On the evolution of growth and senescence

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Abstract

Consistent associations between growth and senescence are seen throughout nature. Whilst a larger size correlates positively with lifespan between species, this relationship is reversed within a species so that the smallest members tend to be the longest-lived. Indeterminate growth - i.e. growth that continues post-maturity - is a strong predictor for an especially slow rate of ageing. A number of interventions which alter the rate of growth, especially at a point early in development, have been shown to have enduring effects on later growth and lifespan. This thesis provides a theoretical examination of why relationships such as these may have evolved.

Two dynamic programming models are here presented. Both consider associations between growth and longevity within a species and ask whether these are compatible with idea of a trade-off between somatic maintenance and other fitness-enhancing functions as predicted by the disposable soma theory. The first reproduces the sexual dimorphism in longevity and in body size seen baboons; it predicts that males should 'choose' a faster rate of ageing and a greater investment in growth than females. The second suggests that a faster rate of ageing may be an optimal response to low food availability in early life in humans.

A critical appraisal is also given to two recent theories of the evolution of ageing which rely explicitly on differences in body size and/or growth to explain differences in lifespan: the hyperfunction theory and the heat dissipation limit theory. What these can teach us about the evolution of senescence and whether they can provide plausible challenges to the disposable soma theory is considered.

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Chapter 1: Introduction

1.1 The evolutionary puzzle of ageing

From even a cursory review of the various life cycles found in nature, it is apparent that diversity in lifespan, rates of senescence and the ageing phenotype is vast. Maximum lifespans range from just days for yeast and bacteria, to weeks for worms and flies, years for guppies, decades for horses, centuries and tortoises and millennia for certain plants. Even for reasonable closely-related species such as mice and humans lifespan can vary over an order of magnitude. Despite experiencing essentially the same cellular and physiological changes with age - and approximately the same lifetime risk of several age-related diseases - mice can live only a few years in a laboratory whilst the maximum recorded human lifespan stands at 122 years[1].

For iteroparous or polycarpic species, senescence is usually a gradual process; there is a steady decline in physiological integrity with age and the productivity and frequency of reproductive events is steadily reduced. In contrast, semelparous or monocarpic species seem to 'fall apart' within a short space of time immediately succeeding reproduction. In the case of Pacific Salmon, individuals can survive between one and five years at sea before reproducing, but always die within a few days or weeks of spawning[2].

For the majority of species - or at least those with which we are most familiar and for which the most age-specific data are available - there is a progressive deterioration in performance with time. This typically manifests itself in an increased rate of mortality and reduced fecundity. However, such deterioration is by no means universal[3]. A number of species, including hydra, hermit crabs and armed saltbush, show no change in mortality rate or fecundity with age; they seem not to age at all. Still others, such as the desert tortoise and white mangrove, experience a reduction in mortality rate and increase in fertility throughout life, a phenomenon which has been termed negative senescence.

Even within a species, differences in longevity can be striking. Some of these differences are 'hard-wired', that is, they are mediated directly by genetic factors. Human longevity, for example, shows a substantial heritable component which has

been estimated at 25% in Western populations[4]. Some differences are plastic: they are elicited by exposure to different environmental factors such as external mortality hazards, food availability and seasonality. As a direct result of differences in nutrition and pheromone exposure during the larval phase, a queen bee is able to live for two-three years whilst her sister workers survive only four-eight weeks[5]. Some differences are stochastic and simply reflect the effect of chance upon the emergence of disease and ultimately upon mortality. In the nematode *C. elegans*, age at death has been shown to vary considerably even between genetically identical individuals grown in a homogenous environment.

Any theory of the evolution of ageing must explain this distribution of senescent phenotypes: why ageing occurs in some species and not in others; why it proceeds at different rates in different species or under different environmental conditions and; why it is sometimes a gradual process throughout adult life and sometimes a rapid decline at the very end of life.

It is not immediately apparent why ageing should occur at all. Since reductions in fertility and increases in mortality are normally opposed by natural selection, we might expect that senescence would have been eliminated. We also know that immortality is possible for at least those of an individual's cells which will be passed on to future generations; the germline cells of every organism alive today are descended via direct cell division from the earliest life forms on Earth and the germline cells of any organisms alive millennia from now will be directly descended from those of current species. In order for life itself to continue, these cells must be able to replicate themselves without the accumulation of defects.

Although it has been suggested that ageing is only seen in protected environments and therefore is outside the influence of natural selection, this is now known not to be the case. Senility may be so rare in the wild that little selective pressure against it exists however senescent changes are seen well before such a time as senility sets in. Decreases in survivorship and fecundity are well-documented in wild populations of birds and mammals and some data are available for senescence in insects, reptiles, fish and in one case amphibians[6]. Athletic records show that people in their thirties - an

age to which many humans live even outside of protected environments - have already started to decline in strength and robustness.

This chapter reviews the most prominent theories which have been put forward to explain why ageing has evolved and the evidence which has been presented for each of them. There then follows a summary of the relationships between growth and longevity and on sex differences in longevity which are the primary focus of this work. We begin with a short discussion on what it means to say an organism ages. Whilst a single definition of age would obviously be desirable, it is difficult to provide a measure that can be applied to all species and that captures the essential differences in how individuals age. For this reason the measure of ageing used will often depend upon the question being addressed.

1.2 How to measure ageing

One of the most rudimentary indicators of ageing rate is the maximum age to which any member of a species (or other group) has been known to live. This can easily identify gross differences between species - as for example between the Galapagos tortoise (106 + years) and the house mouse (3 years) - but is heavily dependent upon the sample size from which the measure is drawn. If sample size is known, some correction for this can be made but this is unfortunately not always the case. An alternative measure, which avoids this bias towards higher values for better-studied species, is life expectancy at birth or any other age. Both of these are, however, measures of *lifespan* which is dependent upon, but not synonymous with, ageing.

Consider the mortality profile seen in humans and many other animals, the early part of which is shown in figure 1.1B (solid black line)[7]. Mortality is rather high in infancy and gradually falls to a minimum at, or just before, the time of reproductive maturity. From then onwards it increases exponentially with age as described by the Gompertz hazard function:

$$\mu(x) = \alpha * \exp(\beta * x)$$

where $\mu(x)$ is the mortality rate at age x units above the age of reproductive maturity; α is the initial mortality rate at the age of reproductive maturity and; β describes the rate of increase in mortality. Whilst α depends upon the range and severity of

extrinsic threats (such as predation, disease or starvation) to which the organism is exposed when in optimal condition, β depends both upon the intrinsic rate of deterioration in physiological condition and the relative susceptibility to external threats as a function of condition (for example, the relative facility with which a predator/pathogen can kill/infect an old rather than a young individual). Since:

$\ln[\mu(x)] = \ln[\alpha] + \beta * x$

plotting Gompertz functions on a logarithmic scale gives a straight line, the elevation of which increases with α and the gradient of which increases with β. Gompertz functions are shown, on both arithmetic and logarithmic scales, for different values of α and β in figure 1.1A.

For any two species or groups which display such a mortality profile - moderately high early life mortality and exponential increases post-maturity - one may enjoy a longer lifespan than the other for at least four different reasons [7](figure 1.1B). Firstly, mortality during early life may be reduced; secondly, the onset of ageing may be delayed; thirdly, initial mortality rate (baseline mortality) may be reduced and; fourthly, the rate of increase in mortality with age may be reduced. As ageing is a description of the rate of deterioration over time, it is only this last reason that corresponds to a difference in ageing rate. For this reason β is often taken as a measure of senescence. An equivalent measure also often used is the mortality rate doubling time (MRDT). This can be derived as a function of β from the Gompertz mortality function above in the following way, and these therefore amount to descriptions of the same property.

If mortality rate at age x_1 is half that at age x_2, then:

$2 * \alpha * \exp(\beta * x_1) = \alpha * \exp(\beta * x_2)$

$2 = \exp(\beta * (x_2 - x_1))$

$MRDT = x_2 - x_1 = \ln[2]/\beta$

Unfortunately, using β or MRDT as measures of ageing is also not without difficulties. They take no account of decreases in fecundity which can be as important as increases in mortality. They may also change as a consequence of environmental factors; as

mentioned above they depend upon the relative susceptibility to external threats as a function of condition and therefore can vary with extrinsic hazards as well as with intrinsic rates of deterioration. Suppose, for example, that a population suffers predation and that it is almost exclusively older individuals that are targeted. If the predator population were diminished, this would lower mortality in the old to a greater extent than in the young so that the rate of increase in mortality with age is reduced. Finally, as stated in section 1.1, not all species display a Gompertz mortality profile.

Relatively low mortality throughout early life with a strong deterioration at very old ages (as seen with a Gompertz mortality function) produces a type I survivorship curve [8](figure 1.2). This is seen in buffalo, red deer, elephants and humans among other species. That this is the survivorship pattern with which we are most familiar may simply reflect a phylogenetic bias in available data sets. A type II survivorship curve is characterised by constant mortality throughout life[8]. Species which display this pattern survivorship - which include hydra, squirrels and songbirds - are said to display negligible senescence. A type III survivorship curve is characterised by high mortality in early life which then declines monotonically with age and species in which this is seen - for example oysters, alligators and redwood trees - are said to display negative senescence[8]. If mortality is our metric for senescence, then these species seem to be growing younger. More complex survivorship curves are also seen and a fourth is sometimes defined (figure 1.2); this is characterised by high mortality during both the developmental and post-reproductive periods and low mortality during the reproductive period. Such a survivorship pattern is seen in the white-tailed deer. As for mortality profiles, reproductive profiles can vary considerably, with bell-shaped, increasing, steadily or exponentially declining and, asymptotic functions all being found in nature[3].

These differences have led Baudisch to distinguish between the shape and pace of senescence, and she suggests that these represent orthogonal axes along which life histories can be classified[3]. Pace is a measure of the length of life and of the reproductive rate (a fast pace referring to a short life and high reproductive rate) whilst shape is a measure of the abruptness with which survivorship and fecundity decline towards the end of life. The latter could be measured by the mortality rate at

the end of life (for example at an age to which 5% of the population survive) divided by the mean adult mortality - a lower value would indicate a more rapid deterioration and a higher value a more gradual one.

A couple of criticisms can be made of defining ageing in terms of mortality rates, as all the measures of ageing thus far considered do. Firstly, there may be differences between subgroups of a population which die young and those which survive to old age and the average mortality rate may not therefore reflect that which some/all subgroups actually experience. If, for example, various strata of a cohort differ in their susceptibility to mortality but each stratum experiences a constant mortality threat over time, the selective survival of those with lower mortality rates would cause the overall rate to decline with age.

Secondly, a change in mortality rate may proceed from causes other than a change in physiological integrity. Many indeterminate growers, for example, experience a reduction in predation risk with increased size and therefore a type III survivorship curve. However, although indeterminate growers are able to maintain physiological integrity more successfully than determinate growers, the fact that they experience a reduction in mortality rate does not imply their condition has improved; this is not a reversal of the sort of deterioration seen in determinate growers with age.

We could instead try to measure ageing in terms of the molecular changes which accompany a progressive, generalised loss of physiological integrity over time. Much work has been done in recent decades on the molecular causes of ageing and an increasingly large set of damage markers are known[9]. In theory, if a complete set of biomarkers of ageing could be defined, they could be used as a reference for biological, rather than chronological, age. However, some of these at least seem to be species-specific and comparing ageing rates between species might therefore be hard without some recourse to mortality and/or reproductive trajectories.

Finally, the question of whether ageing can be separated from age-related disease has implications for how we attempt to measure ageing[1]. It is well known that the probability of contracting a wide range of diseases increases with age. *A priori*, this could be explained in a number of ways: the ageing process itself may predispose individuals to disease; ageing and disease may be caused by the same agents via

independent pathways (so that ageing has no direct effect upon the likelihood of contracting disease) or; they may be completely unrelated phenomena both of which develop over time. To the extent that ageing predisposes to disease, we may consider disease an integral part of the ageing process and one which must be encompassed in any definition of ageing. However, disease itself will have specific effects upon physiology which could obscure those of any underlying ageing process - as it might occur in an otherwise healthy person - and to this end the definition of ageing should confine itself to disease-free individuals. This is a moot, but conceptually important, point. (From what we know of ageing it does make individuals intrinsically more susceptible to age-related disease and is also caused by many of the same factors that cause disease.)

An awareness of the strengths and weaknesses of these different measures of ageing, and of the conceptual issues in defining age, is important for gerontologists. The measure used may depend on available data as well as on the focus of the researcher. For the models presented in chapters 3 & 4 of the current work, the rate of ageing under a Gompertz mortality model (β) is used; the species to which these models apply (baboons and humans respectively) are known to display Gompertz mortality trajectories and it is assumed for both models that, post-maturity, mortality rate varies only as a function of condition.

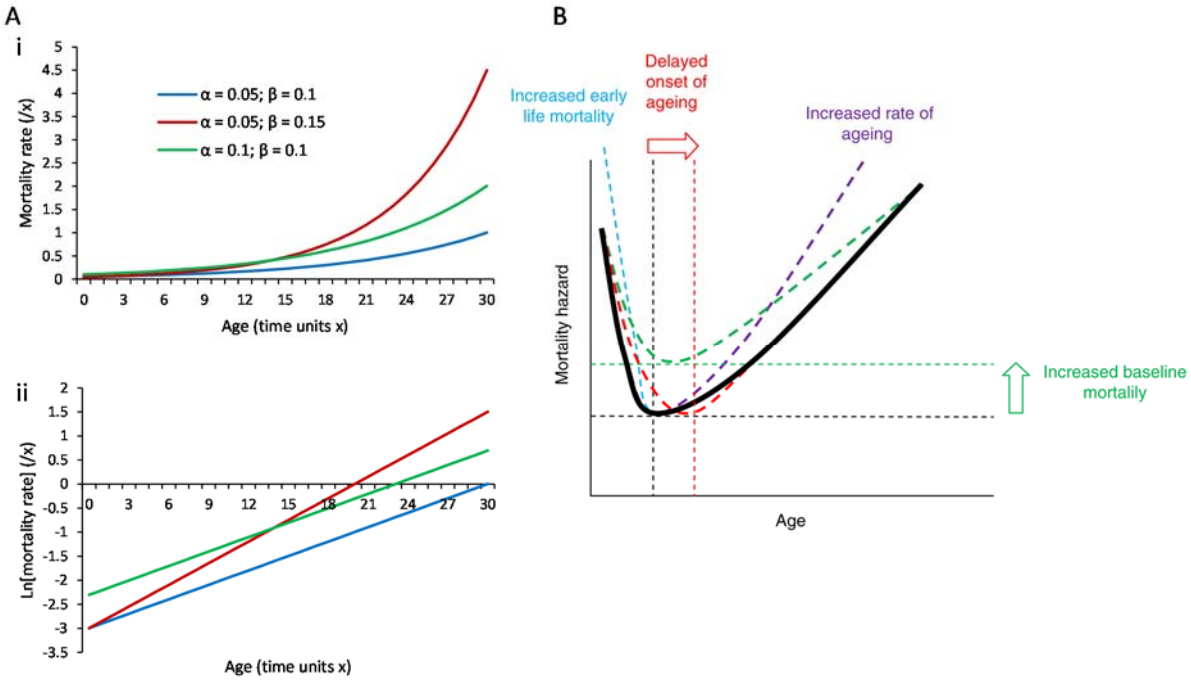

Figure 1.1: A: Gompertz mortality curves plotted on a linear (i) and logarithmic (ii) scale. The effects of varying initial mortality rates (α) and rates of ageing (β) are shown. B: Four possible changes to a species' mortality profile all of which may reduce average lifespan (taken from Selman, Nussey & Monaghan[7]).

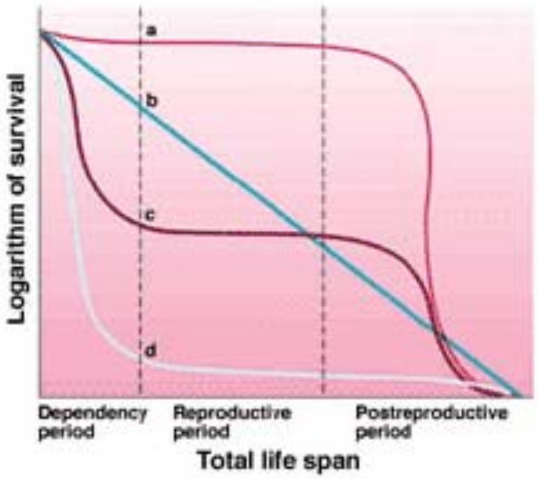

Figure 1.2: Four types of survivorship curve. Type I(a) display low mortality during the juvenile and reproductive periods followed by rapid increases in mortality throughout post-reproductive life. Type II(b) display constant mortality throughout the entire life course. Type III(d) display high mortality during the juvenile period followed by low mortality during the reproductive and post-reproductive periods. Type IV(c) display high mortality during both the juvenile and post-reproductive periods, and low mortality over the reproductive period. (Figure taken from www.cathylaw.com/APES/populationdynamicsnotes.html.)

1.3 Programmed ageing

1.3.1 Ageing for the good of the species

Theories of programmed ageing propose that positive selection exists for the decline - and ultimately the death from purely intrinsic causes - that occurs in many organisms over time. They predict that ageing has evolved because it carries some merit in its own right rather than as the by-product of selection upon some other trait(s) (see section 1.4) or because selection is powerless to act against it in the wild. (As mentioned in section 1.1, ageing is widespread in the wild and its effects can often be seen in early/mid adulthood when selection is still strong.)

It is hard to see how this could be to the good of the individual and proponents of programmed ageing generally argue that it has evolved for the good of the species. The idea was articulated well over a century ago by Weismann[10]; he compared ageing to mechanical wear and tear - an inevitable deterioration with time and use - and postulated a death mechanism which would bring about the controlled termination of life in old and worn out individuals. This removal of the older individuals from a population is said to i) prevent overpopulation; ii) leave more resources for those who are younger and stronger and; iii) by facilitating a more rapid turnover of generations, increase the pace of adaptive change.

Selection at the level of the group is not impossible[2]. Should the survival of the group as a whole be under threat from overpopulation, those groups within the wider population that age may survive while those that don't become extinct. However, it is now widely accepted that since selection at the level of the group must be much slower and weaker than at the level of the gene or individual, it is, except in rare circumstances, an unsatisfactory explanation for behaviour. The criticisms of group selection as applied to ageing may be outlined as follows.

A population which aged as a means of population control would be susceptible to invasion by cheats; a non-ageing mutant would - since it benefits from the ageing of other members of the population but does not itself pay the cost of ageing - be better able to propagate itself and thus spread throughout the population. It is also hard to see how the expression of genes for ageing could be regulated if not by reference to

some underlying progressive decline. As stated by Kirkwood & Melov[2], genes cannot easily 'tell the time'. Argument ii) above may be dismissed on the grounds of circularity as it presupposes that older individuals are less robust and therefore that some age-related deterioration in condition occurs. In response to argument iii) it has been pointed out that the rate of generational turnover is more heavily dependent upon the age of reproductive maturity than on lifespan and selection on germline mutation rate is in any case likely to be more important than generational turnover in determining the speed of adaptive change[2].

Finally, the idea of a program for decline, and in particular death, does not seem to concur with experimental data on senescence. Since senility is hardly ever seen except in protected environments, it is hard to see how a death program could have been selected for in the wild. Furthermore, although the set of changes seen with age are broadly reproducible within a species, it is rarely possible to predict from what intrinsic malfunction any one individual will ultimately die and this again is hard to reconcile with the notion of programmed death. If decline, but not necessarily death, were programmed, we would expect to find some mutation or combination of mutations which inactivate the program. Despite extensive mutant screens in many laboratories, and the identification of a multitude of mutations that affect the rate of ageing, none have ever been found that will eliminate it.

1.3.2 Ageing for the benefit of kin

Leaving aside the idea of group selection, some researchers have suggested that a theory of programmed ageing may be built upon the idea of kin selection instead. In this scenario, ageing will increase the inclusive fitness of the ageing individual, rather than benefitting the population at large. Ageing would evolve by such a means if and only if the fitness benefit provided to kin multiplied by the relatedness between the ageing individual and the said kin exceeds the fitness cost borne by the individual itself.

Some experimental support for the idea of programmed ageing for the benefit of closely-related individuals has come from work in the yeast *Saccharomyces cerevisiae*[11]. Yeast colonies typically grow under conditions of tight spatial constraint. When cell density is low and nutrition plentiful, the colony experiences

rapid growth; once cell density is high and nutrition limited, the colony transitions into a low-metabolic stationary phase. The majority of cells within such a colony die in a manner resembling mammalian apoptosis (programmed cell death), with surviving members then utilising nutrients released from their dead neighbours to initiate regrowth. These colonies have been likened to a super-organism - cells are, barring mutations, genetically identical - so the cells that die are certainly helping their kin. Moreover, the subpopulation which regrows seems to be better adapted to the nutrient-poor environment, so programmed ageing could in this case provide a means of hastening adaptive change. The theoretical plausibility of attributing this phenomenon to programmed ageing has been confirmed by means of computational models[11].

1.4 Non-programmed ageing

Although theoretically possible, the conditions under which programmed ageing may evolve do not apply to the vast majority of species. Other explanations are therefore needed as to why senescence is so widespread.

Theories of non-programmed (non-adaptive) ageing assume no fitness benefit from ageing *per se*; all other things being equal, a non-ageing organism will be fitter than an ageing one. There is consequently no positive selection for a well-defined program of ageing. The three major theories of non-programmed ageing which have guided most recent research are Medawar's mutation accumulation theory[12], Williams' antagonistic pleiotropy theory [13]and Kirkwood's disposable soma theory[14]. These three are not mutually exclusive and, in the case of the latter two in particular, share a number of common features.

1.4.1 Medawar's mutation accumulation theory

Medawar's theory hinges on one key insight which is also fundamental to the two later theories. He realised that, if organisms are always at risk of death from extrinsic hazards (such as drought, starvation or predation), survivorship will be a decreasing function of age even in a non-senescing population[12]. Since a lower contribution to overall fitness is thus made by older age classes, the ability of selection to optimise performance shows a monotonic decline with age. A 'selection shadow' is said to exist at ages which an organism is unlikely to survive to in the wild, with late-acting germline

mutations escaping selection and accumulating in the genome. The vast majority of such mutations will be deleterious and in a protected environment they manifest themselves as a progressive deterioration in condition.

Mutation accumulation is able to explain the well-documented correlation between extrinsic and intrinsic mortality rates. Cross species comparisons reveal slower rates of ageing in species subject to fewer external hazards, for example, in those who are able to evade or repel predators in consequence of their large size, capacity for flight or protective armour. Birds and bats are significantly longer-lived than non-flying mammals of the same size. Hibernating bats - who are protected from predation, starvation and extreme weather over winter - live an average of six years longer than non-hibernating species[15]. This correlation between extrinsic and intrinsic mortality rates seem to hold within a species as well. Following the separation of Sapelo Island from mainland South Carolina, opossums on the island - which was unable to support their mammalian predators and therefore provided lower rates of extrinsic mortality - have evolved slower rates of increase in age-specific mortality over time[16].

The gene for Huntington disease seems a good contender for one that has been retained despite deleterious effects because its action is seen at ages where selection is weak (usually in post-reproductive individuals). There is also some evidence of greater heterogeneity in behaviour in late life as would be expected if late-acting genes evaded selection; variability in the timing of reproduction within the breeding season increases with age in mute swans.

However, mounting evidence that pathways affecting ageing are highly conserved seems to argue against mutation accumulation since any random accumulation of late-acting mutations would be lineage-specific. One of the criticisms of programmed ageing made above applies also to mutation accumulation, namely that the theory seems incomplete without some explanation of what governs the age-of-expression of a gene. If late-acting genes are switched on in response to changing cellular micro-environments with age then the theory assumes rather than explains senescence. In the case of Huntington disease a decline in, or for women cessation of, reproduction with age must be presupposed in order for the gene - which then acts post-reproduction - to escape selection.

1.4.2 Williams' antagonistic pleiotropy theory

Williams postulated the existence of genes which conferred a selective advantage in early life (for example, increased early life fecundity or survivorship) and a selective disadvantage in late life in the form of ageing[17]. A declining force of selection with age means that selection for the early benefit outweighs that against the later harmful effects and such genes will therefore spread. The ultimate rate of senescence for a species or population will depend upon the strength of selection for increased vigour in early life relative to that against deterioration in later life, this being largely a question of the extrinsic mortality rate. As extrinsic mortality increases, the probability of survival to old age is reduced (regardless of whether senescence occurs) and the force of selection in favour of robustness in old age is similarly reduced.

Leroi *et al*. compiled a list of potential candidates for genes displaying antagonistic pleiotropy[18]. These fell into five broad classes: those involved in gonadal regulation; insulin and insulin-like signalling (IIS); free radical scavenging; the response to heat shock and; apoptosis. A couple of examples are here outlined: dauer formation in the nematode *C. elegans* and p53 which can induce apoptosis. Dauer formation represents a type of developmental diapause in worms, seen under conditions of overcrowding and starvation. Although worms normally survive for only a few weeks, a decision may be made at the end of the second larval stage to enter a highly stress-resistant state known as a dauer in which the individual can survive for about three months before developing into an adult. Genetic mutations affecting genes involved in dauer formation extend lifespan (without the formation of the dauer) at the expense of reduced fecundity.

The apoptosis of defective cells within a multicellular organism provides a safeguard against cancer; such cells are removed before they become so damaged that they no longer respond to anti-growth signals from the rest of the body. However, excessive apoptosis will deplete the stem cell pool and, as the body's regenerative capacity is impaired, hasten senescence. Activity levels of the protein p53, an inducer of apoptosis, represent a trade-off between selection for longevity and against cancer. A gain-of-function mutation in p53 produced a premature ageing phenotype but an increased resistance to tumours in mice[18].

There is a lot of additional evidence for antagonistic processes; many interventions (genetic, environmental and pharmacological) which prolong lifespan do so at the expense of early life fitness. However, although such findings seem consistent with the theory, it is often not known whether these effects can be attributed to individual genetic loci.

Importantly, this theory does not suffer from the problem of how genes tell the time. The postulated antagonistic pleiotropic genes would have different effects in different somatic environments and the effects of a gene in early life may change the somatic environment in which it will then be during late life.

1.4.3 Hamilton's model of the declining force of selection with age

Formal mathematical underpinning of the theories of mutation accumulation and antagonistic pleiotropy was provided by Hamilton, who developed a quantitative model of how fitness is affected by a change in survival at any age[19]. The intrinsic rate of increase r - that is the rate of increase in the absence of any density-dependent checks on population growth - was taken as the measure of fitness. The probability of survival to age x (l_x) was defined as the product of the survival probabilities for each time interval between 0 and x, so $l_x = p_0 * p_1 * \ldots * p_{x-1}$ where p_a is the probability of surviving the ath time interval. Differentiation of r with respect to the logarithm of p_a gives a measure of the force of selection. This derivative was shown to decrease as a increases so that the strength of selection declines steadily with age: genes with positive effects earlier if life will be selected for more strongly than those with positive effects in later life.

Hamilton then considered the effect of this on the survival curve. If a genetic mutation reduces mortality at a specific age it will be favoured by natural selection and over subsequent generations spread through the population. This initially causes a rise in population size which is subsequently checked by environmental constraints on the carrying capacity, i.e. the maximum population size that the environment can sustain. The environmental constraints will slightly elevate the mortality rate at all ages so that the advantage initially felt at one age is effectively compensated for by increased mortality at other ages. As mutations with positive effects in early life are selected for more strongly, the mortality curve will over time be lowered in early life with a

compensatory increase at later ages. Hamilton concluded from this line of reasoning that ageing is inevitable.

Different mathematical formulations have more recently been developed which question the inevitability of ageing[20]. They suggest instead that, if there is a rise in fecundity post-maturity of sufficient magnitude to offset the decline in survivorship, then older age classes need not necessarily make a lower expected contribution to overall fitness and the force of selection need not decline with age. The biological validity of these formulations has, however, yet to be verified.

1.4.4 Kirkwood's disposable soma theory

The disposable soma theory [14]is perhaps the most influential theory for the evolution of ageing guiding current research. It assumes that organisms have evolved strategies for optimal allocation of a finite resource (usually energy) obtained from the environment. Importantly, it states that somatic maintenance - the suite of processes for the repair and replacement of damages molecules and cells - is energetically costly. Immortality would be achievable if sufficient energy were invested in somatic maintenance, however, this would come at the expense of other fitness-enhancing functions notably reproduction. A trade-off emerges between the fitness gains from immediate reproduction and the potential fitness gains from future reproduction, the latter depending on physiological condition in future breeding seasons. Since maintaining somatic integrity for longer than the organism is likely to survive in the wild (even if it does not senesce) is energetically wasteful, evolved investment in somatic maintenance will be less than is required for immortality[14, 21, 22].

According to this theory, ageing is the result of accumulated molecular damage, the rate of which is regulated through the effectiveness of somatic maintenance processes. The name derives from the fact that it is only the soma in which somatic defects can be allowed to amass; accumulated defects in the germline cells would not be compatible with the long-term survival of the lineage (although see section 1.4.6 below about ageing in unicellular organisms which do not have a separate germline and soma). Whilst the soma is required for reproduction to take place, the fact that it is not itself transmitted to the next generation means that it is in a sense disposable.

This theory is reminiscent of antagonistic pleiotropy in that early life benefits are traded against late life costs. However, whilst antagonistic pleiotropy is a genetic theory which postulates antagonistic effects resulting from the action of a single gene, the disposable soma theory predicts trade-offs at the physiological level for which a single gene need not be responsible. It is founded upon the basic premise of life history theory: the principle of allocation and the idea of trade-offs between biological functions. It therefore fits readily with a wealth of research into life history theory which demonstrate that trade-offs are ubiquitous in nature and that organisms will optimally partition limited energetic resources between competing biological functions.

A couple of conceptual points relating to all three of the theories of non-programmed ageing which have been outlined here are discussed below. The first is what we mean by extrinsic versus intrinsic mortality and what consequences extrinsic mortality may have for evolved rates of senescence. The second considers the implications that ageing in unicellular organisms has for our understanding of the evolution of ageing. Experimental and theoretical support for the disposable soma theory is then outlined in section 1.5.

1.4.5 Extrinsic and intrinsic mortality

Extrinsic mortality is defined as that which results from environmental hazards and to which all ages are equally susceptible. It is distinguished from intrinsic mortality which results from internal causes and to which individuals become more susceptible with age.

It has already been stated that rates of extrinsic and intrinsic mortality are positively correlated between species (section 1.4.1) and this correlation is considered one of the key predictions of the three theories of non-programmed ageing outlined above. This seems to follow directly from Medawar's insight that the force of selection declines with survivorship. If the probability of surviving to old age is reduced independent of an organism's behaviour - as would be the case when extrinsic mortality is increased - the ability of natural selection to optimise performance in late life and the value of late life performance relative to that in early life is reduced. This idea is accepted and quoted by many gerontologists and seems to concur with laboratory experiments in

which rates of extrinsic mortality have been manipulated as well as with what we know of senescence in the wild.

However, the idea has been challenged on theoretical grounds in a paper by Abrams[23]. He based his argument on indicators of the force of selection with age derived by Hamilton, which give the change in fitness associated with a change in survivorship or fecundity at a given age. Abrams showed that if the survival probabilities for all ages are multiplied by a constant factor, and no density-dependent effects on population growth occur, there will be no change in the optimal rate of senescence. The mathematics is not reproduced here but the logic behind this is as follows.

Consider an individual of age x, whose behaviour is optimised for a given environment and who now experiences an increase in extrinsic mortality rate. Assume that the probability of survival to age (x + 1) is reduced by a multiplicative factor þ. The expected number of offspring produced at age (x + 1) relative to those produced at age x is therefore decreased by a multiplicative factor þ also. Let us take as a measure of fitness the number of descendants far into the future. Since the survival of descendants at each age is multiplied by þ and those resulting from offspring produced at age x will have had to survive one period longer than those produced at age (x + 1), the fitness accrued from an offspring produced at age x relative to that from an offspring at age (x + 1) is reduced by a factor þ. These two changes - in the probability of producing an offspring at one age relative to another and in number of descendants far into the future which result from an offspring at one age relative to the other - cancel out. The relative fitness contribution of all age classes and therefore the rate of senescence remains the same.

The argument is mathematically sound but seems to be at odds with experiments in *Drosophila* and the nematode *C. remanei* that have manipulated extrinsic mortality and observed changes in evolved rates of senescence [24-26]. This discrepancy may stem from a difference in how extrinsic mortality has been defined. A change in extrinsic mortality should affect all ages equally and Abrams has taken this to mean that survival at all ages is altered by the same multiplicative factor. If some age-dependent increase in mortality was already at play, such a change would increase

absolute mortality at younger ages to a greater extent than at older ages. (To see why this is the case, suppose that mortality rates for ages m and n are 0.2 and 0.8 initially and then consider the effect of reducing survival by a factor of two. The survival probabilities now become 0.5*0.8 = 0.4 and 0.5*0.2 = 0.1 respectively and the corresponding mortality rates are 0.6 and 0.9. The additive increases in mortality are therefore 0.4 and 0.1 respectively.) This does not seem to be how many researchers have intuitively thought about extrinsic mortality and in the experiments referred to above it is an additive increase in mortality at all ages that is considered. The age structure of a population and consequently the strength of selection against senescence will change in response to an additive change in mortality rates as it will not in response to a multiplicative change in survival rates. It still seems reasonable therefore to consider differences in extrinsic mortality as one of the key factors driving differences in senescence.

Although it can sometimes be useful to consider the effects of purely extrinsic or intrinsic mortality separately, death will often be attributable to a combination of both extrinsic and intrinsic factors[27]. The risk of infection will, for example, depend upon the distribution of pathogens as well as the immune status of the potential host. Similarly, the threat of predation will depend both upon predator density and the susceptibility of an individual to attack, the latter usually being a function of age. Changes to age-dependent mortality, such as changing the relative facility with which a predator can overcome an old rather than a young individual, can have complex effects on the optimal rate of ageing. If mortality in older individuals alone were increased, this could favour a slower rate of ageing if the fitness costs from reduced fecundity in early life are outweighed by the fitness gains from delaying the onset of this age-dependent mortality. There is some evidence for this in guppies and worms[24, 25, 28]. If, on the other hand, the costs associated with delaying the onset of this age-dependent mortality are especially great, it may be better to accept a short life and simply invest heavily in reproduction in early life.

1.4.6 Ageing in unicellular organisms

The disposable soma theory in its original formulation saw the separation of germline and soma as a necessary condition for ageing to evolve. Although it was for a long time thought that unicellular organisms (which do not have a distinct germline and

soma) were immortal, it has now been shown that they do indeed age[29]. This holds true when their division is morphologically asymmetric - in which case one daughter is smaller and requires a protracted period of growth before dividing again - and also when it is morphologically symmetric - in which case no juvenile phase can be distinguished.

Stewart *et al*. showed that there is a *functional* asymmetry in the way an *E. coli* cell divides[30]. It stems from the fact that at one end of each rod-shaped cell a new pole is formed upon division whereas at the other end an old pole is retained. Over a number of generations, heterogeneity builds up with respect to the ages of the poles carried. Lineages which receive older poles exhibit a reduced growth rate compared to those with newer poles. When the cell divides there is initially a choice as to whether damages molecules are preferentially retained by one daughter or evenly distributed between both. An asymmetric segregation of damaged molecules is expected if the increased vitality of the daughter who doesn't inherit damage exceeds, in fitness terms, the loss in vitality incurred by the other. Each daughter then chooses how much of her energy budget to invest in repair and replacement of any damaged molecules and how much in growth and division. A greater investment in repair will reduce the number of daughter cells in the near future but reproductive capacity will be retained for longer. Despite the lack of any division between germline and soma, we still see a division of labour here; one cell carries the greater responsibility for future growth and the other bears the burdens of repair and reduced efficiency.

Once the potential for division of labour is realised, it is easy to reconcile this with the disposable soma theory. The reasoning behind the disposable soma theory - that all organisms sustain damage, that investment in maintenance is a choice and, that investment decisions have evolved so as to maximise fitness in the environment in which an individual's ancestors lived - applies to unicellular as well as multicellular organisms.

The feasibility of this explanation for ageing in unicellular organisms has been confirmed computationally. Watve *et al*. developed a Leslie matrix model of growth in unicellular populations with a view to assessing whether conditions could arise under which we would expect to see immortality[31]. (The Leslie matrix gives the age

structure of a closed population at time t in terms of the age structure at time (t - 1) and the mortality and fecundity rates for each age class.) All cellular components were subject to ageing and there were assumed to be m distinct age classes to which components could belong. The growth rate of the cell (the rate of synthesis of new components) was therefore dependent upon the age distribution of its cellular components, with an older age distribution corresponding to a slower growth rate. The authors considered the consequences of two limiting cases: those of perfectly asymmetric and perfectly symmetric division. In the first case, one daughter cell receives completely new components at each division and all old components (and therefore the cell carrying them) die upon reaching age class m. In the second case, both daughters receive an identical age distribution of components at each division and upon reaching age class m components either accumulate or are repaired at a cost to the cell. Simulations for these two division strategies were run for 100 generations by which time a stable age distribution was attained. The asymmetric division strategy gave a higher growth rate whilst the symmetric division strategy gave a higher growth yield, that is, a higher ratio of living components to total components synthesised. It was therefore concluded that more symmetric division is advantageous under nutrient poor conditions when optimising growth yield may be more important for survival than growth rate. This has subsequently been confirmed experimentally; *E. coli* grown in oligotrophic environments show greater morphological and functional symmetry[32]. Immortality may therefore be possible for *E. coli* when resources are scarce.

1.5 Testing the disposable soma theory

1.5.1 Experimental support for a trade-off between maintenance and other functions

The existence of a trade-off between somatic maintenance and other fitness-enhancing functions may be tested in at least three ways. Firstly, correlations may be sought between selective pressures and life-history characteristics (or simply between different life-history characteristics) in natural populations. Secondly, selective pressures may be applied to laboratory populations and subsequent evolutionary change recorded. Thirdly, the effect that interventions which target a given characteristic have upon other life-history traits may be observed. These three lines of

evidence are considered in turn with reference to a trade-off between maintenance and reproduction (which has received more attention than trade-offs with other functions). Some evidence of a trade-off between maintenance and growth rate is presented in section 1.6.

Evidence for an inter-specific correlation between extrinsic and intrinsic mortality has already been presented (see sections 1.4.1 & 1.4.5). A higher extrinsic mortality constitutes a selective pressure in favour of higher early life fecundity, as the probability of surviving to reproduce at later ages is lower regardless of investment in maintenance, and a concomitant reduction in longevity is therefore consistent with a trade-off between the two functions.

Correlated changes in life-history traits may also be seen in a single species in response to environmental changes. The developmental of a long-lived dauer in *C. elegans* under conditions of low nutrition and overcrowding has already been described (section 1.4.2). Two distinct longevity phenotypes are seen in worker honey bees; during the summer months when the colony reproduces worker lifespan is greatly reduced compared to during the winter months[5]. In both cases we see a switch in strategy away from reproduction and in favour of survival in response to unfavourable conditions.

An inverse correlation between early life fecundity and lifespan has also been observed in humans[33]. Analysis of data from the British aristocracy found that for women who survived until menopause (and therefore from whom death in childbirth could not skew survival) the longest-lived had the fewest children and tended to be older when they birthed their first child. These trends remained after correction for spousal lifespan which was used as a proxy for environmental impact on survival. The positive correlation between age at first birth and lifespan also remained after correction for total number of offspring and was therefore not simply the result of a lower total reproductive investment.

In laboratory-based experiments, selection for early reproduction in Drosophila - by only allowing offspring born to parents in early adulthood to contribute to the next generation - led to a significant reduction in lifespan[26]. Conversely, when only eggs laid in late adulthood were allowed to contribute to the next generation, flies evolved

with extended lifespan and reduced early fecundity. Experiments such as these are, however, unable to distinguish between the effects of parental age at breeding and longevity; if the act of reproduction itself incurs some disadvantageous age-independent effect, a difference in longevity between early and late reproducing flies may emerge without any difference in underlying ageing rate. One elegant experiment by Zwaan, Bijlsma & Hoekstra controlled for any effects of early life history by selecting directly for longevity[34]. The progeny from pairs of flies were split into two groups. One was kept at 20^{o}C at which temperature they age slowly and the other at 29^{o}C at which they age rapidly. The second group were not allowed to reproduce and their lifespans (necessarily independent of any differences in early life fecundity) were recorded. The siblings of those who were especially long-lived or short-lived were then separated and bred. Assuming only that lifespan at 20^{o}C correlates with that at 29^{o}C, longevity was thus selected for directly. After six generations of this selection, a significant negative correlation emerged between lifespan and fecundity over the whole of adult life.

A range of interventions, genetic, pharmacological and environmental, are known to have opposing effects upon lifespan and reproductive rate. The daf-2 mutation in *C. elegans* doubles the lifespan of worms in the laboratory and confers a reduction in fertility in early adulthood[35]. Importantly, these mutants are rapidly out-competed by wildtype worms in their natural environments suggesting that the evolved balance between survival and reproduction in the wildtype in indeed optimal.

One group of researchers in the Netherlands manipulated clutch sizes in the European kestrel *Falco tinnunculus* as a means of altering investment in reproduction (ref in [36]). When clutch size was increased, the number of offspring reared from that breeding season rose as did the number of grandoffspring later produced from that clutch. This implies that a greater parental investment was elicited rather than simply a reduction in offspring quality. However, survival to the next breeding season was lowered so a greater investment in current reproduction came at the expense of lifespan and consequently future reproduction. When clutch size was reduced the opposite was seen: the reduced investment in current reproduction was associated with increased survivorship to the next breeding season. Birds whose clutches were manipulated (in either direction) recorded a lower lifetime reproductive success when

compared to controls. Clutch size therefore seems to be adapted to maximise Darwinian fitness.

Dietary restriction is perhaps the most consistent intervention known to extend lifespan. It works in worms, flies, mice, some rotifers and fungi and, possibly rhesus macaques[37]. From an energetic viewpoint, this may at first appear counterintuitive. If ageing occurs because investment in maintenance is lower than required for immortality, it may not seem that a reduction in the total energy budget can be compatible with longer life. However, an adaptive explanation for lifespan extension under dietary has been offered. If the probability of successful reproduction is very low during periods of scarcity, it may benefit the adult to reduce or completely suspend reproductive activity until conditions improve thereby freeing up a considerable resource which may be diverted to somatic maintenance. The probability of surviving a period of food shortage is thereby increased and reproductive capacity is retained for longer so the organism can breed when conditions are again favourable. If this response is indeed adaptive, we expect to see it only in species frequently exposed to short-term food shortages and in whom juvenile survival is reduced to a greater extent than adult survival by such shortages. In keeping with this, lifespan extension under dietary restriction is not seen in tropical squirrel monkeys who are not exposed to famine in their natural habitat[38]. For sporulating fungi and certain species of rotifer, adult mortality exceeds juvenile mortality during times of famine and in these species reproduction is maintained or increased and survival decreased under dietary restriction[39].

1.5.2 Evidence for molecular damage as the proximate cause of ageing

The disposable soma theory makes specific claims as to the proximate as well as the ultimate cause of ageing. Senescence is said to result from the accumulation of a host of stochastic molecular defects which directly underlie the ageing phenotype at the level of the organism.

The majority of gerontologists are now in agreement that molecular damage causes ageing and telomere erosion; protein misfolding and aggregation; DNA copying errors and base modifications and; lipid peroxidation, have all been linked to senescence. Defects may result from both exogenous and endogenous insults, for example

ultraviolet radiation and free radicals created as by-products of the electron transport chain. Damage accumulation is countered by a wide spectrum of somatic maintenance systems, notably the proteasome system (responsible for the degradation of old and misfolded proteins); the DNA repair systems (responsible for the excision of faulty bases/nucleotides and the repair of single and double strand breaks) and; the antioxidant system (responsible for the enzymatic removal of free radicals)[21].

There is correlational evidence for an accumulation of somatic defects with age, for more efficient repair in longer-lived species and for lower absolute damage levels and/or a shallower increase in damage load with age in longer-lived species. When cell lines from eight mammalian species were challenged with a range of oxidative and non-oxidative stressors, a clear correlation emerged between stress resistance and the lifespans of the species from which cells were taken. Cell lines from two long-lived mouse strains - the Ames dwarf and Snell dwarf - are more resistant to cytotoxic treatments than are those from standard laboratory strains. No detectable reduction in telomere length with age occurs in the long-lived storm petrel, although it is well documented in many shorter-lived species of bird. The naked mole rat, a subterranean eusocial rodent noted for being exceptionally long-lived given its size, displays a much higher level of proteasome activity than the closely-related mouse and furthermore no detectable increase in carbonylated proteins with age[15].

If damage does cause ageing, we would also expect that interventions which alter the damage load or the efficacy of the repair systems would bring about a change in lifespan. It is perhaps surprising then that the administration of antioxidants has repeated proved unsuccessful at extending longevity[40]. However, it is not always clear whether oxidative status within cells is actually changed by such interventions and they do, in any case, target a single function without changing the overall setpoint for levels of repair and maintenance. There is some evidence that manipulations to reproductive effort are accompanied by the expected changes in molecular damage or repair. In zebra finches, protection from oxidative damage was compromised by artificial increases in brood size[41]. Similarly, the artificial stimulation of egg production impaired resistance to oxidative stress in female *Drosophila*. Gene expression profiling has also indicated a broad spectrum upregulation of somatic maintenance functions in response to dietary restriction[42].

1.5.3 Theoretical support for the disposable soma theory

As for other areas of biology, mathematical models can provide valuable insights into the evolution of ageing. Two such models have already been described: Hamilton's model of the declining force of selection with age has provided one of the cornerstones of how we understand ageing and Watve *et al*.'s model of symmetric and asymmetric division in unicellular organisms has demonstrated that senescence may be understood in terms of optimal energy allocation for unicellular as for multicellular organisms. Models may be useful for a number of reasons. They can serve to clarify our thought on a particular issue since we are forced to make our assumptions explicit. They can provide quantitative predictions for how a system should behave which may guide experimental work. For complex problems it is not possible to intuit how a system will behave so we must make predictions based on mathematical simulations. Models can also be a cost- and time-effective means of exploring the effect upon a system of systematic changes to assumptions or parameters.

Drenos & Kirkwood developed a mathematical model to simulate the effects of varying investment in maintenance[43]. They assumed that a greater investment in maintenance carried both benefits and costs. The former were an increased longevity and slower decline in reproductive capacity throughout adulthood. The latter were a delayed attainment of reproductive maturity (since maintenance is traded-off against growth during the juvenile period) and a reduced peak reproductive rate (since maintenance is traded-off against reproduction during adulthood). They allowed for the theoretical possibility of immortality; if sufficient energy were invested in maintenance no increase in mortality nor decrease in fecundity would be experienced with age. However, such a strategy of immortality was, for a wide range of model parameters, always less fit than a strategy of senescence. This confirmed that the basic logic behind the disposable soma theory is sound and that it is robust to a range of environmental and physiological parameters.

Whilst models such as this are useful in understanding optimal behaviour in broad terms, if we want to explore differences between individuals differentiated by state, a different approach is required [44]. By state, is meant any characteristic of the organism or its environment that may affect its behaviour such as age, size or season. The relative costs and benefits from a particular action may vary with state; as an

organism gets older, for example, its chances of survival are reduced regardless of current investment in maintenance and it may do better to invest a greater proportion of its available energy in reproduction. State may also constrain the set of allocation decisions from which an individual must choose. Individuals in better condition may, due to an increased foraging success, have more energy available for both maintenance and reproduction. We do not therefore necessarily see a negative correlation between the levels of investment in each even if there is a trade-off between them[45]. Only if one function were manipulated would we see a change in the other as, for example, was seen following brood size manipulations in the kestrel (see section 1.5.1).

Dynamic programming - the principles of which are outlined in chapter 3 - is a means of computing a series of state-dependent decisions made at regular time intervals throughout an individual's life and its usefulness in modelling optimal ageing rates in particular has already been established. It has been used to confirm that a suspension of reproduction and increased investment in maintenance in response to temporary caloric restriction may be selectively advantageous in the house mouse[46]. It has also been applied to the problem of human menopause[47] and of seasonal morphs whose longevity varies considerably in the butterfly *Bicyclus anynana[48]*.

The application of dynamic programming to other questions of optimal allocation will likely be a fruitful avenue for future research. Of particular interest would be the consideration of more complex trade-offs than that between maintenance and reproduction alone; the dependency of optimal behaviour upon that of others and; the impact of decisions upon inclusive rather than individual fitness. The current work provides some contribution to this end. The particular focus is on the relationships between growth and ageing and two dynamic programming models are presented in chapters 4 and 5. The first seeks to determine whether differences between the sexes in terms of both growth and longevity can be explained by optimal energy allocation between growth, maintenance and reproduction in baboons. The second asks whether accelerated growth at the expense of reduced longevity may be an optimal response to early life adversity in humans. An overview of the evidence and theory motivating each of these pieces of work is given in the relevant chapters but some background on the associations between size/growth and longevity is covered in the

next section followed by an outline of the competing hypotheses that have been put forward to explain differences in lifespan between the sexes in section 1.7.

1.6 Growth and longevity

1.6.1 Inter-specific relationships between size and longevity

It has been suggested that inter-specific correlations between the major life history traits are so strong and predictable that almost all life forms can be arranged along a one-dimensional fast-slow continuum. 'Fast' species have short lifespans and fast rates of ageing; are small; mature at relatively young ages and have high fecundity. The latter is manifest as both a high productivity per reproductive event and a short interval between successive reproductive events; the proportion of offspring from a fast species which survive to maturity is typically low and the production of a large number of offspring each of whom receive relatively little parental investment represents a strategy of bet-hedging. 'Slow' species show the opposite set of traits: they are long-lived; large; mature at relatively old ages and; have fewer offspring in each of whom they invest heavily. For them the emphasis is on quality rather than quantity of offspring.

The correlation between size and longevity in particular has long been appreciated[49]. The rate of living theory sought to explain this with reference to metabolic rates. Larger organisms have lower metabolic rates per unit body mass and some early comparisons between species suggested that lifetime metabolic rate per unit mass might be invariant. It was therefore hypothesised that metabolism directly influenced the rate of ageing. Not all species fall neatly into the arrangements predicted by either the fast-slow continuum or the rate of living theory - despite being large and long-lived trees produce many low-quality offspring and birds live longer than mammals of the same size despite having higher metabolic rates - however, the consistency with which many species do is still striking[49].

If we consider the costs and benefits associated with growth, some insight into why these traits are so often correlated can be gained. The cost of growth in terms of a longer developmental period and an older age at first reproduction could not be borne by species in whom - either due to a high rate of extrinsic mortality or because

investment in maintenance was compromised by that in other functions - average lifespan was short. We would therefore expect to see a large body size only in species who are long-lived. Size is often its own protection against extrinsic mortality and may be selected in part for this reason; larger species tend to have fewer predators, enhanced fasting endurance and can be better able to outcompete other species for contested resources. (In some instances it is, however, possible that size can bring an increase in extrinsic mortality due to an increased detectability, parasite burden or heat stress.)

