ENERGETICS, ENCEPHALISATION AND WEANING:

MODELLING GROWTH AND MATURATION

IN PRIMATE AND HUMAN EVOLUTION

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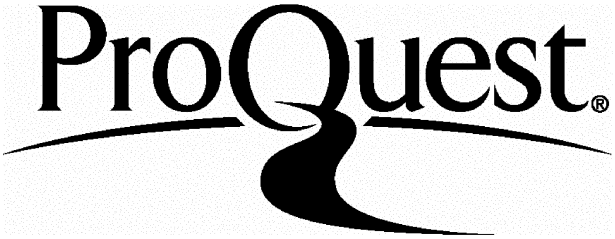

ABSTRACT

The evolution of encephalisation (relatively large brains) in primates has been linked to the evolution of other unusual aspects of primate life histories, in particular, slow infant growth. Mothers supply the majority of the infant's energetic needs during infancy, and infant growth in the lactation period reflects maternal investment. Weaning represents the cessation of this investment, and is an important developmental milestone. This thesis investigates the impact of encephalisation on growth, lactation and weaning strategies in a variety of haplorhine primate taxa, with particular emphasis on a relatively encephalised species, *Cebus*.

The results of both inter- and intraspecific analyses show that weaning in primates coincides with the inflexion of the postnatal brain growth allometry i.e. when the majority of brain growth is completed. This suggests that weaning is associated with brain, as well as body, growth. Furthermore, capuchin brain and body growth can be dissociated under nutritional stress, with brain growth protected at the expense of body growth. Brain growth is also shown to be associated with specific weaning behaviours, e.g. suckling frequency, as well as more general measures of infant behavioural, locomotor and physiological independence.

The energetic and life history correlates of brain and body growth are also examined. Maternal metabolic capacity (inferred from maternal mass) influences fetal brain growth and postnatal infant body growth separately. Paternal mass influences fetal body growth. Weaning age is associated with postnatal brain growth such that infants which undergo more brain growth wean later. Humans and capuchins are shown to be similar in some aspects of ontogeny, and capuchin growth and maturation are discussed in the context of human adaptation and life history.

CONTENTS

CONTENTS

CONTENTS

CONTENTS

CONTENTS

TABLES

page

TABLES

page

FIGURES

page

FIGURES

FIGURES

FIGURES

FIGURES

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INTRODUCTION

i.i Aims

This thesis examines primate growth in the context of encephalisation. As we shall see in this introductory chapter, mammalian mothers must support all the nutritive and energetic needs of their infant for the majority of the lactation period. Infant brain and body growth prior to weaning are largely determined by maternal resources, and maternal metabolism is predicted to limit the rate of nutrient transfer, and therefore growth. This investment in infant growth places an energetic burden on the mother, and reflects an optimisation of maternal resources, i.e. represents a trade-off between investment in current and future offspring. Weaning will occur when the mother's priorities switch to the next reproductive event. In many mammalian taxa, infants are weaned when they reach a certain body mass relative to birth mass. This suggests that the energetic demands of the infant influence maternal investment decisions. When a mother can no longer meet the energetic demands of her infant, and the infant is large enough to support itself, it is weaned.

This weaning 'threshold' is also seen primates. However, compared to other mammals, primate infants are unusually 'expensive': they have relatively large brains compared to non-primate taxa at similar body sizes, and the majority of brain growth occurs in the gestation and lactation periods. As brain tissue is expensive to maintain and grow, it is likely that encephalisation increases the energetic burden on the mother during lactation. It can therefore be expected that encephalisation will influence primate investment and growth via the increased energetic demand of a large brains. Brain size is therefore expected to play a role in determining the weaning threshold in primates.

The infancy period in primates is characterised by slow growth, low milk energy content and a late age at weaning. More generally, primate life histories are slow: they grow, reproduce and mature at a slow rate. Previous studies have noted the tight correlation between brain size and life history variation. One explanation for these associations might

be that primate mothers spread the energetic cost of their expensive offspring over a long period. Other models of primate life history suggest that non-metabolic (i.e. behavioural/physiological) mechanisms underlie the relationship between lager brains and slow growth.

This thesis investigates the relationship between relative brain size and maternal investment during lactation in the context of life history variation. It asks: how does the size of the primate brain influence primate strategies of lactation and weaning? Several issues are investigated:

- Do different primate species share a common pattern of postnatal brain and body ontogeny, and how does this pattern compare with that seen in other mammals?

Necropsy data from nine haplorhine taxa are used to test an allometric model of mammalian growth. The model predicts that the rate of brain mass increase relative to body mass increase is constant during the postnatal period i.e. that the primate brain allometry is linear, and that the exponent describing brain and body growth is similar to that seen in other mammalian taxa. This model is tested at both the inter- and intraspecific level. Data for the intraspecific analysis come from two species of capuchin monkey (*Cebus*). After humans, capuchins are the most encephalised primate taxon, and encephalisation is predicted to be an important determinant of growth in *Cebus*. Capuchin brain allometries are modelled using longitudinal endocranial vault area and body mass data taken from a group of captive animals.

- How do ontogenetic changes in brain and body size relate to lactation and weaning strategies?

Several predictions about brain and body size at weaning are tested against the inter- and intraspecific data. Firstly, the prediction that brain size is an important component of the

weaning threshold in primates is tested. Weaning data are integrated with the allometric model of brain and body growth. Secondly, the prediction that brain growth is associated with weaning mass independently of body growth is investigated. Data for this part of the analysis come from a comparison of normal and nutritionally-stressed capuchins.

- How do ontogenetic changes in brain and body size relate to other aspects of primate life history?

The behavioural and physiological aspects of weaning that are specifically linked to brain growth are investigated using various data from the literature, and the hypothesis that brain growth is important in setting the pace of primate life histories is tested. The energetic costs of brain and body maintenance are modelled, and their relationship to maternal size parameters examined. The implications these results have for the evolution of a highly encephalised species, *Homo sapiens*, are discussed at the end of the thesis.

i.ii Life history theory

Before discussing primate ontogeny, the underlying theoretical framework on which this thesis rests must be examined. The analyses presented here examine growth and investment in the context of life history. Life history theory models the suite of time and size parameters that characterise a species' 'pace' of life – the speed at which it lives, reproduces, and dies – as a function of intrinsic fitness trade-offs and extrinsic mortality pressures (Stearns, 1989; Stearns, 1992). As Hill (1993) notes, "the basic tenet of life history theory... is the principal of allocation, which states that energy used for one purpose cannot be used for another" (p79). For example, energy invested in body tissues during growth cannot be used for reproduction, and energy invested in one infant cannot be invested in another. Hill therefore suggests that "the two most fundamental trade-offs, which are at the centre of all life history theory, are those between current and future

reproduction and between the number of and fitness of offspring" (p79ff.). Thus the age at which an animal reaches reproductive maturity (and by extension, the patterns and pace of growth), the length of the reproductive career, the frequency with which it reproduces, and the length of the lifespan are seen as adaptive responses to selection pressures, and life histories reflect the fitness of the phenotype for reproductive success (Promislow & Harvey, 1990; Charnov, 1991; Stearns, 1992). This thesis tests the hypothesis that the fitness trade-offs a mother makes during lactation (measured as investment in growth of the infant) are directly influenced by encephalisation.

