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The Evolution of Avian Growth Rates in Variable Environments

A thesis presented in partial fulfillment of the requirements for the degree of

Ph.D.

in

Evolutionary Ecology

at Massey University, Palmerston North, New Zealand.

John Clive Ashton

2000

ERRATA

Chapter 1

(i) Page 3, line 9, "glow growers" should be "slow growers".

(ii) Page 3, lines 30-31 imply that the Fieldfare is a ground nester, whereas it actually has cup nests in trees.

(iii) Page 6, line 10, "that a the" should be "that the"

(iv) Page 9, line 13, "ideals" should be "ideal"

Chapter 2

(i) Equation 2.1 should be:

$$Mt = Ma - (Ma - Mo).\exp\{-\ln(\frac{1-\frac{Mo}{Ma}}{0.5}).(\frac{t}{t_{50}})^p\}$$

(ii) In regard to the section in the methods that describes the use of phylogenetic contrasts, it should be noted that all regressions were forced through the origin.

Chapter 3

(i) Table 3.3 legend, "all three independent variables. *V*, *FF* and *PD*..." should be "both independent variables. *V* and *FF*..."

(ii) Fig. 3.3 legend, last sentence refers to an earlier draft.

(iii) Page 54, second paragraph, reference to Fig. 2.2 should be to Fig. 2.1.

Chapter 4

(i) In Fig. 4.9 *Junco hymenalis* is classified as a protected nester, whereas nest sites are in fact variable for this species, sometimes cavities and sometimes in the open.

(ii) Page 57, line 20, "it not specify" should be "it does not specify"

(iii) Fig. 4.5, "horizonal" should be "horizontal".

Chapter 5

(i) Page 82, 3rd paragraph "If energy is....periods of starvation" is a repeat of the previous paragraph.

(ii) Fig. 5.6 legend, "horizonal" should be "horizontal".

Chapter 6

Page 122, 2nd sentence, should read "With an ideal food supply, as the maximum fat deposition rate increases, *GRI* increases, and the probability of fledging decreases by a small amount"

Chapter 7

(i) The methods should state the fact that approval from the Massey University Animal Ethics Committee was obtained for the brood swapping and deprivation experiments.

(ii) "Control" chicks in the deprivation experiment are not controls in the sense that conditions are normal for those chicks (e.g., they may have increased food supply during the experiment) and should be referred to as "non-deprived" chicks.

(iii) Table 7.4 legend, "Treatment chicks (C)..." should be "Treatment chicks (T)..."

Chapter 9

Page 179, paragraph 2; this argument applies to *case* probabilities only. The frequency interpretation of *class* probabilities is compatible with determinism and is an objective theory of probabilities.

Appendix 1

Parus major, *P. Montanus*, and *Sturnus vulgarus* are wrongly recorded as unprotected nesters in Table A1.2, whereas in fact they are protected nesters.

Abstract

Tubenoses and swifts develop slowly, and often have a variable food supply. Lack (1968) attributed this to convergent evolution, arguing that slow growth is an adaptation allowing survival in environments with a variable food supply. In this thesis, I test whether there is a general relationship among bird species between slow growth rate and variability in food supply. I analysed data on nestling period, growth rates for mass and wing length, and variability in food supply in birds using phylogenetically independent contrasts.

Variability in food supply may be correlated with feeding frequency, and growth rate is correlated with predation risk. I included these potential confounds in my analysis. Variability in food supply was correlated with nestling period, and negatively correlated with mass and wing growth rate, taking average feeding frequency and predation risk into account. I show that nest site preference is incompletely coadapted with growth rate. The correlations between growth rate and variability in food supply could also be explained by the proximate effect of environmental variability on growth. I therefore tested predictions of Lack's hypothesis, in comparison to those of growth models assuming facultative growth adjustments in response to variability in food supply. This further supported Lack's hypothesis.

While Lack proposed that slow growth is an adaptation to variability in food supply, he did not explain the underlying mechanism. I examined three possible mechanisms, along with two alternative explanations where slow growth is not an adaptation to a variable food supply, and tested them with comparative data. I developed two of these models using computer simulations which predicted that survival is increased by reducing maximum lean tissue growth rates and increasing maximum fat deposition rates when food supply is variable. I tested predictions from these models using experiments on the Welcome Swallow, corroborating a model that predicts that lean tissue growth is prioritised over fat deposition but that fat deposition is facilitated by reduced lean tissue growth rates. I also tested whether swifts and tubenoses are adapted to an unpredictably, or predictably, variable food supply, and discuss the degree to which chicks of swifts and tubenoses are well designed for survival in environments with a variable food supply.

Acknowledgments

The primary idea in this thesis that slow growth is an adaptation to a variable food supply belongs to Ed Minot. He played a major role in helping me to develop the ideas early on in my study. My doctoral supervisor Doug Armstrong has made more suggestions and provided me with more insights than I can count. I am deeply grateful for the enormous amount of time and effort he has devoted to the role of supervisor. I am also grateful for the confidence he has shown in me, especially in his support for my bid for a scholarship. My wife Louise has read passages of the thesis, helped me with field-work, took the photographs that appear in Chapter 7, and helped me in innumerable other ways. Most importantly I owe to her the opportunity that I have had to undertake a doctoral thesis at all. Her support has been immeasurable. This thesis would also have been impossible if it were not for the financial support I had from a Massey Doctoral Scholarship. Bob Jolly listened patiently to my early ideas and offered helpful advice. He and Aileen Jolly provided me with lodgings free of charge near Massey University, which was a tremendous help. Clare Veltman gave me valuable feedback on my thesis proposal and made numerous helpful suggestions. Ian Jamieson suggested the deprivation experiment that appears in Chapter 7. Discussions with Wayne Linklater ultimately lead to Chapter 3. Conversations with Dave Lambert made me consider more alternative hypotheses and interpretations than I would have otherwise. Many ideas in this thesis are direct responses to criticisms he raised in these discussions. Ian Henderson gave me useful advice on phylogenetic analysis. Suggestions by Brian Springett helped me to select the method that I used to sample insects in Chapter 7. My ideas on adaptation that appear in Chapter 9 have been improved with the help of suggestions and criticisms from G.C.Williams, D.C. Dennett, Richard Dawkins, and John Catalano. Professors Williams and Dawkins took the time to send me important references. Charles Sibley was kind enough to send me up-to-date phylogenies for the tubenoses and other taxa. Thanks are due also to the various orchard-owners of Twyford, Hawkes Bay, who not only tolerated my skulking about under their culverts and bridges in my pursuit of Welcome Swallow data, but also offered me observations on Swallow breeding sites and dates.

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Chapter 1

Introduction

In his 1968 book "Adaptations for Breeding in Birds" David Lack drew a surprising conclusion[1]. In 1951 he and Elizabeth Lack had published a paper in *Ibis* titled "The breeding biology of the Swift *Apus apus*". They had noted that Swift was an ecological puzzle. Only 40 grams full-grown, but its chicks took as long to leave the nest as species several times its size. Early in development the chick appeared little more than a bag of fat with a gut and tiny appendages. This was evidently for good reason, because when storms closed in the parents could spend days before being able to bring a substantial amount of food to the nest. In such harsh conditions the chicks of most species would perish. Most species, but not all species. One group of birds, they realised, had a strikingly similar nestling stage to the swift, a group that, superficially, was as distinct from the swift in other ways as dolphins are from anteaters. That other group was the tubenoses (the Procellariiformes). The tubenoses are a group of birds including the albatrosses that forage almost exclusively in the open ocean, an environment very distant from the middle reaches of the atmosphere where swifts forage. As early as 1888 ornithologists had puzzled over the peculiar growth of the tubenoses. Species such as the White-Faced Storm-Petrel and the Sooty Shearwater had since been intensively studied in New Zealand by Lance Richdale. In good conditions many species of tubenose could put on a huge amount of weight during nesting, only to lose 50% of it as the time for fledging approached. Most of the excess weight was fat, and the chicks of the tubenoses too, it appeared, could survive prolonged fasts. More interesting still, the chicks grew in other respects very slowly - just like the Swift - and took, what is compared to other seabirds, an extremely long time to fledge. The conclusion that Lack drew was that the similarities of the breeding ecology of the Swift and the tubenoses is the result of convergent evolution.

[1] Formal citations are kept to a minimum here, as the works discussed are cited in later chapters.

Convergent evolution and the insurance hypothesis

Textbook examples of evolutionary convergence tend to focus on external shape, hence the sharks, swordfishes, porpoises and ichthyosaurs. But convergence may appear in any aspect of biology, including life history characteristics. Convergence is clearly one of the guiding principles behind general evolutionary explanation. For instance, the classic theory of *r* and *k* selection; species for which conditions suitable for reproduction come fleetingly and erratically should evolve rapid breeding and growth, whereas species in more stable environments should converge on a strategy of slower reproduction and increased ability to compete for other resources.

Lack's theory was also about evolution in a variable environment, but variability over time scales much shorter than that applicable to *r* and *k* selection theory. He argued that while the food sources of swifts and tubenoses are very different on the surface, in another way they are very similar. A spell of bad weather, an unlucky spell of foraging, and the chicks of both groups will go without food for a considerable period. The swifts and tubenoses were, he continued, convergently adapted for growth in environments with a variable food supply. The chicks grew slowly and accumulated fat to avoid starvation when the food supply dried up, as it did periodically.

Closer observation of species of tubenoses seemed to confirm this idea (reviewed in Chapter 2). Most impressive of all the tubenoses are the storm-petrels, tiny birds - no larger than a thrush - that confront the gales and storms of the open ocean, and somehow manage not only to survive, but to bring enough food back to shore to raise a chick. In extreme cases chicks will go up to 10 days without food and still continue to develop to fledging when feeding recommences. Similar sized chicks of land birds would perish after only a fraction of the time that Storm-Petrel chicks regularly wait for provisions.

Whenever researchers looked at species of birds that forage in the open ocean, they discovered a recurring pattern. Compared to other terns and noddies, the sooty tern *Sterna fuscata* and the black noddy *Anous minitus* grow very slowly. J.B. Nelson (1978) found that in the Sulidae (that group of birds that includes the gannets) the boobies have by far the slowest growth rates. All of these birds feed far out to sea while their fast growing relatives forage close to the shoreline.

David Lack turned his attention to the swiftlets, the smaller cousins of the swifts. He found that the further above the ground a species of swiftlet foraged, the less dependable its food supply, and the slower its development. Lack also compared the development of swifts to the development of swallows, which bear a strong physical resemblance to swifts. Swallows and swifts both forage on aerial insects but swallows forage close to the ground, to water, and to vegetation, where flying insects are protected from the storms that rack the middle altitudes - i.e., swallows have a more dependable food supply than swifts. Swallows, whilst not exactly fast developers, were not glow growers like the swifts. It seemed clear to Lack that slow growth was part of a collection of adaptations that insured chicks against starvation when the food supply crashed. If certain species were paying for this insurance with slow growth, then the swifts and tubenoses were paying the highest premiums.

Challenges and complexities

Since Lack's seminal work the case for his insurance hypothesis has become somewhat less secure (reviewed in Chapter 2). Beginning in the 1960's and continuing to the present Robert Ricklefs has published numerous important papers on the ecology, physiology and evolution of growth rates in birds. Some of Ricklefs' findings have supported Lack's ideas, including his insurance hypothesis. However, at other times his findings have contradicted Lack's insurance hypothesis, especially as it applies to the tubenoses.

Some authors have argued that it isn't that swifts and tubenoses have a *variable* food supply, rather that they are fed relatively infrequently but at a fairly constant rhythm. What limits their growth rate is an overall reduction in food rather the alternation of booms and crashes of the food supply. Furthermore, the swifts and tubenoses do not just share similarities in their food supply, but in other aspects of their nesting as well. Both types of bird are protected from ground predators, the swift by nesting in cavities on cliffs, and tubenoses by nesting on remote islands. In only the last decade and largely due to the work of Thomas Martin, evidence has mounted that the vulnerability of chicks to predators is a crucial determinant of their growth rates and other life history characteristics (reviewed in Chapter 4). Where predation is severe the pressure is on to grow rapidly and leave the nest as soon as possible. In some species where ground predators are an especial threat - i.e., the skylark and fieldfare who nest

in open fields, chicks grow at a breakneck rate, achieving up to 90% of their growth in as little as 4 days. So great is the pressure to reach a safer environment, that the chicks fledge whilst their wings have yet to reach full development. This contrasts with the growth of swifts, tubenoses, and even swallows, which first grow to adult weight, but then linger in the nest as their slowly growing wings edge toward maturity.

There are other problems. In some slowly growing species, the energy spent on growth is small compared to the energy spent on general metabolism (reviewed in Chapter 5). Presumably chicks could grow much faster with only a small increase in food. Turning this around, a large reduction in growth rate means only a small reduction in the amount of food needed for the chick to grow. In other words, slow growth doesn't save much energy, and if this is true, then the insurance hypothesis would appear to be in trouble.

These doubts have lead some researchers such as Ricklefs to cast about for alternative theories (reviewed in Chapter 5). Maybe there is some specific nutrient, perhaps an amino acid, that is lacking in the diet of birds that feed in the open ocean? But in that case, why the huge fat deposits in these species? One suggestion is that in obtaining enough of the scarce nutrient, the chicks consume more energy than they can use. This excess energy is deposited in a "sink" - i.e., in fat deposits. But this raises more problems than it answers. It is not uncommon for an organism to accumulate more of a substance than it can profitably use, but the usual strategy is simply to excrete and jettison the excess in some way, a relatively inexpensive strategy. For example, aphids have a low-nutrient/high-energy diet, and excrete dissolved sugar as waste. By contrast, depositing unusable energy as fat involves considerable costs not only in the digestion of dietary energy, the subsequent re-conversion of ATP into fat, and the production of enzymes necessary for fat production, but also the growth and maintenance of fat cells and their associated blood vessels. Furthermore, the fat stores do a good job of supporting chick metabolism during fasts. This requires a complex ensemble of enzymes and biochemical feedback systems. All this suggests that there is much more to fat than an energy dump. Perhaps it is insulation against the cold? The distribution of fat about the chick suggests otherwise, and the fattest chicks are not necessarily in the coldest environments. Alternatively, chicks that do not receive post-fledging parental care may need a reserve of fat whilst learning to forage. This may

well be true, but it cannot possibly explain why the chicks of many species lose most of their body-fat *before* fledging.

An alternative to the slow-growth/insurance hypothesis is that slow growth and large fat deposits are associated merely by coincidence. Under this hypothesis, tubenoses and swifts accumulate fat to avoid starvation, but this isn't a result of slow growth. Slow growth is for some other reason.

One possible reason for slow growth in environments with a variable food supply is that adults may have to abandon their eggs for long periods (reviewed in Chapter 5). To avoid drying out, the eggs must restrict the amount of gas that passes through their shells, and this means that the embryos grow slowly. Perhaps then, slowly growing chicks are simply a carry-over from slowly growing embryos? Indeed, some biologists have argued that different aspects and periods of development are tightly coupled - i.e., no one aspect of development may evolve to any extent independently of any other aspect (reviewed in Chapter 5). Variation among the different aspects of development will be so tightly coupled, genetic variation so dominated by pleiotropic interactions, that selection on one aspect of development will accompanied by a flood of consequences.

If this scenario is correct, then as far as adaptive studies of growth rates that restrict themselves to some isolated period or aspect of development are concerned, experimental analysis is all but impossible. The developing organism must be treated as one interconnecting whole, or not at all. The trouble with this is that there is no shortage of theories that explain the timing of life history events. For instance, some life history theories predict a delayed age at first reproduction (e.g., Charnov 1993). If development can only be changed as a whole, then this means slow growth rates, long incubation, long nestling periods, the pace of the whole of development in other words.

On the other hand, other theorists (reviewed in Chapter 5) have argued that pleiotropic interactions are themselves the result of evolution and can be adjusted by modifier genes. Even if additive variation is limited in the short term, mutation provides ample new additive variation over evolutionary time scales. Perhaps most importantly, development is not a simple system of branching causation that the coupling hypothesis assumes. In mammals, prenatal and postnatal growth rates vary independently among species and in many arthropods the evolution of the early stages of development is virtually independent of the evolution of later stages. Development

is not so much a system of causation in which everything depends on everything else, but one of partially isolated modules that may evolve somewhat independently. As already noted, the relationship between mass and wing growth rate varies hugely among birds. Wings appear to be a "module" that may evolve in growth rate partially independent of overall mass (and if anything, correlations between "modules" are competitive rather than additive - see below). If this is correct, then I cannot automatically assume that the evolution of long incubation periods necessitates the evolution of long nestling periods.

If development is sufficiently modular, then growth rates may vary significantly over the course of development. Williams (1966), for instance, argued that a the duration of a developmental stage should be inversely proportional to mortality rate at that stage. However, the weakness with William's theory is that it different types of mortality have different evolutionary consequences, e.g., deaths from starvation should have a different affect on the evolution of growth rates than deaths from predation (Ricklefs 1969a).

Perhaps more importantly for the insurance hypothesis, it has now become less certain that all species of tubenoses have an variable food supply (reviewed in Chapter 2). Studies on some species have found that, at least during the time that they were observed, chicks had a reliable food supply and did not suffer long spells deprived of food. If most species of tubenose have a reliable food supply, then once again, the insurance hypothesis is in jeopardy.

The insurance hypothesis, and specifically the theory that swifts and tubenoses are ecological analogues by virtue of the insurance effect, has, whether it is right or wrong, all the features of a bold conjecture, the kind of speculation that the philosopher Karl Popper urged scientists to make. It is specific in its predictions yet sweeping in its application. If corroborated, it solves an outstanding problem in ornithology, and it reduces a large number of otherwise unconnected facts to a general explanation. But even if refuted, it gives rise to a profusion of new problems and avenues of research.

Among the problems that the insurance hypothesis has produced, the two main ones appear to be (i) whether the species in question really do have a variable food supply, and (ii) the absence of a plausible physiological theory relating slow growth to avoidance of starvation. In this thesis, I address both of these problems, and in doing so subject the insurance hypothesis to rigorous testing.

Thesis outline

In **Chapter 2**, I test the insurance hypothesis using comparative data for a wide range of bird species. If the hypothesis is correct, then a variable food supply should correlate with slow growth when a range of species are compared. In the course of this chapter I address a major sub-problem, that is, how to measure "variability in food supply". The idea of an undependable or variable food supply is an intuitive one, but for comparative tests it must be turned into a measurable variable. Then there are other sub-problems, the problem of allometry (the scaling relationships between biological characters that may confound comparative studies) and the problem that birds with a variable food supply may also tend to have long average intervals between feeds. I therefore need to invent a set of methods prior to beginning my comparative tests, and assess the reliability of these methods. I used the method of phylogenetically independent contrasts to then address the question of whether (i) species with a variable food supply have evolved slow growth rates, (ii) whether tubenoses and swifts do in fact - as Lack argued - have a variable food supply.

The next problem is that correlations in variables between species, or between phylogenetically independent units, may not be the result of evolution at all. Instead, correlations of this kind may be due to proximate responses to environmental variability. This is a problem that is often mentioned, but to my knowledge has never been directly addressed. Hence, in **Chapter 3**, I invent methods for deciding between "evolutionary and proximate" explanations of the kind of correlations that may arise in comparative studies. I then apply these methods to the results of Chapter 2. My methods, however, assume that I can reliably estimate minimum nestling periods and/or maximum growth rates. Minima and maxima are sample size dependent, and are never as reliably estimated as means (or standard deviations). To deal with this new problem, I invented yet another set of methods. In doing so I constructed a set of models relating lean tissue growth rates to overall growth rates (which includes fat deposition). These models include one that incorporates the assumptions of the insurance hypothesis. I am then in a position to subject the insurance hypothesis, and alternative models, to more rigorous testing than I achieved in Chapter 2.

In **Chapter 4**, I try to control for the effects of variation in nest predation. Having done this, and having confirmed the nest predation hypothesis once again with

new data and with new methods, I ask whether the growth rates evolve gradually or suddenly when there is a change in nest type - i.e., are nesting preferences always tightly coadapted with growth rates, or, is the coadaptation less complete in species that have undergone a recent change in nest type. To answer this question I develop a new use of phylogenetic contrasts, one that tests hypotheses about evolutionary transformations - i.e., a new *directional* comparative method. However, before I draw any conclusions from my data, I subject my method to careful analysis, using simulations to test a range of interpretations of possible results.

Chapter 5 consists of five sections, focusing on five alternative explanations of the results of my comparative tests. Two of the explanations are unrelated to the insurance hypothesis. The first of these is a variety on the "nutrient limitation" hypothesis discussed earlier. To test this hypothesis, I first clarify its predictions, second, invent a method of quantifying one of these predictions, and third, use comparative data to test the prediction. The second of the alternative explanations is the idea - discussed earlier - that the evolution of postnatal growth rates is a developmental byproduct of the evolution of prenatal growth rates. To test this idea I have to invent a general method - using phylogenetic contrasts - for testing the independence of evolution in correlated traits.

The other three hypotheses considered in Chapter 5 are attempts to explain how - physiologically - the insurance effect might work - i.e., why slow growth is correlated with the ability to survive food shortages. The first hypothesis is the simplest, slow growth saves energy which is then converted into fat. The second hypothesis is that metabolic rate is the factor linking growth rates and survival during food shortages. I test this idea by using comparative data to check for correlations between metabolic rates and growth rates. The third hypothesis is my own invention, highly speculative, and a "bold" conjecture in the spirit of David Lack and Karl Popper. I argue that if a chick is to accumulate large deposits of fat, then it must develop specialised tissues for the task. These tissues are not just fat cells, but also blood vessels that support fat cells, and cells that produce the enzymes involved in the building up and breaking down of fat. I argue that these tissues compete with other tissues not only for energy, but for the allocation of cells during differentiation and for a supply of blood vessels. Hence, I first argue that there is a tradeoff between the development of fat, and the development of lean tissue and then test one of the predictions that follow from this using

comparative data. Other researchers have argued that various tissues compete during development, and that this plays a crucial role in overall growth rate, but I am unaware of anyone treating fat as a tissue like any other, and one that may compete with other tissues during growth.

In **Chapter 6**, I return to Lack's original idea - i.e., that a slow growth rate saves energy, and that this saving helps survival during food shortages. Previous work has assumed that the relatively low costs of growth mean that reduced growth rates will save so little energy as to be insignificant. I try a different approach using empirical data from the energy budgets of growing storm-petrels to construct a computer model of a growing chick. In this model, each day involves the intake of energy and conversion of this into lean tissue and fat. Energy is also used up in general metabolism. By varying the growth rate of simulated chicks, I am able to compare the effect of a slow growth rate in simulations where the energy supply is ideals, to simulations to where it is variable. If Lack's insurance hypothesis is correct, then simulated chicks with slow growth rates should have a survival advantage in environments with a variable food supply. I then construct a number of variations on the basic model. First, I determine whether increasing potential fat deposition rates in proportion to decreasing lean tissue growth rates changes the survival rates of simulated chicks with a variable food supply. I then go on to modify my first model to correspond to a range of different assumptions. These assumptions made an initial appearance in the models in Chapter 3. With my computer simulations I am able to assess whether the predictions I draw from the models in Chapter 3 are sound. Having assessed the computer models, I then turn to testing them with empirical data. The data requirements for these tests are stringent. Nevertheless I am able to find to draw some conclusions about the nature of growth in several species of bird.

In **Chapter 7**, I use direct experimental manipulation of a single species, the welcome swallow *Hirundo neoxena*, to test my growth models. This swallow is a good test subject because it occupies an intermediate position in terms of growth rates and variability in food supply. I conducted these experiments over two breeding seasons, making over 3000 measurements of weights and wing-lengths of growing chicks, hence constructing a detailed picture of swallow growth. These data help to explain how the swallows' growth pattern may be adjusted to their food supply.

In **Chapter 8**, I return to the problem of clarifying the concept of variability in food supply. I argue that it is not just variability in food supply that is important, but also the predictability of the variability. "Predictability" is defined as variability that is autocorrelated over the short to medium term. I conjecture that there are two ways to deal with environmental variability, an insurance strategy or a "tracking" strategy, and that tracking strategies will be most effective in predictably varying environments. I conjecture that regulation of provisioning by parents, and the regulation of growth priorities in chicks, are examples of tracking strategies. I then test these conjectures using comparative data, and as a result am able to criticise some current ideas about provisioning strategies in tubenoses.

Throughout the thesis I test hypotheses about adaptation. In **Chapter 9** I argue that my methods are part of a general *comparative* method for studying adaptation. I argue that the study of adaptation is the study of good design, drawing primarily on the ideas of R.A. Fisher and G.C. Williams, but also drawing parallels between good design, problem solving, (im)probability, and information content. I show how my work can be interpreted as an analysis of good design for growth in environments with a variable food supply.

In **Chapter 10** I summarise my findings, draw conclusions about Lack's insurance hypothesis, and suggest pathways for future research.

Chapter 2

Comparative tests of Lack's insurance hypothesis

Introduction

Lack argued that chicks would evolve adaptively to maximise chick survival under different conditions. He therefore predicted that the rate of development of chicks would evolve to become slower in species characterised by low predation risk, low feeding frequencies, and/or high variability in food supply. The role of predation pressure has been tested with broad comparative studies which support Lack's prediction that species with low predation rates tend to have slow development (Ricklefs 1969b, Martin 1995, Bosque and Bosque 1995, Martin and Clobert 1996). Case (1978) showed that families of birds with low feeding frequency (i.e., long feeding intervals) have relatively slow growth rates. A lower feeding frequency may also result in less food being supplied to the chick over the nestling period. The weight of the chicks of some species of the Procellariiformes drops markedly between feeds (see, for example, growth curves in Berruti 1979). The digestive tract must therefore be emptied between feeds in these species, suggesting that feeding interval limits food intake. There have been several intraspecific studies showing that chick growth rates are often limited by food supply (Rice and Kenyon 1962, Lack 1968, Harris, 1966, Ricklefs 1969c, Ricklefs and White 1981, Ricklefs 1983, Shea and Ricklefs 1985, Tarbuton 1987, Martin 1992, Navarro 1992, Klaasen 1994). There have also been several intraspecific studies testing the effect of variability in food supply on chick growth rates (e.g. Zotier 1990, Becker and Specht 1991, Hamer and Hill 1993, Hamer and Thompson 1997, Boersma and Parrish 1998), or the effect of variability in food supply on chick or adult fat reserves (e.g. Schaffner 1990, Navarro 1992, Bednekoff and Krebs 1995). However, there have been no interspecific comparative studies testing for evolutionary changes in growth rate with variability in food supply.

Average food supply and variability in food supply pose related but different problems for the growing chick. A chick with a low average food supply must maximise its growth rate within the constraint of the available energy and nutrients. A chick with a variable food supply also needs to maximise its growth rate within the

constraints of average food supply, but has the additional problem of surviving during food shortages.

The effect of variability in food supply is an important aspect of Lack's theories. Many species have low nest predation or low feeding frequency, but it is the variability hypothesis that Lack put forward to explain the extreme life history and development characteristics of swifts and the Procellariiformes. However, feeding interval and nest predation are both factors that may covary with nestling period and growth rate. It is therefore necessary to take these factors into account when testing for the effect of variability.

Several researchers have addressed Lack's insurance hypothesis using data from one or a few species. Bryant and Gardiner (1979) argued that the large amount of fat in house martin (*Delichon urbica*) chicks is an adaptation to a highly variable food supply, and similar arguments have been made for other species of aerial insectivore (Bryant and Hails 1983, Marin 1997, McCarty and Winkler 1999). Ricklefs *et al* (1985) argue that growth rates in storm-petrels, and perhaps all pelagic seabirds, are not an adaptation to food limitation or a variable food supply, but a result of extreme precocity of thermogenesis due to early maturation of leg muscle tissue. Functionally mature tissue has a correspondingly low amount of embryonic tissue and therefore grows slowly (Ricklefs *et al* 1994). Delayed wing development in pelagic seabirds may be needed to accommodate precocity of development in leg tissue. However, differential growth rates of body components has itself been put forward as an adaptation to a variable food supply. Brood swapping experiments with pelagic seabirds suggest both food limitation and delayed growth of lean tissue in undernourished chicks (Hulsman and Smith 1988, Congdon 1990). These authors argue that delayed wing growth is part of a prioritisation system that ensures that energy is used preferentially for maintaining body mass rather than lean tissue growth. Shea and Ricklefs (1996) studied the growth of six tropical pelagic seabirds with slow development and high lipid deposition, but found no evidence for extreme variability in their food supply. From studies of the development and breeding biology of tropicbirds, Schaffner (1990) argues that pelagic seabirds are adapted to a variable food supply through a suite of a developmental features referred to as "adaptive hyperlipogenesis", which includes slow lean tissue growth rates and increased lipogenesis and adipocyte production. Hamer and Thompson (1997), however, found a

diversity of growth and feeding patterns amongst the Procellariiformes, and argued that there is no unified explanation for lipid deposition and slow growth in that group.

Lack's insurance hypothesis is therefore supported by some studies but not others. However, there has been no systematic attempt to test the hypothesis using broad comparative data, and little consensus over what constitutes a variable food supply. In this chapter, I test Lack's hypothesis that slow growth rate is an adaptation to variability in food supply using data collected from the literature on a broad range of bird species.

Methods

I used data from a total of 102 species in 25 families (Appendix 1). Of these I used data from 52 species in 19 families to calculate variability in food supply from variability in mass growth. I only used altricial and semi-precocial species because the concepts of nestling period and feeding frequency used in this study are not applicable to precocial birds.

I gathered data on growth, nestling period, and provisioning. Growth data were obtained from papers reporting on weight and wing growth. Where data were presented graphically, coordinates were read off using a millimetre grid on a transparency. Papers were not used if growth data came from only one brood, or data did not adequately cover the nestling period. Nestling periods were obtained from papers reporting number of days from hatching to fledging. If data were available from more than one paper, I took the average value. Provisioning data were obtained from papers reporting average number of feeds per day (Case 1978). In some cases both feeds per time and foraging time per day were available, so feeds per day could be inferred.

Parameter Estimation

Growth rate and variability in food supply are complex concepts that have to be reduced to relatively simple indices if they are to be comparable amongst species. The indices that I used in this chapter are described in detail below. Abbreviations and the method of calculation for each index are summarised in the results (Table 2.2).

Growth Parameters

I calculated the growth rates for body mass and wing-length for each species by fitting the Janoschek equation (Equation 2.1, Gille and Salmon 1995) to measurements of weight and wing-length collected over the nestling period.

Equation 2.1 $$M_t = M_a - ((M_a - M_o).\exp(-\ln\frac{(1-\frac{M_o}{M_a})}{0.5}.\frac{t}{t_{50}}.p))$$

where M_t is mass at age t, M_o is mass at hatching, M_a is asymptotic mass, t_{50} is age at 50% of asymptotic mass, and p is a shape parameter adjusting the degree of maturity when growth rate peaks.

I chose the Janoschek model because it is more flexible than other sigmoidal growth models, and does not assume a fixed point of inflection. The logistic model assumes that the growth curve's point of inflection occurs at 50% of the asymptotic value, and this could create a bias. Birds with a variable food supply might tend to have the point of inflection at a late stage. If so, the logistic curve would provide a relatively poor fit for these species, confounding the correlation between growth rate and variability in food supply. By contrast the Janoschek equation has a shape parameter p that is used to calculate the parameter U_i (Equation 2.2), the percentage of the asymptotic value at which point of inflection occurs. The Janoschek curve has no parameter corresponding to growth rate. I used the Janoschek equation to estimate the number of days it takes a chick to grow from 10% to 90% of asymptotic weight (t_{90} - t_{10}).

Equation 2.2 $$U_i = 1 - \frac{1-U_o}{\exp(1-\frac{1}{p})}$$

where U_o is the degree of maturity (percentage of asymptotic weight) at hatching.

This index (t_{90} - t_{10}) is inversely related to growth rate and has the advantage of being comparable between growth curves with different shapes and asymptotes (Ricklefs 1967a). I refer to this index as growth period (*GP*). I calculated growth periods for both mass (*MGP*) and wing-length (*WGP*). It was useful to consider growth

periods as well as nestling periods for two reasons. First, nestling period is only a rough indicator of growth rate. Species vary in their degree of maturity of wings at fledging (Redfern 1994) and vary in their relation between mass and lean tissue growth rates/periods. Each of the parameters is a partially independent indicator of overall developmental rate. Second, I used the relationship between mass and wing growth periods to examine the proximate effect of environmental variability on growth (discussed below).

Variability in food supply

I used two methods to calculate the variability in food supply. First, from growth data, and second, from provisioning data. I initially assumed that both variability in mass growth and variability in feeding frequency were good indicators of variability in food supply. However, during the course of my investigations I decided that variability in feeding frequency was not a suitable indicator of variability in food supply. I found few papers with good data on variability in feeding frequency. Sample sizes were often small, and observations were often recorded over relatively short periods of time relative to the length of the nestling period. For these reasons (and others discussed below) I considered variability of mass growth as a more reliable indicator of variability in food supply than variations in the observed feeding frequencies .

As a result, I only used variability in mass growth in my later investigations involving phylogenetically independent contrasts (see below). However, I have presented my results using variability in feeding frequency to help to show why I considered it a flawed indicator of variability in food supply.

I gathered data on the standard deviation of feeding frequency for 34 species in 8 families. Feeding frequency is the number of feeds counted in a day, and counts tend to have a variance proportional to their mean (Sokal and Rohlf 1995). Hence, I divided the standard deviations by the means to obtain coefficients of variation of feeding frequency (CV_{ff}), which are independent of mean feeding frequencies.

Measuring the variability of mass growth

I constructed several indices of variability in mass growth, both from individual chick growth curves and from scattergrams of chick weights. I used the goodness-of-fit index, which I refer to as V. This index is $1-r^2$, where r^2 is the correlation between the actual weight data for a population of chicks and the weights expected by age from

the Janoschek equation. This index shares a weakness with any measure of variability that depends on a fitted model. Species whose growth doesn't match a particular growth model will have a relatively poor fit, hence the index will overestimate the variability in mass growth. However, growth models differ primarily in the position of the point of inflection (Ricklefs 1968). Because the Janoschek equation fits the point of inflection to the data, it does not provide a better fit to growth curves that have a particular shape (Gille and Salmon 1995).

Another confounding factor is that aerial foraging birds and seabirds often achieve a peak weight during nesting and then begin to lose weight as they approach fledging (e.g. O'Connor 1977, Croxall and Prince 1980). This reduces the goodness of fit for sigmoidal growth equations (Boersma and Parrish 1998). Hence, when calculating V for these species, I only used data up to the age at when weight recession began.

To measure variability in mass growth, I had to assume some model of growth. Sigmoidal equations are generally accepted as good descriptive models of avian growth (Ricklefs 1968). I chose the Janoschek equation for reasons given above. I used two types of data for measuring variability in growth: (i) individual chick growth curves, (ii) growth scatter diagrams from a number of chicks measured at different ages. The latter type of data are reported much more frequently in the literature.

One assumption made in this and following chapters is that most of the variation in mass growth is caused by environmental variation. However, genetic differences among chicks within a population will also contribute to population variability, hence growth scattergram indices may overestimate the variability of individual growth curves. On the other hand, individual chick growth data may underestimate environmental variation. For instance, two chicks born some time apart may be subject to a large long-term change in the food supply that does not show up in short-term fluctuations in growth (types of environmental variability are reviewed in Frank and Slatkin 1990).

Therefore, I investigated whether variation in growth calculated from individual growth curves for a species is closely correlated with variation calculated from growth scattergrams. If the two are closely correlated, I may use a growth scattergram index as an estimate of the variability of individual growth curves.

In some cases growth data for a number of chicks were presented not as a scattergram, but as a graph of mean weights at a given age, together with standard deviations (or standard errors). In these cases, I used a random number generator to generate a dummy scattergram from the mean weights, standard deviations, and sample sizes, then measured variability in growth using the methods described below. I did this in order to measure the variability in growth in the same way for each species.

I checked to see if this method of generating a dummy scattergram was sound, by comparing the parameters of actual scattergrams to dummy scattergrams. I took 15 species where data were presented in the form of scattergrams, and for each calculated the mean and standard deviation in weight for each age. I used these values to generate dummy scattergrams and compared these with the original scattergrams. The variability in growth (V_{pop1} below) calculated on the dummy scattergrams was very strongly correlated with that calculated from the original scattergrams ($r^2 = 0.97$).

Measuring variability in growth from growth scattergrams

I considered several ways of measuring the variability in growth from this type of data.

(i). V_{pop1}; I estimated the best fit Janoschek equation to the data using non-linear modeling in Systat. I assumed the r^2 between the predicted and actual data to be inversely proportionate to variability in growth, subject to the constraints described above I subtracted this figure from one to give us a number proportional to variability in growth. This is the goodness-of-fit index, V, used in this thesis.

(ii) V_{pop2}; This is the conditional variance for weight, where the conditional distribution is calculated from the best fit Janoschek equation with age as the independent variable.

(iii) V_{pop3}; It is conceivable that there is some association between variability in growth (calculated in either of the two ways described above), and the spread of data along the y-axis (age) that is an artifact of the method of calculation, and independent of any biological association. To calculate an index of variability in growth that is independent of growth period, I first calculated the coefficient of variation of weight at 18 days of age. 18 days of growth is a sufficient time for substantial variance to accumulate in species with variable growth, but short enough so that only a few species with a short nestling period were excluded. However, this index is not entirely independent of growth period. Variance among weights in a growth scattergram tends

to accumulate up to the point of inflection, and then decrease nearer to the asymptote (see below). Therefore, this index will tend underestimate variability in growth for species reaching asymptotic weight at approximately 18 days. Therefore I only calculated this index using species with a mass growth period of at least 25 days.

Measuring variability in growth from individual growth curves

An individual chick of a species will have a growth curve that exhibits some degree of fluctuation. I considered three different ways of measuring this:

(i) V_{ind1}; I calculated the goodness-of-fit index (see V_{pop1}) for individual growth curves, and then calculated the mean as above.

(ii) V_{ind2}; For the growth curves of several individual chicks in a species, I calculated the conditional variance, where the conditional distribution is calculated from the Janoschek equation fitted to individual growth curves, with age as the independent variable. I took the mean of these values as an index of the variability of chick growth for the species.

(iii) V_{ind3}; I fitted the Janoschek equation to individual chick growth curves. Using this model, for each point on each growth curve I calculated an expected increment of growth (I_{exp}). Actual growth increments (I_{act}) were then divided by I_{exp} to give standardised growth increments (I_{std}). Since I_{exp} tends to zero as the asymptote is reached, and near hatching, I only used data between 10% and 90% of asymptotic weight. I took the standard deviation of I_{std} as a measure of variability in growth for individual growth curves, and then calculated the mean of these values to give us a species-specific measure of the variability of chick growth.

All six indices are strongly correlated (Table 2.1). I preferred to use V_{pop1} , for four reasons: (i) by using an index measured from growth scattergrams I were able to use more species than if I had used individual chick growth curves, (ii) V_{pop1} gives a measure of variance that is independent of the units in which weights are measured as opposed to V_{pop2}, (iii) V_{pop1} is strongly correlated with all the other indices (Table 2.1), (iv) V_{pop1} allows us to use a wider sample of birds than V_{pop3}.

	V_{pop1}	V_{pop2}	V_{pop3}	V_{ind1}	V_{ind2}	V_{ind3}	**Mean**
V_{pop1}	1						0.78
V_{pop2}	0.92	1					0.78
V_{pop3}	0.88	0.76	1				0.70
V_{ind1}	0.78	0.76	0.59	1			0.59
V_{ind2}	0.76	0.75	0.64	0.38	1		0.61
V_{ind3}	0.54	0.59	0.52	0.43	0.47	1	0.50

Table 2.1 Correlation matrix for population and individual growth variability indices. Values are r^2. All correlations are statistically significant (n = 20 species for correlations among growth scattegram indices, and n = 8 for correlations involving individual growth curve indices.) Mean is the mean of the 5 correlations for each index

Variability measured from individual growth curves as a predictor of variability in growth measured from scattergrams

I also attempted to find out how much of V_{pop1} can be attributed to the short term fluctuations in the growth of individual chicks, by simulating growth scattergram data from individual growth curves and comparing the results to actual scattergrams.

I calculated the parameters of the Janoschek equation for each of eight species for which individual growth data were available. Where I had more than one individual chick growth curve for a species, I estimated the parameters for each individual, and used mean values in my simulation model. I used the parameters estimated for the Janoschek equation to calculate expected increments of growth for each day for a chick. From this I calculated the deviations from expected growth for each day for each chick in a species (i.e., the deviation divided by the expected value). Values above 1.0 represent greater than expected growth, values between 0 and 1.0 represent lower than expected growth, and negative values represent weight loss. Such values have a skewed distribution, and therefore could not be approximated by the normal distribution.

I used the Janoschek equation, with the parameters estimated from the individual growth curves, to simulate the growth of individual chicks. For each species, I simulated the growth of the same number of chicks as were used in the actual growth scattergrams. In this way I obtained a simulated growth scattergram that I could compare to the actual growth scattergram. The simulated data only take into account

the effect of fluctuations in individual chick curves, whereas the actual data should include other factors such as genetic differences among chicks.

To simulate the growth of a chick I started at the mean hatching weight for the species. I calculated expected growth from the Janoschek equation for the chick's mass and drew at random (with replacement) a deviation from the set of observed values. I then multiplied the expected growth increment by the chosen random value. I continued rounds of growth in this way until 90% of the asymptotic weight was reached.

The indices calculated from these were closely correlated with the goodness-of-fit index, V_{pop1}, calculated from scattergrams (r^2 = 0.99, Fig. 2.1). They consistently underestimate population variability (linear regression coefficient = 1.24). which may be explained by genetic variation and/or long term environmental variation in actual chick growth that is not taken into account in the simulated chick growth.

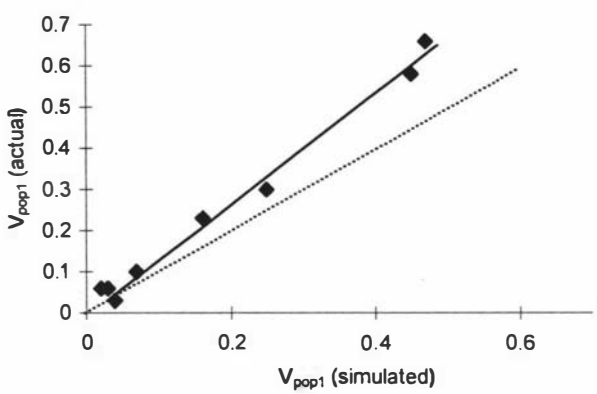

Fig. 2.1 Variability in growth measured from actual scattergrams, and from scattergrams simulated from individual growth curves. Dotted line shows one-to-one relationship.

It is unlikely that this result would be obtained if the simulations did not approximate the way variance in weights accumulates in real growing chicks. However, I tested the accuracy of my approach in a second way. In the simulated scattergrams, the variance of weights increased up until the weight at which the point of inflection occurred. After this, the variance of weights declined, and converged

toward a fairly narrow range of weights near fledging. Hence, to test whether my approach to simulating growth scattergrams was correct, I compared the standard deviation of weight at the end of the nestling period with the standard deviation of weight at 0.75 of the nestling period in 15 species. The standard deviation of weights at fledging was consistently smaller than that at the early measurement. The mean of the ratios for the standard deviation of the early measurements over the standard deviation of the late measurements was 0.53 (*sd* 0.29). The same pattern was obtained both in species that undergo weight recession prior to fledging, and in those that do not.

I concluded that V_{popl} is a good indicator of the variability in food supply faced by chicks in a species, and this is the index I used in this and later chapters. Throughout the rest of the thesis, V_{popl} is referred to as "*V*". The distribution of *V* was heavily skewed, hence I log-transformed measurements of *V* to obtain normally distributed data.

Habitat and variability in food supply

Lack argued that the Procellariiformes and the swifts have similar life history strategies because of convergent evolution. Specifically, he argued that the habitat of both types of bird is extremely variable, and the chicks of both types undergo periodic fasts. However, other authors (e.g., Shea and Ricklefs 1996) have argued that the food supply of the Procellariiformes is not extremely variable.

I tested the hypothesis that the habitats of swifts and Procellariiformes are highly variable using phylogenetic contrasts (see below). I classified species into four habitat categories: (i) aerial insectivores, (ii) terrestrial non-aerial insectivores, (iii) inshore-foraging seabirds, (iv) offshore-foraging seabirds. Swifts and Procellariiformes fall into the first and fourth categories respectively. Ancestral nest types were estimated by the criterion of parsimony using MacClade (Maddison 1986). These were then assigned scores according to the scheme described above and entered into the phylogeny. I assigned aerial insectivores and offshore-foraging seabirds a habitat score of 1, and the others a score of 0. I then compared phylogenetic contrasts for variability in food supply (*V*) with the contrasts in degree of habitat type.

Eliminating confounds among parameters

Interspecific correlations between variability in food supply and growth paramaters may contain a component from covariation with mass, from shared phylogeny, and from covariation with other ecological factors such as feeding frequency. These effects confound tests of the prediction that growth periods and nestling period are species-specific adaptations to a variable food supply.

Confounds due to scaling relationships

Growth parameters vary allometrically with adult mass (Ricklefs 1968, 1973). Large birds grow more slowly and take longer to fledge than small birds, and mass itself may be an adaptation to a harsh or variable environment. To control for a possible confound, I calculated the expected value for each species from the allometric function for each growth parameter using the data in Appendix 1. I used a larger sample size to estimate the allometric relationship between mass and growth periods than that used by Ricklefs (1968, 1973). The residual nestling and growth periods obtained in this way are independent of mass (Harvey and Pagel 1991), and are referred to as relative nestling period (*RNP*) and relative growth period (*RGP*).

The confound of feeding frequency

A low feeding frequency may result in a decrease in the amount of food delivered to the chick over the course of development (Case 1978). Hence, species with a low feeding frequency can be expected to evolve a slow growth rate due to food limitation. There are several reasons for expecting feeding frequency to be correlated with variability in food supply. First, the mean frequency of feeds that a chick receives in a given interval may be correlated with the variance in feeding frequency. Data in the form of whole numbered counts may be distributed according to a Poisson distribution, and tend to have a variance proportional to their mean. Second, foraging niches that require long return trips on each foraging bout (e.g., pelagic feeding and aerial insectivory at high altitudes) are also niches that tend to be vulnerable to changes in the weather.

To control for this confound I used the mean number of feeds per day (*FF*) as a covariate of variability in food supply (*V*) in my multiple regression analysis of phylogenetically independent contrasts (see below). The distribution of feeding frequency (*FF*) was heavily skewed, hence I log-transformed feeding frequencies to obtain normally distributed data.

The confound of phylogeny

Data for individual species are not independent measures of evolutionary events. Because of this, interspecific comparisons that do not take into account phylogenetic relationships risk both high rates of Type I error and low statistical power (Grafen 1989, Martins and Garland 1991). To remove the possibility of obtaining spurious correlations in this way, measurements must be independent of phylogenetic relationships.

I used two methods to control for phylogenetic dependence, (i) using family means in place of species means, (ii) phylogenetically independent contrasts. I used family means in the early part of my investigation - i.e., for both CV_{ff} and V (see above). However, I rejected CV as an indicator of variability in food supply before I carried out phylogenetically independent contrasts. However, I have presented both my results using family means and phylogenetically independent contrasts in order to include my results using variability in feeding frequency - i.e., to help to show why I considered it a flawed indicator of variability in food supply.

For 102 species in 25 families, I used the family means of feeding frequency (FF), coefficient of variation of feeding frequency (CV_{ff}), variability in food supply (V, variability in mass growth), and relative nestling period (RNP). Mean values for higher phylogenetic nodes, and their taxonomic approximations, are more phylogenetically independent than species values (Harvey and Pagel 1990). I was only able to obtain good growth data for 52 of the 102 species (in 18 families). For these species I was able to construct a working phylogeny and apply Felsenstein's (1985; Garland *et al* 1992) method of phylogenetic contrasts to remove the shared phylogenetic component from sister taxa. To carry out this method, a phylogeny for the group of species being used is constructed, and ancestral or nodal values inferred. Divergence of a trait between two sister taxa is a phylogenetically independent contrast, reflecting the unique evolution of the trait.

I calculated phylogenetically independent contrasts using "COMPARE3.0" (Martins 1999). This programme calculates a set of contrasts from a set of species-specific values and a phylogeny. Ancestral values are calculated as the weighted average of the other taxa on the phylogeny, where the weights are given by phylogenetic distances. Details of the methods used by the programme are given in Martins and Hansen (1997).

Grafen (1989) shows that phylogenetically independent contrasts are suitable for regression analysis. To test Lack's insurance hypothesis I used multiple regression analyses with contrasts for nestling and growth periods as dependent variables, and contrasts for ecological parameters as independent variables, along with other independent variables added to eliminate confounds (below).

I used phylogenies from Imber (1985), O'Hara (1989), Sibley and Ahlquist (1991), McKitrick (1992) Paterson *et al* (1995), Eppley (1996), Martin and Clobert (1996) and Reynolds and Lee (1996) to construct a working phylogeny (Appendix 2). There were some conflicts between Imber's (1985) phylogeny and the other phylogenies. In these cases, I used the more recent phylogenies.

Results

Despite strong allometric correlation between nestling period and adult mass (r^2 = 0.49, Fig. 2.2A), there is a great deal of variation in nestling period unexplained by mass. Much of this residual variation can be attributed to variation between families of birds (Fig. 2.2B).

The residuals from these curves are measures of nestling period relative to mass. These data are therefore independent of scaling relationships, and were used in the comparative tests that follow. Similar transformations were carried out on mass and wing growth rates.

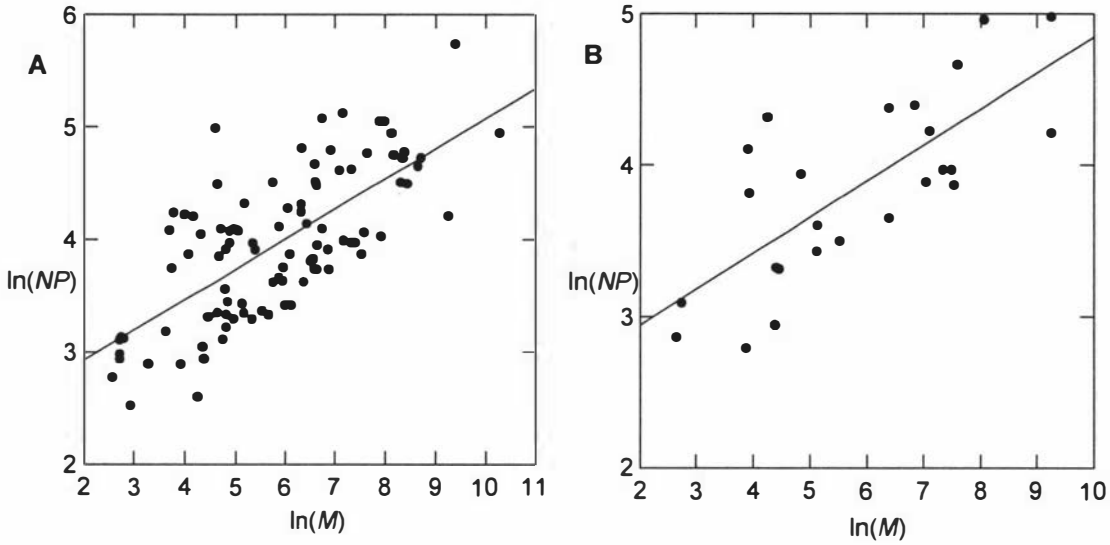

Fig. 2.2 Allometric relationship between nestling period and adult mass. Abbreviations are defined in Table 2.2. (A) linear regression for 102 species ($\ln(NP) = 0.268.\ln(M) + 2.4$); (B), linear regression for 25 family means ($\ln(NP) = 0.238.\ln(M) + 2.5$).

Abbreviation	Name	Calculation
V	Variability in food supply	One minus the goodness of fit (r^2) of the Janoschek equation fitted to the weights of a population of chicks recorded throughout nesting.
FF	Feeding frequency.	Mean feeds/day /chick.
FI	Feeding interval	The reciprocal of *FF*.
CV_{ff}	Coefficient of variation of feeding frequency	Standard deviation of feeds/day/chick divided by *FF*.
RNP	Nestling period	Number of days from hatching to fledging relative to mass.
RMGP	Mass growth period	Number of days from 10% of adult mass to 90% of adult mass, estimated from the Janoschek growth equation fitted to the weights of a population of chicks recorded throughout nesting (relative to mass).
RWGP	Wing growth period	Calculated as for *MGP* using wing-length in place of mass.

Table 2.2 Summary of definitions of parameters.

Family means for variability in food supply (V), feeding frequency (FF), coefficient of variation in feeding frequency (CV_{ff}) and relative nestling period (RNP)

Family means for relative nestling period (*RNP*) were significantly correlated with both feeding frequency (*FF*, $r^2 = 0.36$, Fig. 2.3A) and variability in food supply

(V, $r^2 = 0.36$, Fig. 2.3B) when tested independently. I carried out a multiple regression analysis for relative nestling period as the dependent variable, with variability in food supply and feeding frequency as the independent variables (Table 2.3). Both factors are significant predictors of relative nestling period. However, family means for coefficient of variation of feeding frequency (CV_{ff}) are not significantly correlated with relative nestling period.

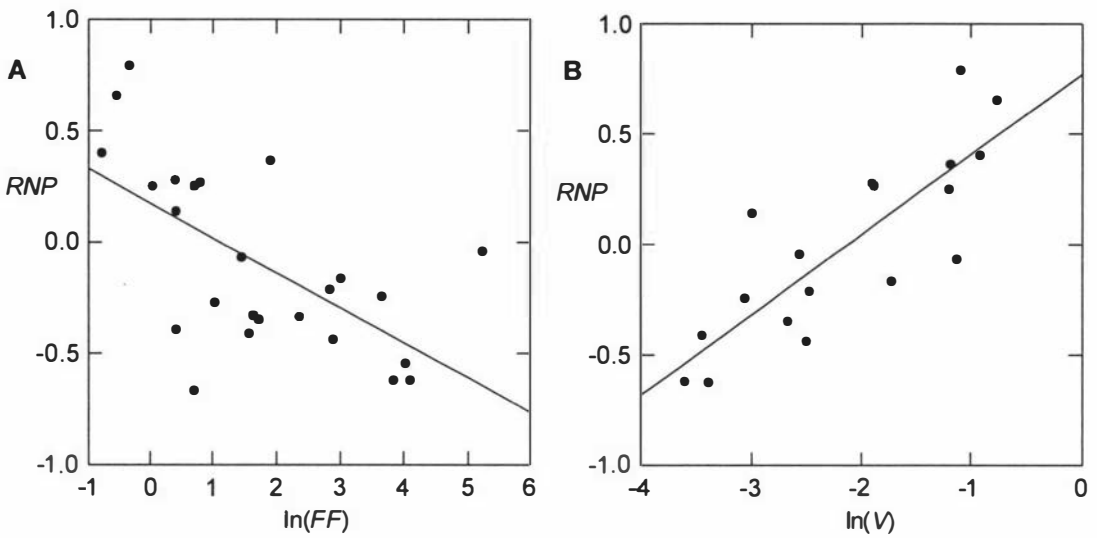

Fig. 2.3 Relationship between family means for (A) relative nestling period and feeding frequency, and (B) relative nestling period and variability in food supply. The methods for calculating the variables are shown in Table 2.2.

	r^2	ln(V)	ln(FF)
RNP	*0.76****	*0.57***	*-0.38**

Table 2.3 Effect of variability in food supply (V) and feeding frequency (FF) on relative nestling period and growth periods, testing using multiple regression analysis on family means. r^2 given for the proportion of variation explained by both independent variables. V and FF show standardised partial regression coefficients. Statistical significance in the overall model and for the partial coefficients is indicated by asterisks: * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$. The methods for calculating the variables are shown in Table 2.2.

Phlyogenetically independent contrasts for variability in food supply, feeding frequency, nestling period and growth periods

Phylogenetically independent contrasts for nestling period and are significantly correlated with both variability in food supply and feeding frequency (Table 2.4, Fig. 2.4). A similar pattern holds for mass growth period (Fig. 2.5). The wings of species with a variable food supply also appear to grow slowly (Fig. 2.6). However, my sample size for wing tissue growth rates were small by comparison with my other data (n = 19).

	r^2	ln(*V*)	ln(*FF*)
RNP	0.50***	0.45***	-0.38**
RMGP	0.45**	0.57**	-0.17
RWGP	0.50**	0.48*	-0.10

Table 2.4 Effect of variability in food supply (*V*) and feeding frequency (*FF*) on relative nestling period and growth periods, tested using multiple regression analysis on phylogenetically independent contrasts. r^2 given for the proportion of variation explained by both independent variables. *V* and *FF* show standardised partial regression coefficients. The methods for calculating the variables are shown in Table 2.2. Conventions as for Table 2.2.

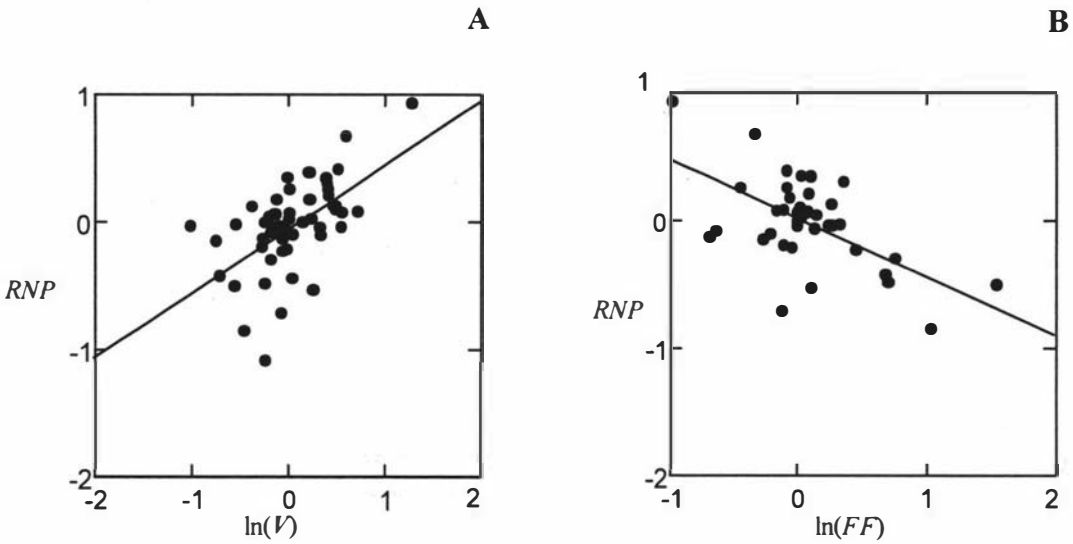

Fig. 2.4 Relationship among species between (A) relative nestling period (*RNP*) and variability in food supply (*V*), and (B) relative nestling period and feeding frequency (*FF*). The actual values shown are phylogenetically independent contrasts. These were obtained for each variable from the values calculated for the 52 species in Appendix 1. The methods for calculating the variables are shown in Table 2.2.

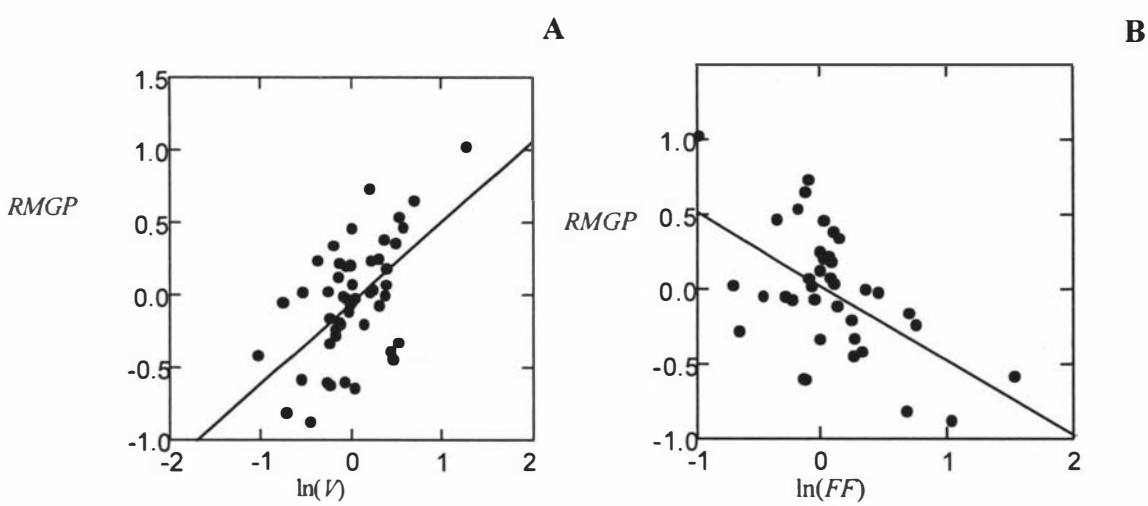

Fig. 2.5 Relationship among species between (A) relative mass growth period (*RMGP*) and variability in food supply (*V*), and (B) relative mass growth period and feeding frequency (*FF*). Conventions are as for Fig. 2.4. The methods for calculating the variables are shown in Table 2.2.