Another clear benefit to size is a greater absolute energetic investment in reproduction. This can manifest itself as a greater expenditure per offspring and a prolonged period of parental investment can allow for greater social learning, the development of more complex structures and therefore a more competitive adult. This can alternatively manifest itself as an increase in offspring number as is seen, for example, in fish. The increase in fecundity with size seems to have favoured the evolution of indeterminate growth in fish, i.e. the continuation of growth post-maturity. The relative fitness contribution of older age classes is increased as a consequence of indeterminate growth and this can (partially) offset the declining force of selection with age due to decreasing survivorship. Many fish show the type III survivorship curve described in section 1.2, follow a strategy of high investment in maintenance and age very slowly. Indeterminate growth is seen in a range of other species including many plants and some crustaceans, lizards and snakes. With their continual investment in growth and slow rates of ageing, these may represent the ultimate slow life history.

1.6.2 Intra-specific relationships between size and longevity

The positive correlation between size and longevity seen between species is reversed within species, with the smallest individuals enjoying the longest lifespan. This is especially well-documented in dogs where artificial selection has produced breeds which differ in size over an order of magnitude. Dogs display a Gompertz mortality curve and analysis of the mortality profiles for different breeds has shown that the difference in longevity is due to an increase in the rate at which mortality rises with age (β in section 1.2) as opposed to an increase in baseline mortality, an earlier onset of ageing or an increased mortality in early life[7].

There are for many species significant differences in body size and longevity between the sexes with the smaller sex usually, although not always, being the longer-lived. In humans, the discrepancy in lifespan between the sexes is attributable to a difference in the elevation rather than the slope of the mortality curve; women do not age more slowly but are more robust at every age[1]. A number of hypotheses have been put forward for the sex difference in longevity not all of which connect this to a difference in size and these are covered in the next section.

The reasons for a negative intra-specific correlation between size and longevity have yet to be fully elucidated, however, two possible explanations have been submitted. Firstly, if cell number is greater in larger members of a species, there are more 'targets' in which carcinogenic mutations may occur. Secondly, if developmental times are essentially the same for smaller and larger members of a species, then the latter must experience a faster rate of growth; evidence for a trade-off between lifespan and growth rate is presented in the next section.

One question of particular interest is whether the reduced longevity at the level of the organism is mirrored by a reduced longevity at the level of the cell. Research in the honey bee *Apis mellifera* suggests that this might be the case[50]. Bee colonies have been likened to a super-organism in which queens and drones are analogous to germline cells and workers to somatic cells. A lesser degree of integration than is seen in unitary organisms makes it easier to observe any differences in the longevity of component parts. By monitoring tagged workers who were randomly assigned to either small or large colonies, worker longevity was found to be reduced as a direct consequence of increased colony size.

1.6.3 The growth rate-lifespan trade-off

When investigating correlations between size and longevity, it can often be difficult to disentangle the effects of final size attained from those of growth rate. If functions are traded-off against each other as predicted by the disposable soma theory then, all else being equal, a faster growth rate would leave less for maintenance and we would expect to see a reduction in lifespan. Faster rates of growth would be favoured when the fitness gains from a younger age at first reproduction become more important than the losses from a shorter reproductive life, as might be the case when a high rate

of extrinsic mortality means that long-term survival is unlikely regardless of allocation strategy.

A direct demonstration of the growth rate-lifespan trade-off has been performed in three-spined sticklebacks[51]. Growth rate is temperature-dependent in these organisms, with the rate of resource acquisition and subsequent utilisation of energy falling as temperature does. A brief cold spell will reduce growth rate at the time it is experienced and trigger subsequent catch-up growth upon return to ambient temperatures; a brief hot spell will increase growth rate at the time it is experienced and trigger a subsequent slowing down of growth upon return to ambient temperatures. Importantly however, the change in growth rate that occurs at the time when a temperature change is experienced does not affect allocation to maintenance and therefore has no effect upon longevity. By applying brief temperature manipulations during the juvenile period, it was shown that catch-up growth induced a 14.5% reduction in lifespan and slowed-down growth a 30.6% increase in lifespan. Temperature manipulations during the growth period do not affect final size attained so these alterations in lifespan could be attributed solely to the rate of growth.

There is also evidence for catch-up growth being associated with decreased lifespan in mammals. In male mice who have experienced foetal growth retardation, a subsequent compensatory acceleration in growth reduces longevity whilst continued growth restriction prior to weaning extends it[52]. In humans too, a short-term period of scarcity in early life followed by a period of catch-up growth upon return to a more plentiful environment results in a significantly reduced life expectancy and a greater susceptibility to a range of chronic conditions including obesity and type II diabetes[53]. It has been hypothesised that the foetus/infant commits to a growth trajectory best suited to its current environment. If the adult environment does not match the early life one, a mismatch between phenotype and environment is said to occur. In a harsh nutritional environment it pays to lay down fat readily, however, in a plentiful environment such a strategy is maladaptive, leaving the individual prone to a number of health risks. The model presented in chapter 4 explores responses to early life adversity and and a fuller account of the evidence for such a response and the theory behind it is presented in the introduction to that chapter.

1.7 Sex differences in longevity

Sexual dimorphism in longevity is widespread, with females usually being the longer-lived sex. In humans this is especially well-documented and the difference is seen across multiple societies, over a number of centuries and in both developed and developing countries. Almost 90% of all known supercentenarians are women and the oldest confirmed person to have lived was a women[1]. Greater female longevity is not universal however and for many species there is not sufficient data available to say which sex is longer-lived.

A number of non-mutually-exclusive hypotheses have been put forward to explain sex differences in longevity and these are outlined below. Some of these derive from genetic differences between the sexes, some from mechanisms of inheritance/information transmission and some from behavioural differences[54].

1.7.1 The unprotected X hypothesis.

According to this hypothesis, the homogametic sex (that which has two copies of the larger sex chromosome) will be on average longer-lived since the effect of any recessive, deleterious mutation on such a chromosome may be masked by a dominant counterpart[54]. In the homogametic sex, such a mutation must always affect phenotype. In keeping with this hypothesis, females (XX) enjoy greater lifespan than males (XY) in humans and many closely-related species; in the majority of bird species, males (ZZ) live longer than females (ZW) and; the *C. elegans* hermaphrodites (XX) lives longer than males (XO). Furthermore, inbreeding depression - in which the genetic similarity of parents means that recessive deleterious traits are more readily manifest - is known to reduce lifespan in many species. However, exceptions to the rule do exist: in golden hamsters and guinea pigs, which share with most other mammals the XX/XY sex-determination system, males are longer-lived than females.

1.7.2 Asymmetric inheritance of cytoplasmic genomes.

Cytoplasmic genomes - namely mitochondria and chloroplasts - are inherited through the female line, that is, each individual receives them only from his/her mother and a male may not pass them on to any of his offspring[55]. This has important consequences for how natural selection can act upon cytoplasmic genomes. Any mutation in such genomes which reduces female fitness will be less successful at

propagating itself into the next generation, but any that reduces male fitness alone will be outside the reach of natural selection. (Male-specific cytoplasmic mutations would affect the success their owner has in getting copies of his nuclear genes into the next generation but this is not to the detriment/benefit of the cytoplasmic genes themselves, since they are not passed on.) It is therefore possible for male-specific cytoplasmic mutational loads to accumulate.

Consistent with the idea that mitochondrial function if optimised for a female background, the comparison of a number of mitochondrial haplotyes in an isogenic nuclear background in Drosophila found that variance in absolute longevity and rate of senescence after correction for initial robustness was greater in males[55]. However, as mentioned above there are species (golden hamsters, guinea pigs) in which males are longer-lived despite the maternal inheritance of cytoplasmic genomes.

1.7.3 Maternal effects optimised for female offspring.

Whilst both sexes make an equal (nuclear) genetic contribution to their offspring, it is often the mother who supplies the immediate environment in which the offspring will develop. She provides, for instance, a host of mRNAs and proteins stored within the ovum and needed for the correct development of the embryo. The genotype and environment of the mother can in this way affect offspring phenotype independent of the genotype and (non-maternal) environment of the offspring. It has been suggested that maternal effects may be optimised for female offspring and that one of the ways in which this shows itself is in the greater lifespan of females[54]. Whether maternal effects have some differential effect on longevity remains to be seen but they could not explain why in some species it is males who are the longer lived.

1.7.4 A protective role for oestrogen.

Female Wistar rats - whose lifespan is approximately 16% longer than that of their males - display higher levels of the antioxidant glutathione peroxidase and reduced oxidative damage in the mitochondria of their livers in comparison to males[56]. Interestingly, oxygen radical production was increased up to the level of males following ovariectomy and this increase could be inhibited by the subcutaneous administration of oestrogen. This seems to imply a protective role for oestrogen in the defence against ROS-induced damage. As for some of the other hypotheses here

listed, it can potentially explain the 'rule' that females live longer than males but not the exceptions where males live longer than females.

1.7.5 Males engage in more risky behaviour.

In many species, males seem more inclined towards a range of risky behaviours. They tend to be the most aggressive sex and are therefore more often injured or killed in fights. In humans, records show that males constitute a disproportionate number of deaths from road traffic accidents, firearms and extreme sports. A difference in longevity between the sexes remains, however, after correcting for deaths directly attributable to risky behaviours, although it is possible that this follows indirectly from behavioural differences[54]. The sex which engages most frequently in dangerous activities and is thus more likely to die young may place a lower premium on somatic maintenance.

1.7.6 Differences in body size.

Males are typically larger than females and as already noted, the larger members of a species do not tend to live as long. It might then be hypothesised that it is the link between body size and longevity that mediates that between sex and longevity. In keeping with this, male golden hamsters, who are longer-lived than females, are also the smaller sex. As previously discussed (section 1.5) it is also well documented that reduced activity through growth factor and insulin signalling cascades can effect an increase in longevity.

An explanation of sex differences in longevity based on body size may overlap with many of the other hypotheses here listed. As larger individuals are usually more dominant within a group, they may be more likely to engage in risky behaviour. Sex-specific selective pressures may favour greater investment in growth in males even at the expense of somatic maintenance (see section 1.7.8). Larger individuals also tend to suffer higher disease burdens, (see section 1.7.7 below).

1.7.7 Differences in disease burden and/or immunity.

A number of suggestions have been put forward as to possible links between ageing and disease. Due to their greater foraging needs, the larger sex - usually males - will be exposed to a higher parasite and pathogen burden. Male Soay sheep are normally

larger, shorter-lived and more heavily infested with intestinal parasites than females. Consistent with the hypothesis, the difference in mortality rates between the sexes disappeared when parasite burdens were artificially reduced[57].

Sex differences in longevity may be in part mediated by testosterone, which acts as a positive regulator of male secondary sexual characteristics (SSCs) and a negative regulator of the immune system. Testosterone levels seem to represent the resolution of a trade-off between the losses in fitness from reduced immune function and the gains (improved mating success) from enhanced SSCs[58, 59]. Interestingly, there is some evidence that castration may increase lifespan in males, which would also be consistent with the idea that testosterone enhances the rate of ageing[60].

However, it seems unlikely that differences in disease burden or immunity could underlie all differences in longevity between the sexes. Many captive populations, who experience essentially disease-free environments, display the same differences as their wild-living counterparts. Human females are, at all ages, less prone to a range of life-threatening conditions such as cancer, heart disease and chronic obstructive pulmonary disease (COPD), none of which can be solely attributed to differences in immune status.

1.7.8 Sex-specific selective pressures.

Sex differences in longevity are expected to evolve when the fitness benefits of long life relative to those of other attributes (for example, size) differ between males and females[61]. Differences in lifespan are then driven by sex-specific selective pressures acting on the allocation of available energy to competing fitness-enhancing functions. (Several of the explanations offered above may in fact imply the existence of sex-specific selective pressures but the idea is presented here with explicit reference to the disposable soma theory and the notion of trade-offs.)

The tendency for females to be the longer-lived may stem from the fact that females often provide the majority of offspring care and do not compete with each other for mates as males do. Since, for many species, a female's fitness is closely correlated with her ability to support successive offspring/litters over a period of time, she places a high premium on maintaining her soma in good condition over a relatively long life. In contrast, a male's fitness is often curtailed by his ability to secure mates and he

therefore invests considerable capital in physical/behavioural attributes which improve his desirability or competitiveness as a mate, even at the expense of somatic maintenance. Males and females are 'choosing' different rates of ageing as part of an optimal life history.

Consistent with this, differences in lifespan between the sexes tend to be lost when differences in reproductive strategies are. In monogamous species, where both sexes invest in offspring care and extra-pair matings are rare, life expectancy for males and females is far closer than for polygamous species[62]. This is often despite sharing the same sex determination system.

1.8 The scope of this thesis

Two models are presented in the current work, both of which explore intra-specific relationships between growth and longevity and ask whether these can be explained within a framework of trade-offs between somatic maintenance and other fitness-enhancing functions in keeping with the disposable soma theory. Both models are of dynamic behavioural games, that is, they apply dynamic programming to the decision processes of individuals who interact with each other and for whom the outcome of a particular strategy will depend on that employed by others. The principles of dynamic programming and game theory, and how they are combined within dynamic behavioural games, are outlined in chapter 3.

Chapter 4 presents a model of energy allocation between growth, maintenance and reproduction in the baboon. This has reproduced the sexual dimorphism in longevity and body size seen in wild baboons and provides support for the idea of sex-specific selection pressures driving different ageing phenotypes in males and females. A number of other interesting findings have also emerged from this model relating to the force of selection with age, the decline in mass which frequently occurs with age and the optimal sex ratio.

A model of human life history in response to a changing environment is presented in chapter 5. This has been constructed in order to explore the possible adaptive basis for an acceleration in life history following early life adversity. Results from this have highlighted both the effect of internal state and prediction of the future environment in explaining the changes to life history seen in times of adversity.

Chapter 6 details possible extensions to the two models which would - by taking greater biological complexity into account - increase their explanatory power. Firstly, an overview and evaluation of two recent theories of ageing - the hyperfunction theory and the heat dissipation limit theory - according to both of which differences in growth and/or size between organisms are considered central to understanding differences in ageing is given in chapter 2 below.

Chapter 2: Recent challenges to the disposable soma theory

2.1 Introduction

It is indicative of the strength of associations between growth and longevity that two recent evolutionary theories of ageing rely explicitly upon differences in body size or growth rate to explain differences in lifespan. The TOR-driven hyperfunction theory of ageing was proposed by Blagosklonny in 2006[114] and has since gained the support of some other prominent gerontologists[115, 116]. It asserts that ageing is caused by the unintended continuation of developmental programs; the same cell signalling pathways that drive growth and development in juveniles go on to drive senescence in adults. The speed of these two processes is therefore correlated, with selection for faster growth resulting in a faster rate of senescence.

The heat dissipation limit theory, proposed by Speakman & Król in 2010[117], was not initially developed as a theory of ageing but rather as a theory of what limits maximum sustained energy expenditure. It hypothesises that, in endotherms at least, this is the capacity for heat dissipation which is principally a function of size. Since its original conception it has been suggested that this could have important implications for the evolution of ageing, in particular that it could explain the fast-slow life history continuum without recourse to the principle of energy allocation.

If either of these were true, they would constitute a revolutionary shift in our understanding of ageing. They provide very different answers to why we age - and in the case of the hyperfunction theory how we age - than the disposable soma theory, which has for some time been one of the most influential in ageing research. An outline and analysis of each theory is given below.

2.2 The hyperfunction theory of ageing

2.2.1 Outline of the theory

Blagosklonny contends that ageing results from the unintended continuation of developmental processes (figure 2.1A). The same pathways that induce necessary cell growth and division during development go no to induce destructive hypertrophy, hyperplasia and hyperfunction post-development and this directly underlies the ageing

phenotype and age-related diseases[118]. At a molecular level, growth/development and later hyperfunction are driven primarily by TOR activity which stimulates anabolic pathways (protein synthesis, ribosome biogenesis etc.) and inhibits catabolic pathways (autophagy, proteolysis, mRNA degradation etc.). This unintended continuation of the developmental program is termed a quasi-program: it was selected for (programmed) during a different time in an organism's life than that at which its effects are now being seen[119].

There is no selection to switch off these growth-promoting pathways once development is complete because very few organisms live to an age where the negative consequences of their continuation are seen. In the wild, the vast majority of deaths result from extrinsic threats such as predation, infection and starvation; only in protected environments have deaths from intrinsic causes become widespread. Organisms have experienced selection for rapid growth and development, so as to maximise their chances of maturing and reproducing before succumbing to external threats, but no selection for the termination of growth-promoting pathways post-maturity[114]. As a general rule, the greater the external mortality threats, the faster the rate of development and therefore the faster the rate of ageing. However, hyperfunction does not require that programs necessarily continue at the same rate they did during development, simply that they continue at a higher rate than required - figure 2.1A shows two cases where functions continue at a faster or slower rate than they did during development. Any reduction in the rate at which the quasi-program proceeds, i.e. a reduction in TOR activity during adulthood, will result in a slower rate of ageing.

2.2.2 Evidence presented for hyperfunction

Two principal lines of evidence have been presented for hyperfunction. Firstly, interventions that increase or decrease TOR activity - either by genetic, pharmacological or environmental means - usually bring about a concomitant decrease or increase in lifespan. It is well established that dietary restriction extends lifespan in species as diverse as yeast, worms, flies and mice. Rapamycin - an inhibitor of TOR - also extends lifespan in all these model organisms; inhibits senescent morphology and the hyper-secretory phenotype in cell culture; rejuvenates haematopoietic stem cells and; has been shown to prevent cancer in humans and atherosclerosis in mice[122].

TOR knockdown more than doubles lifespan in *C. elegans* and over-expression of the tuberous sclerosis proteins (TSC1 & TSC2), which inhibit TOR activity, extends lifespan in *Drosophila*. Ames and Snell mice, which show reduced insulin-like growth factor 1 (IGF-1) signalling and consequently reduced TOR activity, enjoy significantly greater longevity than their wildtype counterparts. Further examples of such interventions - that have opposite effects on TOR activity and lifespan - are plentiful and a more comprehensive summary is given by Blagosklonny[114].

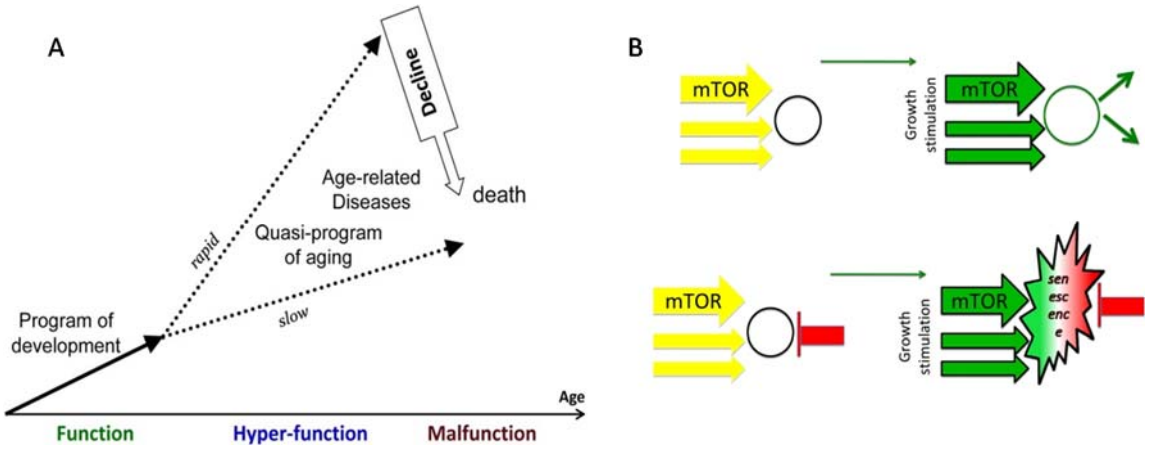

Figure 2.1: A: The continuation of developmental programs post-maturity (hyperfunction) and resultant emergence of age-related diseases and death at old ages (malfunction). B: The dependence of the senescent phenotype upon TOR activation. When TOR activity is low (as indicated by the yellow arrows), the cell will not become senescent regardless of whether or not a block upon the cell division cycle (as indicated by the red inhibitor) is in place. When TOR activity is high (as indicated by the green arrows), the cell will proliferate if no block upon the cell division cycle is imposed and will become senescent otherwise. Diagrams are taken from [120] and[121].

Secondly, hyperfunctions are said to be apparent when examining age-related changes in physiology. A number of examples have been given of age-related cellular changes which could be described as hyperfunctions including the hyperplasia seen in cancerous cells, hyper-secretion in endocrine cells and excess lipid accumulation in adipocytes[40, 114, 123]. Moreover, such changes to cellular phenotypes can often be related directly to changes at the tissue, organ and system levels. Osteoporosis results from the hyperactivity of osteoclasts relative to osteoblasts and atherosclerosis from the proliferation and hypertrophy of smooth muscle cells. Gems & de la Guardia [115]

have argued that a host of age-related changes in *C. elegans* seem to result from hyperfunctionality: the cuticle thickens with age due to the continued activity of the collagen-producing hypodermis cells; extra-neuronal processes are sprouted in mechanosensory and GABAnergic neurones; intrauterine masses appear in later adulthood possibly as a result of runaway endoreplication and; yolk production continues well after oocyte depletion.

It has also been pointed out that the dependency of cellular senescence on high TOR activity which can be seen *in vitro* is consistent with the hyperfunction theory[121]. If the cell division cycle (CDC) is blocked and TOR activity is low, cells will enter the quiescent state. This is reversible: if the CDC block is removed and growth factor stimulation applied, these cells are again able to proliferate. In contrast, high TOR activity when the CDC is blocked will convert quiescence to senescence; cells become hypertrophic, exhibit a range of molecular hyperfunctions such as hyper-secretion and, have permanently exited the CDC (figure 2.1B).

It has been claimed that the hyperfunction theory can explain data that seem incompatible with the disposable soma theory[40, 124]. Blagosklonny claims that the genetic regulation of ageing is evidence for a program. Coupled with the fact that ageing is not widespread in the wild, and therefore does not seem to have been selected for, this is said to constitute a paradox the resolution of which requires the notion of a quasi-program[123]. Explanations based on optimal energy allocation for a) lifespan extension under dietary restriction and b) differences in ageing between the sexes have also been criticised. It seems counterintuitive that when the total energy budget is reduced, the energy allocated to somatic maintenance should increase or that the sex which usually invests more in offspring care should also find more to invest in maintenance. Overexpression of, or dietary supplementation with, antioxidants does not increase lifespan as might be expected if ROS-induced damage causes ageing. Finally, the consistency in the ageing phenotype (including for example atherosclerosis, osteoporosis and loss of skin elasticity in humans) is said to be hard to reconcile with the notion of random molecular damage; under the hyperfunction model, the aimless running on of developmental programs produces non-random organ and system damage. Most of these points have already been addressed in chapter 1 and the reader is referred to sections 1.3, 1.5.1 & 1.5.2. The supposed

paradox regarding the programmed/non-programmed nature of ageing disappears once we allow that the programming of somatic maintenance functions - or more generally resource allocation - will result in many genes whose effects modulate ageing rate without ageing itself being programmed. Austad compares ageing of organisms to that of vehicles in this respect[125]. A vehicle is designed to last a certain length of time and alterations to its design will produce changes in the average length of time it survives. However, this does not imply that the vehicle is designed to fail at a particular age or in a given way.

2.2.3 Contrast with the disposable soma theory

Both the hyperfunction theory and the disposable soma theory rely on Medawar's (1952) insight that the force of selection declines with age. Both are also reminiscent of Williams' (1957) theory of antagonistic pleiotropy. According to the hyperfunction theory, TOR, and all genes whose products converge upon the TOR pathway, are antagonistic pleiotropic genes[126]. If their action increases signalling through the TOR pathway, they increase fitness in early life and decrease fitness in late life; if their action decreases signalling through the TOR pathway, the opposite is true. However, one difference between hyperfunction and conventional antagonistic pleiotropy is worth emphasising. Williams postulated genes whose effects in early life were inseparable from their negative effects in late life. The fitness disadvantage in late life is felt by whatever proportion of the population survives to such ages but this is outweighed by the positive effect in early life since this is felt by a larger proportion of the population. In contrast, Blagosklonny states that selection is essentially neutral to the late-life effects of hyperfunction because their impact is only seen in protected environments. No 'brake' on TOR over-activity has evolved, not because this would be mechanistically impossible without a corresponding brake on TOR activity in early life, but because there is no selective pressure in favour of a brake in the wild[119].

The disposable soma theory predicts antagonistic energy allocation decisions: diverting energy from maintenance to reproduction (or other fitness-enhancing functions) in early life will increase expected fitness gains in the near future at the expense of potential fitness gains in later life and these two effects are mechanistically inseparable. Whilst the disposable soma theory sees ageing as a consequence of optimal energy allocation decisions throughout life, hyperfunction predicts that adults

sub-optimally allocate energetic resources with the continuation of developmental pathways driving useless biosynthesis.

The disposable soma theory is a theory of non-programmed ageing. There is no selection for the decline seen in ageing, but rather there is selection for investment in somatic maintenance for as long as the fitness gains from such investment exceed those from investment in other functions instead. Under the hyperfunction theory, ageing is also not selected for *per se* but results from the unintended continuation of programmed growth/development. Although termed a quasi-program, hyperfunction would be considered a non-programmed theory according to the definition given by Kirkwood & Melov [2]as it does not claim any adaptive benefit (see chapter 1).

These theories make different predictions as to the molecular/cellular causes of ageing: excess biosynthesis on the one hand and stochastic molecular damage on the other. According to Blagosklonny, the accumulation of molecular damage with age is not responsible for the ageing we experience although it would go on to cause ageing and death if we didn't suffer the effects of hyperfunction first.

2.2.4 An evaluation of the hyperfunction theory

A number of challenges to the hyperfunction theory - both logical difficulties with how the theory has been formulated and data which it is hard to reconcile with the theory (but which can be explained by the disposable soma theory) - are outlined in this section. I propose that these constitute sufficient reason to reject the idea of TOR-driven hyperfunction as an explanation for why we age.

2.2.4.1 Selection against hyperfunction in the wild

A central tenet of the hyperfunction theory is that organisms do not need brakes because "in the wild, [they] do not live long enough to experience aging". A selective pressure against hyperfunction is only thought to exist at very old ages when age-related diseases are manifest.

This reasoning seems to be flawed on two counts. Firstly, even if wild organisms do not display any age-related deterioration due to hyperfunction, any energetic resources used in this way could better be diverted to some fitness-enhancing function. An organism which could completely switch off the developmental program

and allocate this energy to reproduction would be fitter in early adulthood when the force of selection is strong. Secondly, senescence is in fact widespread in natural populations[6]. Even though the frailty seen at very old ages is rarely seen in wild organisms, there is still good evidence for age-related increases in mortality rate and decreases in reproductive rate. Therefore, if hyperfunction does cause ageing, we must assume that organisms are diverting energy to a process which actively lowers their fitness, against which there would exist a strong selective pressure.

2.2.4.2 The problem of how to define hyperfunction

From the examples of hyperfunctions given by Blagosklonny and Gems & de la Guardia, it seems that hyperfunctionality is defined only relative to the function of something else. It then becomes hard to think of any dysregulation or loss of homeostasis that could not be termed hyperfunction. For any biological process that is subject to both activational and inhibitory signals, we could attribute a reduction in the process to a hyperfunction of the inhibitory signals and an increase in the process to a hyperfunction of the activational signals. Moreover, it is not clear why such dysregulation should be described as a continuation of developmental processes simply because both the activational and inhibitory signals are present during development.

Osteoporosis is, for example, said to result from the hyperfunction of osteoclasts but this could equally well be described as the hypofunction of osteoblasts. Furthermore, it does not seem reasonable to suggest that osteoporosis is a continuation of programs that exist during development when the relative activity of osteoclasts and osteoblasts is tightly regulated. In young *C. elegans* yolk is found only in the intestine (where it is synthesized) and the gonad (where it provisions developing oocytes). The continued production of yolk after oocyte production stops and its resultant accumulation throughout the entire worm has been described as a hyperfunction, however, this could be equally well described as gonadal hypofunction. Even apoptosis - which seems the epitome of decline rather than over-activity - has been described as the unintended continuation of the programmed apoptosis during development of some cells of the immune system.

2.2.4.3 Hypertrophy as a consequence rather than a cause of cellular senescence

Although the observation that senescent cells are hypertrophic appears at first consistent with the idea of hyperfunction, a theory of ageing really needs to explain why an old cell becomes hypertrophic when TOR activity is high whilst a young cell does not.

Consider a culture of mammalian cells grown in constant high nutrient conditions such that TOR is highly active. At first TOR activity will stimulate both biosynthesis and proliferation - growth is balanced by division so that a constant size distribution is maintained for many passages. This co-ordination between growth and division is only lost towards the end of the cells' replicative lifespans when cells become hypertrophic. So, P1 cells may not differ in size from P10 cells but the latter will have a lower remaining replicative lifespan. Without presupposing that cells can 'tell the time', it does not seem that these different replicative capacities can be explained without reference to some other underlying process of ageing.

2.2.4.4 The distribution of ageing

If ageing is an unintended continuation of growth, we might expect organisms that never cease growing not to age. This is true for some forever-proliferating organisms such as hydra but not so for unicellular organisms or those capable of indeterminate growth.

In the case of *S. cerevisiae* - where a clear distinction exists between the larger mother and smaller daughter cell and where a steady increase in the size of the mother is seen throughout her lifespan - it could be argued that rapid growth is selected for during the juvenile phase and 'runs on' into the adult phase resulting in hypertrophy and senescence. As Blagosklonny [114] claims "cell senescence and longevity merge; when [the] cell cycle is arrested and TOR is activated, cells senesce." Interestingly however, cell size can sometimes be increased without lifespan being shortened indicating that hypertrophy does not always imply senescence. If cell size is increased by the application of mating pheromone which arrest cells in G1, they will exhibit the same replicative lifespan as untreated cells upon removal of the CDC block despite remaining larger than normal throughout their lives[127].

It is acknowledged that in bacteria displaying morphologically-symmetric division ageing must occur via some process other than hyperfunction - probably molecular damage accumulation. It is possible that ageing in bacteria has a different cause to ageing in eukaryotes; however, it is perhaps telling that some of the observations which it is argued support the hyperfunction theory in eukaryotes also occur in bacteria. In *M. extorquens*, there is an increase in cell size with pole age[128]. *E. coli* grown on low nutrient medium seem to age more slowly than those grown on high nutrient medium, if we take division time asymmetry as a measure of ageing rate[32]. Should we therefore take senescent cellular hypertrophy or an inverse relationship between longevity and nutrient availability as good evidence for the hyperfunction theory in eukaryotes?

Senescence in organisms with indeterminate growth, such as fish and some reptiles, is also hard to reconcile with the hyperfunction theory. The assumption that energy assimilated from the environment cannot be challenged into growth-promoting pathways upon attaining reproductive maturity no longer applies here. A selective pressure in favour of growth continues after the juvenile stage and into adulthood as larger adults experience increased fecundity.

In contrast, ageing in unicellular organisms and indeterminate growers can be explained by the disposable soma theory. The fitness advantages to incomplete damage repair and asymmetric accumulation between daughter cells of unicellular organisms with morphologically-symmetric division have been modelled by Watve *et al*. [31]and are described in chapter 1. In the case of indeterminate growers, a trade-off between somatic maintenance and other fitness-enhancing functions exists as for determinate growers. The delayed senescence seen in these organisms and be explained by considering the fitness contributions of different age classes. The increase in fecundity with size offsets the decline in selective force due to extrinsic hazards so that the fitness contributions of higher age classes are higher than for determinate growers subject to the same external threats. Consequently these organisms will invest more in maintenance.

2.2.4.5 The effect of dietary restriction upon lifespan

According to the hyperfunction theory the explanation for lifespan extension under dietary restriction (DR) is purely mechanistic: if you reduce the activity of TOR, then ageing rate should decline. However, lifespan extension under DR, although widespread, is not universal[38]. Organisms that do not respond in this way to DR - for example *Synchaeta pectinata* and, under certain conditions, water striders - pose a problem for the hyperfunction theory.

According to the disposable soma theory lifespan extension under DR should only be seen in organisms for whom a) an increased investment in maintenance at the expense of other fitness-enhancing functions is adaptive and b) frequent relatively short-term food shortages have been experienced in their evolutionary history and therefore an adaptive response to DR can be expected to have evolved. The latter condition may explain why tropical squirrel monkeys appear not to show the lifespan extension seen in temperate rhesus macaques[38]. Quantitative modelling is needed to determine whether the response to DR in species such as rotifers and water striders is adaptive. If they were shown to be so this would certainly support the disposable soma theory over the hyperfunction theory.

2.2.4.6 Changes in cellular physiology with nutrient availability

With explanations based on both the hyperfunction theory and the disposable soma theory having been offered for why lifespan extension is often seen under DR, one way to tell between them is to consider the different predictions they make as to the molecular and cellular changes we should see accompanying this lifespan extension. According to the disposable soma theory we should see an upregulation of somatic maintenance functions. According to the hyperfunction theory we should see reduced 'excess biosynthesis' through growth-promoting pathways. In both cases we see reduced TOR activity so this alone should not be taken as evidence for hyperfunction; as a key nutrient sensor, TOR activity would be reduced under DR regardless of any effects on lifespan. In fact, many somatic maintenance functions are negatively regulated downstream of TOR[42]. If molecular damage does not underlie the ageing we experience we would not expect to see this shift towards maintenance and repair.

2.3 The heat dissipation limit theory

2.3.1 Experimental work from which this developed

This theory arose from a series of experiments that sought to identify the factor(s) that limit maximum sustained energy expenditure in endotherms. Since body stores may only supply any difference between intake and expenditure in the short term, this is equivalent to asking what limits maximum sustained energy intake[129-131].

A constraint on energy intake may be environmentally imposed, as is known to be the case during winter at high latitudes, and organisms exhibit a range of dynamic responses to seasonal energy restriction including migration, hibernation and the depletion of fat reserves built up during times of abundance. It has been suggested that there is often no extrinsic limitation to energy intake during the breeding season when any trade-off between reproduction and somatic maintenance would be apparent[117]. A review of food supplementation studies found that animals do not always increase intake and reproductive output in response to increased provisioning suggesting that intake may be limited by some intrinsic constraint rather than by extrinsic availability.

A conceptual basis developed in the 1980s classed intrinsic limitations as being either central or peripheral[129, 130]. The central limitation hypothesis states that the capacity of the alimentary tract to absorb energy and/or the capacity for excretion of metabolic by-products limits intake whilst the peripheral organs possess an excess capacity for energy expenditure. The peripheral limitation hypothesis states conversely that intake is limited by the capacity of the peripheral organs to generate and utilise ATP whilst the organs of energy assimilation possess an excess capacity. The central limitation hypothesis predicts that ceilings for energy intake will be the same under different modes of energy expenditure; the peripheral limitation hypothesis that intake will vary with mode of expenditure and in particular that if energetically demanding tasks are undertaken by multiple peripheral organs maximum intake will be greater than when each task is performed separately.

Lactating rodents have provided a useful model for exploring constraints on energy intake[130]. Lactation is the most energetically demanding period of the female's life

cycle; food intake and milk production increase steadily during days one-to-ten of lactation and are then reasonable constant between days ten and eighteen. The level of milk production during late lactation seems to constitute an upper bound on the mother's ability to produce milk rather than the pups' ability to convert milk to biomass as pups from smaller litters are larger and females do not vary levels of milk production following manipulation of litter size[130, 131]. Consistent with the central limitation hypothesis, mice were unable to increase energy intake during late lactation when they were made to exercise for their food or were simultaneously pregnant. However, when exposed to cold these mice were able to increase intake suggesting that the absorption capacity of the alimentary tract is not the limiting factor. The peripheral limitation hypothesis - which would predict that the mammary glands are producing milk at the maximum rate of which they are capable during late lactation - also doesn't seem to fit the evidence. When mice were housed in the cold or were shaved they elevated their milk production and were able to wean heavier offspring[130, 131]. These results, together with findings from a number of domesticated animals which show heat production to be a limiting factor in reproductive output, led Speakman & Król [117]to hypothesise that maximum sustained energy expenditure in endotherms is constrained by the capacity for heat dissipation.

As further support of this notion, Speakman & Król cite data on field metabolic rates[117]. Since heat dissipation is a surface phenomenon, and to a good approximation surface area increases by two thirds of a unit for each unit increase in volume, the heat dissipation limit theory predicts that daily energy expenditure should scale with body mass raised to the exponent two thirds. Furthermore, as this scaling exponent is lower than that for basal metabolic rate (which is closer to 0.75), this theory predicts that metabolic scope - that is the maximum metabolic rate relative to the basal metabolic rate - should decline with size. There is evidence that both these predictions hold in endotherms.

2.3.2 Outline of the theory

According to Speakman & Król, if we start from the observation that interspecific variation in body mass exists and then - bearing in mind that size will determine the rate at which heat can be dissipated - consider the consequences this variation has on

the types of life histories that can evolve, the fast-slow life history continuum may be explained without requiring that trade-offs exist between fitness-enhancing functions (figure 2.2)[117].

Their lower surface area-to-volume ratios and hence their reduced capacity for heat dissipation means that larger animals are constrained to having lower reproductive rates. High mortality coupled with a low reproductive rate is incompatible with the long-term survival of a species and larger lineages will therefore go extinct if which occupy biological niches in which external mortality threats are high. Consequently, a negative correlation between size and mortality rate arises without size itself conferring any protection against mortality.

The relationships between size and a) the occurrence of genes with negative effects at older ages; b) the occurrence of genes with positive effects in early life and negative effects in late life and; c) investment in somatic maintenance, that are predicted by the mutation accumulation, antagonistic pleiotropy and disposable soma theories respectively may then arise as a consequence of this correlation between size and mortality (figure 2.2). A low extrinsic mortality rate establishes a selective pressure in favour of lower intrinsic mortality; reducing the rate of ageing will extend longevity to a greater extent in species where it is not otherwise heavily curtailed by external threats. Late-acting deleterious mutations will therefore be selected against and longevity assurance mechanisms selected for. Importantly, however, longevity assurance mechanisms need not be energetically expensive and are not necessarily traded-off against reproduction. Due to their enforced lower reproductive rates, genes which increase reproductive rate in early life at the expense of fitness in late life are selected against in larger organisms. Whilst each of these selective pressures will enhance the association between body size and longevity, they are not the original cause of it which was the heat dissipation limit.

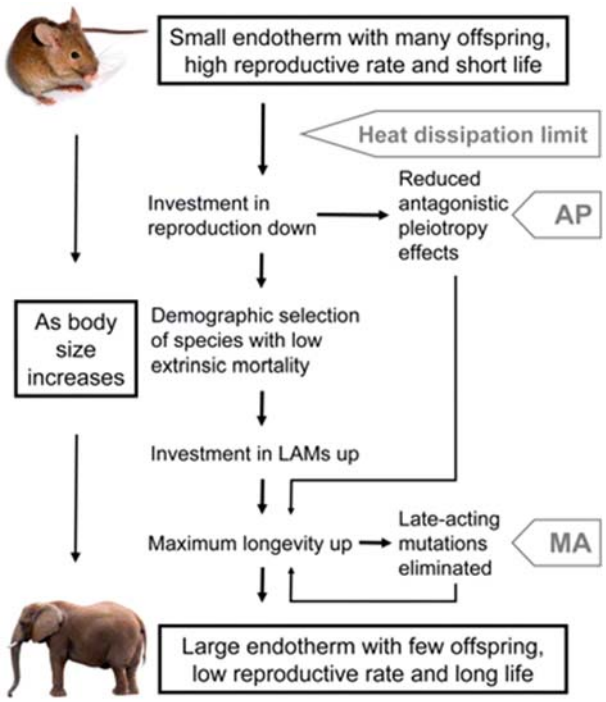

Figure 2.2: Flow diagram showing the changes to life history and genetics which result from an increase in size according to the heat dissipation limit theory. Constraint upon the energy that may be expended in reproduction weakens selection for genes which enhance reproductive rate in early adulthood at the expense of reproduction and/or robustness in later life in larger species. These species will - owing to their lower rates of reproduction - only survive in habitats where external mortality threats are low. Selection favours investment in longevity assurance mechanisms (LAMs) in environments where external mortality threats are low, however, there is no requirement that these be energetically expensive nor traded-off against reproduction. As longevity increases, selection against genes which confer adverse effects in later life grows stronger. Diagram taken from [117].

2.3.3 Contrast with the disposable soma theory

The effect of body mass and extrinsic mortality threats in moulding the rate of ageing has been emphasised in both the disposable soma theory and the heat dissipation limit theory although the routes by which these affect longevity are different. According to the disposable soma theory, extrinsic mortality threats affect the probability of survival to a given age, and therefore the weighting of future reproductive gains relative to those of the current period. This determines the optimal investment in reproduction relative to maintenance and other fitness-enhancing functions, importantly in this case growth. Investment in maintenance and growth in turn alter the probability of

succumbing to extrinsic mortality threats; a greater investment in maintenance slows the rate of increase in susceptibility to many mortality threats and a greater investment in growth provides an enhanced fasting endurance, a reduced risk of predation and - because larger species live at lower densities - a reduced rate of disease transmission. Investment decisions evolve towards an optimum, with larger species generally investing a greater proportion of their energy budgets in maintenance and a lower proportion in reproduction (see chapter 1).

According to the heat dissipation limit theory, larger organisms are, by way of their reduced capacity for heat loss, unable to sustain high reproductive rates and consequently only able to occupy niches in which extrinsic mortality threats are low. (If extrinsic mortality threats were high the population would decline and eventually go extinct.) Size does not in itself provide any protection against extrinsic mortality threats and the correlation between size and lifespan results purely from the elimination of large species with short lifespans.

Under both theories, ageing is determined by investment in somatic maintenance, or what Speakman & Król call longevity assurance mechanisms. Such investment protects against a spectrum of molecular damage including telomere shortening, protein aggregation, oxidative damage and increasing rigidity of cell membranes. The disposable soma theory postulates that somatic maintenance is energetically expensive and, as the energy budget is limited, investment in this is traded-off against investment in other fitness-enhancing functions. The heat dissipation limit theory postulates that somatic maintenance is energetically cheap and therefore investment in this does not come at the expense of other functions.

These theories differ in the range of organisms to which they apply. The heat dissipation limit theory applies only to endotherms: it assumes that metabolic rate and size are the principal determinants of body temperature which must be kept within a reasonably narrow range. The disposable soma theory may be applied to all biological organisms. It is assumed that all organisms are a) subject to a range of molecular insults which may be repaired at a cost and, b) free to choose how the resources available to them are partitioned (or at least free to evolve different allocation

strategies). All species have therefore evolved levels of investment in maintenance which return the greatest fitness.

Ageing is non-programmed under both theories: senescence is not in itself beneficial and there is no selection for the ageing phenotype.

2.3.4 An evaluation of the heat dissipation limit theory

There are a few possible drawbacks to the heat dissipation limit theory. Firstly, it only applies to endotherms when it is known that ageing, and in particular the link between size and ageing, has a much older root. Whilst it is possible that there are different explanations for correlations between size and longevity in different groups of organisms, it would certainly be more parsimonious to have a single explanation that applies to all.

Secondly, Speakman & Król began from the premise that variation in body size exists and then considered what the consequences of this variation would be in terms of the types of life histories that would evolve. However, based on the explanation given, it is hard to see how it would ever be beneficial for a species to evolve a large body size. If there is no inherent advantage to increasing size, in terms of either reduced mortality or increased reproductive rate, then we would expect that smaller individuals always outcompete larger ones.

Thirdly, there is evidence for ageing in the wild across a wide range of species, both small and large, and experiencing both low and high rates of extrinsic mortality. If longevity assurance mechanisms are cheap, so that the rate of ageing could be reduced without any significant loss of investment to other fitness-enhancing functions, we would expect such mechanisms to be strongly selected for in all species to the extent where ageing is (virtually) eliminated. The near ubiquity of senescence in the wild is more easily explained if somatic maintenance makes significant demands upon a limited resource budget.

Lastly, the heat dissipation limit theory was based primarily on data from laboratory rodents. Since these have been selectively bred for high litter sizes, it does not follow that what limits their reproductive output also limits that of wild-living rodents. Similarly, many domesticated species have been bred for high litter size or milk

production and the finding that heat dissipation limits their expenditure does not necessarily apply to their wild-living counterparts.

As a final note, it is worth considering what the implications of studies into the nature of energy limitations are for the disposable soma theory. The authors of the heat dissipation limit theory have stated that the disposable soma theory assumed an environmentally-imposed limitation upon energy intake, however, this is not the case. It would be possible that some intrinsic upper bound upon energy intake exists and within this bound organisms must choose how to allocate resources to competing physiological functions. If this upper bound was dictated by the capacity for heat dissipation, then we expect that the benefit of size is sufficiently great to outweigh the disadvantage in terms of reduced metabolic rate. It is also possible, and indeed is known often to be the case, that intake is itself a behavioural choice. Foraging carries a number of costs including predation, parasitism, exposure to adverse weather conditions, time not spent mating or caring for offspring, ingestion of toxins and food storage costs. These costs must be weighed against the fitness gains from energy assimilation. In effect, an organism's fitness depends on two related trade-offs: that between effort diverted to foraging versus other fitness-enhancing functions and that of the partitioning of assimilated energy between various fitness-enhancing functions. Models have already been developed which derive optimal foraging strategy with explicit reference to the costs of foraging and have predicted that organisms will choose a lower energy intake than the environment is capable of supporting[132]. In fact, if either a central or peripheral limitation upon energy intake was found to apply, this limitation could be deemed a behavioural choice. As the structures of energy assimilation and expenditure have evolved to meet a functional need, and an excess capacity in any of these structures can be assumed to be energetically expensive, it may be supposed that their maximum capacity has evolved to fit their owner's optimal intake.

Chapter 3: Methods

3.1 Dynamic programming

3.1.1 Static versus dynamic optimisation

Evolutionary optimisation models seek to identify, from a set of available behavioural strategies, that which maximises a given definition of fitness. In simple cases, strategies may consist of a single decision, for example the proportion of the available energy budget which is devoted to maintenance. This is known as static optimisation. Such decisions may depend upon environmental conditions - for example, changing the nature of extrinsic mortality threats may favour either a greater or lesser investment in maintenance (see chapter 1) - as well as on the state of the organism - for example, an organism of poor quality at the start of life may do better to select an accelerated life history with a faster rate of ageing (see chapter 5).

Although it has proved useful in exploring the rationale for a variety of behavioural decisions, static optimisation suffers from some significant limitations. Firstly, the relative rewards and costs associated with a particular decision may change over time. Older individuals are, owing to their poorer condition, unlikely to survive for long regardless of current investment in maintenance. Since the weighting of potential future reproductive gains relative to those of the current period would therefore decrease, investment in reproduction is expected to increase at the expense of maintenance. Secondly, the decision set from which an organism may select can change over time. If foraging yield decreases with age, then absolute investment in the whole suite of fitness-enhancing functions is necessarily lower in older individuals than in younger ones and absolute allocation to at least one function must be reduced.

It is therefore more realistic to model organisms as making a series of decisions throughout their lives. The organism may then respond to a) a changing environment, b) stochastic effects and, c) the effect of previous decisions upon current state. To compute an optimal sequence of decisions made over time is known as dynamic optimisation. This can be carried out by means of dynamic programming: an approach

developed by Richard Bellman in the 1940s for solving problems which can be decomposed into overlapping sub-problems and which have an optimal substructure. These two properties mean, respectively, that a single algorithm can be applied recursively to the solution of sub-problems and that the solution to the overall problem may be computed from solutions to these sub-problems. As applied to behavioural decisions in biology, the technique is summarised in the next two sections.

3.1.2 An overview of dynamic programming

Individuals are assumed to choose from a specified set of possible actions at a series of discrete time points. The set of actions available may vary with the state of the organism and over time. Decision processes are Markovian, meaning that the optimal decision at any given time point depends only upon state at that time point and not on any states (or decisions) previous to that. Actions may incur rewards in the time period in which they are employed and also affect the state of the organism in the next period. The value of being in a given state at the start of the next period will depend upon the actions taken at that and all subsequent time points[44, 63].

The decision process is shown schematically in figure 3.1, where an organism in state X(t) at time t, is subject to environmental conditions ∃(t) and performs action A(t) which produces some reward R(t) and leaves the organism in state X(t + 1) at time (t + 1). Note that environmental effects are pictured as separate from state as this is most intuitive. However, these can also be treated as part of the state variable since they reflect an aspect of the situation in which the organism finds itself and upon which a choice of action is made in the same way as would a variable such as size or age. Mathematically these two conceptualisations - considering the environment to be part of the state variable or external to it - are equivalent. Environmental effects may be modelled as acting either before an action is taken or subsequent to it (as indicated by the dotted arrow). They may either be constant or change between time periods; in the latter case the environment at time (t + 1) must be a function only of the environment at time t. The effect of the environment upon state; the reward associated with a particular action and; the change in state which follows a particular action may all be subject to stochastic effects. Obvious examples of this would include the stochastic nature of mortality upon the individual given an environmentally-

imposed mortality risk or of foraging success given the decision to hunt in a particular locale.

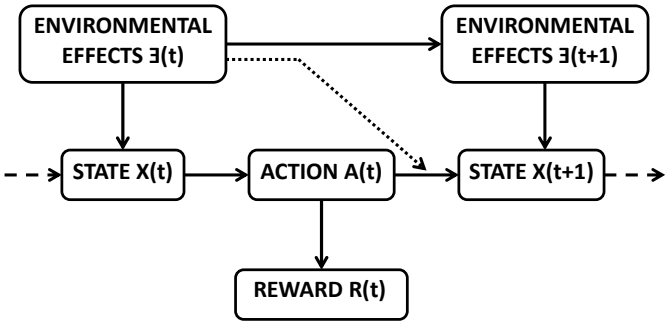

Figure 3.1: Schematic representation of a Markovian decision process as used in dynamic programming.

The optimal strategy at time t is computed by working backwards from some final time horizon T. Reproductive values associated with each of the possible states in which an individual may exist must be specified at this time horizon. This being done, the optimal action for each state at time (T - 1) - i.e. the action which if followed would maximise the reproductive value of an individual in this state at this time - is defined as that which maximises the sum of the expected reward from the action plus the expected reproductive value at time T given the possible state transitions which may follow this action. Having calculated reproductive values and optimal actions for each state at time (T - 1), these may then be calculated for each state at time (T - 2) in a similar manner assuming that the organism will behave optimally at time (T - 1), and so on for as many time points as are being considered. In this way the optimal strategy is computed as a function both of state and time given some objective.

The generalised form of the relationship between reproductive values at one time point and the next, for t < T, is given by the dynamic programming equation[44, 63]. For the system shown above this would be:

$$F(x, \exists, t, T) = \max_A \{E[R] + E[F(x', \exists', t + 1, T)] \}$$

where:

- $F(x, \exists, t, T)$ is the maximum reproductive value of an indiviual in state x, subject to environmental effects $\exists$, at time t and with time horizon T, the maximisation being taken over all possible actions A.
- E indicates that an expectation is taken. This accounts for possible stochasticity is environmental effects, the reward from a given action and the state transition that follows a given action.
- The reward R is a function of state, time, action and the environment.
- x' is state at time (t + 1), as a function of state, action and environment at time t. It is often the case that, unless the functions relating change in state to action and enviornmental effects are chosen very carefully, x' does not fall on one of the discretised values of state in which individuals are supposed to exist. In these cases, the reproductive value associated with the true state is estimated as the weighed average of the reproductive values of surrounding states via linear interpolation. For example, in the case of a one-dimensional state variable, when the precise value x' lies in the interval between discretised values x_1 and x_2, reproductive value is estimated as:

$$F(x', \exists, t, T) \approx \{ (x' - x_1)*F(x_2, \exists, t, T) + (x_2 - x')*F(x_1, \exists, t, T) \}/(x_2 - x_1)$$

with $x_1 < x < x_2$

- $\exists'$ is the environment at time (t + 1) as a function of environment at time t.