Originally developed to describe island biogeography (MacArthur & Wilson, 1967), life history theory has developed into two more wide-reaching models. The first predicts that life history variation is influenced by density-dependent and density-independent mortality (Pianka, 1970). Animals living in environments where mortality is density-independent – i.e. is not linked to the number of individuals in a population – are predicted to evolve 'fast' life histories. Individuals will mature and begin reproducing early, reproductive rates and litter size will generally be high, lives will be short and the population will expand to make use of the available resources. In this environment, there are few limits on population growth and the rate of expansion is primarily determined by the intrinsic rate of population increase, or *r*. Above a certain population size, however, density-dependent mortality becomes an important selective pressure, and population growth will be matched to the carrying capacity (*K*) of the environment. Life histories will then tend to be 'slow', with low birth and growth rates, late ages at maturity and a relatively long lifespan. Reproductive strategies reflect the quality-not-quantity approach: investment in fewer offspring that are more likely to survive until adulthood. As we shall see in Chapter 6, selection along this *r*-*K* continuum is associated with different habitat types (Ross, 1991; Ross, 1992b). *r*-selection typically occurs in relatively unstable and unpredictable habitats where there are periods of plenty interspersed with population crashes, whilst *K*-selection is associated with stable, predictable and resource-rich environments. The association between risky environment and maximal infant growth rates

has been observed in some taxa; for example, differences in growth rates between ape taxa may be a function of ecological risk (Leigh & Shea, 1997), as may variation in interbirth interval in some neotropical primates (Fedigan & Rose, 1995). However, recent research has failed to find statistically-robust support for this correlation of life history and habitat (Ross & Jones, 1999), and some taxa show a mixture of *r*- and *K*-selected life history traits: *Varecia variegata*, for example, is a lemur that has the highest *r* value (corrected for body size) of all the primates, but lives in a tropical habitat more usually associated with slow life histories (Rasmussen, 1985).

These inconsistencies have led some authors to conclude that a simple *r-K* model cannot account for the complexities of life history variation (Martin, 1990; Charnov, 1991; Clutton-Brock, 1991; Charnov & Berrigan, 1993). Charnov (Charnov, 1991; Charnov & Berrigan, 1993) is the main proponent of a second model that uses age-specific, rather than density-dependent/independent, mortality to predict life histories. The infant (birth to weaning), juvenile (weaning to first reproduction) and adult periods are each associated with a characteristic intrinsic mortality rate (Sibley *et al.*, 1997), and Charnov models the effects of these on rate of reproduction and growth in mammals. Mortality is described in a simple function of growth and age at maturity, scaled to body size; adult body size is determined by mortality acting through selection on age at maturity. When adult mortality rates are high relative to infant and juvenile mortality, animals are expected to mature quickly and begin breeding as soon as possible. When mortality is low, there is less pressure to grow quickly, and animals can afford to invest more in their offspring. Both strategies maximise lifetime reproductive success and contribute to the rate at which a population grows to fill a habitat (see Chapter 6 for further discussion). The model makes several assumptions that may or may not be appropriate for all mammalian taxa (e.g. that reproduction begins only when growth stops, and that weaning mass is a constant proportion of adult mass), but in primates, the predicted relationships between life history variables are generally supported by the available data (Western & Ssemakula, 1982). In practice, pre-adult mortality is usually high in stable primate populations, and variation in

life history variables usually result in differences in adult mortality alone (Clutton-Brock, 1991). For example, primates with high rates of adult mortality begin weaning and reproduction early, spend less time in post-weaning and have high birth rates (Harvey *et al.*, 1989; Promislow & Harvey, 1990; Ross & Jones, 1999). Other predictions are less well-supported in an analysis that controls for phylogeny; for instance, low adult mortality is not significantly associated with a long juvenile period, and some of allometric exponents predicted by Charnov's model are non-significant or reversed (Ross & Jones, 1999). Despite these inconsistencies, one feature of primate life histories is apparent across most analyses: primates have very low reproduction rates compared to other animals, and take a long time to grow and reach reproductive maturity. The 'slowness' of primate life histories is discussed further below.

i.iii Body size constraints and adaptation

One important factor in life history variation has not yet been mentioned: body size. In general, animals that reproduce slowly tend to be large and those that reproduce quickly tend to be small (Harvey *et al.*, 1987). Many life history traits therefore show an allometric relationship with body mass, i.e. scale non-linearly with body size (Peters, 1983; Calder, 1984; Kirkwood, 1985). This allometric association of body mass and life history traits is strong: for example, approximately 70 to 80% of variation in female age at first reproduction, birth rate and r value is explained by variation in body mass, although this percentage drops when phylogenetic contrasts are used (Ross & Jones, 1999). Some authors view body mass as a constraining factor in life history evolution (Western & Ssemakula, 1982). Animals that are large take time to grow and reproduce, and will therefore follow relatively slow life histories. However, many life history traits remain highly correlated even when the effects of body size are removed, for example between age at maturity and life expectancy (Harvey *et al.*, 1989; Smith, 1992; Kozlowski & Weiner, 1997). This suggests that life histories are selected for independently of body size. The

models described above assume that life history variation is adaptive, not just a by-product of size selection; Charnov's (1991) model proposes that adult body mass is largely determined "by adult mortality through the evolution of [age at maturity]" (p1135). Thus selection on a late age at maturity produces larger-bodied adults, whilst selection on early maturity favours the evolution of a small body size. As mortality tends to scale with body size (Charnov, 1991), this model may explain the body size effect on life histories.

Kozlowski & Weiner (1997) extend Charnov's model to a more general context of body size evolution. They argue that the life history allometries observed across species are the result of body size optimisation. They note that "interspecific allometries seem to provide very strong support for the idea that optimisation of age and size at maturity based on optimal allocation of resources is indeed a powerful selection force" (p376).

i.iv Primate encephalisation

The empirically-derived allometric exponent of 0.75 that describes the relationship of brain to body size in all mammals (see below) indicates that brain and body masses do not increase linearly with each other; rather, as body sizes get bigger, animals tend to have relatively smaller brains for their body size (Martin, 1990). However, at all body sizes, primates are more encephalised in comparison with the majority of other mammals. On average, primates have twice as much brain tissue per unit body mass (Sacher, 1982; Fig i.i). Only a few non-primate taxa have brains that are relatively as large as primates, for example cetacea which, after humans, are the most encephalised extant mammal (Marino, 1996).

In terms of brain specialisation, primates, like most taxa, show a mosaic of derived features within the order (Barton, 1999). Although the majority of brain structure variation can be explained by variation in brain size (Finlay & Darlington, 1995), residual variation has been linked to functionally-significant adaptive variation (Barton, 1999). The most important source of variation among the primates is probably adaptation to diurnality and