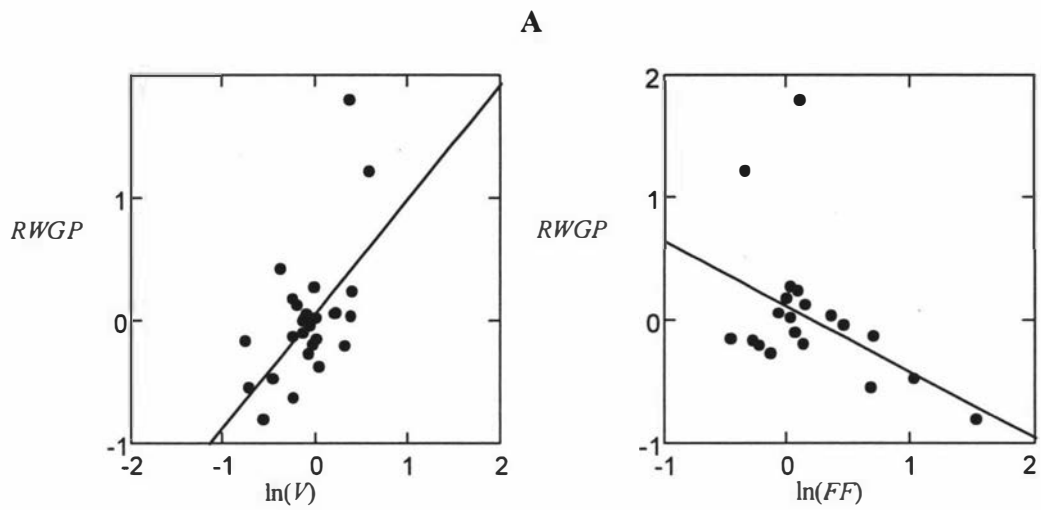

Fig. 2.6 Relationship among species between (A) relative wing growth period (*RWGP*) and variability in food supply (*V*), and (B) relative wing growth period and feeding frequency (*FF*). Conventions are as for Fig. 2.4. The methods for calculating the variables are shown in Table 2.2.

Habitat type and variability in food supply

Each of seven families of offshore-foraging seabirds had more variable food supply than families made up of exclusively inshore foragers (Fig. 2.7A). Offshore foragers also tended to have longer nestling periods than inshore foragers, though there was some overlap. The Sternidae (terns and noddies) contain both inshore and offshore foragers, and occupied an intermediate position. The two families of terrestrial aerial insectivore had longer nestling periods, and generally more variable food supply than the three families of terrestrial non-aerial feeders. However, the Paridae (tits) were very similar to the Hirundidae (swallows). The Paridae had much longer nestling periods relative to their weight, and more variable growth, than the two other families of terrestrial non-aerial feeders.

I gave phylogenetic contrasts from the inshore or terrestrial non-aerial niche to the offshore or terrestrial aerial insectivore niche a score of 1, and the opposite contrasts -1. Where there was neither type of change, I gave contrasts a score of zero. Phylogenetic contrasts in variability in food supply were significantly different among the three types of habitat contrast (Fig. 2.7B, 1-way ANOVA, $P = 0.001$).

This distribution of variability in food supply across habitat types corroborates Lacks hypothesis that swifts and Procellariiformes are both adapted to a similarly variable niche type. It also falsifies the conjecture that offshore-foraging seabirds do

not have a variable food supply but only a long average feeding interval. I found that species with long feeding intervals usually also have a variable food supply as measured by variability in mass growth (Fig. 2.8). Offshore-foraging seabirds have long feeding intervals and variable food supplies.

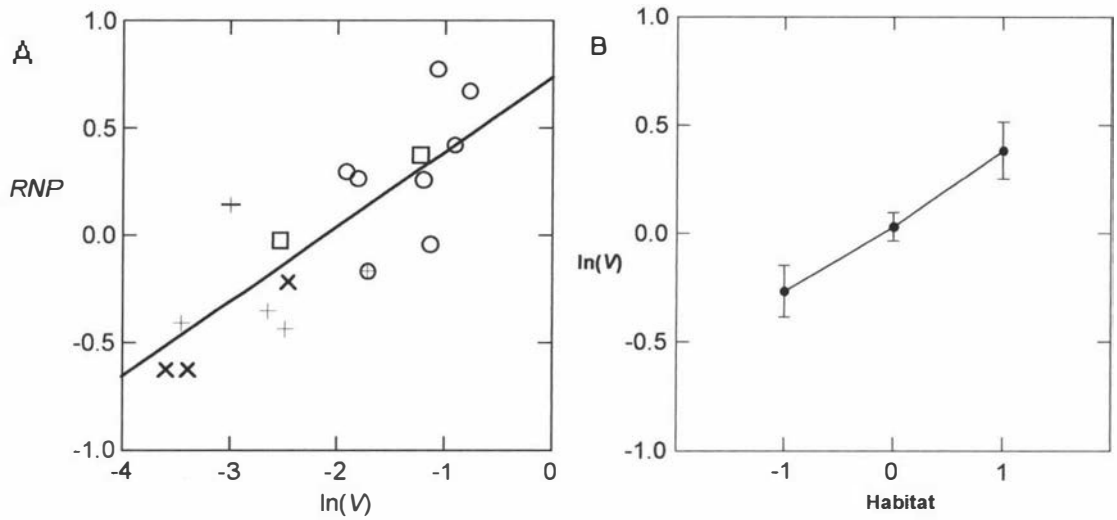

Fig. 2.7 Variability in food supply and relative nestling period in four habitat types. (A), relationship between relative nestling period (*RNP*) and variability in food supply for 14 families in 4 different habitats. ○, offshore-foraging seabirds; +, inshore-foraging seabirds; ⊕, mix of inshore and offshore seabirds (Terns and noddies); □, aerial insectivores; ×, terrestrial non-aerial feeders. Habitats labeled with open symbols (circle and square) have been conjectured to have a highly variable food supply and habitats with cross or plus sign symbols have been conjectured to have more stable food supplies, (B), Relationship between variability in food supply and habitat type in 52 species. Habitat, dummy variables for habitat type (see text); ln(*V*) natural log of variability in food supply. Points are means of phylogenetic contrasts and vertical lines are standard deviations. The methods for calculating the variables are shown in Table 2.2.

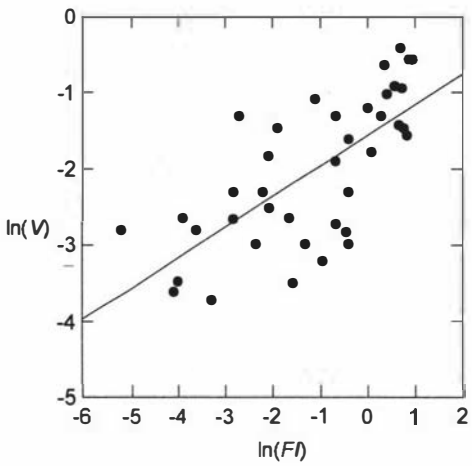

Fig. 2.8 Relationship between family means for feeding interval and variability in food supply. The methods for calculating the variables are shown in Table 2.2.

Discussion

Chicks of species with a variable food supply face the problem of starving before they reach fledging. The developmental features of swifts and the Procellariiformes such as high levels of body fat, slow growth, and torpor in swift (Koskimies 1948) and petrel chicks as listed by Lack and Lack (1951), are intuitively good solutions to this problem. However, Lacks hypothesis has been challenged by various authors, usually working on one or a few species at a time. Procellariiformes chicks are sometimes found to have an apparently stable food supply (e.g. Shea and Ricklefs 1996). However, when I used variability in mass growth as an index of variability in food supply, the relationship between nestling and growth periods and variability in food supply seemed a general one among birds. The effect was independent of mass and phylogenetic constraints.

When I used the coefficient of variation of feeding frequency (CV_{ff}) I could find no consistent relationship with nestling period. This clashes with the correlations found between nestling and growth periods and variability in food supply calculated on the basis of variability in mass growth. Variability in mass growth (i.e., V) was also not correlated with the coefficient of variation of feeding frequency (CV_{ff}). This suggests that the diverging opinions on the variability in the food supply of Procellariiformes, and offshore-foraging seabirds in general, could be a result of problems in the way variability in food supply is measured. CV_{ff} does not appear to be a reliable indicator of the fluctuations in mass gain (hence food intake) that chicks of species with a variable

food supply undergo. This may have several causes. First, in some cases, observational data on provisioning may tend to be restricted to periods of relatively good weather. Second, variability in feeding frequency does not give any indication of the variation in the amount of food delivered per feeding bout, which may vary even as feeding frequency remains relatively constant. Parents may return to the nest according to a fixed schedule, but with varying success at procuring food for the chick. Hence, recorded values of the variability of provisioning are likely to underestimate true values for the variability in food supply. Third, CV_{ff} tends to underestimate the affect of rare but severe drops in food supply, such as may occur during a storm. Fourth, using the standard deviation of feeding frequency assumes that the period over which feeding bouts are counted, is shorter than the average feeding interval for a species. Estimates of the variability of feeding frequency become unreliable for species that are fed less than once a day.

I assumed that a low average feeding frequency causes a low overall food supply (however, see Appendix 3). The feeding interval (the reciprocal of feeding frequency) represent a delay in receipt of food, especially in birds with very long feeding intervals. The weight of the chicks of some species of Procellariiformes drops markedly between feeds (see for example, growth curves in Berruti 1979). Hence, the digestive tract is being emptied between feeds which suggests that feeding interval limits food intake. Furthermore, feeding frequency may covary with variability in food supply because both result from a similar type of environmental constraint (see introduction). Because of this, I used feeding frequency as a covariate of variability in food supply in the regression analysis of phylogenetically independent contrasts to control for a possible confound. However, it is also possible that mean food supply is correlated with variability in food supply irrespective of feeding frequency. Controlling for feeding frequency will not control for an effect such as this, because although a lowered feeding frequency may *cause* a decrease in mean food supply, feeding frequency should not be used as an *indicator* of mean food supply. As noted above, the amount of food delivered to chicks per visit may vary considerably (e.g. Hamer and Thompson 1997). I discuss the role of mean food supply (as opposed to variability in food supply) further in Chapter 3.

Contrary to some authors (e.g. Shea and Ricklefs 1996) calculating variability in food supply from variability in mass growth suggests that offshore-foraging seabirds

do have a more variable food supply than other types of birds, and that swifts have a similarly variable food supply. These types of bird not only have a long interval between feeds, but also a highly variable amount of food delivered to the chicks in any given day. I suggest that the reason this has been controversial, is that there is no standardised way of measuring variability in food supply, and because some methods - such as the variability of feeding frequencies - significantly underestimate the true variability in food supply. In this chapter I developed a standardised method of recording the variability in food supply, using the variability of weight gain as an index of the variability of food intake. Applying this to a broad range of species showed that not only do offshore-foraging seabirds and swifts have a highly variable food supply, but that there is a general, and phylogenetically independent relationship between variability in food supply and growth periods (hence growth rates). Hence, Lack's insurance hypothesis has been corroborated by these tests.

However, there are other problems that remain for Lack's insurance hypothesis. Correlations between variability in food supply and growth periods (hence growth rate) may be the result of a proximate environmental effect, rather than adaptation. This problem is taken up in the next chapter.

Chapter 3

The proximate effect of variability in food supply on growth and nestling periods

Introduction and methods

In comparative analyses, differences in parameters such as nestling period among species are assumed to be due to evolution. An alternative explanation is that parameters may vary due to due to proximate responses to environmental variability. Under ideal conditions, an individual may grow at the maximum rate possible set by the physiology of its species. In comparison, an individual experiencing periodic fasts may grow more slowly and take a longer time to reach fledging. If this is true for many of the species in a comparative study, then even if species are not evolutionarily adapted to variable conditions, there will still be an interspecific correlation between a variable food supply and growth rate and nestling period. Throughout this chapter, I will refer to this as the "proximate effect" (of environmental variability on nestling period and growth rate). I use this term both for the relationship between environmental variability and growth rate within species, and the interspecific correlation that arises as a result.

The problem of proximate effects confounding evolutionary hypotheses is common to all interspecific comparisons (Endler 1986). Because of this, I not only discuss the particular problem faced in this study, but the problem of proximate effects in general. I describe various methods for controlling for proximate effects in interspecific comparisons, and illustrate each of these methods using my own data. Because of this I sometimes present similar data in several different ways.

My general approach to controlling for a proximate effect is to model the intraspecific response for each species. The parameters produced by models of this kind will vary among species and reflect the evolutionarily determined constraints on growth rates or nestling period. In my case, growth rates or nestling period are the dependent variable, and food supply or variability in food supply is the independent variable. As food supply increases nestling period should decrease and growth rate increase toward species-specific minima and maxima respectively (Fig. 3.1).

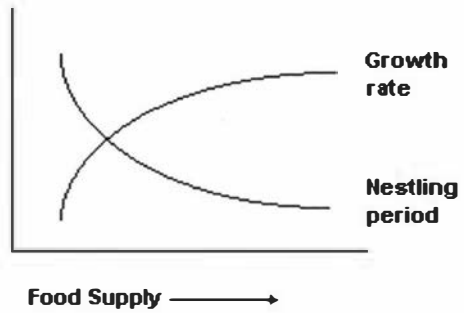

Fig. 3.1 Model of the proximate effect of food supply on growth rate and nestling period.

Parameters of the intraspecific model

Ideally, modelling these "intraspecific response curves" (Fig. 3.1) would use detailed information on food supply, and growth rates and nestling periods for a number of species. Given a range of feeding conditions, and corresponding growth parameters, detailed models of intraspecific proximate effects could be obtained. However, I was unable to obtain such data from the literature. As a result my methods are considerably simplified.

I assumed a linear response of growth parameters to changes in food supply. This is may be unrealistic and the curves shown in Fig. 3.1 are probably more realistic. However, linear models have the advantage of being easily calculated from limited data. In principle, a linear model (Equation 3.1) can be estimated from only two coordinate pairs.

Equation 3.1 $NP = m.V + NP_{min}$

where NP is mean nestling period (for a sub-grouping of chicks of a species, in a particular local environment), V is variability in food supply (measured from scattergrams of weights for a sub-grouping of chicks of a species in a particular local environment, see next section and Fig. 3.3) , NP_{min} is a species-specific minimum nestling period, and m is a coefficient relating nestling period to the local variability in food supply (similar equations can be written for growth periods).

Minimum Nestling and Growth Periods

Equation 3.1 predicts that nestling period (NP) is a variable partially dependent upon proximate food supply (as are mass growth period MGP, and wing growth period WGP). However, minimum nestling period (NP_{min}) is a species-specific constant that, according to Lack's insurance hypothesis, will be correlated with species-specific variability in food supply (V) (and likewise for MGP_{min} and WGP_{min}).

Using the same species as I used in Chapter 1, I was able to record minimum nestling periods for 32 species , minimum mass growth periods for 52 species, and minimum wing growth periods for 19 species (Appendix 1). I converted these data into relative minimum nestling and growth periods (RNP_{min} etc.) to control for mass (discussed in Chapter 2). From these data I calculated phylogenetically independent contrasts and used these as dependent variables in a multiple regression analysis using variability in food supply (V) and feeding frequency (FF) as independent variables (see Chapter 2).

Asymptotic mass is often higher for chicks (or populations) growing at the maximum rate than for those growing near the mean rate. This tended to cause the difference between mean and minimum growth rates/periods to be underestimated (Fig. 3.2). Hence, I calculated minimum growth periods in terms of asymptotic values for *mean* growth rates - i.e., t_{10} and t_{90} for GP_{min} were calculated at 10% and 90% of the mean asymptotic value respectively. In this way, I was able to compare minimum and mean growth periods despite differences in asymptotic values.

Fig. 3.2 Calculating minimum growth periods when asymptotic mass is higher for fast growing chicks. The two curves represent a chick growing at the mean rate (right) and the maximum rate (left). GP_{min} is minimum growth period (corresponding to maximum growth rate), GP_{mean} is mean growth period.

The gradient of the intraspecific model

According to the proximate effect hypothesis, under ideal conditions individuals grow at the maximum possible rate (Fig. 3.3). If a number of individuals in a species are growing under similar (ideal) conditions, then variability in food supply calculated on the scattergram of their weights (V) will be close to zero and mean growth period will be approximately equal to minimum growth period. Where there is a variable food supply, some individuals will grow below the maximum rate, hence V will increase and mean growth periods (calculated from best fit Janoschek equations on scattergrams of weights) will increase.

If the proximate effect hypothesis is correct - i.e., if in ideal conditions variability in food supply V is close to zero, and an increase in V increases nestling period (NP) - then the degree of increase m (Equation 3.2), can be in principle be estimated from a set of coordinate pairs for of the form (V_i, GP_i). Ideally, coordinate pairs would be estimated for a number of sub-populations, each with a different

variability in food supply. Since I do not have these data, I instead estimated m using only two coordinate pairs. I did this by assuming that mean growth and nestling periods correspond to the species value for V, and that minimum nestling period occurs when V is approximately zero (both are assumptions of the proximate effect hypothesis) (equation 3.2).

Equation 3.2. $$m = \frac{NP - NP_{min}}{V}$$

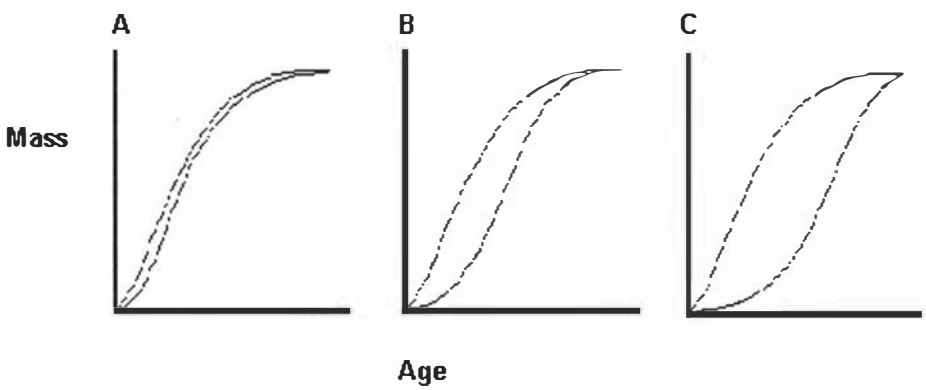

Fig. 3.3 The proximate effect explanation for interspecific correlations between growth rate and V. Within a species with a variable food supply, growth rates and growth periods will vary. At the maximum growth rate (A), conditions are ideal and stable (V = 0). This is the case for species with short nestling periods. However, for species in variable environments (B), typical conditions will show a spread of growth rates. As fluctuations in food supply become greater (C), V rises and mean growth rate declines increasingly further below the maximum growth rate (where V and mean growth rate are measured from population scattergrams). The topmost figure shows minimum, mean and maximum nestling periods corresponding to matching growth rates.

To summarise the procedure so far, the proximate effect hypothesis implies that each species will have a characteristic intraspecific response curve (Fig. 3.1) for mean growth periods on variability in food supply (V). Each curve can be approximated with a linear model, and these linear models can be estimated from minimum nestling or

growth periods (where V is assumed to be zero), and mean growth periods (which correspond to the measured values for V).

I used the parameters of the intraspecific response curves (i.e., NP_{min} or GP_{min}, and m) to compare the two hypotheses that predict a correlation between variability in food supply and nestling period i.e., Lack's insurance hypothesis (which explains growth rates as the result of evolution) and proximate effect, Fig. 3.4). If the proximate effect hypothesis is correct, then the intraspecific response curves will be unrelated to the species-specific variability in food supply (Fig. 3.4B). If Lack's insurance hypothesis is correct, then the intraspecific response curves should line up as shown in Fig. 3.4D.

The method described in the last paragraph does not take phylogeny into account. To solve this problem I devised a slightly different method. The proximate effect hypothesis predicts that phylogenetic contrasts (i.e., in V, RNP, and RGP) between pairs of species can be explained by differing proximate responses to environmental variability in pairs of species. To test this idea I converted minimum and mean nestling periods into "intraspecific contrasts" (analogous to phylogenetic contrasts, which are also linear contrasts, Grafen 1989). If the proximate effect hypothesis is correct, then phylogenetic contrasts will be explained by (i.e., reduced to) intraspecific contrasts. That is, differences in nestling period amongst pairs of species can be explained by species-specific intraspecific responses, and these will not be correlated with variability in food supply. By contrast, *intraspecific* contrasts in nestling period between two species with differing variability in food supply are less than *interspecific* contrasts because of evolution. Therefore, if the proximate effect hypothesis is correct, the relationship between *intraspecific* contrasts for nestling period and variability in food supply (V) should have the same slope as the relationship between *interspecific* contrasts for these parameters.

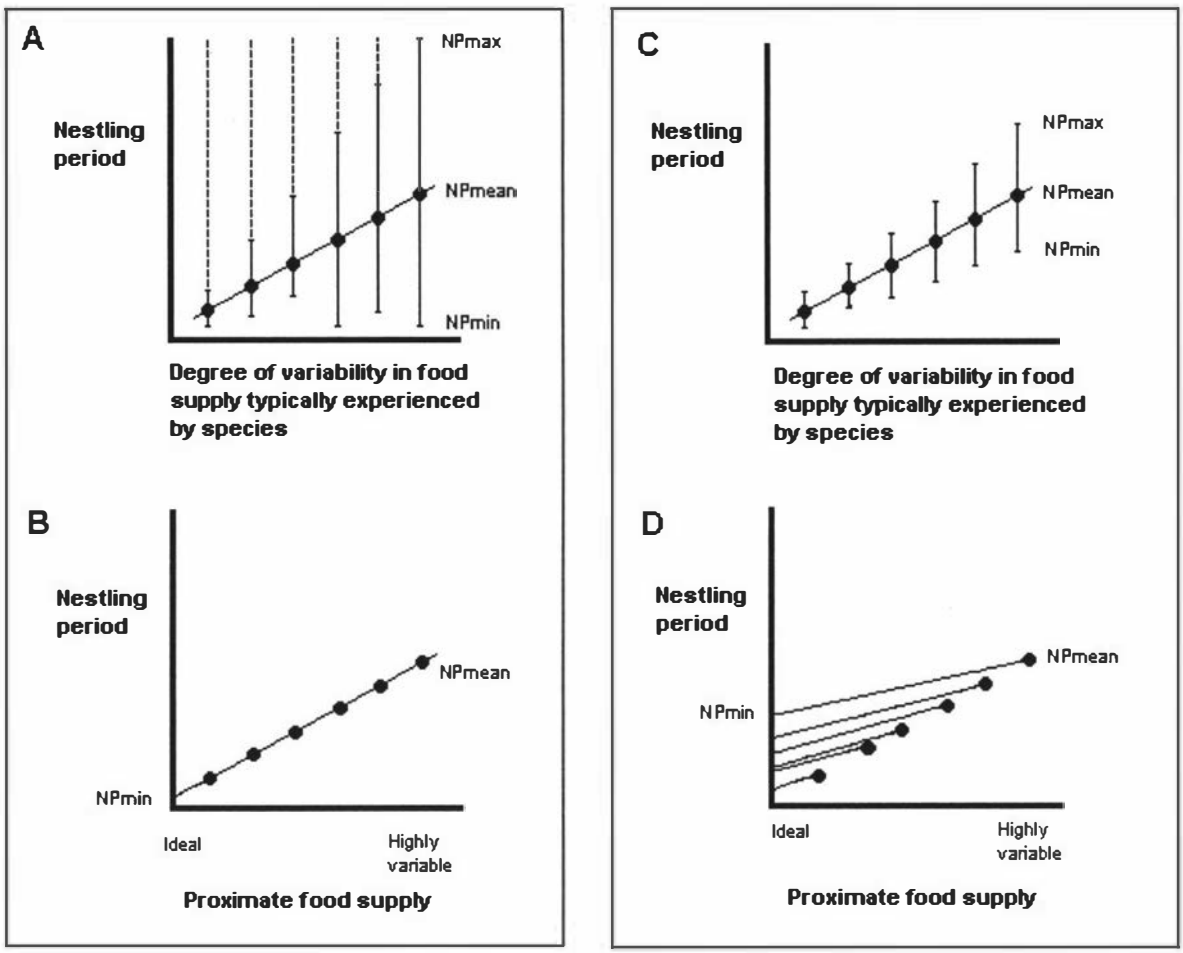

Fig. 3.4 Proximate effect hypothesis (A and B) and Lack's (evolutionary) insurance hypothesis (C and D) explanations of a correlation between variability in food supply and nestling period. (A), vertical lines are ranges of nestling period for six species in increasingly variable environments. Dotted lines are potential nestling periods where food supply is poorer than has been recorded ; (B), dots are the six species in A. Minimum nestling period is unrelated to the variability in food supply typically experienced by species. For simplicity I have illustrated this by showing each species with a similar *minimum* nestling period (y-intercept) and similar *m* (i.e., similar/overlapping intraspecific response curves, represented by the single line). Dots represent *mean* nestling periods calculated from mass growth scattergrams (see Fig. 3.3). (C), species in more variable environments have longer minimum nestling periods. Measured ranges of nestling period are similar to potential ranges; (D), circles are the six species in C. Each species has a distinct intraspecific response curve the parameters of which are correlated with variability in food supply and are the result of evolution according to Lack's insurance hypothesis.

Eliminating the proximate effect hypothesis using growth models

The methods that I have discussed so far assume that recorded estimates of minimum nestling and growth periods accurately reflect nestling and growth periods in ideal conditions. However, this may be incorrect because estimates of minima are sample size dependent. That is, how well *recorded* minimum growth period measure *actual* or *potential* minimum growth periods for a species depends upon the range of conditions under which growth periods were recorded. Consequently I developed another set of methods to eliminate the proximate effect hypothesis.

I did this using a model of avian development developed by Boersma and Parrish (1998) that incorporates both evolutionarily determined developmental constraints and environmental constraints on growth rate. In this model lean tissue growth rate varies with mass growth rate up to a maximum level (Fig. 3.5A). Wing growth can be used as an measure of lean tissue growth. Any increase in mass growth rate above this maximum is due to fat deposition alone.

I used this model to construct versions of Lack's insurance hypothesis and the proximate effect hypothesis that could be tested with comparative data. As before, if Lack's insurance hypothesis is correct, then the maximum lean tissue growth rate for species adapted to variable environments should be lower than that for species in more stable environments (Fig. 3.5B). By contrast, the proximate effect hypothesis predicts that maximum lean tissue growth rate will not be correlated with varibility in food supply, but that species with a more variable food supply will have mean lean tissue growth rates well below their maximum lean tissue growth rate (Fig. 3.5C). Converting this to growth periods, Lack's insurance hypothesis predicts that variability in food supply will be correlated with minimum lean tissue growth periods whereas the proximate effect hypothesis does not.

The proximate effect hypothesis can be eliminated if the ratio of wing growth period to mass growth period ($\frac{WGP}{MGP}$, which I refer to as the Growth Ratio Index, GRI) increases with the variability in food supply (V) (Table 3.1). This correlation is expected under Lack's insurance hypothesis (Hulsman and Smith 1988, Fig. 3.5B) with wing growth rate used as an indicator of lean tissue growth rate, but not under the proximate effect hypothesis.

However, other growth models are also consistent with this effect. In Figure 3.5D, species living in a variable environment have a relationship between tissue and mass growth below the maximum tissue growth rate that is different than that for species living in a more stable environment. This could be due to adaptation, with resources shunted in greater proportion into overall mass growth than lean tissue growth (Congdon 1990). However, it could also be due entirely to environmental effects. If species with a *variable* food supply also have a *poor quality* food supply (where quality of food is the ratio of essential nutrients to energy), chicks may need to consume a large amount of energy to obtain nutrients sufficient to support lean tissue growth (Shea and Ricklefs 1985, Hamer and Thompson 1997, Boersma and Parrish 1998). Alternatively, the relationship between mass and lean tissue growth may have a x-intercept that is greater than zero (Fig. 3.5E). This could be the case if chicks only start lean tissue growth after some minimum amount of fat has been deposited. This model is consistent with Lack's insurance hypothesis, as the position of the x-intercept should be a trait that is evolutionarily determined.

Both of these models (Figs. 3.5D-E) can be eliminated if the ratio of minimum wing growth period to minimum mass growth period ($\frac{WGP_{\min}}{MGP_{\min}}$) becomes higher in comparison to the ratio of mean wing growth period to mean mass growth period ($\frac{WGP}{MGP}$) in species with a more variable food supply (V) (Table 3.1) (Definitions for these and all other the indices used in this chapter are summarised in Table 3.2). This effect is consistent with Lack's insurance hypothesis (Fig. 3.5B). To test for this, I compared variability in food supply (V) with the ratio of $\frac{WGP_{\min}}{MGP_{\min}}$ to $\frac{WGP}{MGP}$. I refer to this ratio as the Growth Adjustment Index (GAI). Because my sample size for minimum wing growth periods is small, I repeated this test using mean and minimum nestling period in place of mean and maximum wing growth rate. I refer to this as the Nestling Period Adjustment Index ($NPAI$). I expect nestling period to be constrained by tissue growth, so $NPAI$ is directly analogous to GAI.

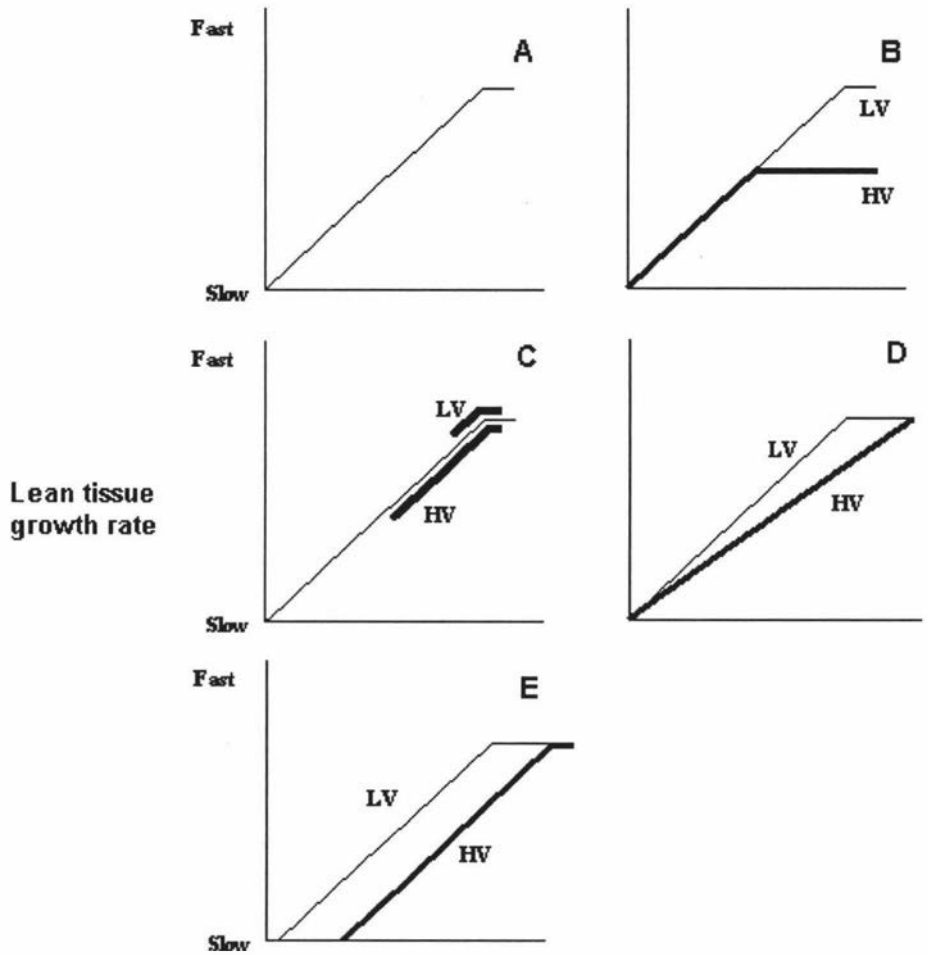

Fig. 3.5 Models of the relationships between lean tissue growth rate and mass growth rate in birds (adapted from Boersma and Parrish 1998). (A) Basic growth model, where tissue growth rate has a maximum value beyond which any gain in mass is due solely to fat deposition (horizontal line) (B) A hypothetical species adapted to a highly variable environment (HV, dark line) has a lower maximum tissue growth rate than a hypothetical species adapted to a environment of low variability (LV, light line). (C) Proximate effect hypothesis: HV species and LV species are similarly adapted and have similar developmental patterns under ideal conditions. The light line represents the *potential* growth rates that are similar for HV and LV species. The dark lines represent the actual or *realized* growth rates that are a result of environmental differences between HV and LV species (and are offset from the light line for clarity). (D) HV species have a similar maximum tissue growth rate to LV species, but have a slower rate of tissue growth for a given degree of mass gain. This could be due (i) to a developmentally regulated pattern of growth in which energy intake is distributed to fat deposition and lean tissue growth according to some set ratio, or (ii) due to the environmental effects of food quality. (E) Chicks do not start tissue growth until some fat deposition has taken place. The amount of fat deposited in greater in HV species.

Predictions	Models based on Lack's insurance hypothesis			Models based on the proximate effect hypothesis	
	Reduced lean tissue growth rate (Fig. 3.5B)	Prioritised fat deposition (Fig. 3.5E)	Energy distribution (Fig. 3.5Di.)	Environmental regulation (Fig. 3.5C)	Food quality Fig. 3.5Dii.)
Variability in food supply (*V*) is correlated with wing growth period (*RWGP*), mass growth period (*RMGP*) and nestling period (*RNP*)	✔	✔	✔	✔	✔
Variability in food supply is correlated with minimum wing growth period (*RWGP*min) and minimum nestling period (*RNP*min)	✔	✘	✘	✘	✘
Variability in food supply is positively correlated with *GRI*.	✔	✔	✔	✘	✔
Variability in food supply is positively correlated with *GAI* and NPAI.	✔	✘	✘	✘	✘

Table 3.1. Growth models and their tests.

Columns are growth models corresponding to Fig 3.5. Rows are comparative tests. Ticks indicate that a model is consistent with the result. Crosses indicate that a model is inconsistent with the result.

Flexibility of growth rate as an adaptation to a variable environment

Each of these growth models assume facultative growth adjustments of mass and wing-length in response to variability in food supply. In Fig. 3.5, the graphs are drawn so that the relationship between mass and wing growth rate varies continuously to the origin - i.e., to zero growth rates. However, the degree to which growth may adjust - i.e., the flexibility of growth - may be an adaptation to a variable environment. Vinuela and Ferrer (1997) argue than an ability to facultatively retard wing growth may be important for chicks of species with a variable food supply. Hence, species feeding with a more variable food supply should have a more flexible growth pattern (Fig. 3.6).

There seems to be no way of applying my methods to test this hypothesis (which I refer to as the flexible growth hypothesis) using comparative data whilst avoiding circularity. My index of variability in food supply is calculated from variability of mass growth. However, variability in growth requires flexibility of growth, hence the circularity. The circularity would be complete if the point at issue was the flexibility of mass growth rate. However, the problem remains even when looking at flexibility of lean tissue growth rate. Hence, it would seem impossible to test the flexible growth hypothesis by comparing variability of mass growth with variability

of wing growth, whilst avoiding circularity. However, the question *may* be at least partially answered in the negative. If variability of wing growth does *not* increase as variability of mass growth increases, then this argues against the flexible growth hypothesis. This could be the case if mass growth rate is free to vary in species adapted to a variable environment, even as lean tissue growth rates are constrained to a narrow range of values. However, low wing growth rate variability relative to mass growth rate variability could result even if wing growth is flexible *if* wings typically have sufficient nourishment to grow at the maximum rate. This would mean relatively stable realized wing growth rates but potentially variable mass growth rates.

The model shown in Fig. 3.5E implicitly assumes that wing growth rate is highly flexible. This model is examined in more detail in Chapters 6 and 7. The flexibility of mass growth rate irrespective of lean tissue growth rate (i.e., due to fat deposition), is examined in more detail in Chapters 5, 6, and 7, using variations on the growth models discussed above.

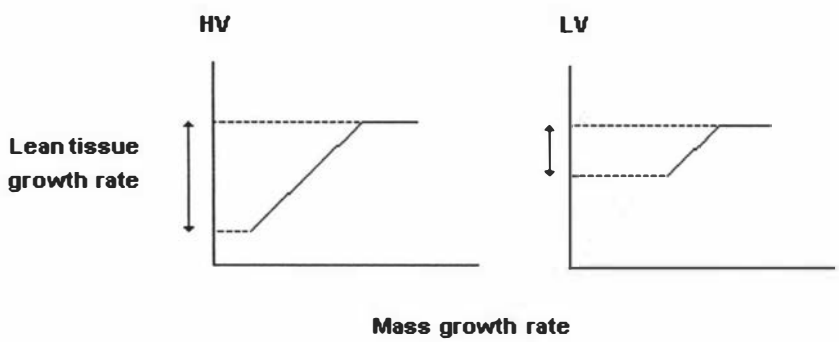

Fig. 3.6 Flexibility in lean tissue growth rate in relation to variability in food supply. A species adapted to a variable food supply (HV) may facultatively adjust lean tissue growth rate over a wide range of values (double headed arrow). A species adapted to a less variable food supply (LV) adjusts lean tissue growth rate over a smaller range.

Results

The relationships between variability in food supply (V) and feeding frequency (FF), and minimum nestling and wing growth period are similar to those described in Chapter 2 for mean nestling and wing growth periods - i.e., phylogenetically independent contrasts for minimum nestling and wing growth period are significantly

correlated with variability in food supply and feeding frequency (Figs. 3.7-9, Table 3.3).

Abbreviation	Name	Calculation
RNP_{min}	Relative Minimum Nestling Period	Shortest nestling period recorded for an individual of a species (relative to mass)
$RMGP_{min}$	Relative minimum mass growth period	Number of days from 10% of mean adult mass to 90% of mean adult mass, estimated from the Janoschek growth equation fitted to the maximum weights of a population of chicks recorded throughout nesting (relative to mass).
$RWGP_{min}$	Relative minimum wing growth period.	Calculated as for $RMGP_{min}$ using wing-length.
GRI	Growth Ratio Index	$\frac{WGP}{MGP}$
GRI_{min}	Growth Ratio Index at maximum growth rate	$\frac{WGP\ \text{min}}{MGP\ \text{min}}$
GAI	Growth Adjustment Index	$\frac{(WGP\ \text{min}).(MGP)}{(MGP\ \text{min}).(WGP)}$
NPAI	Nestling-period Adjustment Index	$\frac{(NP\ \text{min}).(MGP)}{(MGP\ \text{min}).(NP)}$

Table 3.2 Definitions of the parameters used in testing the proximate effect hypothesis with respect to the growth models shown in Fig. 3.5.

Minimum mass growth period is not significantly correlated with variability in food supply (Fig. 3.8, Table 3.3) (in contrast to mean mass growth period, Chapter 2). However, minimum mass growth period is significantly correlated with feeding frequency (Fig. 3.8, Table 3.3).

	r^2	ln(V)	ln(FF)
RNP_{min}	0.75***	0.56***	-0.17
$RMGP_{min}$	0.26**	0.06	-0.34*
$RWGP_{min}$	0.73**	0.56*	-0.59*

Table 3.3 Effect of variability in food supply (V) and feeding frequency (FF) on minimum nestling period and minimum growth periods, tested using multiple regression analysis on phylogenetically independent contrasts. r^2 given for the proportion of variation explained by all three independent variables. V, FF and PD show standardised partial regression coefficients. See Table 2 for definitions of parameters. Statistical significance in the overall model, and for the partial coefficients, is indicated by asterisks: * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

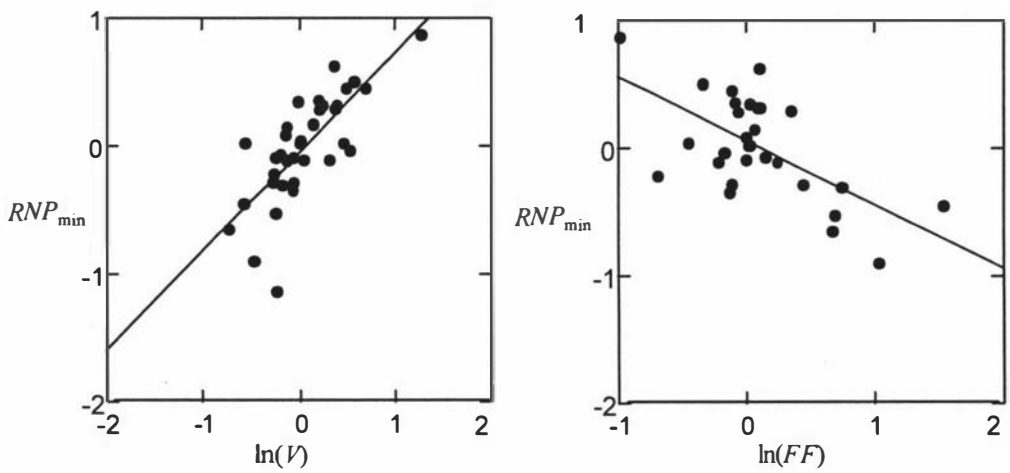

Fig. 3.7 Relationship among species between (A) relative minimum nestling period (RNP_{min}) and variability in food supply (V), and (B) relative minimum nestling period and feeding frequency (FF). The actual values shown are phylogenetically independent contrasts. These were obtained for each variable from the values calculated for the species in Appendix 1. The methods for calculating the variables are shown in Table 2.2 (Chapter 2) and Table 3.2.

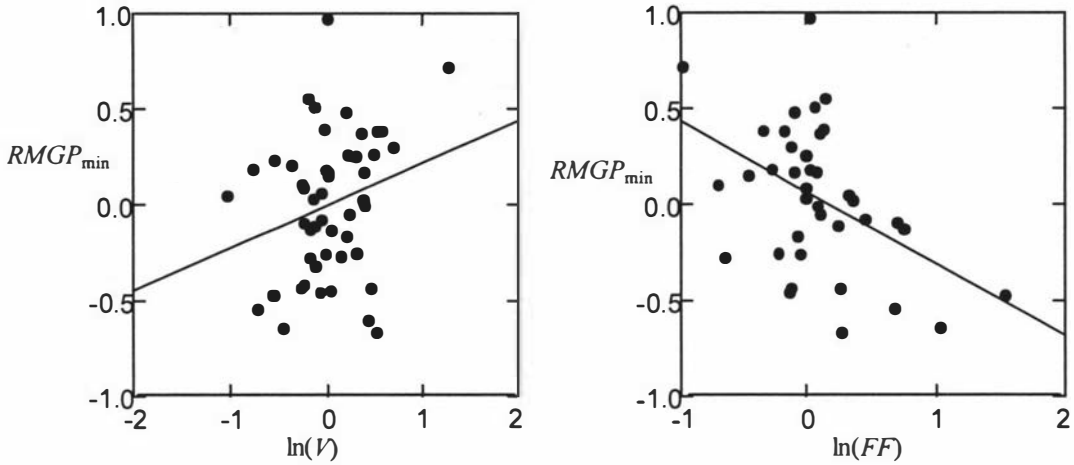

Fig. 3.8 Relationship among species between (A) relative minimum mass growth period ($RMGP_{min}$) and variability in food supply (V), and (B) relative minimum mass growth period and feeding frequency (FF). Conventions are as for Fig. 3.7. The methods for calculating the variables are shown in Table 2.2 (Chapter 2) and Table 3.2.

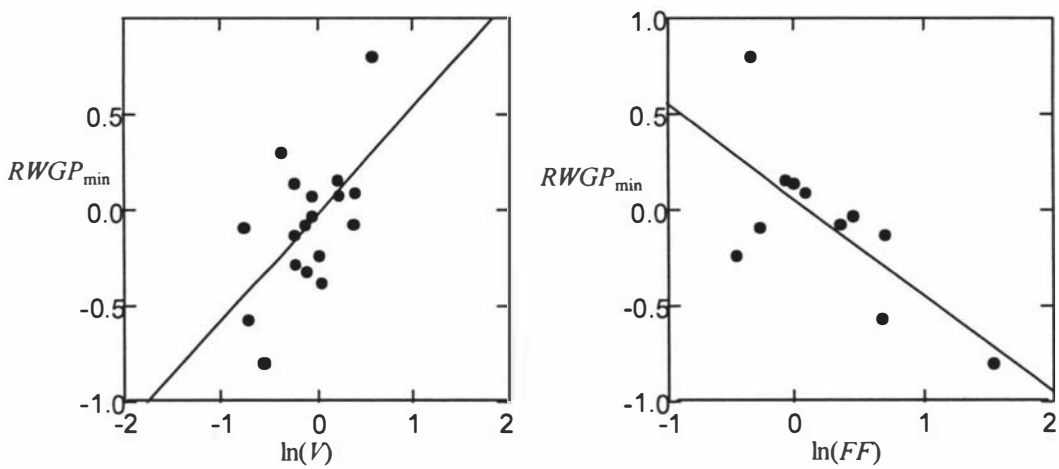

Fig. 3.9 Relationship among species between (A) relative minimum wing growth period ($RWGP_{min}$) and variability in food supply (V), and (B) relative minimum wing growth period and feeding frequency (FF). Conventions are as for Fig. 3.7. The methods for calculating the variables are shown in Table 2.2 (Chapter 2) and Table 3.2.

Differences between (relative) mean and minimum nestling periods within a species are small compared with differences between species with different variability in food supply (Fig. 3.10) (percentage of nestling period expected from mass in place

of the method described in Chapter 2 in order to make the differences in nestling period more comprehensible).

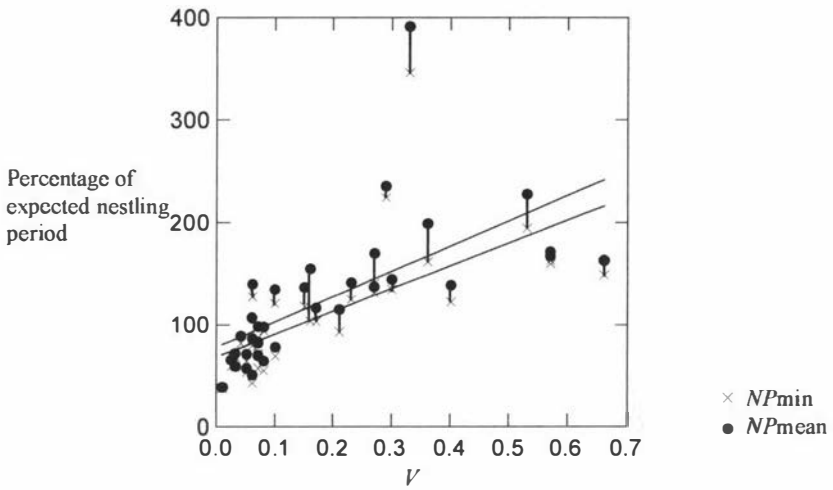

Fig. 3.10 Minimum and mean nestling periods in relation to variability in food supply in 35 species. Percentage of expected nestling period, the percentage of the nestling period expected by adult mass (Fig. 2.2); *V*, variability in food supply. Vertical lines connect minimum and mean values for a single species. The highest values are for *Procellaria cinerea,* an Antarctic winter breeder.

These data resemble the pattern shown in Fig. 3.5C, in keeping with Lack's insurance hypothesis. From these data it appears that in order for the proximate effect hypothesis to be correct, ranges of nestling periods within species would have to be on the order of from 50% to 210% of nestling period expected from mass - i.e., a difference of 160% (disregarding the outlier Grey Petrel *Procellaria cinerea,*). However, the largest range within a species is 69% in the Swift (*Apus apus*). This is twice the size of the next largest range (including the Procellariiformes). Typical values are on the order of 5-20%.

Estimated intraspecific response curves (discussed above) align with variability in food supply (*V*) in a pattern resembling that shown in Fig. 3.5D (Fig. 3.11A). Therefore, the proximate response of nestling period to variability in food supply *within* species, varies predictably *among* species - i.e., in keeping with Lack's insurance hypothesis.

Intraspecific contrasts in variability in food supply are not significantly correlated with contrasts in relative nestling period (Fig. 3.11B). This contrasts with

the *interspecific* contrasts discussed earlier, and suggests that correlations amongst interspecific contrasts cannot be explained by the proximate effect.

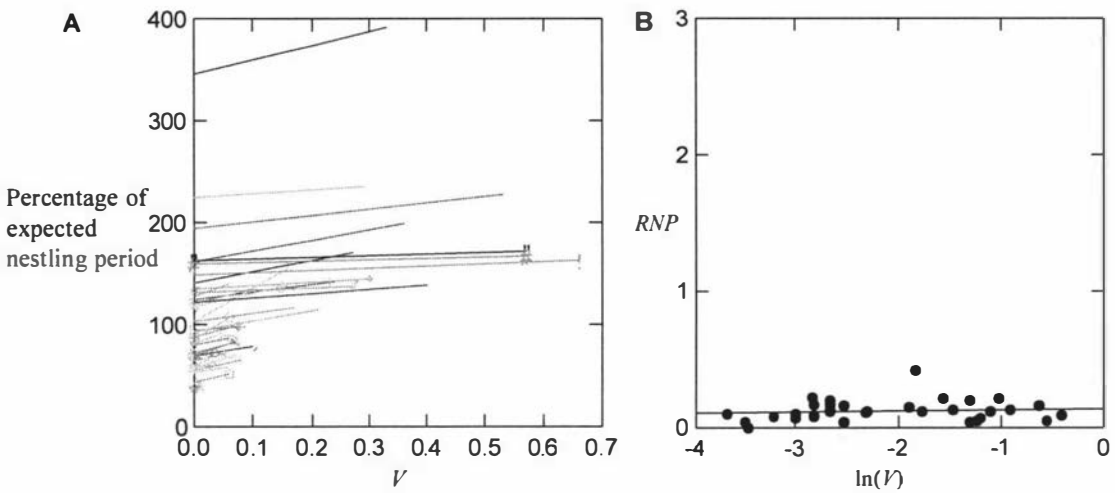

Fig. 3.11 The relationship between intraspecific contrasts in nestling period and variability in food supply. (A), Intraspecific response curves for nestling period on variability in food supply in 35 species; (B), intraspecific linear contrasts for variability in food supply and relative nestling period. The data have been plotted on the same scales as used in Fig. 3.7 for comparison with the relationship between phylogenetic contrasts.

The growth model that best fits my results is that shown in Fig. 3.5B. Species in variable environments (i.e., with a high V) have reduced minimum wing growth periods and nestling periods (Table 3.3). They also have wing growth periods that are long in comparison with their mass growth periods (Fig. 3.12, Table 3.4). Furthermore, the ratio of minimum wing growth period to minimum mass growth period becomes higher in comparison to the ratio of mean wing growth period to mean mass growth period in species with a more variable food supply (V). That is, species in variable environments tend to have a high value for the Growth Adjustment Index (*GAI*) (Fig. 3.12, Table 3.4). The ratio of mean nestling period to mean mass growth period becomes higher in comparison to the ratio of minimum nestling period to minimum mass growth period - i.e., species in variable environments tend to have a high value for the Nestling Period Adjustment Index (*NPAI*, Fig. 3.12, Table 3.4). Only the reduced lean tissue growth rate model (Fig. 3.5B) is consistent with the results of our

comparative tests. The proximate effect model (Fig. 1C) and other models (Figs. 1D-E) are falsified, hence Lack's insurance hypothesis gains additional support.

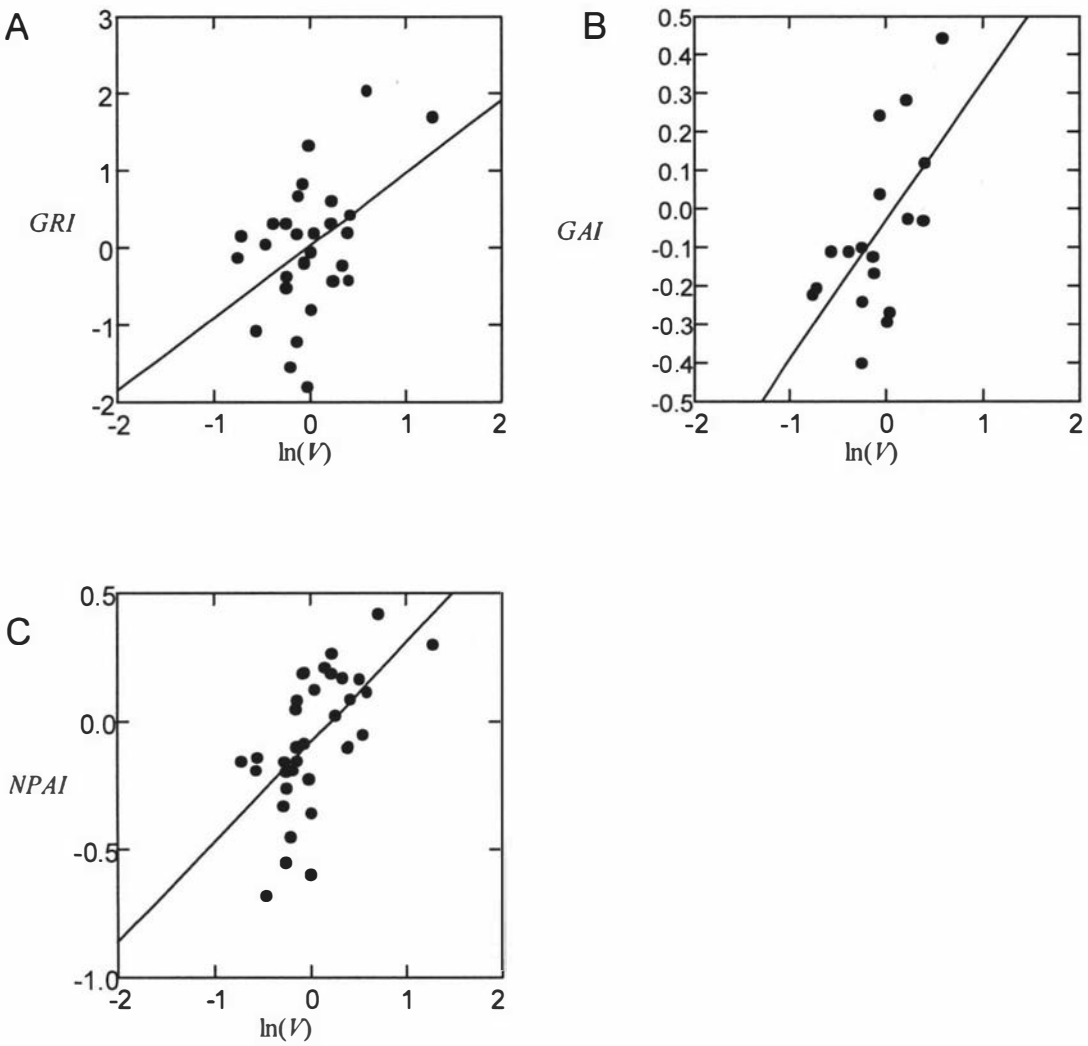

Fig. 3.12 Relationship among species between (A) Growth Ratio Index (*GRI*) and variability in food supply (*V*), (B) Growth Adjustment Index (GAI) and variability in food supply, and (C) Nestling Period Adjustment Index (*NPAI*) and variability in food supply. Conventions are as for Fig. 3.7. The methods for calculating the variables are shown in Table 2.2 (Chapter 2) and Table 3.2.

	r^2	ln(V)
GRI	0.21	0.94**
GAI	0.37	0.36**
NPAI	0.36	0.39***

Table 3.4 Effect of variability in food supply (V) on Growth Ratio Index (*GRI*), Growth Adjustment Index (*GAI*) and Nestling Period Adjustment Index (*NPAI*) using regression analysis on phylogenetically independent contrasts. Conventions are as for Table 3.3. The methods for calculating the variables are shown in Table 2.2 (Chapter 2) and Table 3.2.

Discussion

The proximate effect hypothesis appears to be partially correct in so far as the interspecific correlation between *mass* growth period/rate and variability in food supply appears to be explained to a large extent by a proximate effect. Two species of similar size with a very different *mean* mass growth rate may be much more alike in their *minimum* mass growth periods (Fig. 3.13). However, this cannot explain the interspecific correlation between nestling period, wing growth period/rate and variability in food supply. Intraspecific variation in mass growth rate/period is not closely matched by intraspecific variation in wing growth rate/period nor nestling period. This can be seen clearly by comparing variability in mass growth with variability of wing growth using the goodness-of-fit index discussed in Chapter 2 (Fig. 3.14, variability in mass growth is the index used as an indicator of variability in food supply V, I refer to it as "V_m" here to distinguish it from variability in wing growth, V_w). Hence, much of the variation in mass growth rate in species in variable environments appears to be due to variation in the rate of fat deposition. Lean tissue growth rates by contrast are relatively constant.

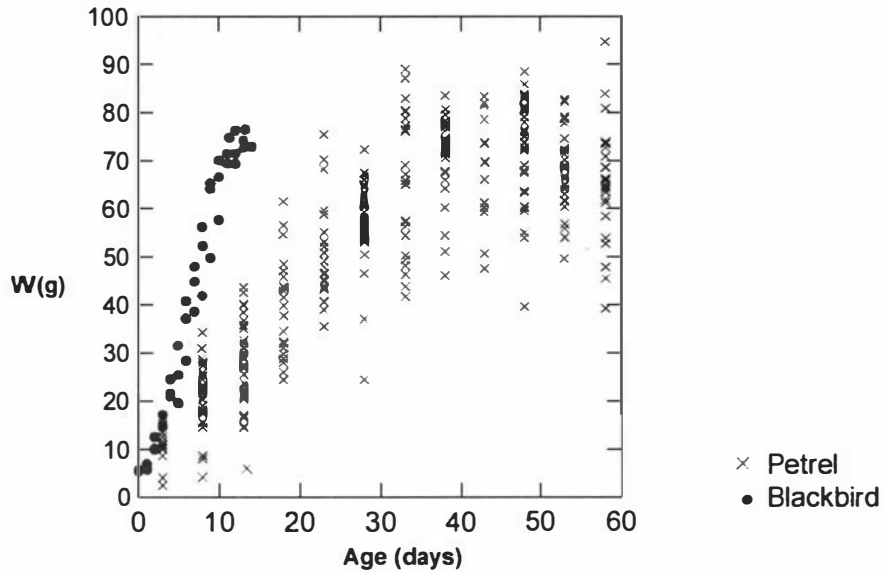

Fig. 3.13 The proximate effect explanation for a correlation between mean mass growth rate/period and V. A species (Blackbird, *Turdus merula*) with a low V (0.05) has a similar mean and maximum mass growth rate. A species (Madeiran Storm-Petrel, *Oceanodroma castro*) with a high V (0.33) has very dissimilar mean and maximum mass growth rates. The difference between mean mass growth rates in the two species is considerably more than that between maximum mass growth rates.