The terminal fitness function, which gives the state-dependent reproductive values at the final time horizon T, would have the form:

$$F(x, \exists, T, T) = P(x)$$

Working backwards in this way provides a computationally efficient means of determining the optimal strategy. If the optimal strategy over n time steps is to be computed, then n*Ж*Ш calculations are required where Ж is the dimension of the state space, Ш is the dimension of the set of possible actions and a calculation refers to the computation over one time step for a single action. In contrast, were an exhaustive forward search employed, the number of possible combinations of

decisions would increase exponentially with the number of time steps. In this case, Ж*Ш^n calculations would have to be carried out to determine the optimal strategy over n time steps[64]. (To see why this is the case observe that there are Ж possible states in which an individual may be at the earliest time point and Ш possible decisions which can be taken at each of the subsequent n time points.)

3.1.3 Components of a dynamic optimisation model

A formal specification of a dynamic optimisation model requires the following to be defined[44, 63].

1. **Fitness**

   This is the quantity which is to be maximised and therefore the metric by which the value of different actions is compared. The most appropriate definition of fitness will depend upon the question under consideration. If - as in the example described in section 3.1.5 - foraging decisions over winter are being modelled, a suitable definition of fitness might be the expected size of the individual at the start of the breeding season. Since this is a reasonable proxy for expected reproductive success, the individual is effectively maximising the probability of surviving over winter multiplied by the probable breeding success if it should survive.

   Where the entire life history is being modelled, the simplest definition of fitness would be the number of offspring born; under this definition an organism seeks to maximise Σ_x l(x)*b(x) where l(x) is the probability of survival to age x and b(x) is the expected reproductive output at age x. A more sophisticated definition may include some measure of offspring quality; the probability that an offspring will survive to reproductive maturity (which could be dependent upon continued parental investment) or; the effect that behaviour has upon kin. In the models detailed in chapters 4 & 5, fitness is defined as the expected number of copies of an individual's genes at some time far into the future. This takes into account differences in quality between offspring as well as the effect of population growth rate upon optimal reproductive scheduling. In a growing population, the expected number of copies of an individual's genes increases per generation and so, all else being equal, it pays to shorten the generation time by having offspring sooner. In a

declining population the reverse is true and individuals do better to have their offspring later.

2. The time interval between successive decisions

The most suitable choice of time interval will depend upon the system being modelled. If reproductive scheduling over the entire life course of a long-lived species such as humans is being modelled then a year may be appropriate whereas a day might be suitable for modelling the foraging decisions made by a bird during the non-breeding season.

3. The state space

This is the set of all possible state variables. A state variable may be either a single value or a vector and may include any characteristics of the organism and its environment which affect the behaviour under study. It may also include other organisms and dependent offspring are often modelled as part of the parental state. Boundaries on the state space must be defined and these will normally extend beyond the range of states that are seen in the field because - presuming state dynamics are a good approximation to biological reality - no simulated individuals should then hit these boundaries and consequently predicted behaviour will not be constrained by them. If the state variables are continuous (for example age or size) an appropriate discretisation of the state space must be chosen. This should be sufficiently fine that changes in state during the forward iteration (see below) are not so crude as to be biologically unrealistic.

4. The decision set

This comprises the set of all possible actions between which individuals must choose. The decision set may vary with state and/or time. If, for example, optimal growth rate during the juvenile period is being modelled, then maximum allowable growth per time interval may be an increasing function of size at the start of the interval. If energy allocation is modelled over successive breeding and non-breeding seasons, then the option to invest in reproduction would only be available during the former.

5. **State dynamics**

   These describe a) the transition between states as a function of action and b) the expected reward from an action as a function of state. As mentioned above, both these functions may be stochastic, that is for a given state and action there may be multiple possible state transitions and rewards. As well as deciding upon the general form of these equations, a suitable parameter set must be selected and any additional constraints defined. Constraints might include things such as maximum weight loss per period (as in the model in chapter 4) or maximum gut capacity of a forager.

3.1.4 Convergence

It will be obvious from the description of dynamic programming given in section 3.1.2 that optimal decisions at any given time point will be a function of the distance of the time horizon (T - t) and the form of the terminal fitness function (P(x)).

Depending on the system being modelled, there may or may not be a natural choice for T and P(x). For the example of overwinter foraging decisions, the natural choice for T would be the start of spring and setting P(x) to be an increasing function of size would be easy to justify since larger individuals generally enjoy a greater reproductive success. However, if energy allocation between reproduction and maintenance over many generations is being modelled, there is no obvious choice for T and P(x) is also harder to define. P(x) requires that the reproductive values of individuals be given as a function of their age and reproductive status; this is, however, what the model seeks to determine.

Fortunately, if a dynamic model is run over a large number of time steps ($t \ll T$), the optimal decision at time t is in the majority of cases independent of P(x) and, when environmental conditions do not vary systematically with time, also independent of t. This property of dynamic models is known as backward convergence. If behaviour remains dependent upon t for $t \ll T$ but not P(x), weak backward convergence is said to hold; if behaviour is independent of both t and P(x), strong backward convergence is said to hold.

So, for the example of allocation between reproduction and maintenance, some naïve function P(x) may be defined - such as P(x) = 1 if the individual is alive and P(x) = 0 otherwise - and predicted behaviour far from the time horizon should still be biologically reliable.

3.1.5 Patch selection

The following example, taken from Mangel & Clark[44], is used to demonstrate the application of dynamic programming to an iterative biological decision process.

Consider a forager who must select, at regular time intervals throughout the non-breeding season, from a set of available foraging patches. The goal of this forager is simply to survive until the start of the breeding season; all individuals who are then alive are considered to be of equal fitness. Foraging patches differ in terms of probability of finding food; energetic value of food if it is found; risk of predation and; energetic cost per-period to the individual. The per-period cost may include travel to and from the foraging patch and any anti-predator behaviour which must be adopted in given patches as well as the cost of vital metabolism. Survival until the breeding season requires that the individual garners sufficient energetic resources to meet these costs and that it does not succumb to predation.

Let x represent the state of a forager; this could simply be the size of an individual but may also incorporate other aspects of quality. State may not exceed a maximum of C nor fall below a minimum of x_c. Should state reach the critical value x_c, the individual dies. The final time horizon T is the start of the breeding season. Fitness at this time horizon is set to one if the forager is alive and zero otherwise. Foraging patch i is characterised by a per-period cost of α_i; predation risk β_i; probability of finding food λ_i and; value of food Y_i. α_i and Y_i are measured in terms of decrement and increment to the state variable respectively, rather than in absolute energetic terms. There are no environmental changes (that is, no systematic changes to the characteristics of all patches) from one time period to the next. Since fitness is defined only in terms of survival, there are no per-period fitness increments (rewards in figure 3.1), simply a change in state. This system is described by the dynamic programming equation:

$$F(x, t, T) = \max_i \{(1 - \beta_i)*[\lambda_i*F(x_i', t + 1, T) + (1 - \lambda_i)*F(x_i'', t + 1, T)] \} \quad \text{if } x > x_c;$$

$$F(x, t, T) = 0 \quad \text{if } x = x_c$$

where:

- $F(x, t, T)$ is the maximum reproductive success for an individual in state x, at time t and given a final time horizon T. The maximisation is taken over the choice of foraging patch.
- $(1 - \beta_i)$ is the probability of survival if patch i is chosen. (Note that whilst the equation is written as if predation acts before the foraging outcome, since there is no per-period fitness reward in this case, there would be no change to $F(x, t, T)$ were these the other way round.)
- The term $\lambda_i*F(x_i', t + 1, T)$ is the probability of finding food in patch i multiplied by reproductive value at the start of period $(t + 1)$ if food is found. The state at $(t + 1)$ if food is found is represented by x_i' where:

  $$x_i' = \min(\max(x - \alpha_i + Y_i, x_c), C)$$

- The term $(1 - \lambda_i)*F(x_i'', t + 1, T)$ is the probability of not finding food in patch i multiplied by reproductive value at the start of period $(t + 1)$ if food is not found. The state at $(t + 1)$ if food is not found is represented by x_i'' where:

  $$x_i'' = \min(\max(x - \alpha_i, x_c), C)$$

The terminal fitness function is:

$P(x) = 1$ if $x > x_c$;

$P(x) = 0$ if $x = x_c$

The authors present results for the case where there are three available patches. Predation risk was positively correlated with probable increase in state variable between patches so that the safest patch yielded the least foraging success and the riskiest patch the most. (If, for any pair of patches, that with the greater predation risk afforded the lower expected increase in state variable, this patch could be excluded from consideration since the other would always be preferable to it.) As expected, the reproductive value of an individual in any given state increased as the time horizon

was approached and for each time point, reproductive value was a non-decreasing function of the state variable. If an individual was in a reasonable state close to the time horizon, survival depended essentially on avoiding predation whereas further away from the time horizon, even an individual in a good state did not have sufficient reserves to last until the end of the season. The risks of starvation relative to those of predation therefore decreased as the time horizon was approached and so the proportion of states in which it was optimal to choose the safest patch increased. Backward convergence was seen, with decisions far from the time horizon being state-dependent but time-independent.

3.1.6 The forward iteration

Calculation of the optimal strategy, by working from the final time horizon backwards according to the dynamic programming equation, is known as the backward iteration. To track the behaviour of a population over time, all members of which follow this optimal strategy, is known as the forward iteration. This can tell us the expected proportion of a population who will be in a given state; perform a given action or; obtain a given reward within each period. It requires that some starting population (and environmental state if applicable) at time t = 0 be defined. For each subsequent time point, the actions followed by all members of the population are read from the optimal strategy and the rewards and state transitions which succeed these actions are computed. If rewards and/or state transitions are stochastic in nature, then one must be selected from the set of possible values according to the probability of each occurring. Similarly for environmental states if these change over time and are subject to stochasticity. As mentioned in section 3.1.2, unless the functions describing state transitions are chosen very carefully, predicted state may not fall upon one of the discretised values which make up the state space. In these cases, an individual may transition to any of the states surrounding the predicted one, the probability of each being determined by its relative closeness to the predicted state.

As for the backward iteration, the behaviour of a population predicted by the forward iteration often shows convergence upon some stable behaviour for $t >> 0$ and again two forms of convergence are defined. If behaviour remains dependent upon t for $t << T$ but not the initial state of the population, weak backward convergence is said to

hold; if behaviour is independent of both t and the initial state of the population, strong backward convergence is said to hold.

3.2 Game theory

3.2.1 Evolutionary game theory

Game theory is the analysis of interactions between two or more rational decision makers all of whom act so as to maximise some objective and where the outcome of an interaction for any one individual depends upon the behaviour of the others. Much of game theory was developed with reference to decision making within economics but has since been applied to a variety of fields including evolutionary biology. When applied to biology, a decision, or strategy, refers to a behavioural phenotype and the optimal strategy is that which maximises the player's Darwinian fitness.

A formal specification of a game must include some description of the players; the set of strategies open to each player and; the possible outcomes of each strategy to a player as a function of their opponent's strategy[65]. Players may be identical or may differ in some aspect of their state. If players differ in their state, this may constrain the set of actions available to them. If iterative games are being modelled, players may remember previous interactions with particular individuals and select a strategy according to the past behaviour of their opponent and the outcomes of past interactions. The outcomes to all players, given their states and strategies, may be either deterministic or stochastic.

Given information about the players, available strategies and possible outcomes, optimal strategies for all players may be computed. These may be either pure or mixed. A pure strategy is one which specifies a single behaviour which the player should adopt (possibly dependent on their own and their opponents' states and past actions); a mixed strategy specifies multiple behaviours along with the probabilities that each should be adopted.

Evolutionary game theory assumes that the positivity of the outcome from a strategy correlates with the fitness of individuals who employ it and therefore with the degree to which the strategy is represented in future generations. If a game-playing population is modelled over time, with offspring playing the same strategies as their

parents and outcome determining reproductive success, the most successful strategy will sometimes spread through the population to fixation. Such a strategy has been termed an unbeatable strategy by Hamilton or an evolutionarily stable strategy (ESS) by Maynard Smith and Price[66]; when employed by all members of a population, it cannot be invaded by a mutant strategy by means of natural selection. However, for a given set of potential strategies, an ESS need not always exist. Since the positivity of outcomes from a strategy depends upon the distribution of other strategies against which it is played and this distribution changes as certain strategies spread, the relative success of a strategy may change over time. If successful when played against other strategies but unsuccessful against itself, a strategy may never be eliminated from a population (because it does well when adopted by a minority) nor spread to fixation (because it does badly when adopted by the majority). A stable distribution of various strategies may evolve at the population level or the strategy set may cycle periodically with first one predominating and then another.

The following example, first presented by Maynard Smith and Price[66], is used to illustrate how a biological problem may be formulated as a game; the dependency of optimal strategy upon how others behave and; the meaning of an ESS.

3.2.2 Hawks and doves

Consider two contestants who compete over a resource, the attainment of which would increase their fitness by V. Each player may adopt one of two strategies: hawk or dove. Under the hawk strategy, an individual will always launch an escalated attack on an opponent. If the opponent backs away immediately, the individual gains the contested resource; if the opponent attacks, the pair will fight until one of them is injured and retreats leaving the victor to claim the contested resource. Fitness is decreased by C should an injury be sustained. All individuals are assumed to be of equal fighting ability so that the probability of winning a fight is always 50% for each player. The dove strategy involves a display but no escalated attack. If the opponent attacks the individual will back away immediately; if the opponent also displays but does not attack they will share the resource equally between them.

The expected payoffs to individuals playing each strategy, dependent upon their opponent's strategy, are shown in figure 3.2. If two hawks play against each other, the

expected payoff for both is (V - C)/2 since their chances of winning and losing are the same. If two doves play each other, the expected payoff for each is V/2 since they share the resource. If a hawk and dove play, their expected payoffs are V and 0 respectively since the dove will retreat leaving the hawk to claim the resource.

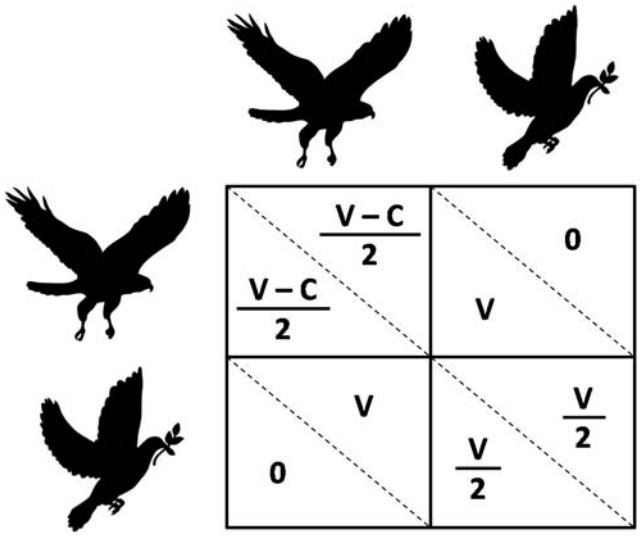

Figure 3.2: The payoff matrix for the hawk-dove game described by Maynard Smith & Price[66].

Consider first the relative rewards of these two strategies when V > C. Against an opponent who plays dove, an individual will do better to play hawk; the expected payoff if playing hawk is V compared with only V/2 if playing dove. Against an opponent who plays hawk, an individual will do better also to play hawk; the expected payoff if playing hawk is (V - C)/2 which, as V > C, is greater than the 0 payoff from playing dove. Therefore, the hawk strategy is optimal regardless of an opponent's strategy and constitutes (for a single play) an ESS. Note that, by playing the ESS all contestants actually do worse than they would if they all agreed to play the alternative strategy since V/2 > (V - C)/2. However, a population all members of which played dove would be susceptible to invasion by hawks. A mutant hawk would be able to claim the full value of a contested resource and the strategy would therefore spread. On the other hand, a population of hawks is not susceptible to invasion by doves as a mutant dove would gain none of the value of a contested resource.

If such games are played iteratively, with individuals repeated encountering the same opponents, there is potential for co-operation to evolve between contestants. A co-operator would play dove when they first encounter a given opponent. Provided the opponent reciprocates and also plays dove, they then do the same when they meet the same opponent next and this continues for as long as they both cooperate. If an opponent played hawk, co-operation would be withheld from them in future. Co-operators will on the whole do better than non-co-operators in this scenario; although they lose out in their first encounters with non-co-operators, they do equally well as non-co-operators in their subsequent encounters and gain increased payoffs in their encounters with other co-operators.

Consider now the relative rewards of these two strategies when $V < C$. Against an opponent who plays dove, an individual does better to play hawk as before; the expected payoff if playing hawk is again V compared with only V/2 if playing dove. Against an opponent who plays hawk, an individual will now do better to play dove; the expected payoff if playing hawk is $(V - C)/2$ which, as $V < C$, is less than the 0 payoff from playing dove. In this case, a population of hawks is susceptible to a dove invasion and a population of doves to a hawk invasion. There is no ESS and the population would either a) settle at some stable distribution with given proportions of the population playing each strategy and the expected fitness of all players being the same or b) cycle between periods in which one strategy and then the other is dominant.

The basic hawk-dove game may be extended to allow for differences in state between contestants. As a simple example, suppose that individuals differ in the fitness cost of an injury - say that a subgroup of the population carry a gene which confers better protection against an attacker (although not a greater chance of winning a fight). If the cost of injury was less than the fitness value of the resource to this subgroup but more than the fitness value of the resource to the remainder of the population, the subgroup is expected always to play hawk whilst the rest of the population would employ a mixture of hawk and dove.

Differences in energy reserves between players who compete for food could also be considered; the relative value of a food item would be greater to those close to starvation and we would expect them to employ a more aggressive strategy than their

well-fed conspecifics. If a game is extended to allow for both iterative plays and differences in state, it is termed a dynamic behavioural game and these are described in section 3.3.

3.2.3 Types of game

- **Zero-sum versus non-zero-sum games**

  Zero-sum games are ones in which the payoffs to all players always sum to zero. An example is the game of poker: one player must win the amount that the other players lose (assuming the house does not take a cut). Non-zero-sum games are ones in which the payoffs to all players may be more or less than zero. The basic hawk-dove game outlined above is a non-zero-sum game: if at least one contestant plays dove, payoffs to both contestants sum to V; if both contestants play hawk, the sum of payoffs is non-zero provided $V \neq C$.

- **Symmetric versus asymmetric games**

  A symmetric game is one in which the payoffs from a particular strategy do not depend upon which player employs them. For a two-player game this means that player 1 receives the same payoff from playing strategy A against player 2 playing strategy B as player 2 would receive from playing strategy A against player 1 playing strategy B. The basic hawk-dove game is a symmetric game (figure 3.2). An asymmetric game is one in which the payoffs from a particular strategy depend on who employs it. The suggested extension to the hawk-dove game in which a subgroup of the population is better protected against injury is an asymmetric game: if a well-protected contestant and a poorly-protected contestant both play hawk, the expected outcome for each is different. Games in which different strategy sets are available to each player (as, for example, is often the case in parent-offspring games) are also asymmetric.

- **Simultaneous versus sequential games**

  Simultaneous games are ones in which all players must choose their strategy without knowledge of what their opponents' strategies are. This may mean playing strategies at the same time or that the strategy of the player who is first

to act is hidden from its opponents until after they have chosen their own strategies. An example is the game of rock-paper-scissors in which both players must show at the same time. A sequential game is one in which one player must act first and later players may choose their own strategies on the basis of what the first player's is. The game of chess is sequential since players move in turn.

- **Pairwise versus n-player games**

  A pairwise game is one in which there are two contestants, such as the basic hawk-dove game. An n-player game is one with some finite number of players, n. The game of poker may, for example, be anything up to a fourteen-player game. Evolutionary games are often n-player games where n is assumed to be very large. Individuals are then said to 'play the field', that is, they must base their behaviour upon the expected behaviour of the rest of the population as a whole.

3.3 Dynamic behavioural games

3.3.1 The application of dynamic programming to evolutionary games

The model presented in chapter 4 of this thesis is of a dynamic behavioural game. Individuals play a series of games against one or more other individuals at discrete time points with the outcomes depending upon time, the states of all players and the strategies they employ.

The easiest way to solve a dynamic behavioural game - that is, find the ESS - would seem to be to work backwards from the time horizon, in the same way as for a simple dynamic optimisation problem. At each time point, all possible combinations of states in which the various contestants may be and all possible actions that they may employ would be considered in order to calculate the optimal action for each. If past actions gave any indication of future behaviour, all possible actions and outcomes from past encounters would also have to be considered. Unfortunately, besides this approach being computationally formidable, dynamic behavioural games cannot always be solved in this way as the following example illustrates.

Consider the iterative hawk-dove game, played at each time point against a randomly selected opponent from within a population, with V > C. It may be supposed that contestants are defined by their energetic reserves x, however, assuming that the outcome of any fight is independent of x, state is actually unimportant here. At time (T - 1), the optimal strategy for both contestants is to play hawk and the reasoning here is the same as given in section 3.2.2 for a single game. If an opponent plays dove, an individual does better to play hawk; if an opponent plays hawk, an individual does better also to play hawk. Working backwards to time (T - 2), and then to all subsequent time points, it is by the same reasoning predicted that all contestants should play hawk all the time. However, as stated above, this does not in fact represent an ESS. It has been shown by forward simulations - starting from a mixture of strategies and assuming that payoff from a strategy correlates with the frequency of the strategy in the next generation - that co-operative strategies do better, provided that co-operation is withheld from those who do not reciprocate. A population whose members only played hawk would be susceptible to invasion by co-operators; expected payoffs accrued over the lifetime of a co-operator would exceed those for a non-co-operator. A population of co-operators would, however, be immune to invasion by non-co-operators.

There are nevertheless certain classes of dynamic behavioural games that can be solved by a backward iteration including ones in which co-operation is predicted. The following example of an anti-predator game between two players was formulated by Lima [67]. It differs from the above in that each player's fitness depends upon the fitness afforded the other as a consequence of their actions.

It is assumed that the players suffer regular attacks from a predator. They may either co-operate (C) with each other in escaping/repelling these attacks or defect (D). The chances of survival for one period, dependent upon the strategies employed by both players, are shown in figure 3.3. If a player survives and their partner dies, they must play alone from then onwards and the chances of surviving an attack if playing alone are $\alpha = 0.5$. The ranking of outcomes for any one period are the same as for the hawk-dove game: if ω_{ij} is the probability of a player surviving given that they play i and their partner plays j then $\omega_{DC} > \omega_{CC} > \omega_{DD} > \omega_{CD}$. So, when played a single period from the time horizon, both players are expected to defect. However, at two periods or more

from the time horizon, the probability of survival until the time horizon is enhanced by ensuring the survival of one's partner and co-operation is thus predicted. Solution of this problem found the optimal strategy for both players was to co-operate with probability 0.66 at time t = (T - 2) and with probability 1.00 when t ≤ (T - 3).

		Player 2	
		C	**D**
Player 1	**C**	**0.9**	**0.8**
	D	**0.95**	**0.82**

Figure 3.3: Payoff matrix for the anti-predator game described by Lima[67]. C represents a strategy of co-operation and D one of defection. The matrix shows the probability of player 1 surviving an attack given that each player plays the strategies shown. The game is symmetric so player 2 is faced with the same set of probabilities.

3.3.2 Convergence upon the ESS by successive approximations

Sometimes a dynamic behavioural game which could theoretically be solved in the manner described above cannot practically be done so. If the number of players is large, it is not computationally feasible to consider all possible combinations of their states and actions, past and present. In some cases, the ESS may instead be converged upon by successive approximations. This procedure is described with reference to an example from Houston & McNamara[68].

Consider a male bird who, during daylight hours in springtime, must decide how to divide his time between foraging and singing. His energetic expenditures during both these activities are increasing functions of his energetic reserves x and his overnight survival also incurs some energetic expense. Should his reserves fall to zero, he starves. If he forages, he gains some random quantity of food R. If he sings, there is some probability that he will attract a mate and this probability may depend upon the behaviour of other males. Cases were considered in which the interaction between males' songs was either positive or negative, that is, the probability of attracting a mate was either an increasing or decreasing function of the number of males singing.

It was initially assumed that all members of a population except for a focal male followed a given state-dependent strategy. (Specifically, they all followed the strategy which was optimal in the case when no interaction effects were considered.) A forward iteration was run for non-focal males, starting from some specified population at time t = 0, to find the proportion singing as a function of time. The optimal strategy for a focal male was then computed, by a backward iteration, under the assumptions that the non-focal strategy was known to him; the local population was large and; his probability of attracting a mate was a function of the proportion rather than the absolute number of males singing. Any stochastic effects in the exact number of males singing at any given time could therefore be ignored. The focal male was modelled as 'playing the field' (see section 3.2.3). It was assumed that a mutant who followed the focal strategy had arisen and that this strategy then spread through the population - since such a mutant would be fitter than other members of the population - so that, at the next stage, it was followed by all non-focal males. Forward iterations for non-focal males and backward iterations for focal males were then run cyclically, with the non-focal strategy updated after each backward iteration.

When interaction between male strategies was positive, the ESS (i.e. the strategy which, when followed by all other members of a population, would be optimal for a mutant) was converged upon in this way. Bouts in which many males sang together were predicted and the frequency and duration of these was higher during the early part of the day; in particular a 'dawn chorus' was seen at the very start of the day in which nearly all males sing. During the latter part of the day, more time was spent foraging in order to obtain sufficient reserves to last the night. Interestingly, one strong bout was seen at dusk. Since the value of food obtained when foraging was unpredictable, individuals began foraging slightly earlier in the day than was usually needed to obtain sufficient reserves to survive the night and many were able therefore to spend the past periods of the day attempting to mate.

When interaction between male strategies was negative, the strategies of focal and non-focal males did not show convergence. Rather, after a number of cycles, strategies began to oscillate with the non-focal strategy from one cycle being that predicted for the focal during the next cycle. This may indicate that no ESS exists or that the ESS is a mixed strategy.

This approach - of computing an optimal strategy by means of successive approximations - is used for the model on sexual dimorphism in baboons described in chapter 4. (As a final note, it is possible that multiple ESSs exist, and that different ones may be reached, dependent on the choice of starting population and initial non-focal strategy. If this is thought to be likely, multiple initial conditions may need to be tried in order to find the complete set.)

Chapter 4: Sexual dimorphism in baboons

4.1 Introduction

4.1.1 Dimorphism and sexual selection

As stated in chapter 1, a number of hypotheses have been put forward to explain the dimorphism in longevity which is often seen between the sexes. These include the unprotected X hypothesis, the accumulation of male-specific deleterious genes in cytoplasmic genomes, differences in disease burden and immunity, the fact that males are more likely to engage in risky behaviour and therefore die from accidents or in fights and, the existence of sex-specific selection pressures which favour differential allocation to maintenance. Some of these relate to explanations for dimorphism in size as well. Insofar as size improves competitiveness in agonistic encounters, it is expected that the sex that engages in fighting more often will be both larger and less long-lived. A higher foraging yield must be achieved by larger individuals and exposure to ingested pathogens and parasites, and therefore mortality, is thus increased. If the rate of ageing is 'chosen' as part of an optimal life history, then the optimal investment in maintenance can only be understood within the wider context of energy allocation decisions between all fitness-enhancing functions, including growth. It is this last explanation - that dimorphism in both size and longevity results from different optimal energy allocation decisions between the sexes - that is considered in the current chapter.

Under this hypothesis, the dependency of fitness upon lifespan and maximum size is different for males and females. In many species, males invest little in offspring care so that costs associated directed with reproduction - essentially the costs of mating - are rather low and concentrated within a narrow window. In contrast, the costs borne by the mother are considerable and span a lengthy period of time: from the point of conception until the offspring attains independence. The expected number of offspring which survive to reproductive maturity will therefore correlate much more strongly with lifespan for a female than a male; a female must survive and provision each successively whilst a male may achieve high reproductive success by securing multiple mating opportunities within a relatively short time frame. To this end males

invest heavily in secondary sexual characteristics, notably a large body size which provides a competitive advantage in agonistic encounters with other males. Whilst a female will make some investment in growth to lay down stores for the future provisioning of herself and her offspring, this is greatly reduced in comparison to a male since she needn't compete with other females for mates. Both sexes are optimally partitioning their available energy budget between growth, maintenance and reproduction however, once it is assumed that females are the chief provisioners of offspring and males must compete for mating opportunities, differences in the functions relating state to fitness will drive selection for greater growth in males at the expense of more rapid ageing.

In the above scenario, the fitness consequences of a particular male's allocation strategy will depend upon the strategies that other males are following. If the majority of males in a population are small, the optimal strategy for a mutant may be to grow to be slightly larger than the average. As this strategy spreads, the mating success associated with this slightly larger size is reduced so that a mutant may now do better to grow slightly larger again. The average size distribution of the population will increase until the benefit of further growth is outweighed by the costs. The latter may include time constraints imposed by the greater foraging effort needed to support a larger size; a reduced probability of survival to breeding age (if it is assumed that breeding is delayed until the completion of growth) and; a reduction in the reproductive lifespan (for the previous reason and/or if investment in maintenance is compromised by additional growth).

If the above hypothesis is true and dimorphism is the product of sexual selection, we should expect to see certain correlated sets of life history traits across species. Species which are highly dimorphic in body size should be more likely also to exhibit dimorphism in lifespan, to show greater levels of competition between males and little male investment in offspring post-conception. There is some evidence from comparative data sets that this is the case. In anthropoid primates, for example, body weight dimorphism is known to be associated with both the frequency and intensity of male-male competition[62]. In those species for which males have the largest body size relative to females males are least tolerant of each other, often engaging in escalated combat and forming clear social dominance hierarchies based on agonistic

encounters. The largest males tend to be the highest ranking and are better able to guard reproductive females against the approaches of rival males.

Evidence such as this gives qualitative support for the proposed sexual selection hypothesis for dimorphism; it shows that size gives a competitive advantage to males, but it doesn't tell us whether the magnitude of this is sufficient to explain the degree of dimorphism seen. This is important since the various hypotheses mentioned above are not mutually exclusive. A quantitative test of whether dimorphism can be explained solely by sex-specific optimal energy allocation requires that the fitness consequences of all possible allocation strategies be computed and that the optimum be shown to reproduce the life histories seen in the wild. The model described in this chapter does this for baboons: it computes the optimal partitioning of energy between growth, maintenance and reproduction for both sexes based on data in the literature.

Baboons were chosen for several reasons. Firstly, they have been extensively studied for a number of decades, both in the wild and in captivity, and much is known about their ageing rates, growth trajectories, social dynamics, reproductive strategies and interactions with the abiotic environment. Secondly, the degree of sexual dimorphism is striking and consistent across almost all populations and species of baboon. Adult females are about half the size of their male counterparts and significantly longer lived. Thirdly, their mating and reproductive behaviour matches that described above: females provide the majority of offspring care and there is fierce male-male competition for mates with size being a clear predictor of success.

4.1.2 Baboon ecology and life history

There are five baboon species which form the genus *Papio*: *P. anubis* (olive baboons), *P. hamadryas* (hamadryas baboons), *P. ursinus* (chacma baboons), *P. cynocephalus* (yellow baboons) and *P. papio* (Guinea baboons). They occupy a variety of equatorial and temperate habitats throughout sub-Saharan Africa, including desert, semi-arid savannah, mountains and forests. However - with the exception of *P. hamadryas* which inhabit arid regions in the horn of Africa - these species are most often found in savannah regions.

4.1.2.1 Baboon social structure

Savannah baboons live in multi-male, multi-female groups, or troops, which typically range in size from 25-200 individuals. They are matrilocal: females remain in their natal groups throughout life whilst males emigrate to new groups as they approach adulthood and may undergo multiple subsequent dispersals later in life[69-71]. No structured sub-groups have been identified within these larger troops. Strict dominance hierarchies exist within troops and have been determined on the basis of dyadic approach-avoidance interactions and supplants over resources. Whilst female dominance hierarchies are stable both between and within generations, with daughters assuming a rank just below that of their mothers, male dominance hierarchies are unstable and age-graded. Males achieve their highest rank in early adulthood when they are at their largest and before the effects of age on robustness emerge. Lifetime reproductive success correlates positively with rank for both sexes although the association is stronger for males. The primary benefit of rank to females is improved foraging efficiency - subordinate individuals may be displaced from prime foraging sites - and to males is increased access to reproductive females. All adult males are dominant to all adult females.

The troop moves as a whole between the foraging grounds that they occupy during the day and the cliffs used as sleeping sites at night. Owing to their height and steepness cliffs provide safety against nocturnal predators - primarily leopards - at a time when baboons would otherwise be especially vulnerable[72]. As travel is restricted to daylight hours, there can be considerable seasonal variation in the distance covered. For GPS-tracked chacma baboons in the Wildcliff Nature Reserve in South Africa, the monthly mean daily path lengths ranged between 3.6 and 6.5km[73]. The choice of foraging ground will depend upon a number of factors including the quality and distribution of food patches; the costs of travel in terms of time and energy; the distance to a waterhole and; the risk of predation during travel and whilst foraging. In particular, evidence for a trade-off between expected foraging yield and predation risk is strong with the optimal decision being largely dependent upon group size.

In the majority of baboon populations the main cause of adult and juvenile mortality is predation[72, 74, 75]. The most common predators are lions and leopards, but baboons may also fall prey to hyenas, crocodiles, pythons, jackals, dogs and

chimpanzees. Individuals in larger groups experience a reduced predation risk through a simple 'dilution effect'. These groups tend to be more dispersed; more likely to accept foraging grounds that are further from refuges or that have lower visibility and; show decreased rates of individual vigilance. It is probable that larger groups, because they contain a greater number of adult males, may also be more effective at repelling an attack. Cooperation between males in mobbing a predator, either as a retaliatory or pre-emptive action, is well-documented and usually successful. Weighed against this reduced predation risk are an increase in foraging competition and possibly in the rate of disease transmission. One study found that an increase of ten adult females in a group of yellow baboons produced a 2.5 month increase in the inter-birth interval (IBI) presumably because maternal foraging efficiency was reduced[76]. The resolution of this trade-off, that is the optimal group size, will likely vary with the specific predator density and food patch distribution within each troop's home range. In cases where troop size has fallen dramatically, for example following heavy flooding or predation, fusion has been observed between neighbouring troops. Conversely, when troops become especially large, fission to produce two equally-sized new groups is seen.

The social structure of hamadryas baboons is markedly different. The smallest social division is the one-male unit (OMU) composed of a single leader male, a number of adult females and their young and sometimes a follower male. Leader males hold exclusive mating rights to the females in their OMU - in whom their interest is intense and sustained regardless of reproductive state - and do not compete for mating opportunities outside of the OMU[77]. Several OMUs led by related males comprise the foraging unit known as a clan and clans are assembled into bands which are analogous to the troops of savannah baboons. A troop of hamadryas baboons comprises a number of bands which occupy the same sleeping cliffs. Both sexes disperse upon maturity. Females will either choose, or be aggressively herded into, an OMU. Males may be solitary for some time before winning/forming an OMU or may join an OMU as a follower whose presence is tolerated by the leader but who possesses no mating rights. OMU takeovers follow a successful challenge to the leader; the death of the leader from predation or disease or; the peaceful transfer of power to the follower male[78]. Hamadryas baboons show the same sexual

dimorphism in size and longevity as savannah baboons but their reproductive strategies are rather different, which may be either a cause or a consequence of their different social structure. Information on hamadryas baboons is included here for completeness - the remainder of this introduction and the model here presented apply only to savannah baboons.

4.1.2.2 Sexual dimorphism

Baboons show significant sexual dimorphism in longevity, body size, reproductive scheduling and various other aspects of behaviour[79]. As already noted, female savannah baboons remain in their natal group throughout life whilst males disperse to join new groups as older sub-adults or young adults. Mating behaviours are starkly different and details of these are given in the next two sections.

The maximum recorded lifespan for a wild baboon is 27 years although ages of 30 and over have been attained in captivity. Overall longevity as well as the lengths of most life history stages are approximately one third what they are in humans[69, 70]. Females are considerably longer-lived than males and from their mid-teens onwards look noticeably younger than their male counterparts. It is not uncommon for a female to live into her third decade in the wild but extremely rare for a male to do so. As for many species, mortality rate increases exponentially with age and can be modelled by a Gompertz function.

Growth trajectories are similar for males and females until about five years of age at which point males begin a sub-adult growth spurt, eventually attaining an adult body mass index (BMI) 50% greater and a weight 100% greater than females[62]. Females are classified as adults when they first birth a live young, typically in their fifth or sixth years; males are classified as adults when they have (almost) achieved their final size and have fully developed canines, typically in their eighth or ninth years. Both sexes experience a reduction in size with age, starting at about ten years. Both the absolute and relative declines in size are greater in males.

From her first conception onwards, a female's reproductive output is fairly constant until her early twenties, at which point the average inter-birth interval (IBI) and the chance of foetal loss increase. A complete cessation of reproduction has only been observed in the very oldest individuals (25 years and over). Males are rather older

than females when their first offspring is born: on average about eight years[80]. Their reproductive output increases steeply during late sub-adulthood and early adulthood to a peak at about ten years. At this point the rate of siring offspring is approximately 2.5 times the birth rate for females of the same age. Reproductive output then declines rapidly throughout the teenage years, until it is effectively zero at age nineteen/twenty.

4.1.2.3 Female reproductive behaviour

Females exhibit highly-visible sexual swellings during the oestral phase of their cycle which advertise their sexual receptivity. Generally females will mate with whichever males win access to them, however, there does seem to be some scope for them to employ their own mating strategies as well[81]. Females may strive to copulate with multiple males by means of sexual presents, post-copulatory darts and copulation calls, the latter being thought to draw attention to a mating pair and so invite competition from other males. It is presumed that these behaviours constitute a strategy of paternal confusion; they serve to increase the number of resident males who have a potential genetic stake in an offspring and will therefore defend such an offspring against infanticide by newly-immigrant males. A male-biased operational sex ratio, relatively long IBI and low rate of infant mortality from other causes make infanticide a chronic threat and the primary cause of death in those under one year. This strategy also has the effect of raising the reproductive success of subordinate males since solicited copulations are not preferentially directed towards high-ranking males. There are no permanent mating bonds and little association between the sexes outside of the oestral period.

Females reach menarche between four and five years of age then experience a period of sub-adult infertility of approximately one year before conceiving their first offspring. Age at first conception is lower in high-ranking females - at least for chacma baboons in the Moremi Game Reserve in Botswana - but only when they have close female kin (mothers or sisters) living. Although there is generally little seasonality in reproduction, there is some evidence that the probability of conception increases in response to the relative abundance of food which follows a period of heavy rainfall. Over the course of a six-month gestation females lay down body stores in preparation for lactation; how this is achieved seems to vary between populations but can involve

a reduction in physical activity and/or an increase in energy intake. The probability of not carrying a pregnancy to term increases with parity, age and weight[82, 83].

Infants are completely dependent upon their mothers until they are weaned at about one year, and then achieve increasing independence throughout their second year. Lactation is the most energetically-expensive time of a female's life and involves a drop in body weight despite an increase in foraging yield[84, 85]. Infanticide is a particular risk during the first six months when young are still being carried by their mothers and is especially high in the offspring of primiparous mothers. The chances of infant survival have been shown to be improved by the existence of a close, enduring friendship between the mother and another female.

Assuming offspring survival, a female will resume cycling during her offspring's second year with a few non-conceptive cycles being common before she conceives again. The choice of IBI represents a trade-off between offspring number and quality; lengthening it will increase the expected reproductive success of the current offspring at the expense of future offspring. An increased IBI is seen in older mothers, presumably because, being smaller, they are unable to invest as much in an offspring per time interval as their younger counterparts and prolong the period of investment in compensation. The IBI is slightly reduced in high-ranking females who enjoy increased access to food and are therefore able to support a more rapid growth in their offspring. The mother will start to put on weight again as soon as her offspring is weaned and this will continue throughout the next pregnancy to peak again just before she gives birth to the next offspring.

The overall reproductive success of a female is dependent upon lifespan; rank; the distribution of predators and foraging sites; choice of IBI; the presence of close kin and maintenance of friendships; the effectiveness of paternal confusion against the threat of infanticide and; the stochastic nature of mortality on offspring. Of these factors, the greatest proportion of the variance in lifetime reproductive success is accounted for by lifespan[76].

4.1.2.4 Male reproductive behaviour

A male's reproductive success is primarily dependent upon his ability to establish consortships with oestrous females. A consortship is defined as 'a continuous, close

spatial association between a male and a sexually receptive female, with evidence of sexual activity by the male'. Success at this is strongly correlated with rank, and hence with size since size is the principal determinant of rank in early- and mid-adulthood. This was recognised by Altmann in his Priority of Access Model [86] according to which a male may only secure a consortship when all higher-ranking males are already engaged in one and are therefore removed from further competition. At least two females must therefore be oestrous at the same time for any but the highest-ranking male to mate. Field studies have shown that whilst high-ranking males certainly consort at higher rates, they do not monopolise matings to the extent that is predicted by the Priority of Access Model. The reasons for this are not fully understood but it is likely that the proportion of days on which a male consorts is constrained by the energetic costs involved. Foraging activity may be constrained both spatially and temporally; the former because of the need to remain close to the female and the latter because a state of vigilance against the approach of potential rivals is incompatible with foraging. There will also be energetic costs involved in the act of mating itself and combat with challenger males. In view of this, high-ranking males do not consort throughout the entire oestrous period but will concentrate most effort within the window when conception is most probable. If the first day of subsidence of the sexual swelling is designated day 0, the conceptive period is defined as [-7, +1] and the period of most likely conception as [-3, 0][86]. Males also seem able to estimate the probability of conception on a given cycle as they consort at a much higher rate on conceptive compared with non-conceptive cycles. It is possible that males can also estimate the quality of potential mates and the highest-ranking will concentrate effort on those on are most likely subsequently to raise any offspring conceived to independence.

Low-ranking adults and sub-adults can still gain some mating opportunities although these are more limited than for high-ranking adults. They may gain 'sneak' matings when the group is highly dispersed or visibility poor, or following a post-copulatory dart by a female[87]. (It is thought that the social structure and mating behaviour of hamadryas baboons evolved in consequence of the fact that they occupy hilly terrain where visibility is often poor and it would be extremely difficult for high-ranking males to monopolise matings via the procurement of consortships.) More opportunities may

be available outside of the period of peak oestrous when the probability of conception is low but non-zero and when interest in the female from higher-ranking males is lower. Two or more low- or mid-ranking males can also form a coalition which is able to displace a single higher-ranking male. Interestingly, this strategy is frequently seen in yellow and olive baboons but not in chacmas. Where it is seen, it tends to be adopted by older males, whilst younger ones rely on solo tactics[88]. When displacement of a male in consort occurs, either by a solo male or a coalition, it can sometimes be a bystander who is the first to reach the temporarily unguarded female and mate with her, and again lower ranks may improve their reproductive success by such a means.

There is some controversy as to whether males can gain mating access by developing friendships with females or through an ownership effect. Reports of a male and female remaining in closer proximity to each other than would be expected by chance outside of a consortship usually follow a successful mating between the pair and may reflect a strategy of paternal care rather than the attempt to solicit future matings. It has been suggested that pre-existing pairings between mutually-preferred partners may be respected by other males in a troop, but there is as yet no strong evidence for this[79].

Albeit the majority of offspring care is undertaken by the female, there is evidence of paternal care in baboons. Yellow baboon males in the Amboseli National Park in Kenya were found to assist their juvenile offspring in agonistic encounters significantly more often than they would unrelated juveniles. These males seemed able to distinguish their own offspring and preferentially direct care towards them[89, 90]. Furthermore, the presence of the father was associated with accelerated maturation in both sexes, although for males it was conditional upon the father holding a high rank at the time of conception.

As stated above, infanticide accounts for the majority of deaths in those under one year. Following the death of an infant, the mother will resume cycling sooner and so the pool of mating females in the near future will be swelled. This will improve a male's reproductive success assuming that the current offspring is not his and is therefore a strategy employed by males that have recently immigrated into a group.

Finally, a male's reproductive strategy includes the decision either to stay in his current troop or leave and seek a new one. Secondary dispersals should only be undertaken when the expected mating opportunities in a neighbouring group exceed that in the current group by a margin large enough to outweigh the costs associated with dispersal. Expected mating opportunities will depend upon the state (size and age) of the male as well as the demography of the current troop and of neighbouring troops. The costs of dispersal include the increased predation risk when travelling alone, the danger of not being accepted into another group and the inability to provide future paternal care to offspring in the group that is left.

The total lifetime reproductive success of a male is a function of lifespan; rank; the distribution of predators and foraging sites; the compositions of the groups of which he is a member; group dispersal and visibility and; his policy of paternal care and infanticide. Some of these factors are ones upon which a female's reproductive success also depends although the nature of the dependency is different. The most important determinant is rank which is heavily dependent upon size[79].

Given the life histories described above, the sexual selection hypothesis seems a good candidate to explain the sexual dimorphism seen in baboons. Males compete for access to oestrous females and females provide the vast majority of infant care. The strongest predictor of lifetime reproductive success for males is maximum size and for females is lifespan. The test this quantitatively, a dynamic programming model was constructed in which males and females make a series of decisions throughout their adult life as to how to split their available energy budgets between growth, maintenance and reproduction. The fitness consequences of all possible decisions were calculated according to a simplified model of the social dynamics and reproductive behaviour outlined above. Optimal state-dependent strategies were calculated for both sexes and a population following such strategies was simulated to determine whether these would reproduce the dimorphism seen in the wild.

4.2 Aims

1. To demonstrate how dynamic programming may be used to create a two-sex model of behavioural decisions, with the decisions made by each sex affecting those of the other.

2. To determine whether sexual dimorphism in body weight and lifespan can be explained in terms of optimal energy allocation between growth, reproduction and maintenance in baboons. More specifically, does a social structure in which females provide all offspring care and males must compete for access to reproductive females drive males towards in higher investment in growth at the expense of maintenance?

3. To quantify the extent to which the differential longevity of males and females can to attributed to differences in the initial mortality threat to which adults are exposed rather than differences in the rate of ageing.

4. To determine how the fitness of suboptimal strategies compares to that of the optimal and therefore how strong the force of selection is for the optimum.

5. To explore how robust the outcome of the model is to changes in parameter values. Are there regions of parameter space for which we expect dimorphism to be enhanced or reduced?

4.3 Methods

4.3.1 Model overview

Consider first the female decision process, shown in figure 4.1 (top). At time t, a female is defined by her size (s), condition/biological age (c) and the age of any dependent offspring she has (d). She is subject to a mortality threat the magnitude of which is dependent upon her state. If she survives this, she achieves a foraging yield, also state-dependent, a given amount of which must be diverted to essential functions. Essential functions are those which must be met to survive the current period; these comprise vital metabolism (breathing, thermoregulation etc.) and the costs of travel in order to reach foraging grounds. The remainder of her foraging yield she may freely allocate between growth, reproduction and maintenance. She may also choose to lose weight and use the energy she has stored in biomass for either reproduction or maintenance.

Her investment in growth, maintenance and reproduction at time t will determine respectively her size (s'), condition (c') and the age any of dependent offspring (d') at time (t + 1), where she is again faced with a similar set of decisions. To raise an offspring to independence requires continual investment in reproduction over the period of pregnancy and lactation. Her optimal decision is that which maximises her fitness gains from the current period - the fitness she accrues in raising an offspring to independence by the end of the current period - and her expected reproductive value at time (t + 1) which will be a function of her state at the start of the next period.

Now consider the male decision process, shown in figure 4.1 (bottom). At time t, a male is defined by his size (s) and condition (c). As for females, he is subject to a state-dependent mortality threat, survival of which leaves him with the choice of how to distribute the remainder of his foraging yield after essential functions have been met between growth, reproduction and maintenance. He may also choose to invest negatively in growth, i.e. he may choose to utilise body stores for reproduction and/or maintenance. Energy invested in reproduction will go towards attempting to maintain consortships with, and so gain mating access to, receptive females. His fitness gains from reproduction will be a function of his success at impregnating females and of the likelihood that these females will subsequently raise his offspring to independence. In

contrast to females, the reproductive opportunities available to him will depend upon his social environment: the distribution of receptive females and the distribution and allocation decisions of other adult males. If there are many larger males with whom he is competing for access to relatively few receptive females, his chances of success will be low. To use the parlance from chapter 3, he is modelled as playing a game against the field. This game is asymmetric (expected success at establishing a consortship will depend upon state), simultaneous (males must all choose their strategy at the start of the period) and non-zero-sum (the fitness gains associated with establishing a consortship need not equal the losses incurred by all males all compete for the said consortship). It is assumed that a focal male knows both the population distribution and the strategy employed by other males and makes his allocation decision accordingly.

His investment in growth and maintenance will determine respectively his size (s') and condition (c') at the start of the next period. The optimal decision is that which maximises his expected reproductive success in the current period plus his expected reproductive value at time (t + 1) the latter being a function of his state at the start of the next period.