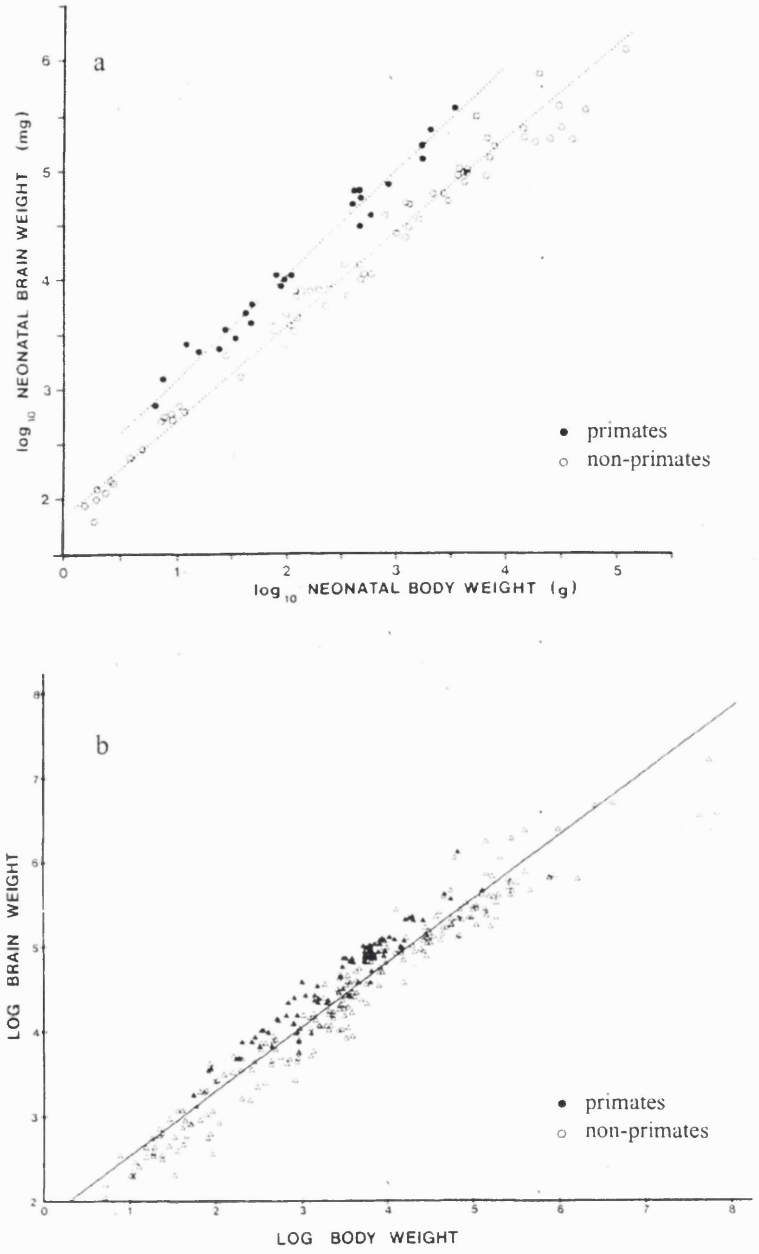


Figure i.i. Primates are relatively encephalised, both as neonates and adults. (a) Neonatal brain weight [mass] over neonatal body weight [mass]. (b) Adult body weight[mass] over adult body weight [mass]. Taken from Martin, 1983. Closed symbols are primates, open symbols are non-primate mammals. Lines fitted are major axes.

diurnal feeding: species that are active in the day have larger visual cortices and smaller olfactory bulbs than do nocturnal primates (see Chapter 2 for further discussion of brain structure variation in primates). These 'compensatory' size adaptations have been noted in non-primate taxa (e.g. Sherry *et al.*, 1989), and probably have only a small effect on changes in net brain size (Barton, 1999).

Brain size has been suggested as an important determinant of life history variation, in addition to the factors discussed in the previous section (Sacher, 1982; Martin, 1983; Harvey & Clutton-Brock, 1985; Pagel & Harvey, 1988; Allman *et al.*, 1993b; Pagel & Harvey, 1993; Martin, 1996; Joffe, 1997; Lee, 1999). In fact brain size is a better predictor of some life history traits than body size in primates: for example, the length of the lifespan, and the timing of developmental markers such as gestation, age at M1 eruption and age at first reproduction, are all highly correlated with brain size when the effects of body size are removed (Sacher, 1982; Harvey & Clutton-Brock, 1985; Harvey *et al.*, 1987; Smith *et al.*, 1995). Some parts of the brain correlate better with life history variation than others; for example, the cerebellum, which decreases in size later in life, is the best predictor of maximum lifespan in primates (Allman *et al.*, 1993a). Some statistical and theoretical problems of analysing residual brain size correlations have been outlined (e.g. the 'Economos effect', which stems from the inherently 'messy' nature of body mass as an estimate of general body size, Barton, 1999). In general, however, when body size is controlled for, animals with large brains grow slowly, live long lives and reproduce slowly.

i.v Brain size and maternal investment

Why might this association of brain size and life histories have arisen? One possibility is that the size of the brain is constrained by the kinds of maternal investment* trade-offs that

*Although the term 'parental investment' can be used to describe any characteristic or action of the parent that increases the fitness of their offspring at a cost to any component of the parent's fitness (Clutton-Brock, 1991), here it is used specifically to denote energetic investment in infant brain and non-brain tissue mass increase.

characterise life histories (Stearns, 1989). Some authors have argued that these investment trade-offs are metabolic; for example, Martin (1983; 1990; 1996) points out that, although relative adult metabolism does not correlate with adult relative brain mass in mammals (McNab & Eisenberg, 1989), adult brain size scales to adult body size with the same exponent as adult metabolism scales to adult body size, i.e. 0.75. Furthermore, an association is observed between metabolic rate, relative brain size and gestation length. In an analysis of data from 53 primate species, Martin (1996) finds that adult brain mass correlates positively with basal metabolic rate and gestation period, with the effects of body size held constant. On the other hand, gestation length and metabolic rate are negatively correlated. From this, he infers that it is the maternal metabolic capacity during the gestation period that regulates fetal brain growth. Brain tissue is relatively expensive to maintain (see Chapter 5), and the rate at which it increases in mass is limited by the rate of nutrient transfer from the mother to the fetus (Martin, 1996). The maternal energy hypothesis also predicts, therefore, that the effects of metabolism on brain mass at birth are mediated by variation in gestation length.

In mammalian taxa, the majority of brain growth is complete by birth. However, in primates and some other altricial taxa, brain growth continues into the postnatal period. Martin points out that the tight association of neonatal brain size and body size becomes much looser at adulthood (Fig i.i), and goes on to suggest:

> "Both the length of gestation and the period of postnatal growth supported by lactation could exert modifying effects [on brain growth], thus explaining why there is no tight scaling between the brain size of an adult and its own basal metabolic rate."
>
> Martin (1996:154).

The maternal energy hypothesis has been criticised by several authors. Although most are now agreed that the exponent linking interspecific values of brain size to body size is 0.75 (Bauchot, 1978; Eisenberg, 1981; Hofman, 1982; Pagel & Harvey, 1988;

Martin, 1996; but see Jerison, 1977), when independent contrasts are used, the slope is closer to the previous consensus estimate of 0.67 (Barton, 1999). Furthermore, despite the well-established links between neonatal brain size and maternal mass found in primates (e.g. Leutenegger, 1977), other analyses have failed to find an association of neonatal brain size and maternal metabolism more generally (Pagel & Harvey, 1988). It is possible that the unusually large size of the primate brain brings to the fore allometric trends that are unimportant in other taxa. For example, the energetic challenge facing avian taxa is not to invest in big brains but in big hearts, important organs in powering flight (Daan *et al.*, 1990). Dietary adaptation may also play a part in obscuring trends: for example, frugivores tend to have large brains but low metabolic rates (Kurland & Pearson, 1986; Elgar & Harvey, 1987). Similarly, the metabolic and size associations observed by Martin become less clear when phylogeny is accounted for; for instance, Barton (1999) finds no association between primate brain size and maternal energy variables (controlling for body size) when an independent contrasts method is used. Thus the link between maternal metabolism and infant brain size remains enigmatic. In human neonates and adults, for example, the brain is extremely large in relation to the body, but metabolic rates are exactly as predicted from body size (Kleiber, 1947) ~~(Ross, 1992a)~~. Humans may cope with the extra energetic costs of growing large brains by cutting the size (and cost) of other 'expensive' tissues such as the digestive tract (Aiello & Wheeler, 1995); this possibility is discussed in more detail in Chapter 7.

i.vi Maternal investment during lactation

Martin's hypothesis focuses on the prenatal period, but he acknowledges that growth in the postnatal period, especially during lactation, is an important source of variation in adult morphology. Lactation evolved as a relatively efficient method of mother-infant energy transfer, allowing females to continue their investment in offspring growth and survival after parturition by providing a relatively constant energy supply that buffers the infant

from nutritional inadequacies (Pond, 1984; Prentice & Whitehead, 1987). The majority of primate subadult mortality tends to occur at the end of the maternal dependency period: in general, juveniles are less efficient foragers than older individuals (Boinksi & Fragaszy, 1989; Janson & van Schaik, 1993), are more susceptible to starvation (Hamilton, 1985), and are more often the subjects of predation (Boinksi, 1988). Weanlings are also less likely to have developed the physiological and/or behavioural capacity to resist mortality pressures, are less able to compete effectively in taxa where conspecific competition is high, and are less able to exploit food resources available to their larger peers (Janson & van Schaik, 1993). Many of these features of survival relate to body size, and infants that have achieved a certain size by the end of the maternal dependency period are likely to have increased survival chances (Lee, 1987). Growth during lactation is therefore vital to survival once lactation has ended.