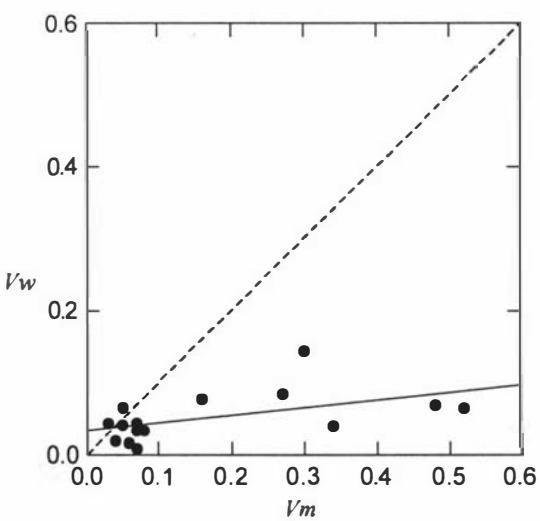

Fig. 3.14 Variability in mass growth (V_m) in relation to variability in wing growth (V_w). Dotted line shows one-to-one relationship expected from the proximate effect hypothesis.

Therefore, species in variable environments have lean tissue growth rates that are slow irrespective of proximate food supply. When food is abundant, the increased supply of energy is channeled not into increased lean tissue growth rates, but into increased rates of fat deposition. In Chapter 5 I will argue that the ability to utilize infrequently abundant food in this way is itself an adaptation of species in variable environments, and that an increased capacity for fat deposition results in a trade-off in potential for rapid lean tissue growth.

However, it could be argued that all that has been shown here is that some species with slow lean tissue growth rates sometimes grow in mass quickly because of periodic *oversupplies* in food supply, but that this doesn't mean that these species suffer food *shortages*. Against this argument, the individual growth curves used to generate the simulated growth scattergrams (Fig. 2.2, Chapter 2) showed periods of food shortages. This problem is discussed more fully in Appendix 3.

The fact that flexibility of mass growth rate does not closely correspond with flexibility of lean tissue (i.e., wing) growth rate answers a possible criticism of the tests carried out in Chapter 2. Using the same data to measure growth period and variability in food supply (hence variability in growth period) is problematic. Variance is correlated with the mean in many data sets. Therefore, *if* the test only involved mass growth period, then there would be a danger of circularity. I considered this problem briefly in Chapter 2 in my discussion of indices of variability of mass growth where I argued that the coefficient of variation of weights at 18 days is an index of variability in food supply that at independent of growth period (with qualifications). I showed that this index is closely correlated with other indices of variability in food supply, suggesting that variability in growth measured by these other scattergrams is also independent of growth period.

The data presented in this chapter support the conclusion that these tests are not circular. Variability of wing growth (V_w) is not correlated with wing growth period, and high values for variability of mass growth (V_m) are not matched by similar values for variability of wing growth. Therefore, the tests that compare nestling and wing growth period with variability in mass growth are not circular.

The model shown in Fig. 3.5B may explain these effects (variable mass growth rates corresponding to relatively constant wing growth rates). If wing growth rates are inflexible in most species, then it suggests that most species have wings that are

usually growing at a rate close to their maximum rate. For some species, this maximum rate is relatively slow, but they may nevertheless grow rapidly in overall mass. If variation between species in mass growth is largely environmental in origin, it follows that some species usually considered slow developers, are in fact capable of assimilating energy and nutrients very rapidly and mobilising them for (mass) growth.

Fluctuations in food supply may have the effect of decreasing total energy intake over the nestling period (over and above the effect of a typically long feeding interval). An alternative explanation to Lack's insurance hypothesis then is that slow growing birds in variable environments have adjusted their growth rate to this decreased level of overall energy intake (but see Appendix 3). However, I will argue below that this hypothesis is not as successful an explanation of the growth patterns discussed above as is Lack's insurance hypothesis.

In Chapter 2 I argued that growth rates may be adjusted to mean food supply. The physiological capacity for faster growth rates that the supply of food allows would be costly in terms of maintenance of the tissues required for faster growth. These costs would not be matched with the compensating benefits of faster growth. Applying this argument to a variable food supply, this means that the capacity for faster growth rates when the supply of food is good does not compensate for the costs of maintaining the tissues required for growth on poorer days. But, it appears that some species in variable environments *do* retain the capacity for fast *mass* growth that matches the supply of food on very good days even though they have a reduced capacity for *lean tissue growth.* This differential between potential mass and lean tissue growth rates, and the variation in this differential between species, is parsimoniously explained by Lack's insurance hypothesis, specifically the model shown in Fig. 3.5B. In this model, the cost of maintaining the capacity for fast lean tissue growth is the inability to grow rapidly in mass (fat) during periods of good food supply, hence survive periods of poor food supply. The hypothesis that growth rates are adjusted to a mean food supply decrease by virtue of variability in food supply (see above) does not explain a capacity for fast *mass* growth during good days - i.e., it does not explain why lean tissue growth rate but not mass growth rate should be reduced in species with a variable food supply. Therefore it, does not explain the differential growth rates of mass and wing (lean) tissue, why this differential is greater in species in variable environments, nor why

maximum wing growth rate but not maximum mass growth rate should be lowered in such species. Hence, it does not explain the correlation of *V* with *GAI*.

The correlation of feeding frequency with maximum mass growth period ($RMGP_{min}$) suggests that mass growth rates are may be adjusted to a decreased overall energy intake that results from an increased feeding interval. This contrasts with *V*, which is not significantly correlated with ($RMGP_{min}$). This in turn suggests that although species with a long feeding interval also tend to have a variable food supply, the correlation is not exact, and, species have evolved in different ways to the two aspects of their food supply.

In this chapter I have argued that the correlation between variability in food supply and growth and nestling periods is not the result of a proximate effect. The possibility remains that the correlation may be the result of variability in food supply and nestling and growth periods both correlating with degree of nest predation. I take up this problem in the next chapter.

Chapter 4

Nest Predation and Nestling Period: Confounds, Correlations, and Coadaptation

Introduction

Nestling predation, food supply, and growth rate

The relationship between variability in food supply and nestling period is complicated by the relationship between nestling predation and nestling period. Species with a low degree of nest protection may reduce nestling predation rates by evolving a shorter nestling period.

The role of nestling predation in the evolution of nestling growth rates has been recognized at least since Lack (1948) studied nestling growth rates to try and explain clutch size. He argued that reduced nestling growth rates would make larger clutch sizes possible. Food availability, variability in food supply, and nestling predation could all affect growth rate, hence clutch size. Cavity nesters tend to have larger clutch sizes, and this may be made possible by reduced nestling growth rates. Cavity nesters are known to suffer lower rates of nest predation than open or cup nesters (Lack 1954, Ricklefs 1969b). Increased growth rate should reduce the time that nestlings are exposed to predators in birds with open nests (Lack 1968).

Williams (1966) argued that natural selection will favour rapid development in species with high juvenile mortality rates. Ricklefs (1969c) criticised this argument, it not specify balancing selective forces. For example, mortality due to starvation will not necessarily be expected to select for rapid development (Case 1978). There seems to be no simple relationship between nestling mortality and growth rate. Nestling predation is one mortality factor among several, including food availability (O'Connor 1984, Martin 1992).

It is generally accepted that there is an correlation between slow growth rate and low nestling predation in cavity nesting birds, the reasons for this are debated (Martin and Li 1992). Lack's mechanism described above is one of several alternative explanations (reviewed in Martin and Li (1992)). Martin (1988) found that clutch size is generally greater in relatively exposed nests, contradicting Lack's expectation. Though Lack's theories about the relationship between clutch size, growth rate, and

nestling predation have found little support, his theories about the conflicting effect of nest predation and food limitation on growth rates continue to gather support.

The aims of this chapter

Evidence for the correlation between slow growth and low nestling predation has been accumulating in recent years (Martin and Li 1992, Bosque and Bosque 1995, Martin 1995, Martin and Clobert 1996). These studies have tested the hypothesis that long nestling period is an adaptation to a high degree of nest protection using standard regressions on interspecific data or phylogenetically independent contrasts to test for an correlation between cavity nesting and life history traits.

In this chapter I aim to study the effect of nest predation on nestling period and the relationship to food supply using original methods. The chapter has three sections. In the first section, I do a comparative analysis of nestling predation and growth rate using similar methods to Bosque and Bosque (1995). However, whereas Bosque and Bosque used standard regressions on interspecific data, I use phylogenetically independent contrasts. I also attempt to disentangle the effect of nestling predation on nestling period from the affect of feeding frequency and variability in food supply. If nestling predation is correlated with nestling period, this could confound the correlation between variability in food supply and nestling period (Chapter 2), and vice versa.

In the second section, I present a model of the selection pressures on nestling period due to nestling predation and to variability in food supply. This model predicts that behaviour (i.e., habitat preference) and physiology (i.e., growth rate and nestling period) are coadapted. In this second section I argue that species that have, in evolutionary terms, recently changed habitats, have relatively incomplete coadaptation between behaviour and growth rates. Specifically, I argue that species that have recently switched nest types will show incomplete coadaptation of nestling period to the degree of nestling predation. In order to test this prediction I modify a phylogenetic method developed by Huey and Bennett (1987).

In the third section, I argue that this method is a special case of a more general method, one that seeks to use evolutionary time-lags in order to test hypotheses about adaptation. In order to study these ideas in detail, I carry out a simulation study of several models of evolutionary change. I show that several models of evolution are compatible with my results. Finally, I apply these ideas to the coadaptation of nest

preference and nestling period and argue that this gives a stronger comparative test of the hypothesis that nestling period is an adaptation to nest predation than do studies of correlation.

Methods

I developed methods for each of the three aims discussed above. Methods and results are presented independently for each section. Sections one, two and three are labeled with roman numerals.

I. Relationship between nest predation, nestling period and growth rates

Nest predation and growth rate

To test for a relationship between degree of nest predation and growth rate, I followed the methods of Bosque and Bosque (1995). I first classified each species in the phylogeny (Chapters 2, 3 and Appendix 1) according to a simple indicator of degree of nest protection that I derived from Bosque and Bosque (1995). Species that have open nests and usually nests on continents were given a score of 2. Cavity nesting species that usually nest on continents were given a score of 1, open nesting species that usually nest on islands were given a score of -1, and cavity nesting species that usually nest on islands were given a score of -2. I also classed swallow species that nest on the undersides of vaulted places as "cavity" nesters. Ancestral nest-types were estimated by the criterion of parsimony using MacClade (Maddison 1986). These were then assigned scores according to the scheme described above and entered into the phylogeny. I then compared phylogenetically independent contrasts for nestling and growth periods with the contrasts in degree of nest protection using 59 species (Appendix 1). Contrasts in degree of nest protection were divided into three categories: those where the degree of protection increased (*P*) and those where it decreased (*U*). If there was no change in the degree of nest protection, I removed the contrast from the data-set. If protected nesters evolve relatively slow growth rates (hence long growth periods, see Chapter 2), then the changes in nestling and growth period in protected contrasts should tend to be greater than those for unprotected contrasts. I tested this using 1-tailed 2-sample t-tests. This method is derived from that used by Bosque and Bosque (1995), but with four differences. First, I used phylogenetically independent contrasts rather than using species as replicates, therefore controlling for phylogenetic

dependence. Second, I used the residuals from allometric curves (see Chapter 2), whereas Bosque and Bosque controlled for scaling relationships using the parameters (slope and y-intercept) from allometric regressions of nestling period on adult mass for protected and unprotected nesters. Third, I used mass and wing growth period as well as nestling period, and used minimum as well as mean growth and nestling periods. Fourth, I used a data-set consisting of different species to those used in earlier studies

Nest predation as a confound of variability in food supply

To examine the relationship between feeding frequency, variability in food supply, and growth and nestling periods independently of nest predation, I used the scores described above as proxies for the degree of nest protection (*PD*). These scores were used in a multiple regression among phylogenetically independent contrasts with variability in food supply (*V*) and feeding frequency (*FF*) as the other independent variables, and relative nestling and growth periods as dependent variables. A statistically significant partial regression coefficient for an independent variable indicates an correlation with growth or nestling period that is not explained by the other independent variables in the regression.

II. Coadaptation of nest site preference, nestling period, and variability in food supply

Species adapted to a variable food supply may, by increasing their nestling period, increase the amount of time over which the nestling is vulnerable to nest predators. A model of the proximate causal pathways between growth rate, nestling period, habitat preferences, nest predation, and variability in food supply, along with pathways of selective feedback is illustrated in Fig. 4.1.

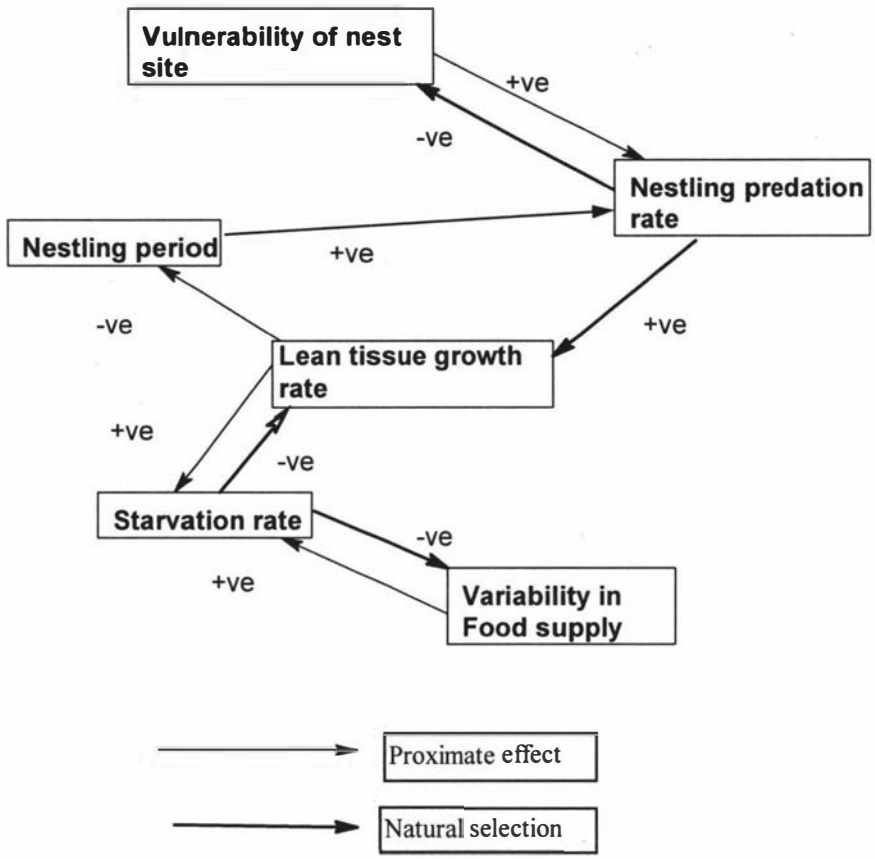

Fig. 4.1 Model of causal and evolutionary interactions between growth rate, nest predation, and nestling predation. Proximate effects are immediate causes. Arrows for natural selection indicate the resulting evolutionary feedback. Effects are either positive (+ve) or negative (-ve).

The model predicts conflicting selection pressures on growth rate by nest predation and variability in food supply. In environments with a variable food supply an increased growth rate not only reduces the time over which the chick is vulnerable to nest predators but also increases the probability of starvation (i.e., Lack's insurance hypothesis). Species should evolve toward an optimal growth rate that minimizes chick mortality due to predation and starvation. A reduction in mortality caused by starvation will cause increase in mortality due to predation. Hence there should be a trade-off between adaptation to avoid nest predation and to avoid starvation. The two evolutionary processes will tightly constrain one another, resulting in an correlation between nest predation and variability in food supply. Only those species with low nest predation should be able to adapt to a variable food supply through a slow lean tissue growth rate. Similarly, only those species with a relatively stable food supply should be

able to adapt to a nesting habitat with a low degree of nest protection through a fast lean tissue growth rate. Therefore, I predicted from the model that species that have evolved to a greater variability in food supply will also have evolved a higher degree of nest protection. To test for this, I used the three categories of change in degree of nest protection (*PD*) discussed above as the independent variable and variability in food supply (*V*) in a 1-way ANOVA analysis.

The model predicts that nesting behaviour and foraging behaviour will be coadapted with developmental physiology. The degree of protection of the nest site and the variability in food supply are both partially under behavioural control. Species show preferences for certain foraging habitats and techniques, and preferences for places to nest. High nestling predation may be mitigated by an increase in growth rate, or, by a behavioural change to preference for a more protected nest site. A more protected nest site may reduce predation risk to the extent that a species can evolve a slow growth rate and exploit more variable food supplies. Similarly, a high starvation rate may be reduced by a decrease in growth rate, or by a change in foraging behaviour. Conversely, a change in foraging behaviour to a more stable food supply may allow the evolution of shorter nestling periods, and less protected nest sites. One weakness in the model is that there is no clear incentive to evolve an unprotected nest. I assumed that unprotected (i.e., open or continental) nest sites have some advantages, e.g., access to a wide range of niches and release from competition or avoidance of pathogens and parasites which may reach high densities in enclosed nests.

I examined the idea that nest site preference is coadapted with nestling period by modifying a technique developed by Huey and Bennett (1987). When behavioural traits are coadapted with physiological traits, if an environmental change occurs, natural selection should initially favour adaptive changes in behaviour. If behavioural changes result in diminished physiological adaptation, selection for corresponding changes in physiology should result, thereby restoring coadaptation (reviewed in Huey and Bennett 1987).

Huey and Bennetts' example is the evolutionary relationship between optimal temperatures for locomotion, and temperature preference in lizards. Using an optimality model, they calculate evolutionary changes in optimal temperature (due to physiological adaptation) and corresponding evolutionary changes in preferred temperature (due to behavioural changes). Changes in preferred and optimal

temperature were derived from a phylogeny for lizards. Evolutionary changes in the lizards' physiologically optimum temperatures were generally less than - but in the same direction as - changes in temperature preference. Hence, the coadaptation between behavioural preference and physiological optimum was incomplete, and this partial coadaptation was evolutionarily derived. The general principle underlying Huey and Bennetts conclusions is illustrated in Fig. 4.2A.

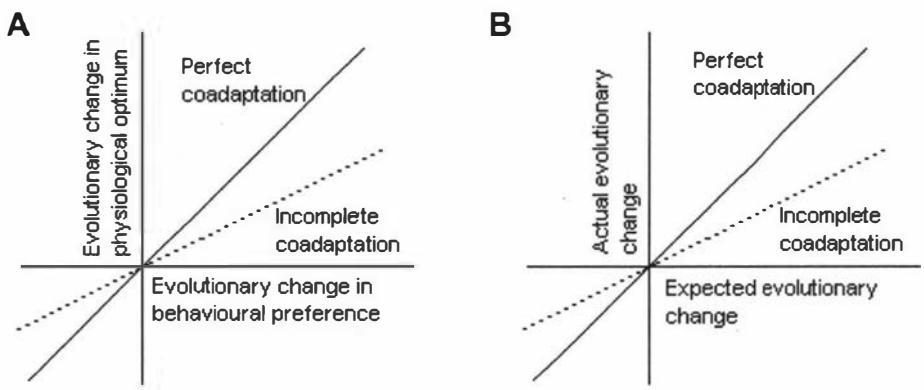

Fig. 4.2 (A) Coadaptation between behavioural and physiological traits. This figure shows incomplete coadaptation that may result when natural selection initially favours change in a behavioural trait (see text). (B) Evolutionary change in a physiological trait is less than that expected given an evolutionary change in a behavioural preference. Expected values are calculated from the relationship between the behavioural preference and physiological trait in species where the behavioural preference is the ancestral condition.

I modified these methods to examine the degree of coadaptation between nest type preference and nestling period. If the correlation between cavity nesting and long nestling periods is a result of evolution, rather than coincidental correlation, then species that have changed nest site preferences relatively recently should be relatively less coadapted than species that have a long evolutionary history in the same nest type. This is a stronger test of the evolutionary model than phylogenetically independent contrasts, as it tests for the degree, direction, and potentially the rate of evolutionary change between ancestral and daughter nodes, rather than just correlations of variables among species and clades.

In order to apply Huey and Bennetts method, behavioural and physiological traits must be measured with respect to single variable, in their case temperature. In order to relate nest type preference and growth rate to a single variable (nestling period), I first conjectured that in species where the nest type preference (cavity or open) is in the ancestral condition, coadaptation between nest type preference and nestling period would be more complete than in those species where the nest type preference is recently derived. I refer to former type as "old" cavity or open nesters, and the latter type as "new" cavity or open nesters. I then calculated allometric regression equations for the log of nestling period on the log of adult mass for both old cavity and old open nesters. I assumed these equations to be an estimate of the relationship between mass and nestling period in species where the coadaptation of nestling period to nest type is relatively complete. I used the regression equations to calculate the expected change from the ancestral nestling period for species where the nest type is recently derived. This is the change in nestling period expected from a change in behavioural preference (for a particular nest type). This modified procedure is illustrated in Fig. 4.2B.

If "new" nesters have a similar degree of coadaptation between nestling period and nest type as do "old" nesters, then actual evolutionary change should be consistently as great as expected evolutionary change. If, however, actual changes are consistently less than expected changes, then new nesters are relatively less coadapted than old nesters, and this is evidence of gradual adaptive evolution.

To calculate the regression equations for "old" nesters I used the extensive data base provided by Bosque and Bosque (1995) for data on mass, nestling period, incubation period, and nest type. This was supplemented with data from Bateman and Balda (1973), Murphy (1983), Cramp and Simmons (1980), and Redfern (1994). Since one nest type was overwhelmingly dominant in each family, I took the predominant nest type for that family as a taxonomic approximation of the ancestral (or "old") condition (see also Fig. 4.9). Species in a rare nest type for a family were classified as "new". In some cases I had information on incubation period but not nestling period for a species. In such cases I estimated nestling period from incubation period using a log-log regression model. Raw data for adult mass, incubation period, nestling period and nest type are given in Appendix 4.

To calculate evolutionary changes, both present-day and ancestral values are required. I used the family means for nestling period as estimates of ancestral nestling period. Expected present-day nestling periods in new nesters were calculated from adult body mass using the regression equations for old nesters - i.e., if the new nest type was cavity nesting, then the expected present-day nestling period was calculated from adult body mass using the allometric regression for old cavity nesters.

A problem with this method of analysis is that there are necessarily few members of the new categories. I compiled data for 139 species in 14 families. Of these, 86 were old open nesters, 46 old cavity nesters, 5 new cavity nesters and only 2 open. However, this compares favourably with Huey and Bennett, who examined 6 pairs of evolutionary changes.

III. Partial coadaptation and evolutionary time-lags

The method described in the last section seeks to explain partial coadaptation as the result of an evolutionary time-lag. The evolution of physiology lags behind that of behaviour. Other researchers have predicted an evolutionary time-lag when faced with incomplete coadaptation between a behavioural preference and a physiological trait. Davies (1992) argued that the degree of egg mimicry of Cuckoo eggs to the eggs of host species could be explained by an evolutionary time-lag. Host preference has presumably evolved faster than egg-shell physiology. If phylogenetic analysis were to show that the degree of egg mimicry increases with the time spent with a host species, then this would be evidence of evolution in the direction of increasing coadaptation between host preference and egg mimicry.

This argument may be generalised. Evolutionary hypotheses not only predict "horizontal" patterns in a phylogeny (phylogenetically independent contrasts), but also "vertical" patterns (phylogenetic transformations). Hence, evolutionary hypotheses predict the correlation of lengths of phylogenetic branches with the magnitude of change of variables along the branches. Phylogenetic analyses that show evolutionary transformations have been called "directional methods" by Harvey and Pagel (1990). Directional methods provide stronger tests of evolutionary hypotheses than non-directional tests, but require more information.

In this section I describe a general directional method for testing evolutionary hypotheses. I examine the logic of this method using a simulation study. I also criticise

Huey and Bennetts argument that showing a time-lag between a behavioural and a physiological trait implies that behavioural changes are the initiators or "drivers" of evolutionary change. Instead, I argue that although behavioural traits may evolve at a faster rate than physiological traits, the initiative for evolutionary change may come from either behavioural or physiological changes. In the results, I apply the conclusions of this analysis to the nest preference and nestling period data discussed above. I argue that nest preference evolves more rapidly than nestling period, but that it is not possible to conclude that behavioural evolutionary change in nest preference always precedes an evolutionary change in nestling period.

A general directional method

Phylogenetically independent contrasts are usually used in non-directional tests. Contrasts for one trait are compared with those for another trait. However, the comparison of contrasts with the time since the divergence of sister species or clades from a common character state, can provide the basis of a transformational or directional test (Fig. 4.3).

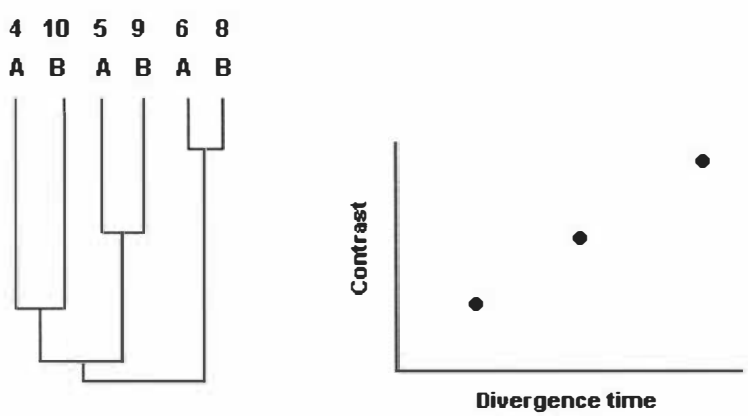

Fig. 4.3 The correlation of phylogenetically independent contrasts with time since divergence from a common ancestor. A and B are two character states, and the numbers are values for a continuous variable. The contrast between species for the continuous variable increases with the time since the species diverged from a common character state.

This method requires relatively precise data on phylogenetic branch lengths. A less precise method may use "old" (ancestral) and "new" (recently derived) categories

as rough approximations of branch lengths (as discussed above). For a binary character state, such as cavity and open nesting, this gives four categories; old cavity nesters, old open nesters, new cavity nesters, and new open nesters. If, when nest preference has changed, the evolution of nestling period is a gradual transformation from the typical state for the ancestral nest type toward the typical state for the new nest type, then intuitively, the following pattern should result. Old cavity nesters will have a long nestling period and old open nesters a short nestling period. New cavity nesters will have similar nestling period to their ancestral condition - i.e., that of old open nesters. However, they will show some change toward the characteristic nestling period for old cavity nesters. A similar pattern will hold for new open nesters (Fig. 4.4A).

However, this pattern should only occur if the rate of evolutionary change of nestling period is relatively slow. If nestling period evolves relatively quickly, then new nesters will be very similar to old nesters of the same category (Fig. 4.4B). I refer to this as punctuationistic evolution (Gould 1982), according to which, when there is a change in nest type, the nestling period rapidly changes toward a new equilibrium. The method of comparing phylogenetically independent contrasts with divergence times depends upon the rate of evolution being relatively slow compared to the span of time over which evolutionary change can be measured. If change from one equilibrium state to another takes place over a period shorter than the average time between speciations in a phylogeny (or shorter than the period of time since "new" nesters changed from the ancestral condition), then evolutionary change is punctuationistic. The gradualistic model and the punctuationistic models are not mutually exclusive models of evolutionary change. Instead, they are the result of my lack of detailed knowledge of the timing of speciation events.

It is possible that for some branches in a phylogeny, evolution has proceeded relatively slowly (relative to my knowledge of the phylogeny), whereas in other branches, evolution has proceeded relatively rapidly. This will give data that are a mix of the pure gradualistic and punctuationistic models (Fig. 4.4C), which I refer to as the "variable rate" model of evolution. According to this model, there should still be a time lag between the evolution of nest preference and evolution of nestling period, but this will not be as clear as it would be if the rate of the evolution of nestling period took place at a relatively constant and slow rate.

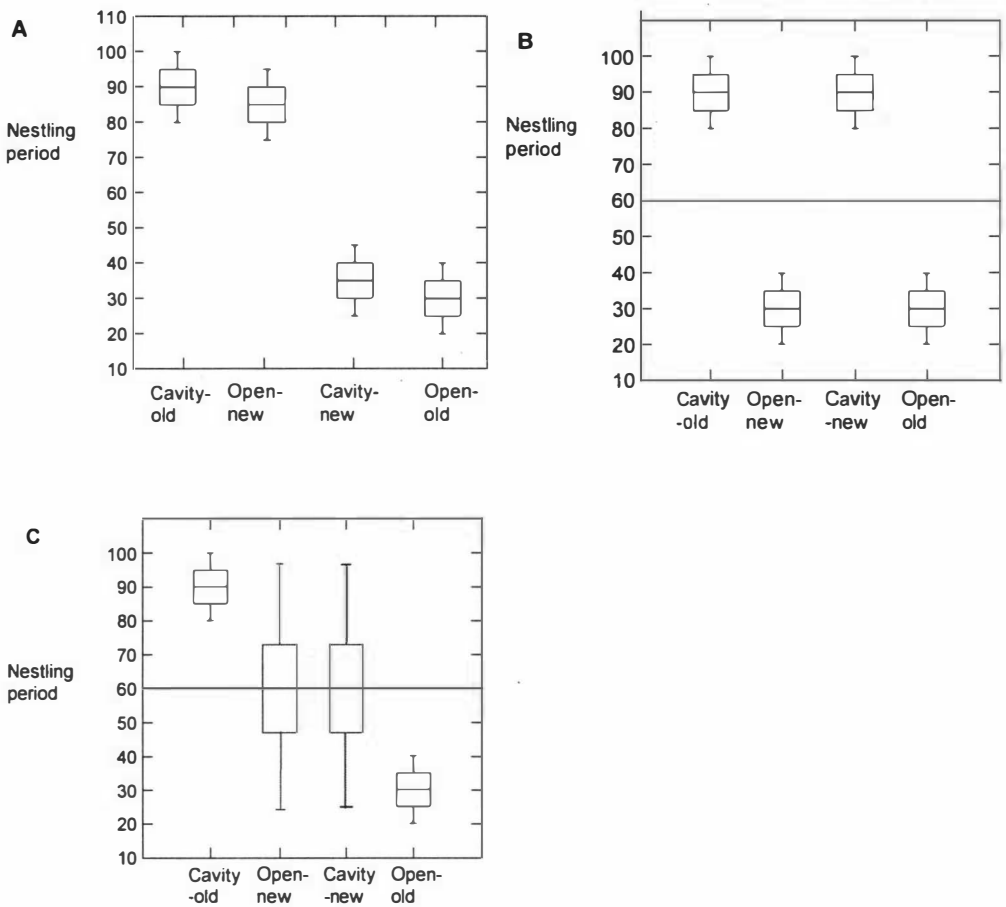

Fig. 4.4 Models of evolution when nest preference is the "cause" and nestling period is the "effect". (A), nestling period changes gradually and at a relatively constant rate. The rate of evolution of nestling period is slow relative to the how recent change in nest preference is for "new" nesters, (B), nestling period changes rapidly or punctuationistically - i.e., the rate of evolution of nestling period is rapid relative to the how recent change in nest preference is for "new" nesters, (C), nestling period evolves at different rates in different species - i.e., the rate of evolution of nestling period is variable relative to how recent change in nest preference is for "new" nesters.

All three models of evolution discussed so far (gradual, punctuationistic, and variable rate), assume that nest preference is the "cause" of evolutionary change, and that nestling period evolves in response to nest preference. However, the direction of evolutionary cause and effect may run in the other direction. An initial change in nestling period may change the selective pressures on nest preference. If the method of identifying time-lags in the coadaptation of traits developed here is sound, then it should not be important which trait changes initially. All that is required for

directionality to be detected by this method is that once evolutionary change is underway, one trait is able to respond more rapidly than the other. Nestling period may undergo an initial change, which sets up the conditions for selection for evolution in nesting behaviour which then proceeds more rapidly than the evolution of nestling period.

To investigate this idea, I constructed several models of evolution in which nestling period is the independent or causal variable and nest preference is the dependent or effect variable. These are not detailed models of natural selection, but coarse grained models of evolutionary change. Nestling period changes randomly, and this in turn causes nest preference to change. The models fall into two sets of categories according to (i) the way in which nestling period changes over time, (ii) the way in which nest preference responds to change in the nestling period. This gives four combinations, hence four models. The simulation results are shown in Fig. 4.5.

(i) Nestling period evolves by random *discrete* changes (here discrete change means that changes in nestling period can be up to 40% of the maximum nestling period in one round) Nest preference changes when the nestling period crosses a threshold value (Fig. 4.5A)

(ii) Nestling period evolves by random but *gradual* change (where gradual means that changes in nestling period are less than 4% of the maximum nestling period in one round). Nest preference changes when the nestling period crosses a threshold value. (Fig. 4.5B) .

(iii) Nestling period evolves by discrete changes. The probability of the nest preference changing increases as the nestling period changes - i.e., as nestling period gets longer the probability of switching to cavity-nesting increases (Fig. 4.5C).

(iv) Nestling period evolves by gradual change. The probability of the nest preference changing increases as the nestling period changes (Fig. 4.5D).

I wrote programmes to simulate each of these processes (code in Appendix 5). Each program generates a set of cavity and open nesters and their nestling periods. These are further classified into "new" and "old" according to the number of rounds since the last change in nest preference.

In the simulations, minimum and maximum nestling period were set at 5 and 15 days respectively. In each round of a simulation, a random nestling period between 5 and 25 "days" is generated. If less than 15, then it is initially classified as an open

nester. If over 15 days it is initially classified as a cavity nester. Then evolution in nest preference and nestling period proceeds according to the model being simulated. Each round of the simulations represents a period of evolution over which nestling period and nest preference have changed. In the models where nestling period evolves by discrete changes, in each period (round) nestling period changes randomly by a number of days between 0 and 10. In the models where nestling period evolves by gradual changes, nestling period changes randomly in each round by a number of days between 0 and 1. In the models where nest preference changes when nestling period crosses a threshold value, the threshold is 15 days. If nestling period evolves to greater than 15 days nest preference switches to cavity nesting, and if nestling period evolves to less than 15 days nest preference switches to open nesting. In the models where the probability of switching nest preference to cavity nesting increases as nestling period gets longer (and the probability of switching to open nesting increases as nestling period gets shorter) the probability of switching to cavity nesting from open nesting (Ps) was calculated from a Equation 4.1 which treats the probability of switching nest type as a linear function of nestling period (NP). The probability of switching to open nesting from cavity nesting is $1 - Ps$.

Equation 4.1 $$Ps = \frac{5.NP - 25}{100}$$

"Evolution" continues for a random duration of between 50 and 250 rounds, representing evolution along a branch in a phylogeny. For each model I simulated 100 such "species" and for each recorded the final nestling period, nest type, and time (number of rounds of simulated evolution) in the final nest type. Those runs that had spent more rounds than a number "T" in the same nest type leading up to "present" were classified as "old". In the simulation results presented below (Fig. 4.5) T was set at 20 rounds of evolution. In other simulations I set T at higher values. This had the effect of reducing the distinctions between the old and new categories.

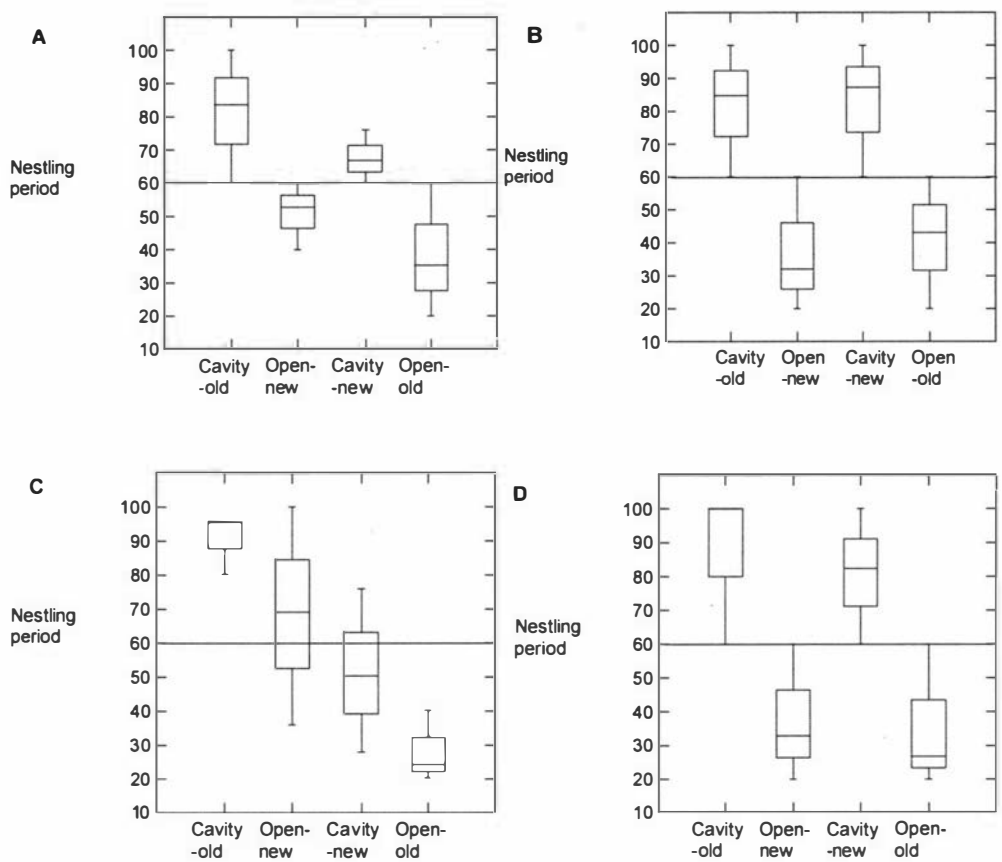

Fig. 4.5 Simulation results produced by four models of evolution in which nestling period is the "cause" and nest preference the "effect". (A) nestling period changes gradually and nest preference changes at a threshold nestling period, (B) nestling period changes in discrete and nest preference changes at a threshold, (C) nestling period changes gradually and the probability of nest preference changing varies with nestling period; D, nestling period changes in discrete jumps and the probability of nest preference changing varies with nestling period. Horizonal lines are median values; boxes are 1st and 3rd quartiles; vertical bars are 95 percentiles.

The results shown in Figs. 4.5C are similar to the pattern that I predicted for the "variable rate model" (where nest type is the causal variable and nestling period is the effect variable, Fig. 4.4C). This suggests that a similar evolutionary lag between the coadaptation of traits can result irrespective of which trait undergoes the initial change. Whether it is nest preference or nestling period that changes first is irrelevant to the argument that partial coadaptation will result from evolutionary time-lags. In either

case it is the relatively slow evolution of nestling period that is the factor responsible for incomplete coadaptation. Hence, the phylogenetic methods for studying partial coadaptation that are discussed in this chapter should be valid irrespective of whether behavioural preference, or physiology, initiates change. The crucial point is the differential *rate* of behavioural and physiological evolutionary change, irrespective of which factor changes first. Huey and Bennett contend that an evolutionary lag between physiology and behavioural preference shows that behaviour is the initiator of evolutionary change. If my analysis is correct, then this need not be the case.

I re-analysed the data discussed above for nestling period in cavity and open nesters using the diagrams shown in Figs 4.4 and 4.5. If the data are similar to the pattern shown in Figs. 4.4C and 4.5C, then it may be concluded that nestling period evolves relatively slowly (rapid evolution of nestling period means either the patterns shown in Fig. 4.4B, Fig. 4.5B or Fig. 4.5D) but not that nest preference is the initiator of evolutionary change. However, even if the data show the pattern described by Figs 4.4C and 4.5C it is possible that this is due to chance alone. This is especially likely in this example, because the sample sizes for that new nest types are small. It is possible that, for nestling period, new cavity nesters are simply a random sub-sample of old cavity nesters. To test for a significant difference between old and new cavity nesters I carried out a randomization test. In each round I combined data for relative nestling period for all cavity nesters into one population and drew 5 random species and measured the difference in means between the sub-sample and the remaining data. I then measured the frequency at which this re-sampled difference was equal to or greater than the observed difference (Simon 1995). From this, I calculated the probability that the new open nesters are a sub-sample of old open nesters.

Results

The results are divided into three sections corresponding to the three sections discussed in the introduction and in the methods.

I. Nest predation and growth rate

Species that have evolved more protected nest sites have evolved longer nestling periods and slower growth in comparison to species that have evolved less protected nest sites (Table 4.1). That is, species changing to a protected nest sight

decrease growth rate and increase nestling period, or, species changing to an unprotected nest site increase growth rate (hence decrease growth period) and decrease nestling period. The correlation between changes in relative wing growth period (*RWGP*) with changes in nest protection (*PD*) were only marginally significant. However, there were few contrasts in wing growth period where there was also a contrast in nest type, an unavoidable effect of this method.

	Mean		Std. dev.		N	P
	P	*U*	*P*	*U*		
RNP	0.29	-0.39	0.36	0.34	19	0.0005
RNP_{min}	0.30	-0.38	0.37	0.36	17	0.001
RMGP	0.17	-0.36	0.45	0.41	18	0.01
$RMGP_{min}$	0.11	-0.28	0.34	0.28	18	0.01
RWGP	0.60	-0.29	0.86	0.34	12	0.02
$RWGP_{min}$	0.16	-0.29	0.45	0.38	9	0.06

Table 4.1 Phylogenetically independent contrasts in mean and minimum nestling and growth periods for contrasts that had evolved and increased degree of nest protection (*P*) and contrasts that had evolved a decreased degree of nest protection (*U*). See Tables 2.2 (Chapter 2) and 3.2 (Chapter 3) for abbreviations. N, combined sample size. P indicates the statistical significance of the difference between means using a 1-tailed, 2-sample t-test.

When contrasts for variability in food supply (*V*), feeding frequency (*FF*) and degree of nest protection (*PD*) were simultaneously used as independent variables, all were significantly correlated with relative nestling period (*RNP*, Table 4.2). Minimum nestling period was significantly correlated with variability in food supply and nest predation, but not feeding frequency. Wing growth period was also significantly correlated with degree of nest protection. This suggests that although variability in food supply and degree of nest protection are (negatively) correlated, they both have an effect on the evolution of growth and nestling periods.

	r^2	ln(V)	ln(FF)	PD
RNP	0.60***	0.33**	-0.31**	-0.31*
RNP_{min}	0.74***	0.58***	-0.16	-0.35**
$RMGP$	0.54***	0.49***	-0.30*	-0.14
$RMGP_{min}$	0.24**	0.07	-0.36*	-0.22
$RWGP$	0.50**	0.47*	-0.08	-0.29*
$RWGP_{min}$	0.72**	0.55*	-0.58*	0.03

Table 4.2 Effect of variability in food supply (V), feeding frequency (FF) and degree of nest protection (PD) on nestling period and growth periods, tested using multiple regression analysis on phylogenetic contrasts. r^2 given for the proportion of variation explained by all three independent variables. V, FF and PD show standardised partial regression coefficients. See Tables 2.2 (Chapter 2) and 3.2 (Chapter 3) for definitions of parameters. Statistical significance in the overall model, and for the partial coefficients, is indicated by asterisks: * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

II. Coadaptation of nest site preference, nestling period, and variability in food supply

The degree of protection of the nest (PD) was strongly correlated with variability in food supply (V) (1-way ANOVA, $P = 0.001$, Fig. 4.6). Where there was no contrast in the degree of protection of the nest site, the average contrast in variability in food supply was close to zero. Where there was a positive or negative contrast in the degree of protection of the nest site, there was an inverse contrast in variability in food supply. This suggests that the evolution of nest site preference is coadapted with the variability of the foraging niche.

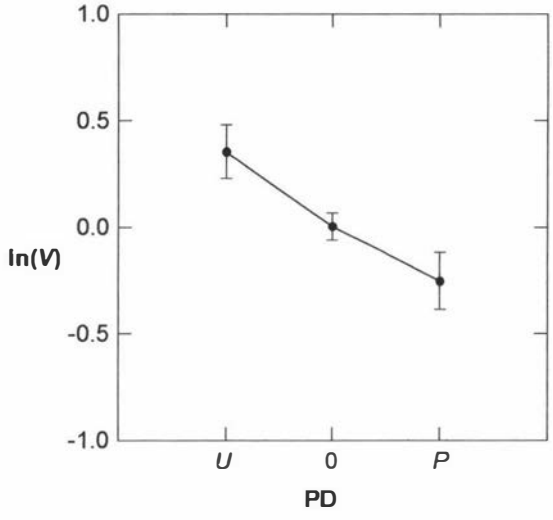

Fig. 4.6 Relationship between phylogenetic contrasts in degree of nest protection and variability in food supply in 52 species. *PD*, dummy variables for nest protection (see text); ln(*V*), natural log of variability in food supply. Points are means of phylogenetic contrasts and error bars show standard deviations.

Coadaptation of nest preference and nestling period

I compared the natural log of nestling period with the natural log of adult mass for "old" cavity nesters and for "old open nesters (Fig. 4.7). "Old" cavity nesters had longer nestling periods at a given adult mass. I used 6 families of cavity nesters and 9 families of open nesters. Hence, these results the follow should be independent of phylogeny.

I calculated allometric regressions for this data to estimate the expected nestling period for species of a given adult mass in cavity nests and in open nests (old cavity nesters, Equation 4.2; old open nesters Equation 4.3).

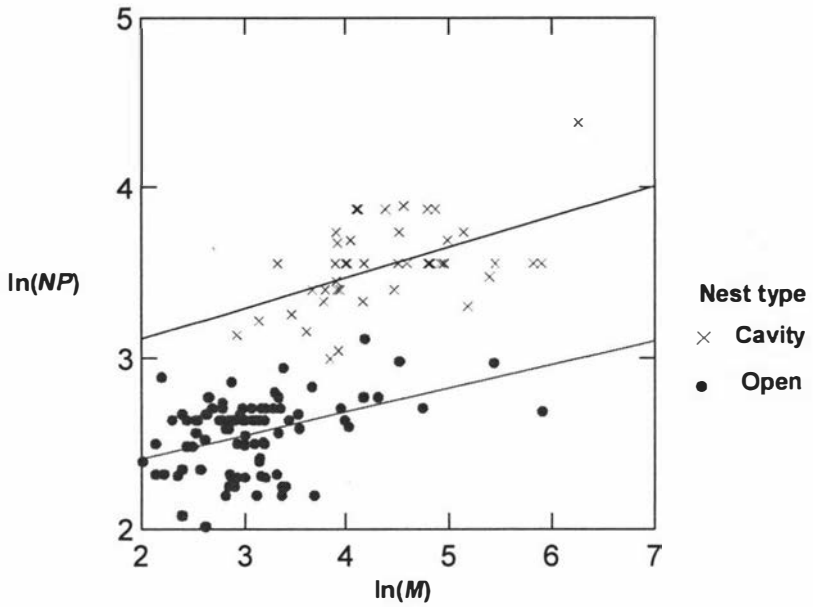

Fig. 4.7 Adult mass and nestling period in species for which cavity nesting and open nesting are the ancestral condition. ln(NP) is the log of nestling period; ln(M) is the log of adult mass; lines are linear regressions of log-transformed variables.

Equation 4.2 $\ln(NP) = 0.18.\ln(M) + 2.76$

Equation 4.3 $\ln(NP) = 0.14.\ln(M) + 2.13$

The actual change in nestling period from ancestral to the present condition in new nesters was consistently less than that expected from Equations 4.1 and 4.2 (Fig. 4.8). The change in nestling period in each case was in the expected direction, though of a lesser degree than expected. This supports the hypothesis of evolutionarily-derived incomplete coadaptation between nest preference and nestling period.

In the five cases where species had changed to cavity nesting, nestling period changed much less than expected with respect to Equations 4.2 and 4.3. In the two cases where species had changed to open nesting, nestling period had changed by an amount very close to that expected. Hence, coadaptation between nest preference and nestling period *may* be more complete in new open nesters than new cavity nesters.

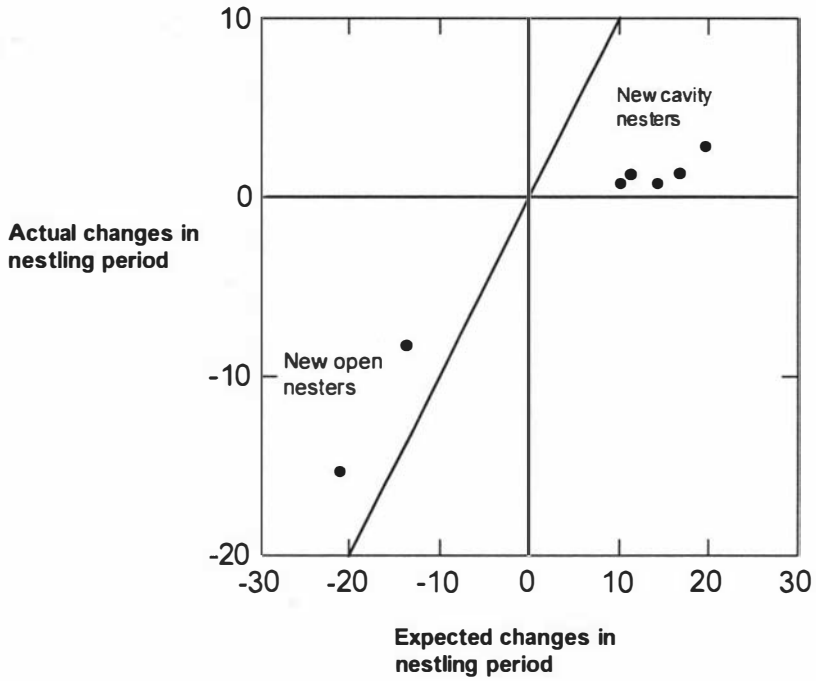

Fig. 4.8 Evolutionary changes in nestling period expected from change in nest type preference with respect to Equations 4.2 and 4.3, and actual changes in nestling period. The line represents perfect coadaptation.

III. Partial coadaptation and evolutionary time-lags

When I constructed a phylogeny of new nesters, and closely related species or genera, I was unable to find precise estimates of branch lengths (Fig. 4.9). Also, of the five contrasts where I had good information on divergence times, variation in divergence times was small. Hence, I could not use this phylogeny to test the hypothesis that contrasts between nestling period in open and cavity nesters increases with the amount of time since divergence from a common nest type.

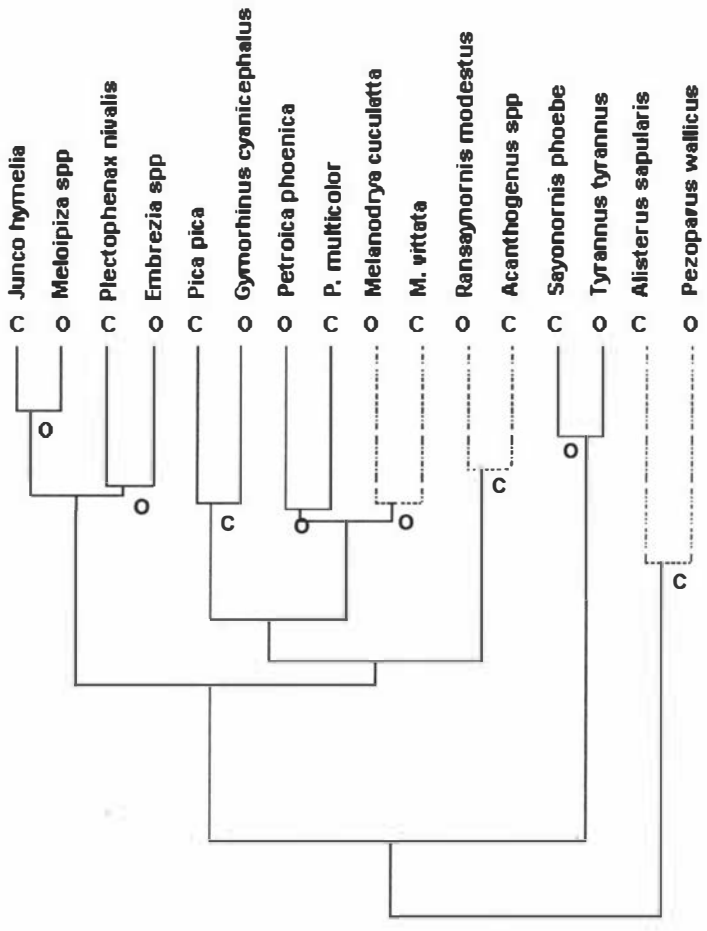

Fig. 4.9 Phylogenetically independent contrasts for nest type. C, cavity nester; O, open nester. Dotted lines show maximum estimates for branch lengths only. Actual divergence times may be much shorter. Ancestral nodes have been classified on the basis of parsimony applied to the data-set in Appendix 4, which has been treated as a taxonomic approximation of phylogenetic relationships between species. *Junco hymenalis* is an occasional cavity nester.

I plotted relative nestling period (see Chapter 2) for old and new, cavity and open nesters (Fig. 4.10). Relative nestling period was calculated from allometric regression equations. However, as shown above (Fig. 4.7, Equations 4.1 and 4.2), the relationship between adult mass and nestling period is itself dependent on nest type, and this could bias the results. Hence, I repeated the test using relative nestling period calculated form the allometric regression for open nesters only, and from the allometric regression for cavity nesters only (Fig. 4.10).

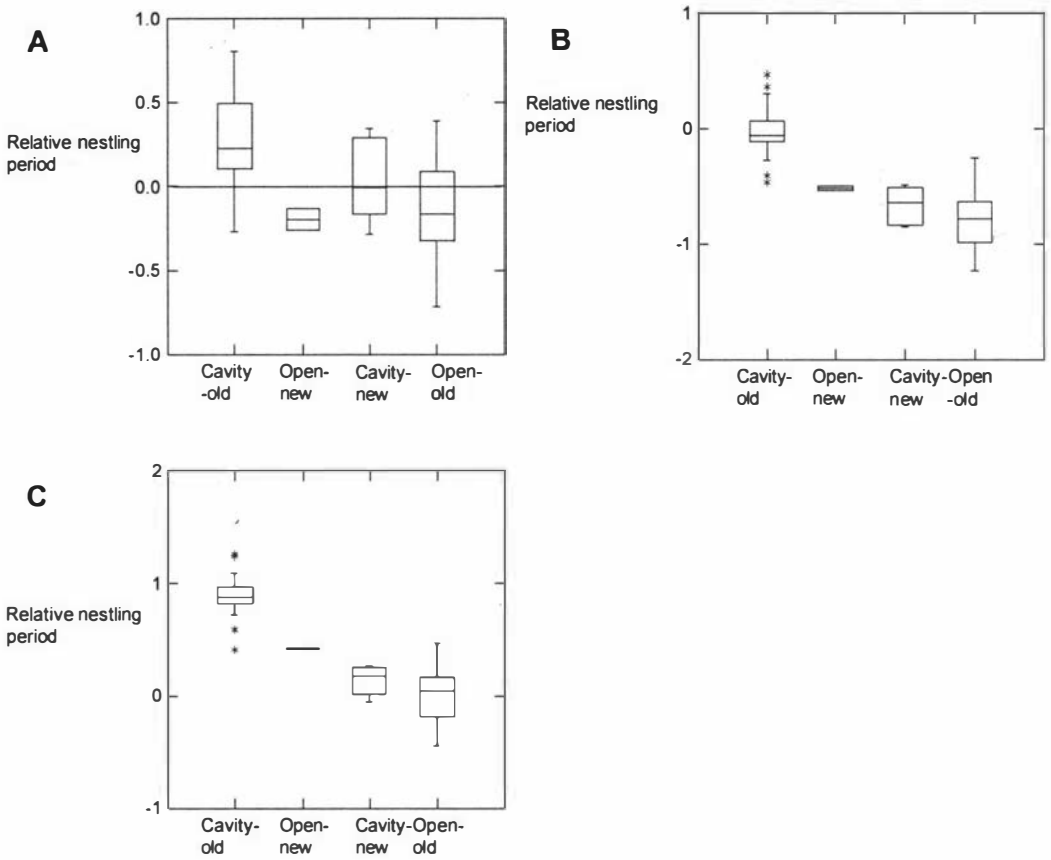

Fig. 4.10 Relative nestling period in old and new, cavity and open nesters. (A) relative nestling period calculated the allometric regression for pooled data for cavity and open nesters, (B) relative nestling period calculated from the equation for open nesters only (C), relative nestling period calculated from the equation for cavity nesters only. Conventions are as for Fig. 4.5.

By using the randomisation test described above, I calculated for Fig. 4.10A that the probability that the relative nestling periods for new cavity nesters are a sub-sample of those for old cavity nesters is 0.004. The data support the patterns shown Figs. 4.4A, 4.4C, 4.5C, and 4.5A. Hence, either nest preference changes and nestling period gradually responds, or, nestling period evolves gradually and nest preference evolves in response. That is, in all the models, nestling period evolves relatively slowly with respect to changes in nest preference.

Discussion

The correlation between nest type and nestling period in my data-set corroborates recent studies on nest predation and life-history (Martin and Li 1992,

Bosque and Bosque 1995, Martin 1995, Martin and Clobert 1996). This analysis however, has several original points. First, it is not just nestling period, but also measures of growth rate (i.e., growth periods) that correlate with degree of nest protection. Second, the correlation remained when I used degree of nest protection as an independent variable along with variability in food supply and feeding frequency in a multiple regression analysis using mean and minimum nestling and growth periods as dependent variables. Third, these effects appear to be independent of phylogenetic dependence.

Phylogenetically independent contrasts in variability in food supply are strongly, and negatively, correlated with phylogenetically independent contrasts in degree of nest protection. This suggests that nest predation and variability in food supply cause conflicting selection pressures on growth rates and nestling period. Hence, nestling periods may represent a trade-off between these two selection pressures.

An interaction of different selection pressures on nestling period suggests that growth rate will be coadapted with both nest preference and foraging behaviour. The degree of coadaptation between nest preference and nestling period appears to be dependent upon the amount of evolutionary time that a species has spent with the same nest preference. Hence, incomplete coadaptation between nest preference and nestling period appears to be evolutionarily derived, thus corroborating the hypothesis that the correlation between nest type and nestling period is the result of adaptive evolution.

Coadaptation between nest preference and nestling period appears to be more complete in new open nesters than new cavity nesters. This may be because the evolution of nestling period in new open nesters may is faster than that in new cavity nesters. This in turn may be the result of stronger selection pressures for changes in nestling period in open nesters than cavity nesters. This would be the case in species where selection pressure due to nest predation is relatively stronger in comparison to that due to the food supply.

Further analysis of partial coadaptation of nest preference with nestling period showed the data to be compatible with two different models of evolution. Either nest preference initially changes, and nestling period gradually evolves in response, or nestling period initially changes, and this causes evolution in nest preference. Both scenarios are compatible with the model of evolutionary interactions shown in Fig. 4.1.

The question as whether nest preference or nestling period first responds to an environmental change, is irrelevant to this model. Hence, an evolutionary lag between behavioural and physiological traits cannot support that idea that behavioural changes are the "drivers" of evolution in the sense that behavioural changes initiate new evolutionary transformation (Baldwin 1896). However, it is consistent with the weaker hypothesis that behaviour may evolve more quickly than physiology in response to earlier changes in the environment, physiology, or other aspects of behaviour.

However, there are two points that qualify the conclusions drawn from these data. First, are the small and very uneven sample sizes, combined with the fact that the distinctions between the the models are qualitative patterns, do not facilitate the use of quantitative statistical tests. However, the null or a-directional hypothesis would seem to be refuted by the low probability that new cavity nesters are a subsample of old cavity nesters, and the qualitative predictions of the alternative models seem inadequate to explain the data. Second, species are not randomly distributed for mass with respect to nest type. In this data set cavity nesters tend to be larger than open nesters. Since relative nestling period is calculated on the basis of a model fitted between mass and nestling period, this compounds the problem of finding a good baseline from which to calculate expected nestling period. A more severe test of the hypothesis will require more evenly distributed data.

The use of directional or transformational phylogenetic tests as developed in this chapter may represent an advance on phylogenetically independent contrasts. These methods use the idea of phylogenetic inertia, or evolutionary gradualism, to show the correlation of some trait, or the contrast between traits in related species, with evolutionary time. Phylogenetically independent contrasts test for a correlation between traits. Transformational methods also test the prediction of evolutionary change over time, hence providing for more a powerful test of evolutionary hypotheses.

Chapter 5

Explaining further the correlation between variability in food supply and prolonged nestling period; mechanisms of growth

Introduction

According to Lack's insurance hypothesis, slow growth insures chicks against starvation. Lack did not fully explain how this may work, although he did expect that slower growth should save energy, and that this is instrumental to surviving food shortages (Lack 1968). In this chapter I test this model (which I call the "energy savings model") and two other possible explanations for Lack's insurance hypothesis, the "slow metabolism model" and the "cell allocation model". I also test two models that may explain the correlations described in Chapter 2, but have no relationship to the Lack's insurance hypothesis. The first is a version of the "nutrient limitation model" and the second is the "developmental coupling model". The introduction, methods, and results sections are divided into five sections corresponding to the five models. Predictions from the five models are summarised in Table 5.1 at the end of the methods.