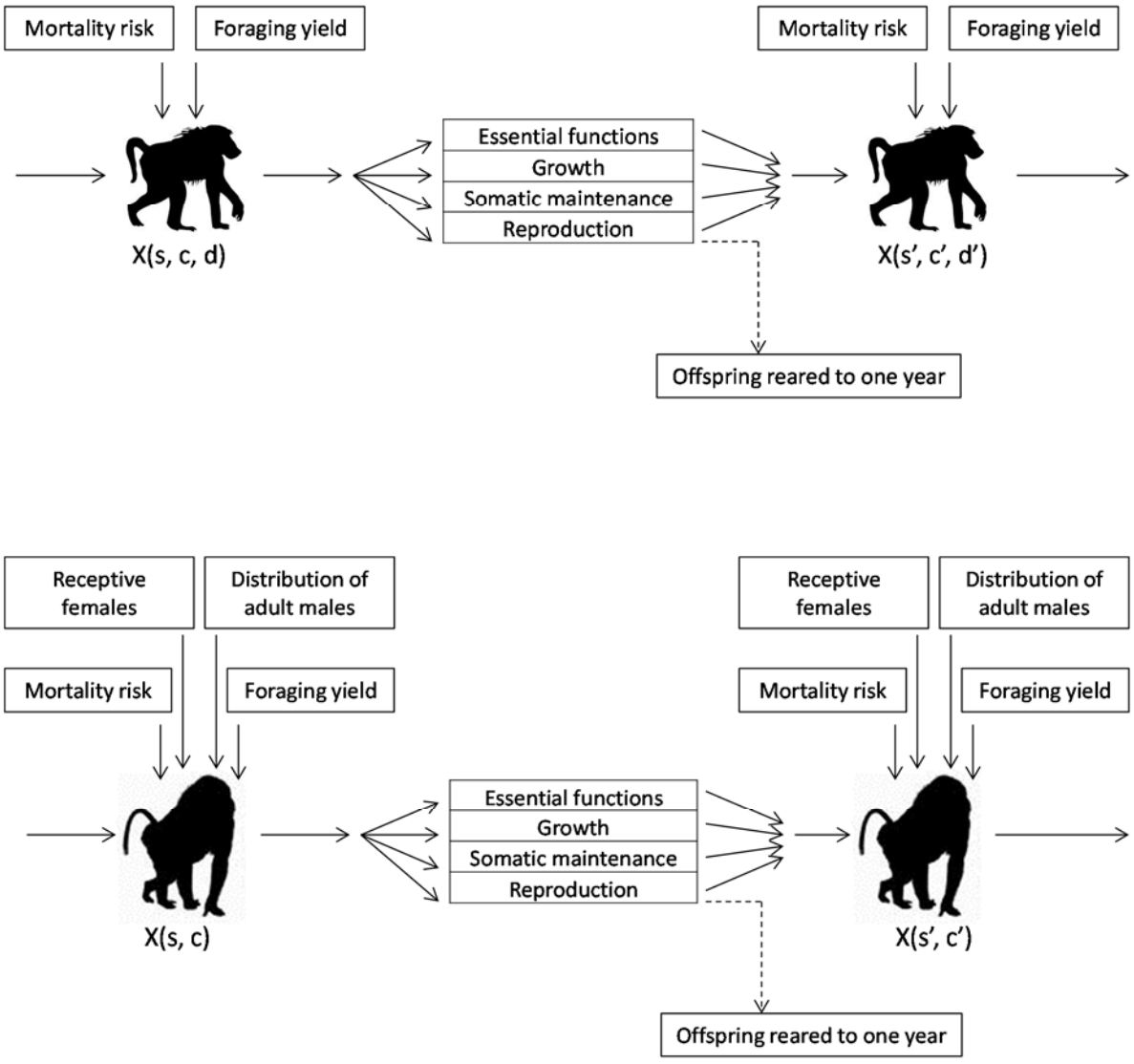

Figure 4.1: Schematic representation of the decision process for females (top) and males (bottom). Size, condition and age of dependent offspring are indicated by s, c and d respectively at time t and by s', c' and d' at time (t + 1).

4.3.2 Model structure

As described in chapter 3, a formal specification of the model requires the following: a measure of fitness; the time interval between successive decisions; the state space; a decision set and; the state dynamics as given by dynamic programming equations.

Fitness is defined as the expected number of copies of an individual's genes at some time point far enough into the future such that the optimal strategy is independent of time.

Time periods are of length $\tau = 36$ days; the average duration of the menstrual cycle in baboons. This is the length of time between which a female (if she is not already pregnant or lactating) can make successive decisions whether or not to begin investment in reproduction.

4.3.2.1 State space

A female is described by the state variable $\mathbf{x} = (s, c, d)$ where s is size (in kg), c is condition (in time periods τ) and d is the age of any dependent offspring (in time periods since conception). A male is described by the state variable $\mathbf{x} = (s, c)$.

Size can take the values 8.0, 8.5, 9.0, … 20.0 kg for females and 8.0, 8.5, 9.0, … 30.0 kg for males. This is consistent with the range of weights reported in the literature[62]. Upon reaching maturity, all individuals are of size 8.5kg. If weight subsequently drops to 8kg the individual dies. The lower maximum size boundary for females was imposed to reduce the model run time following a preliminary simulation to confirm that individuals did not hit this boundary and therefore that any predicted size dimorphism is not attributable to it.

Condition can take the values 50, 51, 52, … 300τ. For the purpose of this model, reproductive maturity is said to be reached at 50τ (a biological age of approximately five years) for both sexes. At this age, males are classed as sub-adults, only attaining full adult status - and generally only beginning reproduction - a few years later. However, males are capable of producing viable sperm at five years and offspring have been sired in colonies where all males over five have been vasectomised as a means of population control. It can be assumed then that this is the age at which they are first able to make energy allocation decisions between growth, reproduction and

maintenance. Importantly, any choice to delay reproduction until maximum size and full adult status are reached will be an outcome rather than an assumption of the model. At 300τ the individual is no longer able to survive; this corresponds to a biological age of approximately 30 years, an age to which no wild baboon has been known to live.

Age of dependent offspring can take integer values between 0 and 14 inclusive, with d = 0 at conception and d = 5 at birth. Pregnancy therefore lasts five periods (180 days) and lactation ten periods (360 days). A female will only care for one offspring at a time. It is assumed that to raise an offspring requires a fixed schedule of energetic investments so that all offspring of a given age are of equal quality. Partitioning of energy between growth and maintenance by the offspring is not considered.

The state space is of size |s|*|c|*|d| = 25*251*15 = 94,125 for females, and |s|*|c| = 45*251 = 11,295 for males.

4.3.2.2 Dynamic programming equations

Each individual has an available energy budget equal to their foraging yield plus that energy which can be made available from weight loss minus that which must be spent on essential functions. The dynamic programming equations tell us how this available energy budget should be optimally allocated by relating current state and decision to immediate reproductive gains and future state.

The maximum weight loss per period is either three kilograms or the difference between current weight and the minimum allowable weight of eight kilograms, whichever of these is lower. Although three kilograms may be a significant proportion of an individual's total size, baboons do seem to be able to experience such rapid weight loss without it being obviously injurious to health. In one study, lactating mothers exposed to 60% DR, lost on average 2.7kg per month and still appeared healthy[91]. As an equation, we have:

growth ≥ max(8 - s; - 3) kg

The dynamic programming equation for females is as follows:

$$F(s, c, d, t, T) = (1 - \beta(c))*[\max_i \max_j E\{F(s + \gamma(j), c + \zeta(k), d'(d, i), t+1, T) + 0.5*\Psi(d, i)*\phi*F(8.5, 50, 0, t + 41, T)\}]$$

where:

- F(s, c, d, t, T) is the maximum expected reproductive value for a female of size s, in condition c and with dependent offspring d at time t, given a final time horizon T. The maximisation is taken over the set of possible decisions for partitioning available energy between growth, maintenance and reproduction.
- β(c) is the probability of succumbing to mortality during the period (t, t + 1). It is assumed that mortality acts before allocation decisions are made and that risk is a function only of condition.
- i is the energy (in kcal) invested in reproduction. This may take the values zero or ρ(d), where ρ(d) is the energetic requirement of an offspring of age d.
- j is the energy (in kcal) invested in growth. This may take values in the range:

  $$v*\max(8 - s; - 3) \leq j \leq \xi(s) - i - \alpha(s),$$

  where v is the energy obtained from the loss of one kilogram of body weight; ξ(s) is the foraging yield and; α(s) is the cost of essential functions.
- k = (ξ - i - j - α) is the energy (in kcal) invested in maintenance.
- E indicates that an expectation is taken over d'(d, i) and Ψ(d, i) defined below. This reflects the stochastic nature of current fitness gains associated with a particular strategy resulting from the fact that offspring mortality is modelled as acting after the maternal allocation decision.
- γ(j) returns the change in weight resulting from a given energetic investment in growth.
- ζ(k) returns the change in condition resulting from a given investment in maintenance.
- d'(d, i) is the age of any dependent offspring at time (t + 1). If the mother dies or chooses not invest in a dependent offspring, the offspring will die. If the mother survives and invests in the offspring, the offspring will survive the current period with probability σ(d). So:

  if i = 0, d' = 0

  if i= ρ(d), d' = (d + 1)mod15, with probability σ(d)

  d' = 0, with probability (1 - σ(d))

- One half is the relatedness between mother and offspring.
- $\Psi(d, i)$ is set to one if an offspring is successfully reared to independence at the end of the current period.

  if $i = \rho(d)$ and $d = 14$, $\Psi(d, i) = 1$, with probability $\sigma(d)$

  $\Psi(d, i) = 0$ otherwise
- ϕ is the probability that a juvenile will survive from age 10τ to maturity at age 50τ.

The terminal fitness function for females is as follows:

$F(s, c, d, T, T) = 1$ if $s \geq 8.5$ and $c < 300$

$F(s, c, d, T, T) = 0$ if $s = 8.0$ and/or $c = 300$

Note that in the equation above, it is assumed that all offspring are female. For the initial simulation this assumption had to be made because there were as yet no figures calculated for male reproductive value. After the male backward iteration was run, the calculated time-dependent reproductive values for males upon reaching maturity were used to run the female backward iteration again with the above equation updated so that half a female's offspring are male and half female. Male and female simulations were then run cyclically until the stable strategies calculated for both did not vary between one run and the next (see section 4.3.4).

The dynamic programming equation for males is as follows:

$$F(s, c, t, T) = (1 - \beta(c))*[\max_i \max_j E\{ F(s + \gamma(j), c + \zeta(k), t+1, T) + 0.5*\chi(s, c, i, \Omega_f, \Omega_m)*\lambda(\Omega_f)*\phi*[0.5*F(8.5, 50, 0, t + 55, T) + 0.5*F(8.5, 50, t + 55, T)]\}]$$

where:

- $F(s, c, t, T)$ is the maximum expected reproductive value for a male of size s and in condition c at time t, given a final time horizon T. As above, the maximisation is taken over the set of possible decisions for partitioning available energy between growth, maintenance and reproduction.
- $\beta(c)$, i, j, k, $\gamma(j)$, $\zeta(k)$, ϕ and $F(s, c, d, t, T)$ are defined in the same way as for females.

- i may take values in the range:

  $$0 \leq i \leq \min(\xi(s)*\eta, \xi(s) - \alpha(s) - v*\max(8 - s; - 3))$$

  where ξ(s), α(s) and v are defined as above. It is assumed that investment in reproduction is equivalent to a loss of foraging yield; for all days upon which a male attempts to hold a consortship, he loses a fixed proportion η of the original foraging yield for that day. So, ξ(s)*η is the investment in reproduction for a male who attempts to hold a consortship every day of the period (t, t + 1). The maximum possible investment in reproduction is less than this if and only if the cost of essential functions exceeds the proportion (1 - η) of the original foraging yield plus the energy available from weight loss.
- j may take values in the range:

  $$v*\max(8 - s; - 3) \leq j \leq \xi(s) - i - \alpha(s)$$
- Ω_f is the stable distribution of adult females.
- Ω_m is the stable distribution of adult males. For the first male backward iteration, a strategy to be followed by all non-focal males was chosen arbitrarily and the stable distribution of individuals following this strategy computed. The optimal strategy calculated for a focal male then became the strategy employed by non-focal males in the next iteration and successive iterations were run until the focal and non-focal strategies were identical, as described in chapter two and in section 4.3.4.
- $\chi(s, c, i, \Omega_f, \Omega_m)$ is the expected mating success of the focal male.

The terminal fitness function for males is as follows:

$F(s, c, T, T) = 1$ if $s \geq 8.5$ and $c < 300$

$F(s, c, T, T) = 0$ if $s = 8.0$ and/or $c = 300$

4.3.2.3 Decision set

In order to give a finite decision set, a discrete number of allocation decisions for reproduction and growth are specified, with the remainder of the energy budget going towards maintenance.

An adult female can decide either to invest in reproduction or not. As stated above, if an investment is made it is always of a fixed amount, dependent on the value of d at

the start of the period. The investment will go towards provisioning of a foetus (if d = 0-4) or lactation (if d = 5-14).

An adult male must decide the number of days upon which he will attempt to maintain a consortship with a female. This may be zero or some multiple of four up to a maximum of either a) 36 or b) the highest multiple of four for which the foraging yield forgone does not exceed the available energy budget, whichever is lower. Multiples of four days were chosen as this is the length of peak oestrous, when a female is most likely to conceive. Each four-day period in which a male attempts consortship is therefore an attempt to mate with a single female. The energetic cost of attempting to maintain a consortship is independent of success.

Investment in growth is required to be some multiple of 500kcal. The maximum investment in growth is the integer quotient of the foraging yield minus the cost of essential functions minus what is spent on reproduction, and 500. The minimum investment in growth is the integer quotient of the maximum amount of energy which can be obtained from weight loss and 500.

4.3.3 Parameterisation

4.3.3.1 Adult mortality rates

Mortality rates are assumed to follow a Gompertz distribution; they are modelled by the hazard function $\beta(x) = \delta * e^{\varepsilon * x}$ where δ is the initial mortality rate (IMR), ε is the rate of ageing (RoA) and x is age in years since attaining maturity. Values for δ and ε are taken from Bronikowski et al.[92]. These authors report an IMR of 0.0285/year for females and 0.0371/ year for males, and a RoA of 0.123 for females and 0.213 for males.

In the model, it is assumed that both sexes experience the RoA quoted for females. That is, both sexes will age at the same rate if they invest an equivalent amount in somatic maintenance. Any difference in ageing rate is therefore a behavioural choice and an outcome of the model rather than an a priori assumption. Differences in IMR are assumed to be externally imposed and independent of behavioural decisions. The validity of this assumption is discussed in section 4.5 however it is noted here that males are more likely to be injured in aggressive encounters and to the extent that

these are outside of an attempted consortship they may be said to be independent of strategy. There is also evidence that males are more susceptible to predation. We have the following equations for adult mortality rates per period:

$\beta(c) = 0.00285e^{0.123*(c-50)/10}$ for females

$\beta(c) = 0.00371e^{0.123*(c-50)/10}$ for males

4.3.3.2 Energetic costs of essential functions

Following Shanley & Kirkwood [46], vital metabolism - that required for immediate survival - is estimated at half the basal metabolic rate (BMR). This is broadly consistent with data on starvation; when caloric intake falls to below half the BMR, organisms will die from a failure to meet immediate physiological needs. It is assumed that the other half of BMR is usually diverted to maintenance.

For mammals, the major determinant of BMR is body mass, with which metabolic rate typically scales to the ¾ power (Kleiber's law). The daily energetic requirements of fasting adult primates have been estimated as $70W^{0.75}$kcal where W is body mass in kilograms. This makes the per-period cost of vital metabolism:

$(70/2)*36*W^{0.75} = 1260*W^{0.75}$

Pebsworth et al. [73] report daily travel of between 1.7 – 11.7km to and from foraging grounds, with monthly means of 3.6 - 6.5 km/day and with most ground covered in the summer when the number of daylight hours is greatest. Seasonality is not considered in this model and an average daily journey of five kilometres is assumed. The energetic cost of travel is estimated by the following equation from Taylor, Heglund & Maloiy [93]:

$VO_2 = 0.533*v*W^{-0.316} + 0.3*W^{-0.303}$

where VO_2 is mass-specific oxygen consumption (in $mlO_2s^{-1}kg^{-1}$), W is body mass (in kg) and v is speed (in ms^{-1}). Assuming a constant speed of $1ms^{-1}$ - giving a total daily travelling time of 5400s - and a caloric consumption of $5*10^{-3}$ for every millilitre of oxygen, we have:

$MSDEE = 5*10^{-3}*5400*(0.533*v*W^{-0.316} + 0.3*W^{-0.303}) = 14.391*W^{-0.316} + 8.1*W^{-0.303}$

where MSDEE is mass-specific daily energy expenditure (in $kcalkg^{-1}day^{-1}$). The per-period energetic cost of travel is therefore:

$$36*W*(14.391*W^{-0.316} +8.1*W^{-0.303}) \approx 518*W^{0.684} + 292*W^{0.697}$$

The total energetic cost of essential functions is given by:

$$\alpha(s) = 1260* W^{0.75} + 518*W^{0.684} + 292*W^{0.697}$$

4.3.3.3 Ageing rate

Following Shanley & Kirkwood [46], the change in age per period was modelled by the equation:

$$\zeta(k) = (0.5*BMR/k)^{\theta}$$

As stated above, it is assumed that an individual will generally invest half his/her BMR in somatic maintenance. Under this investment strategy, rates of biological and chronological ageing are equal, i.e. an organism will go from condition c to condition (c + 1) in one time period. If investment in maintenance is lower than half the BMR, the rate of biological ageing exceeds that of chronological ageing and for each decrement in k, the increase in ζ(k) becomes greater. If investment in maintenance is greater than half the BMR, the rate of chronological ageing exceeds that of biological ageing and for each increment in k, the reduction in ζ(k) becomes lower. Figure 4.2 shows the shape of the function ζ(k) and the effect of varying the ageing rate exponent (θ) within this function. θ was initially set to one, and the effect of changing this on optimal strategy was later explored via sensitivity analysis.

4.3.3.4 Foraging yield

Foraging yield is modelled as an increasing function of size, with an upper limit imposed by the environment. Although I am not aware of any study which has attempted to derive an equation for foraging yield in terms of size, it is known that larger individuals are more successful foragers, being able to displace smaller individuals from prime foraging sites, and it is reasonable to suppose the existence of some maximum rate of intake dependent upon the distribution of foraging sites within the home range. Intake is given by the following equation:

$$\xi(s) = 48{,}000*(1 - e^{-s/15})$$

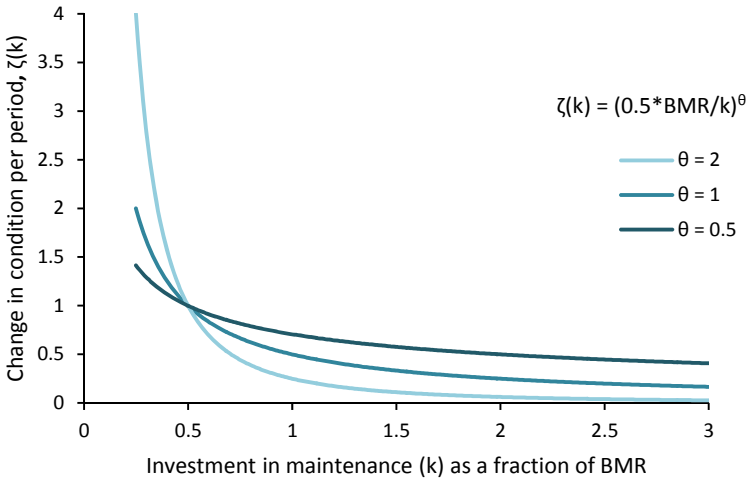

Figure 4.2: The rate of ageing as a function of investment in maintenance and dependent upon the choice of parameter θ.

which has the desired shape (figure 4.3) and which gives a range of foraging yields consistent with those reported in the literature. Leonard & Robertson (1997) calculate the daily caloric intake for a 13kg baboon to be 53.8kcal/kg, equivalent to 25178kcal/period. Rosetta, Lee & Garcia [85] recorded an intake of 832-834kcal/day, equivalent to 29952-30024kcal/period, in a group of eight females whose average size varied between 15.5 and 16.2kg. Muruthi, Altmann & Altmann [94] observed an intake of 690-826kcal/day, or 24840-29736kcal/period for wild-feeding females, although they gave no average size. These three sets of figures all fall in the range of foraging yields given by the above equation, and where given they agree well with the foraging yields predicted for individuals of the size reported. Roberts, Cole & Coward [84] give a higher figure of 1169-1375kcal/day, or 42084-49500kcal/period, for baboons kept in captivity and weighing on average 19-20kg. Whilst this is outside of the range of foraging yields used in the model, these individuals were provided with food ad libitum and their intake is unlikely to be representative of that of their wild-living counterparts.

In figure 4.3, the energy that must be diverted to essential functions is shown in blue, and the investment in maintenance for which biological and chronological ageing will be equivalent (BMR/2) is shown in red. The green bars show the remaining forging

yield and the yellow bars represent the energy which can be made available for reproduction and/or maintenance through weight loss.

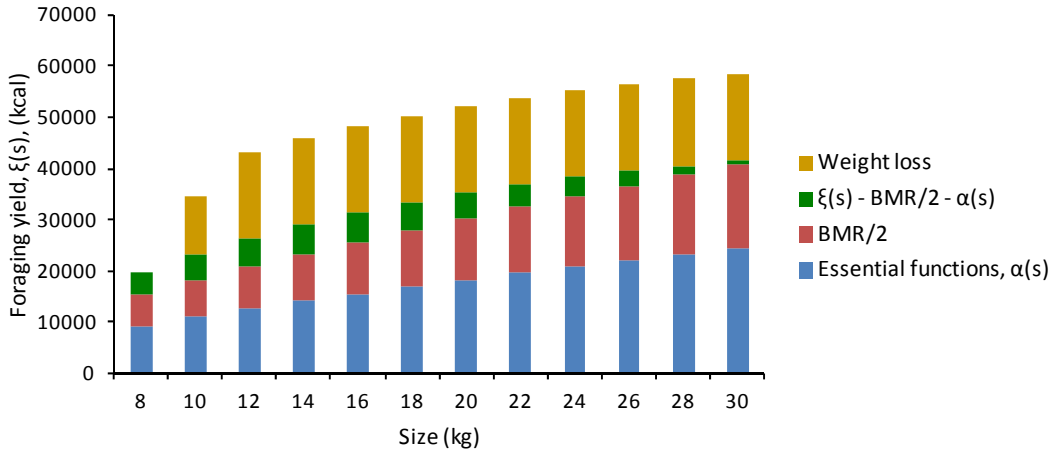

Figure 4.3: Energy budget as a function of size. The blue bars show the costs of essential functions: these costs must be met in order for the individual to survive the current period. The red bars show half the BMR: this is the amount that must be invested in maintenance for biological and chronological ageing rate to be equal. The remaining foraging yield is shown in green. The energy which can be made available through the mobilisation of nutrient stores is shown by the yellow bars.

4.3.3.5 Costs of reproduction

For a female, the costs of reproduction are principally those of provisioning an offspring during pregnancy and lactation. The maintenance of sexual swellings during oestrous and other mating behaviours will also entail some energetic outlay although it is supposed that this is comparatively low.

Lactation is the most energetically demanding time in a female's life and the reproductive investment for which there are most data available. Roberts, Cole and Coward [84] report an increase in voluntary energy intake two-to-ten weeks' post-partum of 206kcal/day in comparison to the non-reproductive weight-maintaining phase. This was accompanied by an average body nutrient mobilisation of 76kcal/day, giving a total cost of 282kcal/day and representing an increase in energetic expenditure of 27% over that of a non-reproductive female. Rosetta, Lee & Garcia [85] estimated the costs borne by eight females in provisioning their offspring at one month post-partum: the absolute costs ranged from 0-264kcal/day with a mean of

82kcal/day, whilst the percentage increase in energetic expenditure ranged from 0-30% with a mean of 13%. Muruthi, Altmann & Altmann [94] report an increase in energy intake of 393kcal/day, or 57%, in females who were either pregnant or lactating when compared to non-reproductive individuals.

Clearly there is a wide range in the estimated cost of lactation, both in terms of absolute expenditure and in the percentage increase over non-lactation. The differences between studies could be explained by a number of factors including the methods by which costs are estimated; differences in feeding regime (wild-feeding versus *ad libitum* provisioning); behavioural changes such as reduced activity that might offset the costs of lactation and; tailoring investment in offspring to the energetic status of the mother so that offspring may be reared more rapidly or be of better quality when conditions are favourable.

The model does not treat foraging as a behavioural decision and therefore energy intake can't be increased at will to meet the costs of pregnancy and lactation. Likewise, the investment schedule in offspring is fixed and may not vary with the size or condition of the mother. The cost of lactation was set at 250kcal/day, or 9000kcal/period. That this falls approximately in the centre of the range of absolute costs quoted above is clear. The cost of lactation as a percentage of the costs of a non-reproductive female can be estimated by the interval:

$$(9000/\xi(s), 9000/[\xi(s) - 9000])$$

where the first term corresponds to the case where foraging yield only meets the requirements of a non-reproductive individual and the second term where foraging yield includes the entire cost of lactation for the period. This interval is shown in figure 4.4 as a function of size and it can be seen that it falls roughly in the middle of the range of percentage increases in energetic expenditure quoted in the literature.

As far as I am aware, there has been no study which has estimated the cost of pregnancy in baboons. (The study by Muruthi, Altmann & Altmann mentioned above does not distinguish between pregnancy and lactation, but only between reproductive and non-reproductive individuals.) It is however understood that the energetic burden of supporting a foetus throughout gestation is significantly lower than that of suckling

an infant. For human females the average daily energetic cost of pregnancy is estimated at a little below half that of lactation[95]. As humans and baboons are closely related, it is assumed - in the absence of data from baboons - that the relative costs of pregnancy and lactation are much the same. The cost of provisioning a foetus was therefore set at 125kcal/day, or 4500kcal/period and the percentage increase in energetic expenditure compared to a non-reproductive individual (figure 4.4) is estimated by the interval:

$(4500/\xi(s), 4500/[\xi(s) - 4500])$

The total reproductive costs incurred by a female are given by the following vector:

$\rho(d) = (4500, 4500, 4500, 4500, 4500, 9000, 9000, 9000, 9000, 9000, 9000, 9000, 9000, 9000, 9000)$

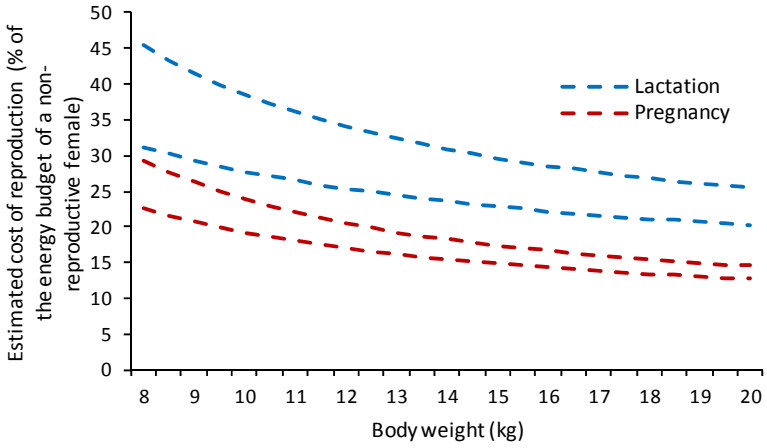

Figure 4.4: The relative cost of pregnancy and lactation as a function of size. The absolute cost is invariant but the percentage of the energy budget of a non-reproductive female of the same size changes and should fall within the intervals shown. These are $(4500/\xi(s), 4500/[\xi(s) - 4500])$ and $(9000/\xi(s), 9000/[\xi(s) - 9000])$ for pregnancy and lactation respectively (see text for justification).

For males, the costs of reproductive behaviour are much harder to estimate and I am again aware of no study which has attempted rigorously so to do. However, there is evidence to suggest that the energetic costs of establishing a consortship with a female are significant[86]. The proportion of foraging yield lost in attempting to

consort (η) was set to 0.5. Although it is difficult to justify the precise value used, this should not be taken as invalidating the model as the primary model outcome - the degree of sexual dimorphism - did not alter significantly with changes to η (see results from sensitivity analysis in section 4.4.7). There will also be direct energetic costs associated with aggressive encounters with other males which, for simplicity and due to lack of quantitative data, were not considered.

4.3.3.6 Probability of offspring loss

Both the overall rate of miscarriage and its relative likelihood at various stages of gestation vary significantly between baboon populations. Overall rates have been recorded at 4.82-20%[83]. Some authors have found that the probability of foetal loss is higher during the latter part of gestation and others that it is reasonably constant throughout. It is likely that all figures reported in the literature slightly underestimate the rate of miscarriage as it is not possible to determine pregnancy, and therefore foetal loss, before three or four weeks' gestation.

Figures in the model are taken from Beehner *et al*. [81]; these data were chosen because the authors were able to validate their visual method of pregnancy detection using faecal hormone profiles. The probability of foetal loss was set to 0.02 in periods zero-to-four of pregnancy and 0.04 in period five making the overall proportion of pregnancies which do not result in a live birth:

$1 - (0.98^4)*0.96 = 0.114$

Cheney et al. [76] report that approximately one fifth of infants do not survive their first year, further stating that of the infants known or suspected of having been killed by adult males, nearly two thirds were under three months. If o is the probability of infant death in each of the first three periods of lactation (d = 5, 6, 7) and υ that in each of the last seven (d = 8, …, 14), then to a good approximation we have:

$(1 - o)^3*(1 - \upsilon)^7 = 4/5$

$[1 - (1 - o)^3]/[1 - (1 - o)^3*(1 - \upsilon)^7] = 2/3$

Solving these equations gives $o \approx 0.04$ and $\upsilon \approx 0.01$. For simplicity it is assumed that the probability of infant mortality does not vary with the condition of the mother. So we have:

$\sigma(d) = (0.02, 0.02, 0.02, 0.02, 0.04, 0.04, 0.04, 0.04, 0.01, 0.01, 0.01, 0.01, 0.01, 0.01, 0.01)$

The probability of juvenile mortality is also taken from Cheney et al.[76], who report an average annual mortality rate for both sexes of 0.04. The probability of juvenile survival over the four-year period from gaining independence to reaching maturity is therefore given by:

$\phi = (1 - 0.04)^4 = 0.849$

4.3.3.7 Conversion of energy to biomass

Following Roberts, Cole & Coward [84], an energy density of 7kcal/g or 7000kcal/kg is assumed. (The energy density of fat is 9kcal/g and that of proteins or carbohydrates is 4kcal/g.) The efficiency with which energy is converted to/from biomass is taken to be 80%. This is based on research referenced by Roberts, Cole & Coward giving the efficiency with which body stores are converted to milk as between 80-90% for a woman. In the absence of any data from baboons, it is assumed that humans and baboons are sufficiently closely related for this to be similar. (At least it should be rather closer to the value for women than that of 58% quoted for rats.) Change in size as a function of energy invested in growth is therefore given by:

$\gamma(j) = j*0.8/7000 = j/8750$ if j is positive
$\gamma(j) = j*0.8/7000 = j/5600$ if j is negative

4.3.3.8 Male mating success

A male's mating success (χ) is a function of his state, the number of days upon which he chooses to compete for access to reproductive females and, the distribution of adult females (Ω_f) and males (Ω_m) within the population. For each four-day period upon which consortship is attempted, the probability of successfully impregnating a female is directly proportional to the expected number of females looking to conceive within those four days and the relative fighting ability of the focal male in comparison

to other competitor males. It is assumed that the timing of the reproductive cycles of any two females are independent so that the expected number of females looking to conceive within any four day interval is the number for the whole period divided by nine. Similarly, the days upon which any two males choose to compete for mating access are independent so that the expected distribution of competing males is the same for all four-day intervals within a period. Males are not able to judge the quality of females. High-ranking males are therefore not able preferentially to consort with those most likely to raise an offspring to independence nor are low-ranking males able to select those of poorer quality for whom competition might be lower. Conception is equally probable on each of the four days of peak oestrous.

Fighting ability (π) is given by the following equation in s and c:

$$\pi = 1/\{[1 + e^{0.5*(25 - s)}][1 + e^{0.6*(c - 150)}]\}$$

Although quantitative data on how π varies with state are lacking, the relationship given by this equation fits qualitative reports well. The effect of size on π is given by the expression in the first square bracket and shown in figure 4.5a (where condition is kept constant at 50τ). Within this expression, 0.5 describes the rate of increase in π with size and 25 is the size at which the increase in π is greatest. If the former was larger, the increase in π would occur within a narrower window so that the curve in figure 3.5a appears 'tighter'. If the latter was larger, the curve in figure 4.5a would be shifted to the right. Smaller males are effectively excluded from mating opportunities because their π is close to zero. There is a sharp increase in π as size increases over 20kg followed by a slight tailoring off towards the maximum size of 30kg which can be interpreted in two ways. Firstly, the proportional increase in size becomes lower with each absolute increment and the increase in π may be correspondingly lower. Secondly, if we make the assumption that any increase in size is primarily muscle mass, then any benefit from increased strength may begin to be offset by limitations on what the skeleton can reasonably support.

The effect of condition on π is given by the expression in the second square bracket and shown in figure 4.5b (where size is kept constant at 30kg). Within this expression, 0.6 describes the rate of decrease in π with condition and 150 is the condition at which the rate of decrease in π is greatest. If the former was larger the curve in figure 4.5b

would appear tighter and if the latter was larger it would be shifted to the right. During early adulthood, slight deteriorations in condition have little effect upon overall physiology and π is left largely unchanged. During mid adulthood the decrement in π becomes more pronounced with each successive increment in condition before gradually levelling off again in old age.

The combined effects of changing size and condition on π are shown in figure 4.5 c & d. Here π is plotted against time in the cases where size increases by either two, four or eight kilograms per period up to the maximum of thirty (this size being maintained thereafter) and condition increases by one (figure 4.5c) or two (figure 4.5d) τ per period. The benefits of rapid growth can clearly be seen in both graphs: by reaching the maximum size before the negative effects of age are felt, a higher peak fighting ability can be attained. This is most apparent when ageing is more rapid (figure 4.5d). The steep inclines and declines in fighting ability over time mirror reports in the literature for rate of siring offspring, as would be expected.

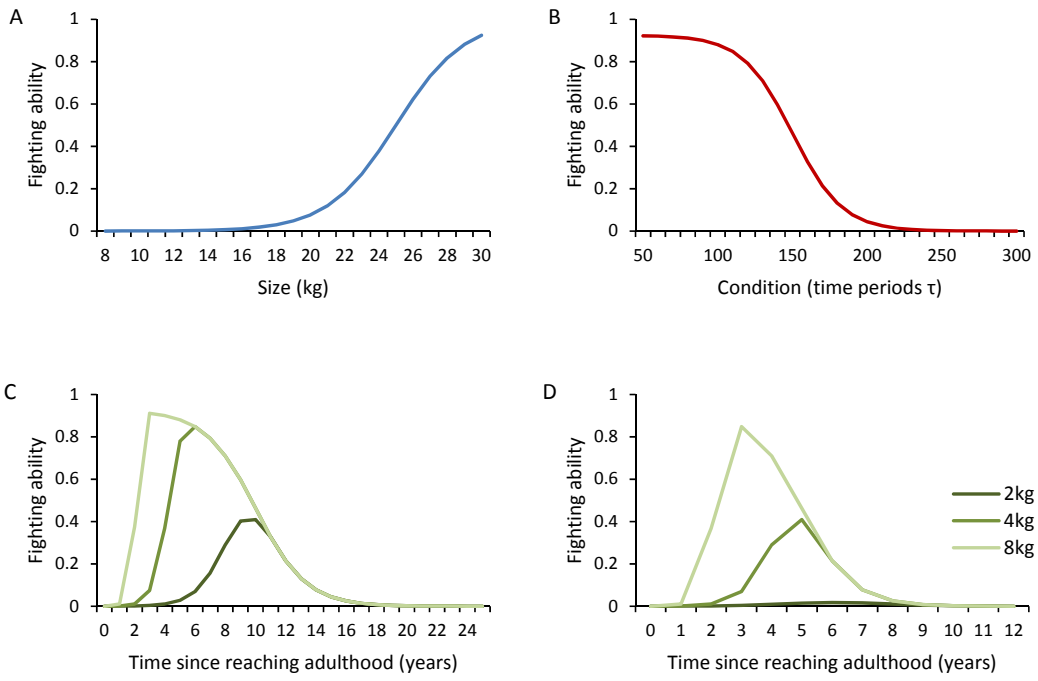

Figure 4.5: A: Variation in fighting ability as a function of size (with condition held constant at 50t). B: Variation in fighting ability as a function of condition (with size held constant at 30kg). C & D: The change in fighting ability over time when size increases by two, four or eight kilograms per period up to a maximum of thirty and condition increases by one (C) or two (D) τ per period.

4.3.3.9 Dispersal mortality

Dispersal mortality is the mortality risk experienced by males when leaving the natal group and seeking a new one. In the absence of any estimate of what this might be, it was initially excluded from the model; effectively it was set to zero. The effect of including this was later considered and is discussed in section 4.4.7.

All parameters used in the model along with their units and the literature upon which they are based are shown in table 4.1.

Parameter	Unit	Value	Reference
energy density	kcal	7000	Roberts, Cole & Coward (1985)
conversion efficiency	none	0.8	
initial mortality rate	$period^{-1}$	0.00285 (female), 0.00371 (male)	Bronikowski et al. (2011)
rate of ageing	none	0.123	
juvenile survival	$4yr^{-1}$	0.849	Cheney et al. (2003)
probability of loss of a dependent offspring	$period^{-1}$	(0.02, 0.02, 0.02, 0.02, 0.04, 0.04, 0.04, 0.04, 0.01, 0.01, 0.01, 0.01, 0.01, 0.01, 0.01)	Beehner et al. (2006); Cheney et al. (2003)
energetic requirements of dependent offspring	kcal	(4500, 4500, 4500, 4500, 4500, 9000, 9000, 9000, 9000, 9000, 9000, 9000, 9000, 9000, 9000)	Muruthi, Altmann & Altmann (1991); Roberts, Cole & Coward (1985); Rosetta, Lee & Garcia (2011); Dufour & Sauther (2002)
intake multiplicand	kcal	48000	Muruthi, Altmann & Altmann (1991); Roberts, Cole & Coward (1985); Rosetta, Lee & Garcia (2011); Leonard & Robertson (1997)
intake exponent	none	-15	
BMR multiplicand	kcal	1260	Committee on Animal Nutrition et al. (2001)
BMR exponent	none	0.75	
travel multiplicand1	kcal	518	Pebsworth et al. (2012); Taylor, Heglund & Maloiy (1982)
travel exponent1	none	0.684	
travel multiplicand2	kcal	292	
travel exponent2	none	0.697	
consort cost	none	0.5	
dispersal mortality	none	0	
fighting ability 1	none	0.5	
fighting ability 2	none	25	
fighting ability 3	none	0.6	
fighting ability 4	none	15	
ageing rate multiplicand	period	0.5	Shanley & Kirkwood (2000)
ageing rate exponent	none	1	

Table 4.1: Parameters used in the model and, where applicable, the publications from which they are taken.

4.3.4 Finding the optimal strategy

Backward and forward iterations for females and males were coded in Python according to the specification above. The first simulation run was a female backward iteration under the assumption that all descendants were female - or at least that the reproductive values of male and female offspring were equivalent. It was also assumed, in this and all later female simulations, that there were always at least as many males competing for mates in any period as there are females seeking to conceive so that a female can always become pregnant if she wishes to. Later simulations of the male population confirmed that this assumption held. As the backward iteration was run, the difference between the strategies followed by an individual in state X at one time point and the next was calculated for all X. When the average of these differences was consistently (over 50 consecutive periods) less than 0.1% of the available energy budget (equivalent to approximately 20kcal), the simulation was terminated. This was achieved after 180 time points.

A female forward iteration was then run starting from an arbitrarily chosen population of ten newly-matured adult females and following all juvenile and adult female descendants. All offspring now had a 50% chance of being female and a 50% chance of being male. All adult females followed the stable strategy calculated from the backward iteration; juveniles were not able to make any energy allocation decisions but all experienced an increase in condition of one τ per period and a linear increase in size up to 8.5kg at the start of adulthood. The number of males reaching maturity in each period was recorded for use in the male backward iteration; mature male individuals were then deleted from the simulation. The forward iteration was run until the percentage difference in the age distribution from one period to the next - that is $\Sigma_i (p_i - q_i)$ where p_i and q_i are the percentage of the population in age class i at times (t + 1) and t respectively - was consistently less than 1%. This was realised after 2000 periods. The number of females seeking mates and the average probability that these females would go on to raise any offspring conceived to independence (which depends upon the distribution of their states) were calculated at this final time point for use in the male simulations. The stable population growth rate ($\omega_{f.j}$) was defined as the total

number of juveniles and adult females at the final time point divided by the total at the penultimate time point.

The population size at the final time point was over 100,000, exceeding that found within most baboon troops by a factor of at least 500. It was necessary to assume that the composition of troops within a wider population was near homogeneous and that slight stochastic variations in troop structure did not significantly affect the optimal strategy. To treat differences in troop structure as a variable which may affect allocation strategy was too computationally demanding for the current work.

Data from the forward iteration were used to check that the boundary conditions used did not artificially constrain behaviour, i.e. relaxing them would not produce a change in the optimal strategy. In this, as in all forward iterations subsequently run, the boundary conditions for maximum size, minimum size, maximum condition and maximum weight loss per period were not hit by any of the simulated individuals.

Simulations were then run to find the stable strategy for males given that all females follow the strategy determined from the above. As stated previously, male strategy will depend on how other males behave as well as how females behave. Initially, the strategy followed by all non-focal males was chosen arbitrarily: they all attempted to consort for 18 days per period and made no investment in growth so that the remainder of the available energy budget was invested in maintenance. The stable distribution of males following such a strategy was computed by a forward iteration starting from an arbitrarily chosen starting population, specifically the empty set. At the end of the first time point, ς newly-matured males were added to the population where ς was non-zero but otherwise arbitrary; at all subsequent time points t, the states of all existing males were updated and $\varsigma^*(\omega_{f.j})^t$ newly-matured males added to the population. At the final time point, the number of females seeking mates was given by $\kappa^*(\omega_{f.j})^t$ where ς:κ is the ratio of newly-matured males to mating females calculated from the female forward iteration above. The stable distribution and strategy of non-focal males and the number of mating females were then used as inputs to the backward iteration for a focal male.

The male backward iteration was run until the mean difference in strategies from one time point to the next was consistently less than 0.1% of the available energy budget.

As for females, this was achieved after 180 time points. It was assumed that each offspring that a male sired had a 50% chance of being male and a 50% chance of being female. The expected reproductive value of a female offspring conceived at time t was taken from the female simulations described above; the expected reproductive value of a male offspring was computed within the backward iteration. Having obtained a stable strategy for the focal male, this was then compared to that of non-focal males. If the mean difference between their strategies - taken over all possible states in which a male may exist - was less than 0.1% of the available energy budget, then the stable strategy was deemed to have been reached. If this condition was not met, then the non-focal strategy was updated to that of the focal and another cycle of male simulations was carried out (i.e. another forward iteration for non-focal males followed by a backward iteration for focal males). Three cycles were required to converge upon the stable strategy.

With this knowledge about optimal male strategy, consideration returned again to that of females. Calculation of female optimal strategy relied upon the predicted behaviour of descendants and it had originally been assumed that these were all females. In particular, optimal reproductive scheduling was tailored to a population growth rate calculated under the assumption that all offspring were female. If population growth rate is positive then, all else being equal, it pays to have offspring sooner; if population growth rate is negative then it pays to have them later. If the growth rate is zero then, all else being equal there is no advantage or disadvantage to a parent in changing the average age at which offspring are born. It was known from the male and female forward iterations that the population growth rate was positive and that males were on average younger when they sired offspring than females were when they gave birth. Therefore, the reproductive value of males at birth was slightly higher than that of females and the expected reproductive value of offspring had been underestimated in the original female simulation. This has the potential to change optimal reproductive scheduling and so the female backward iteration was run again, assuming now that all offspring had an equal chance of being male or female. Male simulations were then also rerun given the updated female strategy and taking the focal male's strategy from the previous cycle as the initial strategy for non-focal males. (At this point it was assumed that the only adjustment a female can make to the higher

reproductive value of sons was in her energy allocation decisions. The implications that this has for optimal sex ratio are considered in section 4.4.7.)

This second cycle of female-and-male simulations only partially corrects for the difference in reproductive value at birth between the sexes. This is because the second female backward iteration uses figures for male reproductive value calculated under the assumption that a male's daughters will only have female descendants. Only the error due to in a female's first generation of descendants has therefore been redressed at this stage. Repeated cycles of female-and-male simulations correct for the error in subsequent generations.

In practice the effect on the current generation of differences in reproductive value between male and female descendants several generations away becomes so insignificant that it has no bearing upon strategy. Only two cycles of female-and-male simulations were required before the mean difference between female reproductive strategies from the current and previous cycles was less than 0.1% and it was deemed that the stable strategy for both sexes had been reached. A plan of the simulations run to find the optimal strategies, is shown in figure 4.6 below.

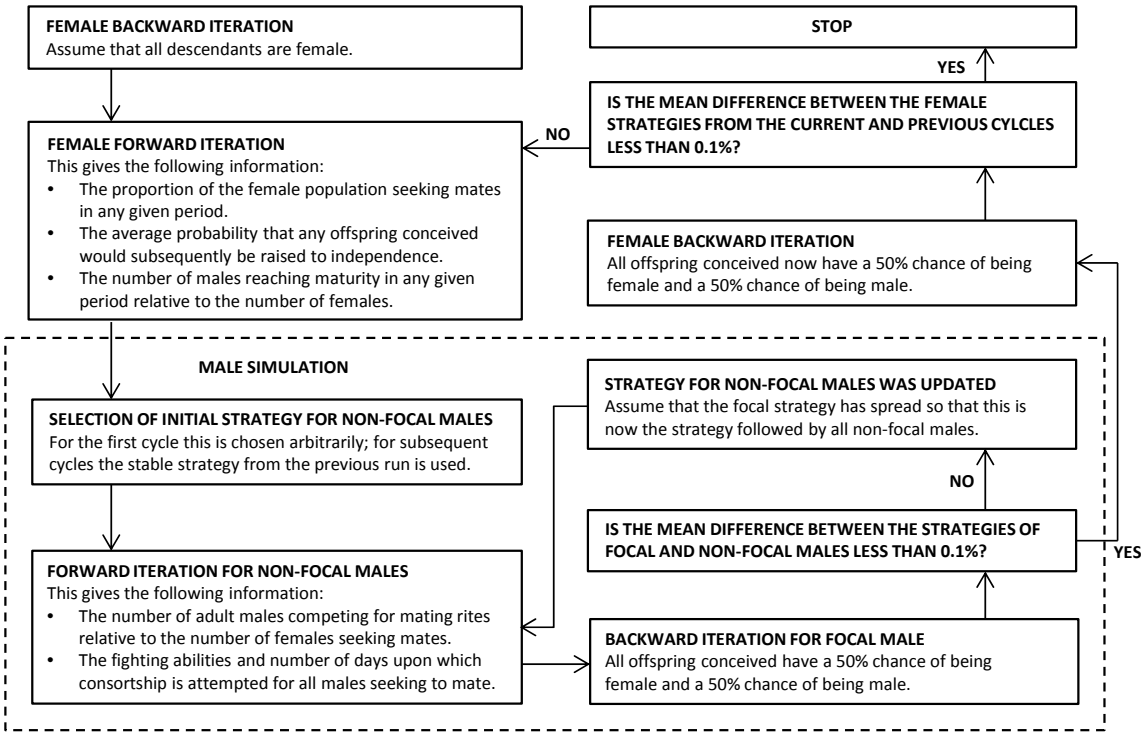

Figure 4.6: Schematic representation of the simulations run to find the optimal strategy.

Having found the optimal strategy, further forward iterations were run to determine whether this strategy predicted the individual- and population-level behaviour that is seen in real baboons. The model was then used to address a number of related issues regarding dimorphism in this species. Firstly, can the difference in lifespan between the sexes be partitioned into that resulting from different IMRs and that resulting from different RoAs? Secondly, how does the relative fitness of suboptimal strategies compare to the optimal and what can be inferred from this about the force of selection in favour of the optimum? Thirdly, what implication does the difference in the average age at which males and females become parents have for the optimal sex ratio? And fourthly, how robust is the behaviour predicted by the model to changes in parameter values? The first three of these are described in the results and no further details are given here. This last point is addressed by means of sensitivity analysis. Some justification is needed of the quantities varied within this analysis and this is given in the next section.

4.3.5 Sensitivity analysis

A number of systematic perturbations to model parameters were defined. Simulations to find the optimal strategy, as described in section 4.3.4, were repeated under each perturbation followed by forward simulations following cohorts of 100 females and 100 males. The average lifespan, growth and reproductive success of these cohorts were then compared to those from cohorts under the original model parameterisation. In most cases eight perturbations were considered corresponding to increases in the parameters of -30, -22.5, -15, -7.5, 7.5, 15, 22.5 and 30%. Only the latter four were considered for dispersal mortality as this can't fall below its initial value of zero. Only seven were considered for conversion efficiency because a 30% increase would give a value greater than one.

Perturbations to the available energy budget as a whole (i.e. the foraging yield plus the energy available through weight loss minus the cost of essential functions) were considered instead of perturbations to each parameter which affects it in turn. So, separate sensitivity analysis wasn't carried out on those parameters that determine foraging yield, BMR, travel costs and the proportion of the BMR that must be invested in maintenance for biological and chronological ageing rates to be identical. An

increase or decrease in any of these produces, with one exception, only minor differences to the shape of the function of energy budget against size (see figure 4.7). Therefore, changes in these parameters are to a good approximation reducible to multiplication of the available energy budget by a fixed term. The one exception was BMR exponent; a significant increase, but not decrease, in this will change the shape of the function of available energy budget as shown in figure 4.6A. However, it can be said with some surety that the values for which this becomes a problem do not arise in nature. Considerable work has been done on how BMR scales with body mass in mammals and the three-quarter power scaling coefficient is widely supported. Where its use has been criticised, it is generally in favour of the lower two-thirds scaling power.

Ideally, a combinatorial approach to sensitivity analysis would have been employed in which systematic variations are applied to multiple parameters. However, given the numbers of parameters and perturbations here considered, this would have produced in excess of 1.9×10^{12} combinations. Even perturbing all possible pair of parameters simultaneously would give 5308 combinations which was still too computationally demanding for the present work. Only alterations to a single parameter at a time were therefore considered.

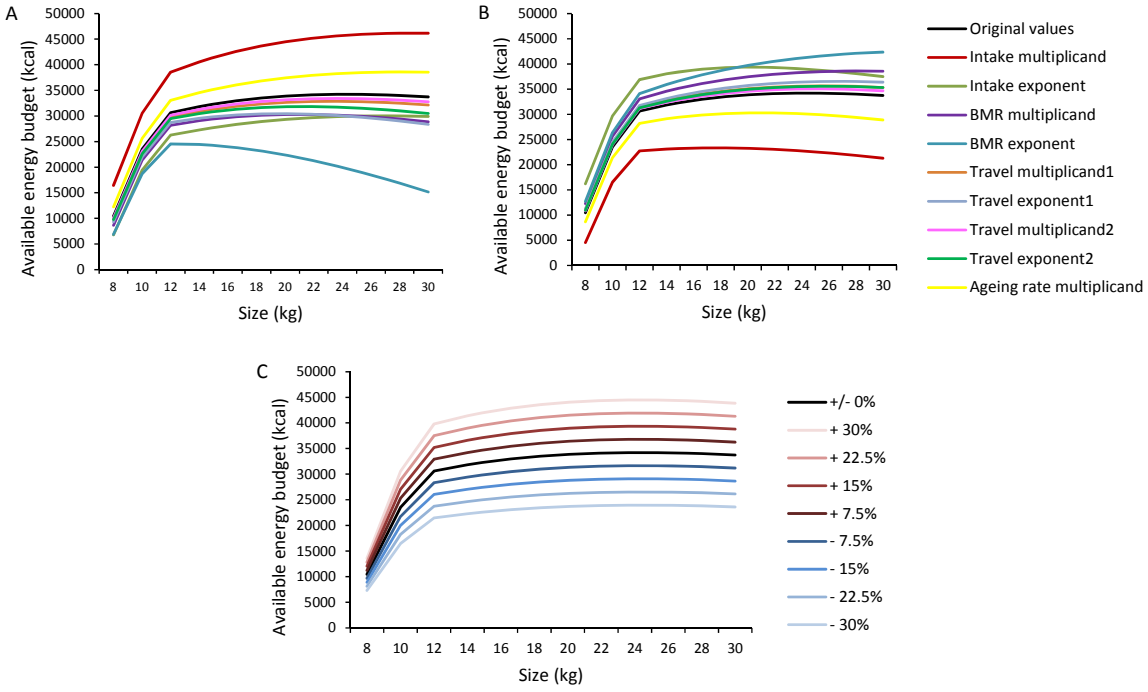

Figure 4.7: Available energy budget as a function of size. A & B: The function used in the model is shown along with those which would result from a 30% increase (A) or 30% decrease (B) in each of the parameters indicated. C: The functions for available energy intake used in sensitivity analysis. These result from multiplication of the original values by the percentages indicated.