Although lactation did not necessarily arise as a means to regulate infant growth (Pond, 1984), the metabolic constraints observed in gestation are likely also to be important in the postnatal growth period (Martin, 1996). For example, the ratio of littermass to maternal metabolic capacity is a good predictor of milk energy output in most mammalian groups (Oftedal, 1984). However, milk energy output is protected in poorly-nourished human females (Prentice *et al.*, 1986), suggesting that infant energy needs are met even in sub-optimal nutritional conditions. In extreme cases, these largely obligate energy demands can be to the detriment of the mother's own survival chances (Mattingley & McClure, 1985) or to subsequent reproduction (Prentice *et al.*, 1986). Mothers and infants are therefore competing for the same maternal energy resources, and throughout lactation a mother is faced with a life history decision: to continue investing in her current infant, or to invest in the potential for further reproduction (Altmann *et al.*, 1978; Lee, 1987). For the mother, a decline in milk output signals the resumption of normal cycling; for the infant, the same decline represents the inability of the mother to meet the infant's energy needs (Lee *et al.*, 1991). Conflict between infant and maternal interests arises when maternal priorities switch from current to future offspring (Trivers, 1974). Weaning can be

seen as the resolution of this conflict: when the mother no longer meets the infant's demands, the infant must turn to other sources and weaning is initiated (Lee, 1987).

One indicator of the energetic burden placed on a female during lactation is the weaning threshold observed by Lee *et al.* (1991). Weaning tends to occur once an infant's body mass weighs four times birth mass; this suggests that 1] the mother is no longer capable of supplying the infant's energetic needs above this birth mass multiple, and 2] that infants bigger than four times birth mass are generally able to support themselves energetically. Although the weaning threshold is variable across taxa, the relationship is generally true across a wide range of different mammalian species. In other words, species with different life histories follow a similar different body sizes and adaptations (e.g. pinnipeds, ungulates; Lee *et al.* 1991). In lactation trend: the termination of maternal investment is determined to some extent by the metabolic demands of the infant, and weaning occurs during a 'window' of infant body size increase (see below and Chapter 1 for further discussion). It can be predicted that taxa with relatively large, expensive brains will grow in such a way to accommodate the energetic constraints placed on them by such a threshold.

In primates, as we will see below, these constraints have probably been met by extending the time over which lactation occurs (Lee, 1996). However, the direct association of brain size and lactation length is likely to be mediated by other variables. In her analysis of primate growth and lactation, Lee (1999) finds that the duration of lactation is not directly linked to brain growth when maternal and infant body masses are controlled for (Fig i.ii). Rather, lactation length is positively correlated with weaning mass and negatively correlated with the infant growth rate. In other words, infants that suckle for longer grow slowly but are bigger at weaning. The link with brain size comes from the fact that both weaning mass and the infant growth rate are correlated with brain growth: infants that are bigger at weaning and that grow slowly tend to undergo more postnatal brain growth. Thus body size and rate of growth both mediate the effects of lactation length on brain growth.

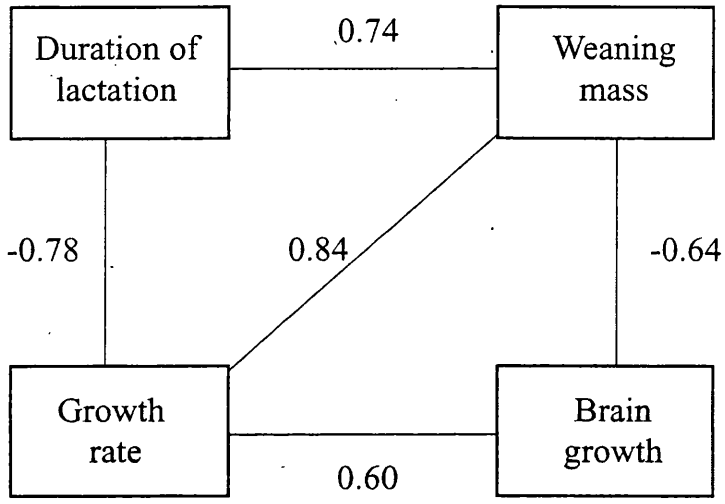


Figure i.ii. The association of lactation and brain growth is mediated by other variables in primates. Based on Lee (1999). Values are partial correlation coefficients. Infant and maternal mass size is held constant in each partial correlation, as well as other independent variables entered into the analysis.

i.vii Primate lactation: the 'long and slow' approach

It was noted above that primates have slow life histories. Infant growth conforms to this pattern of 'long and slow' development. Weaning occurs late in relation to body size in most primate taxa, and infant growth occurs over a long period and at relatively low rates (Kirkwood, 1985; Charnov, 1991; Ross & Jones, 1999). Within the haplorhines, different taxa show different growth trends even when body size is taken into account: apes and old world monkeys tend to show the slowest growth rates followed by the large new world monkeys, with the litter-bearing callitrichids growing at a rate comparable with the fast-growing strepsirhines (Ross, 1991).

A comparison with another relatively large-sized, single-infant bearing taxa shows that, prior to weaning, primate infants grow approximately ten times more slowly than do ungulate infants (Fig i.iiia). For example, olive baboon infants (*Papio anubis*) and Dorcas gazelle infants (*Gazella dorcas*) are born to mothers that weigh approximately the same (13kg – 14 kg), yet gazelle infants grow over thirteen times faster during lactation than do baboon infants (4.8g/day vs. 64.0g/day; data from Lee *et al.*, 1991). Lactation in baboons is thirteen times longer than in gazelles (Fig i.iiib; 600 days vs. 45 days). Gorilla (*Gorilla gorilla*) pre-weaning growth rates are twelve times slower than reindeer (*Rangifer tarandus*) growth rates (19.7g/day vs. 240.0g/day), even though adult female gorillas and female reindeer weigh approximately the same (just over ninety kilograms). Similarly, gorilla infants take almost eight times as long to reach weaning age as reindeer infants (900 days vs. 120 days).

Concurrent with these low growth rates, milk quality in primates is relatively poor (Pond, 1984). Although milk composition varies from feed to feed, lactation to lactation, and individual to individual (Oftedal, 1984), in general composition is very highly conserved across different mammalian taxa and dietary regimes (Pond, 1984). Milk solids consist mostly of carbohydrates in the form of lactose; proteins (e.g. casein and whey); amino acids; and lipids, including essential fatty acids. Vitamins, minerals and various