I. The energy savings model

The energetic costs of growth are relatively low, biosynthesis appears to makes up only a small component of the total metabolism in growing chicks. If energy is the main limiting factor on avian growth rate then calculations from energy budgets show that chicks of Leach's Storm-Petrel could double their lean growth rate with only a 5% increase in energy intake (Ricklefs *et al* 1980, 1985). Hence, even a marked reduction in growth rate should not be expected to significantly reduce energetic requirements during periods of starvation.

If energy is the main limiting factor on avian growth rate, then calculations from energy budgets show that chicks of Leach's Storm-Petrel could double their lean growth rate with only a 5% increase in energy intake Hence, a reduction in growth rate should not be expected to significantly reduce energetic requirements during periods of starvation. Similarly, Ricklefs and White (1981) argue that food limitation is not a significant constraint on growth rate of the Sooty Tern (*Sterna fuscata*), which could be

raised considerably with a small increase in energy intake. However, these authors also argue that the chicks of species with faster growth rates than these very slow growing seabirds spend a larger proportion of their total energy budget on growth. The energy saving from reducing lean tissue growth rates for fast growing species would be greater than those for slow growing species. Such savings might therefore explain variation in growth rates among species with relatively high growth rates, but can not explain the extremely slow growth rates of the Procellariiformes.

In this chapter I take a different. I estimate the amount of fat that can accumulate in a chick that saves energy by growing slowly. From this I estimate the length of time that slowly growing chicks can survive without food. I argue that even a small savings in energy, if accumulated over several days, might nevertheless have a large affect on the chicks ability to survive food shortages.

II. The slow metabolism model

According to this model, chicks with a slow rate of development have a correspondingly slow metabolic rate. During periods of starvation they use fewer calories to stay alive than chicks that have high a metabolic rate.

This model is plausible because metabolic rate scales with body mass in almost identical fashion to growth rate (Ricklefs 1968,1973; Lilja 1983). This suggests that the physiological processes involved in growth and general metabolism are closely related. Metabolism does not simply use up energy that subtracts from energy used for growth: the biochemical and physiological processes that are involved in growth are dependent upon those processes involved in general metabolism. For instance, respiratory metabolism generates the ATP that supplies energy for growth and cell division (Case 1978).

A rapid growth rate may demand endothermic metabolic rates. Growth rate and metabolic rate are closely correlated amongst vertebrate classes; endothermic animals growing faster than exothermic animals. However, the correlation between growth rate and metabolic rate breaks down within classes (taking mass into account, Case 1978). Trevelyan *et al* (1990) found no correlation between nestling period and metabolic rate within birds once they took mass into account. However, they did not use any other index of growth rate other than nestling period and incubation period. In Chapters 2,3 and 4 I used a several indices of growth rate (minimum and mean mass and wing

growth periods and nestling period). Each was slightly differently related to variability in food supply. A similar diversity of relationships might hold between these indices and metabolic rate. Hence, to further test the slow metabolism model in this chapter I compare the metabolic rate of several families of birds with indices of growth rate for mass and wing-length.

III. The cell allocation model

During growth a chick can build only a limited amount of tissue. This must be distributed between particular tissue types in such a way that the overall body organization is efficient for further growth and survival (Lilja 1983). Such trade-offs in development are well documented in birds (Clum 1995). Each body component has a specialized function during development, and the organism must satisfy each of these functions through the allocation of cells and tissue (Ricklefs *et al* 1994).

One type of trade-off in development is between embryonic and functionally mature cells within a given tissue type. Growth rate is limited by the proportion of available resources that can be allocated to growth after other costs have been met. Growth rate may decline with age because the proportion of mature tissue in the nestling as a whole has increased and pre-empted the diversion of resources to continued growth (O'Connor 1977). Hence, growth rate should be inversely related to degree of functional maturity. As birds acquire more mature function, their growth slows (Ricklefs 1979b). Similarly, there may be a trade-off between increases in cell number (hyperplasia) and cell size (hypertrophy) (Clum 1995).

Another type of trade-off is in the allocation of cells to different types of tissue early in development. Overall growth rate is partly determined by the distribution of cells between different tissues (Lilja 1983). For instance, slower overall growth rates in some species of seabird may be a result of a decrease in maximum wing growth rate (Congdon 1990). Different developmental patterns are associated with changes in the pattern of cell allocation between tissue types (Clum *et al* 1995). Tissues with a high initial cell allocation continue to be a proportionally large part of the body until the tissue reaches maturity. Such tissues successfully compete for a greater fraction of nutrients and energy than other tissues and therefore increase in size at the expense of other tissues.

The relative growth rates of different tissues should be affected by natural selection (Ricklefs 1979a). Therefore, the relative growth rate of a tissue should be proportional to its functional priority during development. Tissues with a high functional priority should receive a large supply of nutrients and energy at the expense of other tissues, (O'Connor 1977) and have a large initial allocation of cells. Such a pattern of evolution is most likely to occur when the resources available for tissue growth are limited, either through a low food supply, or through physiological constraints in the distribution of materials from the digestive system to growing tissues (O'Connor 1977). For instance, in nestling Egyptian vultures (*Neophron percnopterus*) the feathers compete with the tarsus for nutrients late in development (Donazar and Ceballos 1989). By contrast, in birds that fledge at an early stage of maturation, early maturation of the flight feathers is achieved by starting flight-feather growth at an early stage and by having a high rate of feather growth (Redfern 1994).

Correlated with relative growth rates are relative sizes of body structures during development. In most species of bird, because the pectoral muscles do not begin to function until close to fledging, their relative size remains small early in development. Wings and pectoral muscles have little utility until fledging, and their delayed development permits available food to be devoted to body components with a higher functional priority (O'Connor 1977, Ricklefs 1979b). In contrast, the heart functions from an early developmental stage and its size relative to overall mass remains approximately constant with age (Ricklefs 1979b).

The mass of the digestive system is relatively large early in the development of fast growing birds (Latimer 1924, Dunn 1975, Ricklefs 1975, O'Connor 1977, Lilja 1981, Lilja 1983, Clum 1991 Clum *et al* 1995). A large mass of digestive tissue makes a large amount of energy and nutrients available for overall growth, including further growth of the digestive system. A relatively high cell allocation to such organs as the liver and intestine during the early stages of development results in delayed development for other components of growth such as the plumage and pectoral muscles (Lilja 1983).

It is my conjecture that there is also a trade-off between lean tissue growth and the growth of body components that insure against starvation, such as adipose tissue and tissues involved in lipogenesis and fat storage. The allocation of cells to functions involved in fat production and accumulation results in a decreased allocation of cells to

tissues involved in lean tissue growth and this in turn results in fat tissue successfully competing with lean tissue for a large proportion of the energy supply. This is plausible because insulin stimulates both the conversion of glucose into fat in fat cells *and* protein synthesis in other cells (as well as suppressing the conversion of glycogen to glucose in the liver and stimulating the conversion of glucose to glycogen). The relative rates of protein and fat synthesis will therefore depend on cell allocation to fat and lean tissue.

Chicks of offshore-foraging seabirds have long been observed to have a peak mass well in excess of adult mass Richdale (1943, 1954). Croxall and Prince (1980) argue that this reflects the extent of fat reserves and hence the degree to which the chicks are insured against variability in food supply. In order to accumulate and maintain such a mass of fat a chick must be physiologically equipped to do so. Increased lipogenesis and increased adipocyte number has been linked with slower lean tissue growth in adult and growing animals (reviewed in Schaffner 1990). Similarly, Hulsman and Smith (1988) argue that slow flight feather development in offshore-foraging seabirds may be the result of a prioritised growth, with low priority tissues being those not essential for survival during food shortages - i.e., the early development of lipogenetic tissues and adipocytes results in a relatively slow lean tissue growth rate.

The chicks and adults of some species increase fat production and decrease lean tissue growth as a proximate response to fasting (reviewed in Schaffner 1990). Tepperman and Tepperman (1965) refer to these effects as "adaptive hyperlipogenesis," where adaptive means facultative (rather than adapted). I conjecture that certain species of birds have *obligate* hyperlipogenesis, hence a pattern of growth involving high fat production and a low lean tissue growth rate irrespective of the proximate food supply. The chicks of species with obligate hyperlipogenesis will be able to survive food shortages and have slow maximum lean tissue growth rates.

If this is true, then the ratio of the maximum rate of lean tissue growth to the maximum rate of overall mass growth should be inversely proportional to variability in food supply.

IV. The nutrient limitation model

An alternative explanation for slow lean growth rates and high lipid deposition has been suggested for offshore-foraging seabirds; parents oversupply their chicks with energy to achieve adequate supplies of some limiting nutrient (Ricklefs 1976, Hamer and Thompson 1997). The ratio of essential nutrients to energy or "food quality" in the diet may be lower than that required by a rapidly growing chick (Fig. 3.5D, Chapter 3, Boersma and Parrish 1998). Growth processes are limited by the rate at which the limiting nutrients are gathered at the tissue where they are required (Ricklefs 1969c, O'Connor 1977). By slowing growth, a chick reduces its requirement for the limiting nutrient(s). The supply of these nutrients will be accompanied by an energy supply that is surplus to the chicks' requirements. This surplus energy supply is deposited as fat (Ricklefs and White 1981, Shea and Ricklefs 1985).

The limiting nutrients may be amino-acids such as cystiene and methionine that are required for feather, and other tissue, development (Shea and Ricklefs 1985). Both of these amino-acids occur in low concentrations in fish (Ricklefs 1983a). Feathers contain about 90% crude protein, which is mostly keratin (Strettenheim 1972). Fault bars are commonly observed in the rectrices of young birds with a variable food supply. When food supply is low there may be an insufficient quantity of nutrients available for the deposition of keratin (Grubb 1989).

The flight feathers of inshore feeders develop much more rapidly relative to growth of body mass than in offshore and pelagic feeders (Hulsman and Smith 1988). The quality of diet may account for this difference. This may be an entirely environmentally induced effect (Fig. 3.5D, Chapter 3, Boersma and Parrish 1998). Another possibility is that the growing chick is adapted to direct the flow of nutrients to tissues having functional priority at each stage of development. The flow of nutrients to tissue such as muscle, organs and body feathers should be met before that of the flight feathers because the former are more important for the initial survival of chicks. This pattern should evolve where some nutrient is in limited supply. Limited availability of nutrient resources for tissue maturation will be reflected by limitation of flight-feather growth to the latter part of the nestling period (Redfern 1994). In offshore-foraging species the rapid development of the wings would have to wait until more of the limiting nutrients could be made available - i.e., when other body

components had developed to the stage where their demand for the limiting nutrient has decreased (Hulsman and Smith 1988).

This model is similar to that of the cell allocation model in that both place a low functional priority on early wing development. According to the cell allocation model, this results in a decrease in cell allocation to lean tissue growth. Accordingly, wing growth should follow a slow but relatively constant pattern of growth through the nestling period. This is because cell allocation involves the allocation of totipotent embryonic cells to particular functions, rather than the allocation of functionally mature cells to new functions - i.e., cell allocation should be fixed relatively early in the development of body components. By contrast, *adaptive* (i.e., facultative) hyperlipogenesis means an adjustment in lean growth and fat deposition rates closer to maturity. However, this is a relatively small effect. Fat deposits induced by intermittent feeding in the chicks of relatively lean species are small relative to the fat deposits in the chicks of species that appear genetically predisposed to accumulate fat; e.g., Procellariiformes and swifts.

By contrast with cell allocation, the distribution of nutrients to tissues should be more easily altered over the course of development; e.g., by increasing the number of capillaries supplying a body component as the function it carries out becomes a priority. Limiting nutrients would become more available to relatively slow growing body components when other body components approach functional maturity - i.e., the wings should display accelerating growth rate in the latter stages of the nestling period.

The cell allocation and the nutrient limitation models are difficult to disentangle. Nutrient availability at growing tissues may be limited by the relative size of the digestive system, which in turn is the result of cell allocation. This would seem to imply that the predictions of the nutrient limitation model will be confounded with the predictions of the cell allocation model. However, I conjecture that there are two distinct ways in which nutrient limitation and cell allocation may be related. First, cell allocation may be increased to the digestive tissue. This will result in (i). an increase in nutrients available to overall growth, (ii). a decrease in cell allocation to selected tissues, such as those of the wings. Hence, cell allocation rather than nutrient supply will be the main constraint on wing growth. Second, cell allocation may be decreased to the digestive system, resulting in the opposite of the effects listed above. Wing growth will now be largely constrained by nutrient supply.

The cell allocation model states that an increase in cell allocation to the adipose system results in a general decrease in allocation to other tissues. The trade-off could conceivably be with the digestive system. However, the digestive system is crucial to gathering a surplus of energy for storage as fat early in chick development. Hence the trade-off should be at least in part, with other lean tissue. If this is correct, then wing growth in chicks that develop a large supply of fat should be constrained more by low cell allocation than by a restricted supply of nutrients.

V. The developmental coupling and parallel selection models

There exists a close correlation between relative incubation and nestling periods amongst bird species (Lack 1968). Lack suggested that this is because the evolution of a change in growth rate of the nestling requires a change in the developmental pattern that begins in embryogenesis. Hence, a change in the growth rate of the nestling makes necessary a parallel change in the growth rate of the embryo in the egg. Similarly, Ricklefs (1969c) argues that the overall rate of development is under genetic control, rather than each section of development being under separate genetic control. I refer to this idea that a change in the growth rate of the nestling requires a similar change in the growth rate of the embryo in the egg as the developmental coupling model.

If this model is correct, then the evolution of slow nestling development may be a by-product of selection pressures on the incubation period. For instance, species that live with a variable food supply will tend to spend long periods foraging for food when the food supply is low. Long foraging trips during incubation require parents to be absent from the egg for prolonged periods (Lack 1968, Case 1978, Boersma 1982). This requires eggs that do not lose water rapidly and are insulated against temperature changes. Two possible adaptations are to decrease shell porosity and increase egg-shell thickness. Both of these changes will decrease the evapotranspiration and thermal conductivity of the egg, resulting in an embryo that is adapted to slow growth in an environment of low egg-shell permeability to oxygen and carbon dioxide (Case 1978). Whittow (1980) has found evidence that the eggs of birds with a relatively long incubation period have the properties that Case predicts; low dehydration rate, low porosity, low water conductance of egg shell, slow embryo growth rate, reduced gas transfer, and correlation with a pelagic habit.

Case (1978), however, questions the developmental coupling model. He points out that mammals show little correlation between prenatal and postnatal growth rates. A major difference between the development of altricial birds and mammals is that whereas eggs and nestlings are exposed to a similar environment, in mammals the embryonic young are contained in the uterus that is a very different environment from that which infants are exposed to. Hence, Case argues that the correlation between incubation and nestling periods is due to parallel selection pressures that have a similar though physiologically independent evolutionary effect on both egg and nestling. I refer to this as the parallel selection model.

Methods

I tested all the models discussed above using interspecific comparisons (predictions summarised in Table 5.1). I used a variety of methods depending on the nature of the data relevant to each model. I have combined the methods for the cell allocation model and the nutrient limitation model because some of the tests are applicable to both of these models.

I. The energy savings model

If the energy savings model is correct then the relatively small savings in energy that slow growth provides will nevertheless produce a large enough deposits of fat to survive long fasts. To test this, I compared four energy budgets over the first half of the nestling period for four hypothetical chicks based on the energy budget of Leach's Storm-Petrel (Ricklefs and Schew 1994).

I first calculated the daily energy budget for a hypothetical growing chick that does not deposit any fat. On each day it uses energy for growth and general metabolism. I assumed that the chick grows according to the logistic equation with an asymptotic mass of 80g and a growth rate (K in Equation 5.1) of 0.5 (this is approximately the growth rate of a moderately fast growing passerine of this mass).[1]

I further assumed that dry lean tissue is 0.33 the mass of wet lean tissue (see Chapter 6), hence I was able to estimate growth increments for dry lean tissue for each day. Each gram of lean tissue growth requires 20kJ of energy (see Chapter 6). I then

[1] I use the abbreviation K for growth rate following the convention for *physiological* growth models rather than r which is used for growth rate in models of *population* growth (and where K is the population asymptote).

calculated amount of energy used on each day for general metabolism (Equation 6.2, Chapter 6), hence I estimated the amount of energy used by the chick on each day. I then calculated similar budgets for chicks with growth rates (K) equal to 0.4, 0.25, and 0.12 (that last is approximately that of a species of Procellariiforme weighing 80g). For each day I subtracted each of the chicks energy requirements from the energy requirements of the fast growing chick ($K = 0.5$) to obtain an estimate of the amount of energy saved by reducing growth rate. I converted this into fat (each gram of fat requires 38kJ of energy, Chapter 6) and added this to the total mass. Finally, I estimated for each of the growth rates and at each stage of the nestling period. the maximum number of days that a chick would be able to survive without food. I did this by assuming that a fasting chick will use energy (supplied by fat deposits) only for general metabolism, and that chicks will survive until they exhaust their fat reserves.

II. The slow metabolism model

I compared family means for metabolic rate (resting metabolic rate in kcal/day) with family means for mean and minimum nestling and growth periods (see Chapters 2 and 3) using 23 families (data for growth and nestling periods in Appendix 1, and data for metabolic rates is from Bennet and Harvey 1987). I chose to compare families because most of the variation in metabolic rates among birds is at the family level (Bennet and Harvey 1987).

Allometric curves for growth rate and for metabolic rate are very similar (Ricklefs 1968,1973; Lilja 1983), hence both these variables were standardised for mass using the methods discussed in Chapter 2.

Individual species data do not represent independent evolutionary events, hence a standard regression of individual species data is susceptible to low statistical power and a high probability of Type 1 error (see Chapter 2 for a discussion). Data from higher nodes in a phylogeny are more phylogenetically independent than individual species. Taxonomic families are reasonable approximations of higher phylogenetic nodes and so should be less susceptible to confound by phylogenetic dependence (Harvey and Pagel 1991).

III. and IV. The cell allocation and nutrient limitation models

These models both predict that wing growth rate will be slower than mass growth rate (hence wing growth period longer than mass growth period) in similar species of birds, e.g., those foraging in an offshore environment. In Chapter 3, I developed an index of the ratio of wing growth rate to mass growth rate; the Growth Ratio Index or *GRI*. I found that *GRI* is correlated with *V*, an index of variability in food supply. Since many of the species of birds with a high *GRI* are offshore-foraging species, this could be explained well by either the nutrient limitation or cell allocation models.

The two models differ in a crucial way. As discussed above, cell allocation should be fixed relatively early in the development of a body component. Hence, if the cell allocation model is correct, then the rate of wing growth should be held below a maximum rate throughout wing growth. By contrast, if the nutrient limitation model is correct, the wing growth rate should be very slow only during the earlier stages of development when the wing tissues are competing with other growing tissues for the limiting nutrient (Hulsman and Smith 1988).

I designed two comparative methods to distinguish between these predictions. The first method requires a large amount of high quality data, and I was able to find sufficient data in the literature for only five species. For these species I calculated an index of the maximum *incremental* wing growth rate to the maximum *incremental* mass growth rate. An increment of growth is the growth that takes place in a 24 hour period. For each increment of growth for an individual in a species, I calculated an estimate of *K*, the growth rate parameter in the logistic (Equation 5.1).

Equation 5.1 $$\frac{dx}{dt} = K.x.(\frac{A-x}{A})$$

where t is time, x is the variable that is growing, and A the asymptotic value.

By rearrangement I obtained;

Equation 5.2 $$K = \frac{dx}{dt} \cdot \frac{A}{x.(A-x)}$$

An estimate of K can therefore be calculated each growth increment. As the asymptotic value is approached, the denominator term in the equation tends toward zero and the estimate of incremental growth rate becomes increasingly unreliable. I therefore only used data below 90% of the growth asymptote. Next, using the asymptotic value, I calculated an increment-specific growth period for each 24 hour stage of growth. According to the nutrient limitation model, species with a variable food supply (e.g., offshore-foraging species) have a limited supply of a nutrient necessary for wing growth and in these species wing growth rate should be low relative to mass growth rate only during the earlier stages of wing growth. By contrast, the cell allocation model predicts that in these species wing growth rate calculated should be low relative to mass growth rate throughout wing growth - i.e., the maximum wing growth rate should be low relative to the maximum mass growth rate in species adapted to a variable food supply.

To test for this effect, for each species where I had a data for individual growth for both wing and mass, I calculated the maximum incremental wing growth period and the maximum incremental wing growth period. Maxima are dependent upon sample size. However, within each species I only used mass growth data for individuals where I also had wing growth data. This insured that the sample sizes within a species for mass and wing growth rate maxima were the same, so that there would be no bias toward either. I used the ratio of the maximum incremental wing growth rate and the maximum incremental wing growth rate to obtain a Maximum Incremental Growth rate Index; *MRI*. According to the cell allocation model, *MRI* should be correlated with V (see Chapter 2). The nutrient-limitation model makes no such prediction.

My second method used less complex data and allowed me to gather a larger sample size for phylogenetically independent contrasts (see Chapter 2 for a discussion of this method). I could use many of the species used in the analyses in Chapters 2 and 3. Hence, I could use the same phylogeny used in those chapters (Appendix 2). According to the nutrient limitation model, species with a variable food supply (e.g., offshore-foraging species) have a limited supply of a nutrient necessary for wing growth and in these species wing growth rate should be faster in the latter part of the wing growth. Ricklefs (1967a) discusses a shape index for growth curves that can quantify this effect; the time taken to grow from 50% of the asymptote to 90% of the

asymptote (t_{90}-t_{50}), divided by the time taken to growth from 10% of the asymptote to 50% (t_{50}-t_{10}). A simpler index is U_i; the degree of maturity (fraction of the asymptotic value for a body component) at which the point of inflection of the growth curve occurs. When the rate of growth is relatively fast in the second half of development U_i should be relatively large, and vice versa (Ricklefs 1967a). I calculated both Ricklefs index and U_i for wing growth, which I call the Wing Growth Index (*WGI*) and Wing-U_i respectively.

The nutrient limitation model predicts an increase in wing growth rate in the latter stages of development (Hulsman and Smith 1988). Therefore, a relatively high value of Wing-U_i for wing growth should be correlated with nutrient limitation, hence with variability in food supply (*V*). I calculated *WGI* and Wing-U_i for 30 species in 22 genera using the Janoschek equation (Equations 2.1 and 2.1 , Chapter 2, Gille *et al* 1995) to wing growth data.

V. The developmental coupling model

If incubation period and nestling period are correlated because of developmental coupling then following a change in the environment they should evolve to the same extent. By contrast, if they are correlated because they are affected by similar selective pressures then it is unlikely that these selective pressures will be equally strong for the incubation and the nestling period, therefore following an environmental change, one period (nestling or incubation) should evolve to a greater extent than the other.

To test the developmental coupling and the parallel selection models, I used egg and nestling predation as an example of a shared selection pressure on incubation and nestling periods. Eggs and nestlings share very similar exposure to predation. In both cases, an acceleration of development to leave the vulnerable environment (the nest) is likely to evolve (see Chapter 4). On the other hand, subtle differences exist. Eggs do not have to be provisioned, and so are less likely than nestlings to be left unattended. Eggs produce no sounds that may attract predators, unlike nestlings. Hence, there should be stronger selection pressure from nest predation on the nestling period than on the incubation period. If the parallel selection model is correct, then following a change in the degree of nest protection nestling period should evolve

(longer or shorter depending on the type of change in degree of nest protection) to a greater extent than incubation period.

I used the scheme discussed in Chapter 4 to designate 54 species in 38 genera as either protected or unprotected nesters. For each species I then calculated relative nestling period and relative incubation period. I used relative nestling and incubation period because absolute nestling and incubation period scale in very similar fashion with adult mass (see Chapter 2). I used the same species for which phylogenetic contrasts were calculated in Chapter 2 (Appendix 1). Hence, using the same phylogeny, I calculated phylogenetically independent contrasts for both relative incubation and nestling period. In this way any problems with phylogenetic dependence were dealt with.

If the effect of nest predation falls more severely on nestlings than eggs, and if incubation and nestling period are not completely developmentally coupled, then unprotected nesters should have a shorter nestling period relative to incubation period than protected nesters. I tested for this by calculating linear regressions for phylogenetically independent contrasts for relative nestling period on phylogenetically independent contrasts for relative incubation period. I did this separately for protected and unprotected nesters. If the parallel selection model is correct, then the regression of contrasts for relative nestling period on contrasts for relative incubation period should have a higher y-intercept and/or steeper gradient than for protected nesters than for protected nesters

Model	Prediction
Energy savings	Reduced lean tissue growth rates save sufficient energy to convert into fat and insure against starvation during food shortages.
Slow metabolism	Metabolic rate is correlated with growth and nestling periods.
Cell allocation	The ratio of mass to wing (i.e., lean tissue) growth rates is high throughout development (in species adapted to a variable food supply), hence maximum mass growth rate is high relative to maximum wing growth rate.
Nutrient limitation	Wing growth rate (relative to age) increases relative to mass growth rate as the chick approaches fledging (in species where the supply of some nutrient essential for lean tissue growth is low - e.g., offshore-foraging seabirds). Therefore, the point of inflection for wing growth curves in these species will occur at a late stage in wing growth (i.e., *WGI* and Wing-*U*i should be correlated with *V*).
Developmental coupling	Nestling period relative to incubation period is not correlated with degree of nest protection.

Table 5.1 Summary of model predictions.

Results

The results follow the format in the introduction and methods - i.e., they are divided into five sections corresponding to the models.

I. The energy savings model

Using equations from the energy budgets used Ricklefs and Schew (1994) I found that a relatively small energy savings results from a reduced growth rate, but that this may nevertheless have a significant affect upon the accumulation of fat (Fig. 5.1). Each of the hypothetical chicks in Fig. 5.1 receives the same amount of energy at each day as a chick growing with a growth rate (*K*) of 0.5 and convert any surplus to their requirements into fat. The chick with the slowest growth rate accumulated a very large amount of fat in the first 13 days of growth. At all growth rates, reserves peaked at an age of 8-14 days and then declined. This was because I assumed that large fat deposits themselves have a maintenance cost, hence the older chicks with large fat deposits have a higher mass and therefore higher metabolic costs than faster growing, hence leaner chicks. I had assumed that the maintenance cost for fat was the same as for lean tissue (Equation 6.2, Chapter 6). In fact, fat has a lower maintenance cost than lean

tissue, hence these results are conservative, fat reserves may in fact be maintained at a higher level for longer in the nestling period than is shown in Fig. 5.1. Even so, the peak fat reserves in the slowest growing chick are still sufficient for the chick to survive for over 10 days.

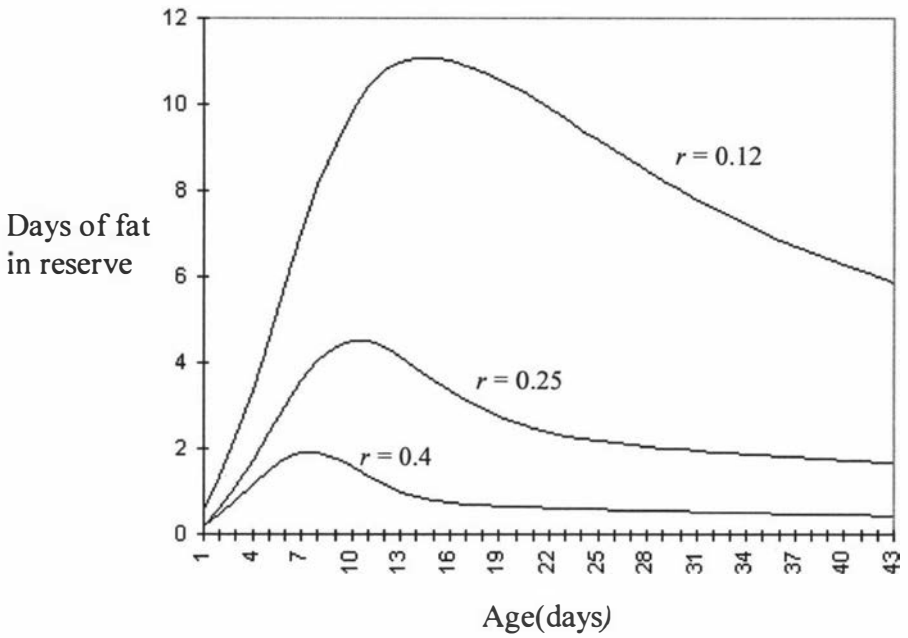

Fig. 5.1 Fat reserves accumulated by chicks of different growth rates over the nestling period. Energy supply for all chicks is equivalent to that required for growth and metabolism in a chick with lean tissue growth only and a growth rate (K) of 0.5.

II. The slow metabolism model

I did not find a definite negative correlation between metabolic rate and growth and nestling periods (standardised for mass) amongst families of birds. Although relative growth and nestling periods show a similar regression line when compared with relative metabolic rate, only nestling period is statistically significant (Fig. 5.2). However, Frigate-birds have extremely low metabolic rates and growth rates and when they are removed from the analysis the regression of relative nestling period on relative metabolic rate is no long statistically significant.

Each growth index measures a different aspect of growth, hence we can infer different things from them. Mass growth period includes the effects of fat deposition whereas wing growth rate does not. Nestling period will include inputs from aspects of lean growth other than wing growth. Growth may be controlled differently during ideal conditions than during poor conditions. Therefore, minimum growth periods and mean

growth periods do not reflect identical developmental processes. Moreover, since there may be a trade-off between different components of growth, no one of these indices may adequately reflect overall constraints on growth rate.

I conjectured that, although the different aspects of growth may be partially independent and in partially in conflict, all must be subject to some overall constraint on the accumulation of tissue. If this is correct, then some single factor should account for much of the variation in the mass growth rate, wing growth rate, and nestling period. If the slow metabolism model is correct, then metabolic rate should be strongly correlated with this factor.

To test for this, I used principal component analysis (*PCA*). Principal component analysis transforms a number of correlated variables into a (smaller) number of uncorrelated variables (principal components). I found that the first principle component explained 81% of the variation amongst families in a *PCA* analysis of relative nestling period (*RNP*), relative mass growth-period (*RMGP*), and relative wing growth-period (*RWGP*). However, I found that relative metabolic rate (*RMR*) was not significantly correlated with this factor ($r^2 = 0.13$, $P = 0.19$).

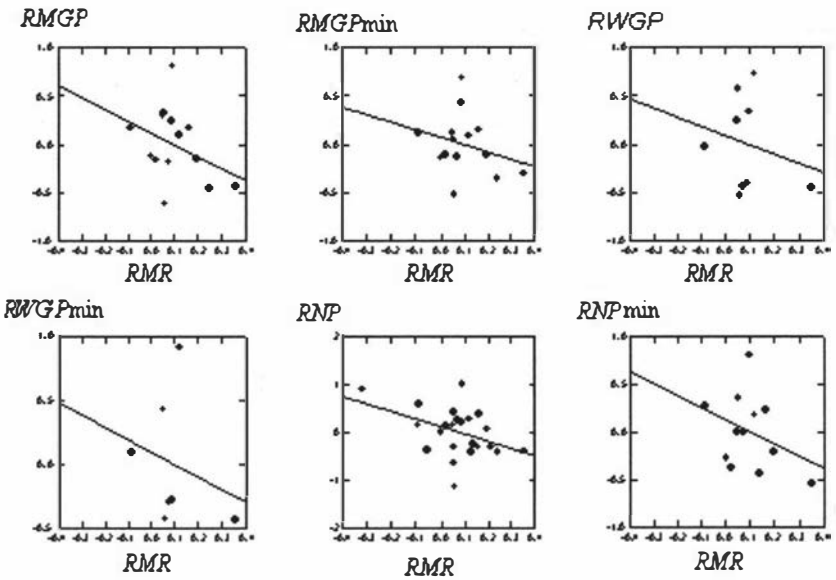

Fig 5.2 The relationship between metabolic rate and nestling and growth periods in families of birds. All data points are the family means and are relative to mass - i.e., residuals from allometric curves. *RMR*; relative metabolic rate, *RMGP*; relative mass growth period $RMGP_{min}$; relative minimum mass growth period, *RWGP*; relative wing growth period, $RWGP_{min}$; relative minimum wing growth period *RNP*; mean nestling period; RNP_{min}; minimum nestling period. For a full explanation of indices see Tables 2.2 and 3.2 Chapters 2 and 3. Only *RNP* is statistically significant ($P = 0.02$).

III. and IV. The cell allocation and the nutrient limitation models

For the five species for which the maximum incremental growth ratio index (*MRI*) could be calculated *MRI* is strongly correlated with variability in food supply (*V*) (Fig. 5.3). This is consistent with the cell allocation model but not the nutrient limitation model. These species were mostly in different families so phylogenetic dependence should be reduced (Redfern 1994). However, a low sample size together with the problems of estimating maxima discussed above, makes this result inconclusive.

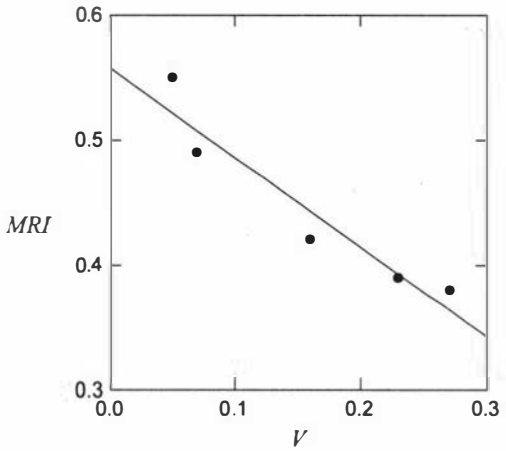

Fig. 5.3 Relationship between variability in food supply (V) and the Maximum Incremental Growth Rate Index (*MRI*). *MRI* is the ratio of the maximum incremental wing growth rate to the maximum incremental mass growth rate. $r^2 = 0.91$, $P = 0.006$.

Wing Growth Index (*WGI*) was strongly correlated with Wing-U_i (Fig. 5.4), confirming Ricklefs (1967a) conjecture. *WGI* and Wing-U_i both measure a change in wing growth rate over the course of development.

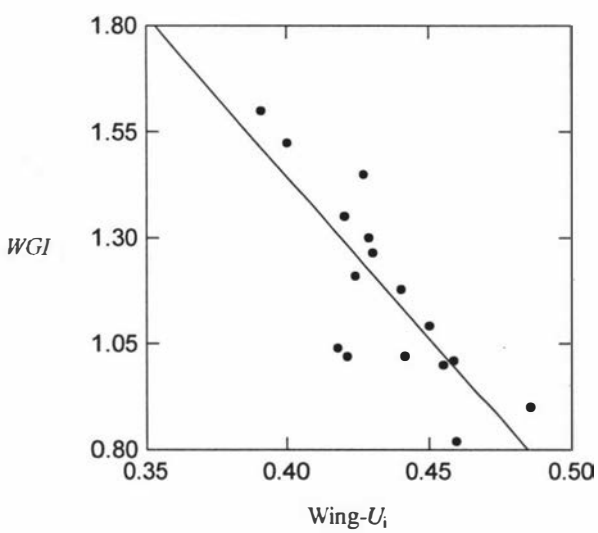

Fig. 5.4 The relationship between two ways of measuring changes in wing growth rate over development. *WGI*; $\frac{t_{90} - t_{50}}{t_{50} - t_{10}}$ for wing growth, Wing-U_i; the degree of maturity of the wings at which the point of inflection of the growth curve occurs. $r^2 = 0.65$, $P < 0.0001$.

For the 30 species for which I measured Wing-U_i there was no significant relationship between Wing-U_i and variability in food supply (V) (Fig. 5.5). This is inconsistent with the nutrient limitation model which predicts that birds with a variable food supply should have an increasing wing growth rate close to fledging, hence Wing-U_i should be greater than 0.5. Also, when I compared 6 inshore-foraging species with 15 offshore-foraging seabirds (Fig. 5.6) there was no significant difference between mean Wing-U_i and the distributions were very similar (1-tailed 2-sample t-test P = 0.36). The similarity of patterns of wing growth in different habitats argues against the nutrient limitation model.

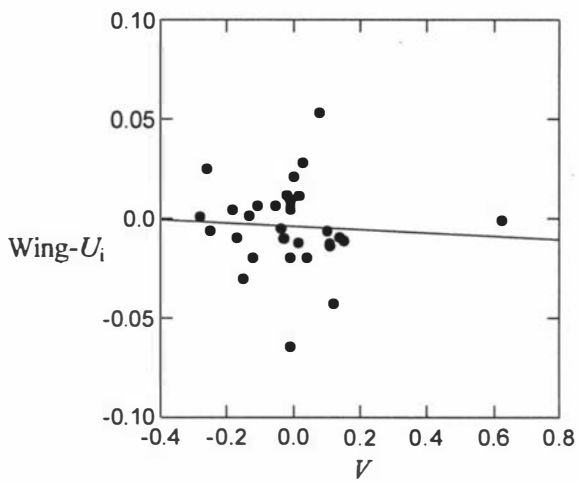

Fig. 5.5 Relationship among species between variability in food supply (V) and the degree of maturity at which wing growth is at the point of inflection (Wing-U_i). The actual values shown are phylogenetically independent contrasts. These were obtained for each variable from the values calculated for 30 species from Appendix 2. Values of Wing-U_i below 0.5 mean that wing growth is relatively fast early in development, and vice versa.

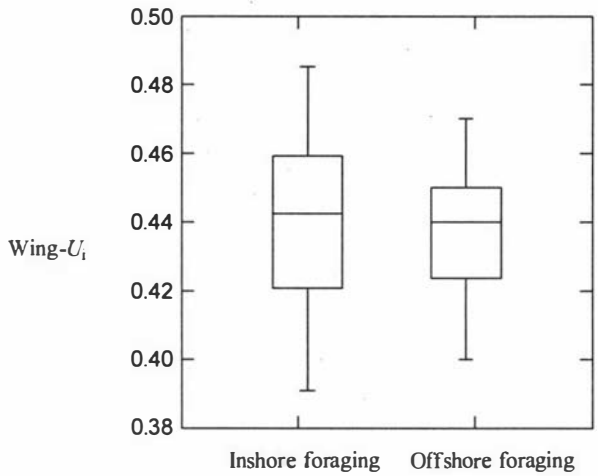

Fig. 5.6 Degree of maturity at which wing growth is at the point of inflection (Wing-U_i) in inshore and offshore-foraging seabirds. Horizonal lines are median values; boxes are 1st and 3rd quartiles; vertical bars are 95 percentiles.

V. The developmental coupling model

Nestling period appears to have an evolutionary response to changes in the degree of nest protection that is partially independent of the evolution of incubation period Birds have tended to evolve longer nestling periods relative to incubation period in protected than in unprotected nests(Fig. 5.7). The difference between the slopes of the linear regression of *RNP* on *RIP* for the two groups was 0.39. I tested the difference between the two slopes using a permutation test. I allocated at random the coordinate pairs (of *RIP* and *RNP*) into a set of 39 open nesters and 14 protected nesters. When I calculated regression lines for the two sets, I found a difference between the gradients equal to or greater than 0.39 in only 2% of permutations. I then used *RIP* and *RNP* as independent variables and nest-protection type as the response variable in a multiple regression analysis Only relative nestling period was a significant predictor of nest protection (Table 5.2) This suggests that selection pressure is different for nestling and incubation period. It also suggests that any developmental coupling between nestling period and incubation period is not completely fixed, but is itself under evolutionary control.

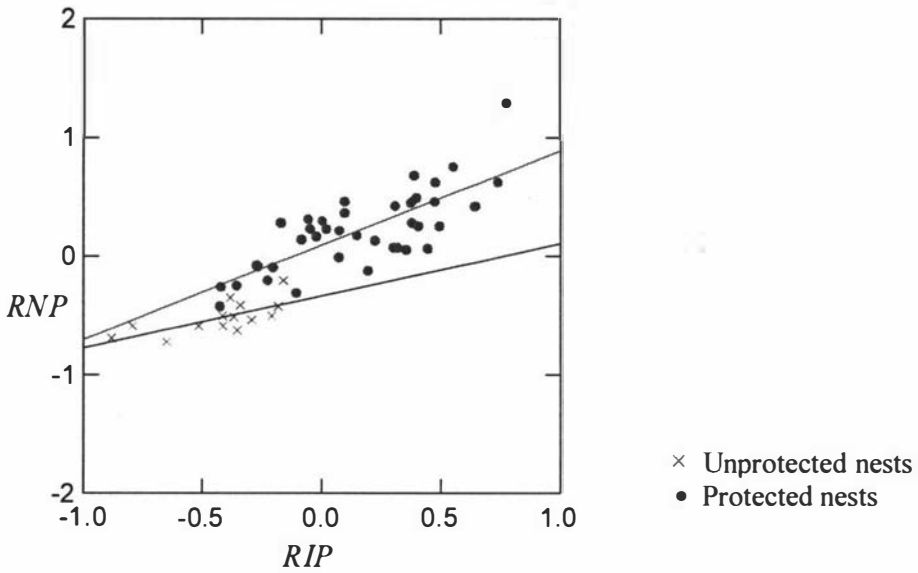

Fig. 5.7 Relationship among species between relative incubation period (*RIP*) and relative nestling period (*RNP*) in protected and unprotected nests. The actual values shown are phylogenetically independent contrasts. These were obtained for each variable from the values calculated for 52 species from Appendix 2.

	r^2	*RIP*	*RNP*
PD	0.75***	-0.06	-0.70***

Table 5.2 Effect of relative incubation period (*RIP*) and relative nestling period (*RNP*) on degree of nest protection (*PD*), tested using multiple regression analysis on phylogenetic contrasts. r^2 given for the proportion of variation explained by all three independent variables. *RIP* and *RNP* show standardised partial regression coefficients. Statistical significance in the overall model, and for the partial coefficients, is indicated by asterisks: * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

Discussion

Slow growth saves a small amount of energy, suggesting that, at least among very slow growing birds, growth rate is not adjusted by evolution to match the average energy supply (Ricklefs *et al* 1980, 1985, Ricklefs and White 1981). My analysis suggests that small savings in energy can nevertheless have a significant affect on the

accumulation of fat. This suggests that if evolution adjusts growth rates to the variability rather than the mean level of food (i.e., energy) supply, then the small energy saving that result from small growth may be adaptive. However, if a small surplus in energy can have a significant affect on fat reserves, then it isn't clear why couldn't this be accomplished by small increases in provisioning rather than by slowing growth rates. One possibility is that the limiting factor in the supply of energy to the chick during periods of good food supply is not provisioning, but physiological limits on the assimilation and utilization of energy. If for example, the amount of energy available for physiological processes is limited not by provisioning but by the size of the digestive organs, then the only way of saving energy may be to reduce growth rates. This complicates further analysis because the growth of digestive tissue is itself part of lean tissue growth - i.e., reducing lean tissue growth rates may reduce the size of the digestive tissue, hence the supply of assimilated energy. I examine this possibility in an appendix to the computer models in Chapter 6 (Appendix 7).

The lack of a strong correlation between metabolic rate and growth rate agrees with earlier work. Trevelyan *et al* (1990) found that nestling period and incubation period were not correlated with metabolic rate among birds once the effect of mass and phylogenetic dependence had been removed. Contrary to the slow metabolism explanation, marine birds tend to have a high resting metabolic rate (Bennett and Harvey 1987). Seabirds that feed by "plunge-diving" tend to have high metabolic rates compared to other seabirds (Ellis 1984), yet this mode of foraging is vulnerable to bad weather (Dunn 1975). Bryant and Furness (1995) found no consistent and clear relationships between avian metabolic rate and habitat type.

A flexible rather than just a constantly low metabolic rate may increase chick survival during food shortages (Ricklefs 1976). More work needs to be done on this, and a possible relationship with growth rates.

Removing mass from consideration may be problematic, given that mass itself may be an adaptation to a variable food supply. However, many of the birds with long growth periods considered here are small (e.g. Storm-Petrels) and the relationship between variability in food supply and development is independent of mass.

A full explanation for the relationship between metabolic rate and growth rate may come from a better understanding of the physiological basis of scaling relationships. West *et al* (1997) have presented a promising approach that seeks to

explain physiological scaling properties by the fractal geometry of the circulatory system.

There are a number of variations on the nutrient limitation model (e.g., Ricklefs 1976, Hulsman and Smith 1988, Hamer and Thompson 1997, Boersma and Parrish). The interpretation that I have discussed is not supported by my data. The cell allocation model was not falsified though, and may explain some of the data presented in this and earlier chapters.

There is indirect confirmation of a physiological trade-off between fat production and lean tissue growth from experiments on the proximate effect of intermittent feeding on growth. A sizable literature exists for such experiments carried out on poultry, laboratory animals, and humans (reviewed Schaffner 1990). Induced intermittent feeding has been linked to increased lipogenesis, increased adipocyte number, and slower lean tissue growth in adult and growing animals (Cohn 1963, Schaffner 1990). Intermittent eating increases fatty acid synthesis and lipogenic enzymes (Nir *et al* 1979). Pinchasov and Nir (1985) report that in intermittently fed poultry chicks, there is a gradual increase in lipid synthesis on days when the chicks are fed, and with increased lipolysis on the days that the chicks are not fed. In this experiment, tissue deposition during the days when the chicks were fed, consisted mainly of protein before intermittent feeding, and shifted gradually to mainly consisting of fat, following the commencement of intermittent feeding. Fat is better utilized than protein for maintenance purposes. Hence, intermittently fed chicks have improved energy utilization from endogenous energy stores on days when the chicks are not fed.

These experiments demonstrate a facultative response to intermittent feeding, which has been referred to as "adaptive hyperlipogenesis" (Tepperman and Tepperman 1965). Williams (1966) has argued that complex facultative responses are evolutionarily derived from obligate adaptations. It takes more information to specify a facultative response than a fixed response. If this is correct, then adaptive hyperlipogenesis is evolutionarily derived from obligate hyperlipogenesis

Further indirect evidence for a trade-off between fat production and lean tissue growth comes from studies of the avian liver. Birds with a fast rate of lean tissue growth have a relatively large liver (Lilja 1983). The avian liver is also the main lipogenic site in avian species, and increases in liver mass can arise from increased

lipogenesis (Pinchasov and Nir 1985). The lipogenetic functions, and the lean tissue growth functions of the liver involve specialized cells, Hence, cell allocation to lipogenetic functions in the liver may require a reduction in the amount of tissue in the liver involved in lean tissue growth (Schaffner 1990).

The cell allocation model may help to explain another feature of some slow growing nesting birds. Some species of nesting birds have a very similar developmental pattern to precocial birds. Ricklefs *et al* (1985) argues that the precocial pattern of development explains the slow growth rates of storm-petrels. Precocial birds generally grow more slowly than altricial birds (Ricklefs 1973). Costs in energy from activity, e.g., locomotion, are higher for precocial chicks than for altricial chicks (Ricklefs 1968). Also, cell allocation to the early development of functioning leg tissue may reduce the development of other body components, resulting in an overall decrease in growth rate (Ricklefs 1979b). Relative to their size, petrels grow at a similar rate to the Galliformes (fowl) of and are similarly capable of mature function (such as thermogenesis) at hatching.

Case (1978) however, argues that precociality is not an adequate explanation of avian growth rates, or for the growth rates of vertebrates in general. He lists numerous counterexamples. For example, Diomedeidae (albatrosses) and Fregatidae (frigatebirds) are provisioned by their parents and are altricial, but grow relatively slower than parentally fed precocial or semi-precocial species in the families Laridae (gulls), and Alcidae (auks) Scolopacidae (sandpipers, knots). The altricial Diomedeidae and Fregatidae grow more slowly than most species in the precocial families Charadriinae (plovers) and Anatidae (waterfowl) whose chicks are self-feeding. Furthermore, in mammals, there is no correlation of growth rate with the level of maturity at birth. The highly precocial young of Artiodactyls (even-toed ungulates) exhibit rapid growth. Ursidae (bears) produce young that are less developed at birth yet their growth rates are slower. The growth rates of Marsupials born in a foetal condition are also relatively slow.

Precocial birds are those in which the chicks are relatively independent and have well-developed powers of locomotion early in development. But generally, species may be "precocial" for any function that matures early relative to other functions. Hence, slow growing precocial species may be a special instance of a more general. The early maturation of body components that do not supply the body with

resources for further growth compromise overall growth rate. In this sense, obligate hyperlipogenesis is the precocial development of body components that decrease the likelihood of chick starvation during food shortages.

My model of obligate hyperlipogenesis (i.e., the cell allocation model) involves a trade-off in the allocation cells to lean tissue and to fat development. Cell allocation is likely to be fixed early in the development of a body component. Hence, growth rate later in development should be constrained by the same factors that constrain growth rate early in development. This seems to imply the developmental coupling of incubation and nestling period. However, incubation period and nestling period seem to undergo at least partly independent evolutionary changes in response to changes in degree of nest protection. Future work on the evolution of avian development should work toward a resolution of this apparent contradiction. One possibility is suggested by Williams (1992), who notes that a common assumption is that development is a process that can be represented by branching causation. In such a model, whatever happens early in development must have a strictly correlated effect in later development. This model of development is falsified by organisms with indirect maturation such as crustacea and metamorphosing insects. Williams argues that development is the timed growth of modules of tissue. The maximum growth rate of a body components or "module" may be limited by its initial allocation of cells early in development. However, this does not mean that all modules of tissue compete for nutrients as soon as they are assembled, e.g., in metamorphosing insects, the cells that become the butterfly do not begin to divide and differentiate until pupation. I conjecture that different tissues have different priorities in the prenatal and postnatal stages and that this is reflected in both the initial allocation of cells to tissues (late priority tissues having a smaller allocation of cells) and that the share of nutrients that a tissue acquires as its growth rate quickens relative to the rest of the body is in turn a reflection of initial cell allocation. The growth of different tissues have different affects on overall growth rate - i.e., if development is modular then the changing priorities of chicks through development should be reflected by a changing overall growth rate. Hence, the evolution of the overall growth rate of one stage of development need not be strictly (and positively) correlated with that of other stages of development.

In this chapter I have examined a number of models that may either explain or replace Lack's insurance hypothesis. I the next chapter I develop the energy savings model further using mathematical models.

Chapter 6

Mathematical models of growth in environments with a variable food supply

Introduction

Lack conjectured that a slow growth rate saves energy and that stored energy helps survival during food shortages. In Chapter 5 I named this the "energy savings model" and reviewed the argument that the low cost of growth means that reducing growth rate will save so little energy as to be insignificant. However, I concluded that even small daily savings in energy may have a significant effect on a chicks ability to survive during food shortages. To do this I used equations derived from the energy budgets of Leach's Storm-Petrel chicks. In this chapter I use these equations in computer simulations to model growth under various assumptions about how energy is used by growing chicks. Each model is a refinement of the energy savings model.

My simulations are similar to those of Henstridge and Tweedy (1984) and Ricklefs and Schew (1994). Parents deliver a variable amount of energy to the chick each day, some of which is used in general metabolism and some of which is allocated to lean and/or adipose tissue. Shortfalls in the energy intake required for metabolism (maintenance costs) are met by the conversion of fat into energy. To more realistically model the affect of slow growth on mortality I included in the simulations a daily probability of death by predation. Chicks with slow growth rates may be better able to avoid starvation, but since they take longer to fledge then they should also have an increased probability of death by predation.

I simulated growth under both ideal and variable energy supplies. I could then see how varying the growth rates under differing model assumptions affected survival in a variable food supply. Hence, I was able to assess more accurately whether - under various assumptions - slow growth rates help chicks to avoid starvation during food shortages.

The secondary purpose of this work was to check the predictions that I used to test the growth models in Chapter 3 (Fig 3.6). Though I made predictions from these models, this involved some jumps in logic (e.g., the assumption that the ratios between *incremental* mean and maximum lean tissue and mass growth rates shown in the

graphs recur when growth rates are measured over the longer term). Therefore, by turning these graphical models into computer simulations I was able to check my logic more thoroughly.

To describe the models I will (i) give a verbal description of each model, (ii) give a mathematically detailed description of the model "skeleton" that is common to all the models, (iii) give mathematically detailed descriptions of the ways in which the various models deviate from this basic pattern

The growth models are of three main types (summarised in Table 6.1). The first type are the "lean-priority" (or "lean tissue prioritisation") models. I have borrowed the term "priority" or "prioritisation" from Hulsman and Smith (1988) and Congdon (1990). In prioritisation models, energy from food is allocated preferentially to the growth of high priority tissues. This is partially independent of the initial allocation of cells to a tissue, (i.e., cell allocation), with which prioritisation may be confused. In the lean-priority models, fat deposition only occurs if there is more than enough energy to meet the requirements for maximum lean tissue growth. Therefore the lean tissue growth rate is equal to the mass growth rate up to a maximum value. Additional mass gain is due to fat deposition.

The second type is the "uniform distribution" model in which energy is distributed according to a fixed ratio between lean growth and fat deposition.(Fig. 3.6D, Chapter 3). The third type are the "fat-priority" models (Fig. 3.6E, Chapter 3) under which lean tissue growth only occurs if energy intake is more than enough for metabolism and the maximum fat deposition rate. I have put details of two more models that are variations on the ones described in this chapter in Appendix 7. These models, though pertinent to some tangential issues arising in other chapters (specifically, issues discussed in Appendix 3 and in Chapter 8) are not directly relevant to testing the growth models described in Chapter 3 and the models discussed in Chapter 5.

Type of model	Modifications on the basic model types	Description
Lean-priority	Fixed maximum fat deposition rate (Fixed Ak)	Reduced lean tissue growth creates a savings in energy which is converted into fat.
	Variable maximum fat deposition rate (Variable Ak)	Reduced maximum lean tissue growth rates allows more rapid maximum fat deposition rates.
	Supply-tissue model (Appendix 7)	Reduced lean tissue growth rate is by reduction in digestive tissue growth rate.
Uniform distribution		Energy is distributed according to a fixed ratio between lean growth and fat deposition.
Fat-priority	Fat-priority	Fat deposition takes place prior to lean growth
	Facultative fat-priority (Appendix 7)	The prioritisation of fat deposition depends upon chick condition.

Table 6.1 Summary of the growth models

Lean-priority model

This model builds upon the model described in Fig. 3.6B (Chapter 3) which was initially derived from Boersma and Parrish (1998). Under this model, species that are adapted to live in a variable environment have a reduced maximum lean tissue growth rate. This saves energy which is converted into fat. This model approximates Lack's (1968) thinking on how slow growth could help adapt a species to life with a variable food supply.

In the simulations, I ran two different versions of this model. In the first version I assumed that the maximum fat deposition rate is not affected by the maximum lean tissue growth rate. Therefore, the maximum fat deposition rates were the same for species with different degrees of variability in food supply. In the second version I assumed that decreasing the maximum lean tissue growth rate allows a corresponding increase in maximum fat deposition rate - i.e., I assumed that the physiological constraints on lean tissue growth are in a trade-off with those for fat deposition.

Uniform distribution model

In this model, lean tissue growth and fat deposition compete for a share of the energy assimilated by the chick. In the lean-priority model, fat deposition only occurs if there is more than enough energy for maximum lean tissue growth rate. The early accumulation of a fat store is made possible by a reduction in maximum lean tissue growth rate. In the uniform distribution model, energy intake is divided into two parts according to a fixed ratio and distributed to lean tissue growth and fat deposition (Fig. 3.6D, Chapter 3).

Fat-priority model

Hulsman and Smith (1988) suggest that in some species, fat deposition takes precedence over lean tissue growth, especially wing growth Therefore lean tissue growth only occurs if there is more than enough energy to meet the requirements for fat deposition (Fig. 3.6E, Chapter 3). Brood swapping experiments by Congdon (1990) suggest that the chicks of the off-shore foraging Black Noddy (*Anous minutus*) grow in this way. However, Congdon's discussion, by suggesting that wing growth rate in the Black Noddy is kept below some maximum (an assumption of a lean-priority model), does not distinguish between the fat-priority and the lean-priority model. However, if my analysis is correct then the results of Congdon's brood swapping experiment only support the fat-priority model

Methods

In this section I describe the mathematical details of the models. Rather than give a complete description of each model, I will describe in detail only the lean-priority model. The other models are modifications on this model, hence I will limit my description of them to the modifications.

Fig. 6.1 shows the main steps in the three models in the form of a flow-chart. Some steps have been abbreviated in the diagram, and details of each step are given in computer code in Appendix 8. Definitions of the all the parameters used in the models are given in Table 6.2.

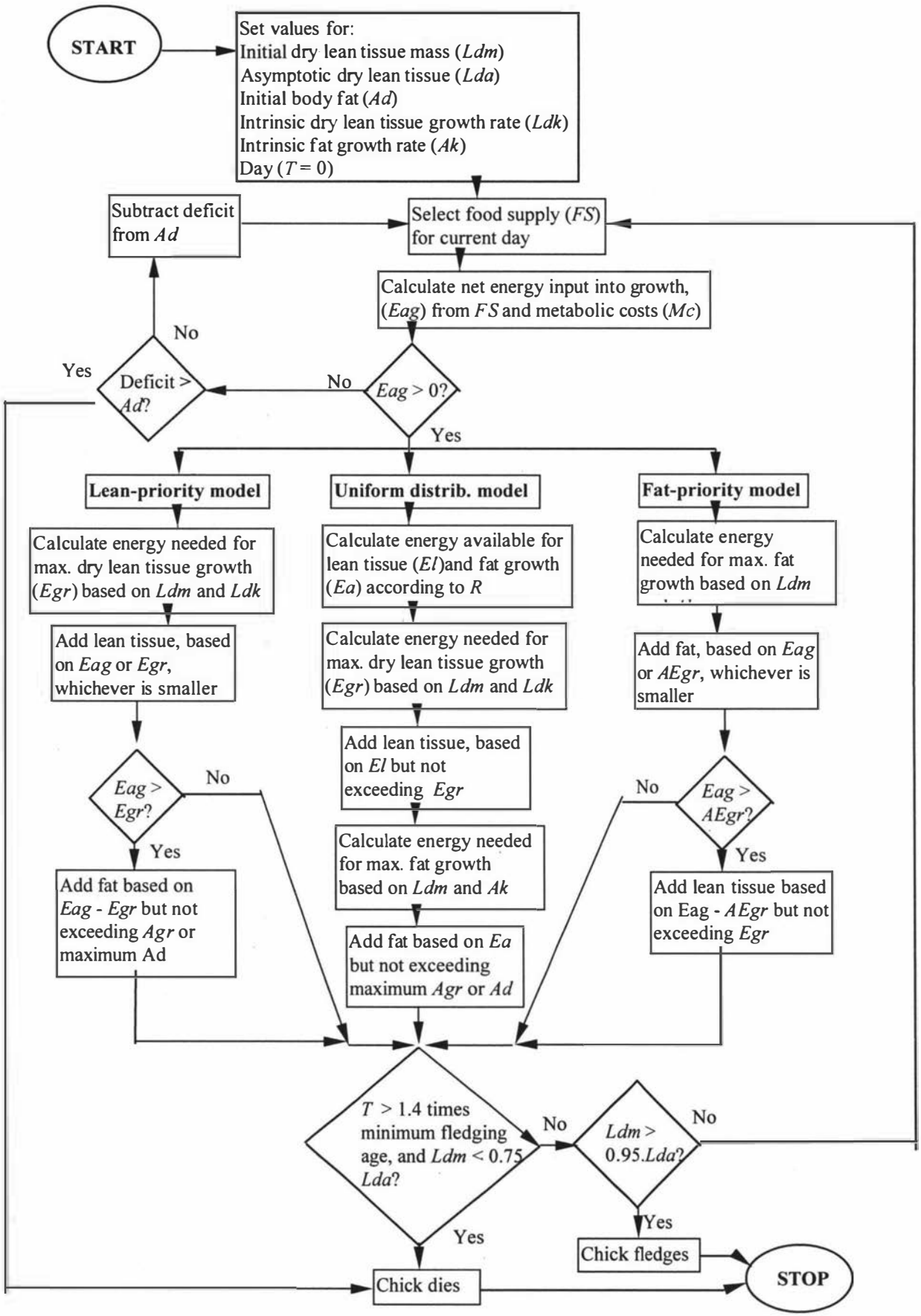

Fig. 6.1 Flowchart of the growth models

Aea	(Adipose energy available) The amount of energy available for fat deposition on a given day (kJ)
Ad	(Adipose) body fat (g)
Adi	(Adipose deposit increment) The actual amount of fat deposited on a given day (g)
AEgr	(Adipose Energy growth rate) The maximum amount of energy that can be deposited as fat on a given day (kJ)
Agr	(Adipose growth rate) Maximum fat deposition increment on a given day (g)
Ak	(Adipose intrinsic growth rate) Maximum fat deposition rate
Ea	(Energy for adipose) The portion of *Eag* available for fat growth on a given day (kJ)
Eag	(Energy available for growth) The portion of energy available for growth on a given day (kJ)
Ef	(Energy in fat) The amount of energy used in fat deposition on a given day (kJ)
Egr	(Energy growth rate) Energy content of maximum dry lean tissue growth increment on a given day (kJ)
El	(Energy for lean tissue) The portion of *Eag* available for lean tissue growth on a given day (kJ)
Eld	(Energy in lean tissue deposited) Energy content of actual dry lean tissue growth increment on a given day (kJ)
FS	(Food supply) Energetic content of food supplied on a given day (kJ)
Lda	Dry lean tissue asymptotic mass (g)
Ldi	(Dry lean tissue increment) Actual dry lean tissue growth increment on a given day (g)
Ldk	Dry lean tissue intrinsic growth rate.
Ldm	Dry lean tissue mass (g)
Ldo	(Dry lean tissue mass optimum) Dry lean tissue mass for a chick growing under ideal conditions on day T (g)
LWGR	Wet lean tissue growth rate calculated over the nestling period from simulated growth curves
Lgr	(Lean tissue growth rate) Maximum dry lean tissue growth increment on a given day (g)
Lwm	Wet lean tissue mass (g)
M	Total mass
Mc	(Metabolic costs) Energetic costs of metabolism on a given day (kJ/day)
MGR	(total) Mass growth rate calculated over the nestling period from simulated growth curves
o	A constant of integration defining the position of the logistic curve relative to the origin.
R	(Distribution ratio) The fraction of energy available for growth on a given day that is directed to lean tissue growth in uniform distribution model.
T	(Time) day of simulation.