4.4 Results

4.4.1 The backward iteration

Output from the backward iteration gives optimal strategy as a function of state. For females this is summarised in figure 4.8 and for males in figure 4.9.

In almost every state in which a female may find herself, she should invest in reproduction (figure 4.8A). The exceptions to this are regions of the state space where s = 8.5kg, d = 0 and c is low ($c \leq 78\tau$) or where c is very high ($c \geq 273\tau$). The former condition only arises upon first attaining adulthood; size then increases during early adulthood (see figure 4.11A) and only falls to 8.5kg again in some individuals late in life when c is much greater than 78τ. The latter describes regions of the state space that are rarely entered: less than 1.5% of the adult female population will attain a condition of 273τ or more (see next section). Within a simulated population, there are therefore only a small minority of females who will not invest in reproduction in any given period.

The optimal change in condition per period varies markedly with size and reproductive status but not with condition itself. Results in figure 4.8B, for which condition is held constant at 60τ, resemble those seen with a higher or lower condition. Change in condition decreases with size and is higher during lactation than pregnancy. Moreover, the difference between pregnancy and lactation is seen most clearly in smaller individuals. This reflects the fact that lactation is more energetically demanding than pregnancy and investment in some other function must be reduced to 'foot the bill'. Without substantial nutrient stores to draw upon, smaller individuals will struggle more than larger ones to meet the costs of reproduction and can only raise an offspring to independence at the expense of rapid ageing.

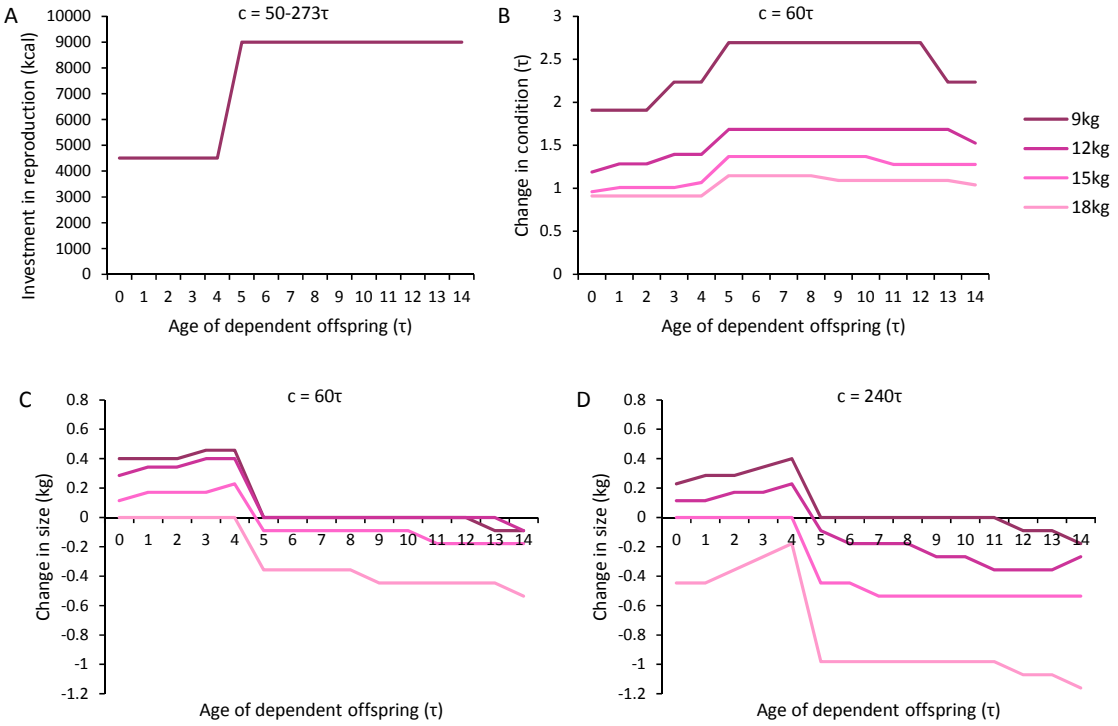

Figure 4.8: The optimal investment in reproduction (A), change in condition (B) and change in size (C & D) for females per period, as a function of state. Note that in A, the functions for the four sizes are identical.

When condition is low, weight is put on during pregnancy at all but the largest sizes: at 18kg weight is maintained and at 18.5kg and above it is lost. Heavier individuals will then lose weight throughout lactation whilst lighter individuals will maintain weight for the majority of lactation and only experience a slight loss at the end (figure 4.8C). As condition is increased, the optimal weight gain during pregnancy is steadily reduced and the optimal weight loss during lactation increased. For an old individual (figure 4.8D), the graphs for optimal change in size have shifted downwards considerably in comparison to those for a young individual. These results suggest cycles of weight gain during pregnancy followed by weight loss during lactation and a general reduction in size with age. This was later confirmed by a forward simulation following a cohort (section 4.4.3).

For males, reproduction should usually only be attempted if size exceeds 25kg (figure 4.9A). Exceptions to this are seen at the very oldest ages; if $c \geq 297$ for example, individuals will attempt to consort when $s < 20$kg. Although the probability of success in these states is tiny, the weighting of potential future reproductive gains relative to

current ones is so small it is still best to try and mate in the current period. These exceptions are essentially academic: they represent regions of the state space which are not entered in a forward simulation (see section 4.4.2). The optimal number of days spent attempting to mate increases with both size and condition; for each successive increment in size, the lowest condition at which a male should attempt to mate is reduced and the number of days he should attempt to mate for a given condition is increased. Only at the largest sizes of 28kg or more does a male attempt reproduction in all possible conditions.

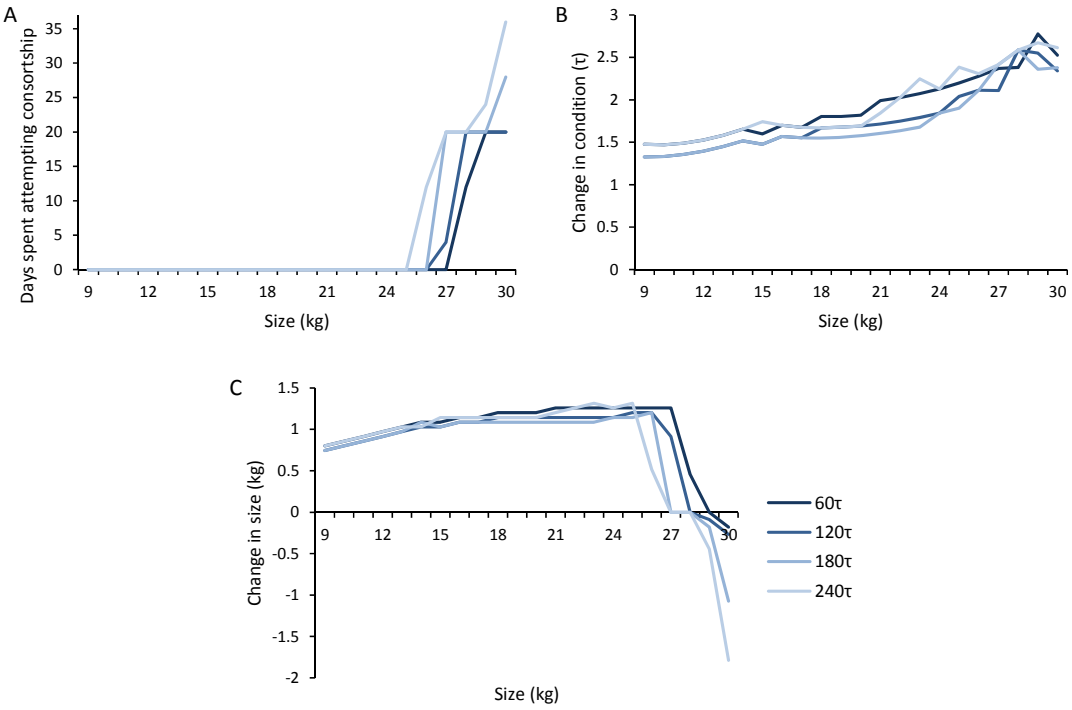

Figure 4.9: The optimal number of days spent attempting to maintain a consortship (A), change in condition (B) and change in size (C) for males per period, as a function of state.

In contrast to what is seen in females, the optimal change in condition increases with increasing size (figure 4.9B). This reflects the fact that a male's investment in reproduction increases with size and the cost of this is met at the expense of rapid ageing. Condition itself has little effect on the optimal change in condition.

Optimal change in size is an increasing function of size, which is largely independent of condition, until approximately 25kg (figure 4.9C). At larger sizes, change in size is

condition dependent with a higher size being maintained at lower conditions: when c = 240τ, the optimal change in size decreases with each increment in size above 25kg, whereas at c = 60τ, this decrease with size is only seen at sizes greater than 27kg. This makes sense if we consider that condition increases more rapidly in larger individuals (figure 4.9B) and a given increment in condition will reduce fighting ability to a greater extent in an old individual than a young one. (Recall that the dependency of fighting ability on condition is described by the sigmoidal curve shown in figure 4.5B.) The optimal change in size is non-positive for all states at 29kg and negative for all states at 30kg, from which it can be inferred that the boundary for maximum size should never be hit during forward simulations.

4.4.2 The stable population distribution

Beginning with an arbitrarily chosen starting population, a forward iteration was run until a stable population distribution was reached. This was achieved after 2000 periods. The equilibrium age, condition and size distributions were computed for both sexes along with the distribution of female reproductive statuses and the stable population growth rate.

The stable size distribution for mature individuals is shown in figure 4.10A and it can be seen that simulated individuals exhibit the same degree of size dimorphism as real baboons. Mature females ranged in size from 8.5 to 16.5kg, with a mean of 12.7kg (standard error 6.06×10^{-3}); males ranged from 8.5 to 28.5kg with a mean of 23.0kg (standard error 2.91×10^{-2}). Size distributions for both sexes show sharp peaks around the mean. If we restrict consideration to those who have reached full adult status i.e. females who have birthed at least one offspring and males who have almost reached their maximum size, then the average sizes for females and males are 12.9kg and 26.8kg respectively. For comparison, Plavcan & Schaik [62] reference eleven studies in four species of baboon which give adult mean sizes of 9.40-16.85kg (average 13.60kg) for females and 17.24-29.20kg (average 23.50kg) for males. The ratio of mean female size to mean male size, for all simulated individuals of five years or over, was 0.55. If we again restrict consideration to adult individuals this ratio becomes 0.48. Plavcan & Schaik give ratios for adults of between 0.44 and 0.83 with an average of 0.57.

The numbers of simulated adult females with dependent offspring of age d is a decreasing function in d. Within a given period, 39.6% of females will be pregnant and 58.6% lactating, leaving just 1.75% who are non-reproductive (figure 4.10B).

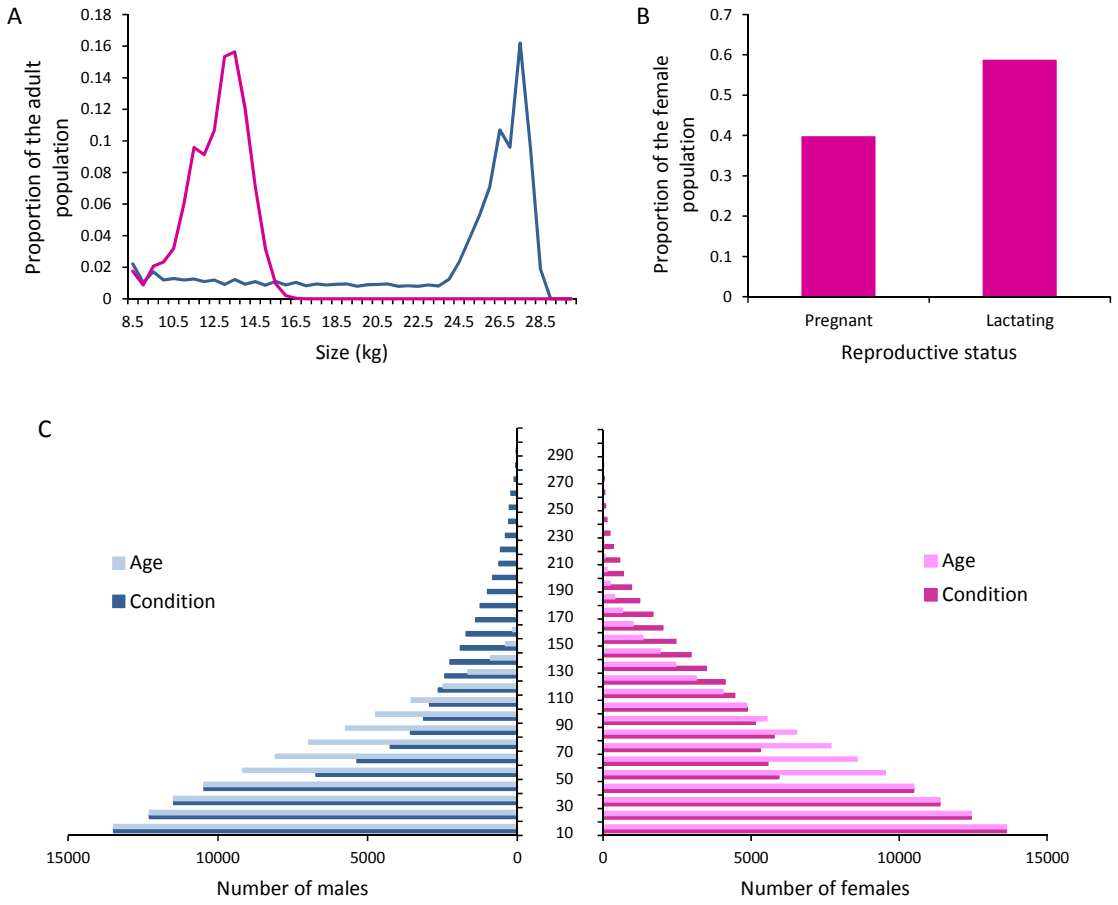

Figure 4.10: The stable adult size distribution (A); proportions of pregnant and lactating females (B) and; distributions of condition and chronological age for a population following the optimal strategy (C). Data for males is shown in blue and for females in pink.

The population pyramid in figure 3.10C shows both the distribution of chronological ages and of biological conditions at equilibrium. The age distribution is typical of a growing population, with a broad base - indicating a relatively high birth rate - and concave sides. Growth rate was calculated as $1.0046 period^{-1}$ or $1.046 year^{-1}$. There are a greater number of females than males in all age groups of five years or over and the oldest female (240τ) is considerably older than the oldest male (180τ). Both sexes tend to invest less than half their BMR in maintenance, so that distributions of condition are shifted towards the higher end of the scale. Condition increases more rapidly with age

in males than in females with the result that, although the average age for an adult male (76τ) is lower than for an adult female (87τ), the average condition of the two is essentially the same (104τ and 105τ respectively). If condition is plotted as a function of age the graphs produced are to a good approximation linear (data not shown).

4.4.3 Characteristics of a cohort

Cohorts of 100 individuals of each sex were followed from five years until death and their behaviour characterised in terms of size, longevity and reproductive success (figure 4.11 & table 4.2). T-tests were conducted to determine whether significant differences existed between males and females in maximum size, age when offspring are born and lifespan. All three measures were significantly different at the 0.0001 level, when applying a Bonferroni correction for multiple comparisons.

As mentioned in the previous section, in terms of both absolute size attained and degree of size dimorphism, simulated individuals match what is reported in the literature for real baboons (figure 4.11A). The model also reproduces the changes in size with age seen in real baboons. Simulated males undergo a rapid growth spurt between five and seven years to attain a maximum size of about 28.5kg. This size is maintained until about nine years after which there is a steady decline, with some of the oldest baboons weighing less than 25kg. Simulated females also experience growth in early adulthood; however this is much shallower and peaks slightly later at about 8.5 years. Average size is then fairly constant (around 14kg) until around 12 years after which there is again a steady decline. I am not aware of any published quantitative data for the reduction in size against age however the relative declines in size predicted by the model are very similar to the declines in BMI reported by Altmann *et al*. [70].

The reduction in size with age is a genuine behavioural response and not the consequence of physiological changes that occur with age. This cannot, for example, be attributed to deterioration in foraging efficiency or reduced muscle mass because - although these are seen in real baboons - they are beyond the scope of the model here presented. The explanation for this reduction in size is slightly different for males and females. For females, since the probability of survival to produce offspring at some future time decreases with condition, the value of nutrient stores - which could be

used to provision such future offspring - is reduced relative to the value of investment in maintenance, assuming that the latter improves survival prospects for at least long enough to raise any current offspring. For males, any reproductive success now starts to depend more heavily upon condition than at younger ages. Fighting ability changes very little as condition is increased from 50τ to 100τ but falls off steeply with further increases to condition. A young male will therefore prioritise investment in growth in order to maximise his fighting ability whilst an older male will do better to accept some reduction in size so that more can be invested in maintenance.

Females underwent cycles of weight gain during pregnancy and loss during lactation which are masked by taking the average size but are clearly seen in the size trajectory of a single female (figure 4.11B). As is the case for real baboons, weight generally rises to a peak at birth (indicated by diamonds) and falls to a local minimum when the offspring is weaned (indicated by crosses). In early adulthood, when there is a general trend towards increasing size, the gains during pregnancy exceed the losses during lactation. At older ages, this is reversed and the losses during lactation exceed the gains during pregnancy.

The model predicts a clear difference in survivorship between the sexes with females being significantly longer lived than males (figure 4.11C). The longest-living female in the cohort died at age 21.2 years whilst the longest-living male died at only 16.2 years. The life expectancy for a female at five years is 12.88 years compared with 10.42 years for a male. The female figure is very close to the 12.1 years reported for yellow baboons at Amboseli in Kenya but rather lower than the 14.7 years seen in olive baboons at Gombe in Tanzania[96]. Analogous results for male baboons are not available.

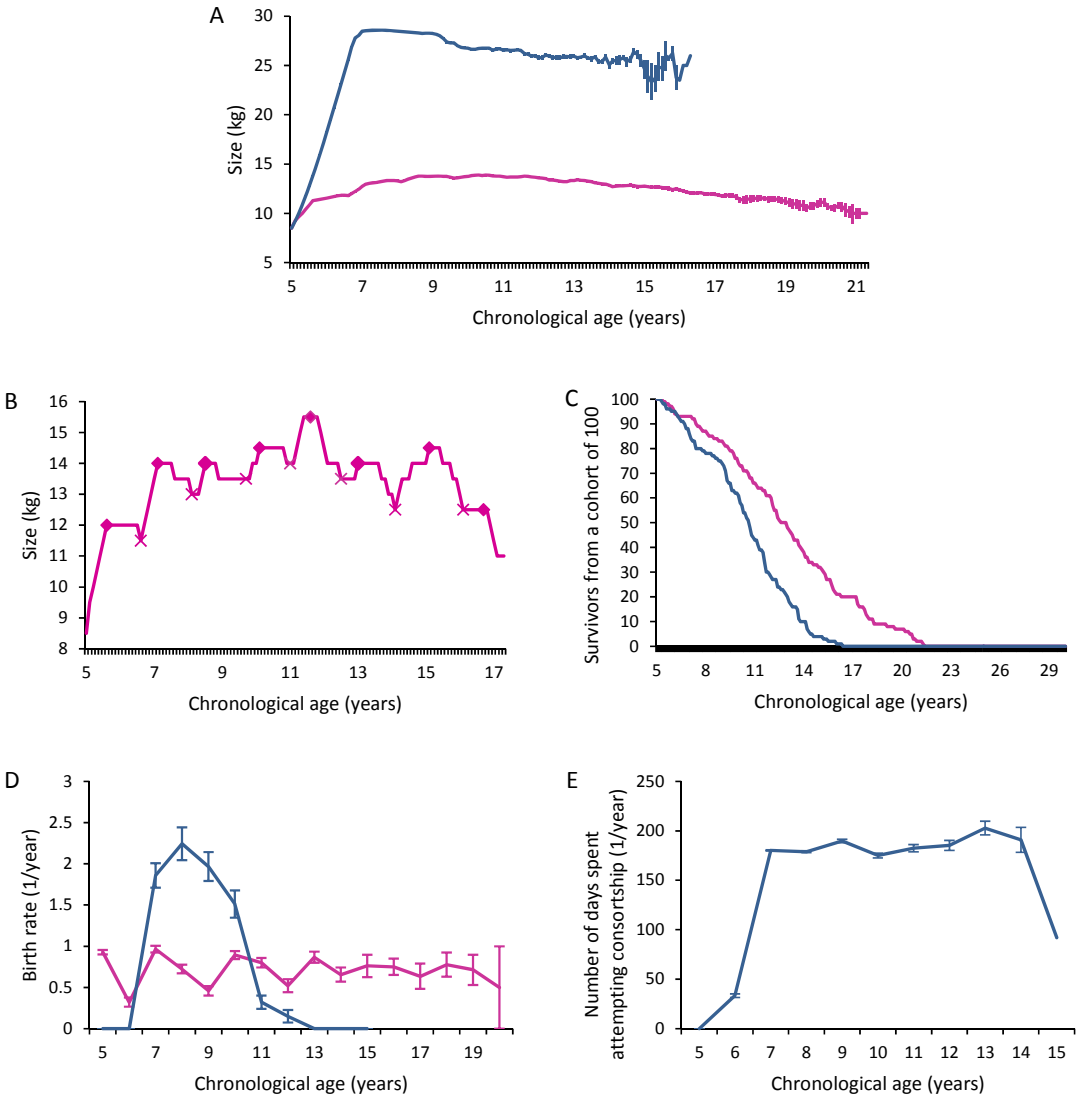

Figure 4.11: The size (A), survivorship (C), birth rate (D) and, for males only, number of days on which mating is attempted (E) as a function of chronological age. B shows the size trajectory for a single female, showing times at which she gives birth (diamonds) and at which she successfully weans an offspring (crosses). Data for males is shown in blue and for females in pink. All error bars are standard errors.

	Female	Male
Lifespan (years)	12.88 (0.4081)	10.42 (0.2672)
Rate of ageing	0.16	0.27
MRDT (years)	4.18	2.55
Maximum size (kg)	14.38 (0.1157)	27.42 (0.3861)
Offspring born	5.58 (0.2811)	5.31 (0.4123)
Offspring weaned	3.93 (0.2405)	3.83 (0.2871)
Age at first birth (years)	5.56 (0.0191)	7.65 (0.0406)
IBI (years)	1.42 (0.0181)	0.39 (0.019)

Table 4.2: Life history characteristics of a cohort of 100 males and 100 females. MRDT is the mortality rate doubling time and IBI the interbirth interval. Rates of ageing and MRDTs refer to single figures calculated for the cohort as a whole. For all other characteristics, the mean is given followed by the standard error in brackets.

By fitting Gompertz curves to the mortality data, the rates of ageing were calculated as 0.16 for females and 0.27 for males. The corresponding mortality rate doubling times (MRDTs) are 4.18 years and 2.55 years. (Note that the parameter 'rate of ageing' was defined as the increase in mortality with age when investment in maintenance was always half the BMR; as individuals may choose their investment in maintenance, their true rate of ageing - obtained from the mortality curve of the cohort - may have been greater or lesser than this.) The female rate of ageing falls in the middle of the range of values quoted in the literature: 0.123 and 0.1774 for wild-living yellow baboons in Amoboseli; 0.1964 for wild-living olive baboons in Gombe and; 0.1457 for captive yellow and olive baboons and their hybrids in San Antonio, Texas[96]. The male rate of ageing is higher than what has been quoted in the literature: 0.213 for wild-living yellows in Amoboseli and; 0.1691 for the captive mixed population in San Antonio. However, it is probable that the populations from which data were available for both sexes experienced lower than average mortality since these gave the two lowest figures for rate of ageing in females.

Birth rates for simulated individuals are shown in figure 4.11D. The average age at first birth is 5.56 years for females and birth rate remains fairly constant from the start of adulthood through to 20 years with the average age at which members of the cohort became mothers being 9.85 years. The mean number of offspring born was 5.58 and the mean number successfully weaned 3.93 (table 4.2). The average interbirth interval was 1.42; this is slightly shorter than the length of time required to raise an offspring

from conception to independence as some offspring died as foetuses or infants. Variance in birth rate increases with age as the number of surviving females decreases.

For males, the function of offspring sired against age shows a sharp incline and decline so that births are concentrated within a much narrower window than for females. The average interbirth interval is correspondingly much shorter at 0.39 years. Males tended to be older when their first offspring was born, with an average age of 7.65 years (table 4.2). No male in this cohort sired offspring in his fifth or sixth years. Reproductive success was considerably higher than that of females between ages seven to ten - with a peak at eight years - before falling below that of females from age eleven onwards. The average age at which members of the cohort became fathers was 8.29 years. Mean numbers of offspring born and weaned were very similar to females at 5.31 and 3.83 respectively. This is as expected; since all offspring must have one mother and one father, the birth rate for both sexes must be the same at the population level and any differences between the two cohorts are attributable solely to stochastic effects.

Where male reproductive success was non-zero, that is between ages seven and twelve, individual variation was rather greater than for females of the same age. Although this may partially reflect differences in state between males, the predominant reason for this is that competition involves a degree of chance. Males of the same fighting ability and following the same strategy can have very different reproductive successes because the outcome of any competition between them is random.

These results generally approximate the behaviour of real baboons well. In the field, most females are known to become parents for the first time in their fifth or sixth years and most males in their seventh or eighth years. Johnson [97] gives the expected number of offspring for chacma baboons as between 4.88 and 5.71. The birth rate for females is known to be stable from maturity until the early twenties and that of males is known to peak at about eight or nine years at which point it is more than double that of females[70]. The model predicts a more rapid reduction in male reproductive success throughout the second decade than is seen in real baboons and possible reasons for this are given in the discussion. I am not aware of any data for IBI

in males in the field but that of females is a little higher than predicted by the model: Cheney et al. [76] observed an average IBI of about two years if the infant survived and 1.3 years if not. This likely reflects the fact that investment in offspring is not abruptly terminated at one year but gradually reduced thereafter.

Figure 4.11E shows the average number of days per year that males spent attempting to maintain a consortship with a female. None attempted to mate during their fifth year when their comparatively small size meant that their chances of success were effectively zero. Some effort to mate was made in their sixth year - 33 days on average - although the chances of success were still small. During their seventh and eighth years, when fighting ability is at its peak, males attempt to consort for an average of 180 days per year, or 18 days per period. This then remains reasonably constant throughout the rest of life, with only a slight increase at the very oldest ages. Once males reach their teens, their fighting ability is rather low and their chances of success correspondingly small. In this cohort no male sired offspring at age thirteen or above. However, since fighting ability will not improve (condition cannot become lower), it is still better for males to compete for access to females than not to.

Weingrill et al. [86]found that the highest-ranking males within four troops of chacma baboons consorted an average of eight or nine days per month: approximately half the number of days predicted by the model. However, if we consider number of consorted days as a proportion of the available receptive days the model and field observations seem to be in agreement. The expected number of oestrous females is non-zero for all 36 days of a period, therefore simulated high-ranking males consort 50% of available receptive days. In the study by Weingrill et al., 50-75% of available receptive days were consorted. Two caveats are worth mentioning here. Firstly, the number of receptive days per month was negatively correlated with the proportion that was consorted by high-ranking males in the field. When there were around 12 receptive days per month, 75% of these were consorted by high-ranking males; when this rose to around 17.5 receptive days, only 50% were consorted. We might therefore predict that if all days are receptive the proportion consorted would fall below the 50% predicted by the model. Secondly, and potentially offsetting this first point, the numbers of receptive days calculated by Weingrill et al. included a) all nine days of oestrous and b) cycles which did not result in conception. Males do seem able to

distinguish between conceptive and non-conceptive cycles and the highest-ranking concentrate mating effort within the four days of peak oestrous when fertilisation is most likely. If consorted days had been given as a percentage of the number of days of peak oestrous during conceptive cycles - which would then be comparable with model output - this would have been higher than 50-75%.

4.4.4. The effects of IMR and RoA on longevity

The model was used to investigate the extent to which the differential longevity of males and females is attributable to differences in IMR - which are coded into the model - as opposed to RoA - which are a behavioural choice. This can't be determined directly from the forward simulations above nor from data on real baboons because in both cases IMR and RoA are changed simultaneously. However, forward simulations could be run for males which artificially imposed upon them either the same IMR or the same RoA as females. Comparison can then be made between cohorts who experience the same IMR but differ in their RoA and vice versa.

The survival curves and life expectancies at five years for such cohorts are shown in figure 4.12. The female and male A cohorts are identical to those shown in the previous section. They both follow an optimal RoA given their respective IMRs. The cohort male B was exposed to the same IMR as females (0.00285/period) and experienced the RoA of males (0.27). Life expectancy at five years for this cohort was almost identical to that for the male A cohort: 10.64 compared to 10.42 years (figure 4.12B). The cohort male C was exposed to the IMR for males (0.00371/period) and experienced the same RoA as females (0.16). At 11.71 years, the life expectancy at five years for this cohort was closer to the female value of 12.88 years than to that of the male A cohort (figure 4.12B). This increase in longevity is driven by a reduction in mortality from about 11 years onwards; prior to this, age-specific survivorship is indistinguishable from that of the male A cohort (figure 4.12A).

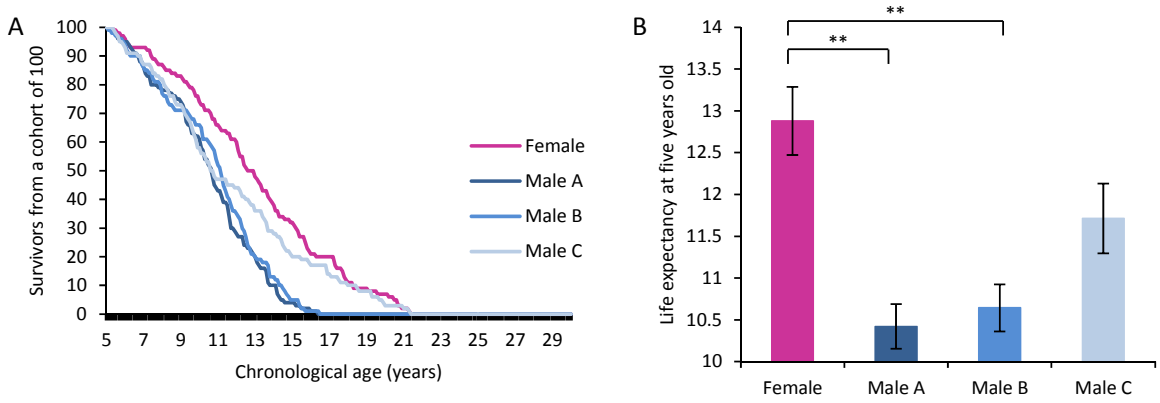

Figure 4.12: The survival curves (A) and life expectancies at five years (B) of four cohorts. The female and male B cohorts were exposed to an IMR of 0.00285/period; the male A and male C cohorts were exposed to an IMR of 0.00371/period. The RoA for the female and male C cohorts was 0.16 and for the male A and male B cohorts was 0.27. Error bars in B show the standard error and ** indicates significant differences at the 0.01 level between the cohorts indicated.

Analysis of variance (ANOVA) - using a reciprocal transformation to homogenise variances - found a main effect of RoA on lifespan at the 0.01 level. There was no significant main effect of IMR on lifespan, nor any significant interaction between IMR and RoA. Pairwise comparisons using a Bonferroni correction found significant differences at the 0.01 level between the female and male A cohorts and between the females and male B cohorts. Most of the variation in longevity between the sexes is therefore explained by a different level of investment in maintenance.

4.4.5 The fitness of suboptimal strategies

The relative fitness of suboptimal strategies gives an indication of the strength of selection in favour of the optimum. If fitness drops rapidly with small perturbations away from the optimum, selection is strong and little variation in strategy is expected in the wild. Conversely, if there are large regions of the strategy space for which fitness is close to its maximum value, selection there will be relatively ineffective and a range of different strategies are expected to persist in a wild population.

Genetic variants were considered which; a) caused their owners and all their descendants to follow strategies which consistently deviated from the optimum by a prescribed amount, or b) caused only members of one sex to follow such a strategy.

The fitness of these variants was defined as the expected number of copies 20 years into the future arising from a pair (one male and one female) of newly-matured individuals possessing the said variant. It was assumed that the rest of the population followed the optimal strategy.

The first set of such genetic variants caused investment in growth to be increased, and investment in maintenance to be correspondingly decreased, by a given percentage of the available energy budget each period. Investment in reproduction was unaltered. When such strategies were followed by both sexes, we see a clear peak in fitness at the optimum with even relatively small perturbations (up to four percent of the available energy budget) causing a noticeable reduction in fitness. Furthermore, underinvestment in growth was more costly in terms of reduced fitness than overinvestment (figure 4.13A, black line). Interestingly, the decrement in fitness from underinvesting in growth in mainly attributable to the effect such a strategy has in females whilst the decrement from overinvestment is driven largely by the effect in males. This can be seen from the curves for sex-limited suboptimal strategies (figure 4.13A, blue and pink lines). In females, overinvestment in growth by up to four percent of the available energy budget has little effect on fitness; selection against such strategies would be weak and they are expected to persist within a population. In contrast, underinvestment in growth significantly reduces fitness and would be strongly selected against. For males, overinvestment in growth is more costly than underinvestment but the difference is small. The efficiency with which natural selection can eliminate such strategies from a population is expected to be about the same.

Size trajectories for cohorts following these suboptimal strategies were computed to ensure that overinvestment in growth did not cause the maximum size boundaries to be hit. As well as confirming that this was not the case, these raise an interesting point about the change in size we should expect under a suboptimal strategy. It might be supposed that consistent overinvestment in growth would result in a greater size however, if the overinvestment is defined relative to a state-dependent optimal strategy rather than to a fixed schedule of investments, this need not be the case. Having overinvested in growth during period t, an individual will only continue to move away from the optimal size trajectory if the prescribed overinvestment in period (t + 1)

exceeds the optimal weight loss in that period given the individual's state. For females following suboptimal strategies, average size trajectories drift significantly from the optimal in this way (figure 4.13C). If however, optimal weight loss exceeds prescribed overinvestment in growth, the individual is pushed back towards the optimal size trajectory, as is seen in males (figure 4.13E).

Survival curves for these same cohorts are shown in figure 4.13 B & D and again these are not as might have been expected. For females, all suboptimal strategies are associated with reduced lifespan and overinvestment in maintenance at the expense of growth actually causes a greater reduction in lifespan than vice versa. This is because underinvestment in growth keeps individuals small post-maturity and the optimal change in condition per period is higher in smaller individuals (figure 4.8B). For males, survival is unchanged by an overinvestment in maintenance and reduced slightly by an underinvestment.

The strength of selection acting on the trade-offs between a) growth and reproduction and b) maintenance and reproduction were harder to quantify. Computing the fitness associated with consistent over- or underinvestment in reproduction by females does not produce meaningful results because the model assumes that dependent offspring have fixed energetic requirements. If investment in reproduction is always below optimal, offspring will never receive sufficient energy for survival, and a female's reproductive value will always be zero. If investment in reproduction is always above optimal, the extra energy can't be utilised by the offspring so a realistic fitness consequence of this cannot be computed. For males, consistent over- or underinvestment in reproduction post-maturity is not possible. Optimal investment in reproduction is zero during much of the subadult growth spurt, so that investment can obviously not be reduced. There are also occasions - although rather fewer - in late life when the optimal decision is to attempt consortship on all 36 days of a given period, and at these times it is not possible to invest more than the optimal amount in reproduction. The fitnesses of male-limited suboptimal strategies that deviated from the optimal by a prescribed amount in all states where this was possible were calculated. A steady decline in fitness was seen with increasing deviation from the optimal strategy (data not shown), but for the reasons stated above these results should be treated with caution.

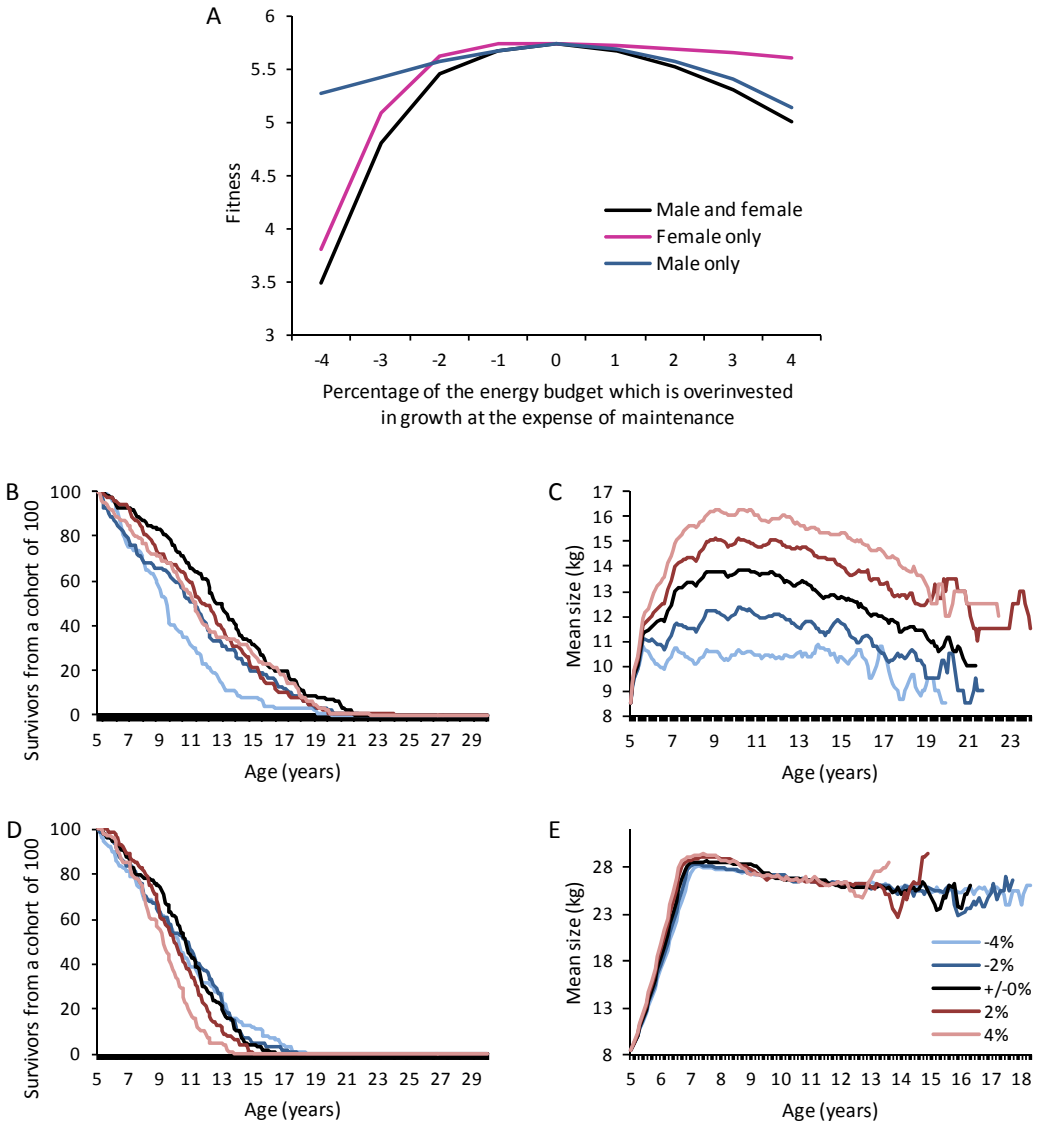

Figure 4.13: A: The decrement in fitness associated with genetic variants which cause a given percentage of the available energy budget to be overinvested in growth at the expense of maintenance. Fitness is here defined as the expected number of copies of the gene for the suboptimal strategy 20 years into the future arising from a pair (one male and one female) of newly-matured individuals. The black line indicates fitnesses when the pair and all their descendants follow the prescribed suboptimal strategy, the blue line when only males follow a suboptimal strategy and the pink when only females do. B-E: Survival curves (B: females and D: males) and average size trajectories (C: females and E: males) for cohorts of 100 individuals following strategies in which investment in growth always exceeds the optimum by the percentages of the available energy budget shown.

By calculating the fitness of genetic variants that cause their owners to deviate from the optimal strategy only during specified windows in condition, the relative force of selection over the mature life course was quantified. Consideration was restricted to variants that affected the trade-off between growth and maintenance, acted within a window of length 250τ and produced a deviation from the optimal strategy equivalent to four percent of the available energy budget. Results are shown in figure 4.14 for overinvestment in growth (A) and overinvestment in maintenance (B). Interesting differences can again be seen between male-limited and female-limited strategies. The force of selection declines monotonically with age in females. This is as expected since survivorship decreases with age so that a strategy which is suboptimal at older conditions will affect a far smaller proportion of the population than one which is suboptimal earlier in life. For males the decrement in fitness associated with following a suboptimal strategy between conditions 75 - 100τ exceeds that between 50 - 75τ, most noticeably when growth is overinvested in. Thereafter it is a decreasing function of condition. This is unexpected, since it is generally accepted that the force of selection declines with age for most organisms, and possible reasons for this are given in the discussion (section 4.5).

Note that deviations from the optimal strategy were calculated as a fixed percentage of the available energy budget. This is greater for baboons of condition 75 - 100τ than for those of condition 50 - 75τ, since the former are on average larger. To check that this alone didn't account for the results shown, simulations were repeated with the absolute energetic deviation from the optimal held at a fixed amount (equivalent to four percent of the available energy budget of a 30kg baboon). When growth was overinvested in, the same result was obtained; when growth was underinvested in, this result was lost and the force of selection became monotonically decreasing with age in males (data not shown).

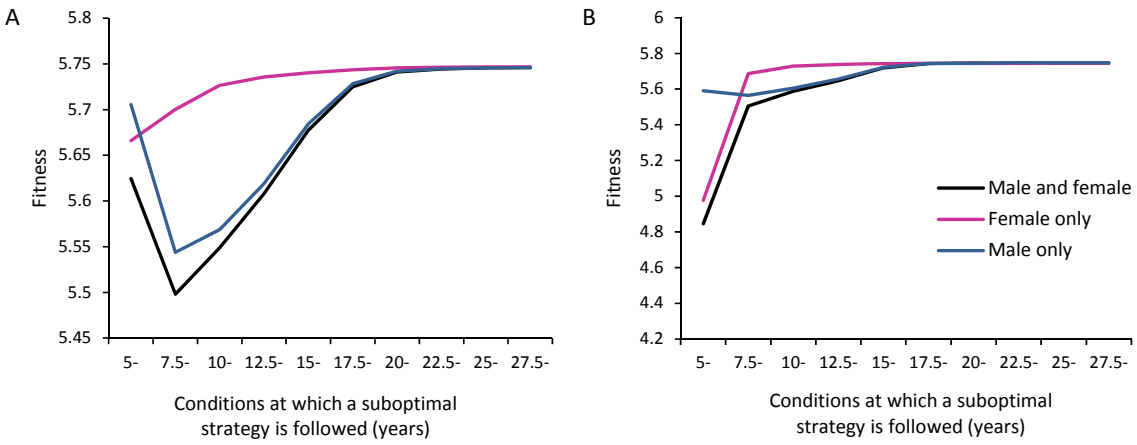

Figure 4.14: The fitness of genetic variants that cause their owners and all their owners' descendants to follow suboptimal strategies between the conditions indicated. In A the suboptimal strategy followed is an increase - by four percent of the available energy budget - in the energy invested in growth and a corresponding decrease in maintenance. In B there is an increase of the same magnitude in the energy invested in maintenance and a corresponding decrease in growth.

4.4.6 Sensitivity analysis

By recalculating the optimal strategy under a series of systematic perturbations to model parameters, the dependency of predicted behaviour upon the original parameter set was determined. Generally, eight perturbations to each parameter were considered, corresponding to increases of -30, -22.5, -15, -7.5, 7.5, 15, 22.5 and 30% of the original values. Only seven perturbations to conversion efficiency were considered since an increase of 30% gives a value greater than one. Only increases to male dispersal mortality were considered since the original value was zero; these could not be given as a percentage difference to the original value and are simply given as a percentage mortality risk. The energy available for growth, maintenance and reproduction was varied as a whole rather than varying each parameter that affects it separately and a justification for this is given in the methods (section 4.3.5). Backward iterations were run to find the optimal strategies under each perturbation followed by forward iterations for cohorts of 100 males and 100 females. Average lifespans, maximum sizes and number of births for these cohorts are shown in figures 4.15 (for females) and 4.16 (for males). Ratios (female-to-male) for average lifespan and maximum size, which reflect the degree of sexual dimorphism, are given in table 4.3.

Ratios for number of offspring are not given. As mentioned in section 4.4.3, this ratio must always be one at the population level, since each offspring must have one mother and one father. Any deviation from one between the male and female cohorts would therefore be attributable to stochastic effects.

Perturbations to the following parameters produced no significant difference to the three traits considered in either sex: probability of offspring loss, juvenile mortality, fighting ability 1, fighting ability 3, fighting ability 4, consort cost, dispersal mortality and ageing rate exponent. In some cases this may seem surprising. Increasing the probability of offspring loss does not, for example, lead to a reduction in the number of births. Whilst more offspring are lost before birth, more are also lost during their first year with the result that the mother falls pregnant again sooner and the mean interval between conceptions is reduced. The overall effect is a reduced number of offspring weaned but little change to the number born. There is also little change in lifespan under perturbations to the ageing rate exponent. In this case optimal investment in maintenance is adjusted to counter the change in this parameter.

Lifespan and number of births decrease with increasing IMR and RoA in both sexes (figure 4.15A & O; figure 4.16A & O). There is also a trend, especially in females, towards reduced maximum size. A greater proportion of the cohorts die before the age when maximum size would otherwise be achieved and the fact that this is most clearly seen in females probably reflects the fact that for them size peaks at a later age. For lifespan and maximum size the ratio between females and males stays much the same in both cases.

An increase in energy density leads to a reduction in maximum size in both sexes but no change to lifespan or numbers of births (figure 4.15D & 4.16D). Both sexes make approximately the same energetic investment in growth so that body stores have about the same caloric value, but the weight of these is reduced. In contrast, an increase in conversion efficiency leads to an increase in maximum size in both sexes (figure 4.15D & 4.16D). Again there is no change to lifespan or numbers of births. In this case, the same caloric investment in growth translates into a greater increase in size and consequently greater reserves to draw on for provisioning offspring for

females and a greater fighting ability for males. In both these cases the degree of sexual dimorphism shows no consistent change (table 4.3).

The most striking changes to female characteristics are seen under perturbations to the energetic requirements of offspring and the energy available for growth, maintenance and reproduction. When the energetic requirements of offspring are increased, the female responds by putting on more weight before conceiving and by investing less in maintenance. If a female waited until body stores were sufficiently large such that she could provision an offspring without compromising maintenance, the chances that she will succumb to mortality before raising any offspring would be increased. Her response is therefore a compromise which results in greater maximum size; reduced lifespan and a reduced number of offspring (figure 4.15L). Increasing the energy available for growth, maintenance and reproduction has the opposite effect (figure 4.15M). The reasoning here is much the same; a greater available energy budget effectively reduces the relative cost of raising an offspring.

Male maximum size and lifespan do not change significantly under perturbations to the energetic requirements of offspring (figure 4.16L) so that sexual dimorphism in both these characteristics is reduced (table 4.3). A larger energy budget for growth, maintenance and reproduction has little effect on lifespan in males and produces a slight increase in maximum size so that dimorphism in both these characteristics is enhanced (table 4.3). The changes to dimorphism seen in these two cases are likely artefacts, resulting from the stipulation that mothers cannot vary investment in offspring in response to state or the environment. In reality, energetically stressed mothers can make a series of smaller investments over a longer period. When resources are abundant they can make larger investments so that offspring are of better quality and/or reach independence at younger ages.

Lastly, variation in fighting ability 2 has no effect upon female life history characteristics, nor lifespan or number of offspring in males, but has a marked effect upon maximum size in males. Recall that this parameter determines the size at which the increase in fighting ability with size is maximal. By increasing this, larger males are more able to monopolise mating opportunities and this drives all males to attain a greater maximum size. Dimorphism in size is correspondingly increased. (Note that

for simulations where fighting ability 2 was increased by 15% or more, the maximum size boundary for males was increased to 34kg because the lower boundary of 30kg was being hit during forward iterations.)

Since quantitative support for the sigmoidal relationships between size/condition and fighting ability (see section 4.3.3.8) was lacking, other functions were also considered. Specifically, linear, quadratic and cubic dependencies of fighting ability upon size and/or condition were modelled. Changing the dependency of fighting ability upon condition had little impact upon male behaviour however changing the dependency upon size produced some interesting results. When fighting ability was a linear function in size the maximum size that males attained, and consequently the degree of size dimorphism, were considerably lower than they were with sigmoidal, quadratic or cubic functions in size (data not shown). They were also lower than has been reported for any baboon population that I am aware of. It can therefore be concluded that the benefit of increased size to a male's fighting ability must be more than linear. Since the probability of a male winning a fight is proportional to his fighting ability relative to his rival (see section 4.3.3.8), this means that if two males of sizes x and 2x fight the probability of the former winning must be lower than one third. When fighting ability was a polynomial function in size (be it linear, quadratic or cubic) the reduction in size with age was much more pronounced than for a sigmoidal function with weights in later life falling to 15kg or less (data not shown). This reduction in size is far greater than is seen in real baboons and may indicate that a) a sigmoidal curve, which excludes smaller individuals from mating more completely, is a more accurate characterisation of the dependency of fighting ability upon size or b) that there is some physiological constraint on so drastic a weight loss.