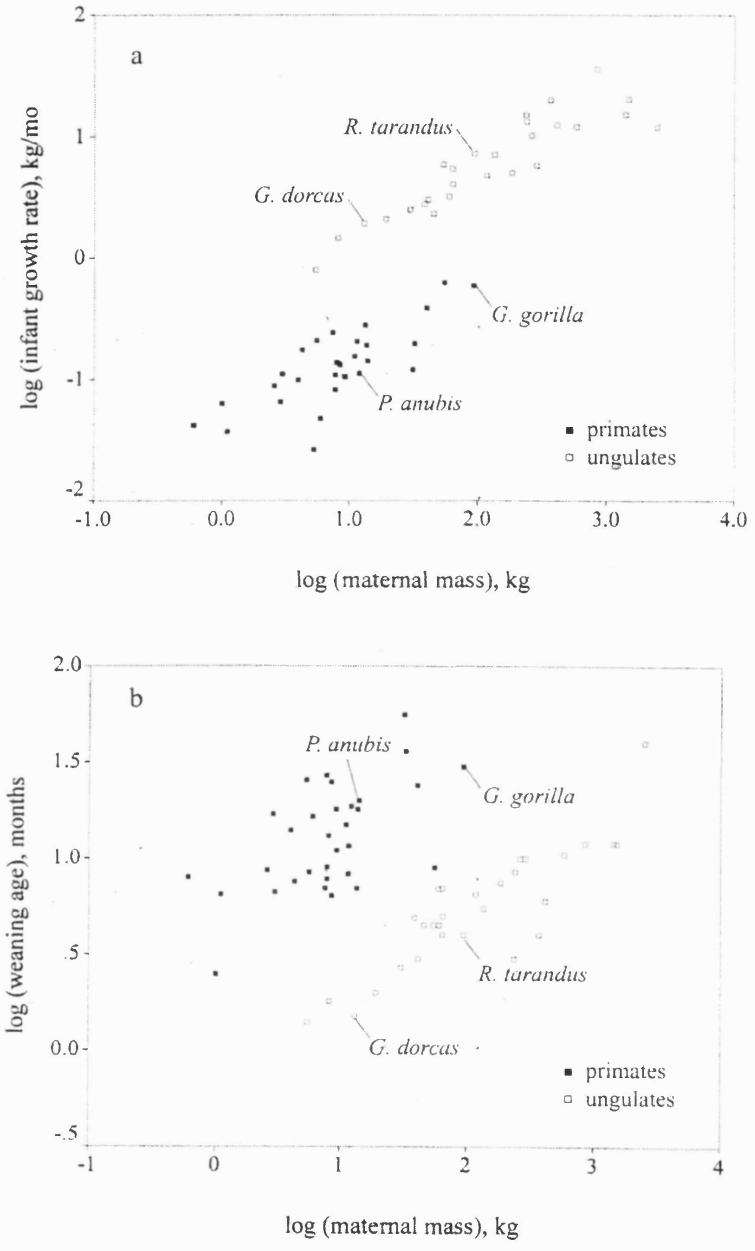


Figure i.iii. Primate and ungulate lactation lengths and infant growth rate. (a) Lactation length by by maternal mass; (b) infant growth rate by maternal mass. Data from Lee *et al.*(1991). See text for discussion of highlighted taxa.
In general, primates grow more slowly than ungulates of similar body size (a), and are weaned at a later age (b).

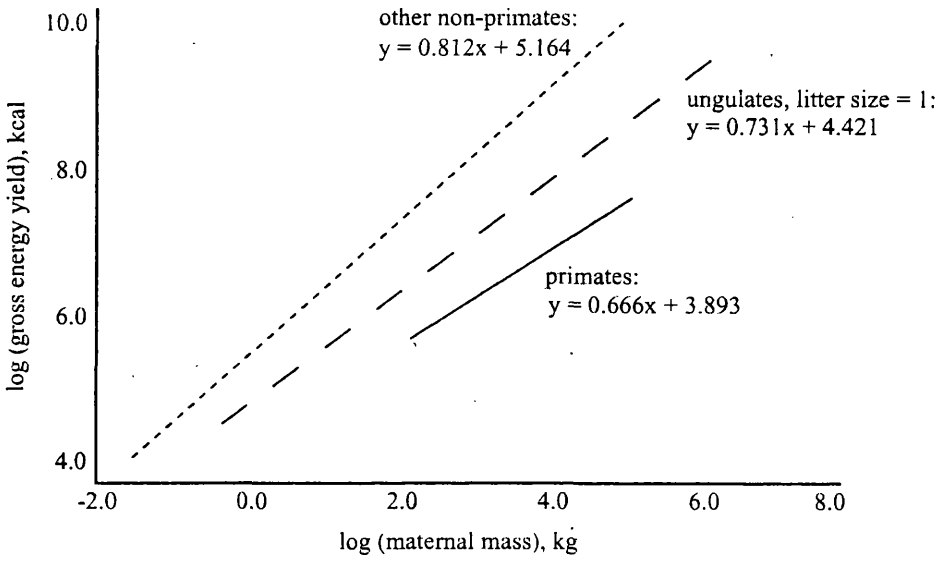


Figure i.iv. Energy content of milk in mammals. Lines fitted are least squares regression. Based on Oftedal (1984). Primates tend to have lower-yielding milk than other non-primates of similar body mass.

products of the endocrine system e.g. hormones and growth factors are the other important components of milk (Hartmann *et al.*, 1984). Lipids are the most variable constituent of milk across mammals, and primate milk has a very low fat content when compared with other terrestrial mammals (Glass & Jenness, 1971; Oftedal, 1984). It is especially poor in comparison with marine mammals (e.g. pinnipeds), whose milk is notably lipid-rich (Oftedal, 1984). Primate milk is also dilute, and solids account for approximately 12% - 14% of the milk volume by weight; this compares with a minimum of 30% volume by weight in the terrestrial mammals (e.g. carnivores), and 30% - 60% in cetaceans (Oftedal, 1984). This dilute, lipid-poor milk has a low energy-content: at peak lactation and per kilogram maternal mass, primate milk provides approximately one quarter the energy that milk from single-infant-bearing non-primates of similar body masses provides, e.g. ungulates (Fig i.iv).

i.viii Brains and slow growth

Despite producing poor-quality milk, the energetic requirements of female non-human primates increase by at least 50% during lactation (Key & Ross, in preparation ; Clutton-Brock, 1989), even though the energetic costs of primate milk synthesis are low compared to those in other mammalian taxa (Prentice & Prentice, 1990). Although mothers are able to accommodate some of this extra energy requirement through behavioural and physiological mechanisms (e.g. seasonal breeding, Wasser & Barash, 1982; reducing metabolic rate, Prentice & Whitehead, 1987), extending lactation prolongs the period in which a female's energetic needs are substantially elevated compared to her non-pregnant or pregnant condition (Key & Ross, in preparation). Why should primates slow infant growth and extend lactation? Martin (1996) suggests that maternal metabolic capacity is an important regulator of infant brain growth both during gestation and lactation. The low growth rates, low milk energy content and late weaning ages seen in primates might, therefore, reflect a maternal strategy aimed at spreading the energy costs of lactation over a

longer period of time (Lee, 1996). Thus, although absolute investment in growth is high, relative costs per unit time are low. Mothers may then be better able to meet the daily energy requirements of lactation, especially when resources are unpredictable, or fluctuate over time (as they do in many primate habitats, Smuts *et al.*, 1987). Lee (1999) finds that, although larger-brained primates tend to have longer reproductive events (conception to weaning), when maternal mass is accounted for, only a weak association remains between relative brain size and the length of reproduction. This is also true of the correlation between length of reproduction and amount of postnatal brain growth: with maternal mass held constant, the association is weak. Both these results highlight the importance of maternal mass – and by inference, maternal metabolism – in influencing both infant growth and brain size.

Several other non-metabolic mechanisms linking large brains with slow development have been suggested. Large brains might simply take longer to mature compared to small ones. Postnatal brain mass increase occurs mostly through the myelination of axons (see Chapter 1). It is during myelination that neural pathways are 'fixed' and that behaviours are canalised; large brains contain more neurons than smaller ones, and require an absolutely longer period of time to mature (Nolte, 1999). Furthermore, brain evolution in primates is characterised not only by increased size, but increased neocortical mass (Finlay & Darlington, 1995). The neocortex is the last part of the brain to develop to full maturity (Jacobson, 1978), and selection acting to lengthen the period in which primate brains grow has favoured an disproportionate growth of the neocortex in relation to 'older' structures, e.g. medulla oblongata (Finlay & Darlington, 1995). The neocortex plays an important in role in many of what are often thought of as the 'higher' mental functions: forward planning, decision making, memory formation, recognition of objects, complex spatial awareness, language capabilities etc. (Nolte, 1999), and it is precisely these functions that develop in the later stages of brain mass increase and myelination (Mize & Erzurumlu, 1996).