Table 6.2 Summary of parameters used in the growth models.

The lean-priority model

The mathematical simulation consists of a sequence of "days" (rounds) of feeding and growth. The simulation begins with starting values for dry lean tissue mass (*Ldm*), and body fat (*Ad*). At the beginning of each day, the amount of energy supplied to the nest in a day in kilojoules (*FS*) is calculated (discussed in detail in Appendix 6). Next, the maximum increment in dry lean tissue for the present day (*Lgr*) is calculated from a logistic growth equation:

Equation 6.1 $$Lgr = Ldm.Ldk.(1-\frac{Ldm}{Lda})$$

where *Ldm* is the current dry lean mass, *Ldk*, is the maximum lean tissue growth rate for the species, and *Lda* is the asymptotic lean mass.

Lgr is a mass and must be converted into an equivalent quantity of energy. Ricklefs and Schew (1994) give a conversion factor for energy to dry lean tissue of 20 kJ/g. Hence, multiplying *Lgr* by 20 gives the maximum amount of energy that a chick can convert into lean growth in the day, *Egr*.

Metabolic costs are then calculated on the basis of mass (total wet mass, *M*). For simplicity, I have ignored the lower metabolic costs for maintaining fat as compared to lean tissue. I have been unable to find any consistent estimates of the relationship between dry lean mass (*Ldm*) and wet lean mass (*Lwm*). The relationship varies between species and within species for chicks of different ages. However, examination of water indices in lean tissue in Bryant and Gardner (1979), Bryant and Hails (1983), and Ricklefs and Schew (1994), shows that an estimate for *Lwm* of three times *Ldm* will be approximately correct, and hence is the conversion factor used here.

Mass (*M*) is then equal to wet lean mass (*Lwm*) plus body fat (*Ad*). To calculate metabolic costs I use the allometric equation relating metabolic costs (in kJ/day) to mass (*M*), given by Ricklefs and Schew (1994);

Equation 6.2 $$Mc = (6.1.M^{-0.39}).M$$

The total amount of energy available for growth (*Eag*) is then energy from food (*FS*) minus metabolic costs (*Mc*). If *Eag* is greater than zero then the amount of energy used in lean growth (*Eld*) is calculated; *Eld* is equal to either *Eag* or *Egr* (the maximum amount of energy that can be converted to lean tissue) whichever is the smaller. *Eld* is then divided by 20, giving the amount of dry lean mass to be added to *Ldm* in the present day (*Ldi*).

If energy from food (*FS*) is insufficient to meet metabolic costs (i.e., if *Eag* is negative), then no lean growth occurs, and this metabolic energy deficit is drawn from adipose tissue. To convert energy to fat I used a conversion factor of 38 kJ/g (Ricklefs and Schew 1994).

If the energy from food is greater than that required for metabolic costs and maximum lean tissue growth (i.e., $Eag > Egr$) then this surplus (Aea) is converted into fat and added to body fat (Ad). I have assumed that there is a limit on the amount of fat a chick of a given mass of lean tissue can carry. I have set this arbitrarily large (40% of wet lean mass) because the purpose of this model is to analyze the consequences of changing lean growth rate, rather than adipose storage capacity. If there is insufficient adipose the chick starves. For simplicity, starvation metabolism of lean tissue is not taken into account in this model.

The rate at which chicks can deposit fat is limited. I have assumed that, like lean tissue growth rate, the maximum fat growth increment for a day (Agr) is proportional to lean mass (Equation 6.3), but that the rate decreases as the maximum fat carrying capacity is approached (140% of Lwm, see above).

Equation 6.3 $$Agr = Ak.Ldm.(1-\frac{M}{1.4.Lwm})$$

where Ak is the maximum fat deposition rate for a species.

If Aea is greater than zero then the amount of energy used in fat deposition (Ef) is calculated; Ef is equal to either Aea or $AEgr$ (the maximum amount of energy that can be deposited as fat - i.e., Agr multiplied by 38kJ) whichever is the smaller. Ef is then divided by 38, giving the amount of body fat to be added to Adm in the present day (Adi).

In my first set of simulations, I fixed Ak at 0.05 for all values for maximum lean tissue growth rate (Ldk). In the second set of simulations I varied Ak in inverse proportion to Ldk. I started by using a high maximum lean tissue growth rate equal to the fastest growing chick in the first set of simulations (0.9) and a maximum fat deposition rate of 0.05. For a number of slower maximum lean tissue growth rates, I then calculated proportionately increased maximum fat deposition rates according to Equation 6.4. Therefore, given a starting maximum lean tissue growth rate (Ldk_1), and starting maximum fat deposition rate (Ak_1) and a new maximum lean tissue growth rate (Ldk_2), I calculated the new maximum fat deposition rate (Ak_2).

Equation 6.4 $Ak_2 = Ak_1 + (Ldk_1 - Ldk_2)$

I assumed that chicks neither have an indefinite amount of time in which to development, nor that their lean tissue growth can be indefinitely delayed without serious developmental problems. Hence, if a chick has not reached a certain state of lean tissue development in a certain amount of time then the chick is judged to have died due to stunted development and/or abandonment. To do this, the simulated chick is compared with a chick of the same age that has been growing under ideal conditions. First, the lean mass that would be expected for a chick growing under ideal conditions is calculated on the basis of the logistic equation (Equation 6.1). The integral of that equation gives the expected lean mass for a chick of a given age (Equation 6.5)

Equation 6.5 $$Ldo = \frac{Lda}{1 + \exp(o - (T.Ldk))}$$

where *Ldo* is the dry lean mass expected for a chick growing under ideal conditions at time *T*, and *o* is a constant defining the position of the logistic curve relative to the origin (I use a value for *o* of 1.5 in all simulations, a value that is representative of *o* calculated from real growth curves).

If lean tissue mass (*Ldm*) is less than 75% of asymptotic dry lean mass (*Lda*) after a period that is 140% of the time taken to reach 95% of asymptotic lean mass under ideal conditions (calculated by rearranging Equation 6.4) then the chick is dead. This criterion is arbitrary, and introduced for the requirements of the model. In reality, different species will have varying degrees of flexibility to their development.

The uniform distribution model

This model requires only a simple modification of the lean-priority model. In the lean-priority model, a quantity of the energy available for growth (*Eag*) is consumed by lean tissue growth. Any surplus energy is converted into fat. In the uniform distribution model, a fixed fraction *R* of *Eag* is available for lean growth (*El*), and the remainder (*Ea*) is available for conversion into fat (subject to the same constraints as the lean-priority model with respect to maximum lean tissue and adipose growth increments), hence:

Equation 6.6 $El = R.Eag$

Ldk and *Ak* are fixed at 0.9 whilst *R* varies from 0.2 to 1.0.

The fat-priority model

The fat-priority model reverses the priority of energy flow between lean growth and fat deposition. In the lean-priority model maximum lean tissue growth rate (*Ldk)* is calculated first and any surplus energy is converted into fat. In the fat-priority model, if the energy intake is enough for general metabolism and the maximum fat deposition rate then the remainder is converted into lean tissue. I ran several simulations at various values for *Ak* with *Ldk* fixed at a value of 0.9.

Results

The results are divided into two sections. The first section consists of the results of the simulations and the second section consists of tests of the models using comparative data.

Simulations

I translated each of these models into computer code using the programming language "RESAMPLING STATS" (Simon 1994) (see Appendix 8 for computer code), a simple language that is designed for monte carlo statistical modeling.

A simulation run generates a large number of growth trajectories for individual "chicks". Simulated data for each chick is generated using the iterative approach described above. At the end of a simulation run, summary statistics for the "chick population" are calculated. New simulated "populations" are then generated by varying the parameters of the models.

I ran simulations for each of the models by first varying *Ldk* in the lean-priority model with *Ak* fixed at 0.05, and then with *Ak* varying in inverse proportion to *Ldk* (Equation 6.4). For the uniform distribution model I ran simulations at varying values for *R*, and in the fat-priority model for varying values of *Ak*. I simulated the risk of predation by building into the models a probability (0.015) of a chick being killed in a day Lima (1986) . The choice of 0.015 was arbitrary. For each set of simulations I then calculated a probability of fledging that takes into account nestling mortality due to starvation and due to predation. For each "population" of simulated chicks I also

calculated the realised mass growth rate (*MGR*) and realised wet lean tissue growth rate (*LWGR*) by fitting the logistic equation to scattergrams from simulated growth curves for weights and wet lean masses respectively. The ratio of *MGR* to *LWGR* ($\frac{MGR}{LWGR}$) is directly analogous to *GRI* ($\frac{WGP}{MGP}$) in Chapter 3, hence for simplicity I will refer to it also as *GRI*. Growth rates were simpler to calculate from simulated growth data than growth periods.

For each simulation, asymptotic dry lean tissue mass (*Lda*) is 100g, and hatching dry lean tissue mass is 1g. Chicks are born with a body fat (*Ad*) equal to 25% of wet lean tissue mass (0.75g). Food supply in ideal and variable conditions are discussed in Appendix 6.

Simulation results

I first compared the results of varying the maximum lean tissue growth rate (*Ldk*) in the lean-priority model, with an ideal food supply and with a variable food supply (Figs. 6.2-3). With an ideal food supply, the probability of fledging increased steadily with growth rate. With a variable food supply, the probability of starvation increased with an increase in lean tissue growth rate. This meant that there was a lean tissue growth rate that optimised the probability of fledging, and that probability of fledging was lower than under ideal conditions.

However, when maximum fat deposition rate (*Ak*) was fixed, the reduction in starvation rate due to reduced growth rate was relatively small, hence, the probability of fledging at the optimal *Ldk* was only marginally greater than for other values. The highest probability of fledging was achieved when *Ak* increased in inverse proportion to *Ldk*.

The ratio of mass growth rate to lean tissue growth rate (*GRI*) decreased with increasing lean tissue growth rate (Fig. 6.3). However, the effect was very weak when *Ak* was fixed. A variable food supply depressed mass growth rate more than lean tissue growth rate when *Ak* was not fixed, hence the ratio was consistently higher with an ideal food supply.

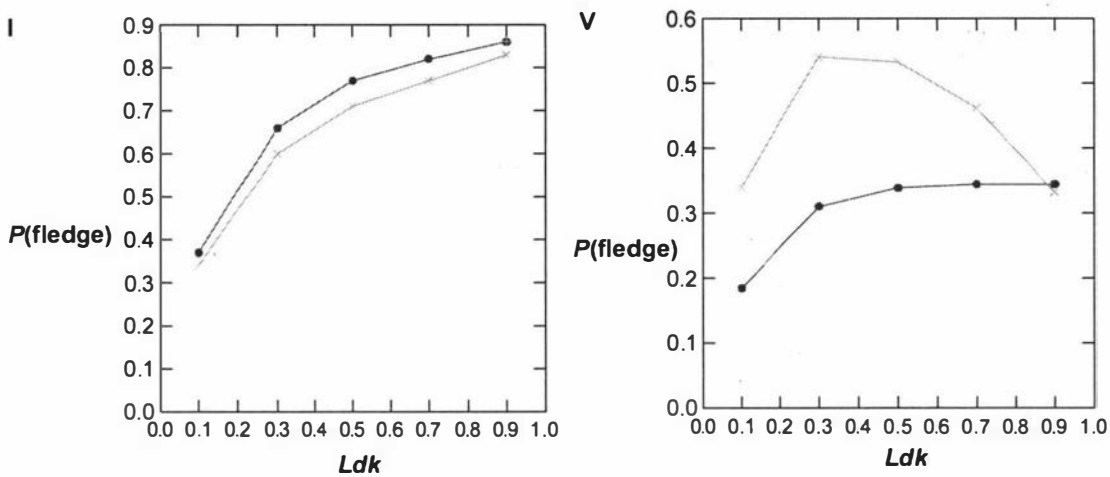

Fig. 6.2 Probability of fledging in lean-priority growth models in relation to maximum lean tissue growth rate. *P*(fledge), the probability of successfully fledging calculated as the frequency of fledging in 250 simulated chicks. Successfully fledged chicks had fat levels that stayed above zero and avoided a 0.015 chance per day of death by predation. *Ldk*; dry lean tissue growth rate (*Ldk* = 1 means that chicks initially double in weight in one day). Asymptotic lean mass (*Lda*) is 100g, initial dry lean mass (*Ldm*) is 1g, and initial fat (*Ad*) is 0.75g.; *I*, ideal food supply (see Appendix 6), *V*, variable food supply; ● fixed *Ak*; × *Ak* varies in inverse proportion to *Ldk*.

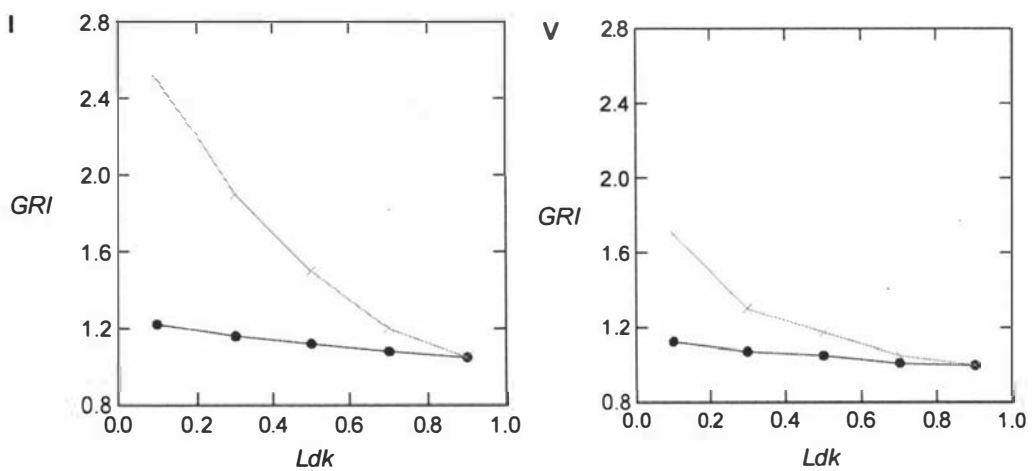

Fig. 6.3 Lean tissue growth rate and *GRI* in relation to the lean-priority model.
See text and table 6.2 for an explanation of the variables . Points are mean values for 250 simulated chicks. Conventions as for Fig. 6.2

The results for the uniform distribution model were similar to the results for the lean-priority model when *Ak* was not fixed (Fig. 6.4). Under ideal conditions, the

probability of fledging increased, and *GRI* decreased, with increasing *Ldk*. With a variable food supply the probability of fledging was maximised at lower growth rate. The trend in *GRI* was very similar with a variable food supply to that obtained with an ideal food supply. Unlike the lean-priority model, where fat is only deposited if energy intake crosses a certain threshold, in the uniform distribution model fat deposition occurs at all levels of energy intake.

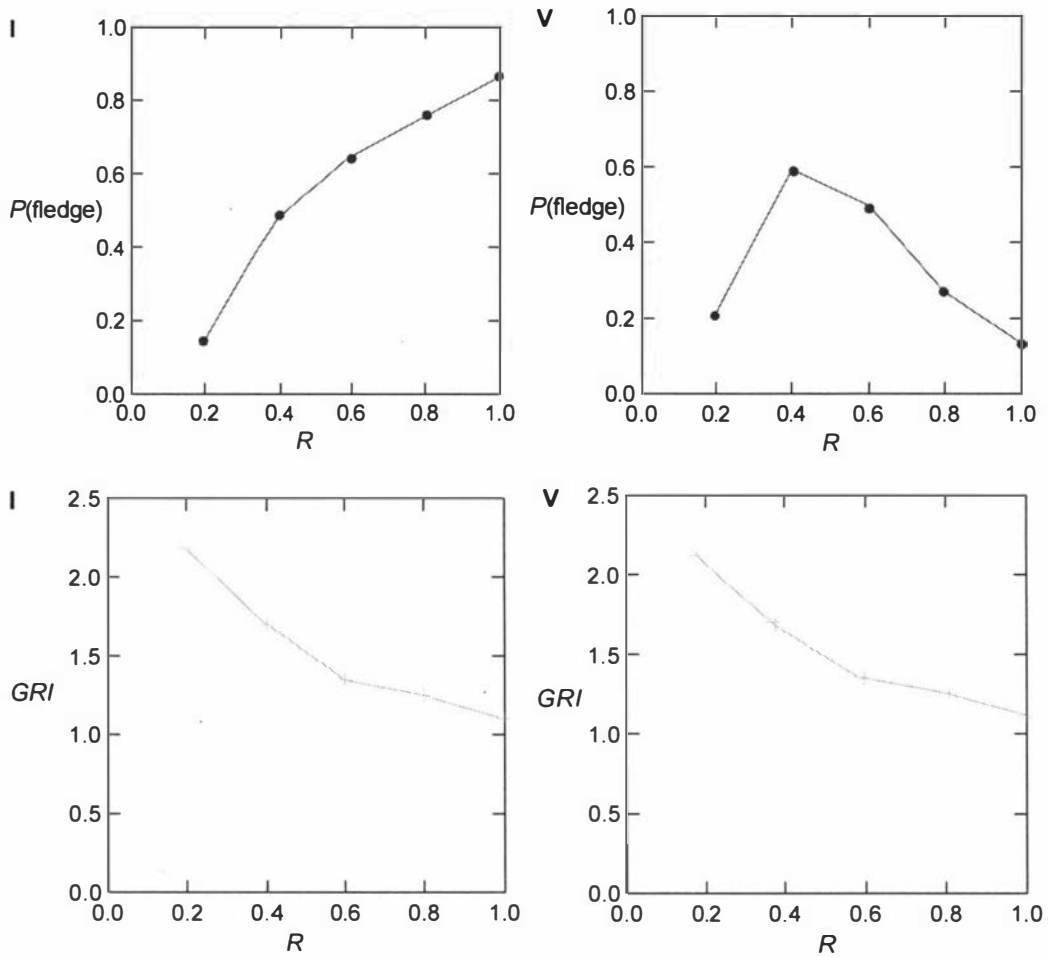

Fig. 6.4 Probability of fledging and *GRI* in the uniform distribution model in relation to the fraction of energy intake distributed to lean tissue growth *R*. *Ldk* and *Ak* are fixed at 0.9. Conventions are as for Fig. 6.2.

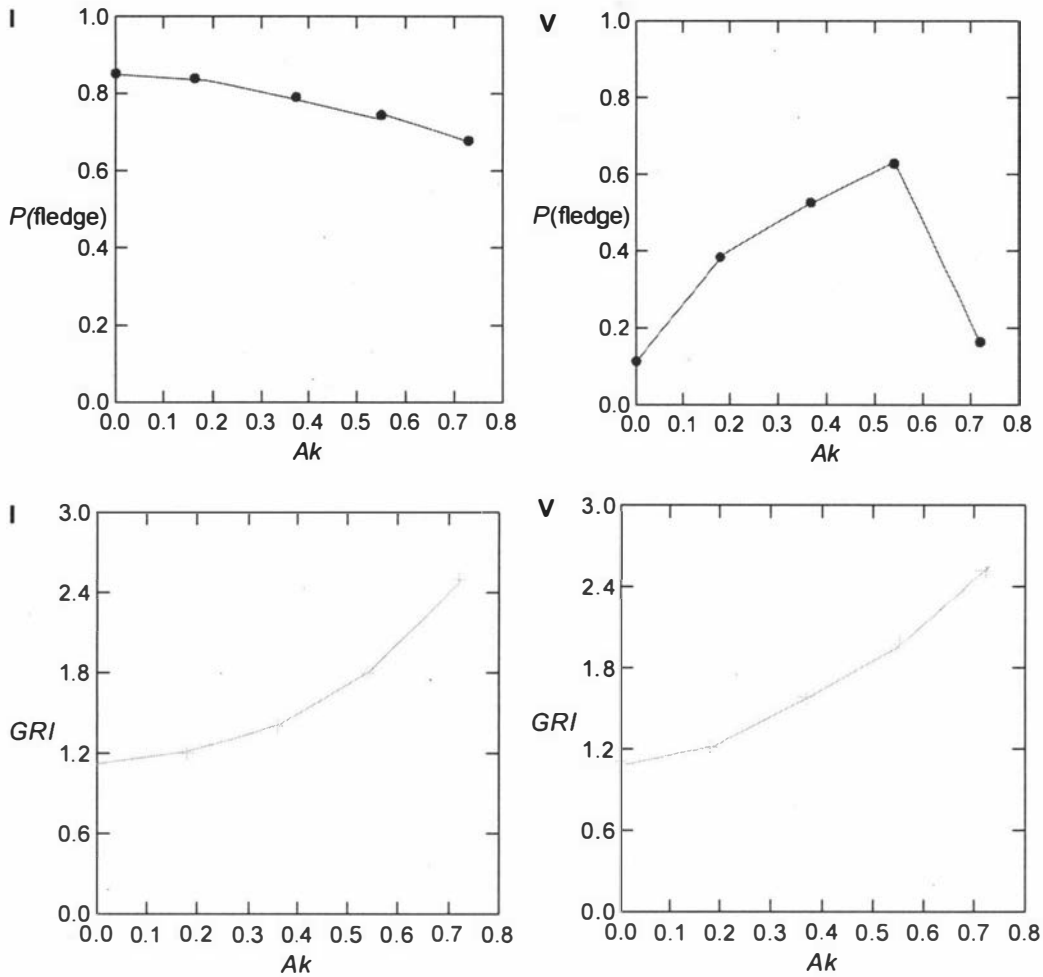

Fig. 6.5 Probability of fledging, and *GRI* in the fat-priority model in relation to maximum fat deposition rate *Ak*. *Ldk* is fixed at 0.9. Conventions as for Fig. 6.2.

Results were slightly different for the fat-priority model (Fig. 6.5). With an ideal food supply, as the fat deposition rate increases, the probability of fledging increases, *GRI* increase, and the probability of fledging decreased by small amount. With a variable food supply, resistance to starvation increases with increasing fat deposition rate. Probability of fledging peaked at an optimum value (Ak = 0.55). A major difference with the earlier models is that *GRI* is almost as large in environments with a variable food supply as in environments with an ideal food supply. This is because in this model, body fat is kept consistently high even under poor conditions. When energy intake is insufficient for a significant increase in lean tissue, fat deposition may still occur.

The growth models and comparative data

In Chapter 7 I will discuss experimental tests that can be used to discriminate between the lean-priority, uniform distribution, and fat-priority models. In this section I will discuss the models with respect to comparative data using both data from earlier chapters and new data.

Probability of fledging and lean tissue growth rate

In the lean-priority model, optimal lean tissue growth rates were relatively low when maximum fat deposition rate varied in inverse proportion with lean tissue growth rate. The probability of starvation is lowest at the lowest lean tissue growth rate, and only predation alters this - i.e., with a relaxation of predation pressure optimal lean tissue growth rates would be lower still. Furthermore, optimal values for R in the uniform distribution model and Ak in the fat-priority model correspond to relatively slow lean tissue growth rates.

However, when Ak is fixed, the lean-priority model does not predict a low optimal lean tissue growth rate. This may be because I have used a relatively low fixed value for Ak, but regardless, this suggests that a relatively high maximum fat deposition rate is an important factor for surviving a variable food supply.

The ratio of mass and lean tissue growth rate (GRI)

In Chapter 3 I listed predictions from several growth models (Table 3.1). The models shown in Figs. 3.6B, 3.6D, and 3.6E (Chapter 3) correspond to the lean-priority, uniform distribution, and the fat-priority models respectively. I predicted from each of the models that mean and minimum wing growth periods (hence, mean and maximum wing growth rates) would be relatively slow in species with a variable food supply. I also predicted from each of the models that the ratio of wing growth period to mass growth period (*GRI*, hence the ratio of mass growth rate to wing growth rate) would be higher in species with slow wing growth rates (hence in species with a variable food supply).

Assuming that wing growth rate is an indicator of lean tissue growth rate, the reliability of these predictions are supported by my simulations. *GRI* was negatively correlated with lean tissue growth rate in all the simulations.

In Chapter 3 I also predicted from the lean-priority model (Fig. 3.6D) that *GRI* would be greater with an ideal than with a variable food supply and that this difference would be greater in species with slow wing (hence lean tissue) growth rates. Therefore

the ratio of *GRI* with an ideal food supply and a variable food supply (*GAI*) would be greater in species with slow wing (hence lean tissue) growth rates (hence species with a variable food supply).

The reliability of these predictions are also supported by my simulations. *GRI* was greater with an ideal food supply than with a variable food supply for the lean-priority model, but not for the uniform distribution and fat-priority models. However, this was only true for the variable *Ak* lean-priority model (i.e., where maximum fat deposition rate (*Ak*) varies in inverse proportion to maximum lean tissue growth rate (*Ldk*)).

Mass growth rate with an ideal and with a variable food supply

In the fat-priority, uniform distribution, and the variable *Ak* lean-priority model, mass growth rate (*MGR*) is relatively high in ideal conditions regardless of *Ldk* (this can be seen from the high values for *GRI* at low *Ldk* for these models). In the uniform distribution and the variable *Ak* lean-priority model, mass growth rate is maintained at a high level by rapid fat deposition. Overall growth rates only decline significantly if lean tissue growth rates are set very low, hence fat deposition itself is constrained by the slow rate of lean tissue growth. By contrast, the fixed *Ak* lean-priority model has a slow mass growth rate with an ideal food supply if *Ldk* is set at a low value.

In Chapter 3, I found that mean mass growth period (hence mean mass growth rate) was correlated with variable food supply. However, the minimum mass growth period (hence maximum mass growth rate) was not significantly correlated with variability in food supply. Even if species are adapted to a variable environment their mass growth is still generally rapid in ideal conditions. In contrast, the wings of species in variable environments generally grow slowly even with an ideal food supply

The rapid maximum growth rate of species adapted to variable environments falsifies the fixed *Ak* lean-priority model. If species with a reduced maximum lean tissue growth rate do not have a corresponding increase in maximum fat deposition rate, then their maximum mass growth rate will be relatively slow.

The correlation of mass and lean tissue growth increments

Boersma and Parrish (1998) make a distinction between developmental and environmental regulation of lean tissue growth (Fig. 6.6). When lean tissue growth is developmentally constrained it is not affected by food supply and does not vary in

proportion to food supply and mass growth rate. When it is environmentally constrained, it is correlated with both food supply and mass growth rate.

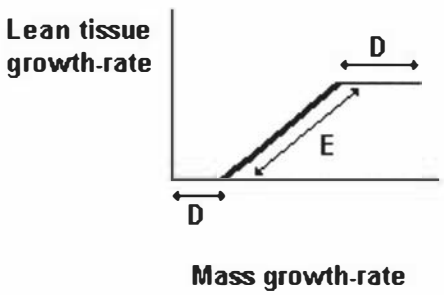

Fig. 6.6 Developmental and environmental regulation of lean tissue growth. Developmentally constrained lean tissue growth occurs in the regions labeled *D*. In the region labeled *E*, lean tissue growth rate is correlated with mass growth rate, hence the supply of food from the environment. Conventions are as for Fig. 3.6, Chapter 3.

According to the fixed *Ak* lean-priority model and the uniform distribution model, lean tissue growth rate should be almost exclusively environmentally regulated, hence mass growth increments and lean tissue growth increments should be correlated. The variable *Ak* lean-priority model (when *Ldk* is relatively low) and the fat-priority model allow for developmental regulation of lean tissue growth rate, and predict that mass and lean tissue growth increments will be less strongly correlated (Hamer and Hill 1993, Boersma and Parrish 1998)

I looked for correlations between daily mass and wing growth increments in growth data for five species (Table 6.3, sources in Appendix 1). I assumed that wing growth rates indicate lean tissue growth rates. Mass and wing growth vary with age, and according to different patterns. To detect a correlation between mass and wing growth it is necessary to convert growth increments into a form independent of age. I therefore converted growth increments into standardised growth increments - i.e., growth increments that have been standardised relative to initial values. This procedure is discussed in detail in Chapter 7. This method of comparing standardised growth increments is similar to that used by Shea and Ricklefs (1996) and Vinuela and Ferrer (1997).

Species name	Common name	r^2 of standardised wing and mass growth increments	Variability in food supply (V)
Apus apus	Swift	0.05	0.23
Catharacta maccormicki	Polar Skua	0.34*	0.05
Gygis alba	Fairy Tern	0.20	0.16
Hirundo neoxena	Welcome Swallow	0.29**	0.08
Puffinus ilherminieri	Audubon's Shearwater	0.32	0.27

Table 6.3 Regression analysis of daily mass and wing growth rates in five species. r^2 is calculated from the regression of standardised wing growth increments on standardised mass growth increments, * $P < 0.05$, ** $P<0.01$.

The Polar Skua (*Catharacta maccormicki*) and the Welcome Swallow (*Hirundo neoxena*) had significant correlations between standardised daily mass and wing growth increments, suggesting environmental regulation of lean tissue growth rate. This is consistent with the fixed *Ak* lean-priority model, the variable *Ak* lean-priority model at relatively high *Ldk*, and the uniform distribution model. The other three species did not have significant correlations between daily mass and wing growth rate suggesting developmental regulation of lean tissue growth rate. This is consistent with these species growing according to either the fat-priority model or the variable *Ak* lean-priority model at a low *ldk*.

The two species with evidence of environmental regulation of lean tissue growth rates have the lowest variability in food supply (V). However, the differences between species in both the correlations between growth increments and variability in food supply are too small to be able to draw any compelling conclusions.

Pauses in wing growth

In the fat-priority model wing growth will cease altogether under certain conditions. In the fat-priority model, wings do not grow if food intake is low (hence when mass growth rate is relatively low). I tested this using growth data for each of the five species listed in Table 6.3. I judged wings to have not grown on a given day if wings grew less than 5% of the expected daily growth increment. Expected wing-length growth increments were calculated from a best-fit logistic equation for wing growth data for each species. I estimated food intake using standardised mass growth increments (see above).

Only the Swift showed instances where wings did not grow (see also Appendix 7). In the Swift, there was no wing growth in five cases and wing growth in 44 cases. However, standardised mass growth (hence food supply) was not significantly lower in cases with where wings did not grow than cases where wings grew (1-tail t-test, P = 0.87). Therefore, pauses in wing growth are independent of mass growth (hence food supply) on a given day (in Appendix 7 I examine the hypothesis that pauses in wing growth are related to poor body condition). These results are inconsistent with the fat-priority model, but consistent with all the other models.

Discussion

The models suggest that a slow lean tissue growth rate can help chicks to avoid starvation in environments with a variable food supply. However, a more important factor appeared to be the maximum fat deposition rate (*Ak*). Reducing maximum lean tissue growth rates (*Ldk*) in the lean-priority model did not reduce the probability of starvation by a great deal when *Ak* was fixed, but it did when *Ak* varied in inverse proportion to *Ldk*. This suggests two possibilities. First, I may have set *Ak* too low in the fixed *Ak* model. Second, reduced lean tissue growth rates aid survival in environments with a variable food supply by allowing increased allocation of cells to tissues involved in fat deposition. Which of these conclusions is correct can only be answered empirically - i.e., by measuring actual fat deposition rates in species with various lean tissue growth rates. If the variable *Ak* model is correct, then these measurements should show that maximum fat deposition rates are inversely proportional to maximum lean tissue growth rates.

If this is correct, then this agrees with the "cell allocation model" discussed in Chapter 5. According to the cell allocation model, the growth rate of a body component is constrained by its initial allocation of cells. Early in development dividing cells are "allocated" to different functional roles. Some of these cells remain in an embryonic stage and continue to divide, and others differentiate into a functionally mature form. Tissues with a high initial allocation of cells continue to make up a proportionally large part of the body until the tissue reaches maturity. Such tissues will successfully compete for a greater fraction of resources than other tissues and grow relatively rapidly. A corollary of this hypothesis is that the evolution of increased cell allocation to one type of tissue will mean fewer cells allocated to other types of tissue. If this

applies to fat tissue and other tissues associated with fat deposition then increasing the maximum rate of fat deposition will require a larger allocation of cells to tissues of the adipose system and a corresponding decrease in allocation to the tissues of other systems.

Furthermore, because the cell allocation hypothesis assumes there is a direct trade-off between maximum lean tissue growth rate and maximum fat deposition rate, the maximum mass growth rate is similar in species adapted to variable to species adapted to stable conditions.[1] The is consistent with the results of Chapter 3 - i.e., minimum mass growth period (hence maximum mass growth rate) was not significantly correlated with variability in food supply. However, decreased cell allocation to lean tissue may also increase the *intrinsic* constraints on the supply of energy and nutrients to the growing tissues. This possibility is examined in Appendix 7.

Using data such as those presented here, it is possible to construct a picture of the growth processes involved in chicks of a given species. In this regard, it is instructive to compare the Swift and the Welcome Swallow.

The Swift has slow maximum lean tissue growth rate and a relatively fast maximum mass growth rate - i.e., mass growth rate is fast relative to wing growth rate and this is even more pronounced when the chick has an ideal food supply (GAI = 1.2, see Chapter 3). This supports the lean-priority model, assuming that Ak is set relatively high. Furthermore, daily wing and mass growth rates are virtually independent of one another, hence the Swift appears to carry out much of its growth in the D areas in Fig. 6.6. That is, wing growth rate is constrained more by developmental than environmental factors. This either supports the fat-priority model or means that Swift chicks often have more than enough energy to achieve their maximum wing growth rate. Swift chicks also show pauses in wing development. These occur independently of mass growth rate, contradicting the fat-priority model. However, in Appendix 7 I show that pauses in the wing development only occur in chicks in very poor condition - i.e., supporting the *facultative* fat-priority model.

The Swallow has a faster, but still relatively slow, lean tissue growth, does not appear to pause wing growth, has relatively fast mass growth rate relative to wing

[1] This is a simplification, i.e., the assumption that growth in lean tissue and in fat have similar costs in energy, whereas in reality fat has a much higher energy density that lean tissue (cf. Appendix 3).

growth rate, and has daily wing growth rates that are weakly correlated with mass growth rates. This suggests a lean-priority model, with only a moderate decrease in maximum lean tissue growth rate (this hypothesis is examined further using experiments in Chapter 7). Therefore, Swallow chicks may be adapted to survive a moderately variable food supply. By contrast, Swift chicks appear to possess a combination of strategies for dealing with a highly variable food supply.

Various authors (e.g., O'Connor 1978, Vinuela and Ferrer 1997) have suggested that the ability to retard lean tissue growth, including feather growth, may be important for chicks of species with an variable food supply. These models suggest that there are several ways in which this may occur. Depending on the model, lean tissue growth rate may be retarded in step with mass growth rate, or, mass growth rate may be relatively independent of lean tissue growth rate. This has implications for ptilochronology, the study of variations in the food supply by examination of fault bars in feathers (Grubb 1989, 1992). Fault bars may occur in the rectrices of birds that have sporadically insufficient food supplies during feather growth. Ptilochronology, however, rests on the assumption that fluctuation in food supply is a good predictor of short term variation in feather growth (McCarty and Winkler 1999). This assumption has been criticised by Murphy and King (1991) and Murphy (1992), who argue that that body reserves might moderate any reduction in the growth rates of feathers. Murphy and King (1991) point out that in some species feather growth is not affected until the third or fourth day after a decline in food supply. They argue that feather growth differs from fat deposition by involving the synthesis and differentiation of a complex structure and therefore feather growth is relatively immune to the effect of food supply on the day of growth and may be influenced more by environmental conditions several days prior to the actual growth (McCarty and Winkler 1999).

My analysis suggests feather growth (hence the distribution of fault bars) is only a good predictor in food supply in species with environmental regulation of feather growth. The assumptions for ptilochronology will not be met in species with developmental regulation of feather growth (Fig. 6.6). Therefore ptilochronology may not be reliable for species adapted to a variable food supply.

The simulations and data presented in this chapter support the view presented in earlier chapters that species with low lean tissue growth rates and with a variable food supply have fast maximum mass growth rates (hence fast maximum fat deposition

rates). High maximum fat deposition rates (*Ak*) help survival in environments with a variable food supply. The variable *Ak* lean-priority model is consistent these conclusions and is also consistent with the cell allocation model of hyperlipogenesis discussed in Chapter 5. In the next chapter I will test the lean-priority, uniform distribution, and fat-priority models using experimental data on one species, the Welcome Swallow (*Hirundo neoxena*).

Chapter 7

Testing growth models using the Welcome Swallow as an experimental system

Introduction

Lack argued that some of the similarities between the life histories of swifts and Procellariiformes exist because both types of bird are adapted to raise young under a variable food supply. I examined this hypothesis using comparative data in Chapter 2. In Chapter 3 I developed models of the relationship between mass growth rate and lean tissue growth rate. Three of these models (Figs. 3.5B, 3.5D, and 3.5E) were advanced as interpretations of Lack's insurance hypothesis. Using comparative data, I tested these models through their predictions about the maximum lean tissue growth rates of species with a variable food supply. In Chapter 5 I studied the "energy savings model" in an attempt to explain why slow lean tissue growth rates could be adaptive in environments with a variable food supply. In Chapter 6, I developed this model further using computer simulations. I considered three variations, using assumptions corresponding to Figs. 3.5B, 3.5D, and 3.5E, which I called the lean-priority model, the uniform distribution model, and the fat-priority model respectively. I also considered two variations on the lean-priority model, showing that the version in which maximum fat deposition rate varies in inverse proportion to maximum lean tissue growth rate gives the best probability of fledging in environments with a variable food supply. I interpreted this variation of the lean-priority model as being consistent with the cell allocation model examined in Chapter 5.

In the lean-priority model mass growth is due solely to lean tissue growth up to a maximum rate of tissue growth. When enough energy is available for the maximum tissue growth rate, any addition energy is converted into fat. In the uniform distribution model mass growth rate (which includes lean tissue growth and fat deposition) is a relatively constant multiple of tissue growth rate. In the fat-priority model; mass growth rate is due solely to fat deposition when the food supply is low. Each of these models predicts a different effect on the relative growth rates of mass and lean tissue resulting from food deprivation. These predictions are examined in more detail in the methods below (Fig. 7.2).

These predictions can't be tested with the comparative data that are available, but require experiments on individual species. Ideally, these experiments would be carried out on a number of species, and the results compared for species living in different environments. However, a single species can be used for a less comprehensive test of the models, so long as the species has a variable food supply. An experiment carried out by Congdon (1990) on Black Noddy (*Anous minutus*) chicks can be interpreted as an experiment of this type. Congdon swapped chicks between broods to induce food deprivation in artificially large broods. The chicks in larger broods underwent a decrease in wing growth rate, but not weight growth rate, thus corroborating the fat-priority model.

I used the chicks of the Welcome Swallow (*Hirundo neoxena*) as an experimental system to test my growth models (Plate 1). This species is suitable for several reasons. Like all swallows, the Welcome Swallow is an aerial insectivore. Aerial insect abundance is dependent on the weather, so swallows are faced with the problem of a food supply that varies with the weather. However, swifts forage in open spaces high in the air column (Lack and Lack 1951), whereas swallows feed much closer to the ground and to cover. Swallows are therefore able to forage in areas that provide shelter for aerial insects even in bad weather, such as near hedges and trees. For this reason, the swallow is not as vulnerable to variations in food supply due to the weather as the Swift. Any adaptation of the chick to a variable food supply would therefore be expected to be less extreme than that of the Swift. The nestling period of the Welcome Swallow is 22 days on average, which is relatively long for a bird of its size of 15g (10-15% longer than the nestling period expected by mass from allometry) but short relative to the 42 days of the 43g Swift (*Apus apus*) (50% longer than the nestling period expected by mass from allometry). The Welcome Swallow is similar to the Swift and Procellariiformes in that its weight peaks well above its fledging weight (up to 134% of fledging weight in this study). The Welcome Swallow is also similar to swifts and Procellariiformes in that the body mass reaches fledging weight well before the wings reach their mature length. This may be due to the demands of developing wings for aerial foraging, or part of a growth priority strategy as described below. The suitability of the Welcome Swallow as an experimental system is examined in more detail below.

Methods

This chapter consists of two main parts. First, I test the suitability of the Welcome Swallow as an experimental system upon which to test the growth models. Second, I test the growth models themselves using experiments.

The Welcome Swallow study population

The Welcome Swallow is indigenous to Australia and colonised New Zealand sometime in the early 20th Century. The transformation of New Zealand from a forest-clad to a pastoral landscape has facilitated the success of the species (Robertson 1985). In its native range the species is found in open areas such as farmland, grassland, and partly cleared woodland, often associated with water. The distribution of Welcome Swallows in Australia has closely matched the expansion of human activity (Marchant and Fullagar 1982). The breeding biology of the Welcome Swallow in Australia is detailed by Marchant and Fullagar (1982), who note its close similarity to that of the Barn Swallow *Hirundo rustica.* Nest, nest site, growth rate, and division of duties between parents are closely similar to that given for *H. rustica* in Britain by Adams (1957).

This study was carried out on the Heretaunga Plains, Hawkes Bay, in the North Island of New Zealand in the summers of 1997/1998 and 1998/1999. Irrigation ditches run across the Heretaunga Plains. Swallows nest under bridges and culverts crossing these ditches, and are easily accessible for experimental manipulation (Plate 2). They are, however, well protected from ground predators. Only one pair nests at a site at one time, and up to three broods may be reared at one site per breeding season. The breeding season in this study was found to be much the same as in Tasmania, which lies at a similar latitude to the study area (Marchant and Fullagar 1982). The earliest nestlings observed in 1997 were about 13 days old on 6 October, and most study sites were producing eggs by this date. In 1998 no nestlings were found hatched earlier than October. In both seasons, no nestlings were observed later than February.

Plate 1 Welcome Swallow chicks at 3 days after hatching, at the Raupare Road nest site

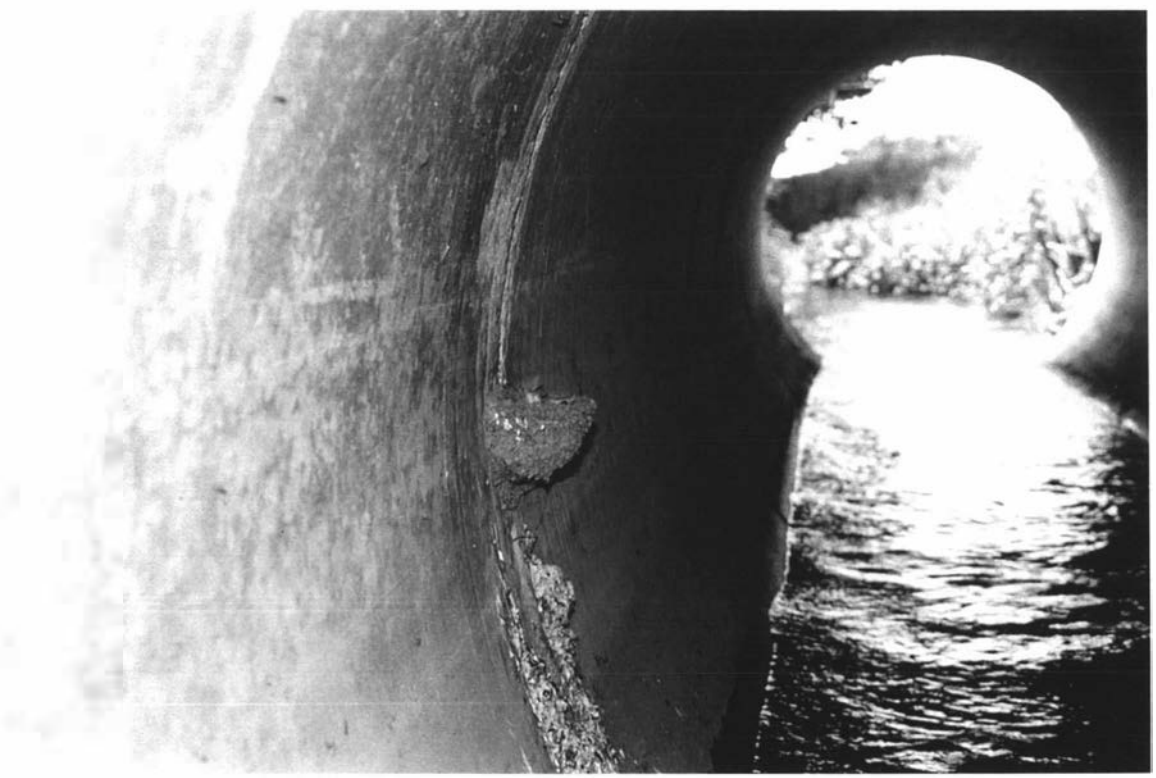

Plate 2 The nest site at Tywford Road, a typical nest set site for Welcome Swallows' in the Heretaunga Plains population

Testing the suitability of the Welcome Swallow as an experimental system

The assumption that the Welcome Swallows are suitable for testing my growth models is itself a hypothesis. To be a suitable experimental system, the Welcome Swallow must have a food supply that is temporally variable, and its rate of growth must be affected by variations in the food supply. To facilitate testing these assumptions, I constructed a model of the relevant causal pathways in swallow breeding biology (Fig. 7.1), based upon a similar model that McCarty and Winkler (1999) tested for Tree Swallows (*Tachycineta bicolor*). This model summarises assumptions about the breeding system of the Welcome Swallow. To avoid confusion with the growth models, I refer to it as the "system model".

The system model assumes that the weather (rain, wind, and temperature) affects the growth of nestlings indirectly though its affect upon the food supply. Lack and Lack (1951) have shown that feeding frequency of the Swift is negatively correlated with rainfall. Bryant (1975) found that aerial insect abundance in Berkshire, England is negatively correlated with rainfall and wind intensity, and positively correlated with sunshine hours. McCarty and Winkler (1999) found that aerial insect abundance is positively correlated with maximum daily temperature in Tree Swallow habitat. The model also assumes that the weather affects nestling growth more directly. Chicks may grow more slowly in lower temperatures because: (i) more energy is spent on thermogenesis, leaving less for growth, (ii) parents may brood the chicks more, hence feed them less. Hence, the model attempts to account for the direct effects of the weather when determining the effect of the food supply on growth.

The model also assumes that chicks in large broods will have a decreased food supply, and that this will have a negative affect upon growth. The assumption that brood size affects food intake is an important assumption of the brood swapping experiment described below.

To test the system model, I followed the methods of McCarty and Winkler (1999), who used path analysis to test their system model for Tree Swallows. Path analysis is a method designed for applying multiple regression analysis to systems where independent variables are correlated, and the determinants of an ultimate dependendent variable (mass growth rate in this case) may affect one another (Wright 1920). Path analysis has been widely used in ecology, to test hypotheses where experiments are not feasible (e.g., Kohn and Walsh 1994, Wooton 1994).

The model is tested by multiple regression analysis for each dependent variable (i.e., a variable to which an arrow, or arrows, point). Standardised partial regression coefficients are entered onto the path diagram as path coefficients. These represent effect sizes, and the sign (positive or negative) of the effects. The statistical significance of path coefficients is indicated by asterisks. No asterisk indicates no significant effect ($P > 0.05$), * indicates $0.05 > P > 0.01$, and ** indicates $P < 0.01$. The effects of unmeasured variables (i.e., U_m and U_a for mass change and insect abundance) are represented by error path coefficients, calculated as $\sqrt{1-r^2}$, where r^2 is the proportion of variation in the dependent variable that is explained by all the independent variables (McCarty and Winkler 1999).

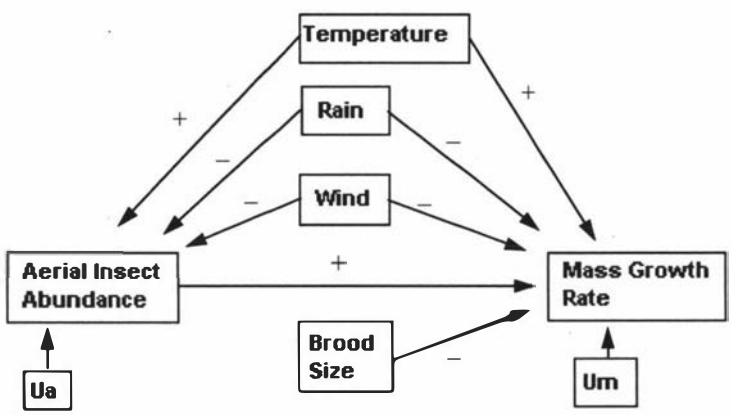

Fig. 7.1 Model of relationships of environmental variables to nestling growth in Welcome Swallows. Sign (+/-) shows the effect of an increase in the variable at the base of the arrow on the variable at the tip of the arrow. Ua and Um show effects of all unmeasured variables on aerial insect abundance and nestling mass growth rate respectively.

I collected data in the 1997/1998 season differently than in the 1998/1999 season, hence I was unable to pool the data for two seasons. I sampled insect abundance only in the 1998/1999 season. Also, in 1997/1998, data collection was driven by the requirements for a brood manipulation experiment (see below). As a result, I recorded data throughout the nestling period. In the 1998/1999 season, data collection was driven by the requirements of a deprivation experiment (see below), and I recorded data only during the initial stage of the nestling period.

To further test the system model I carried out a two-way ANOVA of standardised growth increments from October-November 1997. At this date 17 chicks were nesting at the same time. As a result, I was able to examine the amount of variance in growth increments both among chicks and among days. A relatively large amount of variance in the growth increments among days indicates a temporally varying environment. A low value could indicate that most of the variability in chick growth was due to differences in foraging ability amongst the parents or spatial variability of the food supply.

Parameter estimation for testing the system model

Measuring growth

In both breeding seasons, I measured the gain in mass of a chick as the difference between its weight on successive mornings (referred to as "growth increments"). I weighed the chicks with 30g persola scales. In the 1997/1998 season, I collected data from 68 chicks in 18 nests, over the entire nestling period. In the 1998/1999 season, I collected data from 67 chicks in 19 nests, only over the first 5 days of the nestling period.

Mass growth increments are dependent upon the age of the chicks. Studies of the effect of the environment on chick growth have not always taken this into account. Dunn (1975) found that in Sandwich tern (*Sterna paradisea*) chicks, mass growth increments were negatively correlated with wind-speed, but did not take into account the dependence of growth rate on age. McCarty and Winkler (1999) attempted to account for age-dependence by using age as a variable in their path analysis. They were able to do this because they used chicks only differing in age by a few days. Hence, in their model, growth rate could be treated as an approximately linear function of age. I was able to do this with my 1998/1999 data, which consisted of measurements taken from chicks aged 1 to 5 days. However, my 1997/1998 data covers the entire nestling period, over which the relationship between age and mass growth rate is not linear, as assumed by the regression model (this follows from the sigmoidal dependence of mass on age). It meant that I could not use chick age as a variable in the path analysis for my data. I instead converted growth increments into standardised growth increments that were independent of age.

In order to calculate standardised growth increments, I first pooled the data for chick weights and ages, and fitted the logistic equation to the data. From this equation I calculated expected growth increments for chicks of given masses. Standardised growth increments are the residuals of the regression of actual growth increments on expected growth increments. Bryant (1975) used a similar method to measure growth of House Martin (*Delichon urbica*) chicks, except that he used percentage of expected growth. I did not use this index because it becomes extremely variable near fledging because expected growth approaches zero. The index used here allows use of data for chicks much closer to fledging than does Bryant's index. Standardised growth increments are indicators of *incremental* growth rate - i.e., the amount of growth per day (cf. Chapter 5).

Weather data

I obtained daily data on rainfall, average wind-speed, and maximum temperature for the Hawkes Bay from the New Zealand meteorological service. Daily rainfall may be misleading however, because it doesn't accurately indicate the amount of foraging time lost. I therefore ranked days according to the number of hours without rain, recognising three levels 0-5, 6-10, and 11.

Aerial insect abundance

I measured aerial insect abundance throughout the 1998/1999 season with a hand net, using the same method as Jones (1986). One hundred sweeps of the net were made at each of three sampling sites each day, and the mean number of insects caught was used as an index of abundance.

Experimental tests of the growth models

The three growth models (lean-priority, uniform distribution, and fat-priority) each predict a different effect of food deprivation on the relative (see Chapter 5) growth rates of mass and lean tissue (Fig. 7.2, Table 7.1).

If food deprivation results in a relatively greater decrease in mass growth rate than lean tissue growth rate this supports the lean-priority model. If the opposite is true this supports the fat-priority model. The uniform distribution model is effectively the null model of no significant difference between the effects on incremental lean tissue and mass growth rate. Logical problems arise when the logical prediction of a model corresponds to the null statistical hypothesis (Underwood 1990). The uniform

distribution model may be falsified by rejection of the statistical null hypothesis, but it cannot be supported by provisional acceptance of the null statistical hypothesis. Hence, only the lean-priority model or the fat-priority model can gain logical support from these experiments.

I carried out two experiments to test the growth models. In the first experiment, I swapped chicks between broods to obtain artificially large and artificially small broods, and compared growth rates calculated over the entire nestling period. Larger broods are assumed to have less food delivered per chick (this assumption is tested along with the system model). In the second experiment, I compared the daily growth increments for chicks that had been deprived of food in the previous day and control chicks that had not been deprived of food.

Nests were observed from the date when eggs were discovered until the fledging of the chicks. Upon fledging the chicks were observed to leave the nest and stay in a family group for at least a day. Early on in the study, some chicks fledged while still poor fliers, probably due to handling. Handling of chicks was subsequently restricted to those aged less than 18 days.

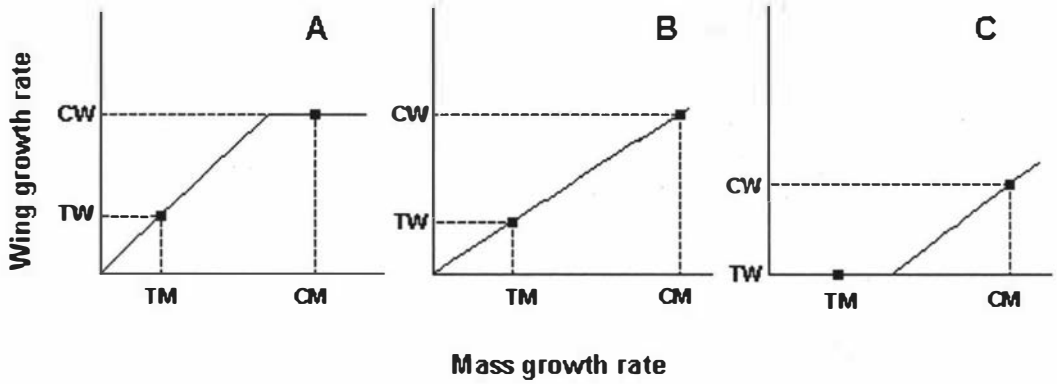

Fig. 7.2 The effect of food deprivation on mass and wing growth rates in three growth models. Dots represent well fed (control) and treatment (i.e., food deprived) growth rates. *CW* = control wing growth rate, *TW* = treatment wing growth rate, *CM* = control mass growth rate, *TM* = treatment mass growth rate. (A) lean-priority model, (B) uniform distribution model, (C) fat-priority model.

Model	**Prediction**
Lean-priority	$\frac{TM}{CM} < \frac{TW}{CW}$
Uniform distribution	$\frac{TM}{CM} = \frac{TW}{CW}$
Fat-priority	$\frac{TM}{CM} > \frac{TW}{CW}$

Table 7.1 Predicted effects of food deprivation from three growth models. Abbreviations are defined in Fig. 7.2.

Brood Swapping Experiment

In the 1997/1998 season I swapped chicks between nests to artificially increase and decrease the size of broods. Chicks were transferred when 3 days old, and were all accepted into their new broods. Enlarged broods had 4 or 5 chicks (both resident and transferred), and small broods had 1 or 2 chicks (all resident). Chicks in the enlarged broods correspond to the "control" chicks in Table 7.1 and Fig. 7.2. I swapped a total of 8 chicks among 11 nests (Table 7.3).

To estimate the incremental growth rates for wing-length and mass, I first fitted linear regressions to each chick, taking age as the independent variable, and mass and wing-length as the dependent variables. I calculated the regressions using data from the period during nesting when growth is approximately a linear function of age; 4 to 12 days for mass, 6 to 16 days for wing-length. I took mean linear coefficients for each brood, as indicators of growth rate for both mass and wing-length. I tested whether mass growth rates and wing growth rates differed between reduced and enlarged broods using two-sample t-tests. I also pooled all enlarged-brood chicks, and all reduced brood chicks, in order to compare their mass and wing-length growth patterns. I first fitted the logistic equation to each set of pooled data. I then plotted all four equations on the same graph. In order to represent wing and mass growth on a common scale, I plotted wing-length and mass as fractions of the asymptotic values for reduced broods.

There were two problems with the brood swapping approach. First, there was little synchronisation of nesting between pairs. This made it difficult to match nests with chicks of similar age for brood swapping. This meant that I was unable to achieve an adequate sample size. Second, both the constraints of matching similar aged chicks in different broods, and the capacity of the nests to hold chicks, meant that I was unable to increase brood size beyond the natural range. Brood size ranged naturally from 2 to 5 chicks (☒ 3.8, sd. 0.98) in my study, and broods of 6 chicks have been occasionally recorded in Australia (Marchant and Fullagar 1982). Jones (1986) managed to artificially increase brood size to 8 chicks in *Hirundo rustica*. It was not clear whether the amount of food provided to individual chicks in large broods in my study was appreciably smaller than that in small broods. To counter these problems, in the 1998/1999 season I directly manipulated the access of parents to the chicks, to induce short term deprivation.

Deprivation Experiment

I induced deprivation in chicks by placing a barrier over half the chicks in each nest. The other chicks in the nest were the control group, and parents continued to feed these chicks. The presence of accessible chicks in the nest probably minimised the risk of desertion by the parents, and no nests were deserted.

Chicks were deprived of food for 6.5 hours, when they were 6 days old. This amount of time proved sufficient to test the models without placing undue stress on the

chicks. All treatment chicks subsequently fledged. At 6 days of age brooding had ceased, but the chicks were still increasing in mass. Chicks were weighed and measured at the start of deprivation, at the end of deprivation, the following morning, and on the second morning after deprivation.