	Deviation from the original value								
	- 30%	- 22.5%	- 15%	- 7.5%	+/- 0%	+ 7.5%	+ 15%	+ 22.5%	+ 30%
Lifespan									
Initial mortality rate	1.29	1.32	1.32	1.27	1.24	1.32	1.32	1.21	1.29
Offspring mortality	1.32	1.35	1.26	1.35	1.24	1.21	1.25	1.27	1.24
Juvenile mortality	1.28	1.33	1.30	1.21	1.24	1.32	1.23	1.28	1.32
Energy density	1.45	1.29	1.27	1.37	1.24	1.26	1.28	1.31	1.19
Conversion effeciency	1.28	1.27	1.26	1.24	1.24	1.30	1.23	1.26	
Fighting ability 1	1.23	1.27	1.20	1.23	1.24	1.24	1.25	1.28	1.16
Fighting ability 2	1.21	1.28	1.06	1.20	1.24	1.24	1.17	1.15	1.38
Fighting ability 3	1.20	1.15	1.20	1.24	1.24	1.22	1.19	1.19	1.12
Fighting ability 4	1.21	1.10	1.22	1.30	1.24	1.15	1.25	1.24	1.28
Cost of consortship	1.28	1.27	1.32	1.19	1.24	1.19	1.33	1.30	1.21
Dispersal mortality					1.24	1.24	1.32	1.28	1.22
Offspring energy needs	1.38	1.35	1.27	1.41	1.24	1.31	1.25	1.23	1.19
Energy for G/M/R	1.09	1.22	1.22	1.31	1.24	1.40	1.25	1.24	1.39
Ageing rate exponent	1.37	1.28	1.33	1.36	1.24	1.30	1.19	1.27	1.28
Rate of ageing	1.17	1.14	1.33	1.19	1.17	1.21	1.17	1.22	1.23
Maximum size									
Initial mortality rate	0.54	0.54	0.53	0.54	0.52	0.53	0.54	0.53	0.53
Offspring mortality	0.56	0.55	0.53	0.54	0.52	0.53	0.53	0.52	0.52
Juvenile mortality	0.53	0.54	0.55	0.54	0.52	0.55	0.53	0.55	0.53
Energy density	0.57	0.55	0.54	0.55	0.52	0.55	0.54	0.56	0.53
Conversion effeciency	0.52	0.52	0.51	0.53	0.52	0.53	0.53	0.56	
Fighting ability 1	0.54	0.56	0.53	0.52	0.52	0.55	0.52	0.53	0.52
Fighting ability 2	0.67	0.64	0.59	0.56	0.52	0.50	0.47	0.45	0.47
Fighting ability 3	0.52	0.51	0.52	0.54	0.52	0.54	0.54	0.52	0.52
Fighting ability 4	0.53	0.52	0.53	0.54	0.52	0.52	0.53	0.53	0.52
Cost of consortship	0.53	0.55	0.55	0.51	0.52	0.53	0.54	0.54	0.51
Dispersal mortality					0.52	0.53	0.55	0.53	0.54
Offspring energy needs	0.45	0.47	0.48	0.52	0.52	0.58	0.59	0.62	0.62
Energy for G/M/R	0.65	0.64	0.61	0.56	0.52	0.53	0.49	0.48	0.47
Ageing rate exponent	0.55	0.52	0.56	0.54	0.52	0.55	0.53	0.55	0.57
Rate of ageing	0.56	0.55	0.54	0.54	0.53	0.53	0.54	0.53	0.54

Table 4.3: The ratios (female to male) of lifespan, maximum size and number of births for the cohorts shown in figures 3.15 & 3.16. Energy for G/M/R is the available energy budget which is to be split between growth, maintenance and reproduction.

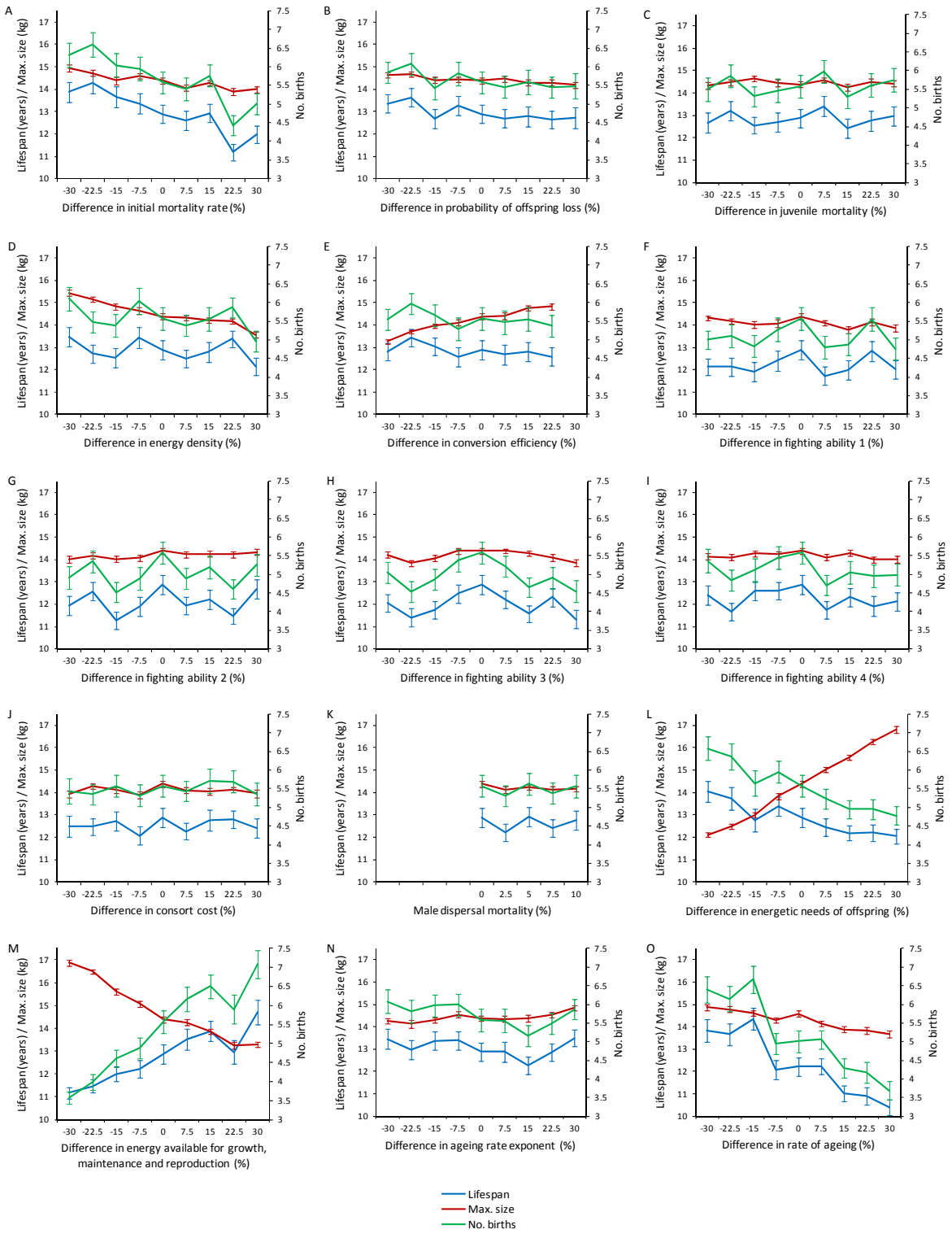

Figure 4.15: The lifespans, maximum sizes and number of births for cohorts of 100 females who follow strategies that are optimal under the perturbations to model parameters indicated. Error bars represent standard errors.

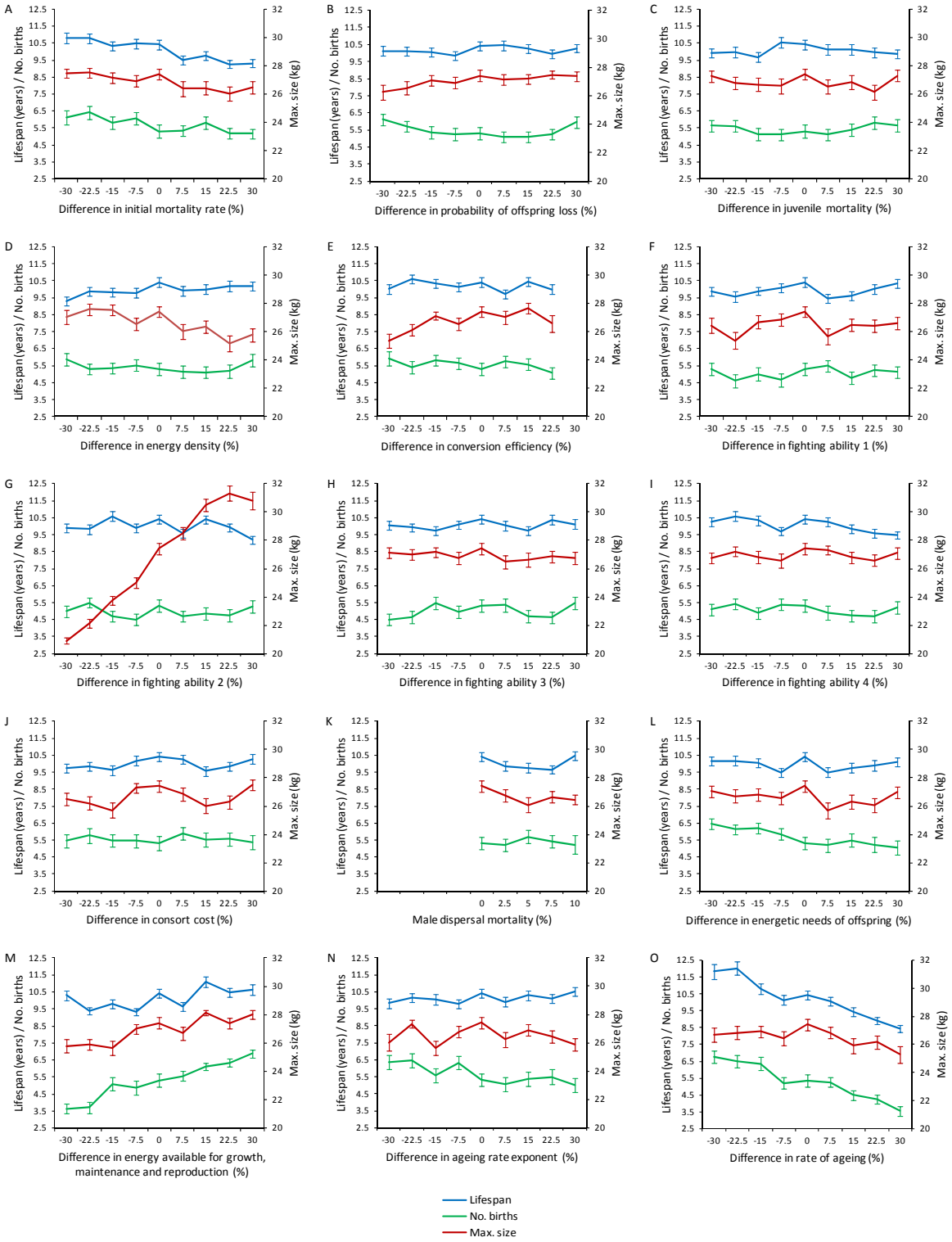

Figure 4.16: The lifespans, maximum sizes and number of births for cohorts of 100 males who follow strategies that are optimal under the perturbations to model parameters indicated. Error bars represent standard errors.

4.4.7 The optimal sex ratio

As mentioned in section 4.3.4, if the population is growing and one sex is on average younger when they produce offspring, this sex will have a higher reproductive value at birth (and upon attaining maturity) assuming an equal sex ratio. It is therefore in a parent's interests to produce slightly more of this sex, in the present case males. In doing so the difference in fitness between the sexes is reduced as there are now more males competing for fewer reproductive females. The sex ratio will settle at an optimum where the advantage from having offspring sooner is balanced by the disadvantage from being the more prevalent sex at birth.

The optimal sex ratio was determined under the assumption that its choice was independent from that of energy allocation: females were allowed to choose a different sex ratio at conception but both sexes were required to follow the energy allocation strategies calculated under an equal sex ratio. Initially, it was known that the optimal sex ratio (the proportion of males) had a lower bound of 0.5 and an upper bound of 1.0. Convergence upon the optimum was achieved by repeated cycles of the following: selection of a sex ratio half way between the current lower and upper bounds; determination of the reproductive values for both sexes upon maturity under this sex ratio and; the updating of either the upper bound (if females were fitter) or the lower bound (if males were fitter). This was terminated when the optimum had been calculated correct to three significant figures. The optimal sex ratio predicted by the model showed only a very slight bias towards males, at 0.504.

4.5 Discussion

4.5.1 A two-sex model of energy allocation which supports the sexual selection hypothesis for dimorphism

A two-sex model of energy allocation decisions has been built with the decisions of both sexes affecting those of the other. Both sexes follow a strategy the optimality of which is based upon the predicted behaviour of the rest of the population in the current and/or in future time points. For males, the expected mating success within any given period will depend upon the distribution of receptive females; the distribution of mature males and; the number of days that other males will attempt to consort. For females, allocation decisions depend in part upon the predicted reproductive success of sons and therefore upon the future population distribution and the strategies that both sexes will employ in the future. Whilst dynamic programming always assumes that descendants will follow an optimal strategy, in this case it is therefore also assumed that the rest of the population will. As far as I am aware, this is the first attempt to apply dynamic programming to the decision processes of two groups of individuals who interact with each other and whose optimal strategy depends upon the strategy employed by members of the other group.

The model reproduces the sexual dimorphism in longevity, body size and reproductive scheduling that is seen in real baboons. Life expectancy at five years is almost two and a half years greater for females than for males and this is driven primarily by differences in RoA - which are a behavioural choice - rather than differences in IMR - which are externally imposed. Simulated males undergo a sub-adult growth spurt to attain a maximum size approximately twice that of females. Male reproductive output is concentrated within a much narrower window than a female's is; attempts to establish a consortship are for the most part delayed until after the sub-adult growth spurt and the age-related decline in reproductive success begins sooner and is more pronounced than in females.

Predicted dimorphism is robust to changes in most parameter values. This is important since the same degree of dimorphism is seen across multiple populations and species of baboons, each experiencing a different external environment. Baboons inhabit forest, desert and mountain regions as well as savannah and these various

terrain differ in, among other things, food patch distribution, rainfall, visibility, refuge distribution and predator density. Temporal variation in habitat quality can also be considerable with some years seeing much heavier predation, disease, flooding or drought than others. For one population of chacma baboons in Botswana, annual mortality rate varied four-fold among adult females (0.04 - 0.16) and nineteen-fold among infants (0.03 - 0.57) over a ten-year period[76]. Despite these differences, sexual dimorphism in size, longevity and reproductive output is largely unaltered.

Whilst not excluding other hypotheses, these results are consistent with the sexual selection hypothesis for dimorphism. The differences that are seen between males and females in the field are as expected if both sexes are optimally partitioning their available energy between growth, maintenance and reproduction.

4.5.2 Discrepancies between the model and experimental data

Simulated individuals of both sexes choose to age more rapidly than those in the wild population from which mortality figures were taken. This was evident in the population pyramid showing both condition and age distributions at equilibrium (figure 4.10C) and in the rates of ageing calculated from cohort mortality data (table 4.2). This might result from simplifications of baboon biology that were used in the model. For example, female provisioning of offspring was compulsorily terminated at one year and males were unable to invest in offspring post-conception. Were these constraints relaxed and both sexes choose to increase the time span over which they provided parental care, the dependency of fitness upon longevity - and hence the optimal investment in maintenance - might increase. Alternatively, the optimal ageing rate predicted by the model may differ from that of the population in question simply because other parameters in the model were drawn from multiple sources and do not exactly match those of the said population. The predicted rate of ageing for females was, at 0.16, still lower than that reported in two other populations of wild-living baboons (0.1964 and 0.1774) and is therefore well within the range that is experimentally plausible. (I am not aware of any other estimates for IMR and RoA in wild-living male baboons.)

Other predicted aspects of male life history are accelerated in comparison what is seen in the field. The length of the sub-adult growth spurt is shorter than in real baboons so

that maximum size is achieved sooner[70]. The value of a strategy in which growth is slower and lifespan a little longer (because investment in growth is lower and in maintenance higher during sub-adult life) may only become apparent when males are able to provide offspring care or gain mating access in later life through the formation of coalitions. Alternatively, as it requires that males learn to fight and that they repeated challenge other males in the group, the establishment of rank may take some time in the field and may therefore favour a more protracted period of development. The drop in male reproductive output with age begins sooner and is more rapid in simulated than in real individuals. This may again result from the fact that simulated individuals can't form coalitions and so their mating opportunities in later life are reduced to a far greater extent than is seen in most real baboon populations.

The model has also failed to reproduce the time lag between a female weaning an offspring and falling pregnant again. However this is unsurprising when we consider that, in the field, offspring independence is gradually achieved and newly-weaned offspring will continue to receive some maternal investment which would be compromised by another pregnancy.

4.5.3 The fitness of suboptimal strategies

A number of interesting findings emerged from simulations in which individuals deviated from the optimal strategy in a prescribed manner. Firstly, overinvestment in growth at the expense of maintenance was more costly in males than females whilst underinvestment in growth at the expense of maintenance was more costly in females than males. Secondly, suboptimal investments in growth and maintenance do not necessarily produce significant differences in lifespan or size when compared with the optimal. Thirdly, the force of selection upon the trade-off between growth and maintenance is a monotonically decreasing function of age in females but not in males. Importantly, for each of these findings, suboptimal strategies were defined relative to a state-dependent optimal rather than a fixed schedule of investments. (In fact, it would not be possible to define a fixed schedule of overinvestments in one function and underinvestments in another. If, for example, growth were overinvested in and maintenance underinvested in by some amount x during a given period, then foraging yield - and hence available energy budget - in the next period would be reduced (as this is an increasing function of size). If the same overinvestment in growth were

repeated in the next period, the underinvestment in maintenance would necessarily have to greater.)

For females, underinvestment in growth at the expense of maintenance was much more costly than the reverse. For males, overinvestment was more costly but the difference was much smaller. Should a range of strategies persist in a population, it is therefore to be expected that those followed by females will tend toward greater than optimal investment in growth with a corresponding reduction in maintenance, whilst those of males will show an even spread around the optimal investment in both. For females, underinvestment in growth resulted in a reduced weight gain between ages five and nine and consequently a significantly smaller size throughout the mature life course (figure 4.13B). The benefit of size in terms of increased foraging yield was never fully realised so that these individuals were always more energetically stressed than those following an optimal strategy. Optimal changes in condition are greater in smaller individuals (figure 4.8B) and the prescribed overinvestment in maintenance was not sufficient to overcome this; lifespan was actually reduced in those who overinvested in maintenance.

For males, the prescribed suboptimal investments were not sufficient to produce the same drift in average size trajectory from the optimal that was seen in females so that the effects of different foraging yields throughout mature life were not seen. Differences in lifespan between cohorts following suboptimal and optimal strategies were also much smaller.

This raises an interesting point about the degree of phenotypic variation we might expect to see in a population where a range of suboptimal strategies are followed as well as the optimal. A shallower slope in the fitness landscape implies more strategies will be followed in the wild but - if these do not produce significant differences in the characteristics of interest - does not imply a greater variation in phenotypes seen in the wild. For example, selection against a 4% underinvestment in growth in males is approximately as strong as against a 2.5% underinvestment in growth in females (figure 4.13A) so we expect such strategies to persist in a population to about the same extent. However, the range of sizes and lifespans seen within the female population would still be greater (figure 4.13B-E). These results also reiterate a point that has

been previously made, that the existence of a trade-off between maintenance and growth does not imply that smaller individuals within a population will be longer-lived. It has been pointed out that smaller individuals may be of lower quality and therefore have less for maintenance as well as growth (see chapter 1.5.3). It may also be the case that, by following a strategy of underinvestment in growth, individuals will obtain a reduced foraging yield and therefore have less for both functions.

Selection upon the trade-off between growth and maintenance is stronger between 75-100τ than 50-75τ in males, and then declines monotonically with age from 100τ onwards. One potential explanation for this initial increase in the force of selection may be that it is more costly to follow a suboptimal strategy in certain states than in others. If these states happen to be those most commonly occupied in the second condition bracket, the force of selection appears strongest here. Overinvestment in growth may, for example, always be more costly at larger sizes because the associated increase in foraging yield relative to the increased cost in essential functions is lower than at smaller sizes.

Alternatively, the explanation for this might lie in the fact that the reproductive values of the older individuals tend to be greater. The vast majority of reproduction is concentrated within the latter period, so that a suboptimal strategy here brings an immediate cost in terms of fitness. In contrast, although a suboptimal strategy during the earlier period will affect state at the time of first reproduction, this may to some extent be compensated for by adherence to the optimum thereafter so that the absolute loss of fitness is smaller. As females begin reproduction almost as soon as they attain maturity at five years, results for them would also be consistent with strength of selection peaking at the time of maximum reproductive value. The force of selection acting upon the trade-off between growth and maintenance would then be different is in this regard from that acting upon genes conferring a fixed mortality risk, such as were considered by Hamilton (see chapter 1.4.3).

It is hard to tell between these possibilities from the data here presented. However, it would be interesting to return to this point in future using the model in chapter 5. Since this can model the fitness of suboptimal strategies throughout the entire juvenile

period, it can be seen more clearly whether the force of selection does indeed rise to a peak at (or just before) the point of maximum reproductive value.

4.5.4 The optimal sex ratio

When the sex ratio at conception (or equivalently at birth at upon maturity) was 0.5, the reproductive value of males at each of these junctures was higher than that of females. This was at first unexpected since there are no differences in quality or mortality between males and females prior to maturity. It results from two features of the simulated population: a positive growth rate and the fact that males were on average younger when they sired offspring than females were when they gave birth. In a growing population, the expected number of copies of an individual's genes within a population increases with each generation and so a reduction in generation time increases the expected number of gene copies at any specified future time point. All else being equal, a parent does better to have offspring sooner; in this case, the sex which is able to have offspring sooner does better. When the mother was allowed to adjust the sex ratio to increase the likelihood of a male birth, the operational sex ratio became increasingly male-biased and the advantage to males from being younger when their offspring are born was offset by a reduction in their expected number of offspring. The sex ratio settled at an optimum of 0.504. Whilst the effect of population growth rate and a difference in the average age of the sexes when they become parents upon the optimal sex ratio was very slight in this model, the point is theoretically interesting - it represents an exception to Fisher's principle [98]that natural selection will favour an equal investment in both sexes - and it may have important implications for other species/populations.

The argument for an equal sex ratio as laid out by Fisher can be summarised as follows. Since both sexes make an equal contribution to the genetic material of the next generation, the total reproductive value of all males within a population at the time at which parental investment ceases must equal that of all females. Consequently, any systematic bias in the parental expenditure directed towards a given sex would establish a selective pressure in favour of increased expenditure towards the other; parents who invested more in the other sex would contribute a larger fraction of the genetic material of future generations. Equal expenditure on both sexes corresponds to an equal sex ratio if the average investments required to raise individuals of both

sexes to independence are the same. Should one sex suffer higher mortality during the period of parental investment, the average energetic investment required to raise an individual of this sex to independence will be greater since, for every offspring of this sex successfully raised, there will be a greater number who have requisitioned parental resources but not survived to independence. The average parental expenditure per birth is, however, lower for this sex since a lower proportion will appropriate parental investment for the full length of time needed to attain independence. In order for the total expenditure in both sexes to be equal, natural selection will favour a higher frequency of this sex at birth but a lower frequency at the age when parental investment is terminated. Any difference in mortality between the sexes after the period of parental investment has no effect upon the sex ratio; although higher mortality may mean that fewer members of one sex will survive to reproduce, those that do survive will enjoy a higher reproductive rate and the expected reproductive value accrued to the parent from offspring of this sex per unit expenditure is unchanged.

There has, since the publication of Fisher's argument, been recognition of the fact that optimal investment in each sex may vary between subgroups of a population defined by quality. Trivers and Willard [99]realised that if offspring quality correlates with that of the mother and the dependency of reproductive success upon quality is steeper for one sex than the other, mothers of good quality should produce more of this sex and mothers of poor quality less. For many species in which the majority of offspring care is undertaken by the female, the operational sex ratio is male-biased; as a consequence competition between males for mates is intense with those of highest quality often monopolising access to receptive females. It follows that the reproductive value of a good-quality male is greater than that of a good-quality female since the latter's reproductive output is constrained by the need to provide prolonged investment to each offspring whilst the former's is only constrained by the number of receptive females. A good-quality male can sire offspring at a much faster rate than a good-quality female can birth them. The reproductive value of a poor-quality male is, however, lower than that of a poor-quality female since the latter can usually secure a mate despite her quality whilst the former is frequently excluded from mating altogether by higher-quality males. Assuming a correlation between the quality of

mother and offspring, females of good quality will do better to produce more sons than daughters and females of poor quality more daughters than sons. As quality is defined relative to other members of a population, these deviations from an equal sex ratio (strictly from an equal investment in both sexes) should cancel out at the population level.

Crucially, the slight male bias in the sex ratio predicted by the model is seen at the population level, is independent of quality - as maternal investments followed a fixed schedule - and independent of differences in mortality during the period of maternal investment - as mortality was identical for males and females prior to maturity. Rather, this bias is driven simply by a positive population growth rate and the fact that males are younger on average when their offspring are born. The potential impact of these factors on the sex ratio in other species/populations would be interesting to explore, perhaps in particular in populations which undergo regular cycles of growth and decline and in which the sex ratio may therefore be sequentially skewed toward one sex and then the other.

4.5.5. Limitations to the model

A number of simplifications were made in constructing this model, either due to lack of experimental data or in order to make the system computationally tractable, and many of these have already been alluded to. Since a model is only as good as its assumptions, the results here presented can be considered reliable only insofar as these simplifications constitute a close approximation to biological reality; should they deviate significantly from what is seen in the field, the model will necessarily provide much more limited insight into the behaviour under consideration. The most pertinent simplifications are therefore outlined below along with some justification of their use.

The reproductive strategies available to simulated individuals of both sexes are an abstracted set of those available to real baboons. Females were assumed to mate with whichever males gained access to them and had no scope to employ their own mating strategies. Furthermore, their energetic investments in offspring always followed a fixed schedule. Related to this second point, there was no discrimination between females on the basis of rank which is known to affect the infant growth rate that a mother can support. Male reproductive success depended solely on the ability to

secure access to receptive females by means of individual challenges over consortships; there was no opportunity to form coalitions, provide paternal care, develop friendships or undergo secondary dispersals. Consortship was assumed to entail a cost only in terms of foraging yield lost as a consequence of maintaining close proximity to the female - any direct cost from aggressive encounters was not considered. With infanticide modelled as a uniform threat to all those under one year, adult males were unable to protect their own offspring nor to target those of other males.

These simplifications are justified in that they exclude subsidiary components of the reproductive strategy whilst retaining those which are the primary determinants of reproductive success. Although females were not permitted to vary their investment in offspring, the important point is that a large investment in made over a significant period of time and a female's ability to make such prolonged investments is the primary determinant of her fitness. Rank has been shown to have comparatively little effect upon female reproductive success and, since the most likely father of an offspring is whichever male establishes a consortship with the mother during the period of peak oestrous, the value of a strategy of paternal confusion in terms of reducing the risk of infanticide can also be assumed to be comparatively low. For males, the majority of reproductive success is known to be accrued via solo tactics during early adulthood when size - and therefore rank - is at a peak. Some post hoc justification for overlooking the strategies by which smaller or older males may gain mating rights (for example, through sneak matings or the formation of coalitions) is obtained from sensitivity analysis for the parameters 'fighting ability 1' and 'fighting ability 3'. These parameters describe respectively the rate of increase in fighting ability with size and the rate of decrease with condition. By reducing the former smaller individuals enjoy a greater reproductive success relative to larger ones and, by reducing the latter older individuals enjoy a greater reproductive success relative to younger ones. Predicted sexual dimorphism was essentially invariant to decreases of up to 30% in both parameters (table 4.3). Although the exact nature of the cost of consortship may vary slightly from what is assumed in the model, the key point here is that a significant cost is entailed and therefore the choice of whether to attempt to consort is one of weighing this cost against the potential reproductive gains. Finally,

since the vast majority of offspring care is undertaken by the mother, it is reasonable to suppose that the fitness a male accrues via paternal care will comprise a relatively small fraction of his overall fitness.

Although not modelled explicitly here, foraging effort is also a behavioural decision, made partly at the level of the individual and partly at the level of the troop. The gains from an increased foraging effort - a greater available energy budget - must be weighed against the costs - a greater exposure to predators and parasites; the energetic costs of digestion and of seeking new foraging sites and; for males, the fact that insofar as increased foraging effort is incompatible with maintaining a consortship current reproduction may suffer. Since baboon troops move to and from feeding sites as one, the decision over which foraging grounds to exploit must be made at the level of the troop. As mentioned in the introduction, larger troops experience a diluted threat of predation and increased feeding competition and are therefore more likely to select richer foraging grounds even at the expense of greater predator density, reduced visibility or longer distance to a refuge. At the individual level foraging choices may include location within the group (with those at the periphery experiencing a greater risk of predation than those in the centre); level of vigilance and; willingness to challenge others for prime foraging sites.

To have included foraging effort as a behavioural decision would have greatly increased the model run time. This is especially true for decisions made at the level of the troop - an individual's foraging yield and mortality risk would become a function of the size and composition of the troop to which (s)he was a member. Sensitivity analysis is again useful here in assessing the extent to which dimorphism arising from differential allocation between growth, maintenance and reproduction can be studied without explicit reference to foraging effort. Perturbations to the initial mortality rate (which may reflect risk of foraging strategy) had no significant effect upon dimorphism in size and longevity. Perturbations to the available energy budget (equivalent to changes in the foraging yield) were positively correlated with size and to a lesser extent longevity, although for all perturbations considered females were still substantially longer-lived and smaller than males. (That results obtained under perturbations to the available energy budget may be an artefact arising from the

constraint that maternal investment in offspring must follow a fixed schedule was discussed in section 4.4.6.)

It was assumed that differences in IMR between males and females were externally imposed whilst differences in RoA were a behavioural choice. The latter resulted from differential investment in maintenance, and therefore different rates of increase in condition. RoA is also dependent upon some environmentally-determined rate at which mortality rate increases per increment in condition but this was assumed to be the same for both sexes. In reality, IMR may also depend partly upon behavioural choices. If the higher IMR for males results from an increased risk of predation and of injury during agonistic encounters, then this is externally imposed only to the extent that a) aggressive encounters are initiated by other males seeking to gain access to females or establish their rank within the dominance hierarchy and b) any preference which predators show for males is independent of male behaviour such as the riskier foraging strategy which they seem to employ in order to support their larger size. However, in support of the notion that IMR depends at least principally upon extrinsic threats and RoA upon behavioural choices, Bronikowski et al. [96]report markedly different IMRs but very similar RoAs between three baboon populations (one captive and two wild). Whilst external environments can differ substantially between populations which have only recently diverged - as is the case for captive and wild baboons - behavioural choices, which are the product of natural selection, are not expected to alter significantly between them.

It is also worth noting that inasmuch as mortality is due to disease, it may vary with investment in immunity rather than maintenance and may also depend upon group size and cohesion since transmission probability is generally an increasing function of the density of conspecifics. However, for most baboon populations, predation accounts for the majority of deaths in adults and juveniles.

Finally, male allocation decisions were calculated under the assumptions that troop composition was invariant throughout the wider population and that females' menstrual cycles were independently timed. If a troop contains a greater proportion of receptive females than average and/or a smaller proportion of mature males (especially of males at their peak fighting ability), smaller males will find their chances

of fathering an offspring should they attempt to consort improved and may alter their allocation strategy accordingly. Conversely, if a troop contains a smaller proportion of receptive females than average, higher-ranking and older males may not be able to consort for the number of days predicted by the model because there may not sufficient receptive days available, especially if the troop is small. If female cycles tend to be synchronised then, as a male cannot consort with more than one female at a time, higher-ranking males will be less able to monopolise matings and smaller males may again increase the number of days on which they attempt to consort. This model is therefore best applied to larger baboon troops in which stochastic differences in troop structure, and possibly cycle synchronicity, will be lower.

4.6 Summary

1. A two-sex model of energy allocation between growth, maintenance and reproduction in baboons has been built with the optimal decisions for each sex depending upon the behaviour of the rest of the population.
2. The model reproduces the sexual dimorphism in size, longevity and reproductive scheduling seen in real baboons.
3. By computing the fitness of suboptimal strategies, it has been shown that the force of selection acting upon a trade-off need not decline monotonically with age.
4. The optimal sex ratio is skewed slightly towards males; this results from the fact that the population is growing and males are on average younger when they reproduce than females.
5. The model is generally robust of changes in most parameter values.

Chapter 5: Accelerated life history following early life adversity in humans

5.1 Introduction

5.1.1 An adaptive basis for the acceleration in life history which follows early life adversity

It is well documented that individuals who have suffered from any of a range of hardships when very young seem to live life at a faster pace. These individuals mature at a smaller size and a younger age, begin reproduction sooner and experience reduced longevity and poorer health in later life[53, 100, 101]. The ability of nutritional restriction *in utero* to accelerate life history in this way has received particular attention, however, a similar response is seen under a variety of different stresses including disruption to the family unit, parental addiction disorders, abuse and neglect. Short duration of breastfeeding, frequent residential moves, paternal absence and separation from the mother have all been shown to be independently associated with a younger age at first pregnancy in a cohort of British women[100]. Moreover, those females who would go on to be young mothers showed, throughout development, consistent differences to their peers in growth and psychology: they were smaller at birth, displayed poorer emotional adjustment at ages 7 and 11, attained a shorter final stature and by 16 years judged the optimal age for starting a family to be younger than did controls[101].

Studies in a number of animal species have demonstrated a causal relationship between early adversity and accelerated life history. The association remains when environmental factors other than the hardship of interest are controlled for and cross-fostering designs have been used to show that it is the environmental stress rather than any genetic effect to which this attributable (references in [102]).

The explanation for this early programming of reproductive strategy and health in later life has been the subject of some debate. It is possible that adversity, at the time when it is experienced, compromises physiology in some way that cannot be subsequently overcome and this imposes some mechanistic constraint on the later phenotype of the individual. Alternatively, if different life history traits are fitter in

different environments, the genotype may have evolved the capacity to form different phenotypes depending upon the prevailing environment, with a particular life history trajectory then being 'chosen' in response to cues received in early life[53, 102]. This is one way in which a genotype may be said to exhibit adaptive phenotypic plasticity.

Consistent with this idea, there is evidence that the environment *in utero* or very shortly after birth is of particular importance[103]. The life history trajectory which is committed to during this critical window is fairly robust to later changes in the environment and adversity during the later juvenile period is unable to elicit the same shift in life history traits as early adversity does. A plentiful environment in later life also seems to exacerbate the effects of a scarce one in early life, implying that the phenotype is in this case mismatched to the environment. A range of metabolic disorders were common in survivors of the Dutch Hunger Winter (1944-45), who experienced rapid subsequent improvement in nutrition, but not so in survivors of the siege of Leningrad (1941-44), for whom nutritional availability remained low[53]. As stated in chapter 1, when a period of scarcity in early life is followed by one of plenty, any early growth retardation may be compensated for by a period of catch-up growth and it is this catch-up growth, more so than the early growth retardation, that is most strongly associated with a reduction in lifespan.

A number of theories that give adaptive explanations for an early commitment to a given speed of life history are summarised in the following sections. For any of these theories to hold, several conditions must be met [104]and these are listed here.

- The population must - over the course of its evolutionary history - have faced a range of different environments.
- The optimal set of history characteristics must vary as a function of environment.
- The individual must receive reliable cues in early life of the current environment.
- There must be sufficient temporal correlation between environments for the cue to provide a good predictor of the environment at the time when any benefit from commitment to the chosen life history trajectory is felt.

- The fitness gains from making a correct prediction - the increase in fitness from being well-matched to the environment multiplied by the probability that the environment is predicted correctly - must exceed the costs - the fitness losses from an incorrect prediction multiplied by the probability that the individual will not be well-matched to its environment.
- The fitness costs of maintaining plasticity throughout life (i.e. the ability to vary phenotype in response to a changing environment) must exceed the fitness costs associated with commitment to a trajectory which is not the best-matched to the environment.

5.1.2 The thrifty phenotype hypothesis

The thrifty phenotype hypothesis of Hales & Barker [105]was the first adaptive explanation of an association between early life undernutrition and later life disease. It was developed as a result of the finding that type II diabetes was far more prevalent in men who had been of low birth weight. In response to malnourishment *in utero*, it is proposed that the individual will exhibit a range of metabolic adjustments suited to an environment of nutritional scarcity; these include prioritising the growth of vital organs such as the heart and brain at the expense of less important ones and a greater propensity to lay down fat stores. These adjustments improve survival prospects in the short term but leave the individual at greater risk of a number of chronic conditions - including type II diabetes, cardiovascular disease and obesity - in later life or when the scarcity is lifted. Whilst this describes adaptation to a *nutritional* environment, a similar explanation could perhaps be offered for the acceleration in life history following other kinds of adversity, i.e. the development of a phenotype which improves short-term (juvenile) survival in the adverse environment at the expense of long-term health.

5.1.3 The predictive adaptive response hypothesis

The term 'predictive adaptive response' describes the selection of one of a set of possible life history trajectories which, based on cues received in early life, is predicted to be best suited to the adult environment[53, 102]. If a hostile environment at the start of life provides a reliable indication of hostility to come in the adult environment and thus poor future survival prospects, it may be adaptive to bring forward to time of

maturation, thereby reducing the probability of not surviving to reproduce at all, even at the expense of a faster rate of ageing and poorer health in later life. With respect to the nutritional environment in particular, if scarcity at the start of life predicts scarcity in the adult environment, it may be adaptive to make a series of metabolic adjustments such as increased insulin resistance and a greater propensity to lay down fat. (The idea of a predictive adaptive response is far broader than applies to the behaviours under consideration here and has been invoked to explain variation in a wide range of characteristics in nature.)

This differs from the thrifty phenotype hypothesis outlined above in that it applies to a range of adversities other than malnutrition and, importantly, in that it is the breeding environment which is predicted and to which the phenotype is tailored rather than the short-term future environment. Whether there is sufficient temporal correlation between environments for a predictive adaptive response to evolve in species with long developmental times such as humans is debatable and will obviously depend upon the environmental factor in question. An analysis of annual rainfall in India found no systematic correlation between one year and the next, nor between one year and that ten or forty years into the future (see references in [102]). However, it has been suggested that early life prediction of the reproductive climate could well have evolved during the pre-Holocene period when strong oscillations occurred over the course of thousands of years (see references in [102]). Predictors of other characteristics of the future environment such as disease prevalence and social adversity could also be important. The cues by which social status might be relayed to an infant - such as maternal and paternal investment, stability of the family unit and frequency of residential moves - may be of particular interest since this often seems to be more enduring than the abiotic environment.

In a recent paper by Nettle, Frankenhuis & Rickard[102], the feasibility of the predictive adaptive response hypothesis was demonstrated and an estimate of the environmental correlation between successive years which would be required for this to have evolved in humans was computed. The authors modelled a single environmental variable which changed annually, and of which the optimal adult phenotype was a function. They then determined under what conditions it would be fitter for the organism to commit to a phenotype best suited to the environment of

early life rather than the long-term average environment. It was concluded that a near perfect correlation in successive values of the environmental variable would be required for the evolution of a predictive adaptive response in humans and therefore that, for most environmental characteristics at least, early life prediction of the adult environment is unlikely.

5.1.4 The role of internal state

In the same paper, it was also suggested that deterioration in somatic state rather than the prediction of a poor future environment could explain the acceleration of life history following early life adversity. If an individual whose internal state is weakened as a result of exposure to stress in early life faces reduced long-term survival prospects, it would again be advantageous to bring forward the age of first reproduction so as to reduce the probability of not surviving to reproduce at all. This was termed as internal predictive adaptive response - internal state in early life being supposed to provide a cue for internal state in adulthood - and conceptually this is much the same as the external predictive adaptive response. Both internal state and external environment are aspects of the situation in which an individual finds itself and in relation to which a behavioural strategy may therefore be chosen. It may reasonably be supposed that the individual can sense its own state accurately and that any weakening of state as a result of early adversity could not be fully overcome even if conditions subsequently become more favourable. It is also clear that a genotype will, over the course of evolutionary time, have found itself in individuals of different state so that a selective pressure would exist to tailor life history to state. When the effects of internal state were superimposed upon those of the external environment in the model described above, a predictive adaptive response was far more likely to evolve[102].

5.1.5 Parent-offspring conflict

Life history acceleration has its roots in early life experience when the offspring is heavily dependent upon its mother. The life history strategy selected at this time may therefore be particularly susceptible to maternal effects as well as to effects of the abiotic environment. An offspring can be buffered against adversity by an increased investment from its mother or exposed to greater adversity as a result of reduced

maternal investment. Moreover, the mother's investment decision may reflect not simply the current environment but the cumulative environment to which she has been exposed and her wider family structure. In a series of recent papers, Wells has argued that an understanding of how the maternal phenotype and maternal investment strategy impact the offspring phenotype will be crucial to understanding the early life programming of the speed of life history[103, 106, 107].

An increase in investment in any one offspring will typically cost the mother in terms of expected future reproductive success. As the mother shares the same proportion of her genes with current and future offspring whilst the offspring shares only half its genes with siblings (assuming they share the same father), conflict arises between mother and offspring as to the optimal level of maternal investment. Whilst such conflict arises in both good and bad environments, its degree and ultimate resolution may differ between the two. (If siblings do not share a father, conflict will be greater and in this case there is even conflict between maternally- and paternally-derived genes within the offspring since the former will share half and the latter none of their genetic ancestry with the mother's other offspring.)

The exact degree of conflict between the two will vary with the expected fraction of the mother's reproductive value which is derived from the current offspring. If the mother is in such poor condition that she is unlikely to raise another offspring, this value approaches one and both mother and offspring agree that the mother should invest as much as possible in the current offspring. Conversely, if the offspring is in such poor condition (or juvenile mortality is so high in the current environment) that its chances of surviving to reproduce are very low, this value approaches zero and both mother and offspring agree that it is best for the mother to conserve her resources to invest in other offspring. When the relative fitness prospects of mother and offspring are between these extremes, there is conflict over the degree of maternal resources which the offspring should requisition. Importantly, the expected fraction of the mother's reproductive value which is derived from the current offspring may change under different environments. It will be reduced in an adverse environment which raises juvenile mortality to a greater extent than adult mortality and increased in one which raises adult mortality to a greater extent than juvenile mortality.

This has two implications when we consider how offspring are affected by early adversity. Firstly, if the offspring becomes less valuable to the mother in a certain environment, such that she lowers the proportion of her available resources invested in it, any resultant deterioration in offspring condition and consequent acceleration of life history is attributable to maternal strategy as much as external environment or internal state. Secondly, we should not seek to explain offspring strategy simply in terms of the offspring maximising its own reproductive success since it actually seeks to maximise its own plus that of its mother. In some circumstances, an offspring may commit to a growth trajectory with a shorter period of dependency on it mother and earlier maturation because the improvement in the mother's future reproductive success outweighs the reduction in her own.

That maternal phenotype affects that of the offspring and that there is conflict between mother and offspring are not new ideas, however, that accelerated life history in response to early adversity could be (partially) understood as a maternal adaptation is. As evidence for this, Wells cites studies of previously well-nourished mothers who are subjected to a brief famine whilst pregnant or chronically-malnourished mothers who enjoy a short time of plenty during pregnancy[103, 106]. In both cases, the offspring phenotype seems to depend more strongly upon the overall nutritional status of the mother (i.e. the cumulative environment to which she has been exposed) than upon food availability at the time of pregnancy itself.

5.1.6 Discriminating between hypotheses

The above hypotheses are not mutually exclusive and determining the relative importance of each of them is not straightforward. In some cases at least, a predictive adaptive response may be ruled out on the grounds of too weak a temporal correlation between environments, as was the case for rainfall in India. However, even in cases where a sufficiently strong correlation does exist and the life history trajectories chosen in early life do seem to be matched to the breeding environment, it might be hard to conclude that a predictive adaptive response had evolved. Correlation between early and later juvenile environments would necessarily also be strong and if the phenotype seemed suited to this environment as well it is hard to say which is being predicted.

It has been suggested that the real test of the predictive adaptive response hypothesis is whether an individual who has experienced an adverse environment throughout life (and whose phenotype should therefore be matched to the adult environment) is fitter than one who a) experienced adversity in early life and advantage as an adult or b) experienced advantage in early life and adversity as an adult (both of these then being mismatched to their environments)[53]. If there are no effects of state, then a) would hold; however, it is possible that a bad start weakens an individual so comprehensively that it's maximal reproductive success even when following an optimal strategy cannot meet that of one who has had a good start and does not follow an optimal strategy. The true test should then be whether those who experience adversity throughout life would be less fit if they followed the strategy of one who experiences advantage in early life. By a similar line of reasoning b) also need not hold; an advantageous adult environment may so comprehensively improve survival or reproductive prospects that all individuals in such an environment will, regardless of their strategy, enjoy a greater reproductive success than those in an adverse adult environment.

Disentangling the effects of state versus environment and of maternal versus offspring adaptation requires quantitative modelling of the fitness consequences, for both mothers and offspring, of different life history strategies under different environments. The model presented below has been constructed to this end. This model is, to my knowledge, only the second to apply dynamic programming to the decision processes of both parents and offspring, (the first being a model developed by Clark & Ydenberg of parental feeding decisions and offspring fledging decisions in the dovekie[108]).

5.2 Aims

1. To determine what is the optimal response - in terms of speed of life history - to fluctuating food supply in humans.

2. To determine whether any association between early life adversity and accelerated life history can be best explained as an adaptation to the predicted future environment or as a consequence of the deterioration in state incurred in a harsh environment.

3. To compute a lower bound for the cost, in terms of fitness, of maintaining plasticity throughout life. This is given by the reduction in expected fitness associated with predicting the future environment and constraining life history accordingly early in development rather than allowing both allocation strategies and predictions of the future environment to be updated each period.

4. To explore the effect of conflict between mother and offspring upon the speed of life history in different environments. Are there particular environments or particular states in which conflict is enhanced or reduced?

5. To explore the sensitivity of the model to changes in parameter values. How does the value of prediction change with the correlation between environmental states in successive years? Are there certain situations in which current state is most important in explaining accelerated life history and others where environmental prediction is?

5.3 Methods

5.3.1 Model overview

5.3.1.1 State dynamics

Consider first the maternal decision process, shown in figure 5.1 (top). At time t, an adult female is defined by her condition (c_m). She is subject to a mortality threat (β) the magnitude of which depends upon her condition. If she survives this, she achieves a foraging yield (ξ), the magnitude of which depends upon the current foraging environment and a given amount of which must be diverted to essential functions: those which must be met in order to survive the current period. If she has no dependent offspring at the start of the period, she must choose how to distribute the remainder of her foraging yield between reproduction and maintenance. This decision may depend upon the current environment; her condition and; whether the environment she experienced in early life constrains her subsequent allocation strategy. If she has a dependent offspring at the start of the period, she must maintain the same rate of ageing (i.e. the same investment in maintenance) that was chosen at the start of that offspring's life. Since the foraging environment may change over this time, allocation to the offspring may therefore also change. It is assumed that an offspring will be dependent upon the mother for three years from conception and that the mother may not care for multiple offspring simultaneously.

The state of any dependent offspring at (t + 1) depends on both the maternal allocation to reproduction and the allocation decision at time t of the offspring. The mother's condition (c_m') at time (t + 1) is determined by her investment in maintenance. She is, at time (t + 1), again faced with a particular foraging environment (which may or may not be the same as at time t - see below) and must again distribute her available energy budget, after the costs of essential functions have been met, between reproduction and maintenance.

Consider now the decision process for an offspring in receipt of energetic resources from their mother (figure 5.1 top). At time t, she is defined by her size (s) and condition (c_o). She is subject to a mortality threat (β) the magnitude of which depends upon her condition and age, the latter age-dependent component to mortality risk being included to describe early-life mortality. If she survives this, she will distribute

the energetic resources invested in her by her mother minus the cost of essential functions between growth and maintenance. In her first period of life she may make this allocation decision freely, taking into account the current foraging environment; for the remaining two periods that she is dependent upon her mother she must maintain the same rate of ageing that was chosen in the first period of life.

Finally, consider an offspring living independently of her mother (figure 5.1 bottom) who is again defined at time t by her size (s) and condition (c_o). She is subject to a condition-dependent mortality threat (β). If she survives this, she obtains a foraging yield (ξ) the magnitude of which depends upon her size and environment and the remainder of which, after the costs of essential functions have been met is distributed between growth and maintenance. Her allocation decision may depend upon the current environment; her size and condition and; whether the environment she experienced in early life constrains her subsequent allocation strategy.

An offspring's investment in growth and maintenance at time t determines respectively her size (s') and condition (c_o') at time (t + 1) when she will again be faced with a particular foraging environment and must again distribute her available energy budget, after the costs of essential functions have been met, between growth and maintenance. A greater investment in growth allows her to mature sooner and start reproduction at a younger age; however, this comes at the expense of more rapid deterioration in condition and consequently reduced long-term survival. All individuals mature at the same size, and size remains constant throughout adulthood.

There are two foraging environments in which individuals may find themselves. These environments and the probabilities of transitioning between them are shown in figure 5.2; the good environment (the high foraging environment) is prefixed with a 1 and the bad environment (the low foraging environment) is prefixed with a 2. It is assumed that both mother and offspring can accurately sense the state of the environment and know the transition probabilities between environmental states.

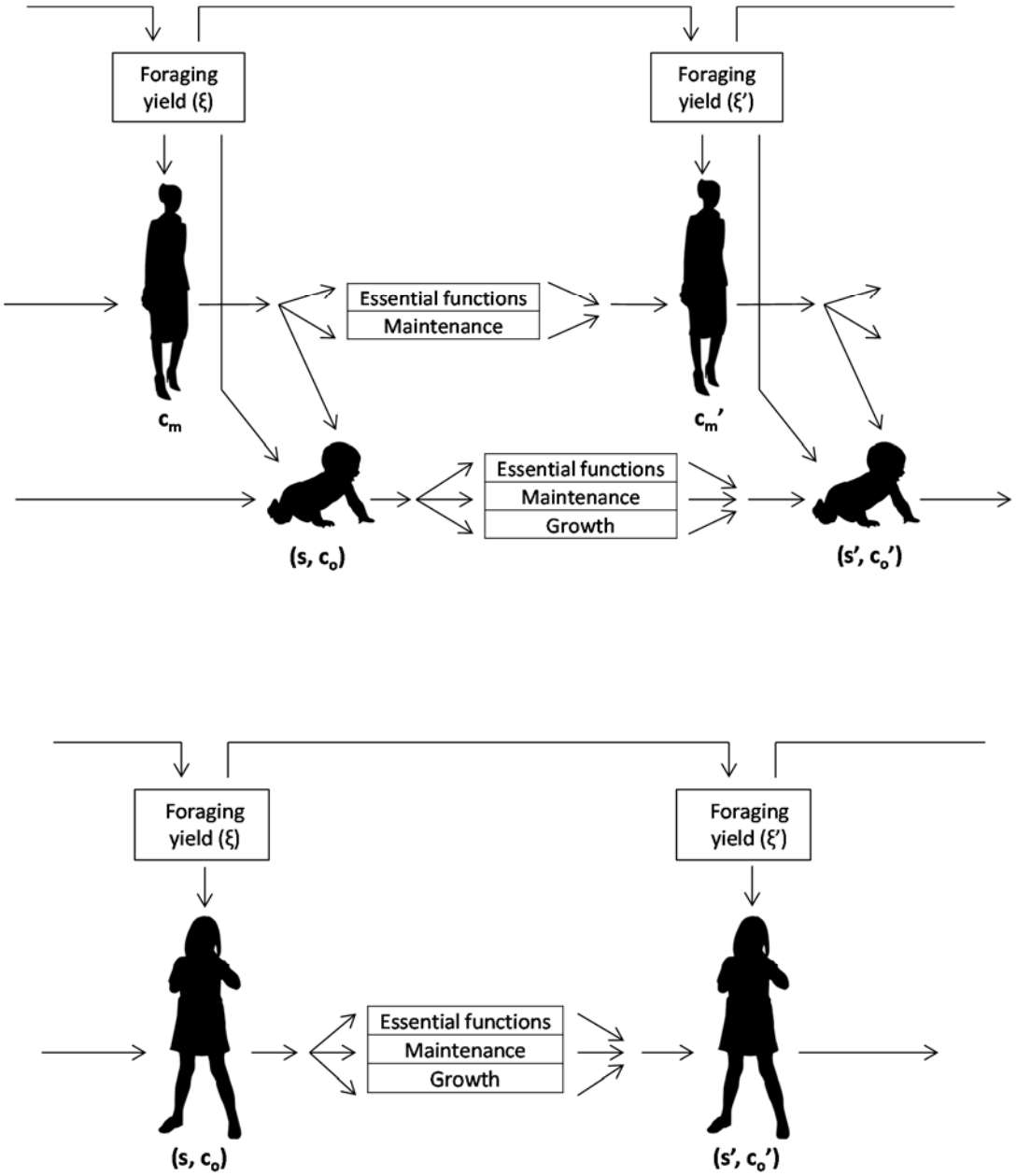

Figure 5.1: Schematic representation of the decision process for mothers and dependent offspring (top) and for offspring living independently (bottom). Maternal condition, offspring size and offspring condition at time t are represented by c_m, s and c_o respectively and at time (t + 1) by c_m', s' and c_o'. Foraging yield at time t is represented by ξ_t and at time (t + 1) by ξ_{t+1}'.