Late ages at maturation might therefore function to ensure these aspects of behaviour are properly developed, especially in the complex physical and social environments occupied by primates (Deacon, 1990). For example, the increase in primate brain size have been linked to the evolution of extractive foraging techniques that characterise some taxa's dietary adaptations, e.g. frugivory (Harvey & Clutton-Brock, 1985; Foley & Lee, 1991; Barton, 1999). The need to develop or even learn the behaviours involved in these complex techniques (e.g. mental mapping of clustered food patches, integration and memorisation of information about ripeness and availability etc.) may lie behind the long period of juvenility. This link between brain size and diet has been shown to hold true in another encephalised taxon, bats (chiroptera; Eisenberg & Wilson, 1978), although some primates which use extractive foraging techniques (e.g. strepsirhines) do not show low growth rates when body size is controlled for (Ross, 1992b).

More compelling is the evidence that the relative size of the neocortex correlates positively with social group size in a range of primate and non-primate taxa (Dunbar, 1992; Dunbar, 1998). It is possible that the suite of social skills and behaviours important in primate group living require a long period of development to acquire. Relative non-visual neocortex size has been shown to correlate directly with the relative length (to lifespan) of the juvenile period in primates (Joffe, 1997), suggesting that the rate at which behaviours develop place a constraint on the speed with which growth occurs. Joffe (1997) also shows that the absolute length of the juvenile period correlates with group size. Ross & Jones (1999) point out that this analysis failed to account for body size, which is correlated with both the size of the neocortex and the length of the juvenile period; body size may, therefore, be responsible for the association of the latter two variables. However, it is likely that "the absence of higher post-natal growth rates in primates probably results from a selective disadvantage to very rapid growth because other maturational rates are not growth-linked but time-linked: they are age- or experience limited, and rapid growth would therefore result in higher mortality" (Altman & Alberts, 1987:19).

These behavioural models assume that the extended juvenile period of experience-related maturation is adaptive as it helps to increase reproductive fitness via increased breeding success later in life (Ross & Jones, 1999). Alternative explanations for the low growth rates and late ages at weaning observed in primates model juvenility as an adaptation to avoid mortality, and involve encephalisation only indirectly. Infant mortality has been shown to be correlated with temporal aspects of growth (gestation length, weaning age, growth rates) independently of body size (Promislow & Harvey, 1990). One proposal is that infants grow slowly in order to avoid the high energy requirements associated with large bodies and spend more time in predator avoidance (Janson & van Schaik, 1993). This contrasts with strategies in which growth is rapid in order to minimise the time spent in the risky juvenile period. This model assumes that the link between slow growth and encephalisation is indirect, and mediated by a third variable, diet (Ross & Jones, 1999). Growth rates are generally suppressed in food-limited populations (Altmann & Alberts, 1987), suggesting that lowering the infant's metabolic requirements does reduce starvation risk. It is likely that both behaviour and size are important in reducing mortality: for example, foraging efficiency in baboons approximates adult efficiency long before adult size is achieved, and may allow infants to remain small for longer (Janson & van Schaik, 1993). In a phylogenetically-controlled analysis that tested between the three main brain constraint models – the energetic model, behavioural/social ontogeny, and the juvenile risk aversion model – Ross & Jones (1999) find some support for the first: brain size is positively correlated with age at maturity. Group size shows a positive relationship with age at maturity when body size, brain size and diet are controlled for, contrary to the prediction of the behavioural/social ontogeny model. On the other hand, the correlation between amount of folivory in the diet and the length of the juvenile period is negative and only just non-significant in their analyses, possibly indicating support for the extractive foraging hypothesis. More evidence that folivory is associated with high growth rates and a short age to maturity is provided by Leigh's (1994) large interspecific comparison.

i.ix Summary

In this chapter we have seen that life history theory models the interactions of a suite of size and time variables that characterise the 'pace' of an animal's lifecycle. Primates are noted to have slow life histories, in particular, slow infant growth and long periods of lactation. They also have relatively large brains in relation to body size. Mothers must meet all the energetic needs of their infants during lactation, and the large size of the primate brain is predicted to influence maternal investment in infant growth. This thesis investigates the relationship between brain ontogeny and lactation/weaning strategies in primates. It examines how primate postnatal growth differs from that of other mammalian taxa, and whether primate strategies of weaning and lactation are linked to brain and body growth. It also investigates encephalisation, growth and lactation in the context of life history variation.

- Chapter 1 investigates interspecific variation in primate brain and body mass ontogeny. The chapter tests a brain allometry model against data from a variety of haplorhine taxa and examines whether primates differ from other mammals in the pattern of postnatal brain and body ontogeny. It also examines weaning brain and body masses in the context of the brain allometry model.
- Chapter 2 tests the observation that body mass at weaning coincides with the body mass at which the majority of brain growth is completed. It does this at the intraspecific level with a sample of longitudinal data from two species of capuchin monkey (*Cebus*).
- In Chapter 3, an 'artefact hypothesis' – that brain growth and weaning are linked because body growth and weaning are linked – is tested using growth data from a population of nutritionally-deprived capuchins.
- Chapter 4 investigates the behavioural and physiological aspects of weaning that are specifically linked to brain growth independently of body growth. Data from the

literature are integrated with brain and body growth trajectories from Chapter 2, and correlations between variables examined.

- Chapter 5 analyses the energetic costs of brain and body maintenance, and models interactions between infant size, maternal size and energetics. It asks: is investment in infant growth related to energetics?
- Chapter 6 examines capuchin growth in the more general context of life history. Principal components analysis (PCA) is used to outline the main sources of variation in the dataset, and the results variable groupings investigated.
- Finally, Chapter 7 addresses the implications these results have for capuchin lactation strategies, and for the evolution of another highly encephalised species, *Homo sapiens*.

CHAPTER 1

BRAIN AND BODY MASS ONTOGENY IN HAPLORHINE PRIMATES

The Introduction presented the idea that encephalisation is an important determinant of primate growth and life histories. This chapter investigates interspecific variation in haplorhine primate postnatal brain and body growth and tests the hypothesis that primate patterns of growth are similar to those found in other, non-primate taxa. It also examines variation in weaning mass and asks: is brain growth associated with lactation strategies in primates? The first section outlines the changes that occur in brain and body size during ontogeny, and discusses the functional implications of using mass as a proxy for size (Section 1.1). An allometric model of brain and body growth is introduced (Section 1.2), and its validity tested with brain and body size data gathered from various sources (Section 1.3). Finally, the results of an analysis that investigates how variation in brain and body growth relates to variation in weaning mass are presented (Section 1.4).

1.1 Patterns and processes of growth

This section introduces the concepts of 'growth' and 'development' at different structural levels, from cell to organism. It discusses the functional implications of mass increase, and describes the general course of mammalian growth from conception to adulthood.

1.1.1 Growth at the cellular level

Growth is a complex process that involves changes in both size and organisation (i.e. complexity or function) of tissues at all structural levels of the body (Sinclair & Dangerfield, 1998). 'Development' and 'maturation' are more ambiguous terms that can be applied to different levels of structural and behavioural organisation (Tortora & Grabowski, 1996). In

this chapter, the terms 'development' and 'maturation' are used to refer to the attainment of mature or adult function.