Whereas growth rate was measured over a period of 10 days in the brood swapping experiment, in this experiment growth was measured over days and hours. I could not compare mass growth rates for control chicks with treatment chicks either during or immediately following deprivation, because treatment chicks continued to defecate during deprivation. Hence, treatment chicks lost mass by partially emptying their digestive system during deprivation. Immediately after deprivation, treatment chicks probably gained mass immediately by re-filling their digestive tracts. I therefore used the differences between measurements taken at the start of deprivation and on the second morning after deprivation to compare treatment and control chicks. I assumed that the gut of treatment chicks would be re-filled by the second morning after deprivation, hence the difference in mass between the two measurements in treatment chicks would not be due to the amount of material in the gut.

For each chick, I calculated mass and wing growth rate as the percentage change from the mass and wing-length measured at the start of deprivation, to the second morning. For each brood, I took the means for control and treatment chicks. I used these means to calculate the test statistic shown in Table 7.1. Each brood is taken as one replicate.

A potential confound is that treatment chicks may lose mass during deprivation due to metabolism, creating a bias toward the lean-priority model. I checked for this by removing 5 chicks from nests in November 1999 and measuring their weight loss over 6.5 hours. I collected each chicks feaces produced over the 6.5 hour period and compared the weight of the feaces with the weight lost by the chick over the period of deprivation.

Another potential confound was the re-use of nest sites. Different broods subsequently occurring at the same nest may have been from the same pair of adults (Fig. 7.3). However, when I took the means of the test statistics for each nest site, and used the nest site means as replicates, the result was not significantly different from that obtained by taking broods as replicates.

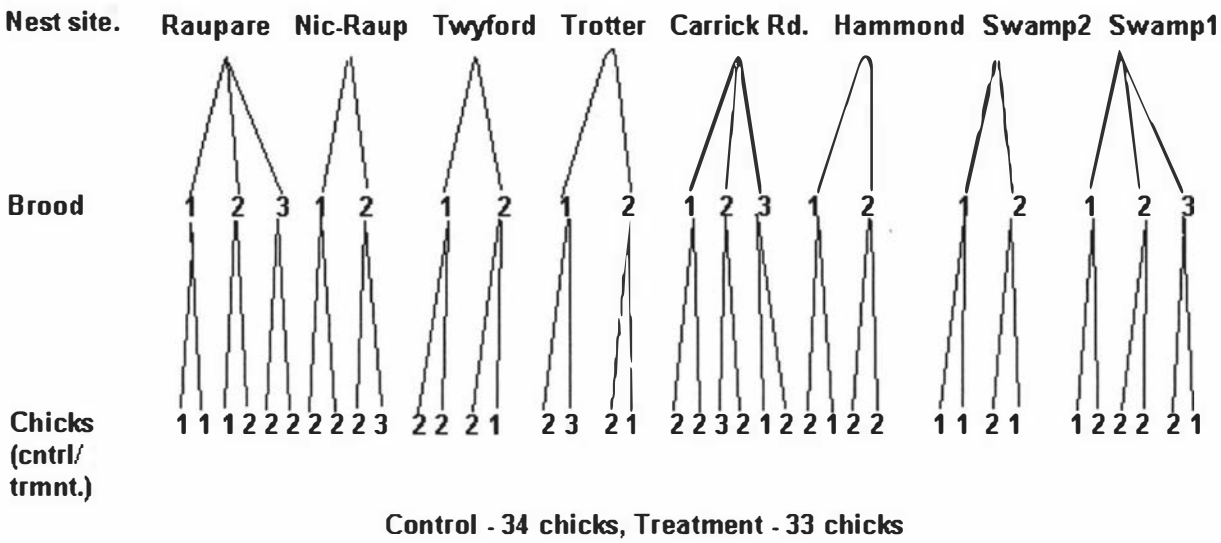

Fig. 7.3 The experimental design for the deprivation experiment. Nest site = the nest in which broods were raised, Brood = successive broods at the same nest site, Chicks = the number of control chicks (left) and treatment chicks (right) in a brood.

Results

This section consists of three parts, the results of the tests of the system model, the brood swapping experiment, and the deprivation experiment.

Testing the system model

In 1997/1998, mass growth rate was significantly correlated with maximum daily temperature (Fig. 7.4). Without data for insect abundance, it is impossible to know if the this was due to the direct affect of temperature on chick physiology and/or parent behaviour, or whether it was due to an effect on the food supply.

Contrary to the system model, rainfall and wind did not have a significant effect on mass growth rate. However, the summer of 1997/1998 was a drought, with only a few days with rainfall, and these only consisted of brief showers. The 1997/1998 season therefore lacked sufficient variation in rainfall to adequately test the model. Brood size was negatively correlated with nestling growth. This supports the assumptions of the brood swapping experiment.

Most (58%) of the variation in growth (once the age of the chick is controlled for) was accounted for by day to day variation, and this variation is statistically significant ($P < 0.01$, Table 7.2). Variation among chicks made up only 24% of the

total variation in growth, and was not statistically significant. This supports the use of the Welcome Swallow as an experimental system upon which to test the growth models

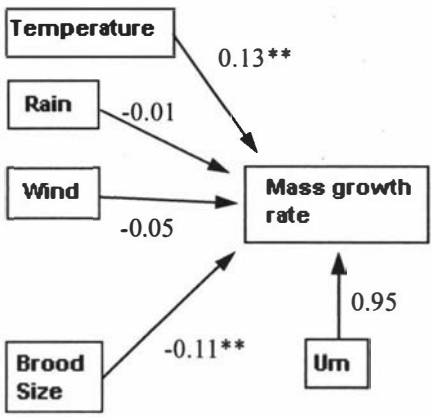

Fig. 7.4 Path diagram showing the effects of environmental variables on mass growth rate in 1997/1998. Numbers for independent variables are standardised coefficients using multiple regression analysis, the coefficient for U_m is $\sqrt{1-r^2}$ where r^2 is the proportion of variation mass growth rate that is explained by all the independent variables, * indicates $0.05 > P > 0.01$; ** indicates $P < 0.01$. Conventions are as for Fig. 7.1.

Source	MS	df	F	P	r^2
Chick	0.14	16	1.4	> 0.10	0.24
Day	0.32	14	3.2	< 0.005	0.58
Error	0.10	224			

Table 7.2 Two-way ANOVA of standardised growth increments in 1997/1998. Variation is partitioned into that due to the day on which measurements were taken (Day), and that due to the chick measured (Chick).

In 1998/1999, change in nestling mass was still significantly correlated with maximum daily temperature (Fig. 7.5). Unlike 1997/1998, however, brood size was a positive, rather than negative predictor of mass growth rate. This may be because the 1998/1999 data included only chicks up to and including 9 days old, whereas the 1997/1999 data included chicks close to fledging. Brood size may only become an

important negative factor in mass growth when the chicks are at a more advanced stage of development (see also Fig. 7.6 below for evidence of this in 1997/1998).

Rainfall was a significant direct predictor of mass growth rate in 1998/1999. Nestlings growing from mid October to January experienced 9 days of relatively heavy rain, in contrast to the drought conditions in the previous season. All weather variables were strongly correlated with aerial insect abundance, especially rainfall. Aerial nsect abundance, in turn, was a significant predictor of mass growth rate. Indirect effects may be estimated by multiplying path coefficients. This gives an indirect path coefficient of -0.16 for the affect of rainfall on growth through its effect on insect abundance.

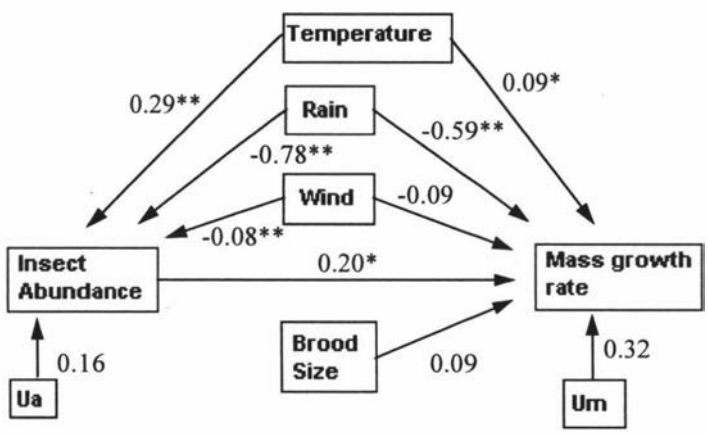

Fig. 7.5 Effects of environmental variables on mass growth rate in 1998/1999. Conventions are as for Figs. 7.1 and 7.4.

Brood Swapping Experiment

Chicks in enlarged broods grew more slowly in mass than chicks in reduced broods (1-tailed two sample t-test, P = 0.004, Table 7.3), and attained a lower peak mass (P = 0.007). Neither wing growth rate, nor the timing of feather emergence, were significantly different between enlarged and reduced broods.

The effect of brood size on mass growth rate is greater for older chicks than for younger, smaller chicks. There was no difference between mass growth rates in enlarged and reduced broods until chicks reached the ninth day of nesting (Fig. 7.6). The growth rate of chicks in enlarged broods began to decline at 9-10 days, whereas the growth rate of chicks in reduced broods remained high for a further 2-3 days. Wing

growth rate remained similar in enlarged and reduced broods in older as well as younger chicks.

Nest	Initial brood size	Expt. brood size	Mass growth rate	Peak mass	Wing growth rate	Feather emergence
Enlarged broods						
Hammond	3	4	1.66	17.5	4.8	7.0
Nic-Raup	4	5	1.72	17.3	4.9	6.4
Carrick2	3	4	1.65	17.5	5.0	6.5
Raup2	3	4	1.76	17.6	5.1	6.5
Trotter	3	4	1.65	17.8	5.0	6.5
Swmp2	2	4	1.61	17.6	5.2	6.5
Raup3	4	5	1.74	17.7	4.9	6.2
Mean	3.1	4.3	1.7	17.6	5.0	6.5
sd	0.7	0.5	0.06	0.16	0.13	0.24
Reduced broods						
Carrick	3	2	1.77	18.5	5.2	7.0
Twfd	3	2	2.02	20.5	5.1	7.0
Swmp	5	2	1.81	18.2	4.8	7.0
Carrick3	4	1	1.80	18.5	4.9	6.0
Mean	3.75	1.75	1.85	18.9	5.0	6.75
sd	0.96	0.50	0.11	1.06	0.18	0.50

Table 7.3. The effect on growth parameters for nestlings by artificial brood enlargement. Nest = road where the nest was located, Initial brood size = the size of the brood before transferring chicks, Experimental brood size = the size of the brood after transferring chicks, Mass growth rate = the brood mean of linear regression coefficients for individual chick mass over time from days 4-12, Peak mass = the brood mean of the peak masses(g) attained by individual chicks, Wing growth rate = the brood mean of linear regression coefficients for individual chick wing-length from days 6-16; Feather emerge, the mean age of the emergence of the first primary wing feather.

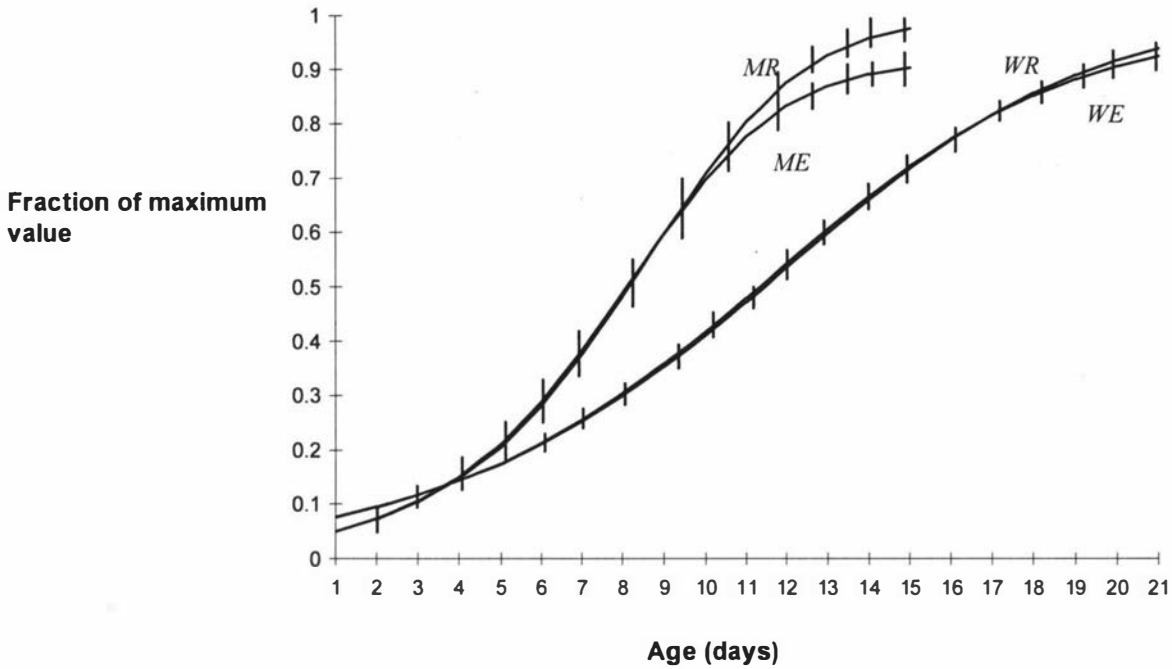

Fig. 7.6 Growth of mean mass and wing-length in enlarged and reduced broods. Curves are logistic equations fitted to pooled data. Vertical lines are standard deviations for individuals of a given age in enlarged and reduced broods. Mass growth is only plotted to day 15 because at day 16 mass starts to decline toward fledging mass (0.75 of maximum mass). Mass and wing-lengths have been divided by the asymptotic mass and wing-length for small broods in order to compare mass and wing-length growth on the same scale. *MR* = mean mass in reduced broods; *ME* = mean mass in enlarged broods, *WR* = mean wing-length in reduced broods, *WE* = mean wing-length in enlarged broods.

This result supports the lean-priority model, and falsifies the other two growth models. Wing growth is not affected by brood size to the same extent as mass growth, which suggests that wings are a priority in the growth of swallow chicks.

Deprivation Experiment

Mass growth rate was lower for treatment chicks than control chicks for each of the 19 broods (paired sample t-test, $t_{18} = 16.8$, $P < 0.0001$, Table 7.4). In contrast, there was no significant difference in wing growth rate between treatment and control chicks (paired sample t-test, $t_{18} = 1.0$, $P = 0.32$). The ratio of the mass growth rate of treatment chicks to the mass growth rate of control chicks, was less than the similar ratio for

wing growth rate - i.e., $\frac{TM}{CM}$ was less than $\frac{TW}{CW}$ in each brood (paired sample t-test, t_{18} = 17.4, $P < 0.0001$). Because the same nest sites were reused by successive broods, I calculated the mean ratios, $\frac{TM}{CM}$ and $\frac{TW}{CW}$, for each nest site (Table 7.5) - i.e., using nests rather than broods as replicates. The result was highly significant (paired sample t-test, $t_7 = 15.3$, $P < 0.0001$).

The mean amount of weight lost by 5 chicks over 6.5 hours in November 1999 was 0.84g (sd, 0.13). Over the same period, chicks produced a mean of 0.79g of faeces (sd, 0.11) (paired sample t-test, $t_4 = 1.8$, $P = 0.14$). I concluded that most of the weight lost during deprivation was due to defecation, and that mass lost due to metabolism during should not confound the experiment.

Chicks briefly deprived of food continue to grow wings at a normal rate but undergo a reduction in mass growth rate. This result corroborates the prediction of the lean-priority model (Table 7.1) and is inconsistent with the uniform distribution and fat-priority model

Brood	*TM*	*CM*	$\frac{TM}{CM}$	*TW*	*CW*	$\frac{TW}{CW}$
Raup1	0.18	0.41	0.45	0.52	0.60	0.87
Raup2	0.14	0.56	0.25	0.73	0.66	1.1
Raup3	0.17	0.51	0.33	0.63	0.61	1.03
Nic-Raup1	0.07	0.39	0.19	0.48	0.55	0.88
Nic-Raup2	0.22	0.43	0.5	0.6	0.62	0.97
Twyf1	0.1	0.26	0.4	0.66	0.56	1.17
Twyf2	0.13	0.38	0.33	0.54	0.6	0.9
Trotter1	0.11	0.46	0.24	0.74	0.67	1.1
Trotter2	0.07	0.28	0.25	0.57	0.61	0.94
Carrick1	0.06	0.46	0.14	0.66	0.69	0.95
Carrick2	0.17	0.45	0.37	0.55	0.56	0.98
Carrick3	0.13	0.48	0.28	0.53	0.68	0.78
Hammond1	0.23	0.53	0.44	0.57	0.72	0.79
Hammond2	0.22	0.43	0.5	0.65	0.74	0.88
Swamp.1.1	0.11	0.28	0.41	0.81	0.69	1.18
Swamp.1.2	0.21	0.48	0.43	0.56	0.57	0.99
Swamp.2.1	0.17	0.45	0.37	0.72	0.67	1.07
Swamp.2.2	0.14	0.46	0.31	0.56	0.68	0.83
Swamp.2.3	0.18	0.51	0.35	0.59	0.55	1.07
Mean	0.15	0.43	0.34	0.61	0.63	0.97
sd	0.05	0.08	0.1	0.09	0.06	0.12

Table 7.4 Growth increments for mass and wing-length in treatment and control chicks in 19 broods. Treatment chicks (C) were deprived of food for 6.5 hours. *TM* = percentage gain in mass in treatment chicks over 48 hours, *CM* = percentage gain in mass in control chicks over 48 hours, *TW* = percentage gain in wing-length in treatment chicks over 48 hours, *CW* = percentage gain in wing-length in control chicks over 48 hours. The experimental design is shown in Fig. 7.3.

Nest site	$\frac{TM}{CM}$	$\frac{TW}{CW}$
Raup.	0.34	1.00
Nic-Raup.	0.35	0.93
Twyf.	0.37	1.04
Trotter	0.25	1.02
Carrick	0.26	0.90
Hammond	0.47	0.84
Swmp1	0.42	1.09
Swmp2	0.34	0.99
Mean	0.35	0.98
sd	0.07	0.08

Table 7.5 Growth increments for mass and wing-length in treatment and control chicks in 8 nest sites. Conventions are as for Table 7.4.

Discussion

Welcome Swallow chicks are able to fast for at least 6.5 hours without a significant effect on wing growth. This suggests that Welcome Swallows prioritise the growth of the wings, yet the wings grow slowly. It also gains a lot of mass, well above fledging mass, during nesting. It loses some of this mass in bad weather, and when food deprivation is induced, but grows to fledging without apparent ill effect. By contrast, many other species of bird of the same size as the Welcome Swallow may store enough fat only for overnight survival (Lima 1986). This pattern of growth can be explained by the lean-priority model.

These results contrast with similar experiments done by Congdon (1990) on the Black Noddy (*Anous minitus*). Chicks of the Black Noddy continue to gain mass at a fast rate in enlarged broods, but have a reduced wing-length growth rate. These experimental results are better explained by the fat-priority model

Hulsman and Smith (1988) and Congdon (1990) argued that the Black Noddy not only prioritises the growth of body mass, but also constrains wing growth rate to a maximum. These are two distinct strategies, however, corresponding to the fat-priority and the lean-priority models respectively. This is not to say that some mixed strategy may occur, but this need not be the case.

If the chicks of the Black Noddy and the Welcome Swallow both accumulate fat in order to survive in variable environments, the question arises as to why they should do so by different methods. One possibility is that the fat-priority model is able to build up an adipose buffer relatively quickly, hence chicks may be able to survive in conditions where the food supply is frequently rising and falling. The lean-priority model on the other hand, accumulates fat more slowly. This may be a sufficient strategy for environments where there are relatively long periods of good food supply, interspersed with infrequent food shortages.

The Welcome Swallow may fit this pattern. The weather seems to be the key factor affecting the Welcome Swallows' food supply. In the warm temperate climate, there are long mild periods during the summer with infrequent spells of poor weather and low aerial insect abundance. The lean priority model allows reasonably rapid growth, but diverts enough energy from growth to support an adipose buffer against periods of poor food supply.

One problem, however, is that the population used in this study is not in its ancestral environment. The variability of the food supply in the Welcome Swallows' ancestral environment may differ from that in the study environment. However, this only affects the assumptions made in constructing the growth models, not in testing them. The models are about chick physiology, which can be assumed to be largely the same as in the ancestral population. The system model is more problematic, though the description of the Welcome Swallow habitat in Australia by Marchant and Fullagar (1982) is very similar to that of the Heretaunga Plains population.

It is not just the frequency of food shortages that is important, but also their degree and duration. The Swift is also vulnerable to rain, but its chicks put on far more fat and grow much more slowly relative to their mass than the Welcome Swallow. In Appendix 7, I argue that the Swift prioritises fat and restricts the growth of wings when it is in poor condition. This contradicts the conclusion of Lack (1954), who thought that the Swift maintained feather growth at the expense of mass. The Swift forages in a niche that is more vulnerable to rain than are swallows, because it forages high in the air column. In comparison, swallows feed much closer to the ground and to cover. Swallows are therefore able to forage in areas that provide shelter for aerial insects even in bad weather (Lack and Lack 1951). The Swift chick must be able to sustain an adipose buffer much larger than that of the swallow. The Welcome Swallow may only

have slow *wing* growth rather than slow lean tissue growth in general, a "minimal" version of the lean-priority model. This may be a strategy for the accumulation of an adipose buffer to cope with a moderately variable food supply. By contrast the extremely slow development of chicks of the swifts and Procellariiformes can not be explained in this way. To explain their growth patterns, a general reduction in lean tissue growth rates in accordance with the cell allocation model or the fat-priority model seems necessary. These models allow for the accumulation of large amounts of body fat, which may be necessary in environments where the food supply declines precipitously and frequently.

One possible advantage of the lean-priority model is given by Newton (1968), who argues that it is important for aerial feeders to avoid the development of fault bars in their flight feathers. Fault bars occur in the rectrices of young birds that have sporadically insufficient food supplies during feather growth. During poor nutritional conditions, there may be insufficient energy or nutrients available for the adequate deposition of keratin (Grubb 1989). By maintaining feather growth in lean periods at the expense of body mass, fault bars may be avoided. For this to be correct, all aerial insectivores should show this pattern of growth. Whether this is true of the Swift, however, is debatable (see Chapter 6 and Appendix 7).

The lean-priority model could also help explain the results of Murphy and King (1991), who studied the affect of food deprivation on wing growth in doves. They observed that the rate of feather growth was not affected until the third or fourth day of deprivation, after the onset of total starvation. However, this also suggests that body reserves might supply energy and nutrients for wing growth during fasts in some birds (Murphy 1992). To take into account this possibility, the lean-priority model would have to be modified to allow for the movement of energy and nutrients from bodily reserves into wing growth.

Chapter 8

Facultative and obligate strategies for growth in environments with a variable food supply

Introduction

Most of the possible adaptations to environmental variability conjectured in previous chapters are obligate adaptations. In Appendix 7 I consider the possibility that prioritisation of body components may be facultatively adjusted during development depending on conditions. In this chapter I examine this idea in more detail, and also consider the idea that parents will regulate provisioning as a facultative adaptation to environmental variability.

In previous chapters variability in food supply was treated as a unified and simple concept. However, food supply may vary in different ways. Environmental variability can be classed into relatively predictable variability, and relatively unpredictable variability (Plotkin 1994, Fig. 8.1). The predictability of variability is important when considering facultative adjustments in growth strategies or parental provisioning.

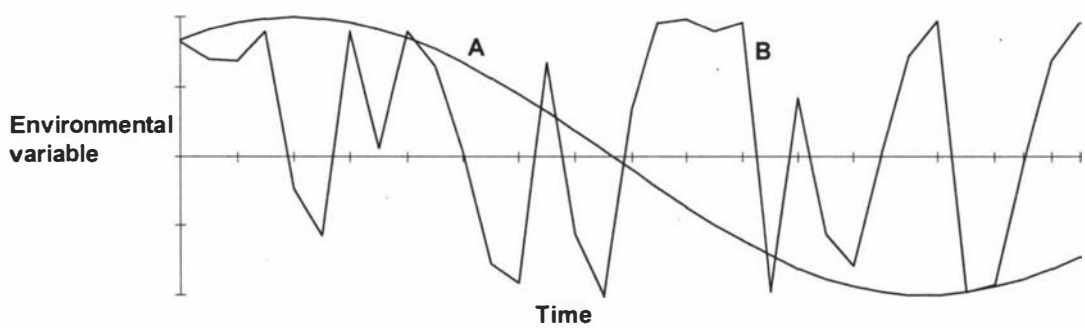

Fig. 8.1 Predictable and unpredictable environmental variance.
A; An environmental character is highly variable over time, but change is gradual and predictable, B; the same character in another environment is similarly variable, but changes unpredictably and relatively rapidly.

Factors that change predictably are highly auto-correlated over the short to medium-term. The relevant time scales are relative to potential response rates, and so

cannot be defined in absolute terms. Because change is gradual, organisms may be able to adjust their responses by using statistically informative environmental cues (Stephens 1989). Factors that change unpredictably are auto-correlated only over the very short-term. Environmental cues will be less informative and will not facilitate facultative adjustments to proximate conditions.

If informative cues are available - i.e., if the cues are correlated with the (near) future state of the environment - then organisms may use them to adjust their responses if the costs involved in reading the cues and in making the adjustments are not too great. As the amount of information in the cues increases, the strength of the relationship between cue and response should also increase (Berrigan and Seger 1998). However, if only poor information is available (i.e., if change is unpredictable over the short term) then organisms should follow an obligate strategy (Berrigan and Seger 1998).

As the cues organisms use to determine their responses become less reliable, their investment in characters correlated with these cues should evolve towards a fixed value, one that maximises average fitness in the face of uncertainty. This is not bet-hedging in the strong sense (which involves the production of variable offspring in the face of uncertainty, Seger and Brockmann 1987, Frank and Slatkin 1990), hence I will refer to this type of strategy as an (obligate) "insurance strategy" (Fig. 8.2). As cues become *more* reliable, characters may be adjusted to some element of predictable change in the relatively short term. I refer to this type of strategy as a (facultative) "tracking strategy" (Plotkin 1994).

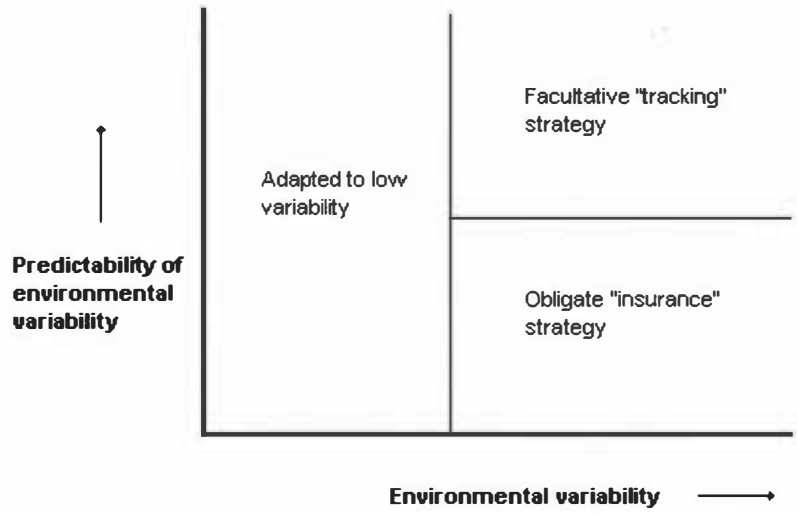

Fig. 8.2 Adaptive strategies in a variable environment

Two tracking strategies; facultative growth prioritisation model and regulation of provisioning

I will consider two models of nesting ecology where tracking of environmental factors may evolve (summarised in Table 8.1). First, I develop a model that predicts the conditions under which the facultative adjustment of growth priorities to proximate conditions should evolve. Second, I develop a model that predicts the conditions under which regulation of provisioning by parents should evolve. Growth and provisioning are physiological and behavioural aspects of nesting ecology respectively. I assumed that the general considerations discussed above apply equally to both. However, behavioural responses are far faster than physiological responses, hence facultative behavioural adjustments should generally be able to respond to faster rates of environmental change than physiological adjustments (Plotkin 1994).

Facultative growth prioritisation model

As discussed in earlier chapters, growing chicks may avoid starvation in environments with a variable food supply by accumulating fat. They may do this either by prioritisation of fat deposition, and/or, by the reduction in lean tissue growth rate. Chicks may benefit by consistently prioritising fat or lean tissue (i.e., an "insurance strategy"), or by a strategy of adjusting their growth priorities depending upon body condition (i.e., a "tracking strategy"). For example, Great Tits (*Parus major*) may

regulate their weights by comparing their average weight over the previous few days to an optimal weight (Bednekoff and Krebs 1995).

A chick following the insurance strategy will deposit fat during good periods and draw upon it during poor periods. A chick following the tracking strategy will only prioritise the fat deposition when food supply becomes poor and intermittent and, hence, it loses body condition (see also the discussion of "adaptive hypolipergenesis" in Chapter 5). It will only be beneficial to slow the growth of lean tissue if these conditions of declining food supply are likely to continue - i.e., food supply is auto-correlated over the short to medium-term. If food supply changes unpredictably, then a change in growth priorities in response to food supply will not increase chick survival. In such a case the insurance strategy should evolve.

A chick can obtain information on food supply from the provisioning it receives from its parent(s). When a parent returns with a feed to the chick, it provides information on (i) the amount of food it was able to supply in the recent bout of feeding, and (ii) the amount of time that was required to provide a feed of this size. These allow an estimate of the amount of food obtainable per unit time during the bout. Also, if the chick is able to compare the most recent feeding bouts with previous bouts, it may be able to estimate whether the food supply is trending upward or downward, and if so, at what rate. If food supply is trending downward, it will be beneficial for the chick to increase the priority put on fat deposition. A problem the chick then faces is depositing fat at a sufficiently fast rate to survive the coming lean spell. The rate at which the food supply changes must be relatively low compared to the rate at which the chick can respond if the chick is to benefit from a tracking strategy.

Regulation of provisioning model

Parent birds may respond to loss of body condition in their chick(s) by increasing provisioning effort. (e.g., in inshore-foraging seabirds, reviewed in Ydenberg and Bertram, 1989). Regulation of provisioning is an environmental tracking strategy in the sense discussed above

Various authors have argued that some species of Procellariiformes do not respond to increased food demand at the nest (Rice and Kenyon 1962, Prince and Ricklefs 1981, Ricklefs 1987, Hamer and Hill 1993). This may be due to a constraint of their foraging niche (Ricklefs *et al* 1987, Ricklefs 1992). This means that if the chick loses body condition, the parent will be unable to increased provisioning effort to

increase the chance of the chick regaining body condition. Instead, these species may chronically overfeed the chicks according to an intrinsic feeding rhythm (Ricklefs 1990). Natural fluctuations in provisioning success may be buffered by keeping the mean provisioning success above the chicks requirements - i.e., an "insurance strategy".

The problems faced by a parent attempting to regulate provisioning are similar to those of a chick regulating its growth priorities. Both must track changes in the food supply and respond quickly enough to increase the probability of the chick surviving food shortages. To regulate provisioning, the parent must be able to predict the body condition of the chick(s) at its next feeding. This will not only depend on current body condition, but also on the amount of food fed to the chick in the current feeding, the likely amount of time it will take to gather food and return for another feeding, the amount of food delivered in the next feeding, and in the case of bi-parental care the provisioning success of the other parent. One possibility is that the most informative predictor of future body condition is current body condition. However, regulation of provisioning causes changes in chick body condition proportional to the degree to which body condition differs from neutral body condition (where mass equals expected mass). Therefore, regulation should *reduce* the auto-correlation (hence reduced predictability) in body condition, hence the regulation of provisioning model does not predict a positive correlation between the degree of regulation of provisioning and predictability of chick body condition.

Another prediction of the regulation of provisioning model is due to Shea and Ricklefs (1985), who argue that pelagic species that feed their chicks more than once a day regulate provisioning, whereas those that feed chicks at longer intervals do not. If the feeding interval is relatively long and the chick fluctuates in body condition relatively quickly, then parents should not be able to match provisioning to the body condition of the chick.

Model	Predictions
Facultative growth prioritisation	Predictability of the food supply is correlated with the degree to which growth priorities are facultatively adjusted to chick body condition.
Regulation of provisioning	Predictability of chick body condition is negatively correlated with the degree to which parents facultatively adjust provisioning in response to chick body condition (i.e., degree to which they regulate provisioning). Degree of regulation of provisioning is negatively correlated with average feeding interval.

Table 8.1 Models of evolution of facultative and obligate adaptations to a variable food supply. Facultatively adjustable growth priorities (of fat and lean tissue) and parental regulation of provisioning should evolve in environments with a predictably variable food supply and with predictably variable chick body condition respectively.

Methods

This section consists of two sections. The first consists of methods for testing the facultative growth prioritisation model and the second consists of methods for testing the regulation of provisioning model. Both sections contain methods for estimating the parameters relevant to the models. In both cases, parameters were derived from individual chick growth curves (both mass and wings) for a number of chicks which I pooled to get species values for interspecific comparisons. Parameters and the method of calculation for each are summarised in Table 8.2.

Facultative growth prioritisation model

To test the facultative growth prioritisation model, we need to test the prediction that the predictability of the food supply is correlated with the degree to which growth priorities are facultatively adjusted to body condition. Therefore, we need to *measure* the predictability of the food supply and the degree to which growth priorities are facultatively adjusted to body condition.

Measuring predictability of the food supply

I estimated food supply from increases in the mass of growing chicks. The amount of mass that a chick puts on in response to a good supply of food varies with its stage of development. Hence, I used mass growth increments standardised for age

(discussed in Chapter 7). Standardised mass increments (*SMI*) varies above and below zero, with zero being the average amount of growth for a chick of a given age.

Chicks lose mass between bouts of feeding due to defecation and metabolism. If the chicks are weighed more frequently than they are fed, then the growth curve will erroneously show short periods of declining food supply. Hence, I replaced the standardised growth increments, with the moving average of standardised growth increments (Bednekoff and Krebs 1995, Boersma and Parrish 1998). I then calculated the moving average for each day for a chick using data from the period starting on the current day and including the number of subsequent days equal to the average feeding interval. I used this moving average of *SMI* as an index of the (changing) food supply.

I then calculated the correlation of these data with the same data lagged by a period of 1 day more than the average feeding interval (1 day in species with an average feeding interval of less than 1 day). A strong correlation means that proximate food supply is relatively stable, and hence may be responded to by the chick with a shift in growth priorities. I chose this particular time lag for several reasons. First, I assumed that if food supply was not a good cue (predictor) for food supply over this relatively short period, then it would be unlikely to be a good cue for subsequent days. Second, moving averages will necessarily be auto-correlated for a lag less than this. Third, I assumed that auto-correlations at shorter time scales would not indicate the stability of conditions necessary for facultative growth prioritisation to be effective.

Measuring the degree to which growth priorities are facultatively adjusted to body condition

Chicks of species with a facultative growth prioritisation strategy will show a positive correlation between body condition (*Cd*) and wing-length growth rate (using wing-length growth rate as a proxy for lean tissue growth rate, see Chapter 3). For a number of chicks in each species I calculated body condition for each day using an index derived from one used by Hamer & Hill (1993);

Equation 8.1 $$Cd = \frac{M - M_{exp}}{M_{exp}}$$

where M is mass and M_{exp} is expected mass.

M_{exp} is the mass expected for an average chick of a species given some degree of lean tissue development, hence the greater the amount by which M exceeds M_{exp} the better chick body condition. To calculate M_{exp} I used Equation 8.2. This estimates expected (average) mass (M_{exp}) for given wing-lengths. It does this by calculating expected age for a given wing-length (wl) and then expected mass from expected age. To obtain Equation 8.2 I substituted logistic equations that relate wing-length to age into logistic equations that relate mass to age. I calculated the parameters of the logistic equations for wing-length and mass growth by fitting best bit logistic equations to - growth data (discussed in Chapter 2).

Equation 8.2

$$M_{exp} = \frac{Am}{1 + \exp\{Om - (\frac{Owl - \ln(\frac{Awl}{wl} - 1)}{kwl}).km\}}$$

where Am and Awl are adult mass and wing-length respectively; Om and Owl are constants defining the position of the logistic curves for mass and wing-length relative to the origin; km and kwl are mass and wing-length growth rate.

I pooled data for a number of chicks within a species and calculated the correlation (r^2) between body condition and wing-length growth increments standardised for age (discussed in Chapter 7). Standardised wing-length increment ($SWLI$) varies above and below zero, with zero being the average amount of growth for a chick of a given age. I used r^2 value as an index of the degree of facultative adjustment of growth priorities for the species.

To calculate the degree to which growth priorities are facultatively adjusted to body condition, I needed data from individual chick wing-length growth curves. Good data of this type were rare in the literature, hence I was only able to obtain good data for 5 species (listed in Table 6.3, Chapter 6). However, I also gathered information on the predictability of changes in the food supply for 15 species where I had good data on individual mass growth curves, each in a different genus, and in 5 different habitats. Because the species used in the comparisons are taxonomically diverse, phylogenetic dependence should be reduced.

Regulation of provisioning model

To test the regulation of provisioning_model, we need to test the prediction that the degree of regulation of provisioning is correlated with the predictability of chick body condition. Therefore, we need to *measure* the degree of regulation of and provisioning the predictability of chick body condition. I also compared the degree of regulation of provisioning with average feeding interval (*FI*) and variability in food supply (*V*). If Shea and Ricklefs (1986) are correct, then species with a feeding interval of greater than one day should not regulate. I included variability in food supply, because it is correlated with average feeding interval and may confound the test (regulation of provisioning may be difficult in a highly variable food supply). To calculate these indices, I used 15 species where I had good data on individual mass growth curves, as explained in the last section.

Degree of regulation of provisioning

Regulation of provisioning has been studied using a variety of mathematical methods (e.g., Ricklefs 1987, Hamer and Hill 1993, Shea and Ricklefs 1996). None of these methods are easily adapted to growth data extracted from the literature. My approach begins with the observation that a chick whose parents regulate provisioning will tend to return toward neutral body condition (where mass equals expected mass) more strongly the greater its deviation from neutral body condition. I measured this using correlation (r^2) between body condition and *subsequent change in body condition* taking the change as the difference in body condition over the mean feeding interval. A negative correlation indicates that the parents are regulating provisioning. Because good data on wing-length growth were often unavailable I estimated expected mass by using the chick's age, rather than wing-length.

However, there is a potential problems with this index. Given a similar sized meal, chicks in very good body condition will not be able to increase in condition to the same degree as chicks in poor body condition, and chicks above the asymptotic mass may tend toward the asymptotic mass irrespective of provisioning (e.g., in chicks in some species that undergo weight recession in the later stages of nest). This may reduce the power of the test by increasing the degree to which all species appear to be regulating provisioning, irrespective of whether they are actually regulating provisioning. To minimise these effects I calculated the degree of regulation of provisioning (i.e., the correlation of body condition with change in body condition)

using only data where body condition was equal to or below 50% of the maximum body condition. Also, in species where weight recession occurs prior to fledging, I only used data up until the commencement of weight recession. However, removing chicks in good condition from the calculation reduces the sample size, which also reduces the power of the test.

Predictability of body condition

I calculated the predictability of body condition using similar methods to those discussed above for food supply. Body condition will appear to fluctuate between feeds in birds with a long feeding interval. Hence, I calculated a moving average of body condition. For a number of chicks in each species, I calculated the moving average over a period of days equal to the average feeding interval. I then calculated the correlation of these data with the same data lagged by a period of 1 day more than the feeding interval, for similar reasons to that given above for predictability of the food supply.

Parameter	Method of calculation
Predictability of the food supply	Auto-correlation of the moving average of standardised mass increments lagged by 1 day more than the average feeding interval.
Degree to which growth priorities are facultatively adjusted to body condition	Correlation between body condition (measured as mass relative to expected mass) and standardised wing-length growth increments
Degree of regulation of provisioning	Correlation between body condition and subsequent change in body condition (over 1 day more than the average feeding interval) using chicks prior to weight recession and less than or equal to 50% of the maximum body condition.
Degree of predictability of chick body condition	Auto-correlation of the moving average of body condition lagged by 1 day more than the average feeding interval.

Table 8.2 Parameters used to test the models and methods for calculating them

Results

The results consist of two sections, the first consisting of results of tests of the facultative growth prioritisation model and the second of the results of tests of the regulation of provisioning model.

Facultative growth prioritisation model

The Swift (*Apus apus*) contradicts is the only species to show evidence of facultative adjustment of growth priorities. That is, of the 5 species tested, it is the only species with a significant positive correlation between body condition and wing-length growth rate (Fig. 8.3). This contradicts my hypothesis because the Swift has a highly variable but very unpredictable food supply (Fig. 8.4). By contrast, two other species (*Gygis alba* and *Puffinus ilherminieri*) with variable but relatively predictable food supplies showed no evidence of a facultative growth prioritisation strategy.

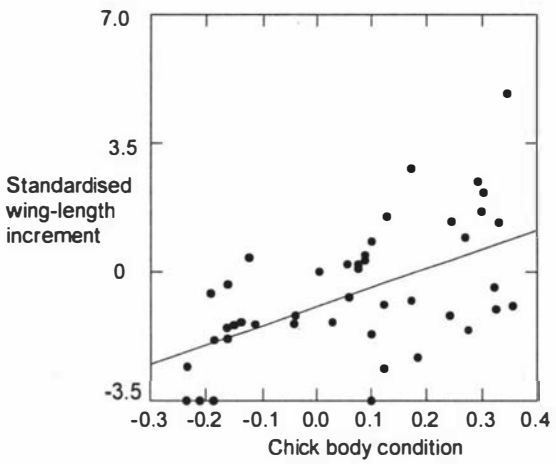

Fig. 8.3 Wing-length growth in relation to chick body condition in three Swift chicks (r^2 = 0.32, P = 0.0001). See text for calculation of body condition and Chapter 7 for calculation of standardised wing-length increments.

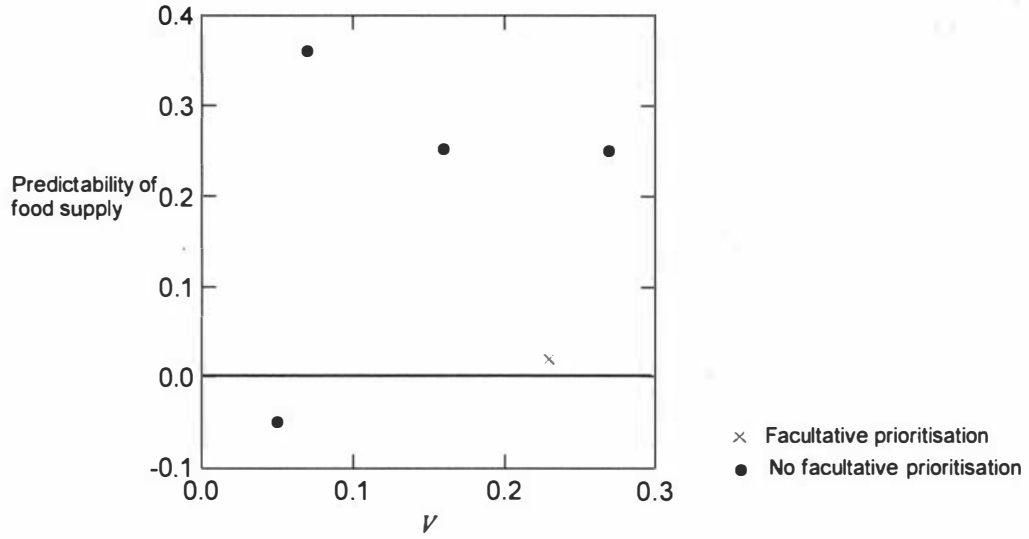

Fig. 8.4 Growth strategies of species in relation to variability and predictability in food supply. Individual points are species values. "Facultative prioritisation" means there is a positive correlation between body condition and wing-length growth rate for the species, whereas "no facultative prioritisation" means no such correlation exists. Refer to text for explanation of variables and Fig. 8.2 for the hypothetical distribution.

Compared to species in several other types of habitat, offshore-foraging seabirds had a relatively predictable food supply (Fig. 8.5). These species have a variable food supply (Chapter 2) but the amount of food delivered is more stable over the short to medium term than for inshore-foraging seabirds. This cannot be explained by the greater variance of growth increments in offshore-foraging seabirds. Using the species in all the habitats, variability in food supply (V) was not significantly correlated with predictability of the food supply. Average feeding interval (FI) was also not correlated with predictability of the food supply.

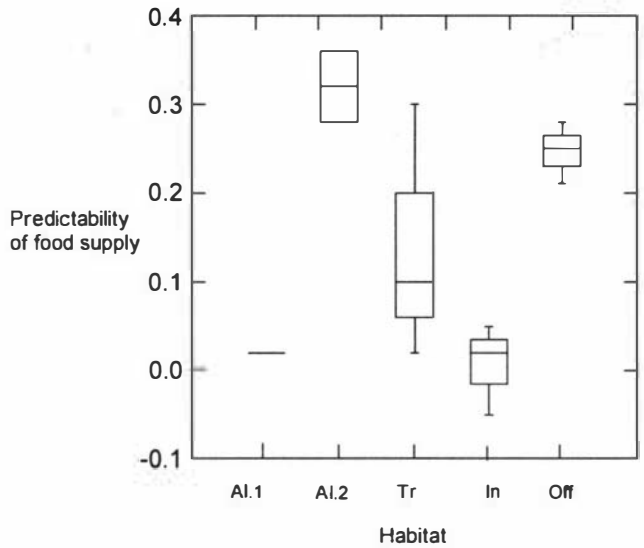

Fig. 8.5 Predictability of the food supply in five habitats.

(AI.1) high aerial insectivores (Swift), (AI.2) low aerial insectivores (Swallows), (Tr) terrestrial non-aerial insectivores, (In) inshore-foraging seabirds, (Off) offshore-foraging seabirds

Regulation of provisioning

Degree of regulation of provisioning was negatively correlated with predictability of body condition (Fig. 8.6A, Table 8.3). Regulation was not significantly negatively correlated with feeding interval (Fig. 8.6B). All species showed significant (negative) correlations between body condition and subsequent change in body condition. That is, all species tended to regulate chick body condition, including three species with a feeding interval greater than 1 day. However, regulation was negatively correlated with variability in food supply (Table 8.3). The lowest score on the regulation index was for the Swift (0.13), and this was the only species for which this score was not statistically significant.

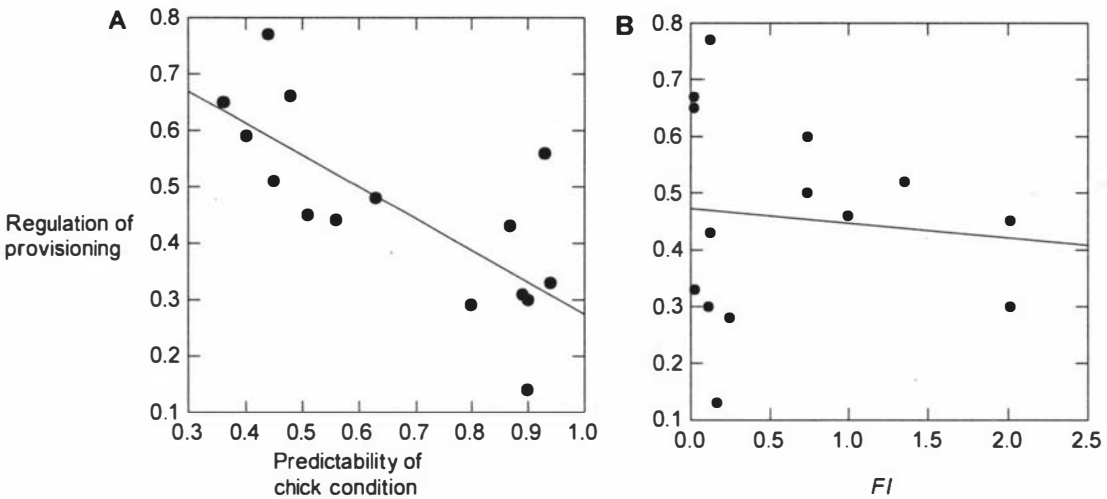

Fig. 8.6 The relationship between predictability of chick condition, average feeding interval, and regulation of provisioning. Regulation of provisioning is the correlation (r^2) between body condition and subsequent change in chick condition. (A) predictability of body condition is negatively correlated with regulation, (B) ln(FI) (natural log of feeding interval) is not significantly correlated with regulation. See text for explanations of parameters.

	r^2	Predictability of body condition	ln(V)
Degree of Regulation of Provisioning	0.73***	-0.70***	-0.44**

Table 8.3 Effect of predictability of body condition and variability in food supply (V) on degree of regulation of provisioning, testing using multiple regression analysis on species means. r^2 given for the proportion of variation explained by both independent variables. Predictability of body condition and variability in food supply show standardised partial regression coefficients. Statistical significance in the overall model and for the partial coefficients is indicated by asterisks: * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

Discussion

According to Berrigan and Seger (1998), increasing information should facilitate increasingly fine-grained facultative responses. The growth pattern and food

supply of the Swift contradicts this model when applied to the facultative growth prioritisation model. There are a number possible explanations for this.

First, the model may be false. One possibility is that different species of bird are able to respond with different speed in adjusting their growth priorities. Some species may be able to change growth priorities only very slowly and so be unable to respond to a worsening food supply. Also, if growth priorities in a species are primarily constrained by tissue allocation, then they will only be able to be slightly modified during development.

Second, the methods used may be insufficient to test the hypothesis. The assumptions underlying the calculations may be flawed - i.e., I may not be measuring the effects that I want to measure. Quantifying the predictability of the food supply is particularly problematic. Predictability of variability in food supply is a complex concept that is not easily captured by a single figure.

Third, the model and methods may be correct, but the database is insufficient to adequately test the model. The data requirements for testing these models are stringent. Therefore, I was only able to use data for a small number of species. Furthermore, the conditions that suit a facultative strategy may occur relatively rarely. It is possible that only the Swift chicks in my database were at sufficiently low body condition to exhibit retarded wing growth. Retarded wing-length growth has been reported by various authors (e.g., Grubb 1989, 1992) but without comparing this to body condition this cannot be taken as evidence of facultative growth prioritisation

Fourth, the hypothesis may be partially correct. The Swift chick differs from the other species with a variable food supply studied in that it has a relatively short feeding interval. Feeding rate may function as a sampling rate for tracking food supply. A facultative growth prioritisation strategy may require a short feeding interval relative to the rate of change in food supply.

Offshore-foraging seabirds had relatively predictable food supplies. This may mean that the variable food supply in these type of species (Chapter 2) is the result of long-term differences in the food supply between chicks (i.e., predictable conditions). These are the conditions under which I conjectured that a facultative strategy would evolve. However, it may also mean that variable growth in these species is due to infrequent declines in food supply that have a disproportionate effect on overall growth rate. These infrequent crashes in food supply could be unpredictable. Using my index,

however, these species would appear to have a predictable food supply because food supply is relatively stable over most of the nestling period. Hence, the predictability of the food supply may be overestimated for these species.

All species except the Swift appeared to regulate chick mass to some degree. Contrary to Shea and Ricklefs (1985), species that feed their chicks less than once a day regulated chick masses to a similar degree to species with shorter feeding intervals. However, the strength of regulation became weaker in species with highly environments with a variable food supply. This may be because, chick masses are able to vary to a greater degree in species with a lesser degree of regulation. That is, species that show a tendency to return toward expected mass are necessarily species where masses do not vary significantly from expected mass.

However, lack of regulation may also be the result of environmental constraints (Hamer and Hill 1993). Provisioning effort may only be a weak determinant of provisioning success beyond some minimum level of effort in species in environments with a variable food supply. Another possibility is that these birds have lost the behavioural characters required for regulation. Hence, lack of regulation may be environmental or genetic in origin. If the environmental explanation is true, then lack of regulation is non-adaptive and may be the result of an evolutionary time-lag. But if the genetic explanation is correct, then lack of regulation is an adaptive mode of feeding; an obligate constant provisioning effort. For Ricklefs (1990) theory of obligate overfeeding to be correct, lack of regulation must be an adaptation to an environment that restricts the ability of the parent to respond to poor body condition with an increased foraging effort.

Ricklefs (1990) argues that obligate overfeeding is an alternative to Lack's insurance hypothesis, and that variable provisioning success is due to lack of regulation rather than an intrinsically variable environment. However, obligate overfeeding is insufficient to account for the accumulation a large amount of fat during nesting. The chick must be physiologically capable of storing fat. Storage of large amounts of fat has no function unless it buffers the chick against a variable food supply (or acts as insulation). It is irrelevant whether this variability in food supply is due to temporal fluctuations in the environment, or by variable provisioning success by the parent.

Ricklefs (1992) has suggested that the regulation of provisioning may result in the negative auto-correlation of growth increments. If true, then my index of regulation

should be correlated with this figure. This would confound and complicate my conclusions, as it would mean that regulation by parents reduces the apparent predictability of the food supply for the chick (as measured by my index). However, I did not find a significant correlation between these figures. If my reasoning is correct, then the cue for regulation of feeding is body condition rather than previous meal size (which may not be a good predictor of body condition). This may explain why my conclusions for regulation of feeding in species with long feeding intervals differs from those of Ricklefs (discussed above).

It is also possible that the "regulation effect" that I have measured may not be due to parental behaviour. The chick may regulate its own intake of food so that if it receives a large meal during one interval or is in particularly good body condition, it may solicit or accept less food during the next interval (Shea and Ricklefs 1986). A chick in good condition may assimilate less energy or convert assimilated energy into lighter tissues than chicks in poor condition (Doug Armstrong Pers. Comm.). Also, the logical difficulties with my index that I discussed in the methods may not have been eliminated. Therefore, the tendency of body condition to return to a mean value may not imply any specific adaptations for regulation of masses.

Parental behaviour and chick growth physiology strategies, as well as predictability of the food supply and predictability of body condition, may interact in complex ways. For example, the body condition of Swift chicks is stable over long periods even though the food supply appears to vary unpredictably. The Swift has a relatively short feeding interval and a food supply that is highly variable. It seems to have facultatively adjustable growth priorities, little or no regulation of provisioning, an obligate slow maximum wing-length growth rate (Chapter 3), and the ability to accumulate a large amount of body fat. Therefore, the swift appears to have a combination of obligate and facultative strategies for survival in an environment with a variable food supply.

A further complication to the theory of the evolution of "tracking" and "insurance" strategies is that the time-scale of environmental change is relative to the rate at which biological processes are carried out in a species. This may determine in which direction - i.e., toward a insurance or tracking strategy - evolution will proceed (Fig. 8.7). An environment may change relatively quickly from the perspective of a large and/or slow developing organism. It may evolve an insurance strategy, whereas a

smaller species with a fast developmental rate may evolve a tracking strategy in the same environment. The auto-correlation index used in this study does not make these distinctions.

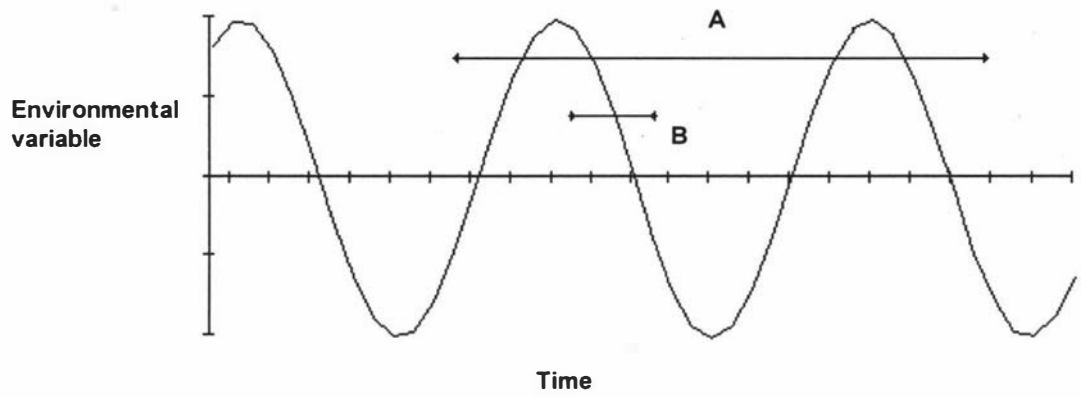

Fig. 8.7 Initial values and subsequent evolution in a variable environment.
A; a species has a long and slow development relative to the rate of change of the environment (e.g., from large size). It may an insurance strategy and a longer and slow development, B; a species has a short and fast development relative to the rate of change of the environment. I may evolve an a tracking strategy and/or faster development.

Chapter 9

The nature of adaptation and the adaptedness of slow growth in environments with a variable food supply

Introduction

Lack tried to explain what appears to be convergent evolution between swifts and Procellariiformes. To do this he advanced a conjecture about the adaptedness of slow growth in environments with a variable food supply - i.e., his insurance hypothesis. He argued that natural selection should favour slow growth in environments with a variable food supply because slow growth promotes chick survival during food shortages. In this chapter I aim to review the methods that I have used to test this hypothesis. In order to do this, I first review what is required for the demonstration of adaptation in general.

If species in environments with a variable food supply are growing according to Lack's insurance hypothesis (as I have argued in earlier chapters), then clearly slow growth is of *benefit* for chicks in these species. However, the demonstration of adaptation requires more than simply demonstrating that something is of benefit to an organism (Williams 1966). In this chapter, I will review a modern version of the argument from design and show how the argument from design is connected with the comparative approach to studying adaptation. I will then argue that the methods used in earlier chapters meet the requirements for demonstrating adaptation, and therefore that my results show that slow growth is an adaptation to environments with a variable food supply.

Methods for studying adaptation

Ridley (1996) argues that adaptation is a clear and objective concept in theory - i.e., a character is an adaptation if natural selection works against mutant alternatives. However, he argues that this criterion is difficult to apply in practice (and at times impossible). Furthermore, Harvey and Pagel (1991) and Williams (1997) argue that natural selection should not and need not be used in a definition of adaptation. Therefore, Ridley (1996) lists three main methods for studying adaptation (i) the

comparison of the (conjectured) functioning of a character with that of a mutant form, (ii) the comparison of the functioning of a character with that of an experimentally manipulated form, (iii) the comparison of species with respect to characters that vary between species in functioning and/or environment.

Each of these methods involves the comparison of some character with alternative forms. The last of the three methods is discussed in detail by Harvey and Pagel (1991) who not only consider comparisons between species, but also between phylogenetically independent units. There is another criterion of adaptation, however, that at first sight appears to be quite distinct from the comparative approach to adaptation. That is, the criterion of good design. Williams (1992, 1996) argues that adaptation is most rigorously demonstrated when some structure or process is shown to be conform to good design principles. Arguments for good design in biology date at least as far back as Aristotle, Galen, Hume and Paley (reviewed in Williams, 1986 1992). However, a standard set of conventions for testing hypotheses about good design does not seem to exist. I will attempt to solve this problem by arguing that good design is also comparative, and that design arguments for adaptation are comparative arguments. To do this, I will show the logical connection between comparative methods and design arguments. This will involve a discussion of design in biology, and the relationship of design to problem solving.

Design in biology

Various authors have argued that in order to demonstrate adaptation, it is necessary but not sufficient to show that the possession of some character is beneficial to the individuals of species that possess it. For a character to be identified as functional or adaptive it is also required that the character is shown to be "well designed for" or "good for" some particular task (Williams 1966, 1985, 1992, Dawkins 1986, John Maynard-Smith 1993, Dennett 1995). The use of the term "design" in biology has been criticised as anthropomorphic. However, the same can be said for much of the vocabulary of science (e.g., law, regulate, mechanism, work). *Design* in biology may have retained anthropomorphic connotations for longer than these other words in science, but these connotations have no relevance to modern evolutionary biology. All that is required for a concept to be used in science is that it be sufficiently

well understood to be used in the construction and testing of hypotheses (Popper 1959, 1983).

Optimality models have been one approach to studying design in biology. One problem with optimality models is that they are based on finding the best solution to a problem within a set of constraints. However, in evolutionary biology it is not always possible to specify what aspects of some structure or process have been constrained by available variation, and what aspects have been free to vary and be optimized by natural selection. However, if only minor quantitative variation is being considered, there is seldom any significant constraint (G.C. Williams Pers. Comm.).