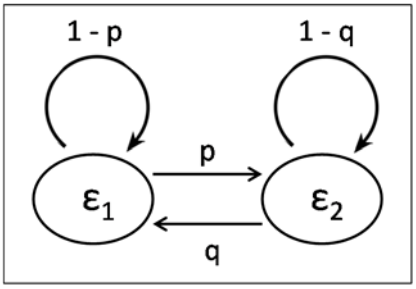

Figure 5.2: Schematic representation of the transitions between environmental states. Good and bad foraging environments are represented by ε_1, and ε_2, respectively. The probabilities of experiencing each of these environments at time (t + 1), given that they are experienced at time t, are given by (1 - p) and (1 - q) respectively.

5.3.1.2 Calculation of optimal strategy under different assumptions

By comparing simulations in which the optimal strategy is calculated in different ways, we can tease apart the effects of state and environmental prediction in explaining any acceleration in life history following early life adversity and determine the cost which must be associated with maintaining plasticity if a strategy of commitment to a given trajectory early in life is to evolve. Simulations will be compared in which the optimal strategy is calculated in the four ways listed below.

1) Plasticity is retained throughout life and no prediction of future environmental state is made.

   In each period, allocation strategy may be chosen freely by all independent offspring and by mothers who conceive at the start of the period and their dependent offspring; there is no constraint imposed by earlier decisions. Strategies may therefore be altered in response to both the current environment and to state. It is always assumed that the probability of being in a given environment in the next period is equal to the long-term probability of being in that environment; the current environment is not used to predict the future one.

2) Allocation strategy is fixed in early life and no prediction of future environmental state is made.

   An organism chooses an allocation strategy based on the investment her mother makes in her during the first period of life. Her strategy is the rate at

which she ages and this is then fixed throughout the rest of her life. In all subsequent periods she will age at the same rate regardless of the environment in which she finds herself, and the remainder of her available energy budget (after the relevant amount is invested in maintenance) goes towards growth during development and reproduction during adulthood. Although she is aware of her environment in the first period of life, she does not use this information to predict any deviation in the environments she will experience subsequently from the long-term average.

3) Plasticity is retained throughout life and current environmental state is used to predict that in the future.

   Allocation strategy can vary not only with current state and environment but also with predicted deviations of the future environment from the long-term average. Plasticity is maintained throughout development and adulthood; prediction of the future environment is updated at each time period and allocation strategy - for all independent offspring and all mothers who conceive at the start of the period and their dependent offspring - is adjusted accordingly.

4) Allocation strategy is fixed in early life and a prediction of future environmental state is made based on that experienced in the first period of life.

   An organism chooses an allocation strategy based on the investment her mother makes in her during the first period of life and predicted deviations of the future environment from the long-term average based on the environment she experiences in the first period of life. Her strategy is the rate at which she ages and this is then fixed throughout the rest of her life.

When prediction about future environment is not made (1 and 2 above), any change in the pace of the life history in response to adversity is explained solely in terms of a change in state at the time when adversity is experienced. By comparing model output from these cases with those from 3 and 4 we can see the effect of prediction on speed of life history over that of state alone. By comparing the results of simulations where plasticity is maintained to those where the life history is constrained we can infer the costs which maintaining plasticity must entail if constraint in early life is to evolve.

5.3.2 Model structure

As for the model in chapter 4, fitness is defined as the expected number of copies of an individual's genes at some time point far enough into the future such that the optimal strategy is independent of time. Time periods between successive decisions are of length one year.

5.3.2.1 State space

An adult female is described by the state variable c_m, this being her condition in time periods of one year. An offspring (juvenile female) is described by the state variable (s, c_o) where s is her size in kg and c_o is her condition in time periods of one year.

Condition may take the values 0, 1, 2, …, 45 years. At a condition of 45 years the individual is no longer able to survive. Since investment over three periods is required for an offspring to reach an age at which she can survive without her mother, this means that the highest condition at which a mother can conceive and the offspring have a chance of survival is 42.

Juvenile size may take the values 0, 1, 2, …, 60kg. Maturity is reached at the maximum size of 60kg and this is then maintained throughout adult life. The adult mass of 60kg represents a healthy size for a female of average height.

As already stated, there are two foraging environments in which an individual may find herself. The state space for an offspring living independently of her mother is of size $|s|*|c_o|*|\varepsilon| = 61*46*2 = 5612$, where $|y|$ represents the size of dimension y. The state space for mothers and dependent offspring is of size $|s|*|c_o|*|c_m|*|\varepsilon| = 61*46*46*2 = 258152$.

5.3.2.2 Notation used in dynamic programming equations

The following notation is used for biological functions, investment decisions, changes to state and reproductive values.

Biological functions:

- $\beta(c_m)$ is the per-period mortality risk for a mother who is at the start of the period of condition c_m.

- $\beta(a, c_o)$ is the per-period mortality risk for an offspring who at the start of the period is of age a and condition c_o. Note that for ages of three or more, the age-dependent component of mortality risk is zero.
- $\eta(s_m)$ is the cost of essential functions over one period for an adult.
- $\eta(s, s')$ is the cost of essential functions over one period for an offspring of size s at the start of the period and size s' at the end of the period.
- $\xi(\varepsilon_t)$ is the energetic resources a mother obtains from an environment ε_t.
- $\xi(s, s'; \varepsilon_t)$ is the energetic resources an offspring is able to obtain over one period from an environment ε_t given that she is of size s at the start of the period and size s' at the end of the period.
- $\Omega(s; \varepsilon_t, \alpha)$ is the maximum growth per period for an offspring who is of size s at the start of the period, given that she experiences environment ε_t and receives energetic resources α from her mother. (In the case that the offspring is living independently, $\alpha = 0$.)
- s_m is the size at which maturity is reached. As stated above s_m = 60kg.

Investment decisions:

- $\alpha(c_m; \varepsilon_t)$ is the investment a mother in condition c_m makes in an offspring conceived at the start of period t, in an environment ε_t.
- $\gamma(s, c_o; \alpha, \varepsilon_t)$ is the investment an offspring of size s and in condition c_o makes in growth, given that she experiences environment ε_t and receives resources α from her mother. (In the case that the offspring is living independently, $\alpha = 0$.)

Changes in state:

- $c_m'(c_m; \alpha, \varepsilon_t)$ is the condition of a mother at the start of period (t + 1) given that she started period t in condition c_m, and that during period t she experiences environment ε_t and invests α in an offspring.
- $c_m''(c_m; \alpha, \varepsilon_t, \varepsilon_{t+1})$ is the condition of a mother at the start of period (t + 2) given that she started period t in condition c_m, experiences environments ε_t in period t and ε_{t+1} in period (t + 1) and invests α in an offspring during period t.
- $c_m'''(c_m; \alpha, \varepsilon_t, \varepsilon_{t+1}, \varepsilon_{t+2})$ is the condition of a mother at the start of period (t + 3) given that she started period t in condition c_m, experiences environments ε_t,

ε_{t+1} and ε_{t+2} in periods t, (t + 1) and (t + 2) respectively and invests α in an offspring during period t.

- $c_m''''(s, c_o; \varepsilon_t, \gamma)$ is the condition of a newly-matured mother at the start of period (t + 1) given that she started period t in condition c_o and of size s, and that during period t she experiences environment ε_t and she invests γ in growth.
- $c_o'''(\alpha, \varepsilon_t, \varepsilon_{t+1}, \varepsilon_{t+2})$ is the condition of an offspring at the start of period (t + 3) given that she is conceived at the start of period t, experiences environments ε_t, ε_{t+1} and ε_{t+2} in periods t, (t + 1) and (t + 2) respectively and that her mother invests α in her during period t.
- $s'''(\alpha, \varepsilon_t, \varepsilon_{t+1}, \varepsilon_{t+2})$ is the size of an offspring at the start of period (t + 3) given that she is conceived at the start of period t, experiences environments ε_t, ε_{t+1} and ε_{t+2} in periods t, (t + 1) and (t + 2) respectively and that her mother invests α in her during period t.
- $c_o'(s, c_o; \varepsilon_t, \gamma)$ is the condition of an offspring at the start of period (t + 1) given that she started period t in condition c_o and of size s, was living independently of her mother at the start of period t and, that during period t she experiences environment ε_t and invests γ in growth.
- $s'(s, c_o; \varepsilon_t, \gamma)$ is the size of an offspring at the start of period (t + 1) given that she started period t in condition c_o and of size s, was living independently of her mother at the start of period t and, that during period t she experiences environment ε_t and invests γ in growth.

Reproductive values:

Reproductive value is defined as the expected number of copies of an individual's genes at some time horizon far into the future which result from descendants the individual raises in the current period onwards.

- $F(c_m, t, T; \varepsilon_t)$ is the reproductive value of a mother in condition c_m, at time t prior to the time horizon T, given that she has no existing dependent offspring at the start of period t and that she experiences environment ε_t.
- $F(0, 0, t, T; \alpha, \varepsilon_t)$ is the reproductive value of an offspring conceived at time t prior to the time horizon T, given that her mother invests α in her during period

t and that she experiences environment ε_t. (The two zeros refer respectively to her size and condition at the start of period t.)

- $F(s, c_o, t, T; \varepsilon_t)$ is the reproductive value of an offspring of size s and in condition c_o, at time t prior to the time horizon T, given that she is living independently and experiences environment ε_t.

5.3.2.3 Dynamic programming equations if the optimal allocation strategy is updated each period

The dynamic programming equation for an offspring living independently of her mother at the start of period t is given by:

$$\mathbf{F(s, c_o, t, T; \varepsilon_t) = (1 - \beta(a, c_o))^* \max_{\gamma=0}^{\Omega} \{ E_{\varepsilon t+1} F(s', c_o', t + 1, T; \varepsilon_{t+1}) \text{ if } s' < s_m;}$$
$$\mathbf{E_{\varepsilon t+1} F(c_m'''', t + 1, T; \varepsilon_{t+1}) \text{ if } s' = s_m \}}$$

This can be understood as follows. The first term in this equation - $(1 - \beta(a, c_o))$ - gives survival probability for the current period. This is multiplied by the offspring's maximum expected reproductive value at the start of the next period, the maximisation being taken over all possible allocation strategies and the expectation over all possible environments. If she remains a juvenile at the start of period (t + 1), her expected reproductive value will be given by the first term in parentheses: $E_{\varepsilon t+1} F(s', c_o', t + 1, T; \varepsilon_{t+1})$. If her investment in growth is sufficient for her to reach maturity during period t, her reproductive value at the start of period (t + 1) is given by the second term in parentheses: $E_{\varepsilon t+1} F(c_m'', t + 1, T; \varepsilon_{t+1})$.

The dynamic programming equation for a mother is given by:

$$\mathbf{F(c_m, t, T; \varepsilon_t) = (1 - \beta(c_m))^* \max_{\alpha=0}^{\xi-\mu} \{A + B + C + D\}}$$

where $\mathbf{A = \beta(0, 0)^* E_{\varepsilon t+1} \{F(c_m', t + 1, T; \varepsilon_{t+1})\}}$

$$\mathbf{B = (1 - \beta(0, 0))^* (1 - \beta(c_m')^* \beta(1, 0')^* E_{\varepsilon t+1, \varepsilon t+2} \{F(c_m'', t + 2, T; \varepsilon_{t+2})\}}$$

$$\mathbf{C = 1 - \beta(0, 0))^* (1 - \beta(c_m')^* (1 - \beta(1, 0'))^* (1 - \beta(c_m''))^* \beta(2, 0'')^*}$$
$$\mathbf{E_{\varepsilon t+1, \varepsilon t+2, \varepsilon t+3} \{F(c_m''', t + 3, T; \varepsilon_{t+3})\}}$$

$$\mathbf{D = (1 - \beta(0, 0))^* (1 - \beta(c_m')^* (1 - \beta(1, 0'))^* (1 - \beta(c_m''))^* (1 - \beta(2, 0''))^*}$$
$$\mathbf{E_{\varepsilon t+1, \varepsilon t+2, \varepsilon t+3} \{F(c_m''', t + 3, T; \varepsilon_{t+3}) + 0.5^* F(0''', 0''', t + 3, T; \varepsilon_{t+3})\}}$$

The reproductive value of a mother at the start of period t equals the probability of her surviving the current period multiplied by her maximum expected subsequent reproductive value with the maximisation taken over possible allocation strategies adopted in the current period and the expectation taken over possible subsequent environments. Expected subsequent reproductive value is defined for four scenarios (A - D), which weighted according to the probability of its occurrence. A gives the probability that the offspring does not survive the current period multiplied by the expected reproductive value of the mother at the start of period (t + 1). B gives the probability of the mother surviving period (t + 1) multiplied by the probability that the offspring survives period t but does not survive period (t + 1) multiplied by the expected reproductive value of the mother at the start of period (t + 2). C gives the probability that the mother survives until the start of period (t + 3) multiplied by the probability that the offspring dies in period (t + 2) multiplied by the expected reproductive value of the mother at the start of period (t + 3). D gives the probability that both mother and offspring survive until the start of period (t + 3) multiplied by the expected reproductive value of the mother plus half the expected reproductive value of the offspring at (t + 3). Under any scenario where the mother dies before the start of period (t + 3), her expected reproductive value is zero.

The terms $F(0''', 0''', t + 3, T; \varepsilon_{t+3})$, $\beta(1, 0')$ and $\beta(2, 0'')$ will all depend upon the offspring's decision. This is calculated, for all possible maternal decisions, under the assumption that the offspring will always act so as to maximise her own reproductive value. When the mother chooses her allocation decision, she does so with a knowledge of the decision her offspring will make in response to hers.

The dynamic programming equation for an offspring at the time of conception is given by:

$$\mathbf{F(0, 0, t, T; \alpha, \varepsilon_t) = (1 - \beta(0, 0))^* \max_{y=0}^{\Omega}\{[(1 - \beta(c_m'))^*(1 - \beta(1, 0'))^*(1 - \beta(c_m''))^* (1 - \beta(2, 0''))^* E_{\varepsilon t+1, \varepsilon t+2, \varepsilon t+3}[F(0''', 0''', t + 3, T; \varepsilon_{t+3})]}$$

Her reproductive value at time t is the product of the probability that she will survive the current period - $(1 - \beta(0, 0))$ - and the maximum probability of subsequently surviving to independence multiplied by her expected reproductive value at the time

of independence. As before, the maximisation is taken over possible allocation strategies and the expectation taken over possible future environments.

Terminal reproductive values for mothers and independent offspring are given by the following:

$F(c_m, T, T; \varepsilon_T) = 1$ **if** $c_m < 45$

$= 0$ **if** $c_m = 45$

$F(s, c_o, T, T; \varepsilon_T) = 1$ **if** $s > 0$ **and** $c_o < 45$

$= 0$ **if** $s = 0$ **and/or** $c_o = 45$

Note that no terminal reproductive values for dependent offspring need to be defined as these are not required for the calculation of reproductive values prior to the time horizon. However, since reproductive values three time periods into the future are used in the dynamic programming equations above, these must also be defined for periods (T + 1) and (T + 2) in order for the algorithm to run but these are simply set to zero.

$F(c_m, T + 1, T; \varepsilon_{T+1}) = 0$

$F(c_m, T + 2, T; \varepsilon_{T+2}) = 0$

$F(s, c_o, T + 1, T; \varepsilon_{T+1}) = 0$

$F(s, c_o, T + 2, T; \varepsilon_{T+2}) = 0$

5.3.2.4 Dynamic programming equation if allocation decisions are constrained in early life

When allocation decisions are constrained in early life, a dynamic programming equation is only required for an offspring at conception; the allocation strategy chosen in this first period of life (specifically the rate of ageing) will then be followed throughout the rest of life. For all organisms at least one period old, reproductive values are calculated within the backward iteration as a function of strategy (σ_i) according to the function below but no longer depend on taking a maximisation over a range of allocation decisions.

The dynamic programming equation for an offspring at conception is given by:

$$F(0, 0, t, T; \alpha, \varepsilon_t) = (1 - \beta(0, 0)) * \max_{\sigma i}\{[(1 - \beta(c_m'))*(1 - \beta(1, 0'))*(1 - \beta(c_m''))* (1 - \beta(2, 0''))* E_{\varepsilon t+1, \varepsilon t+2, \varepsilon t+3}[F(0''', 0''', t + 3, T; \sigma_i, \varepsilon_{t+3})]$$

The reproductive value of an offspring living independently of her mother is given by:

$$F(s, c_o, t, T; \sigma_i, \varepsilon_t) = (1 - \beta(a, c_o))*\{ E_{\varepsilon t+1} F(s', c_o', t + 1, T; \sigma_i, \varepsilon_{t+1}) \text{ if } s' < s_m;$$

$$E_{\varepsilon t+1} F(c_m'''', t + 1, T; \sigma_i, \varepsilon_{t+1}) \text{ if } s' = s_m \}$$

The reproductive value of a mother is given by:

$$F(c_m, t, T; \sigma_i, \varepsilon_t) = (1 - \beta(c_m))* \max_{\alpha=0}^{\xi-\mu} \{A + B + C + D\}$$

where $A = \beta(0, 0)*E_{\varepsilon t+1} \{F(c_m', t + 1, T; \sigma_i, \varepsilon_{t+1})\}$

$B = (1 - \beta(0, 0))* (1 - \beta(c_m')*\beta(1, 0')*E_{\varepsilon t+1, \varepsilon t+2} \{F(c_m'', t + 2, T; \sigma_i, \varepsilon_{t+2})\}$

$C = 1 - \beta(0, 0))* (1 - \beta(c_m')*(1 - \beta(1, 0'))* (1 - \beta(c_m''))*\beta(2, 0'')*$

$E_{\varepsilon t+1, \varepsilon t+2, \varepsilon t+3} \{F(c_m''', t + 3, T; \sigma_i, \varepsilon_{t+3})\}$

$D = (1 - \beta(0, 0))* (1 - \beta(c_m')*(1 - \beta(1, 0'))* (1 - \beta(c_m''))*(1 - \beta(2, 0''))*$

$E_{\varepsilon t+1, \varepsilon t+2, \varepsilon t+3} \{F(c_m''', t + 3, T; \sigma_i, \varepsilon_{t+3}) + 0.5*F(0''', 0''', t + 3, T; \sigma_i, \varepsilon_{t+3})\}$

Terminal reproductive values are given by:

$F(c_m, T, T; \sigma_i, \varepsilon_T) = 1$ if $c_m < 45$

$= 0$ if $c_m = 45$

$F(s, c_o, T, T; \sigma_i, \varepsilon_T) = 1$ if $s > 0$ and $c_o < 45$

$= 0$ if $s = 0$ and/or $c_o = 45$

$F(c_m, T + 1, T; \sigma_i, \varepsilon_{T+1}) = 0$

$F(c_m, T + 2, T; \sigma_i, \varepsilon_{T+2}) = 0$

$F(s, c_o, T + 1, T; \sigma_i, \varepsilon_{T+1}) = 0$

$F(s, c_o, T + 2, T; \sigma_i, \varepsilon_{T+2}) = 0$

5.3.2.5 Decision set

Consider first the case when allocation strategy may be updated each period. An adult female with no existing dependent offspring has an available energy budget equal to her foraging yield minus the cost of essential functions. She must choose from 20 possible ways in which this budget may be split between reproduction and maintenance. Specifically, allocation to reproduction may be the integer quotient of x multiplied by the available energy budget and 19, for x = 0, 1, …, 19. No special significance is attached to the number of potential allocation decisions; they are simply sufficient to capture any differences of interest between individuals in different states but not so many as to make the backward iteration computationally unfeasible.

The decision set for an offspring is a little harder to define. She will have either the investment received from her mother (if dependent) or her own foraging yield (if independent) minus the cost of essential functions to split between growth and maintenance. However, this is not a fixed value since foraging yield and the cost of essential functions are themselves dependent on her allocation to growth; per-period values for each of these are calculated as the mean of that associated with size at the start of the period and that with size at the end of the period. The maximum growth that the offspring may sustain is therefore calculated by implementing the following algorithm.

- Lower and upper bounds on maximum growth are initially defined as [3.5, 9]kg in the first two periods of life and [1.5, min(7, 60 - s)]kg thereafter. These bounds constitute constraints within the model however they should not artificially restrict behaviour as they lie outside the 5^{th} and 95^{th} weight percentiles for girls of the relevant ages. As an offspring approaches maturity, the difference between the adult mass of 60kg and the offspring mass at the start of a period (s) may be less than 7kg and this difference then provides an initial upper bound on maximum growth instead.
- If this upper bound on growth is feasible - that is, if foraging yield (given this growth strategy) plus any maternal investment minus the cost of essential functions (given this growth strategy) minus the cost of growth itself leaves a non-negative investment for maintenance - then this is the maximum possible growth.
- If this upper bound on growth is not feasible, then the midpoint between the lower and upper bounds is considered.
- If the midpoint corresponds to a feasible growth strategy then it replaces the lower bound for maximum growth; if it does not then it replaces the upper bound.
- If the difference between the upper and lower bounds corresponds to a difference in the energetic cost of growth of less than or equal to 20kcal per period, the lower bound is taken as the maximum possible growth. As 20kcal is a tiny difference in energetic investment over the course of a year, it can be assumed at this point that maximum possible growth has been sufficiently well

approximated. If the difference between the upper and lower bounds corresponds to a difference in the energetic cost of growth of greater than 20kcal, the midpoint between the two bounds is computed and the previous step returned to.

Possible allocations to growth are then defined as the integer quotients of x multiplied by the maximum possible growth (in kg) multiplied by the energy required to put on 1kg (Ψ - see section 5.3.3.6 below) and 19, for x = 0, 1, …, 19. Investment in maintenance in each case is calculated as the foraging yield (given the growth strategy) or the maternal investment minus the cost of essential functions (given the growth strategy) minus the cost of growth.

Now consider the case when allocation strategy is constrained in early life. At conception, an offspring must choose between 20 possible ways in which energy may be split between growth and maintenance, in the same way as described above. The rate of ageing associated with her investment in maintenance during this first period is then fixed throughout the rest of her life.

The model was coded - according to the specification which has been outlined above - in Python. As the backward iterations were run, the difference between the strategies followed by an individual in state X at one time point and the next was calculated for all X. When the average of these differences (taken over all possible states in which individuals could be) corresponded to a differential investment of less than 100kcal/period, the simulation was terminated. This was achieved after 120 time points.

5.3.3 Model parameterisation

5.3.3.1 Mortality rates

Three mortality functions are defined within the model: a condition-dependent function experienced throughout life; an age-dependent function experienced in the first three periods of life and; a maternal mortality function experienced in periods when a mother births an offspring. These mortality risks are additive.

Condition-dependent mortality rates are based upon data for females in England and Wales in 1850[111]. Since these published data treat age at birth as zero whilst in the

model the offspring is of condition zero at conception, I first interpolated between the published annual mortality rates to obtain estimates for mortality corresponding to ages used in the model. So, mortality in the second year of life post-conception is estimated as three quarters that in the first year post-birth plus one quarter that in the second year post-birth and so on. Gompertz mortality coefficients were then fitted to data from ages 40-80 post-conception. Although the model only considers conditions up to 45, more accurate estimates for coefficients can be obtained by including data from older ages since Gompertz mortality is adhered to most closely from 40 years onwards. Figures are available for ages up to 110 but data from the oldest ages are not used because sample sizes are smaller and there is some evidence for reduced mortality at the oldest ages. Data for ages below 40 are not used because total mortality rate is heavily affected by maternal mortality and early-life mortality which are modelled as separate mortality hazards. The following function was obtained for condition-dependent mortality:

$\mu(c) = 0.0015*\exp(0.052*c)$

Consider next the function for early-life age-dependent mortality (v(a)). Mortality *in utero* is termed a miscarriage if it occurs prior to 24 weeks gestation (the age at which a foetus could survive independently) and a stillbirth if it occurs after 24 weeks gestation. Estimates of between 0.15-0.20 have been given for miscarriage rates in women who are known to be pregnant, although total miscarriage rates will necessarily be higher than this[109]. Rates of stillbirth in developing countries, where access to antenatal care is limited, are approximately an order of magnitude lower[110]. In the model, a total mortality rate of 0.2 *in utero* was assumed. For simplicity, this was assumed independent of the condition of the mother.

Age-dependent mortality in the first period of life was set at the probability of dying in utero plus the residual mortality rate - after condition-dependent mortality rates as described by the above equation have been discounted - for the first three months post-birth as calculated for the dataset from 1850. Age-dependent mortality risks for the second and third periods of life were simply set as the residual mortality rates from the relevant period.

Maternal mortality - the risk of a women dying from pregnancy-related causes - is considered as a separate mortality hazard (ς). Based on records from 13 English parishes in the period 1800-1850, the probability of maternal mortality is estimated as 0.005[113]. For simplicity this is assumed to be independent of maternal age.

Using the notation for the dynamic programming equations above, we have:

$\beta(c_m) = \mu(c_m) + \varsigma$ if a new offspring is conceived

$= \mu(c_o)$ otherwise

$\beta(a, c_o) = \mu(c_m) + \nu(a)$

5.3.3.2 Foraging yield

Foraging yield is assumed to be dependent on size (see figure 5.4) and independent of condition. For an adult therefore it is dependent only on the foraging environment in which the individual finds herself. In foraging environment 1 she obtains 2100kcal/day equivalent to ξ(1) = 766500kcal/year; in foraging environment 2 she obtains 1800kcal/day equivalent to ξ(2) = 65700kcal/year.

For a juvenile, foraging yield for a given size (s) is given, in kcal/day, by:

$170*s^{0.6}$ in environment 1 and;

$145*s^{0.6}$ in environment 2

These are increasing functions in size with diminishing returns, similar in shape to what is reported in the literature. As a juvenile's size changes over the course of a period, the foraging yield for the period t is estimated as:

$\xi(s, s'; 1) = 365*170*[s^{0.6} + s'^{0.6}]/2$

$\xi(s, s'; 2) = 365*145*[s^{0.6} + s'^{0.6}]/2$

where s is offspring size at the start of period t and s' is offspring size at the start of period (t + 1).

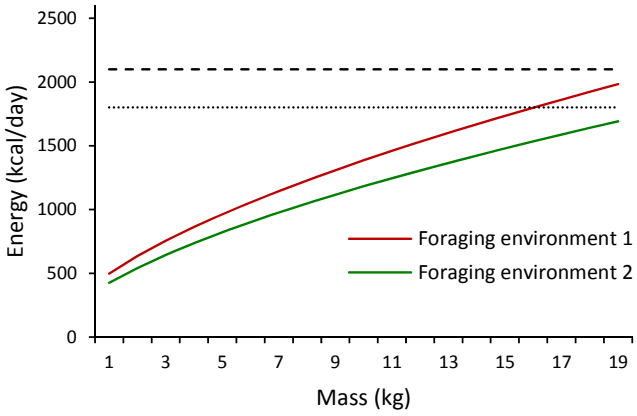

Figure 5.3: Size-dependent daily foraging yields in two environments. Yields for juveniles are indicated by the red and green lines for foraging environments one and two respectively. Adult foraging yields are indicated by the dotted lines, the heavier representing foraging environment one and the lighter foraging environment two.

5.3.3.3 The cost of essential functions

As for the model described in chapter 4, the costs of vital metabolism - those required for immediate survival - are estimated as half the BMR (with the other half typically going to somatic maintenance) and BMR is estimated as $70*W^{0.75}$ per day where W is body mass in kg.

It is reasonable to suppose that some energy must be expended on activities such as travel to foraging sites, seeking shelter and fuel etc. and that this will be required to survive the current period. It is therefore assumed that all individuals over three periods old will expend a certain amount - 365*5*W - on exercise. Estimates for energy expended in walking a set distance in a set time are generally proportional to weight and for a 60kg woman this is approximately equivalent to walking three/four miles (expending 300 kcal) per day. It is assumed that offspring under three periods will usually be carried by their mothers and thus have no exercise-related expenditure of their own.

For an adult the per-period cost of essential functions is therefore given by:

$\eta(s_m) = 365*(70*0.5*60^{0.75} + 5*60) \approx 384907$

For dependent and independent offspring, whose size will change over the course of a period, the costs of essential functions are given respectively by:

$\eta(s, s') = 365(*70*0.5* (s^{0.75} + s'^{0.75})/2)$

$\eta(s, s') = 365(*70*0.5* (s^{0.75} + s'^{0.75})/2 + 5*(s + s')/2)$

where s is offspring size at the start of period t and s' is offspring size at the start of period (t + 1).

5.3.3.4 Rate of ageing

As for the model described in chapter 4, change in age per period ($\phi(k)$) is modelled as:

$\phi(k) = (0.5*BMR/k)^{\Delta}$

with the exponent Δ initially set to one and the effect of changing this being explored by means of sensitivity analysis. As before, this assumes that individuals usually invest approximately half their BMR in somatic maintenance and that when they do so biological and chronological ageing rates are equal (i.e. increments in the state variables c_o and c_m align with time).

5.3.3.5 Transition probabilities

Transition probabilities will obviously be crucial in determining the value of any prediction of the future environment. Figure 5.5 shows the probability of experiencing the same environment at any given age as was experienced at conception for a range of transition probabilities. These curves were plotted under the assumption that transition probabilities are symmetric; to use the notation from figure 4.3, p = q. If the transition probability is 0.5, it is equally likely that either environment will be experienced in the next period and there is therefore no value to predicting the future environment based on the current one. For reasonably modest reductions in the transition probability (0.4 - 0.2), expected future environment returns to the long-term average rather quickly; there may be some benefit to predicting the environment experienced during early life but there can be no value to predicting the adult environment. Only for very low transition probabilities (≥ 0.02) is a significant deviation from the long-term average expected to persist until the end of the reproductive lifespan.

Initially it was assumed that the probability of experiencing the same environment in period (t + 1) as in period t is 0.95, i.e. p = q = 0.05. However, there is no special

significance attached to this value and the impact on behaviour of changing it will be explored via sensitivity analysis.

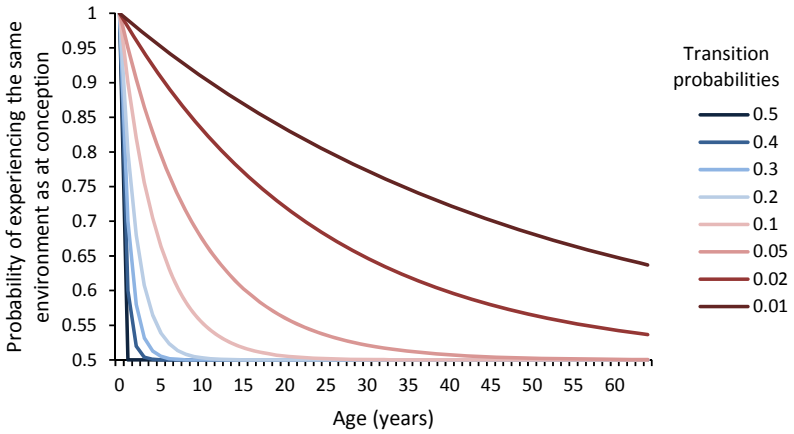

Figure 5.4: The probability of experiencing the same environment as at the time of conception as a function of age and the correlation between one time point and the next. It is assumed that all transition probabilities are symmetric, i.e. if the probability of experiencing environment 1 in the next period given that environment 2 is experienced in the current period is w, then the probability of experiencing environment 2 in the next period given that environment 1 is experienced in the current period is also w.

5.3.3.6 Conversion of energy to biomass

As for the model in chapter 4, an energy density of 7kcal/g, or 7000kcal/kg, is assumed and the efficiency with which energy is converted to biomass is taken to be 80%. (Justification for these figures is given in section 4.3.3.7.) Change in size as a function of energy invested in growth (γ) is therefore given by:

$\Psi(\gamma) = \gamma * 0.8/7000 = \gamma/8750$

Note that weight loss is not permitted in this model so change in size must always be non-negative.

5.4 Results

5.4.1 The effects of state-dependent decision making and prediction of the future environment upon fitness

The stable strategy - that which is state- but not time-dependent - was determined under each of the four scenarios described in section 5.3.1.2 from a backward iteration, run as described in section 5.3.2.5. The relative fitnesses of strategies calculated under these four scenarios are shown in figure 5.5: here the number of copies, at time t = 50, of the genes from 100 individuals conceived at time t = 0 is shown given that half these individuals experience a high foraging environment and half a low foraging environment at conception and given that foraging environment each experiences is updated independently each period of the simulation.

The characteristics of such cohorts will depend upon the investments made by their mothers in early life as well as the foraging environments experienced throughout life and maternal states for these 100 individuals at the time of conception were computed as follows. A forward iteration was run, starting from an arbitrarily chosen initial population each member of which experiences an arbitrarily chosen initial foraging environment. All descendants of this population were tracked over time, with the foraging environment that each experiences being independently updated each period according to the transition probability under which the stable strategy was calculated. (The only exception to the independence of environments experienced by individuals is that dependent offspring must experience the same environment as their mothers.) This simulation was run for 1000 time points. At this point the distribution of foraging environments had stabilised - half the population experiencing a good environment and half a bad one - as had the age distribution (Γ) of the population. Maternal states for members of the cohorts shown in figure 5.5 who experienced a low foraging environment at conception were a random sample of the states of females in Γ who experienced a low foraging environment and who conceived in period 1000; maternal states for members of the cohorts shown in figure 5.5 who experienced a high foraging environment at conception were a random sample of the states of females in Γ who experienced a high foraging environment and who conceived in period 1000.

As expected, the allocation strategy associated with plasticity throughout life and prediction of the future environment is the fittest. The value of predicting the future environment can be seen by comparing columns 1 and 2 and columns 3 and 4: in both cases individuals who predict their future environment based on the current one are fitter than those who do not. Fixing strategy at the time of conception (columns 3 and 4) - so that it cannot be tailored to current state or changes to the predicted future environment based on the current one - results in a marked reduction in fitness. The fitness cost which the retention of plasticity must entail if a fixed strategy is to evolve is the difference between columns 1 and 3 if the future environment is predicted and the difference between columns 2 and 4 if no prediction of future environment in made.

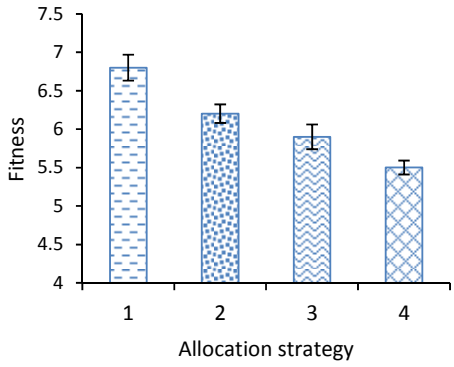

Figure 5.5: The relative fitnesses of allocation strategies calculated under four different assumptions: that plasticity is retained throughout life and future environment is predicted (1); that plasticity is retained but no prediction is made of the future environment (2); that strategy is fixed in early life and the future environment is predicted (3) and; that strategy is fixed in early life and no prediction is made of the future environment. Fitness is here defined as the number of copies of genes at t = 50 from 100 individuals conceived at time t = 0 half of whom experience a high foraging environment and half of whom experience a low foraging environment at conception.

5.4.2 Characteristics of cohorts following strategies calculated under different assumptions

The characteristics of cohorts who follow strategies calculated under the four scenarios described in section 5.3.1.2 are shown in figures 5.6 - 5.9. For each scenario,

cohorts were followed who experienced a) a low foraging environment throughout life(L/L); b) a high foraging environment throughout life (H/H); c) a low foraging environment for the first five years of life and a high foraging environment thereafter (L/H) and; d) a high foraging environment for the first five years of life and a low foraging environment thereafter (H/L). States of mothers at the time of conceiving members of the cohorts were a random sample from Γ (similar to what is described above). Cohorts were characterised in terms of lifespan, rate of ageing, age at maturity and number of offspring. Rates of ageing were calculated for each cohort as a whole by fitting a Gompertz function to mortality data from three years onwards (after which age mortality is a function only of condition). Age at maturity and offspring number are for those who attained the adult size. Age at maturity was equivalent to age at first pregnancy since all females chose to conceive in the first period after they attained the adult size.

Consider first the characteristics of cohorts who retained plasticity in their allocation strategies throughout life and predicted the future environment based on the current one (figure 5.6). Comparison of cohorts who experienced a constant low foraging environment and a constant high foraging environment confirms the optimality of an accelerated ageing rate in times of adversity. This is not however a generalised acceleration in life history since the age at maturity is higher in the adverse environment. When the available energy budget is lower, juveniles will reduce investment in both maintenance and growth and adults will reduce investment in both maintenance and reproduction. The number of births is reduced as a consequence of both the greater age at maturity and the decrease in lifespan.

The impact of early environment upon state and thus upon characteristics throughout the life course is evidenced by the results for cohorts whose environment changes at five years. The L/H cohort matured later and experienced a faster rate of ageing than the H/H cohort; the H/L cohort matured sooner and experienced a slower rate of ageing than the L/L cohort.

Consider next those cohorts who retained plasticity in their allocation strategies throughout life but did not use the current environment to predict the future one (figure 5.7). Characteristics for the L/L and H/H cohorts are generally closer than was

the case above and this is largely mediated by the investment decisions of the cohorts' mothers at conception. If the environment at conception is unfavourable, the mother does not predict a similarly unfavourable environment in the next two periods but rather assumes that favourable and unfavourable environments are equally likely. She therefore invests more in her offspring since this will allow her to take more advantage of a subsequent favourable environment although it disadvantages her more if the environment remains unfavourable. By a similar logic, when the environment at conception is favourable the mother will now invest less in her offspring. As above, the effect of early life environment upon state is apparent: the L/H cohort again matured later and experienced a faster rate of ageing than the H/H cohort whilst the H/L cohort again matured sooner and experienced a slower rate of ageing than the L/L cohort.

The characteristics of cohorts whose allocation strategies are fixed in early life and who use the environment at conception to predict future environment are shown in figure 5.8. Characteristics for the L/L and H/H cohorts are again closer than was the case for those cohorts that retained plasticity and predicted the future environment (figure 5.6). Their strategies are therefore not perfectly suited to their continued respective environments. This reflects a degree of bet-hedging; since individuals cannot be sure that the environment at conception will continue and cannot change their strategy in response to a changing environment, they settle for a compromise between the strategies that would suit each environment, albeit this compromise is weighted towards the environment experienced at conception.

Note that differences in the lifespan and rate of ageing of the two cohorts who experience a poor start (L/L and L/H) - and similarly differences between those that experience a good start (H/H and H/L) - must be due only to stochastic differences between cohorts. This is because all individuals who experience the same environment at conception will (if their mothers are in the same states) follow the same strategy and any differences in their characteristics are the result only the interaction between strategy and environment. When the environment subsequently changes, investment in growth or reproduction will also change - and we can see this reflected in differences in the age of maturity (figure 5.8) - whilst investment in maintenance is fixed.

Lastly, figure 5.9 shows characteristics of cohorts whose allocation strategies are fixed in early life and who do not use the current environment to predict the future one. The allocation strategies followed by members of this cohort are the least successful. Differences between this cohort and the previous one are, however, relatively slight. Those who experience a poor start display a slightly slower rate of ageing and higher age at maturity than above whilst those who experience a good start display a slightly faster rate of ageing and lower age at maturity. Individuals are again hedging their bets but they now assume that both good and bad environments are equally likely from the second period of life onwards regardless of that at conception.

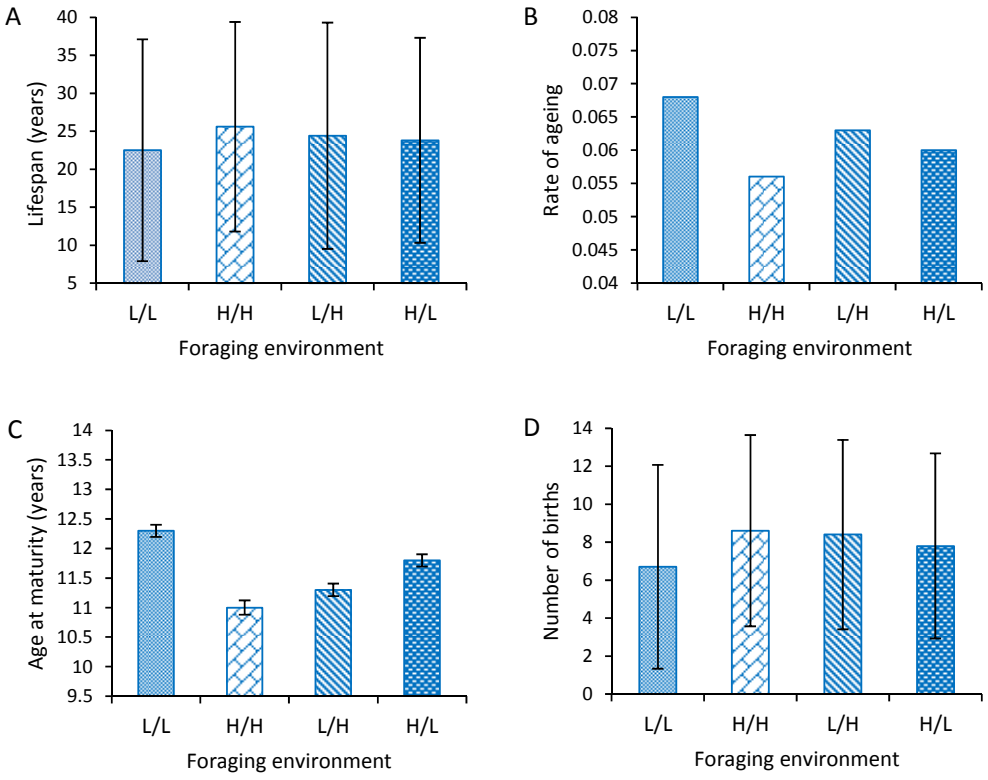

Figure 5.6: Life history characteristics of cohorts of 100 females all of whom retain plasticity in their energy allocation strategies throughout life and who base decisions each period upon their own state, the current environment and the expected environment in the next period given the current one. Cohorts L/L experience a low foraging environment throughout life; H/H a high foraging environment throughout life; L/H a low foraging environment until age five and a high foraging environment thereafter and H/L a high foraging environment until age five and a low foraging environment thereafter. Error bars show the standard error.

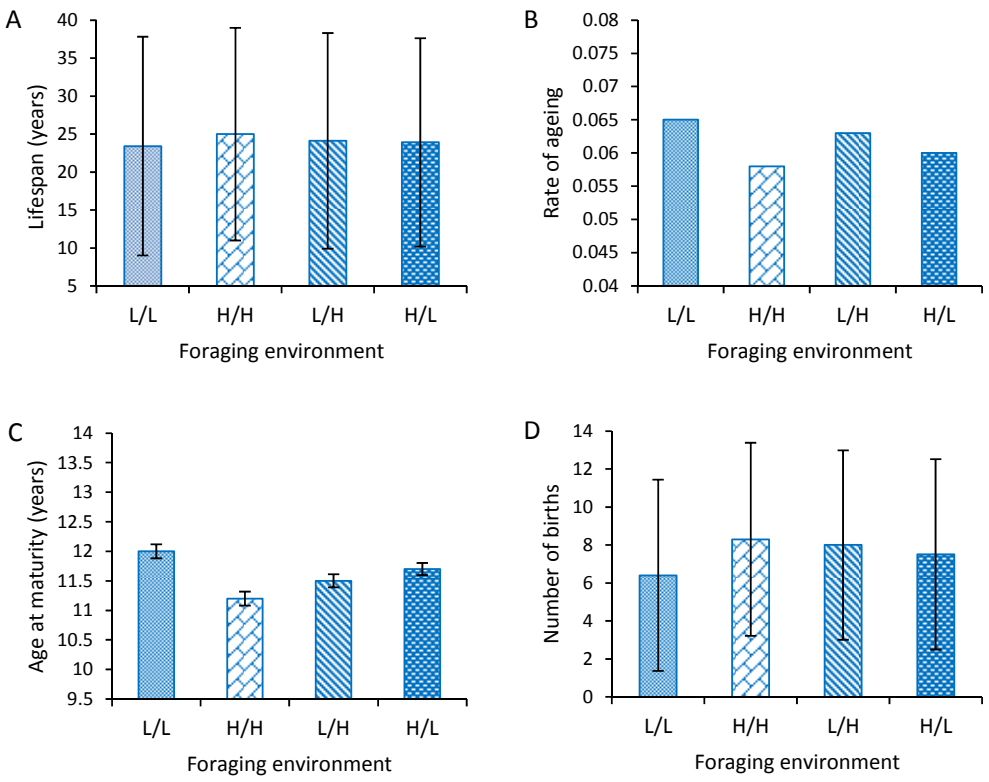

Figure 5.7: Life history characteristics of cohorts of 100 females all of whom retain plasticity in their energy allocation strategies throughout life and who base decisions each period upon their own state and the current environment but do not predict future environment based on the current one. Cohorts L/L experience a low foraging environment throughout life; H/H a high foraging environment throughout life; L/H a low foraging environment until age five and a high foraging environment thereafter and H/L a high foraging environment until age five and a low foraging environment thereafter. Error bars show the standard error.

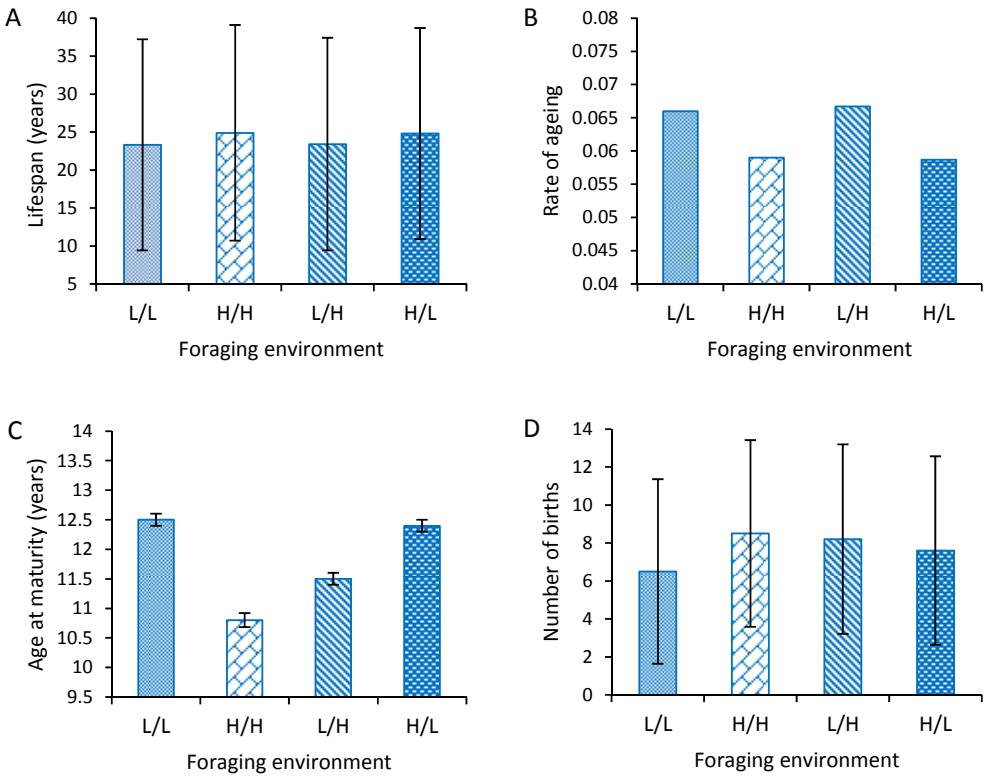

Figure 5.8: Life history characteristics of cohorts of 100 females all of whom fix their allocation strategy at conception and who do so based on the expected future environments that they will experience given that at conception. Cohorts L/L experience a low foraging environment throughout life; H/H a high foraging environment throughout life; L/H a low foraging environment until age five and a high foraging environment thereafter and H/L a high foraging environment until age five and a low foraging environment thereafter. Error bars show the standard error.

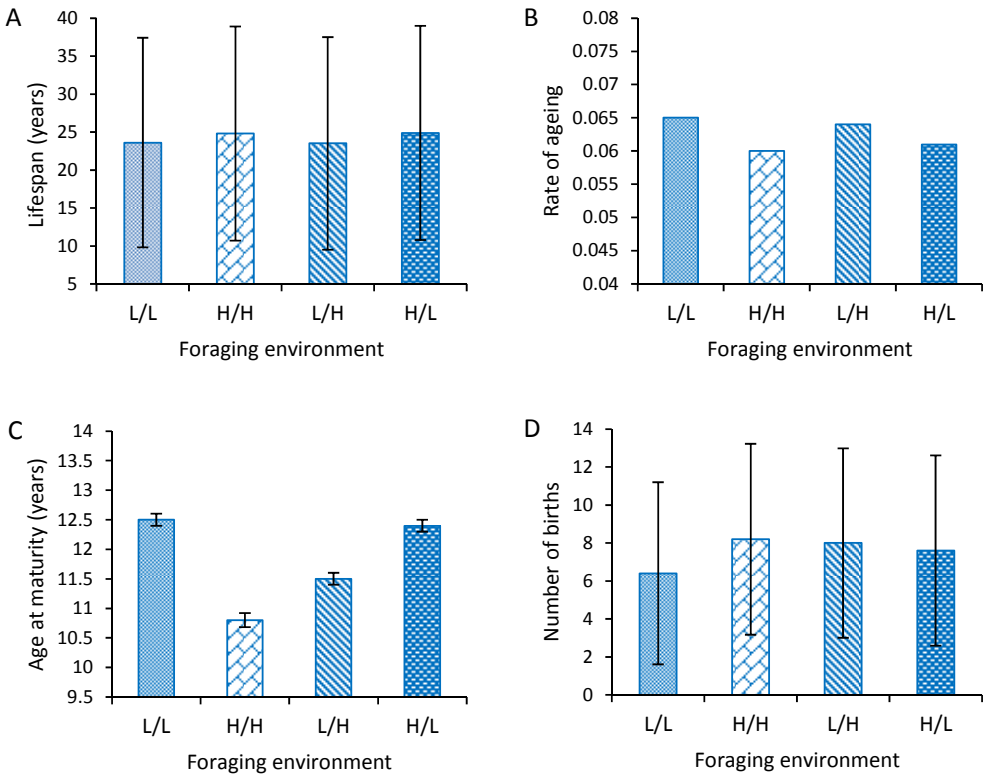

Figure 5.9: Life history characteristics of cohorts of 100 females all of whom fix their allocation strategy at conception and who do not base this strategy on the expected future environments that they will experience given that at conception. Cohorts L/L experience a low foraging environment throughout life; H/H a high foraging environment throughout life; L/H a low foraging environment until age five and a high foraging environment thereafter and H/L a high foraging environment until age five and a low foraging environment thereafter. Error bars show the standard error.