Change in size at the cellular level occurs by the complementary and interdependent processes of cell division (hyperplasia) and cell enlargement (hypertrophy). Total cell mass is therefore a function of both cell size and number (Conlon & Raff, 1999). In the early stages of embryonic growth, cells are multipotent and capable of giving rise to many types of mature cell, but as growth continues most differentiate and become committed to producing only certain cell lines (Tortora & Grabowski, 1996). Differentiation is associated with functional specialisation, and is usually accompanied by changes in organisation and an increase in the complexity of tissues and organs (Sinclair & Dangerfield, 1998). In general, four successive phases of growth characterise the lifespan:

1] A change in cell size and number that is not associated with differentiation.

2] Increases in mass and organisational complexity that continue in step until maturation of the growth system is achieved.

3] Maintenance of the mature system during which damage is repaired and functional activity sustained.

4] Senescence, in which growth is not sufficient to replace lost cells, and function becomes impaired (Sinclair & Dangerfield, 1998).

In mammals, these phases are associated with specific periods in a life history. Embryological growth is characterised by hyperplasia, hypertrophy, and a rapid rate of cell differentiation (phase 1). Fetal and early postnatal growth are dominated by hypertrophic mass increase, and by increasing functional maturity of most of the body's systems (phase 2). The achievement of adulthood (phase 3) marks the achievement of full maturity, and is usually accompanied by the attainment of a relatively stable stature (i.e. skeletal maturity) and body mass (Shea, 1992). Senescence (phase 4) occurs later in adulthood, and may be accompanied by a loss of tissue mass, as well as by a loss of function (Bogin, 1999). The timing of the different stages of development is subject to selection, and varies between taxa (Shea, 1992); this is life history variation (Charnov & Berrigan, 1993). For example, primate growth is

characterised by a long delay between the end of infancy (primary maternal dependence) and the onset of puberty and attainment of adult size (Ross, 1992; Pagel & Harvey, 1993).

In general, prenatal brain and non-brain tissue mass increases keep pace with each other through the effect of pleiotropic genes that regulate hyperplastic growth (Little, 1989). After parturition, hyperplastic growth diminishes and the tight association of brain and non-brain body mass increase is lost. In primates, the brain shows the most rapid rate of growth, and is largely complete early in postnatal life (Smart, 1991). Other organs increase in maturation at a rate comparable with overall body mass or stature (Sinclair & Dangerfield, 1998). For example, the male macaque brain has achieved adult size by the age of 30 months old, whereas the liver and heart only attain adult size a year later, at the age of 40 months old (Fig 1.1). The testes are the last of the organs to reach adult size, two years after the brain.

1.1.2 Nervous system development and brain mass increase

The central nervous system develops from a simple neural plate that emerges from a layer of the ventral ectoderm early in embryological development. The plate thickens and folds inwards, forming a neural 'tube' in a process called neurulation (Nolte, 1999). The cavity of this neural tube will go on to form the ventricular system of the brain. Neural cell precursors along each of the folds (neural crest cells) give rise to different CNS cells, including neurons (Smart, 1991). The differentiation of cells in the neural crest is dependent on cell-cell signalling, and is regulated by a myriad of neurogenic genes and their products (Mize & Erzurumlu, 1996). Factors such as nerve growth factor (NGF) and fibroblast growth factor (FGF) are essential for the growth and survival of neural cell precursors (Nolte, 1999). Hormones that are more generally involved in tissue growth also play an important role in brain growth. For example, T^3 and T^4 (triiodothyronine and thyroxine), corticosterone and somatotrophin all affect various aspects of neural cell development as well as growth of other body systems (Jacobson, 1978). The high lipid content of the brain (Crawford, 1993; Clandinin, 1999) is due in part to the importance of fatty acids in maintaining the structural

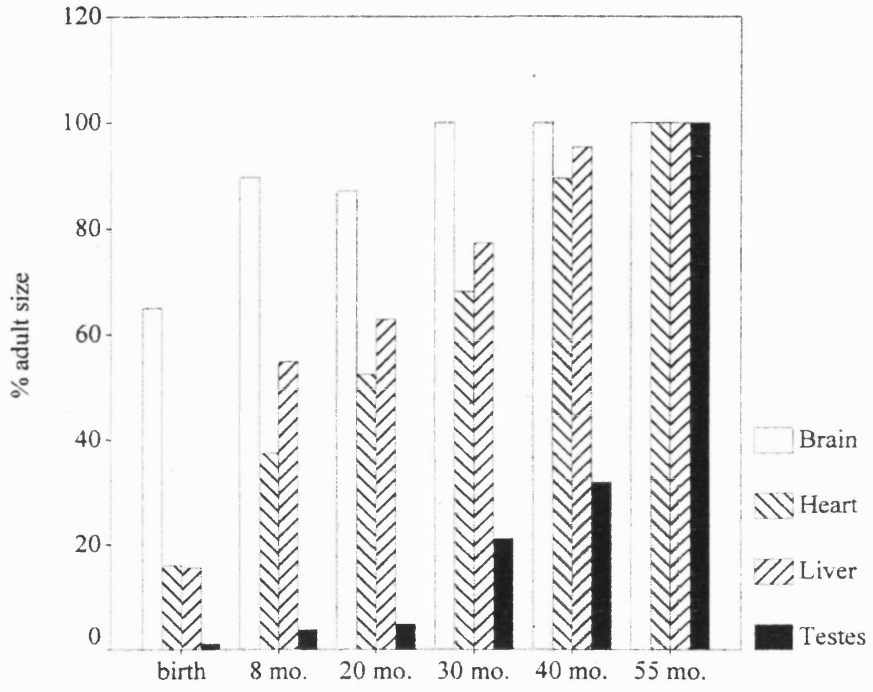


Figure 1.1. Primate brains follow a pattern of advanced growth compared to other organs. Organ masses are expressed as a percentage of adult size (data from Kerr et al., 1974). Data from Macaca mulatta.

integrity of neuronal membranes (Clandinin & Jumpsen, 1997), and in the formation of neural connections during synaptogenesis and myelination (see Chapter 5).

Neurogenesis is largely complete by the third trimester of gestation (Williams & Herrup, 1988). After neurogenesis, the brain increases in mass through the growth of axons and dentrites, the rapid multiplication of oligodenroglial cells, and myelination (Hall & Oxberry, 1977). Brain mass increase is characterised by a sigmoidal growth pattern when plotted against age (Brizzee & Dunlap, 1986), although some primate and non-primate species have been shown to undergo a brain growth spurt around the time of birth (Dobbing & Sands, 1979). The timing of this spurt is variable; in macaques it occurs prenatally, in humans, perinatally (Brizzee & Dunlap, 1986).

By far the most important factor in postnatal brain mass increase is myelination, in which nerve fibres are coated in a myelin sheath that improves the speed and efficiency of signal conduction (Nolte, 1999). Before myelination, neural connections are 'plastic' and open to reorganisation; the completion of myelination signals the achievement of maturity for a particular nerve fibre (Mize & Erzurumlu, 1996). Axons involved in 'primitive' tasks such as gripping and suckling are myelinated early in development (Nolte, 1999). Because myelination further consolidates connections and 'fixes' the axons in place, myelination prevents further neuronal reorganisation (Mize & Erzurumlu, 1996). Postnatal brain growth is also characterised by the large-scale 'pruning' of neurons and their connections, a process that further canalises neuronal pathways in the brain (Smart, 1991).

Recent research suggests that, whilst neuronal population size is largely set before birth, mice, guinea pigs, cats, and some birds and primates (including humans) are able to generate neurons well into adulthood (Hastings *et al.*, 2000). The longevity of these new cells is uncertain, and they may be temporary, functioning for no more than a few weeks (Gould *et al.*, 1999). Postnatal neurogenesis appears to be associated with certain parts of the brain; in birds, for example, the new neurons are associated with brain structures that function in memory formation, and may serve to aid song recognition (Kirn *et al.*, 1999). In rats, the rate of formation of these new neurons is positively associated with environmental enrichment, and

negatively associated with stress (Cameron & McKay, 1999; Gould & Tanapat, 1999). Neocortical neurogenesis has been reported in adult macaque monkeys (Gould *et al.*, 1999), but in humans the regeneration of neurons appears to be restricted to the hippocampus, again as a possible aid to memory function (Fuchs & Gould, 2000).