Good designs are problem solvers

One criterion of good design is that a well designed structure or process solves a "problem" arising from the interaction of an organism with its environment (Popper 1979, Williams 1985, Dawkins 1996). Popper (e.g., 1976) has argued persuasively that problems are not merely subjective frustrations but pertain to objective "problem situations" that are fundamental to understanding all adaptive phenomena. Good designs are good solutions to problems, and problems are situations that arise from the interaction of organisms with their environment in the process of survival and reproduction.

A difficulty with the problem solving criterion of good design is that there does not seem to be any principled way of identifying problems independently of their solutions (D.C. Dennett Pers. Comm.). Popper (1976) argued that problems are failings in existing solutions to meet a certain degree of functioning. He goes on to argue that existing solutions solve previous problems, and that the previous problems are identified with even early solutions and so on. Hence, the idea that adaptations are solutions to problems is one that is if not completely circular then is at least deeply recursive. For the process (of problem solving) to start, the first solution and the first problem have to "arise together" (Popper 1976).

Solutions to problems are theories

One possibility for studying adaptations as problem solvers is by way of analogies with theories, which also solve problems. That is, if adapted structures and processes solve biological problems then they are analogous to articulated theories that

solve articulated problems. Hence, Popper (1976, 1979) refers to biological adaptations as "embodied theories". The problem that an *articulated* theory solves is that of explaining previously unexplained facts (i.e., explaining events that were unexpected with respect to previously held theories).[1] A theory that is close to the truth conforms to some regularity in the world. Similarly, an adapted organic form conforms to some regularity in the environment (Fisher 1958). Theories vary with respect to how much they say about the world - i.e., with respect to their *information content* (hence the degree of complexity of the natural process that the theory conforms to). If adaptations are closely analogous to theories, they should have a quality analogous to information content. Lorenz (1966) noted that;

"All the complicated structures and functions of the chromosomes...evolved in the service of the function of acquiring and storing information on the environment."

Similarly, Williams (1992) argues that DNA is a medium for carrying information. He argues that genes are best understood not as "material" entities, but as "codicial" entities (i.e., with respect to their information content). Campbell (1974) and Plotkin (1994) argue that articulated knowledge is directly analogous to adaptation. Monod (1970) calls the complexity that organisms convey to their offspring by reproduction their "invariance content", a quality directly analogous to information content. Dawkins (1998) argues that the information content of an organism is equivalent to its adaptive complexity.

The concept of information content has been formalised in Logic (Popper 1959), Communication Theory (Shannon and Weaver 1963), Computer Science (Chaitin 1990) and Hypothesis Testing (Burnham and Anderson 1998). I will argue below that both the formal concept of information content and the concept of adaptation are statistical concepts. I will therefore argue that information content is a comparative concept in the same way as adaptation (Popper 1976). I will therefore argue for a comparative theory of problems, solutions, and good designs.

[1] Articulated problems are, like all problems, relative to solutions/theories. The comparative theory of problems discussed in this chapter applies equally to both articulated, conscious problems and embodied theories (adaptations).

Formal ideas about information content have been used in the study of behavioural or sensory adaptations for some time (e.g., Attneave 1954, Barlow 1963). Arguments that behavioural outputs contain more information than can be coded for by environmental inputs (i.e., that perceptions or behaviour involve information processing) have come to be known as *Poverty of Stimulus Arguments*[2] (Fodor 1983, 1985). That is, perceptions involve inference, and behaviours have a degree of computational or algorithmic complexity (see below), both of which are equivalent to information content. The environmental inputs for the perception or behaviour cannot explain the information content of the subsequent processes. I will argue that this applies to adaptations in general, and not just those involving the nervous system.

The formal concept of information content

Theories may evaluated with respect to how good they are at solving problems concerning the explanation of facts. Popper (1959) shows that this "explanatory power" is inversely proportional to the "logical probability" of the theory. That is to say, a good theory excludes a large number of logical possibilities[3] and this is proportional to the specificity (i.e., precision) of the outcomes that the theory predicts and the universality of the initial conditions to which it applies.

In Popper's logic, logical probability is inversely proportional to information content - i.e., the greater the number of logical possibilities that a theory rules out, the more informative it is (Popper 1959). Therefore, explanatory power is proportional to information content.

Theories as algorithms

A theory with high information content "reduces" a large number of facts to an economical description. Indeterminate (hence random) processes generate series that

[2] A detailed and original *Poverty of Stimulus Argument* can be found on in Chapter 26 of Humphrey (1995).

[3] According to the "covering law theory of explanation" in order to explain a set of facts a theory must show that the facts can be deduced from a (set of) general law(s) and initial conditions. Therefore the general law(s) and initial conditions that make up the theory must logically specify the facts to be explained and by doing this exclude alternative possibilities. Hence, the greater the explanatory power of a theory the more it rules out and therefore the less logically probable it is (and as a consequence, the more testable it is).

cannot be reduced to a lawful (hence economical) description. Data generated by determinate (hence patterned or regular) processes are those that *can* be reduced to a lawful description. This idea that pattern in data is characterised by the "reducibility" of the data has been given a formal meaning in Information Theory. Solmonoff (1964) identified theories with algorithms. Algorithms generate strings as output just as theories produce predictions as "output". Patterned data are those that are produced by lawful processes and patterned strings of digits are those that can be produced by algorithms, hence patterned strings may contain repetition or redundancy that can be compressed by an algorithm (Dennett 1991). Hence, "reduction" in Logic corresponds to "compression" in Computer Science.

In Popper, the more specific and detailed the predictions a theory makes, the greater its content. In Information Theory the longer the shortest algorithm that can generate a string, the greater the strings content. A high content prediction in Popper's terms excludes many alternative logically possible outcomes, and a high content algorithm in Information Theoretic terms excludes a large number of alternative output strings. A high content theory in Popper's terms is *logically* improbable. The greater the content of a theory, the less likely *a priori* it is to conform to reality. An algorithm that generates an output with high information content in Information Theoretic terms is also improbable (remembering that a string cannot have more information than the shortest algorithm that can generate it) - i.e., it is unlikely that the output of such an algorithm will conform (i.e., correspond) to a string specified in advance. I will argue below that it is the improbability of "conforming" to some reference point that is a characteristic feature of an adaptation. That is, adaptations are improbable in the same way that theories with a high information content that conform to the facts are improbable.

The statistical nature of adaptations and their information content

I have discussed how the information content of a theory or algorithm (with respect to the theories predictions or the algorithms output) is by nature statistical. That is, information content corresponds to the number of alternative possibilities ruled out by an outcome. The greater the content of a theory, the less probable it is that the theory will conform to reality - i.e., a theory that conforms in many respects to reality is improbable. I will argue that the more complex an organic form, the less likely it is that

it will conform to a *comparatively* high value for some function (i.e., a high value compared to alternative forms, discussed in Fisher 1958, Dawkins 1986, 1996, Dennett 1995, Williams 1997). Hence, an adaptation is improbable with respect to its function in analogous fashion to a theories improbability with respect to its conformity to reality.

Williams (1992) argues that the fundamental comparison for studying adaptation is between an existing character and a variant on the character in the same environment. That is, if a character is *good for* a task, it is better for the task than are most variations on that character. Fisher (1958) argued that;

> ". . . the more complex the adaptation, the more numerous the different features of conformity, the more essentially adaptive the situation is recognized to be. An organism is regarded as adapted to a particular situation, or to the totality of situations which constitute its environment, only in so far as we can imagine an assemblage of slightly different situations, or environments, to which the animal would on the whole be less well adapted; and equally only in so far as we can imagine an assemblage of slightly different organic forms, which would be less well adapted to that environment."

Similarly, Williams (1997) argues;

> "There may be many possible designs [for a particular function]... but their number is infinitesimal compared to the number of designs that would not [be good for the function in question]..."

Dawkins (1986) makes the point most succinctly;

> ". . . however many ways there may be of being alive, it is certain that there are vastly more ways of being dead . . ."

The greater the number of parameters in a biological structure and the range of values over which they may vary, then the greater the number of possible alternative forms. The better the parameter values are for carrying out a function, the narrower the range of values these parameters may take. Therefore, if a biological structure or

process is well designed for some task a random change to one or more of its parameters has a high probability of reducing its capacity or propensity for that task (Fisher 1958, Plotkin 1997). Hence, Lorenz (1966) pointed out that:

> "The more complicated an adapted process, the less chance there is that a random change will improve its adaptedness."

Therefore, adaptations are not only unlikely to arise by chance, they are also unlikely to persist. As Williams (1996) notes, natural selection is required not only to explain the evolutionary origins of an adaptation, but also to explain the prevention of evolution away from an adapted state.

It is important that it is understood that adaptations are only improbable with respect to their functions, or that theories are only improbable with respect to conforming to reality - i.e., to keep in mind the *relational* aspect of adaptations and of knowledge (Plotkin 1994). As Dawkins (1986) notes, adaptations are improbable with respect to some quality that can be "specified in advance". It is not enough that some ensemble or configuration is improbable with hindsight, as this may apply to almost anything.

Chaitin (1990) defines a random string as one that is maximally incompressible, and that nearly all strings are highly random in this sense - i.e., randomly selected strings tend to have a high information content. Similarly, randomly selected observational data will have a high information content. What counts for theories is the *conformity* of some prediction to reality[4], the probability of which *decreases* with the theories information content. What counts for adaptations is the conformity of the adaptations function to a comparatively high value in comparison with alternative forms, the probability of which decreases with the complexity or information content of the form (i.e., with the number of possible forms excluded by the adaptation).

[4] In terms of hypothesis testing, this is equivalent to a model's Akaike Information Criterion (D. Armstrong Pers. Comm., Burnham and Anderson 1998).

The objectivity of improbability

I have argued that idea good design is characterised by comparison with alternative possibilities. That is, most variants on an adapted or well designed process or structure will carry out its function to a reduced degree, or with a reduced reliability. Hence, adaptations are improbable with respect to their function The objectivity of the *improbability* of adaptations depends upon the objectivity of probabilities and possibilities as such. The objectivity of possibilities and probabilities, and the relationship of probability theory to determinism, is discussed by Popper (1959, 1982a, 1982b, 1983) and Von Mises (1964). The distinctions between logical, physical, biological and historical possibilities are discussed by Dennett (1995).

Popper distinguishes between subjective and objective theories of probability (hence possibilities). The subjective theory is the only theory of probability that is compatible with strict determinism. If strict determinism is true then there are no physically possible variants for actual situations - i.e., "only what becomes actual was physically possible" (*actualism*). Hence, strict determinism means that possibilities are entirely subjective, the result of our ignorance of initial conditions. If our knowledge of initial conditions were to become complete, then we would see that there are no physical possibilities other than actualities.

If the subjective theory of probability (and possibility) is correct, then this means that good designs *qua* selections among alternative possibilities are also subjective. At best, good designs would be characterised by comparison with alternative *logical* possibilities. I contend that the subjective theory of probability is incompatible with the comparative study of good design. The reason for this is that we generally need to exclude from the set of "possible variants" those that are *physically* impossible.[5]

Objective theories of probability state that probabilities and possibilities are not the result of our ignorance of initial conditions, but that probabilistic processes are physically real. Popper's "propensity theory" (e.g., 1983, 1990) is an objective theory of probability. Propensities are weighted physical possibilities - i.e., objective probabilities. Propensities are properties of physical situations, ranging in value from

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[5] For instance, is the sun well designed for warming the earth (Williams 1996)? Compared with some physical impossibilities (e.g., replace the sun with the moon) it is. Also, we need to distinguish mere effects that can be explained by physics, from well designed outcomes (Williams 1966, Ridley 1996).

zero (physically impossible) to one (completely determined). The propensity theory of probability treats good designs as objectively improbable. It also refines the idea of comparison with alternative possibilities by giving the alternative possibilities weights. A complete analysis of the probability of an adaptation would take into account this set of weighted possibilities (propensities). However, we needn't go so far as this, but may ignore very low propensities and only consider "possible variants" that have a propensity (before selection) that is similar (relative to prior conditions) to that of the existing character.[6] Hence, the comparative criterion of good design becomes very similar to the first of Ridley's methods (the comparison of adaptations with mutant forms).

The problem solving power of adaptations and theories

The purpose of my discussion of the information content of an adaptation is to show that an adapted characters conformity to regularities in its environment (both the internal and external environment) with respect to comparatively high value for its function, is analogous to a theories conformity to regularities in nature. The purpose of showing this analogy is to show how adaptations can be considered as solutions to problems. Therefore, we are now in a position to discuss the problem solving power of a theory, hence the problem solving power of an adaptation.

The problem solving power of a theory

Popper (1976) argued that problems are logically equivalent to questions. A question asks for a selection (a solution) from a set of alternative possibilities. For example, "Is grass green?" is a question that asks us to select from two possibilities, and "What adjustments to this motor will improve its efficiency?" asks us to select from a much larger range of possibilities. In Information Theory, questions give binary choices - i.e., "1 or 0?". Hence, a binary string n digits in length answers n binary questions, and is a selection from a set of 2^n possibilities. Popper (1976) shows that the number of logical possibilities excluded by a theory corresponds to the number of questions answered (i.e., selections made). He refers to this set as the "problem

[6] This may seem like a conservative a criterion (excluding alternative historical legacies for example) but if "prior conditions" is defined as "the early universe" or at least "at the origin of life" then the set of possible variants may become very large indeed.

content" of a theory. That is, the information content of a theory directly corresponds to the number of questions that it answers (the number of possibilities that it selects from). I will call this the theories "problem solving power". Hence, the problem solving power (i.e., problem content) of an articulated theory is proportional to its information content and its explanatory power. This makes more precise the assertion that I made earlier that articulated theories solve problems pertaining to the explanation of facts.

Theories are improbable with respect to the conformity of their information content to reality. Therefore, theories are improbable with respect to their problem content - i.e., how good they are at solving the problem of explaining some set of facts. Good theories are *comparatively well designed* for solving a problem of explaining some facts. Answers to questions (hence solutions to problems) are selections from sets of alternative possibilities in the same way that good design is a selection from a set of possibilities (Table 9.1). Hence, good design is comparative.

The problem solving power of an adaptation

The comparative criterion of good design may be applied to the analysis of biological problems. That is, a random variation in the parameters of an adapted process is likely to cause a reduction (i.e., a problem) in functioning. The differences between the existing adaptation and effects of variation (that cause a reduction in functioning) are problems for the variants but ones that the existing adaptation solves. That is to say, "problem" and "solution" are comparative concepts as are all corollaries of *good design*. The problems that an adaptation solves exist only in comparison with possible variants in the same way that an adaptation is only *good for* some function in comparison with possible variants. Thus there is no need to identify problems independently of their solutions because problems are characterised with respect to the same set of comparisons as their solutions (hence, to functions or good designs).

It follows from this that the number and extent of the problems that a structure or process solves is inversely proportional to the how probable it is. I conjecture that each problem solved - i.e., each damaging effect of a variant avoided, corresponds to a question answered, or a "design feature."[7]

Therefore, problems are corollaries of good design, and a criterion of good design can also be used as a criterion with which to identify problems. The statements (i) the eye is adapted for sight, (ii) the function of the eye is sight, (iii) the eye is well designed for sight, and (iv) the eye solves the problem of sight, state the same idea in different ways. Therefore, adaptations are embodied theories that solve problems. Furthermore, both articulated and embodied theories evolve by a process of variation and selection (Popper 1963).

An adaptation		**A successful theory**
is complex configuration of parameters, and		has a high a explanatory power, and
	THEREFORE	
is a selection from a large set of possibilities,		is a selection from a large set of possibilities,
	AND	
conforms to a comparatively high function value (or equivalently, conforms to regularities in its environment with respect to a high function value), and	THEREFORE	conforms to regularities in nature, and
is improbable with respect to its function,		is improbable with respect to its predictions,
	AND	
the number of possibilities excluded is equal to the number of bad variants avoided or questions answered (Problem solving power),		the number of possibilities excluded is equal to the number of questions answered (Problem content),
	AND	
this is proportional to the information content of the adaptation,		this is proportional to the information content of the theory,
	AND	
the negation of each bad variant (i.e., each variation that reduces the function value) avoided is a design feature.		the negation of each possibility excluded belongs to the truth content of the theory.

Table 9.1 Analogies between Adaptations and Theories

[7] In a situation that shows good design for some task generally (i.e., it has a high proportion of parameters with values that are better for the task than most variants) then parameter values that may vary without ill effect may be called "epiphenomena" or "spandrels" and those that may vary often with good effect "design flaws". Design flaws are clues to historical legacies (Williams 1992, 1996), i.e., functional analysis is unavoidable if we wish to find *constraints* on good design (Dennett 1995).

Two types of comparisons: problems solved and problems faced

When studying existing adaptations, we may compare an existing adaptation to variants that function more poorly and by doing this we identify problems that the existing adaptation solves. We may call these *retrospective* comparisons (Fig. 9.1). By contrast, when actively trying to solve problems with trial solutions we are aware of problems in the *existing* solution - i.e., the relevant comparison is between the existing solution and possible variants that function *better*. We may call these comparisons of existing solutions with variants with better *prospective*.

Prospective comparisons arise in problem solving and engineering. Awareness of problems in a prospective sense is the basis of *criticism*, and prospective comparisons irrespective of consciousness are the basis of *feedback*. Retrospective comparisons correspond to situational analysis (Popper 1976) and reverse engineering (Dennett 1995).

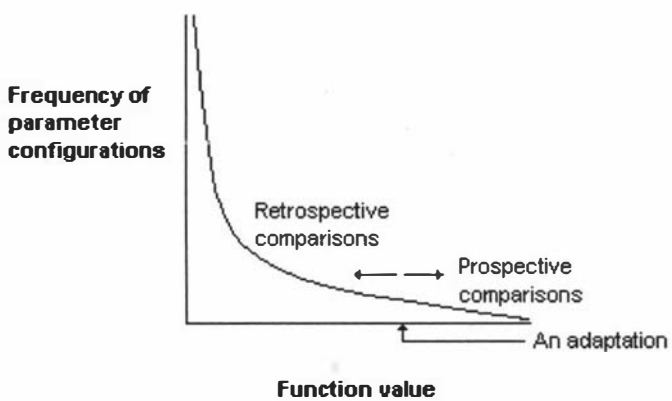


Fig. 9.1 Schematic representation of adaptations as comparatively good designs. Increasing function value means fewer alternative parameter configurations that function as well, hence configurations further to the right of the graph are increasingly improbable with respect to the function. That is, the relationships between the parameters of the situation become increasingly improbable with respect to the function - i.e., there is an increasingly improbable conformity between the parameters. The greater the number of parameters involved, the greater the fewer alternative configurations that will achieve such a function value and the greater the improbability. Good design is a matter of degree characterised with respect to other possible configurations. Likewise, problems that good designs solve are also matters of degree, characterised by the same set of comparisons - i.e., it becomes increasingly sensible to speak of good designs and problems as one moves to the right of the graph. Problems yet to be solved are characterised by prospective comparisons and problems already solved by retrospective comparisons.

How useful is it to characterise adaptations as having an information content?

If the approach to adaptation that I have discussed is accurate, it should help to solve basic problems in biology. Below, I sketch the value of this approach with respect to several fundamental problems.

Levels of analysis and explanation in biology

Information content can only be explained in two ways; (i) by showing how the information content in an output maps or codes to (i.e., is in a one-to-one correspondence with) some input of similar information content (e.g., genetic or cultural inheritance), or (ii) showing how the information content came into being by a selection process (e.g., natural selection or learning). Clearly, the first type of explanation requires further explanation of the second type.

Tinbergen (1963) listed four questions that can be asked about a behaviour (or a character in general) pertaining to its proximate mechanisms, its adaptive value, its development, and its evolutionary history respectively. Answers to each question explain a character in a different way. Lorenz (1966) took a different approach, arguing that what requires explanation in biology are not characters *per se*, but the information content or adaptive complexity of a character.

From this perspective, proximate mechanisms and adaptive significance are not two different levels of analysis, but two sides of the same coin, corresponding to a solution and a problem respectively. Neither can be understood in isolation from the other. In terms of the developmental question, any supposed cause must have sufficient information content to explain the information content of the resulting biological character.

Preformatism and epigenesis

From this perspective, developmental explanations must show how information in developmental inputs map to outputs. Biological structure or form is not preformed in development, but information is (Lorenz 1966). Development is epigenetic with respect to form and structure, but (partially) preformed with respect to information.

Nature and nurture

A shift in emphasis from explaining biological characters to explaining their information content changes the nature and nurture question. Clearly, all biological *characters* require the interaction of intrinsic and extrinsic (i.e., environmental) factors to develop. From the point of view of information content, however, this is irrelevant.

The emphasis shifts to sources of information, e.g., to genetic information, adaptive learning, and cultural transmission. In principle, the relative contributions of these sources of information to a behavioural pattern might be ascertained.

I discussed above how *Poverty of Stimulus Arguments* show that behaviour and perception have more information content in their outputs than in their environmental inputs. Similar reasoning can be applied to development. The environmental inputs involved at an earlier stage of development do not cover the all the aspects of the environment that adapted structures and processes conform to later in development. Therefore development has a high degree of algorithmic complexity (i.e., information content) which cannot be explained by proximate environmental inputs. Neither can this information content be explained by environmental inputs in the previous generation. Variation in the environment in one generation is not heritable, therefore an environment that is selected from a set of possible environmental variants in one generation cannot map to a similar selection in the next generation. That is, one generation (an "output") has more information content than the environmental "input" of the previous generation.

A possible approach to determining the relative contributions of different sources of information (genes, culture, learning etc.) to a behavioural pattern is suggested by the fact that information content is proportional to the degree of selection from a set of possibilities. It may be possible to classify "domains" (Williams 1991, 1996) of information that are characterised by the boundary conditions on a set of possibilities. For example, the deep grammatical structure of human language may be explained in the "genetic domain" (Pinker 1994). That is to say, the neurological structures involved are selected from a set of possible structures, corresponding to a similar degree of selection among possibilities (i.e., information content) in the relevant parts of the genome. However, the vocabulary of a human language is a set of words selected from a finer set of possibilities that is not coded by the genome. The genome lacks the information content to explain the words used in a human language. Hence, the vocabulary of a human language may be explained in a "cultural domain". At an even finer level, individual sentences cannot be explained adequately by cultural transmission. The number of sentences that words in a language can combine into is vast by comparison with the vocabulary of the language. The set of possible sentences forms a new "domain". Hence, the information content of sentences irrespective of

their content with respect to deep grammar and vocabulary, can only be explained by selection processes going on in individual brains and conversations.

The example discussed above suggests that domains of possibilities are organised hierarchically, with one possibility from a wider set of possibilities at one level corresponding to the boundary conditions of a finer set of possibilities at a finer level. The information content sufficient to specify some set of possibilities at a relatively coarse scale, will not necessarily be sufficient to specify an even smaller set of possibilities at a finer scale. In this way it may be possible to distinguish the information content in behaviour due to genetic inheritance to that due to learning and/or other processes.

Extrinsic and intrinsic factors

Extrinsic factors (i.e., the environment) do not contain sufficient information to explain the information content in behavioural processes - i.e., the *Poverty of the Stimulus* (see above). I have further argued that all adaptations have an information content, so this may be generalised to the *Poverty of the Environment*. "Stimuli" are not givens, but are specified by behavioural adaptations. If adaptations are theories, then the environment is the set of regularities in the surroundings of the organism to which it conforms. In other words, the environment is not a *given*, but is *specified by* the organism. The organism can be said to anticipate or expect these regularities (Popper 1976).

Applying this to the classic sensory deprivation experiment (Lorenz 1966), two chicks with different genomes in identical *surroundings* do not have the same *environment*. The differing information content in the two chicks means that they conform to a slightly different set of regularities in the surroundings. However, the fact that the environment can never be identical for two different organisms, does not mean that the difference between the organisms cannot be called innate. The difference in the environments is *due to* the differing information content in the two organisms, and the differing information content cannot be attributed to the surroundings (which are identical).

Well designed organism or well designed environment?

As the quote from Fisher earlier shows, random variations in the environment may reduce functioning in the same way as random variations in the organism

Therefore, in what sense is it correct to say that it is the organism rather than the environment which is well designed?

All life processes involve interactions between an organism and its environment. However, this does not mean that there are no distinctions between the two (i.e., that they can only be treated "as a whole"). It is adaptation that allows an organism to persist in its surroundings (including adaptations for homeostasis). The surroundings cannot explain the information content in an adaptation. The apparent good design in the environment of an organism can only be explained by the information content arising from the genome, learning, or cultural processes. Variation among individual organisms in a species may be heritable, but variation in the environment is not. Clearly it is species that are adapted to their environments rather than vice versa. In other words, where there is homeostatic regulation of the environment, then it is part of the extended phenotype of the organism (Dawkins 1982).

It may be useful to divide the *surroundings* of an organism into two categories, "environment" and "extended phenotype", or even three categories if we include those aspects of the surroundings that are irrelevant to adaptation (*qua* Fishers test, see quote above). This third category can hardly be called part of a species "environment" at all, and this shows that it is the adaptation of species that *specifies* what aspects of its surroundings are part of its environment.

Comparative approaches to studying good design in this thesis

I have argued that adaptations are inherently comparative. I have also argued that adaptations are good designs, are solutions to problems, have an information content, and that all these ways of characterising adaptation are comparative. Furthermore, I have argued that this view of life is supported by the fact that it may help to solve some fundamental problems in biology.

Hypotheses about adaptation can be tested using comparative criteria, and rather than being only weakly falsifiable, such hypotheses specify an improbable state of affairs (good design is improbable), hence are exceptionally falsifiable (see footnote 1). In principle, is relatively easy to demonstrate that something is not well designed

from some specified task.[8] In practice, however, it is not so easy to detect what will be the effect of a random variation on functioning. Often, this requires more detailed physiological knowledge than we possess.

With respect to this thesis, it is not immediately clear that a random variation to the growth processes of swifts or Procellariiformes would increase starvation rates. I addressed this problem in several ways. One approach was consider several mechanisms by which starvation rates and growth rates may be connected (Chapter 5). I incorporated the most successful of these models (the energy savings and cell allocation models) into simulation models in which I varied both growth rate and variability in food supply and measured the effect on probability of starvation (Chapter 6).

The strength of the method described in the last paragraph depends upon the soundness of the models. Ideally, we would like to be able to make real birds change their growth rates in real environments. Since this is practically impossible, I instead compared a range of existing lineages that have evolved different growth rates (Chapters 2 and 3). According to Lack's insurance hypothesis, lineages will only persist if they are well designed for the environments they are in. Therefore, I was able to test Lack's insurance hypothesis by testing whether lineages with variable food supplies consistently have slow growth, and whether lineages in less variable food supplies tend to have faster growth.

Since growth rate is a I simple scalar character, the argument from design or adaptive complexity is hard to apply. By modelling various aspects of growth rate (e.g., minimum and mean mass and wing growth rates), I was able to convert an apparently simple character (growth rate) into a more complex suite of characters. Also, interspecific comparisons (or comparisons between phylogenetically independent lineages) are especially useful for testing hypotheses about the adaptive value of scalar characters such as growth rate, because as Fisher (1958) pointed out, arguments from design or adaptive complexity;

[8] Consider for example how easy it is to falsify the hypothesis that legs are good for seeing with.

"...cannot be applied with full force to such simple characters, considered in isolation; but each may nevertheless be supposed to possess an optimum value in relation to the existing state of the organism and its environment, which we may regard as nearly coincident with the mean value exhibited by the species."

Therefore, comparing species-specific growth rates in relation to species-specific variability in food supply provides a good test of Lack's insurance hypothesis.

By modelling the relationships between minimum and mean mass and wing growth rates I was able to combine the modelling approach with interspecific comparisons. I first constructed models of how I conjectured mass and wing growth rates to be related in species adapted to a variable food supply (Chapter 3). I made predictions from these models (Chapter 3, which I later checked using simulations in Chapter 6) about how the relationship between mass and wing growth rates would vary with changes in the proximate food supply. I tested these predictions by testing whether lineages with variable food supplies showed the predicted changes in the relationship between mass and wing growth rates under a changing proximate food supply (Chapter 3). I also tested these predictions on one species, the Welcome Swallow, by experimentally manipulating the food supply to the nestlings and measuring the effect on mass and wing growth rates (Chapter 7).

Are the developmental patterns of swifts and Procellariiformes well designed for survival in environments with a variable food supply?

It appears that *some* aspects of the development of swifts and Procellariiformes are well designed for survival in habitats where the food supply is variable. Specifically, large deposits of fat in the chick that cannot be explained as insulation nor as reserves for the post-fledging period would have been unlikely to have evolved only in species with a variable food supply if the insurance hypothesis is false. The fact that variability in growth is largely variability in fat deposition does not change this. Species with low variability in growth could have evolved so that a large component of their rapid growth consists of fat deposition - but they have not.

However, although slow lean tissue growth rates have repeatedly evolved in environments with a variable food supply, it is not clear whether this is part of a suite of developmental characters that are well designed for insurance against starvation.

The answer to this question depends partly on our model of growth processes. For instance, if fat deposition and lean tissue growth are a trade-off relationship either by cell allocation or by competition among tissues for energy and nutrients, then a random change in the chicks' lean tissue development is likely to reduce its ability to survive a variable food supply. If, on the other hand, lean tissue growth rates are relatively independent of fat deposition, then it is difficult to say in what way slow lean tissue growth rates are well designed for life in a variable environment.

I have made a first attempt on understanding the relationships between lean tissue growth rates, fat deposition, and avoiding starvation using a number of models tested using phylogenetically independent contrasts and experiments. It appears that the extremely slow lean tissue growth rates, fast fat deposition rates, and the ability to fast for long periods in swifts and Procellariiformes is an improbable combination of factors that is adaptive in environments with a variable food supply. A fuller understanding, however, will require detailed understanding of the interactions between the components of growth in birds.

Chapter 10

Summary and conclusions

Summary of findings

Using variability in mass growth as a proxy for variability in food supply, swifts and offshore-foraging seabirds showed evidence of high variability in food supply. In contrast, inshore-foraging seabirds and terrestrial birds other than aerial insectivores had a relatively constant food supply (Chapter 2). I further found that variability food supply is correlated with long nestling periods and slow growth rates. Foraging in an offshore environment and the swift foraging niche were correlated with a variable food supply. Hence, Lack's insurance hypothesis passed what are critical tests.

Birds with long nestling periods and slow growth rates also tended to have low feeding frequencies and protected nest sites. These trends are in accordance with previous studies and Lack's conjectures, but can also potentially confound Lack's insurance hypothesis. The selection pressures on growth rates by nest predation and variability in food supply should be conflicting rather than concurring (Chapter 4). Birds that have relatively high rates of nestling predation will be unable to adapt to a variable food supply by decreasing growth rates. Therefore, where predation is eased an ecological release for adaptation to a variable food supply may occur. Nesting on offshore islands or in other protected nest sites may be a prerequisite for adaptation to a variable food supply. I found evidence that long nestling periods are coadapted with a preference for protected nest sites The coadaptation is only partial, and probably due to an evolutionary time-lag.

Variability in feeding frequency was not correlated with nestling period, growth rates, or variability of mass growth (Chapter 2). I therefore concluded that variability in feeding frequency is not a reliable measure of variability in food supply. Criticisms of Lack's insurance hypothesis based upon analyses of feeding frequencies are unlikely to be reliable.

I found that proximate variations in food supply appear to strongly affect mass growth rates - i.e., many species in environments with a variable food supply have a relatively fast maximum mass growth rate (Chapter 3). However, wing growth rate was

not affected by proximate conditions in the same way. Wing growth rate and nestling period were relatively uniform in species with highly variable mass growth rates. Hence, the proximate effect hypothesis for long nestling periods in environments with a variable food supply was ruled out in favour of an evolutionary explanation.

The ratio between wing (hence lean tissue) and mass growth periods was higher in species in environments with a variable food supply. This suggests that growth in mass in species in environments with a variable food supply is due in large extent to fat deposition when mass growth rate is at a maximum. Moreover, this ratio was greater for minimum growth periods than for mean growth periods. The difference between the ratios of mean and minimum growth periods was greater in species in environments with a variable food supply.

Species with a variable food supply tend to have low maximum lean tissue growth rates and rapid maximum fat deposition rates. I conjectured that the capacity for rapid fat deposition was physiologically linked to slow wing (i.e., lean tissue) growth rates. I conjectured two ways in which lean and adipose tissue growth might be in a trade-off relationship (Chapter 5). First, low rates of lean tissue growth may incur an energy savings that may be invested in fat deposition and result in lower energy costs during periods of poor food supply. I referred to this as the "energy savings model". Second, lean tissue growth may be in competition for the allocation of cells by differentiation with the growth of adipose tissue and tissues associated with lipogenesis. I tentatively concluded in favour of the latter model (the cell allocation model) following a consideration of chick energy budgets, and having rejected several alternative hypotheses using comparative data.

I conjectured that Lack's insurance hypothesis is a variety of "precociality" theories - i.e., that that swifts and Procellariiformes are "precocial for" the development of adipose tissue, that this ensures survival during fasting conditions, and that a variable food supply places a high functional priority on adipose tissue early in development. Precocial growth has a specific meaning in birds - i.e., it refers to species that have early maturation of locomotion and foraging skills, and leave the nest relatively early. However, it also has a more general meaning in that a species may be precocial for a particular function. Early mature functioning of any tissue may reduce overall growth rate by competing with growing tissues for energy and nutrients, or by

having an increased share of embryonic cells allocated to the tissue (hence fewer cells allocated to other tissues).

When I modelled the growth of chicks using computer simulations, I found that the probability of starvation in environments with a variable food supply was lower in simulations where maximum lean tissue growth rates were set at a low level, and maximum fat deposition rates were set at a high level (Chapter 6). When fat deposition rates were not set at a high level, fat deposition due to energy savings alone was insufficient to ensure low rates of starvation. I concluded that for Lack's insurance hypothesis to be correct, the main benefit of reduced lean tissue growth rates must be an increased allocation of cells to lipogenetic functions, and hence increased potential fat deposition rates.

I also modelled several alternative ways in which fat deposition and lean tissue growth could be related. I concluded that although a reduced digestive system may explain a reduction in overall growth rates, it would not provide the "insurance effect" required for survival in a variable food supply (Appendix 7). This was corroborated by the observation that many species in environments with a variable food supply, with low maximum lean tissue growth rates, have relatively high maximum mass growth rates. I also modelled growth as a priority system involving deposition of fat tissue prior to lean tissue growth (Chapter 6). One version of these models predicts that wing growth rate should correlate with chick condition but not with proximate food supply. I found evidence that the swift growth resembles this pattern. Swift chicks have slow wing growth even when they are gaining mass if they are in poor condition.

I expected that species with a facultative growth strategy, such as the Swift, would have a food supply that is variable but predictable over the short-term (Chapter 8). However, the Swift ranked very lowly on my index of predictability of food supply. I was unable to determine whether my theory or my methods were in error. Similarly, I used comparative data to test the hypothesis that species with long feeding intervals do not regulate provisioning. Again, I was unable to come to firm conclusions because of the highly derived nature of my measurements, and the scarcity of necessary data.

I classified the growth models that I had developed to test the proximate effect hypothesis, and the insurance hypotheses into three types. I used the Welcome Swallow as an experimental system upon which to test the models. I found support for the "lean-priority" model, where lean tissue growth has priority over fat deposition. However,

other authors findings are in agreement with the "fat-priority" model which suggests a diversity of growth strategies among birds

The limitations and strengths of interspecific comparisons

I have subjected Lack's insurance hypothesis to stronger tests than it has been subject to in the past, and found it to be corroborated by comparative data. However, comparative tests cannot control for possible confounds to the same degree as experiments. Nest predation may account for much more of the variation in growth rates in birds than my analysis suggests. Mean food supply (irrespective of feeding frequency) is another possible confound that requires further attention.

Interspecific comparisons are, however, especially useful for testing hypotheses about the adaptive value of scalar characters, such as growth rate, to which arguments form design or adaptive complexity are hard to apply (Chapter 9). Furthermore, hypotheses may not only be rejected because they have been falsified by evidence, but also because they fail to adequately explain (i.e., predict) correlations in the data (Popper 1963, 1983, Humphrey 1995). Interspecific comparisons are useful in this regard because they can be used to not only test hypotheses, but may also reveal patterns in the data that competing hypotheses cannot explain. For instance, the nest predation hypothesis may be sufficiently informative to explain the diversity growth rates. It cannot, however, explain why species with slow lean tissue growth rates in environments with a variable food supply tend to have relatively high maximum mass growth rates. Nor does it explain why lean tissue growth rates are relatively constant whilst mass growth rates are so variable. Lack's insurance hypothesis provides a possible explanation for these data, and hence gains additional corroboration.

Future directions

In this thesis I have followed two general strategies. First, each chapter is an attempt to solve problems arising from the work reported in previous chapters. For example, Chapter 3 is an attempt to solve the problem arising in Chapter 2 - i.e., deciding between an evolutionary (Lack's insurance hypothesis) and a proximate environmental explanation of the correlation between growth and nestling periods and variability in food supply. A corollary of this approach is that I have sought successively more informative models of growth in birds - i.e., models that subsume or

explain earlier models. For example, in Chapter 5 I developed physiological models that may explain Lack's insurance hypothesis, and in Chapter 6 I developed mathematical models that may explain in more detail the models in Chapter 5.

Second, I have been guided by functionalistic, or adaptationistic, reasoning. That is, I have started by testing a hypothesis about adaptation (Lack's insurance hypothesis), and have then sought proximate mechanisms by which the supposed adaptation may be achieved. In other words, I have analysed bird growth as embodying solutions to problems, such as the problem of avoiding starvation in a variable food supply. In Chapter 9, I argued that proximate and adaptative explanations are two sides of the same coin, corresponding to solutions and problems respectively. Therefore, the discovery of proximate physiological processes depends upon prior hypotheses about the problems that such processes may solve. Nesse and Williams (1995) argue that this strategy has been followed for practically all important discoveries in biology. The process is an open ended one. Deeper and more informative explanations may always be sought, more detailed models may always be constructed (Popper 1963). With this in mind, I have listed below a selection of problems that may be addressed by future research. Answers to these problems would increase our knowledge of the details of growth processes. Any such increase in complexity makes it easier to apply the argument from design (which depends upon the complexity of the processes under consideration, Chapter 9) to Lack's insurance hypothesis.

(i) I have postulated that adipose tissue competes with lean tissue during development. This suggests further research into the details of cell allocation and energy distribution during development. Until it is known how fat cells and lean tissue cells are allocated, and how they compete during development, my cell allocation model of obligate hyperlipogenesis (Chapter 5) remains highly speculative.

(ii) The growth models (lean-priority, uniform distribution, and fat-priority) developed in Chapters 3, 6 and 7 are coarse grained models about growth priorities. These models assume the presence of biochemical processes involving complex feedback systems. For example, the fat-priority model assumes that the existence of a factor that regulates the relative rates of fat deposition and lean tissue growth, and that this factor is in turn controlled by earlier fat deposition rates. The details of such a feedback process, and the biochemicals involved, may be worked out by research guided by models, such as the fat-priority model, that predict that such processes exist.

(iii) My mathematical models have been relatively crude and have been constrained by available data on growth energetics. Refinements to the models may include information on the differing maintenance costs of lean and adipose tissue, and on the use of energy by chicks during starvation. Studies on the energy budgets of growing chicks have tended to be on chicks receiving a steady supply of food (e.g., Ricklefs *et al* 1980, 1985). The proportion of energy used for growth as a proportion of total energy budget may change between periods with a good food supply and periods of food shortage, suggesting the need for studies on energy use by chicks in fasting conditions. Energy budgets for growing chicks with more diverse growth rates are also required. Current understanding of the energetics of growth in birds appears to be limited to data obtained from relatively slow growing birds.

My models could be further refined by finding better ways of simulating a variable food intake, and more realistic ways of partitioning of the growing chick into body components.

(iv) My interspecific comparisons could be made more powerful by using more precise data on nest predation rates and more precise measurements of food intake. The relationship between variability in feeding frequency and variability in mass growth could be further scrutinized by researchers with access to detailed field observations of a range of species. By contrast, I was constrained in this study by access only to limited data from published sources. Live chicks (or even preserved specimens) might be studied for a more detailed comparison of variability in wing-length with variability in mass. The role of fat deposits in the variability of mass growth rates could also be confirmed in this way. The concept of variability in food supply is susceptible to refinement, modification and elaboration (something I have attempted to do in Chapter 8).

Are the similarities between the swifts and Procellariiformes the result of convergent evolution?

Lack's insurance hypothesis states the swifts and Procellariformes have undergone a process of natural selection in environments with a variable food supply, wherein individuals with slow growth rates had a reproductive advantage. Lack's insurance hypothesis is part of a general hypothesis about the operation of natural selection in environments with a variable food supply. This implies that (i) the

relationship between variability in food supply and growth rate is general among birds species, (ii) that slow growth rates are adaptive for species in environments with a variable food supply, (iii) that this relationship is the result of independent evolution (convergence) rather than phylogenetic conservatism (homology). The first point has been tested and corroborated in this thesis by comparative data using phylogenetically independent contrasts. I addressed the second point Chapter 9, where I concluded that slow maximum lean tissue growth rates are part of a suite of adaptations for avoiding starvation in environments with a variable food supply. The third point has also been tested and corroborated using phylogenetically independent contrasts, the relationship between growth rate and variability in food supply has apparently evolved independently in a number of lineages.

Prior to developing his insurance hypothesis, Lack considered the idea that the similarities between the swifts and Procellariiformes are due to homology rather than adaptation. The swifts and the Procellariiformes are extraordinary birds. They cover enormous distances and defy unmerciful conditions in order to provide food for their chicks. The chicks themselves are remarkable, able to withstand conditions that would starve other species to death, but growing at a snails-pace. So different are they from other birds, David and Elizabeth Lack (1951) conjectured that their slow growth is an ancestral "reptilian condition" - i.e., it is other birds that have evolved *rapid* growth rates. This is plausible because hummingbirds, which are a member of the Apodidae (the family that includes the swifts), also show certain "reptilian" characteristics such as the ability to go into torpor. But as I showed used phylogenetically independent contrasts in Chapters 2 and 3, it is not just the Procellariiformes and swifts that have slow growth rates and a variable food supply. Growth rate and variability in food supply have evolved together in a number of lineages. In other words, some species in groups where *rapid growth* is the ancestral condition have evolved slow wing growth rates, fast fat deposition rates, and large fat reserves in environments with a variable food supply. Remarkable as they are, the Procellariiformes and swifts appear to be at the extreme end of a continuum of adaptive evolution.

Appendix 1

Tabulated data for phylogenetically independent contrasts

The tables in this appendix contain the data used in phylogenetically independent contrasts. Legends are positioned above the tables.

Table A1.1 Species means 1. *NP*, NP_{min}, and *IP* are species means for nestling period, minimum nestling period, and incubation period respectively. Habitat; a = aerial insectivore, t = terrestrial non-aerial insectivore, i = inshore-foraging seabird, o = offshore-foraging seabird. Phylogeny No. indicates position in the phylogeny in Appendix 2.

Species	Family	Phylogeny No.	Habitat	Adult Weight	*NP*	NP_{min}	*IP*
Accipter cirrhocephalus	Accipitridae			173	28.5		35
Accipter fasciatus	Accipitridae			455	30.5		34
Accipter nisus	Accipitridae			204	27		
Alle alle	Alcidae	26	i	120	28	23	24
Anous minutus	Sternidae	17	o	104	47		35
Anous stolidus	Sternidae	16	i	218	50	40	34
Aptenodytes fosteri	Spheniscidae	40		29000	141		64
Aptenodytes patagonicus	Spheniscidae	41		12000	310		54
Apus apus	Apodidae	3	a	42	42.5	37.5	20
Aquila pomarina	Accipitridae			1952	58		40
Calonectris diomedea	Procellariidae	49	o	746	91	83	58
Catharacta maccormicki	Stercorariina	25	i	1310	54	49	28
Circus aeruginosus	Accipitridae			584	37.5		35
Curcus pygarus	Accipitridae			315	37.5		29
Cypseloides niger	Apodidae	4	a	58.6	48		24
Daption capense	Procellaridae			438	48		44
Diomedea chrystostoma	Diomedidae	59	o	3355	141		73
Diomedea melanophris	Diomedidae	58	o	3505	116		68
Erithacus rubecula	Muscicapidae	12	t	18.7	12.5	10.5	11
Erythrotrichus radiatus	Accipitridae			937			
Falco cenchroides	Falconidae			166	31		29
Forpus passerinus	Pssittacidae	1	t	123.9	31.5	28	35
Fratercula arcica	Alcidae			380	38		40
Fregata aeriel	Fregatidae			842	161		41
Fregata minor	Fregatidae			1287	169		55
Fregetta tropica	Oceanitidae	46	o	54	68		37
Fulmarus glacialis	Procellaridae			713	46		52
Fulmarus glacialoides	Procellaridae			767	52		
Gygis alba	Sternidae	22	o	108	60	40	31
Gypaticus barbatus	Accipitridae			5573	105		58
Halobaena caerula	Procellariidae	47	o	208	53	43	49

Species	Family	Phylogeny No.	Habitat	Adult Weight	*NP*	NP_{min}	*IP*
Hieraetus morphinoides	Accipitridae			850	60		37
Hirundo neoxena	Hirundinidae	8	a	15	22.5	20	16
Hirundo tahitica	Hirundinidae	7	a	15	19.8	18.3	16
Iridoprocne albilinea	Hirundinidae	10	a	15.5	23		
Ixobrychus minutus	Ardeidae			84	27.5		21
Larus argentatus	Laridae	23	i	963	42		27
Larus dominicanus	Laridae	24	i	728	42		27
Larus novaehollandiae	Laridae			288	28		24
Lohocatinia isura	Accipitridae			620	63		40
Macronetes giganteus	Procellaridae			4310	119		60
Macronetes halli	Procellaridae			4179	113		60
Megadyptes antipodes	Spheniscidae	37	o	5891	113		45
Myiozetetes similis	Muscicapidae	15	a	26.4	18		
Oceanites nereis	Oceanitidae			37			45
Oceanites oceanicus	Oceanitidae	45	o	40	59	48	50
Oceanodroma castro	Hydrobatidae	42	o	43.5	69	59	42
Oceanodroma leucorhoa	Hydrobatidae	43	o	63.7	67		42
Oceanodroma tristami	Hydrobatidae	44	o	99.9	89.2	85	
Pachyptila salvini	Procellariidae	48	o	154	59	52	49
Pandion halaetus	Pandionidae			1527	53		37
Parus major	Paridae	5	t	15	19	16	14
Parus montanus	Paridae	6	t	12.9	16		
Pelagodroma marina	Oceanitidae	55	o	74	57	52	50
Pelecanoides goergicus	Pelecanoididae			119	50		47
Pelecanoides urinatrix	Pelecanoididae			129	53		54
Pelicanus crispus	Pelecanidae			10500	67.5		33
Pernis apivorus	Accipitridae			762	42		33
Phaethon aethereus	Phaethontidae	28	o	762	88	79	42
Phaethon lepturus	Phaethontidae	27	o	424	72		41
Phalacrocorax aristotelis	Phalacrococacidae	36	i	1650	53	48	30
Phalacrocorax nivalis	Phalacrococacidae	35	i	2746	56		29
Phalacrocorax punctatus	Phalacrococacidae	34	i	956	50	48	29
Phoebetria fusca	Diomedidae	56	o	2678	157	149	70
Phoebetria palpebrata	Diomedidae	57	o	2950	157	150	67
Procellaria cinerea	Procellariidae	52	o	97	147	130	60
Procellaria parkinsonii	Procellaridae			729	107		57
Pssitirostra bailleui	Pssittacidae	2	t	37.1	24	21	16
Pterodroma brevirostris	Procellariidae	53	o	357	61		49
Pterodroma macroptera	Procellaridae			561	123		55
Pterodroma mollis	Procellariidae	54	o	312	91		50
Puffinus ilherminieri	Procellariidae	50	o	175	75	62	49
Puffinus puffinus	Procellariidae	51	o	557	70	62	51
Pygoscelis adeliae	Spheniscidae	39		557	75		34
Pygoscelis papua	Spheniscidae	38		4500	90		37
Rissa trydactyla	Laridae			387	42.7		27
Sterna albifrons	Sternidae			50	18		20
Sterna anaethus	Sternidae			129	59		29
Sterna bergii	Sternidae			354	39		28
Sterna dougallii	Sternidae			100	28.5		23

Species	Family	Phylogeny No.	Habitat	Adult Weight	*NP*	NP_{min}	*IP*
Sterna fuscata	Sternidae	21	o	140	60	56	30
Sterna hirundo	Sternidae	18	i	120	25		22
Sterna maxima	Sternidae			402	30.5		31
Sterna nereis	Sternidae				21		21
Sterna paradisaea	Sternidae	20	i	112	22.5	21	20
Sterna sandviciens	Sternidae			252	29		25
Sterna virgata	Sternidae			117	35		24
Sterna vittata	Sternidae	19	i	140	27	23	24
Sturnus vulgarus	Sturnidae	11	t	78	19		11
Sula bassana	Sulidae	33	o	4000	91	84	44
Sula dactylatra	Sulidae	30	o	2100	118	113	44
Sula leucogaster.	Sulidae	29	o	1200	101	87	43
Sula nebouxii	Sulidae	31	i	1550	102		43
Sula sula	Sulidae	32	o	1000	121		45
Tachycineta cyaneoviridis	Hirundinidae	9	a	16.3	22.8	22	16
Thalassoica antarctica	Procellaridae			677	45		46
Threskiornis molucca	Plataleidae			1862	48		22
Turdus merula	Muscicapidae	13	t	76	21	21	12
Turdus pilaris	Muscicapidae	14	t	69.3	13.5	13	

Table A1.2 Species means 2. *V*, *FF*, PD, and CV_{ff} are species means for variability in food supply, feeding frequency, degree of nest protection (P = protected, U = unprotected), and coefficient of variation of feeding frequency respectively.

Species	*V*	*FF*	*PD*	CV_{ff}
Accipter cirrhocephalus		6		
Accipter fasciatus		6.5		0.2
Accipter nisus		4.9		0.12
Alle alle	0.07	5.2	U	
Anous minutus	0.34	3	P	
Anous stolidus	0.06	1.6	P	
Aptenodytes fosteri				
Aptenodytes patagonicus				
Apus apus	0.23	6.7	P	
Aquila pomarina		9		
Calonectris diomedea	0.06		P	
Catharacta maccormicki	0.05	3.7	P	
Circus aeruginosus		4		0.21
Curcus pygarus		4		0.31
Cypseloides niger	0.38		P	
Daption capense		0.5		0.22
Diomedea chrystostoma	0.24	0.52	P	
Diomedea melanophris	0.23	0.47	P	
Erithacus rubecula	0.06	37	U	
Erythrotrichus radiatus		7		
Falco cenchroides		10.5		
Forpus passerinus	0.1	17	P	

Species	*V*	*FF*	*PD*	CV_{fl}
Fratercula arcica		6		
Fregata aeriel		0.44		0.34
Fregata minor		0.52		0.58
Fregetta tropica	0.39	0.49	P	0.2
Fulmarus glacialis		1.7		1.2
Fulmarus glacialoides		0.6		
Gygis alba	0.16	8	P	0.89
Gypaticus barbatus		3.5		
Halobaena caerula	0.21	0.44	P	0.33
Hieraetus morphinoides		3.1		
Hirundo neoxena	0.07		P	
Hirundo tahitica	0.06	185	P	
Iridoprocne albilinea	0.1		P	
Ixobrychus minutus		2.8		
Larus argentatus	0.065	2	U	
Larus dominicanus	0.1	9	U	
Larus novaehollandiae		9.8		0.57
Lohocatinia isura		0.5		0.8
Macronetes giganteus		1.9		0.51
Macronetes halli		2		0.46
Megadyptes antipodes	0.05	1.5	P	
Myiozetetes similis			U	
Oceanites nereis		0.62		0.37
Oceanites oceanicus	0.36	0.67	P	0.05
Oceanodroma castro	0.53	0.7	P	
Oceanodroma leucorhoa	0.191		P	
Oceanodroma tristami	0.29		P	
Pachyptila salvini	0.4	0.57	P	0.13
Pandion halaetus		1.5		
Parus major	0.07	50	U	
Parus montanus	0.024	27	U	
Pelagodroma marina	0.66	0.5	P	0.5
Pelecanoides goergicus		2.1		0.12
Pelecanoides urinatrix		1.9		0.17
Pelecanus onocrotalus		2		
Pernis apivorus		8		
Phaethon aethereus	0.099	1.5	P	
Phaethon lepturus	0.2	1.5	P	
Phaethon rubricandra		1.4		
Phalacrocorax aristotelis	0.025		U	
Phalacrocorax nivalis	0.04		U	
Phalacrocorax punctatus	0.03	4.8	U	
Phoebetria fusca	0.57	0.42	P	
Phoebetria palpebrata	0.57	0.39	P	
Procellaria cinerea	0.33		P	
Procellaria parkinsonii		2		0.7
Pssitirostra bailleui	0.07	17	P	
Pterodroma brevirostris	0.52		P	
Pterodroma macroptera		0.33		0.44
Pterodroma mollis	0.48		P	0.2

Species	*V*	*FF*	*PD*	CV_{ff}
Puffinus ilherminieri	0.271	0.75	P	
Puffinus puffinus	0.17	0.92	P	0.46
Rissa trydactyla		50.4		0.62
Sterna albifrons		25.4		
Sterna bergii		9.2		
Sterna dougallii		5		1.4
Sterna fuscata	0.3	1	P	
Sterna hirundo	0.27	15	U	0.38
Sterna maxima		17.2		0.69
Sterna nereis		19.1		
Sterna paradisaea	0.05	10.5		0.44
Sterna sandviciens		10.1		
Sterna sumatrana		11.5		
Sterna virgata		158		0.92
Sterna vittata	0.08	8	U	
Sturnus vulgarus	0.027	60	U	
Sula bassana	0.04	2.6	U	0.28
Sula dactylatra	0.27	2	P	0.34
Sula leucogaster.	0.15	2	P	
Sula nebouxii			P	0.54
Sula sula			P	
Tachycineta cyaneoviridis	0.08		P	
Thalassoica antarctica		0.5		
Threskiornis molucca		56		
Turdus merula	0.031	55	U	
Turdus pilaris	0.01		U	

Table A1.3 Species means 3. *MGP*, MGP_{min}, *WGP*, and WGP_{min} are mass growth period, minimum mass growth period, wing growth period, and minimum wing growth period respectively.

Species	*MGP*	MGP_{min}	*WGP*	WGP_{min}
Alle alle	23.3	20	40.3	32.3
Anous minutus	25	16.7	42.7	36.3
Anous stolidus	28.7	23.7	44.4	38.5
Apus apus	15.8	11.8	29.9	26.5
Calonectris diomedea	60.2	49.6		
Catharacta maccormicki	37.2	29.4	66.6	56.8
Cypseloides niger	21.9	16.5	34.9	30.6
Diomedea chrystostoma	60.2	31.1		
Diomedea melanophris	57.1	24.2		
Erithacus rubecula	9.9	7.2		
Forpus passerinus	17.4	14.6		
Fregetta tropica	29.3	12.4	67.6	
Gygis alba	40.3	22.4	54.9	44.4
Halobaena caerula	29.5	19.8	44.8	
Hirundo neoxena	8	7.3	17.6	15.1

Species	*MGP*	MGP_{min}	*WGP*	WGP_{min}
Hirundo tahitica	9.8	7.7	17.3	16.3
Iridoprocne albilinea	10.8		17	
Larus argentatus	29.1	22.9		
Larus dominicanus	35.7	28.4		
Megadyptes antipodes	64.6	52.3	50.5	42.1
Oceanites oceanicus	24.3	17.1	51.7	
Oceanodroma castro	27.8	16.5		
Oceanodroma leucorhoa	45.3	32.9		
Oceanodroma tristami	42.7	30.8		
Pachyptila salvini	49.9	28.7		
Parus major	8	6.9		
Parus montanus	12	10.4		
Pelagodroma marina	28.2	17.5	52.9	
Phaethon aethereus	32.6	22.8		
Phaethon lepturus	36	25.8		
Phalacrocorax aristotelis	27.3	22.6		
Phalacrocorax nivalis	28.2	22.7	44.4	37.4
Phalacrocorax punctatus	17.6	12.9	34.1	22
Phoebetria fusca	74.5	49.8	122.1	
Phoebetria palpebrata	48.3	19.3	122.1	
Procellaria cinerea	63.7	36.9	102.2	95.7
Pssitirostra bailleui	14.5	11.4		
Pterodroma brevirostris	30.3	22.7	51.7	44.3
Pterodroma mollis	55.6	34.5	72	63.2
Puffinus ilherminieri	58.6	36.1	95.5	81.2
Puffinus puffinus	37.9	29.1	67.6	
Sterna fuscata	42.7	23	57.1	
Sterna hirundo	14.4	8.4	34.3	
Sterna vittata	20.8	16.9		
Sturnus vulgarus	11	9.6		
Sula dactylatra	59.4	31	129.2	115
Sula leucogaster	36.9	29.6	133.2	118.2
Tachycineta cyaneoviridis	10.9	8.5	18.3	17.2
Turdus merula	9.9	7.7		
Turdus pilaris	6.7	5.8	3	

Table A1.4 Sources for species means in Tables A1.1-3.

