & Mueller, L. D. (2002). Evolution of late-life mortality in *Drosophila melanogaster*. *Evolution* **56**, 1982–1991.

Sear, R., Mace, R. & McGregor, I. A. (2000). Maternal grandmothers improve nutritional status and survival of children in rural Gambia. *Proceedings of the Royal Society Biological Sciences Series B* **267**, 1641–1647.

Shanley, D. P. & Kirkwood, T. B. L. (2001). Evolution of the human menopause. *BioEssays* **23**, 282–287.

Smith, A. D. B. & Robison, O. J. (1931). The average ages of cows and bulls in six breeds of cattle. *Journal of Agricultural Science* **21**, 136–149.

Stearns, S. C. (1992). *The Evolution of Life Histories*. Oxford University Press, New York.

Sugiyama, Y. (1994). Age-specific birth rate and lifetime reproductive success of chimpanzees at Bossou, Guinea. *American Journal of Primatology* **32**, 311–318.

Takahata, Y., Koyama, N. & Suzuki, S. (1995). Do the old aged females experience a long post-reproductive life span? The cases of Japanese macaques and chimpanzees. *Primates* **36**, 169–180.

Tardif, S. D. & Ziegler, T. E. (1992). Features of female reproductive senescence in tamarins, *Saguinus spp*, a new world primate. *Journal of Reproduction & Fertility* **94**, 411–421.

Tigges, J., Gordon, T. P., McClure, H. M., Hall, E. C. & Peters, A. (1988). Survival rate and life span of rhesus monkeys at the Yerkes Regional Primate Research Center, Atlanta, Georgia, USA. *American Journal of Primatology* **15**, 263–274.

Trinkhaus, E. & Thompson, D. D. (1987). Femoral diaphyseal histomorphometric age determinations for Shanidar 3, 4, 5, and 6 Neandertals and Neandertal longevity. *American Journal of Physical Anthropology* **72**, 123–129.

Turner, H. N. & Dolling, C. H. (1964). Vital statistics for an experimental flock of merino sheep. *Australian Journal of Agricultural Research* **16**, 699–712.

Vlassara, H., Valinsky, J., Brownlee, M., Cerami, C., Nishimoto, S. & Cerami, A. (1987). Advanced glycosylation endproducts on erythrocyte cell surface induce receptor-mediated phagocytosis by macrophages. *Journal of Experimental Medicine* **166**, 539–549.

vom Saal, F. S., Finch, C. E. & Nelson, J. F. (1994). Natural history and mechanisms of reproductive aging in humans, laboratory rodents, and other selected vertebrates. In *The Physiology of Reproduction* (eds. E. Knobil and J. D. Neill), pp. 1213–1314. Raven Press Ltd., New York.

Walker, M. L. (1995). Menopause in female rhesus monkeys. *American Journal of Primatology* **35**, 59–71.

Walker, P. L., Johnson, J. R. & Lambert, P. M. (1988). Age and sex biases in the preservation of human skeletal remains. *American Journal of Physical Anthropology* **76**, 183–188.

Washburn, S. L. (1981). Longevity in primates. In *Aging, Biology, and Behavior* (eds. J. March and J. McGaugh), pp. 11–29. Academic Press, New York.

Weiss, K. M. (1973). Demographic Models for Anthropology. In *American Antiquity*, Vol. 38, Number 2, Part 2, April 1973.

Weiss, K. M. (1981). Evolutionary perspectives on human aging. In *Other Ways of Growing Old: Anthropological Perspectives* (eds. P. T. Amoss and S. Harrell), pp. 25–58. Stanford University Press, Stanford, California.

Willey, P. & Mann, B. (1986). The skeleton of an elderly woman from the Crow Creek Site South Dakota, USA and its implications for paleodemography. *Plains Anthropologist* **31**, 141–152.

Williams, G. C. (1957). Pleiotropy, natural selection, and the evolution of senescence. *Evolution* **11**, 398–411.

Wise, P. M. (2001). The 'menopause' and the aging brain: causes and repercussions of hypoestrogenicity. *Biogerontology* **2**, 113–115.