5.4.3 Parent-offspring conflict

As described in the introduction to this chapter, conflict can arise between parent and offspring as to the optimal allocation that a mother should make to reproduction, with the offspring wanting this to be that which maximises her (the offspring's) expected reproductive value plus the expected reproductive value of her mother from other offspring at the start of the next period. Since the *total* reproductive value of the mother in period (t + 1) includes that of the current offspring multiplied by the relatedness between them (0.5), this is equivalent to the offspring maximising the total reproductive value of the mother plus half her own at the start of the next period. (In weighting the

reproductive value that her mother has from her mother's other offspring as highly as her own (the offspring's) reproductive value, the offspring assumes that she is as closely related to her sisters as to her daughters. This assumes that siblings share a father and therefore that their relatedness is one half.) Simulations were run in which the mother's allocation strategy was calculated in this way and the results in terms of offspring characteristics are shown in figure 5.10. It is assumed that all individuals retain plasticity throughout life and predict the future environment based on the current one.

Perhaps unexpectedly, there is little difference in offspring characteristics (and consequently offspring fitness) when the offspring can determine the allocation her mother makes in her. This likely reflects the fact that a greater part of the offspring's inclusive fitness will come from her mother descendants rather than her own; since her mother has already survived the period of greatest mortality - i.e. early life - her expected reproductive success considerably exceeds that of her offspring.

5.4.4 Sensitivity analysis

As in chapter 4, sensitivity analysis was carried out to determine the robustness of model output to the underlying parameter values (table 5.1). For the transition probability, values of 0.75, 0.80, 0.85, 0.90, 0.98 and 0.99 were considered. For all other parameters, eight perturbations were considered corresponding to increases of -30, -22.5, -15, -7.5, 7.5, 15, 22.5 and 30% of the original values.

As expected, when the transition probability is increased, the value of prediction increases. For all other parameters, the value of prediction - i.e. the relative fitness of a strategy which predicts future environment compared to one that doesn't - is essentially unaltered. The relative fitness costs of constraining strategy in early life, and therefore the costs which it may be inferred are associated with retaining plasticity, is also essentially unchanged under perturbations to all parameters. This is despite the fact that absolute fitnesses calculated do vary slightly as parameters do. When foraging yield is increased (by increasing either the foraging multiplicand or exponent), fitness increases; when the costs of essential functions or any component of mortality rate is increased, fitness decreases.

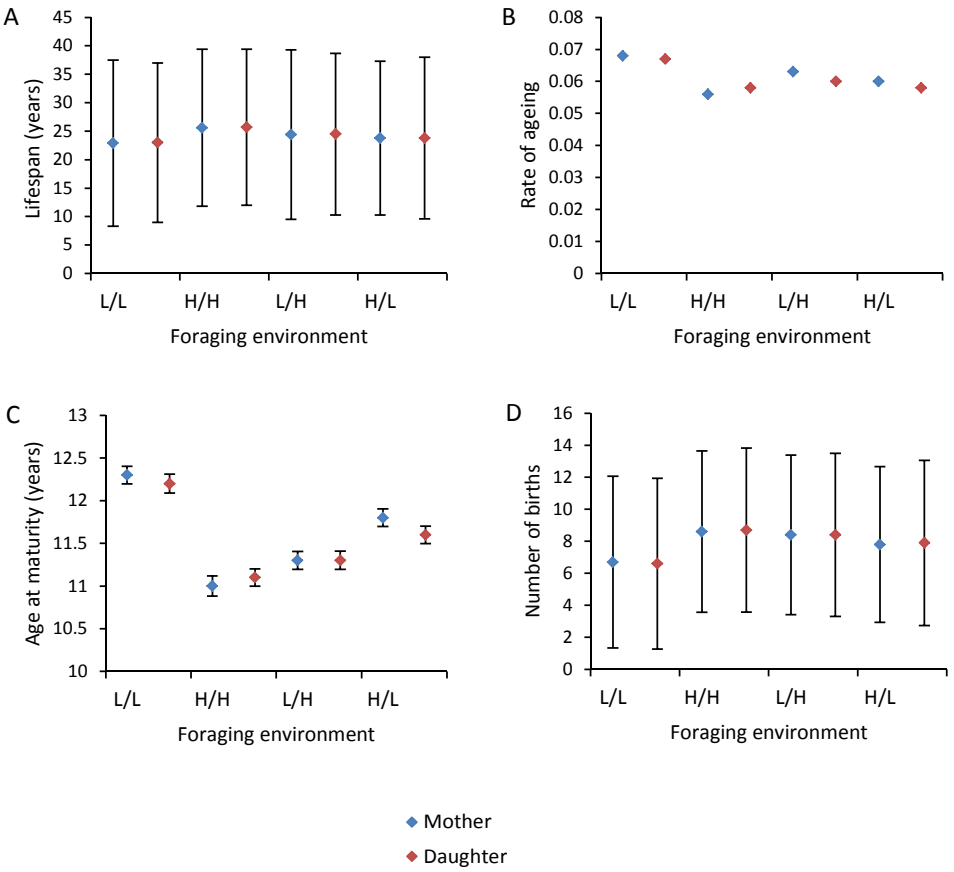

Figure 5.10: Life history characteristics of cohorts of 100 females given that the maternal investment they received at the start of life was chosen so as to maximise their mother's reproductive value (blue) or their mother's plus half their own (red). All individuals retain plasticity in their energy allocation strategies throughout life and who predict future environment based on the current one. Cohorts L/L experience a low foraging environment throughout life; H/H a high foraging environment throughout life; L/H a low foraging environment until age five and a high foraging environment thereafter and H/L a high foraging environment until age five and a low foraging environment thereafter. Error bars show the standard error.

Transition probability

	Prediction		No prediction	
	Plastic	Fixed	Plastic	Fixed
0.75	6.6	5.8	6.2	5.7
0.8	6.7	5.7	6.4	5.7
0.85	6.8	5.8	6.2	5.6
0.9	6.7	5.8	6.1	5.5
0	6.8	5.9	6.2	5.5
0.98	6.9	5.9	6.1	5.5
0.99	6.9	6	6.2	5.4

BMR multiplicand

	Prediction		No prediction	
	Plastic	Fixed	Plastic	Fixed
-30	7.2	6	6.6	5.8
-22.5	7	6.1	6.5	6
-15	7.1	6.1	6.4	5.8
-7.5	6.8	5.8	6.4	5.6
0	6.8	5.9	6.2	5.5
7.5	6.6	5.7	6.3	5.5
15	6.7	5.8	6.1	5.6
22.5	6.4	5.6	6	5.3
30	6.4	5.6	6.2	5.5

BMR exponent

	Prediction		No prediction	
	Plastic	Fixed	Plastic	Fixed
-30	7	6.4	6.7	5.8
-22.5	6.6	6.3	6.8	5.5
-15	7.2	6.4	6.2	5.7
-7.5	6.6	6.1	6.4	5.6
0	6.8	5.9	6.2	5.5
7.5	6.9	5.8	6.1	5.7
15	6.7	6.2	5.8	5.4
22.5	6.6	5.7	5.9	5.5
30	6.7	5.9	5.9	5.5

Ageing rate exponent

	Prediction		No prediction	
	Plastic	Fixed	Plastic	Fixed
-30	7.1	5.9	5.9	5.8
-22.5	6.6	6	6	5.7
-15	6.7	5.8	6	5.7
-7.5	7	5.6	6.1	5.6
0	6.8	5.9	6.2	5.5
7.5	6.6	5.7	5.9	5.6
15	6.7	5.5	6.3	5.4
22.5	6.6	5.7	5.8	5.4
30	6.5	5.8	6	5.2

Exercise multiplicand

	Prediction		No prediction	
	Plastic	Fixed	Plastic	Fixed
-30	6.9	6.2	6.4	5.7
-22.5	7.2	6	6.3	5.9
-15	7.1	6	6.4	5.8
-7.5	6.9	5.7	6.1	5.6
0	6.8	5.9	6.2	5.5
7.5	6.9	5.6	6.2	5.7
15	6.8	5.7	6.3	5.5
22.5	6.7	5.6	5.8	5.9
30	6.8	5.6	5.7	5.1

Exercise exponent

	Prediction		No prediction	
	Plastic	Fixed	Plastic	Fixed
-30	6.7	6.1	6.3	5.4
-22.5	6.9	6.1	6.2	5.6
-15	7.1	5.6	5.7	5.7
-7.5	6.6	5.7	6.1	5.6
0	6.8	5.9	6.2	5.5
7.5	6.5	5.8	5.9	5.8
15	6.7	5.9	6.1	5.4
22.5	6.4	5.5	5.7	5.4
30	6.5	5.7	5.9	5.5

Energy required to put on 1kg

	Prediction		No prediction	
	Plastic	Fixed	Plastic	Fixed
-30	7.1	6.3	6.5	5.7
-22.5	7	6	6.4	5.8
-15	7.1	5.8	6.2	5.7
-7.5	6.9	6	6.3	5.4
0	6.8	5.9	6.2	5.5
7.5	6.7	5.8	6.2	5.5
15	6.8	5.7	6	5.7
22.5	6.6	5.7	5.9	5.6
30	6.6	5.6	6	5.8

Foraging yield multiplicand

	Prediction		No prediction	
	Plastic	Fixed	Plastic	Fixed
-30	6.6	5.5	5.7	5.4
-22.5	6.7	5.8	5.8	5.5
-15	6.7	5.7	5.9	5.6
-7.5	6.9	6.1	6.1	5.6
0	6.8	5.9	6.2	5.5
7.5	7	6	6.1	5.7
15	6.7	5.7	6.3	6
22.5	6.8	5.5	6.5	5.9
30	7.1	5.6	6.3	5.8

Foraging yield exponent

	Prediction		No prediction	
	Plastic	Fixed	Plastic	Fixed
-30	6.4	5.6	5.7	5.6
-22.5	6.6	5.8	5.8	5.5
-15	6.6	5.7	6	5.4
-7.5	6.7	6	6	5.6
0	6.8	5.9	6.2	5.5
7.5	6.9	6.1	6.3	5.5
15	7	6.2	6.3	5.8
22.5	7.1	6	6.5	5.9
30	6.8	6.2	6.4	5.7

Rate of ageing

	Prediction		No prediction	
	Plastic	Fixed	Plastic	Fixed
-30	7.2	6.1	6.3	5.7
-22.5	7	6.1	6.1	5.8
-15	7	6	6.2	5.6
-7.5	6.9	6	6	5.6
0	6.8	5.9	6.2	5.5
7.5	6.8	6	5.9	5.4
15	6.7	5.9	6.1	5.4
22.5	6.7	5.8	6	5.5
30	6.8	5.9	5.8	5.4

Initial mortality rate

	Prediction		No prediction	
	Plastic	Fixed	Plastic	Fixed
-30	6.8	6.2	6.3	5.6
-22.5	6.9	5.9	6.3	5.6
-15	6.9	5.9	6.1	5.7
-7.5	6.7	5.8	6.2	5.6
0	6.8	5.9	6.2	5.5
7.5	6.8	5.8	6.1	5.4
15	6.7	5.7	6	5.5
22.5	6.8	5.8	6.1	5.4
30	6.6	5.9	5.9	5.3

Maternal mortality

	Prediction		No prediction	
	Plastic	Fixed	Plastic	Fixed
-30	7	6	6.2	5.6
-22.5	6.8	6	6.3	5.7
-15	6.9	5.9	6.1	5.6
-7.5	6.7	6	6.2	5.5
0	6.8	5.9	6.2	5.5
7.5	6.7	5.9	6	5.6
15	6.8	5.8	6.2	5.5
22.5	6.6	5.7	6.1	5.6
30	6.7	5.8	6.1	5.6

Early-life mortality

	Prediction		No prediction	
	Plastic	Fixed	Plastic	Fixed
-30	7.3	6.3	6.4	5.8
-22.5	7.2	6.2	6.3	5.8
-15	7.2	6	6.3	5.6
-7.5	7	6	6.1	5.6
0	6.8	5.9	6.2	5.5
7.5	6.7	5.7	6.1	5.3
15	6.7	5.7	6	5.2
22.5	6.5	5.6	6	5.4
30	6.4	5.6	5.9	5.3

Table 5.1: Relative fitnesses of cohorts of 100 individuals following strategies calculated under the four scenarios shown given that parameters are varied from their original values by the percentages indicated. In the case of the transition probability, absolute values are listed rather than percentage differences. Fitness is here defined as the number of copies of genes at t = 50 from 100 individuals conceived at time t = 0 half of whom experience a high foraging environment and half of whom experience a low foraging environment at conception.

5.5 Discussion

A model is presented of energy allocation between somatic maintenance and either growth (during the juvenile period) and reproduction (during the adult period) in humans. Optimal allocation has been computed in response to fluctuating food availability under four different assumptions: firstly that plasticity is retained throughout life and the future environment is predicted based on the current one; secondly that plasticity is retained but no prediction is made; thirdly that strategy is fixed in early life and the future environment is predicted and; fourthly that strategy is fixed in early life and no prediction is made.

In times of low food availability, it is predicted that investment in both maintenance and in growth/reproduction is reduced; in a low foraging environment lifespan decreases and the age at maturity increases. The model suggests that both internal state and prediction of the future environment may be important in explaining the changes to life history which follow early life adversity. Furthermore, the model provides an estimate for the fitness costs which must be associated with maintaining plasticity throughout life if a predictive adaptive response is to evolve. This is given by the average fitnesses of individuals following a plastic strategy minus that of individuals whose allocation decisions are constrained in early life.

By altering the dynamic programming equation so that a mother makes her allocation decision so as to maximise her offspring's fitness rather than her own, it was possible to calculate the degree of parent-offspring conflict over maternal resources and to compute the effects of this upon the offspring's life history characteristics. Little evidence for conflict was found and an offspring's lifespan, rate of ageing, age at maturity and reproductive rate do not change significantly when she is able to control her mother's investment in her. The reason for this is probably that at the point at which the mother's allocation to an offspring is made, the mother's future reproductive success will contribute much more to the inclusive fitness of the offspring than will her own expected reproductive success. It is possible that conflict would be more apparent if iterative decisions between parent and offspring were modelled. Towards the end of the period of parental investment, the offspring will be past the time of greatest mortality and her expected reproductive success relative to her mother's will have increased.

Several further simulations could be run with this model which could address points of biological interest but which due to time constraints are not included in the current work. The relative importance of predicting the juvenile versus the adult environment will be computed by allowing those under 12 say to base their allocation strategies upon the predicted future environment and those over 12 to base their allocation strategies upon the long-term probabilities of being in each state. The reduction in fitness when compared with those who may predict the future environment throughout the entire life course provides a measure of the value of predicting the adult environment. The increase in fitness when compared with those who do not predict the future environment at any age provides a measure of the value of predicting the juvenile environment.

The fitness consequences of following suboptimal strategies could be computed, similarly to as was done for the baboon model in chapter 4. It might be supposed that suboptimal strategies would be more costly when strategy is constrained in early life as there can be no subsequent compensation for a badly-chosen allocation decision and it would be interesting to see whether the model confirms this.

The model could be extended in a number of ways to include greater biological complexity and two possible extensions are detailed in chapter 6: allowing size at maturation to vary and allowing iterative decisions between parent and offspring. It would also be interesting to consider the effect of fluctuations in a different measure of environmental harshness such as mortality rate; to allow mothers to provision multiple offspring simultaneously and; to allow individuals to choose a foraging strategy with a riskier strategy potentially being of more benefit in times of low food availability.

5.6 Summary

1. A model of energy allocation in humans in response to a fluctuating environment has been built.
2. The model shows that both internal state and prediction of the future environment can be important in explaining the changes to life history seen following early life adversity.
3. By comparing simulations in which the strategy is fixed in early life to those where it is plastic, the costs which must be associated with maintaining plasticity for a predictive adaptive response to evolve can be calculated.
4. The degree of parent-offspring conflict is weak in this simple model.
5. With the exception that the value of prediction changes with the transition probability between environmental states, the model is robust to changes in parameter values.

Chapter 6: Future directions

6.1 Summary

This thesis has examined the relationship between growth and ageing from an evolutionary perspective. Since the associations between growth/size and longevity are manifold, any theory for the evolution of ageing must be able to explain why these arise. Chapter 1 outlines the main theories which have guided thinking and research into ageing over the years - in particular the disposable soma theory which is probably the most influential at present - and lists some of the evidence given for each. A summary was given of the relationships between growth and ageing and, since size often differs between the sexes, between sex and ageing.

This thesis has contributed to the literature on three fronts. Firstly, a critique is given of two recent theories which propose novel explanations for the evolution of ageing and both of which rely explicitly upon associations between growth and longevity (chapter 2). Blagosklonny's hyperfunction theory asserts that growth-promoting pathways are not switched off post-maturity and that their continuation drives ageing. Speakman and Krol's heat dissipation limit theory suggests that constraints upon heat dissipation and therefore reproductive rate in larger animals mean that only those species who enjoy a long lifespan may evolve a large body size. The analysis of these theories has also helped clarify some issues with how the disposable soma theory has been thought about, such as the necessity of considering inter-related behavioural trade-offs.

Two models are then presented which test whether certain observed relationships between growth and lifespan are compatible with the disposable soma theory. Both use dynamic programming, the principles of which are outlined in chapter 3. Dynamic programming provides a computationally efficient means of determining an optimal sequence of state- and time-dependent decisions. Computational demand increases exponentially with each time step when an exhaustive forward simulation is required to determine an optimal sequence of decisions. Dynamic programming avoids this combinatorial explosion by working backwards from some time horizon and determining optimal decisions at each time point in relation to those at the next one.

Chapter 4 presents a model of energy allocation between growth, maintenance and reproduction in baboons. There is quantitative agreement between model and field results for dimorphism in size, lifespan and reproductive scheduling, that is, behavioural differences between the sexes are as expected if individuals optimally allocate energy between these three fitness-enhancing functions. A number of other findings emerged from this model. Firstly, a lower selective pressure need not result in a wider range of phenotypes in a population. Secondly, the force of selection upon a trade-off need not be monotonically decreasing with age; that between growth and maintenance in males increased during the sub-adult phase before decreasing during the adult phase. Thirdly, deviations from the 1:1 sex ratio predicted by Fisher are selected for when a population is either in growth or decline and the sexes differ in their average age at reproduction.

Chapter 5 presents a model of energy allocation in a fluctuating environment in humans. It has demonstrated the importance of both internal state and prediction of the future environment based on the current one in explaining the changes to life history which follow a period of early adversity. By comparing simulations in which allocation strategy is fixed in early life with those where it is plastic, the costs that must be associated with maintaining plasticity if a predictive adaptive response is to evolve were inferred.

Both models could be fruitfully extended to consider more complex behaviour. In the final sections of this thesis (6.2 and 6.3 below), two extensions to each of the models are described: allowing maternal investment to vary and allowing for paternal care in the baboon model and; incorporating size at maturation as a behavioural decision and allowing iterative decisions between mother and offspring in the model of early life adversity.

6.2 Extensions to the model for sexual dimorphism in baboons

6.2.1 Allowing maternal investment in offspring to vary

In the model of sexual dimorphism in baboons, it was assumed that maternal investments in dependent offspring followed a fixed schedule. Specifically, offspring

required an energetic investment of 4500kcal/period during pregnancy and 9000kcal/period during lactation. If less was invested, the offspring did not survive; if more was invested the offspring was unable to utilise this extra energy. Since the proportions of an offspring's energy budget that were allotted to growth and maintenance were also fixed, this meant that offspring of the same age were all of the same size and condition.

This assumption was made because it produced a smaller female state space (dependent offspring were assumed to be part of their mother's state) and therefore shorter model run time than a model in which mothers could vary their investment in offspring. It is, however, biologically unrealistic since it is known that energetic investment in offspring varies with maternal age, rank and energy intake[76]. Consequently, the changes to female strategy which accompanied variation in either the energy available for growth, maintenance and reproduction or the total energetic requirements of offspring (section 4.4.6) were not credible. An increase in the former led to a reduction in maximum size; since more energy was available to females per period, they were less dependent upon body stores to provision offspring. This is the opposite response to that seen in the field where a greater foraging yield results in both a larger maternal size and more rapid growth in offspring. An increase in the latter led to a rise in maximum size; in order for the additional energetic demands of offspring to be met, greater body stores were laid down before conception. Again, this is not biologically plausible; the increased energetic burden placed upon the mother by increasing the energetic requirements of offspring is equivalent to a reduction in her available energy budget and the expected response would be to lengthen the period of investment. The following extension to the model would permit mothers to alter the energetic investment per period and/or the length of time over which they provision offspring.

Assume that mothers may invest anything between zero kilocalories and their total available energy budget in dependent offspring and that their choice of investment may depend upon their own state and that of their offspring. Dependent offspring are now represented by two variables: size (s_o) and condition (c_o). For simplicity, let it be assumed that all dependent offspring still face the same mortality threat per period regardless of state; that offspring allocation decisions will be constrained to follow a

fixed rule and; that the mother will cease investment in an offspring when (s)he reaches a critical size (s_c). Behaviour during the juvenile period is unaffected: 40 periods elapse between the times of weaning and of reaching maturity at 8.5kg and condition increases by 40τ during this time. Since offspring may now be of different conditions at the time of weaning, they may also be so at the time of maturity which may affect subsequent allocation decisions. Within the backward iterations for both sexes the minimum condition of mature individuals would have to be reduced to allow for the fact that individuals may be weaned at conditions lower than 10τ.

An offspring's available energy budget is the investment received from the mother minus that required for vital metabolism, (BMR/2). As infants are for the most part carried by their mothers, they incur no travel costs. Allocation to growth is a pre-defined function (Θ) of the available energy budget, the remainder being invested in maintenance. The exact nature of this function is not important here but it could simply be that a fixed proportion of the energy budget is always invested in growth or that the proportion invested in growth changes with the total energy budget, perhaps in the way that was determined by the predictive adaptive response model. The functions relating a) investment in growth to change in size and b) investment in maintenance to change in condition are as defined for adults in sections 4.3.3.3 and 4.3.3.7. (It would be interesting to model allocation decisions in offspring as well, with mother and offspring both playing a strategy the optimality of which is determined with reference to how the other behaves. The simpler extension outlined here is - whilst being more computationally demanding than the original model - significantly less demanding than would be one which incorporated games between mother and offspring.)

As developmental processes cannot be compressed into an arbitrarily short nor extended over an arbitrarily long time frame, minimum and maximum rates of growth per period are specified; these are given in terms of size at the start of the period and represented by $\Xi_{max}(s_o)$ and $\Xi_{min}(s_o)$ respectively. Should allocation to growth - which is dependent upon maternal investment and the function Θ - not meet the requirements for minimum growth, the offspring dies. Should it exceed that for maximum growth, the extra energy cannot be utilised by the offspring.

The dynamic programming equation for females becomes:

$$\mathbf{F(s, c, s_o, c_o, t, T) = (1 - \beta(c))*[\max_i \max_j E\{F(s + \gamma(j), c + \zeta(k), s_o'(s_o, i, \Theta, \Xi_{max}(s_o), \Xi_{min}(s_o)), c_o'(c_o, i, \Theta), t+1, T) + 0.5*\Psi(s_o, c_o, i)*\phi*F(8.5, c_{o(t+1)} + 40, 0, 0, t + 41, T)\}]}$$

where:

- $F(s, c, s_o, c_o, t, T)$ is the maximum expected reproductive value (taken over the set of all possible allocation decisions) for a female of size s and condition c, with a dependent offspring of size s_o and condition c_o at time t, given a final time horizon T.
- $\beta(c)$, i, j, k, $\gamma(j)$, $\zeta(k)$ and ϕ are defined as in the original model.
- i may take values in the range:

  $$0 \leq i \leq \xi(s) - \alpha(s) + v*\max(8 - s; -3)$$

  where v, $\xi(s)$ and $\alpha(s)$ are defined as before.
- j and k may take values in the same ranges as for the original model.
- $s_o'(s_o, i, \Theta, \Xi_{max}(s_o), \Xi_{min}(s_o))$ is the size of any dependent offspring at time (t + 1). $\Xi_{max}(s_o)$ and $\Xi_{min}(s_o)$ represent respectively the constraints upon maximum and minimum growth and Θ is the offspring allocation strategy .
- $c_o'(c_o, i, \Theta)$ is the condition of any dependent offspring at time (t + 1).
- $\Psi(s_o, c_o, i)$ is set to one if an offspring is reared to independence at the end of the current period and is zero otherwise.

The terminal fitness function for females is now:

$$\mathbf{F(s, c, s_o, c_o, T, T) = 1 \quad if\ s \geq 8.5\ and\ c < 300}$$

$$\mathbf{F(s, c, s_o, c_o, T, T) = 0 \quad if\ s = 8.5\ and/or\ c = 300}$$

The dynamic programming equation for males becomes:

$$\mathbf{F(s, c, t, T) = (1 - \beta(c))*[\max_i \max_j E\{ F(s + \gamma(j), c + \zeta(k), t+1, T) + 0.5*\chi(s, c, i, \Omega_f, \Omega_m)*\lambda(\Omega_f)*\phi*[0.5*F(8.5, \Delta(\Omega_f) + 40, 0, 0, t + 55, T) + 0.5*F(8.5, \Delta(\Omega_f) + 40, t + 55, T)]\}]}$$

where:

- $F(s, c, t, T)$, $\beta(c)$, i, j, k, $\gamma(j)$, $\zeta(k)$, Ω_m, Ω_f, $\chi(s, c, i, \Omega_f, \Omega_m)$, $\lambda(\Omega_f)$ and ϕ are defined as in the original model.
- i, j and k may take values in the same ranges as in the original model.
- $\Delta(\Omega_f)$ is the expected condition of a newly-matured offspring.

The terminal fitness function for males is unchanged.

This extended model could be used to address the following questions:

1. Can the increase in IBI with age that is seen in real baboons be reproduced in simulated individuals?
2. If mothers can conserve energy when body stores are low by reducing per-period investment in offspring, what effect, if any, does this have upon the rate of decline in size with age and the change in condition at older ages?
3. Is there any predicted difference in the quality of offspring from older and younger mothers?
4. What effect does condition at maturity have upon allocation strategy thereafter and expected lifetime reproductive success?
5. How does predicted behaviour change with the available energy budget? Do we now see the expected reduction in maternal size and increase in the duration of lactation in response to energetic stress?

6.2.2 Allowing for paternal care

It was assumed in the original model that male fitness depended entirely upon the ability to secure lone consortships with receptive females. The provision of paternal care is one of several other fitness-enhancing strategies which have been documented in male baboons. Males have been shown to recognise their own offspring and preferentially direct care towards them[89, 90]. This care may take various forms including the protection of infants from attacks by newly-immigrant males; providing foraging assistance to juveniles and; support of juveniles during agonistic encounters.

For paternal care to evolve the expected fitness gains to the father from providing it would obviously need to outweigh the losses he incurs by diverting resources away from other functions. It would therefore be interesting to see which functions would

suffer as a result and what effect the allowance of paternal care would have on other aspects of the life-history trajectory. It was suggested in section 4.5.2 that, were paternal care possible, the value of longevity to a male might increase: males who live longer can do more to improve the chances of their offspring. If this were so, then the costs of increased investment in maintenance as well as of paternal care must be paid for by a slower growth rate, reduced maximum size and/or reduction in the number of days upon which consortship is attempted.

The value of paternal care may depend on the states of both father and offspring and on the external environment. Since the chance of impregnating a female per unit energy invested in attempting to consort is a decreasing function of age, the relative fitness gains from investing in paternal care versus attempting to consort would - presuming that the quality of paternal care given by older males is not significantly lower than for younger males - increase with age. We might therefore expect to see more paternal care from older males than from younger ones. The value of paternal care will vary with offspring state and quality (if differences in offspring quality are being modelled). For example, the protection against mortality afforded an offspring when the father remains in close proximity to it is likely greater for those under one year - since they face the highest mortality threat - or for those whose poorer quality would otherwise make them a more likely target for a predator. In a harsh environment, where the probability of offspring survival is low when only maternal care is supplied, paternal care may also be more likely.

An exhaustive model of the decisions a male faces regarding paternal care would be extremely computationally demanding. For all states in which a male may find himself, all possible combinations of offspring he may have would need to be considered and some estimate of the fitness gains that offspring in different states would accrue from a unit of paternal investment would need to be supplied. The fitness consequences of choosing to invest in some or all of these offspring could then be computed. The extension to the original model proposed below allows for a simplified treatment of paternal care. This assumes that males base their decisions upon their expected distribution of offspring given their state rather than their actual offspring set.

Assume that paternal care reduces the mortality risk experienced by infant/juvenile offspring in the period in which it is received. The decrement in mortality risk will be a function of offspring state but for simplicity it is supposed independent of paternal state. As mortality risk decreases with age up until the point of maturity, it is reasonable to expect that the reduction in mortality experienced by a recipient of paternal care is also a decreasing function of age.

Assuming initially that all individuals (male and female) follow the strategy that was optimal under the original model, the expected distribution of offspring that a male will have can be computed as a function of his state by means of a forward simulation following a large cohort. Let $\Pi(A)$ denote the probability that he has A offspring and $\Gamma(A)$ the expected age of his A'th youngest offspring if such an offspring exists (A = 0, 1, …). Now define the increase in the sum of the reproductive values of a male's offspring should he invest ϑ units of paternal care as:

$$\Lambda * \Sigma_{\iota} \{H(\Gamma(\iota)) * \Pi(\iota)\} \qquad \iota = 0, \ldots, \vartheta$$

where Λ is the accuracy with which a male can recognise his own offspring and therefore preferentially direct care toward them and H describes the increase in reproductive value for a single offspring as a function of state. If a male renders one unit of paternal care, this is automatically allocated to his youngest offspring who will gain the most benefit from it; if a second is rendered this will go to his second youngest offspring and so on. Note that this assumes that each offspring can benefit from at most one unit of paternal care. (The exact energetic investment which constitutes one unit of paternal care need not be specified here.)

A backward iteration is now run in which a focal male may split his available energy budget four ways between growth, maintenance, reproduction and paternal care. The dynamic programming equation for males is now:

$$\begin{aligned} F(s, c, t, T) = (1 - \beta(c)) * [\max_i \max_l \max_j E\{ & F(s + \gamma(j), c + \zeta(k), t+1, T) \\ & + 0.5 * \Lambda * \Sigma_{\iota} \{H(\Gamma(\iota)) * \Pi(\iota)\} \\ & + 0.5 * \chi(s, c, i, \Omega_f, \Omega_m) * \lambda(\Omega_f) * \phi * [0.5 * F(8.5, 50, 0, t + 55, T) \\ & + 0.5 * F(8.5, 50, t + 55, T)]\}] \end{aligned}$$

where:

- $F(s, c, t, T)$, $\beta(c)$, i, j, k, $\gamma(j)$, $\zeta(k)$, Ω_f, Ω_m, $\chi(s, c, i, \Omega_f, \Omega_m)$, $\lambda(\Omega_f)$ and ϕ are defined as in the original model.
- l is the investment in paternal care. l may take values in the range:

  $$0 \leq l \leq [\min(\xi(s)*\eta - i, \xi(s) - \alpha(s) - i - \nu*\max(8 - s; - 3))]//\text{ঈ}$$

  where $\xi(s)$, η, $\alpha(s)$ and ν are defined as in the original model, ঈ (in kcal) is one unit of paternal care and // indicates integer division.
- $\iota = 0, \ldots, l/\text{ঈ}$.
- j may take values in the range:

  $$\nu*\max(8 - s; - 3) \leq j \leq \xi(s) - i - \alpha(s) - l$$
- $k = (\xi - i - j - l - \alpha)$
- Λ, $\Gamma(\iota)$, $\Pi(\iota)$ and $H(\Gamma(\iota))$ are defined as above.

The terminal fitness function is unchanged from the original model.

It is assumed that all non-focal males initially split their available energy budget three ways according to the optimal strategy from the original model. A stable strategy is converged upon in the same way as was described for the original model: the time-independent strategy from the backward iteration for a focal male is adopted by all non-focal males; a forward iteration is carried out to determine the population distribution under this strategy and; the backward iteration is run again to recalculate the optimal strategy for a focal male. This process stops when there is no difference between the optimal strategy calculated within one backward iteration and the next. Since male strategy can affect that of females, cycles of female-and-male simulations can now be carried out as described in section 4.3.4 to converge on an optimal strategy for both sexes. The dynamic programming equation and terminal fitness function for females is the same as in the original model. However, the probability of offspring loss ($\sigma(d)$) and the probability of juvenile survival from the time of weaning until maturity (ϕ) must be updated for the female backward iteration in view of the fact that males may now provide offspring care. Expected values for both will depend upon the probability distribution of paternal state and therefore the probability that the father will survive and choose to invest in the offspring at future time points. (Note that in the equation for males above, ϕ is not updated for account for paternal care since the value to the father of providing care is accounted for by the previous term.)

This model could be used to address the following questions:

1. Are males expected to invest in paternal care and if so at the expense of which other fitness-enhancing functions?
2. Does the allowance of paternal care change the expected lifespan for a male?
3. Is paternal care more likely in older individuals?
4. Is paternal care more likely in harsh environments?
5. How do predicted levels of paternal care change with the reliability with which males can identify their own offspring?

6.3 Extensions to the model for early life adversity

6.3.1 Incorporating size at maturation as a behavioural decision

It was assumed, in the model for the effects of early life environment upon speed of life history, that all individuals matured at the same size. In fact, it is known that females who experience adversity in early life tend to be shorter as adults and there may be some adaptive basis for this[100]. Maturing at a smaller size could provide a means - other than accelerated growth - by which the developmental period may be shortened so that reproduction starts sooner. When long-term survival is unlikely, either as a result of poor state or because the environment is unfavourable, maturing at a smaller size reduces the risk of not surviving to reproduce at all. A smaller individual may also be better adapted to a nutritionally-poor environment since her energetic requirements are lower.

It must be assumed, however, that a smaller size also entails some fitness costs since if this were not the case there would never be any incentive to extend the juvenile period and attain a larger size. Smaller individuals may be less successful foragers or less able to secure high-quality mates and the provisioning of offspring may also place a greater energetic burden on a smaller female. This may mean that, even if they are better suited to a poor environment, they are less able to exploit a rich one. If so, this could have knock-on effects for their offspring who may then be of poorer condition than those born to larger females irrespective of their early life environment.

It is here outlined how the model presented in chapter 5 could be extended to include size at maturation as a behavioural decision. The maternal size, which is denoted s_m as before, is now a variable not a constant. Some minimum and maximum size at which females may mature must be defined and these are denoted by s_{min} and s_{max} respectively. For simplicity, let it be assumed that a constant size is still maintained throughout adulthood and that adult mortality rates are still independent of size.

The adult foraging yield and costs of essential functions would now be functions of size - $\xi(s_m; \varepsilon_t)$ and $\eta(s_m; \varepsilon_t)$ respectively - rather than constants. The dependency of foraging yield upon size, at least for adults, could not now be as it was previously for juveniles. For a given decrement in mass, the associated decrement in foraging yield

must exceed that in BMR in order for reproduction to place a heavier energetic burden on a smaller female. This is not the case for the function previously used although it could be for a polynomial function which scaled with mass to a greater power. If a smaller female is assumed to be better suited to a nutritionally-poor environment then a more complex function may need to be considered or a different function could be used for each environment.

Reproductive values for dependent and independent offspring are represented by $F(0, 0, t, T; \alpha, \varepsilon_t)$ and $F(s, c_o, t, T; \varepsilon_t)$ respectively as before. The reproductive value for a mother of size s_m and in condition c_m, at time t prior to the time horizon T, given that she has no existing dependent offspring at the start of period t and that she experiences environment ε_t is now represented by $F(s_m, c_m, t, T; \varepsilon_t)$.

An independent offspring will now decide at the start of a period how much to invest in growth and, should her size at the start of the next period (s') then lie in the interval $[s_{min}, s_{max})$, whether or not to mature at the end of the period. If the expected reproductive success of an adult of size s' at the start of the next period exceed that of an offspring of size s' at the start of the next period (for whatever condition she would enter the next period in), she will mature. When allocation decisions are to be updated each period, dynamic programming equations are now as follows.

The dynamic programming equation for an offspring living independently of her mother at the start of period t is given by:

$$\mathbf{F(s, c_o, t, T; \varepsilon_t) = (1 - \beta(a, c_o))*\max_{\gamma=0}^{\Omega} \{ E_{\varepsilon t+1} F(s', c_o', t + 1, T; \varepsilon_{t+1}) \text{ if } s' < s_{min};}$$
$$\mathbf{\max[E_{\varepsilon t+1} F(s', c_o', t + 1, T; \varepsilon_{t+1}); E_{\varepsilon t+1} F(c_m'''', t + 1, T; \varepsilon_{t+1}) \text{ if } s_{min} \le s' \le s_{max};}$$
$$\mathbf{E_{\varepsilon t+1} F(c_m'''', t + 1, T; \varepsilon_{t+1}) \text{ if } s' = s_m \}}$$

The dynamic programming equation for a mother is given by:

$$\mathbf{F(s_m, c_m, t, T; \varepsilon_t) = (1 - \beta(c_m))* \max_{\alpha=0}^{\xi-\mu} \{A + B + C + D\}}$$

where

$$\mathbf{A = \beta(0, 0)*E_{\varepsilon t+1} \{F(s_m, c_m', t + 1, T; \varepsilon_{t+1})\}}$$
$$\mathbf{B = (1 - \beta(0, 0))* (1 - \beta(c_m')*\beta(1, 0')*E_{\varepsilon t+1, \varepsilon t+2} \{F(s_m, c_m'', t + 2, T; \varepsilon_{t+2})\}}$$
$$\mathbf{C = 1 - \beta(0, 0))* (1 - \beta(c_m')*(1 - \beta(1, 0'))* (1 - \beta(c_m''))*\beta(2, 0'')*}$$
$$\mathbf{E_{\varepsilon t+1, \varepsilon t+2, \varepsilon t+3} \{F(s_m, c_m''', t + 3, T; \varepsilon_{t+3})\}}$$

$$D = (1 - \beta(0, 0))^* (1 - \beta(c_m')^*(1 - \beta(1, 0'))^* (1 - \beta(c_m''))^*(1 - \beta(2, 0''))^*$$

$$E_{\varepsilon t+1,\, \varepsilon t+2,\, \varepsilon t+3} \{F(s_m, c_m''', t + 3, T; \varepsilon_{t+3}) + 0.5^*F(0''', 0''', t + 3, T; \varepsilon_{t+3})\}$$

where c_m', c_m'' and c_m''' are defined as before except that they are now dependent upon the size of the mother as well as on her condition at the start of period t.

The reproductive value for an offspring at the point of conception is given by the same equation as before.

Terminal reproductive values are given by the following:

$F(s_m, c_m, T, T; \varepsilon_T) = 1$ **if** $c_m < 45$

$= 0$ **if** $c_m = 45$

$F(s, c_o, T, T; \varepsilon_T) = 1$ **if** $s > 0$ **and** $c_o < 45$

$= 0$ **if** $s = 0$ **and/or** $c_o = 45$

$F(s_m, c_m, T + 1, T; \varepsilon_{T+1}) = 0$

$F(s_m, c_m, T + 2, T; \varepsilon_{T+2}) = 0$

$F(s, c_o, T + 1, T; \varepsilon_{T+1}) = 0$

$F(s, c_o, T + 2, T; \varepsilon_{T+2}) = 0$

Dynamic programming equations in the case when strategy is fixed in early life are similarly updated if it is assumed that it is only a given rate of ageing and not size at maturation which is committed to in the first period of life. It would be easy to modify these further so that the effect of commitment to maturation size in the first period of life could be explored. The terminal reproductive values for these are the same as above.

This model could be used to address the following questions:

1. Does a harsh environment in early lead to a reduction in the optimal size at which females should mature?
2. How is the reproductive value of a female's offspring affected by the size at which she matures?
3. What effect, if any, does the size of a mother have upon the degree of parent-offspring conflict?

4. Does commitment to a given ageing trajectory in early life make maturation at a particular size more likely than would be the case for an individual who maintains plasticity in her allocation strategy?
5. If a smaller size is better suited to a poor environment and a larger size to a plentiful one, how important is the environment experienced at the time of maturation relative to that in early life in determining final size?

6.3.2 Allowing iterative decisions between mother and offspring

The model could be modified to allow iterative decisions between mother and offspring, with both able to change their allocation strategy over the period of parental investment in response to changes in the environment and the decisions of the other, in the following way.

Any mother who has a dependent offspring at the start of a period now chooses not only the allocation to be made to reproduction but also whether to continue investment in the current offspring or begin investment in a new one. As for the previous model, she may not care for multiple offspring at a time so the decision to begin investment in a new offspring means that any other offspring who has thus far been living under her protection must now live independently. Offspring foraging yield is still defined from the fourth period of life onwards but an offspring is now able to obtain whilst still being in receipt of maternal resources.

The allocation a mother makes to reproduction is now dependent upon the state of any dependent offspring at the start of the period, i.e. $\alpha(c_m; s, c_o, \varepsilon_t)$ rather than $\alpha(c_m; \varepsilon_t)$. The notation c_o' and s' is now used for both dependent and independent offspring. $c_o'(s, c_o; \alpha, \varepsilon_t, \gamma)$ and $s'(s, c_o; \alpha, \varepsilon_t, \gamma)$ describe respectively the condition the size of an offspring at the start of period (t + 1) given that she started period t in condition c_o and of size s, and that during period t her mother invests α in her, she experiences environment ε_t and she invests γ in growth. $c_m''(s, c_o; \alpha, \varepsilon_t, \gamma)$ is now used to denote the condition of a newly-matured mother at the start of period (t + 1) given that she started period t in condition c_o and of size s and that during period t her mother invests α in her, she experiences environment ε_t and she invested γ in growth. Notation for the reproductive values of mothers and dependent offspring is updated as follows and an 'inclusive reproductive value' is now defined for dependent offspring; this is the

expected number of copies of her genes at some time horizon far into the future which result from descendants raised by either her or her mother from the current period onwards.

- $F(c_m, t, T; s, c_o, \varepsilon_t)$ is the reproductive value of a mother in condition c_m, at time t prior to the time horizon T, given that she has a dependent offspring of size s and in condition c_o and that she experiences environment ε_t.
- $F(s, c_o, t, T; c_m, \varepsilon_t)$ is the reproductive value of an offspring of size s and in condition c_o, at time t prior to the time horizon T, given that she is under the protection of a mother in condition c_m and that she experiences environment ε_t.
- $I(s, c_o, t, T; c_m, \varepsilon_t)$ is the inclusive reproductive value of an offspring of size s and in condition c_o, at time t prior to the time horizon T, given that she is under the protection of a mother in condition c_m and that she experiences ε_t. This is half the reproductive value of the offspring herself plus the reproductive value of her mother.

All other notation used in the equations below is as it was in the original model.

The dynamic programming equation for an offspring living independently of her mother at the start of period t is given by:

$$\mathbf{F(s, c_o, t, T; \varepsilon_t) = (1 - \beta(a, c_o))*\max_{\gamma=0}^{\Omega} \{ E_{\varepsilon t+1} F(s', c_o', t + 1, T; \varepsilon_{t+1}) \text{ if } s' < s_m;}$$
$$\mathbf{E_{\varepsilon t+1} F(c_m'', t + 1, T; 0, 0, \varepsilon_{t+1}) \text{ if } s' = s_m \}}$$

and the reasoning behind this is the same as before. The dynamic programming equation for a mother is given by:

$$\mathbf{F(c_m, t, T; s, c_o, \varepsilon_t) = \max(A; B)}$$

$$\mathbf{\text{where } A = (1 - \beta(c_m))* \max_{\alpha=0}^{\xi-\eta} \{ E_{\varepsilon t+1} [(1 - \beta(a, c_o))*}$$
$$\mathbf{[F(c_m', t + 1, T; s', c_o', \varepsilon_{t+1}) \text{ if } s' < s_m;}$$
$$\mathbf{F(c_m', t + 1, T; 0, 0, \varepsilon_{t+1}) + 0.5* E_{\varepsilon t+1} F(c_m'', t + 1, T; 0, 0, \varepsilon_{t+1}) \text{ if } s' = s_m]}$$
$$\mathbf{+ \beta(a, c_o)*F(c_m', t + 1, T; 0, 0, \varepsilon_{t+1})] \}}$$
$$\mathbf{+ \beta(c_m)* 0.5* E_{\varepsilon t+1} F(s, c_o, t, T; \varepsilon_t);}$$

$$B = (1 - \beta(c_m))^* \max_{\alpha=0}^{\xi-\eta} \{ E_{\varepsilon t+1} [(1 - \beta(0, 0))^* F(c_m', t + 1, T; 0', 0', \varepsilon_{t+1}) + \beta(0, 0)^* F(c_m', t + 1, T; 0, 0, \varepsilon_{t+1})]\} + 0.5^* E_{\varepsilon t+1} F(s, c_o, t, T; \varepsilon_t)$$

A is her maximum expected reproductive value at the start of period (t + 1) in the case where, if she survives the current period, she continues to invest in the current offspring. B is her maximum expected reproductive value at the start of period (t + 1) in the case where, if she survives the current period, she begins investment in a new offspring.

A can be understood as follows. The mother will survive the current period with probability $(1 - \beta(c_m))$. If her offspring also survives - which she will with probability $(1 - \beta(a, c_o))$ - the mother's reproductive value in the next period is given by $F(c_m', t + 1, T; s', c_o', \varepsilon_{t+1})$ if her offspring is still a juvenile at the start of the next period and by $F(c_m', t + 1, T; 0, 0, \varepsilon_{t+1}) + 0.5^* E_{\varepsilon t+1} F(c_m'', t + 1, T; 0, 0, \varepsilon_{t+1})$ if her offspring reaches maturity at the start of the next period. If her offspring does not also survive, her reproductive value in the next period is $F(c_m', t + 1, T; 0, 0, \varepsilon_{t+1})$. The mother will not survive the current period with probability $\beta(c_m)$, in which case her expected reproductive value is that of her offspring (now living independently) multiplied by the degree of relatedness between them.

The logic behind B is similar. The mother's survival probability for the current period is the same: $(1 - \beta(c_m))$. The probability that a new offspring will survive the current period is $(1 - \beta(0, 0))$ and the reproductive value of the mother in the next period is then $F(c_m', t + 1, T; 0', 0', \varepsilon_{t+1})$. If the offspring does not survive, which occurs with probability $\beta(0, 0)$, the mother's reproductive value in the next period is $F(c_m', t + 1, T; 0, 0, \varepsilon_{t+1})$. The mother's reproductive value for the current period also includes that of the offspring who left her protection at the start of the period - $F(s, c_o, t, T; \varepsilon_t)$ - multiplied by the relatedness between them.

Given i) that an offspring enters the period under the protection of her mother, ii) that her mother survives and iii) that her mother has chosen to invest in her a given

amount, the offspring's allocation decision - should she survive the period - is determined by the following dynamic programming equation:

$$I(s, c_o, t, T; c_m, \varepsilon_t) = \max_{\gamma=0}^{\Omega} \{ E_{\varepsilon t+1} [0.5*F(s', c_o', t + 1, T; c_m', \varepsilon_{t+1}) + F(c_m', t + 1, T; s', c_o', \varepsilon_{t+1})] \text{ if } s' < s_m;$$
$$E_{\varepsilon t+1} [0.5*F(c_m'', t + 1, T; 0, 0, \varepsilon_{t+1}) + F(c_m', t + 1, T; 0, 0, \varepsilon_{t+1})] \text{ if } s' = s_m \}$$

The offspring will adopt a strategy which maximises her expected reproductive value plus the expected reproductive value of her mother *from other offspring* at the start of the next period. Since the *total* reproductive value of the mother in period (t + 1) includes that of the current offspring multiplied by the relatedness between them (0.5), this is equivalent to the offspring maximising the total reproductive value of the mother plus half her own at the start of the next period. If the offspring's investment in growth is insufficient for her to reach maturity by the start of the next period, then the reproductive value of her mother will be $F(c_m', t + 1, T; s', c_o', \varepsilon_{t+1})$ and her own reproductive value will be $F(s', c_o', t + 1, T; c_m', \varepsilon_{t+1})$. If the offspring reaches maturity at the start of the next period, the reproductive value of her mother will be $F(c_m', t + 1, T; 0, 0, \varepsilon_{t+1})$ and her own reproductive value will be $F(c_m'', t + 1, T; 0, 0, \varepsilon_{t+1})$.

It now remains to define the reproductive value for an offspring who enters the period under the protection of her mother. This is given by:

$$F(s, c_o, t, T; c_m, \varepsilon_t) = F(s, c_o, t, T; \varepsilon_t)$$

if her mother chooses not to continue investment in her.

$$F(s, c_o, t, T; c_m, \varepsilon_t) = (1 - \beta(c_m))*(1 - \beta(a, c_o))*$$
$$\{E_{\varepsilon t+1} F(s', c_o', t + 1, T; c_m', \varepsilon_{t+1}) \text{ if } s' < s_m;$$
$$E_{\varepsilon t+1} F(c_m'', t + 1, T; 0, 0, \varepsilon_{t+1}) \text{ if } s' = s_m\} + \beta(c_m) * F(s, c_o, t, T; \varepsilon_t)$$

if her mother chooses - conditional upon the mother's own survival - to continue investment in her.

If her mother chooses not to invest in her, the offspring's reproductive value is the same as for an individual in the same state who was living independently at the start of the period. The expression for when the mother would choose - conditional upon her own survival - to continue investment in the current offspring rather than begin investment in a new offspring may be understood as follows. The first term gives the probability that the mother survives multiplied by the probability that the offspring survives and her expected reproductive value at the start of the next period given the investment the mother would then make. If the offspring remains a juvenile at the start of the next period her reproductive value is given by $F(s', c_o', t + 1, T; c_m', \varepsilon_{t+1})$; if the offspring reaches maturity at the start of the next period her reproductive value is given by $F(c_m', t + 1, T; 0, 0, \varepsilon_{t+1})$. The second term gives the probability that the mother dies multiplied by the reproductive value of the offspring given that she must now live independently.

Terminal reproductive values are given by the following:

$\mathbf{F(s, c_o, T, T; \varepsilon_t) = 1}$ **if $s > 0$ and $c_o < 45$**

= 0 otherwise

$\mathbf{F(s, c_o, T, T; c_m, \varepsilon_t) = 1}$ **if $s > 0$ and $c_o < 45$**

= 0 otherwise

$\mathbf{F(c_m, T, T; s, c_o, \varepsilon_t) = 1.5}$ **if $c_m < 45$ and $s > 0$ and $c_o < 45$**

= 1 if $c_m < 45$ and ($s = 0$ or $c_o = 45$)

= 0.5 if $c_m = 45$ and $s > 0$ and $c_o < 45$

= 0 otherwise

Equations for the cases where allocation decision is constrained in early life are similarly defined and terminal reproductive values for these would be the same.

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