Species	Sources
Alle alle	Evans (1981)
Accipter cirrhocephalus	Marchant and Higgins 1993
Accipter fasciatus	Marchant and Higgins 1993
Accipter nisus	Cramp and Simmons 1980b
Alle alle	Evans 1981
Anous minutus	Ashmole 1962, Congdon 1990, Higgins and Davies 1996
Anous stolidus	Dorward and Ashmole 1963, Higgins and Davies 1996
Aptenodytes fosteri	Marchant and Higgins 1990a
Aptenodytes patagonicus	Marchant and Higgins 1990a

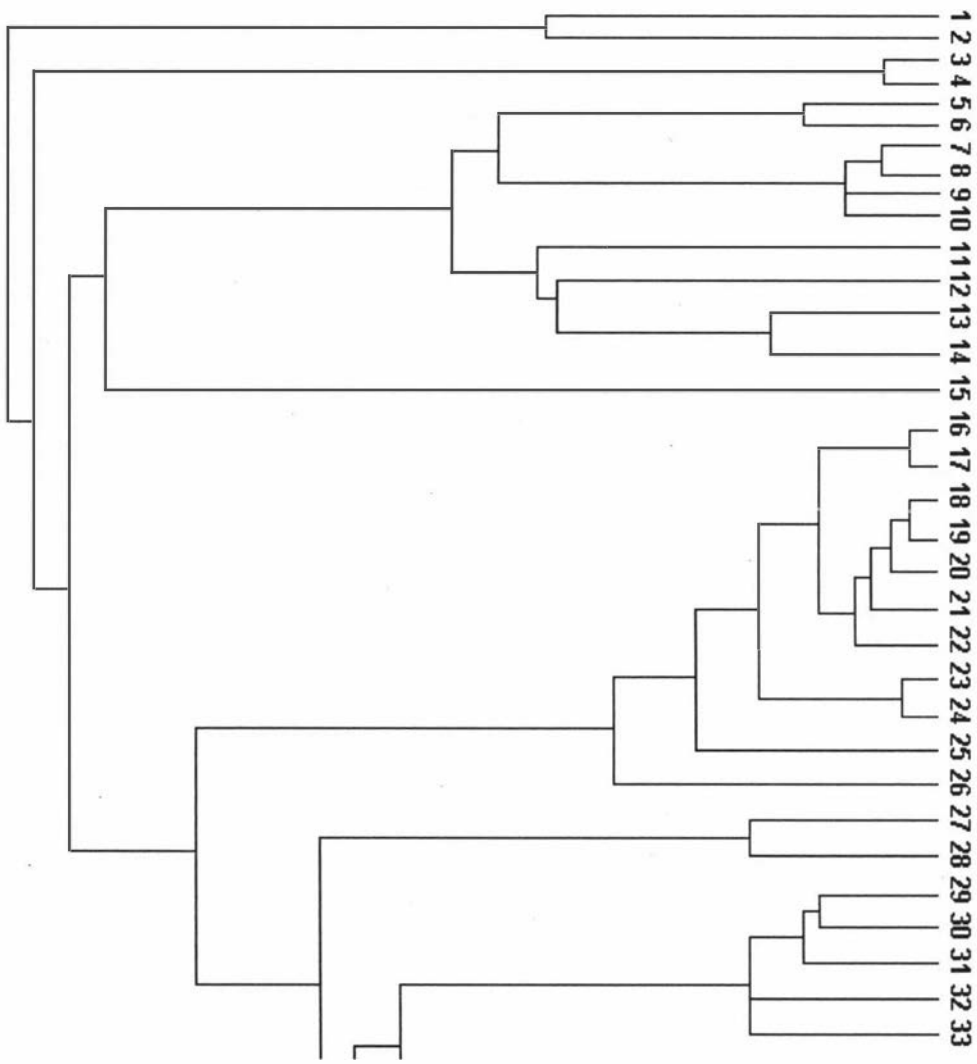
Species	Sources
Apus apus	Lack and Lack 1951
Aquila pomarina	Cramp and Simmons 1980b
Calonectris diomedea	Zino 1971, Round and Swann 1977
Catharacta maccormicki	Young 1963, Higgins and Davies 1996
Circus aeruginosus	Cramp and Simmons 1980b
Curcus pygarus	Cramp and Simmons 1980b
Cypseloides niger	Marin 1997
Daption capense	Marchant and Higgins 1990A
Diomedea chrystostoma	Tickell and Pinder 1975, Marchant and Higgins 1990a
Diomedea melanophris	Tickell and Pinder 1975, Marchant and Higgins 1990a
Erithacus rubecula	Lees 1949, Lack and Silva 1949
Erythrotrichus radiatus	Marchant and Higgins 1993
Falco cenchroides	Cramp 1985
Forpus passerinus	Waltman and Bessinger 1992
Fratercula arcica	Cramp 1985
Fregata aeriel	Marchant and Higgins 1990B
Fregata minor	Marchant and Higgins 1990B
Fregetta tropica	Jouventin et al 1975, Marchant and Higgins 1990a
Fulmarus glacialis	Cramp and Simmons 1980a
Fulmarus glacialoides	Marchant and Higgins 1990A
Gygis alba	Dorward 1963, Higgins and Davies 1996
Gypaticus barbatus	Cramp and Simmons 1980b
Halobaena caerula	Jouventin et al 1975, Marchant and Higgins 1990a
Hieraetus morphinoides	Marchant and Higgins 1993
Hirundo neoxena	This study
Hirundo tahitica	Hails, J. 1984.
Iridoprocne albilinea	Ricklefs 1976
Ixobrychus minutus	Cramp and Simmons 1980b
Larus argentatus	Spaans, A.L. 1971
Larus dominicanus	Fordham 1964
Larus novaehollandiae	Higgins and Davies 1996
Lohocatinia isura	Marchant and Higgins 1993
Macronetes giganteus	Marchant and Higgins 1990A
Macronetes halli	Marchant and Higgins 1990A
Megadyptes antipodes	Richdale 1957
Myiozetetes similis	Ricklefs 1976
Oceanites nereis	Marchant and Higgins 1990A
Oceanites oceanicus	Jouventin et al 1975, Marchant and Higgins 1990a
Oceanodroma castro	Allan 1962, Cramp and Simmons 1990a
Oceanodroma leucorhoa	Wilbur 1969, Ricklefs, White and Cullen 1980
Oceanodroma tristami	Marks and Leasure 1992
Pachyptila salvini	Jouventin et al 1975, Marchant and Higgins 1990a
Pandion halaetus	Marchant and Higgins 1993
Parus major	Gibb 1950
Parus montanus	Pravosudov and Pravosudova, 1996
Pelagodroma marina	Richdale 1943, Marchant and Higgins 1990a
Pelecanoides goergicus	Marchant and Higgins 1990A
Pelecanoides urinatrix	Marchant and Higgins 1990A
Pelecanus onocrotalus	Cramp and Simmons 1980a
Pelicanus crispus	Cramp and Simmons 1980a

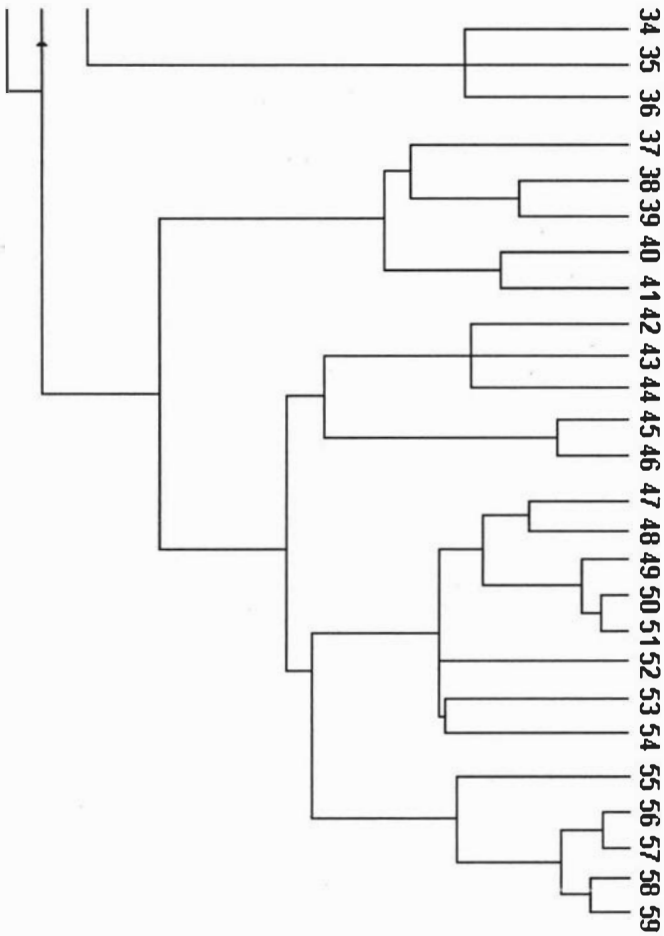
Species	Sources
Pernis apivorus	Cramp and Simmons 1980b
Phaethon aethereus	Harris 1969a, Cramp and Simmons 1980a
Phaethon lepturus	Schnaffner 1990, Marchant and Higgins 1990B
Phaethon rubricandra	Marchant and Higgins 1990B
Phalacrocorax aristotelis	Snow 1960
Phalacrocorax nivalis	Green 1997
Phalacrocorax punctatus	Fenwick 1975, Marchant and Higgins 1990a
Phoebetria fusca	Berruti 1979
Phoebetria palpebrata	Berruti 1979
Procellaria cinerea	Zotier 1990
Procellaria parkinsonii	Marchant and Higgins 1990A
Pssitirostra bailleui	Van Riper 1980
Pterodroma brevirostris	Schramm 1982, Marchant and Higgins 1990a
Pterodroma macroptera	Marchant and Higgins 1990A
Pterodroma mollis	Schramm 1982, Marchant and Higgins 1990a
Puffinus ilherminieri	Harris 1969b
Puffinus puffinus	Harris 1966, Marchant and Higgins 1990a, Hamer and Hill 1997
Pygoscelis adeliae	Marchant and Higgins 1990a
Pygoscelis papua	Marchant and Higgins 1990a
Rissa trydactyla	Cramp and Simmons 1983
Sterna albifrons	Higgins and Davies 1996
Sterna anaethus	Higgins and Davies 1996
Sterna bergii	Higgins and Davies 1996
Sterna dougallii	Higgins and Davies 1996
Sterna fuscata	Asmole 1963
Sterna hirundo	Erwin 1977, Klaassen 1994, Higgins and Davies 1996
Sterna maxima	Cramp 1985
Sterna nereis	Higgins and Davies 1996
Sterna paradisaea	Klaassen et al 1989, Higgins and Davies 1996
Sterna sandviciens	Cramp 1985
Sterna sumatrana	Higgins and Davies 1996
Sterna virgata	Higgins and Davies 1996
Sterna vittata	Klaassen 1994, Higgins and Davies 1996
Sturnus vulgarus	Dunnet 1955
Sula bassana	Nelson 1964, 1968, Cramp and Simmon 1980a
Sula dactylatra	Dorward 1962, Nelson 1978, Marchant and Higgins 1990B
Sula leucogaster.	Dorward 1962, Marchant and Higgins 1990B
Sula nebouxii	Nelson 1964
Sula sula	Marchant and Higgins 1990B
Tachycineta cyaneoviridis	Allen 1996
Thalassoica antarctica	Marchant and Higgins 1990A
Threskiornis molucca	Marchant and Higgins 1990B
Turdus merula	Gurr 1954
Turdus pilaris	Lilja 1983

Appendix 2

Working phylogeny for phylogenetically independent contrasts

Species are labeled with numbers corresponding to "Phylogeny No." in Table A1.1, Appendix 1. Distances between nodes indicate relative evolutionary branch lengths.




34 35 36 37 38 39 40 41 42 43 44 45 46 47 48 49 50 51 52 53 54 55 56 57 58 59

Appendix 3

Estimates of energy supply for fast and slow growing species

I have measured variability in food supply by proxy, i.e., as the variability of mass growth. Species with variable mass growth tend to have slow but relatively constant wing growth. Hence, variability in mass growth is probably mostly variability in fat deposition. It is possible then that species with a so-called variable food supply do not suffer food shortages, but, unlike other species convert windfalls of food into fat for some purpose other than insurance against starvation. The slow mass growth rates in these species, so the argument goes, are due to the fact that the energy density of fat is much higher than protein, hence a unit of fat growth takes more energy than a unit of lean tissue growth. If this is correct, then species with slow wing growth will tend to have a food supply that is greater than that for more slowly growing species.

I tested this idea on two representative species of similar weight but with contrasting wing growth rates and variability of mass growth, the Blackbird *Turdus merula* which has a low variability of mass growth (i.e., a low variability in food supply, $V = 0.05$) and rapid wing growth, and the Madeiran Storm-Petrel *Oceanodroma castro* which has variable mass growth (i.e. high variability in food supply, $V = 0.33$) and slow wing growth.

For both species I estimated the amount of energy required for growth on each day of the nestling period (up to day 43 for the Madeiran Storm-Petrel, prior to weight recession, Fig. A3) For each day I estimated the costs in energy of metabolism and growth. I did this by first estimating the mass growth rate and asymptotic mass of these species (cf. Fig. 3.15, Chapter 3) using the logistic equation. For the Madeiran Storm-Petrel, I did this for maximum, mean, and minimum growth rate. I then used the logistic equations to estimate the mass and growth increments for each day of growth. From these day, I used the equations and conversions described in chapter 6 to estimate daily energy budgets.

I used Equation 6.2 (Chapter 6) for the metabolic costs of the Madeiran Storm-Petrel, for the Blackbird I used values from Bennett & Harvey (1987). I assumed that the costs of lean tissue growth and fat deposition were similar in both species. I assumed that the Blackbird had negligible fat deposition, but for the petrel I divided daily growth increments into components due to lean tissue growth and fat deposition.

These estimates were based on the assumption that the ratio between peak mass (for maximum, mean or minimum asymptotes, corresponding to the respective growth rates) and fledging mass is the approximate ratio of total mass to lean mass throughout fledging.

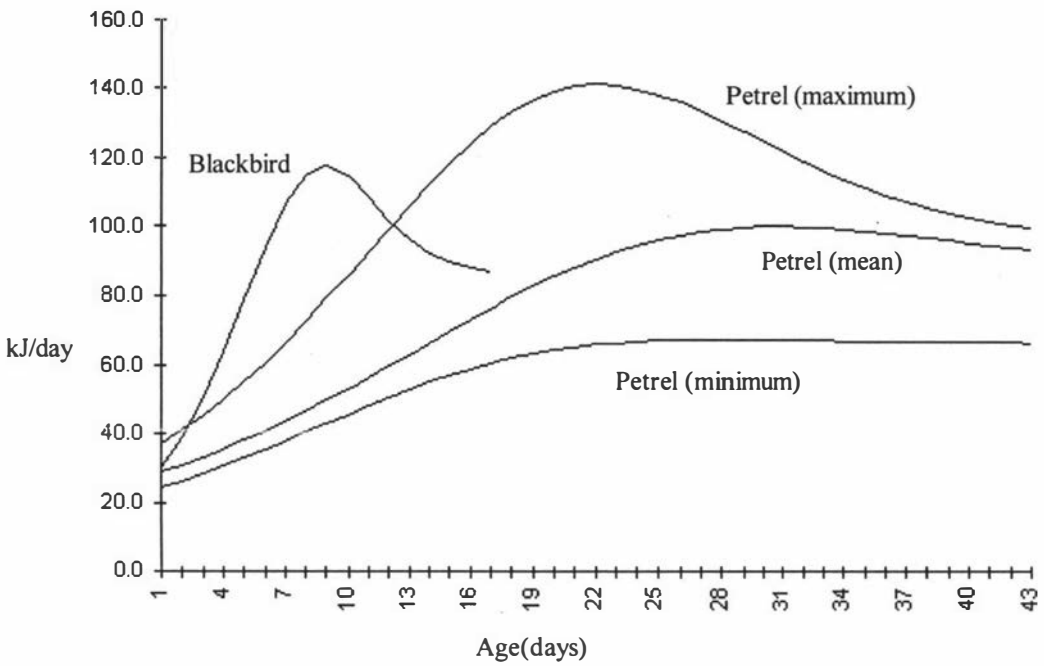


Fig. A3 Hypothetical daily energy requirements for Blackbird and Storm-Petrel chicks. Petrel budgets have been estimated for maximum, mean and minimum mass growth rates.

The curves in Fig. A3 are hard to interpret. One interpretation is that they are lines connecting the maximum, mean, and minimum energy budgets in a collection of chicks of a species for each day during the nestling period. This will only be approximate because the curves average over extremely poor days when chicks are not fed at all. Another interpretation is that the curves are idealised energy-budget curves for individual chicks in ideal, mean, and poor food supplies that persist throughout the nestling period. Again this is only an approximation or an idealisation, food supply will fluctuate from day to day in most chicks (Chapter 2).

However Fig. A3 is interpreted, it has implications for several aspects of this study. First, it appears to be correct that some birds with relatively slow mass growth rates may nevertheless have relatively high energy requirements. However, in this case this is only true when the Storm-Petrel chick growth is at its maximum rate. Clearly, minimum intakes are well below that for the Blackbird, hence petrels appear to suffer serious food shortages. The graph also shows that variability in mass growth rate is probably a good indicator of variability in energy supply.

Second, the high peak in the maximum amount of energy that the petrel can assimilate into its tissues suggests that the size of its digestive system does not limit lean tissue growth rate in the Madeiran Storm-Petrel (see Appendix 7). Also, provisioning of energy to petrel chicks is often below the amount that they can utilize, suggesting that it is factors extrinsic to the chick that limits its energy supply. This adds support to the energy savings model (Chapter 5). Also, the petrel chick appears to be able to both survive food shortages and opportunistically assimilate windfalls of energy from food, as the insurance hypothesis predicts.

Third, the peak of the *mean* energy supply to the petrel chick is approximately 85% of that of the Blackbird, i.e., fairly similar. This suggests that neither a variable food supply nor a long feeding interval necessarily greatly reduce mean food supply.

Finally, detailed estimates of the parameters that I have briefly discussed here could be used in more sophisticated comparative tests of growth models than those I have carried out in this study. For example, the peak of the mean energy requirement curve could be used as a parameter to control for the possible confound of differences between species in mean food supply (that is unrelated to feeding frequency, discussed in chapters 2 and 3) and a better estimate of variability in food supply could be calculated from variability in energy supply.

Appendix 4

Nestling Predation Raw Data

Sources: (1) Bosque & Bosque (1995), (2) Redfern (1994), (3) Cramp and Simmons (1980), (4) Bateman and Balda (1973), (5) Murphy (1983).

Family & species	Weight(g)	Incubation period (days)	Nestling period (days)	Nest type	Sources
Alcedinidae					
Acedo azurea	37.0	21	23.5	P	1
Dacelo novaeguineae	365.0		35	P	1
Dacelo leachii	339.0		35	P	1
Todirhamphus pyrrhopygia	51.0		21	P	1
Todrihamphus sanctus	44.0		28	P	1
Tanysiptera sylvia	50.0		31.5	P	1
Cacatuidae					
Nymphicus hollandicus	91.0	19	35	P	1
Corvus					
Corvus menedula	220.0	17.5	32.4	P	2,3
Pica pica	179.0	21.5	27.2	P	2,3
Gymnorhinus cyanocephala	103.3	17	21.5	O(new)	2,4
Climacteriadae					
Cormobates leucophaeus	23.0	21	25	P	1
Climacteris affinis	18.5		23	P	1
Climacteris picumnus	32.0	17	26	P	1
Emberizidae					
Calcarius mccownii	23.2	12.5	10.2*	O	1
Calcarius lapponicus	29	12	9	O	1
Calcarius ornatus	20	12	10	O	1
Calamospiza meloncorys	27.5	12	9.8*	O	1
Melospiza melodia	22	13	10.6*	O	1
Melospiza lincolnii	17	13.5	11.1*	O	1
Melospiza georgiana	17	12.5	9.5	O	1
Zonotrichia albicollis	30	12.5	9.5	O	1
Zonotrichia leucophrys	18.5	12	10	O	1
Zonotrichia querula	34.5	13.5	11.1*	O	1
Junco hyemalis	20	11.5	11.5	P(new)	1
Passerculus sandwichensis	17.2	12	9.8*	O	1
Ammodramus martimus	22.5	11.5	9	O	1
Ammodramus caudacutus	17.5	11	10	O	1
Ammodramus savannarum	16.5	11.5	9	O	1
Spizella arborea	18	12.5	9.5	O	1
Spizella passerina	13	12.5	10.5	O	1
Spizella pallida	11	10.5	8	O	1
Spizella pusilla	13.6	10.5	7.5	O	1
Pooecetes gramineus	23	12	11	O	1
Chondestes grammacus	29	12	9.5	O	1
Amphispiza belli	18.5	13	10.6*	O	1
Pipilo erythropthalmus	40	12.5	9	O	1
Plectrophenax nivalis	34.8	12.5	13	P(new)	1,2,3

Eopsaltridae					
Microeca fascinans	16.0	16.5	15.5	O	1
Petroica multicolor	13.0	15	15	P(new)	1
Petroica phoenicea	14.0	14	16	O	1
Petroica rosea	8.5	13	13.7*	O	1
Melanodryas cucullata	24.0	15.2	12.3	O	1
Melanodryas vittata	25.0	14	14.8*	P(new)	1
Eopsaltria australis	19.9	15.8	12.1	O	1
Poecilodryas superciliosa	19.0	14	15.3*	O	1
Fringillidae					
Carduelis pinus	13.7	13	14.5	O	1
Carduelis tristus	12.6	13	14	O	1
Carduelis psaltria	9.2	12	10.2*	O	1
Caruelis hornemanni	12.1	14.5	12	O	1
Carduelis flammea	13.5	11.5	12.5	O	1
Leucosticte arctoa	29.5	13	19	O	1
Carpodacus purpureus	24.3	13	14	O	1
Carpodacus cassini	24.2	13	12.2*	O	1
Carpodacus mexicanus	21.3	13	15	O	1
Pinicola enucleator	54.3	13.5	14	O	1
Loxia curvirostra	26.9	14.5	16.5	O	1
Coccothraustes vespertinus	56.4	13	13.5	O	1
Loriidae					
Trichoglossus haematodus	130.5	26.0	48	P	1
Trichoglussus rubritorquis	121.0	26.0	48	P	1
Trichoglossus chlorolepidotus	57.5	22.0	40	P	1
Psitteuteles versicolor	81.0	26.0	48	P	1
Glossopsitta concinna	60.8	24.0	48	P	1
Glossopsitta pusilla	50.5	22.0		P	1
Glossopsitta porphyrocephala	50.0	22.0	42	P	1
Meliphagidae					
Lichmera indistincta	10.0	14	14	O	1
Myzomela sanguinolenta	8.5	12	12.5*	O	1
Certhionys niger	9.0	16	18	O	1
Certhionys variegatus	24.5	13	10	O	1
Meliphaga gracilis	16.0	14	14	O	1
Meliphaga notata	26.5	15	15	O	1
Meliphaga lewinii	34.0	14.5	14.5	O	1
Lichenostomus chrysops	16.5	14	13.3	O	1
Lichenostomus virescens	28.0	13	13	O	1
Lichenostomus leucotis	22.0	14	14	O	1
Lichenostomus flavicollis	31.0	15	14	O	1
Lichenostomus flavescens	12.5	12	14	O	1
Lichenostomus plumulus	18.0	14	14	O	1
Lichenostomus penicillatus	17.0	14	14	O	1
Milithreptus lunatus	14.5	14	15	O	1
Melithreptus affinis	16.0	16	15	O	1
Melithreptus gularis	19.5	15	14	O	1
Melithreptus validirostris	23.5	15	15	O	1
Philemon corniculatus	92.0	16.1	15.8*	O	1
Phylidonyris pyrrhoptera	15.5	14	14	O	1
Phylidonyris novaehollandiae	20.0	13.5	12.8	O	1
Phylidonyris nigra	19.5	15	15	O	1
Phylidonyris albifrons	17.5	14	14	O	1

Ramsayornis modestus	11.5	15	15	P(new)	1
Conopophila albogularis	11.5	12	12	O	1
Conopophila rufogularis	11.5	14	12	O	1
Grantiella pica	23.0	15	14	O	1
Acanthorhynchus tenuirostris	10.5	14	10.1	O	1
Manorina melanophrys	28.5	15	15	O	1
Manorina melanocephala	75.0	15.5	16	O	1
Acanthagenys rufogularis	52.0	14	15	O	1
Anthochaera chrysoptera	65.0	12	16	O	1
Anthochaera carunculata	115.0	15	15	O	1
Anthochaera paradoxa	230.0	15	19.5	O	1
Monarchidae					
Rhipidura leucophrys	21.5	13.7	14	O	1
Rhipidura fulginosa	7.5	14	11	O	1
Rhipidura rufifrons	11.0	14	14.5*	O	1
Monarcha leucotis	11.5	14	14	O	1
Myiagra rubecula	12.5	14.5	13	O	1
Myiagra cyanoleuca	17.5	17.5	17.5	O	1
Myagra inquieta	20.0	14	14	O	1
Pachycephalidae					
Falcunculus frontatus	28.0	17.5	16	O	1
Pachycephala pectoralis	24.5	15.5	1.5	O	1
Pachycephala rufiventris	23.5	15	10.1	O	1
Colluricinla harmonica	66.0	17.1	22.5*	O	1
Psittacidae					
Eclectus roratus	527.5	26	80	P	1
Alisterus scapularis	232.8	20	35	P	1
Aprosmictus erythropterus	141.0	21	35	P	1
Polytelis swainsonii	147.5	20	40	P	1
Polytelis anthropeplus	172.5	21	42	P	1
Polytelis alexandrae	92.0	21	42	P	1
Purpureicephalus spurius	123.5	20	35	P	1
Barnadius zonaris	142.3	19	35	P	1
Barnadius barnadii	124.3	19	35	P	1
Platycercus caledonicus	122.5	19	35	P	1
Platycercus elegans	137.5	19	35	P	1
Platycercus venustus	96.0	19	49	P	1
Platycercus adscitus	121.5	19	35	P	1
Platycercus eximius	99.3	19	35	P	1
Platycercus icterotis	65.5	19	35	P	1
Northiella haematogaster	87.8	19	30	P	1
Psephotus haematonotus	64.8	19	28	P	1
Psephotus dissimilis	55.8	20	35	P	1
Psephotus crysopterygius	55.0	20	35	P	1
Neophema bourkii	46.8	18	20	P	1
Neophema chrystosoma	50.5	20	30	P	1
Neophema elegans	44.8	18	30	P	1
Neophema petrophila	51.8	18	30	P	1
Neophema chrystogaster	49.5	21	35	P	1
Neophema pullchella	39.3	18	30	P	1
Neophema splendida	39.3	18	30	P	1
Psezoporus wallicus	73.5		21	O(new)	1
Melopsittacus undulatus	27.8	18	35	P	1
Lathamus discolor	62.0	20	48	P	1

Tyrannidae					
Tyrannus tyrannus	39.1	15.5	17	O	2,5
Zosteropidae					
Zosterops lateralis	11.0	10	10.5	O	1

Appendix 5

Code for evolutionary models in Chapter 4

All code is in RESAMPLING STATS (Simon 1994). Annotations are indicated by quotations.

Nestling period evolves gradually. Nest preference changes when the nestling period crosses a threshold value

```
'a is np, b is nest with 1 = h and 0 = o"
repeat 15000
'seed a (mp)
generate 1 5,25 a
'set nest type (b), 0 is open, 1 is hole
if a < 15
set 1 0 b
end
if a >= 15
set 1 1 b
end
'evolution along a branch for a
set 1 0 count
'randomise time between speciations
generate 1 50,250 r
repeat r
add 1 count count
generate 1 -1,1 adda
add adda a a
'boundaries of a"
if a>25
set 1 25 a
end
if a <5
set 1 5 a
end
'threshold switching
if a < 15
set 1 0 bb
end
if a > 15
set 1 1 bb
end
'check to see if switch to reset time in nest type
if b <> bb
set 1 0 count
end
'the switch
set 1 bb b
score b countz
end
size countz endz
subtract endz 45 startz
take countz startz,endz lastz
clear countz
```

```
count lastz = b timez
'timez has to be less than 20 to count as new
'rank time in nestype, nh new hole, no new open,oh old hole,oo old open
if count < 15
if timez < 21
if b=1
score  a nh
end
if b=0
score a no
end
end
end
if count >=15
if b=1
score a oh
end
if b=0
score a oo
end
end
end
'print results
```

Nestling period evolves by random discrete changes. Nest preference changes when the nestling period crosses a threshold value.

```
'a is np, b is nest with 1 = h and 0 = o'
repeat 15000
'seed'
generate 1 5,25 a
'set nest type, 0 is open, 1 is hole'
if a < 15
set 1 0 b
end
if a >= 15
set 1 1 b
end
'evolution along a branch for a'
set 1 0 count
'randomise time between speciations'
generate 1 50,250 r
repeat r
add 1 count count
generate 1 -1,1 adda
multiply adda 10 adda
add adda a a
'boundaries of a'
if a>25
set 1 25 a
end
if a <5
set  1 5 a
end
'threshold switching'
if a < 15
set 1 0 bb
end
```

```
if a > 15
set 1 1 bb
end
'check to see if switch to reset time in nest type'
if b <> bb
set 1 0 count
end
'the switch'
set 1 bb b
score b countz
end
size countz endz
subtract endz 45 startz
take countz startz,endz lastz
clear countz
count lastz = b timez
'timez has to be less than 20 to count as new'
'rank time in nestype, nh new hole, no new open,oh old hole,oo old open
if count < 15
if timez < 21
if b=1
score  a nh
end
if b=0
score a no
end
end
end
if count >=15
if b=1
score a oh
end
if b=0
score a oo
end
end
end
'print results'
```

Nestling period evolves gradually. The probability of the nest preference changing increases as the nestling period changes.

```
'a is np, b is nest with 1 = h and 0 = o'
repeat 15000
'seed'
generate 1 5,25 a
'set nest type, 0 is open, 1 is hole'
if a < 15
set 1 0 b
end
if a >= 15
set 1 1 b
end
'evolution along a branch'
set 1 0 count
'randomise time between speciations'
generate 1 50,250 r
repeat r
```

```
add 1 count count
generate 1 -1,1 adda
add adda a a
'boundaries of a'
if a>25
set 1 25 a
end
if a <5
set  1 5 a
end
'probabalistic switching'
multiply a 5 m
subtract m 25 mm
divide mm 100 pr
uniform 1 0 100 u
divide u 100 u
if u<=pr
set 1 1 bb
end
if u>pr
set 1 0 bb
end
'check to see if switch to reset time in nest type'
if b <> bb
set 1 0 count
end
'the switch
set 1 bb b
score b countz
end
size countz endz
subtract endz 45 startz
take countz startz,endz lastz
clear countz
count lastz = b timez
'timez has to be less than 20 to count as new'
'rank time in nestype, nh new hole, no new open,oh old hole,oo old open'
if count < 15
if timez < 21
if b=1
score  a nh
end
if b=0
score a no
end
end
end
if count >=15
if b=1
score a oh
end
if b=0
score a oo
end
end
end
'print results'
```

Nestling period evolves by discrete changes. The probability of the nest preference changing increases as the nestling period changes

```
'a is np, b is nest with 1 = h and 0 = o'
repeat 15000
'seed'
generate 1 5,25 a
'set nest type, 0 is open, 1 is hole'
if a < 15
set 1 0 b
end
if a >= 15
set 1 1 b
end
'evolution along a branch'
set 1 0 count
'randomise time between speciations'
generate 1 50,250 r
repeat r
add 1 count count
generate 1 -1,1 adda
multiply adda 10 adda
add adda a a
'boundaries of a'
if a>25
set 1 25 a
end
if a <5
set  1 5 a
end
'probabalistic switching'
multiply a 5 m
subtract m 25 mm
divide mm 100 pr
uniform 1 0 100 u
divide u 100 u
if u<=pr
set 1 1 bb
end
if u>pr
set 1 0 bb
end
'check to see if switch to reset time in nest type'
if b <> bb
set 1 0 count
end
'the switch'
set 1 bb b
score b countz
end
size countz endz
subtract endz 45 startz
take countz startz,endz lastz
clear countz
count lastz = b timez
'timez has to be less than 20 to count as new'
'rank time in nestype, nh new hole, no new open,oh old hole,oo old open'
if count < 15
```

```
if timez < 21
if b=1
score  a nh
end
if b=0
score a no
end
end
end
if count >=15
if b=1
score a oh
end
if b=0
score a oo
end
end
end
'print results'
```

Appendix 6

Simulating a variable food supply in Chapter 6

In my first attempt at simulation, energy was allocated to chicks according to a normal distribution. I made the mean equal to the amount of energy required for the maximum mass growth rate in the lean-priority model when set at the fastest of the growth rates (that I had modeled) and in a chick of adult weight.

My reasons for doing this were (i) because fast growing birds are relatively unvarying in their growth rate, hence probably growing near their maximum rate, (ii) to provide me with a baseline from which to generate the mean amount of energy allocated to chicks with slower lean tissue growth rates (iii) to provide me with a mean energy set high enough to support the growth and metabolism of older (i.e., larger) chicks (this is realistic because energy requirements increase as chicks grow and parents may be able to provide more than enough food near the start of nesting but struggle to bring enough food to chicks later in nesting).

However, this method of simulating variable provisioning did not tend to produce the runs of poor food supply typical of a the variable food supply of swifts and tubenoses, and so simulated chicks were not in starvation conditions for extended periods.

My second approach, and the one used to generate the results of Chapter 6, was suggested by that used by Lima (1986) and Ricklefs and Schew (1994). Lima modelled a variable food supply (in a study of body fat in adult passerines) as the alternation of "good and bad" days according to a set of conditional probabilities (that specify the probability that a good day will follow a bad day, and vice versa). On good days the bird assimilates all the energy that it can use and on bad days it is not fed at all. In Ricklefs and Schews model, chicks are fed again on an "all or nothing" basis on each day, and this is decided on the basis of a randomly varying interval between each successful day.

On an ideal day the energy supply was equivalent to the total amount of energy a chick of same dry lean mass (*Ldm*) as the simulated chick (which will have different *Ldk* and *Ak*) and with the parameters (i.) $Ldk = 0.9$, (ii.) $Ak = 0.05$, (iii.) $Adm = 0$ could use. This is a chick with no fat, growing according to the lean-priority model at the

highest *Ldk*. This is a "baseline" amount of energy that I assumed to be the maximum amount of energy that a chick optimized for fast growth could assimilate.[1]

With a variable food supply, on each day a chick has 5 possible food supplies, each of equal probability; *a*. as in ideal conditions, *b*. 75% of the ideal conditions *c*. 50%, *d*. 25%, *e*, the chick is not fed.

This rule had the advantage of generating runs of very poor feeding - i.e., less energy than required for metabolism, hence only chicks that were able to convert a large amount of energy into fat on good days survived. However, it also meant that the two feeding conditions differ not only in variability, but also in the overall amount of food supplied to the chicks. However, the same effect occurs with the first approach discussed above when maximum fat deposition rates are relatively low, as in the lean-priority model, and to fast growing chicks in the cell allocation model (increasing the variance of the energy supply can only cause a decrease in the energy used by chicks that are already growing close to their maximum rate).

However, the aim these simulations was not *primarily* to compare chick survival in each model between a stable and a variable food supply. Rather it was to, (i.) compare the survival of chicks under the different models at various lean tissue growth rates in a variable food supply, (ii.) compare the model predictions (e.g., the change in *GRI*) in *ideal* and variable conditions (i.e., corresponding to maximum and mean mass growth rates in Chapter 3).

On average, simulated chicks with a variable food supply receive 50% of the ideal energy intake. Comparing this to Madeiran Storm-Petrel chicks (Appendix 3), this is probably too low, especially considering that my estimate of the ideal energy supply was probably too low (cf. Chapter 5 energy-savings model results). Petrel chicks in average conditions receive approximately 75% of the ideal overall energy intake. Only petrel chicks in very poor conditions may receive as little as 54% of the ideal. However, the results for real birds should be skewed upward because chicks with very low food supplies will starve to death and not be counted. Hence, the average energy supplied to petrel chicks is probably a somewhat smaller percentage of

[1] After having carried out this analysis I realised that this is not correct. Chicks with slower *Ldk* but with higher *Ak* may use even more energy at a given lean mass (cf. Appendix 3). Hence the chicks with slow lean tissue growth rates in the cell allocation model may assimilate even more energy in ideal conditions than I assumed.

the ideal than these figures indicate. By the same token, starved chicks *are* implicitly counted if average food intake is calculated as the expected food intake from the simulation food supply distribution. Calculating the amount of energy that *surviving* simulated chicks receive will give a figure considerably greater than 50%.

Appendix 7

Variations on the growth models in Chapter 6

In Chapter 6 I studied the lean-priority, uniform distribution, and fat-priority models. These are models that I used in phylogenetic comparisons in Chapter 3 and are alternative interpretations of Lack's insurance hypothesis. However, in Chapters 3 and 5 I also discussed the role of intrinsic constraints upon the supply of energy and nutrients to tissues in limiting growth rates. Here I have attempted to model intrinsic constraints on energy and nutrient supply in a modified version of the variable *Ak* lean-priority model. Also, all of these models have been *obligate* models of prioritisation. Also, in Chapter 8 I considered the possibility of *facultative* models of prioritisation. Therefore, here I have modified the fat-priority model to become a model of *facultative* fat prioritisation.

I will introduce each model in turn along with a description of their mathematical details how they differ from the lean-priority model. I will also present the results of simulations and discuss the models with respect to comparative data.

The supply tissue model

Lean tissue can be divided into body components such as wings or digestive tissue. In the lean-priority model, lean tissue is treated as an homogeneous mass. In this model, lean tissue growth is divided into energy supplying and energy consuming tissues.

During growth, the organs of the body can be divided into two main groups, consuming and supplying organs (Lilja 1982, 1983). The supplying organs fulfill the nutritional demands of the growing animal whereas the consuming organs can be adapted to different functional needs. Animals that have a rapid early development of supplying organs in relation to consuming organs ought to be characterised by a high growth rate (Lilja 1983).

High growth rates are correlated with high food consumption which are in turn related to the size of digestive organs (Latimer 1924, Lilja 1981,1982, Clum 1991). Thus, increased cell allocation to digestive tissues early in development could prepare the hatchling to exploit resources necessary for a fast growth rate (Ricklefs 1975, Dunn 1975, Clum et al 1995).

Lilja (1983) found that the relative size of the digestive tract decreases during growth in both geese and quail, and that a there is a close relationship between the weight of the digestive tract and daily food consumption. The energy supply available for growth processes is partly limited by the relative size of the digestive tract which thus limits growth rate. Geese grow very rapidly relative to weight. During the early stages of development the proportion of growth distributed to the digestive tract and to the liver is considerably greater than that distributed to the pectorals and the feathers. During the late stages of development the distribution of growth is biased towards the pectorals and the feathers.

Similarly, O'Connor (1977) found that in passerines the growth of the digestive tract is relatively rapid over the first few days after hatching. This period coincides with rapid overall growth. He therefore argues that growth rate is limited by the availability of energy and nutrient delivered from energy supplying organs.

In this model, the amount of energy made available for growth is limited by the relative size of the supplying organs. The supplying organs make up some fraction of the lean tissue. Reducing lean tissue growth rate involves a reduction in the growth rate of supply organs. This is represented by a reduction in the amount of energy that can be assimilated and distributed to the body at a given mass of lean tissue. The purpose of this model is to help test the hypothesis that species with slow growth rates with a variable food supply have slow growth because of reduced supply organs.

I faced two main problems. First, the way in which the size of the digestive tissue limits the supply of energy to growing tissues. Secondly, how to specify the trade-off between maximum fat deposition rate and maximum digestive tissue growth rate. For simplicity, I ignored the growth rate of other lean tissue, and assumed simply that there is a trade-off between allocation to digestive tissue and adipose

I approached the problem in several stages. First I estimated the amount of energy that a chick can assimilate at a given lean tissue mass. I started with the observation that growth rate varies allometrically with mass. I therefore assumed that the amount of energy that a chick can assimilate varies allometrically with mass. I took several species where mass, energy intake in a day, and growth rate are known (sources: Ashmole 1963, Bryant and Hails 1979, Ricklefs and White 1981, Ricklefs and Schew 1994, Klaasen 1994). I fitted an allometric regression ($r^2 = 0.95$) of energy

intake (*Ae*) to mass (*M*) (all parameters that did not appear in Chapter 6 are defined in Table A7.1) to obtain;

Equation A7.1 $$Ae = (5.3.M^{-0.32}).M$$

Ae	(Assimilable energy) Maximum energy content of food that can be assimilated by a given mass of digestive tissue on a given day (kJ)
Cd	Chick condition; mass relative to lean tissue development.
Df	(Digestive-tissue fraction) The fraction of wet lean tissue mass that is digestive tissue
Df_{max}	Maximum fraction of wet lean tissue that can be digestive tissue.
Dk	Digestive-tissue intrinsic growth rate
Dm	Digestive-tissue mass (g)
P	Fat prioritisation parameter; a constant relating fat deposition rate to chick condition.

Table A7.1 Summary of parameters used in the supply tissue and facultative fat-priority models not listed in Table 6.2, Chapter 6.

I calculated a similar equation for *Ae* relative to wet lean mass (*Lwm*). To do this, I estimated *Lwm* from mass by subtracting lipid mass from (total) mass where the references gave lipid content indices for body composition.

I next estimated a modified equation for a chick of a species optimised for rapid uptake of energy. To do this I used the parameter values that were two standard deviations above the mean in the allometric regression of energy intake on *Lwm*;

Equation A7.2 $$Ae = (8.2.Lwm^{-0.25}).Lwm$$

Next I assumed that the maximum amount of energy that a chick can assimilate in a day is directly proportional to the mass of its digestive tissue (*Dm*). I further assumed that the digestive tissue takes up some relatively constant fraction (*Df*) of the chicks lean mass (*Lwm*);

Equation A7.3 $$Dm = Df.Lwm$$

In reality, *Df* decreases over the course of development (Lilja 1983), but for simplicity I treated it as a constant throughout the nestling period.

I then assumed that the Equation A7.2 would hold in cases where digestive tissue makes up the largest possible proportion of lean mass (Df_{max}). The largest value for *Df* I could find in the literature was for the Greylag goose (*Anser anser*) (*Df* = 0.25, Lilja 1983). I used this as my estimate for Df_{max}. Where digestive tissue makes up a smaller proportion of *Lwm* ($Df < Df_{max}$) then energy assimilation will be proportionally less than that given by Equation A7.2, hence;

Equation A7.4 $$Ae = \left(\frac{Df}{Df_{max}}\right).(8.2.\ Lwm^{-0.25}).\ Lwm$$

This gives the intrinsic constraints on energy uptake in the supply tissue model. If *Ae* is less than energy supplied by the parents (*Ae* < *FS*) then actual intake of energy is *Ae*, otherwise it is equal to *FS*.

I assumed that at fast lean tissue growth rates (*Ldk* = 0.9), then $Df = Df_{max}$. This model assumes that *Ldk* is proportional to cell allocation - i.e., a reduction in *Ldk* means a reduction in cell allocation to lean tissue. This model also assumes that this reduction in cell allocation is entirely due to a reduction of cell allocation to supply tissue(i.e., digestive tissue). This means a reduction in the proportion of wet lean mass that is digestive tissue (*Df*). Therefore, my next problem was calculating *Df*, given an *Ldk* of less than 0.9 (I used the assumptions of the variable *Ak* lean-priority model for setting *Ak*).

To do this I first assumed that digestive *dry* tissue would itself have a maximum growth rate (*Dk*). According to the cell allocation hypothesis the ratio of the *mass* of digestive *wet* tissue to wet lean tissue (*Df*) will be approximately the same as the ratio of the *growth rate* of digestive wet tissue to wet lean tissue, and therefore also the ratio of growth rates for dry tissue - i.e., *Dk* to *Ldk*, hence;

Equation A7.5 $$Df = \frac{Dk}{Ldk}$$

If the reduction in cell allocation to lean tissue is entirely due to a reduction in allocation to digestive tissue, then reducing *Ldk* will be entirely due to a reduction in *Dk*, hence (following the format in Equation 6.4);

Equation A7.6 $$Dk_2 = Dk_1 + (Ldk_2 - Ldk_1)$$

Substituting this into Equation A7.5, this gives the new value for *Df* resulting from a reduction in *Ldk* (Df_2);

Equation A7.7 $$Df_2 = \frac{Dk_1 - (Ldk_1 - Ldk_2)}{Ldk_2}$$

Supply tissue model results and discussion

In the supply tissue model, the probability of fledging was lower than in the lean-priority model (Fig. A7.1). This was because at low supply tissue growth rates, mass growth rate was very slow. This can be seen form the low values for *GRI*.

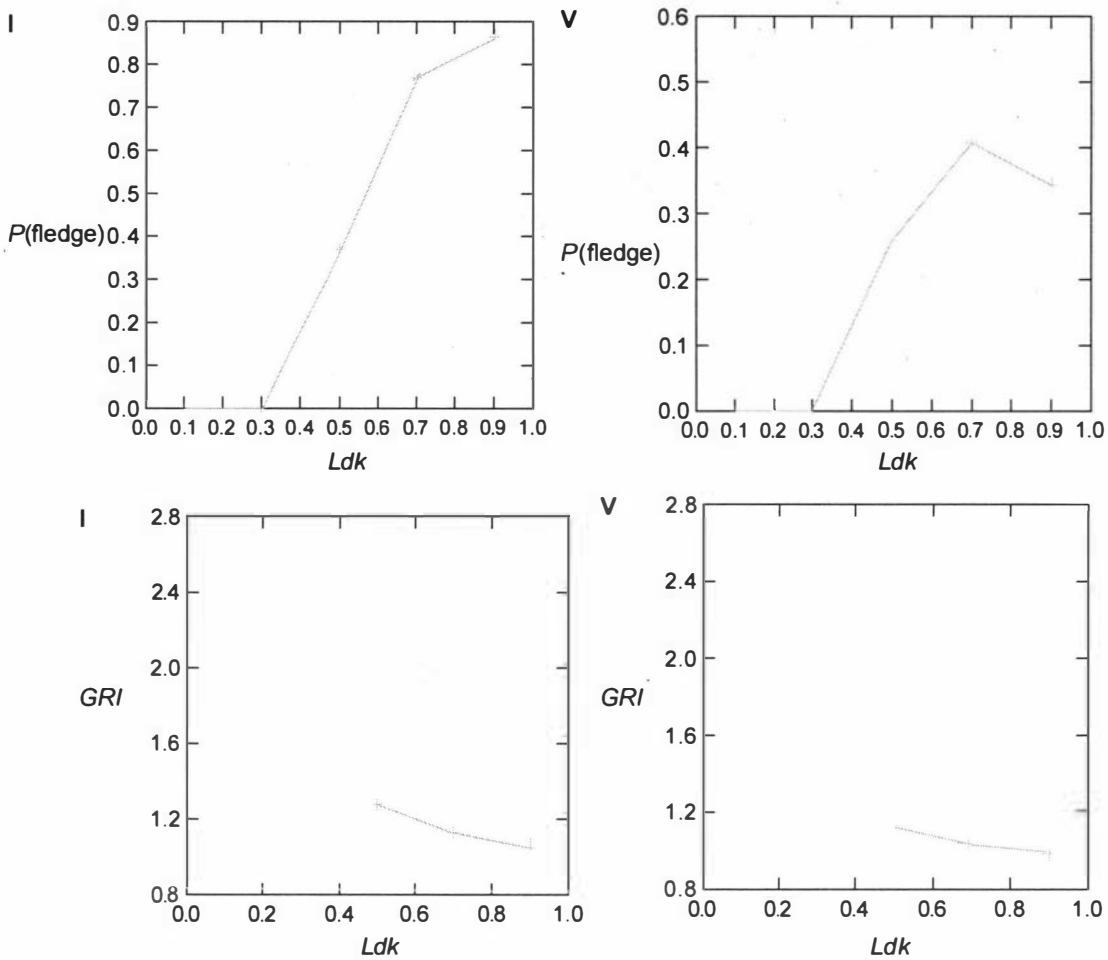


Fig. A7.1 Probability of fledging and *GRI* in the supply tissue model in relation to maximum lean tissue growth rate. See Chapter 6 and Table A7.1 for an explanation of the variables Conventions are as for Fig. 6.2, Chapter 6.

The supply tissue model does not compare well with my data. It predicts that species in a variable environment should have very slow mass growth rates, even with an ideal food supply. This is contradicted by the results of Chapter 3 (minimum mass growth period, hence maximum mass growth rates, are not significantly correlated with variability in food supply). However, increased allocation of cells to supply organs may be responsible for fast growth rates in some species. Fast growth rates require the precocial development of systems involved in the digestion, assimilation, and distribution of the raw materials for growth (Konarzewski *et al* 1989). Various work has supported the hypothesis that high growth rates are correlated with the size and growth rates of the digestive organs and that growth rates are limited by the rate at which energy and nutrients are made available to growing tissues (Latimer 1924,

Ricklefs 1967, Romanoff 1967, Jasper and Brasel 1974, Lodge et al 1977, O'Connor 1977, Lilja 1981, 1982, 1983, Geers et al 1982, Lilja et al 1985,Clum et al 1995). Growth rates will be limited by both extrinsic constraints (i.e., food supply) and intrinsic constraints (i.e., determined by supply organs) on the supply of energy and nutrients). Ricklefs (1969) has referred to this as the nutrient availability hypothesis.

In Chapter 3 I found a correlation between feeding frequency and minimum mass growth period (hence maximum mass growth rate). This raises the possibility that relatively small supplying organs may be an evolutionary response to a low overall food supply rather than a variable food supply. Decreasing allocation to supplying tissues may be a good strategy for species with a poor food supply. However, since this strategy may slow down the rate of adipose accumulation, it may not be a good strategy in a variable environment.

The facultative fat-priority model

Congdon's (1990) model (which I refer to as the "fat-priority model", Chapter 6) can be divided into two categories, a obligate system of prioritised fat deposition and a facultative system of fat deposition. In the facultative model, fat is only a priority when a chick is in poor condition.

In the facultative model the degree to which fat is prioritised can be represented by the x-intercept for the mass-lean growth rate curves in Fig. 3.6E (Chapter 3). Where this model differs from the fat-priority model is that the x-intercept is dependent upon chick condition, rather than fixed throughout development. For a chick in very good condition, fat prioritisation will be almost nil and the x-intercept will be close to zero. This is one possible way of modeling *adaptive hyperlipogenesis* (Chapter 5).

In the fat-priority model, *Ak* is fixed throughout the nestling period, but in this model *Ak* is adjusted according to the chicks condition. A chick in a poor condition places a high priority on fat deposition in comparison to a chick in good condition.

Chick condition (*Cd*) is the mass that a chick has relative to its state of structural (i.e., lean tissue) development (*Lwm*). The maximum amount of fat that a chick can carry in these models is set at 140% of *Lwm*, hence;

Equation A7.8 $$Cd = \frac{M}{1..4Lwm}$$

It is then a simple matter to make the maximum fat deposition rate (*Ak*) proportional to chick condition.

Equation A7.9 $$Ak = -P.Cd$$

where P (the fat prioritisation parameter) is a species-specific constant.

Facultative fat-priority model results and discussion

Results for the facultative fat-priority model were very similar to those for the simple fat-priority model (Fig. A7.2). Increasing the degree to which fat deposition changes with worsening body condition had a very similar effect to that of increasing the maximum fat deposition rate in the fat-priority model. This may be because the assumptions of the simple fat-priority model cause it to behave as if it was facultative. As body condition increases in the fat-priority model, the upper limit of fat deposition is reached. This decreases the maximum fat deposition increment for the day (*Agr*, Equation 6.3). This process is similar to that modelled in the facultative fat-priority model. However, mass growth rate was slightly higher with a variable food supply in the facultative model, and the probability of fledging slightly increased.

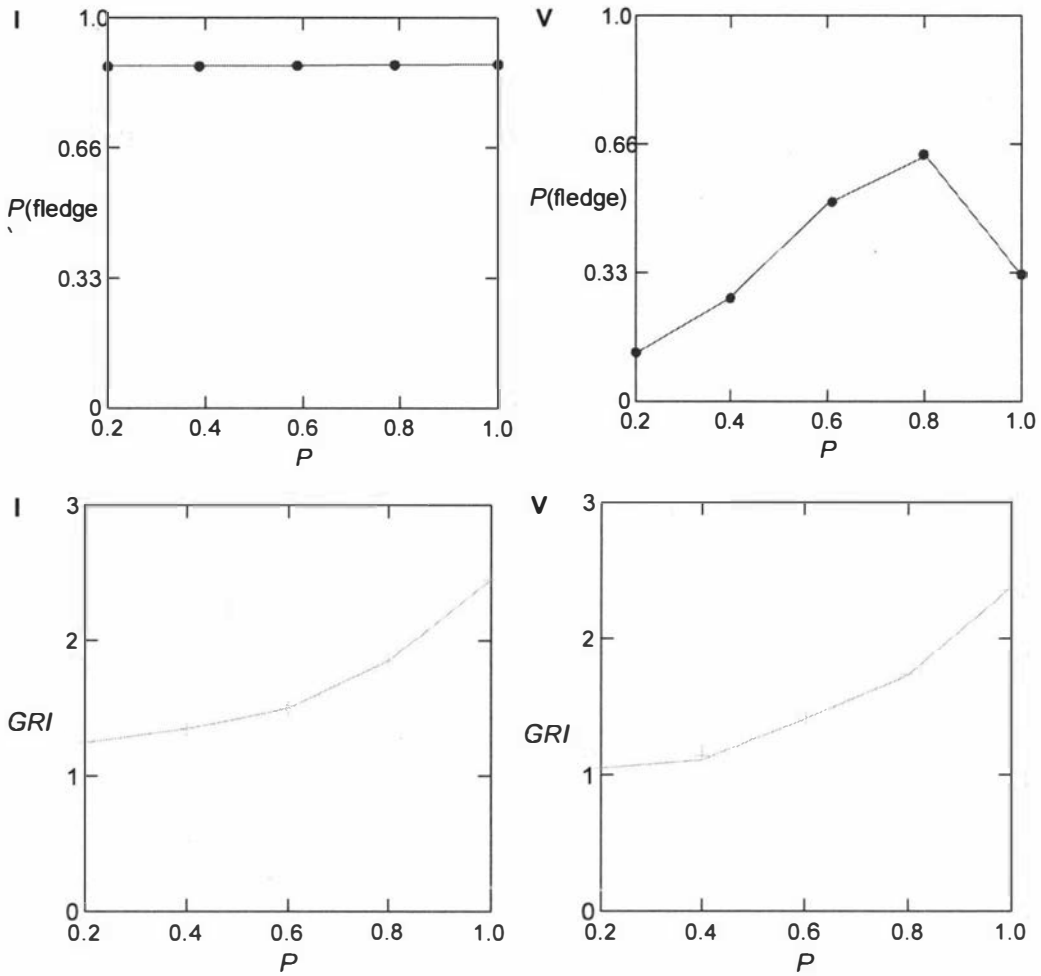


Fig. A7.2 Probability of fledging and *GRI* in the facultative fat-priority model in relation to the fat prioritisation parameter *P*. Higher levels of *P* mean greater increases in fat deposition rate with worsening body condition. Conventions as for Fig. 6.2, Chapter 6.

In the facultative fat-priority model, wings do not grow if the chick's body condition is poor. In Chapter 6 I discussed pauses in wing growth in the Swift, and found that the pauses were independent of mass growth rate (hence food supply). Here I investigated whether these pauses in wing growth are related to chick body condition.

In the facultative fat-priority model I calculated body condition using Equation A7.7. For real chicks I could not estimate either wet lean mass nor maximum possible mass. Therefore, for real chicks I used the index for body condition described in Chapter 8 (Equations 8.1 and 8.2).

I classified chicks as in poor condition if they less than or equal to -0.1. For zero wing growth, I used the criteria described in Chapter 6. Of 18 cases where Swift

chicks were in poor condition, in five cases the wings did not grow. In the 31 cases where Swift chicks were in good condition, wings always grew (Chi-square test, 1 df., $P < 0.01$). The mean condition of chicks where wings grew was 0.05, and the mean condition of the chicks where wings did no grow was -0.21 (1-tailed t-test, $P < 0.001$). Hence, when there was not wing growth, the chicks were in extremely poor condition. Therefore Swift chicks may cease wing growth when in very poor condition even if they have good mass growth (hence food supply, Chapter 6). This is consistent with the facultative fat-priority model.

Appendix 8

Code for growth models in Chapter 6

All code is in RESAMPLING STATS (Simon 1994). Line numbers are for reference only and are not part of the actual code. Annotations are indicated by single quote marks.

Lean-priority model

```
'Set starting parameters'
001     Set I 0.9 Ldk
002     Set I 0.05 Ak
003     Set 1 100 Lda
004     Set 1 1 Ldm
005     Set 1 0.75 Ad
006     Set 1 0 T
007     Repeat ***
'Calculate food supply FS'
008     Multiply Ldm 0.9 FSa
009     Divide Ldm Lda FSb
010     Subtract 1 FSb FSb
011     Mulptiply FSa FSb FSa
012     Multiply FS 20 FSa
013     Multply Ldm 3 Lwm
014     Set 1 Lwm  W
015     Power W -0.39 FSb
016     Multiply W FSb 6.1 FSb
017     Multiply 0.05 Ldm FSc
018     Multiply 1.4 Lwm MaxW
019     Divide W MaxW FSd
020     Subtract I FSd FSd
021     MultiplyFSd FSc 38 FSc
022     Add FSa FSb FSc IFS
023     Set 1 IFS FS
024     Generate 1 0,100 var
025     If var < 20
026     Set 1 0 FS
027     End
028     If var <40
029     If var >19
030     Multiply 0.25 IFS FS
031     End
032     End
033     If var < 60
034     If var >39
035     Multiply 0.5 IFS FS
036     End
037     End
038     If var < 80
039     If var > 59
040     Multiply 0.75 IFS FS
041     End
042     End
'Calculate Egr'
043     Multiply Ldm Ldk Egra
```

```
044     Divide Ldm Lda Egrb
045     Subtract 1 Egrb Egrb
046     Multiply Erga Ergb 20 Egr
'Calculate metabolic costs Mc'
0467    Add Lwm Ad W
048     Power W -0.39 Mc
049     Multiply W Mc 6.1 Mc
'Calculate lean tissue growth'
050     Subtract FS Mc Eag
051     If Eag > 0
052     If Egr < Eag
053     Set 1 Egr Eld
054     End
055     If Eag > Egr
056     Set 1 Eag Eld
057     End
058     Divide Eld 20 Ldi
059     Add Ldm Ldi Ldmm
060     Score Ldmm Ldm
061     End
'Calculate fat deposition'
062     Multiply Ak Ldm Agra
063     Divide W MaxW Agrb
064     Subtract 1 Agrb Agrb
065     Multiply Agra Agrb Agr
066     Multiply Agr 38 AEgr
067     If Eag > Egr
068     Subtract Eag Egr Aea
069     Set 1 AEgr Ef
070     If AEgr > Aea
071     Set 1 Aea Ef
072     End
073     Divide Ef 38 Adi
074     Subtract MaxW Lwm AAd
075     Add Ad Adi ADC
076     If ADC > AAd
077     Subtract AAd Ad Adi
078     End
079     Add Ad Adi Add
080     Score Add Ad
081     End
082     If Eag < 0
083     Divide Eag 38 Adl
084     Add Ad Adl Ad
085     End
'Age'
086     Add T 1 TT
088     Score TT T
'C8eck for death'
089     Generate 1 1,1000 Pr
090     If Pr <= 15
091     Set 1 1 Ldm
092     Set 1 0.75 Ad
093     Set 1 0 T
094     Score 1 Dth
095     End
096     If Ad < 0
097     Set 1 1 Ldm
098     Set 1 0.75 Ad
```

```
099     Set 1 0 T
100     Score 1 Dth
101     End
102     Divide 4.44 Ldk To
103     Multiply To 1.4  Tmax
104     Multiply Lda 0.75 Ldmin
105     If T > Tmax
106     If Ldm <          Ldmin
107     Set 1 1 Ldm
108     Set 1 0.75 Ad
109     Set 1 0 T
110     Score 1 Dth
111     End
112     End
'Check for fledging'
113     Multiply 0.95 Lda F
114     If Ldm > Lda
115     Set 1 1 Ldm
116     Set 1 0.75 Ad
117     Set 1 0 T
118     Score 1 Fldg
119     End
Records
120     Score Lwm Lwmz
121     Score W Wz
122     End
123     Sum Dth Dth
124     Sum Fldg Fldg
125     Sum Dth Fldg Total
```

Uniform distribution model

This program is the same as for the lean-priority model up to and including line 050 and after line 085, hence the following code replaces lines 051 to 084 in the lean-priority program.

```
'Calculate lean tissue growth'
051     Set 1 0.5 R
052     Multiply R Eag El
053     Subtract Eag El Ea
054     If El > 0
055     If Egr < El
056     Set 1 Egr Eld
057     End
058     If El > Egr
059     Set 1 El Eld
060     End
061     Divide Eld 20 Ldi
062     Add Ldm Ldi Ldmm
063     Score Ldmm Ldm
064     End
'Calculate Agr'
065     Multiply Ak Ldm Agra
```

```
066     Divide W MaxW Agrb
067     Subtract 1 Agrb Agrb
068     Multiply Agra Agrb Agr
'Calculate fat deposition'
069     Multiply Agr 38 AEgr
070     If Ea >0
071     Set 1 AEgr Ef
072     If AEgr > Ea
073     Set 1 Ea Ef
074     End
075     Divide Ef 38 Adi
076     Subtract MaxW Lwm AAd
077     Add Ad Adi ADC
078     If ADC > AAd
079     Subtract AAd Ad Adi
080     End
081     Add Ad Adi Add
082     Score Add Ad
083     End
```

Fat-priority model

This program is the same as for the lean-priority model up to and including line 050 and after line 085, hence the following code replaces lines 051 to 084 in the lean-priority program.

```
'Calculate AEgr'
051     Multiply Ak Ldm Agra
052     Divide W MaxW Agrb
053     Subtract 1 Agrb Agrb
054     Multiply Agra Agrb Agr
055     Multiply Agr 38 AEgr
'Calculate fat deposition'
056     If Eag > 0
057     If AEgr < Eag
058     Set 1 AEgr Ef
059     End
060     If Eag > AEgr
061     Set 1 Eag Ef
062     End
063     Divide Ef 38 Adi
064     Subtract MaxW Lwm AAd
065     Add Ad Adi ADC
066     If ADC > AAd
067     Subtract AAd Ad Adi
068     End
069     Add Ad Adi Add
070     Score Add Ad
071     End
'Calculate lean tissue growth'
072     If Eag > AEgr
073     Subtract Eag AEgr El
074     If Egr < El
075     Set 1 Egr Eld
076     End
077     If El > Egr
078     Set 1 El Eld
```

```
079     End
080     Divide Eld 20 Ldi
081     Add Ldm Ldi Ldmm
082     Score Ldmm Ldm
083     End
```

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