Although the majority of brain mass is accumulated in early postnatal life in most primate species, mass increase does continue into ontogeny (Deacon, 1990). An extreme example is *Homo*: five year old human infants have achieved only around 90% or 95% of adult brain mass (Sinclair & Dangerfield, 1998). Some parts of the human brain (e.g. parietal and frontal lobes) are reported to show an increase in mass as late as adolescence, but whether this late postnatal growth has cognitive implications, or simply reflects changes in cerebral blood flow is not yet certain (Sowell *et al.*, 1999). Later still in the lifespan, the human brain undergoes a moderate decrease in mass (Sinclair & Dangerfield, 1998). This degeneration is also observed in chimpanzees, but not rhesus macaques (Herndon *et al.*, 1998; Herndon *et al.*, 1999).

The brain does not grow uniformly across ontogeny, and functional structures in the brain mature at different rates (DeVito *et al.*, 1989). The language centres of the human brain, for example, are not fully myelinated until late childhood, by which time the majority of the sensory and motor processing areas are mature (Nolte, 1999). Whilst there does appear to be some constraint the amount of variation in brain structure size (Finlay & Darlington, 1995), variation in brain mass has been shown to be associated with specific specialisation of certain parts of the brain, e.g. the visuo-spatial pathways in primates (Barton, 1999). Barton (1999) notes:

> "Brain size cannot thus be meaningfully interpreted without reference to specific neural adaptations, which will differ from case to case... Primates combine visual acuity with hand-eye coordination and manual dexterity[,] functions that bring into play the extensive connections between neocortex and cerebellum. Also, the cerebellum is, like the neocortex, significantly larger in primates than in insectivores[...] This suggests that the relevant functional units or modules of the brain, on which selection has acted,

cannot be simply equated with single structures or regions, but constitute networks distributed across major brain regions."

(Barton, 1999:189).

Thus even when an area that is known to be involved in a particular function increases in size, selection is not necessarily acting on that area alone. The existence of these 'global' networks within the brain validates the study of gross brain size as a means of examining the maturity of brain function. A large proportion of variation in the size of brain structures is accounted for by variation in the size of the brain itself (Finlay & Darlington, 1995; Barton, 1999), and the achievement of adult brain size coincides with the attainment of adult patterns of behaviour and cognition in most taxa. Furthermore, brain tissue is relatively homogeneous, and its mass is an excellent predictor of the brain's metabolic costs (Kety & Schmidt, 1945; Schmidt *et al.*, 1945; Passmore & Durnin, 1955). An analysis of brain size variation and function below the gross anatomical level is beyond the scope of this thesis, and in the following discussions brain size, in particular brain mass, is used as a whole-brain maturity indicator.

1.1.3 Growth at the organism level: body mass increase

What aspect of size does body mass represent? Although in general body growth is regular and predictable (Tanner, 1986), body mass itself is relatively labile (Economos, 1980). Mass increase is modulated by a suite of genetic and epigenetic factors (Bogin, 1999). The pituitary gland develops in the second trimester of pregnancy, and growth hormone, or somatotrophin, is detectable in the fetus soon after the pituitary gland is formed (Nolte, 1999) and it is likely that products of all the endocrine glands influence growth at some stage of life (Sinclair & Dangerfield, 1998). For example, insulin-like growth factors, T^3, T^4 and other somatomedins play important roles in regulating protein synthesis (Rechler *et al.*, 1987). Somatotrophins inhibit lipid and carbohydrate synthesis and also promote increased cell division (i.e. cell number) and DNA formation after infancy (Sinclair & Dangerfield, 1998). Androgens

stimulate growth early in postnatal development as well as at puberty and are associated with the development of body mass dimorphism (Leigh, 1992; Shea, 1992).

Growth is also affected by epigenetic factors (Brizzee & Dunlap, 1986; Little, 1989; Sinclair & Dangerfield, 1998). The effects of maternal and infant nutrition on growth are well-documented across many taxa (see Lee, 1996 for review). The adequate diet consumed by provisioned and captive populations tends to promote faster rates of body growth over ontogeny. For example, chimpanzee infants have been reported to wean after about 56 months in a natural environment, but at a little under half that in captivity; weaning masses also differed in the two populations by about 3kg, or about 15% (Lee *et al.*, 1991). This difference in rate of mass increase between wild and captive infants has been reported in many other species (Leigh, 1994). For instance, body mass growth rates in the infants of captive protein-deprived baboons are around half those of infants of adequately nourished baboon females (Buss & Reed, 1970; Altmann & Alberts, 1987). Growth during the period of maternal dependence is also modulated to a large degree by maternal parameters of health, parity, status and psychology (Lee, 1987; Lee, 1996). Even in adults, body mass itself can vary across the course of months, weeks and days. Alberts & Altmann (1987) report a 6% difference in body mass in free-ranging baboons between nightfall and morning: mass gained through feeding was lost overnight, probably through thermogenesis. Long and short term fluctuations in body mass are therefore part of normal, as well as ill, health. The Introduction showed that postnatal variation in growth is an important determinant of adult brain and body proportions (Martin, 1996). In most mammal species, the majority of brain growth is complete by birth, and neonatal body mass is a good predictor of encephalisation (Martin, 1990). Because body growth is completed much later in life, differential body growth can result in differing levels of encephalisation between individuals that are similarly-encephalised as neonates (Martin, 1996).

1.2 Modelling brain allometries over ontogeny

How do brain and bodies grow in relation to each other? Few large, good-quality comparative brain growth datasets exists for species other than humans and macaques (Falk *et al.*, 1999), and only recently have routine necropsy procedures in primate centres and zoos been established (see below). Comparing necropsy data is a useful way of examining how brain and body size vary in relation to each other across a range of different age groups i.e. a comparison of brain allometries (Shea, 1985; Shea, 1992). Empirical evidence suggests that the ontogenetic brain allometry curve can best be modelled as a convex quadratic function (Brizzee & Dunlap, 1986); log-transforming the data allows the comparison of relative growth rates during ontogeny (Holt *et al.*, 1975). All animals are predicted to follow this ontogenetic brain allometry curve, which describes growth from conception to adulthood (Fig 1.2).

The model presented here describes how brain size changes with increasing body size. It can be seen that body size (x-axis) continues to increase after brain size increase (y-axis) has ceased or slowed. Two periods of growth can therefore be distinguished. In the pre-inflexion brain/body growth phase, the slope of the curve is equal to 1.0, and the brain and non-brain tissues grow in step with each other. The value of this slope has been empirically derived, although in some litter-producing species, the exponent appears to be slightly lower than isometry (Lande, 1979; Deacon, 1990; Martin, 1990). For simplicity, and because the analysis presented here only includes species with a modal litter size of one, the brain allometry is modelled with a slope equal to 1.0. In this period, the brain increases in size through neurogenesis and some gliogenesis and includes all prenatal growth from conception to birth (Lande, 1979); in some taxa such as humans, some postnatal growth is also included (Deacon, 1990). Where birth occurs along the curve depends on the altriciality of the species i.e. how much brain growth occurs pre- or postnatally. In Figure 1.2, B marks the position of birth along the body growth axis for a typical mammal.

In the post-inflexion period, the slope of the curve is predicted to be much lower than in the pre-inflexion period. Deacon (1990) estimates an exponent of 0.1 to 0.2, based on

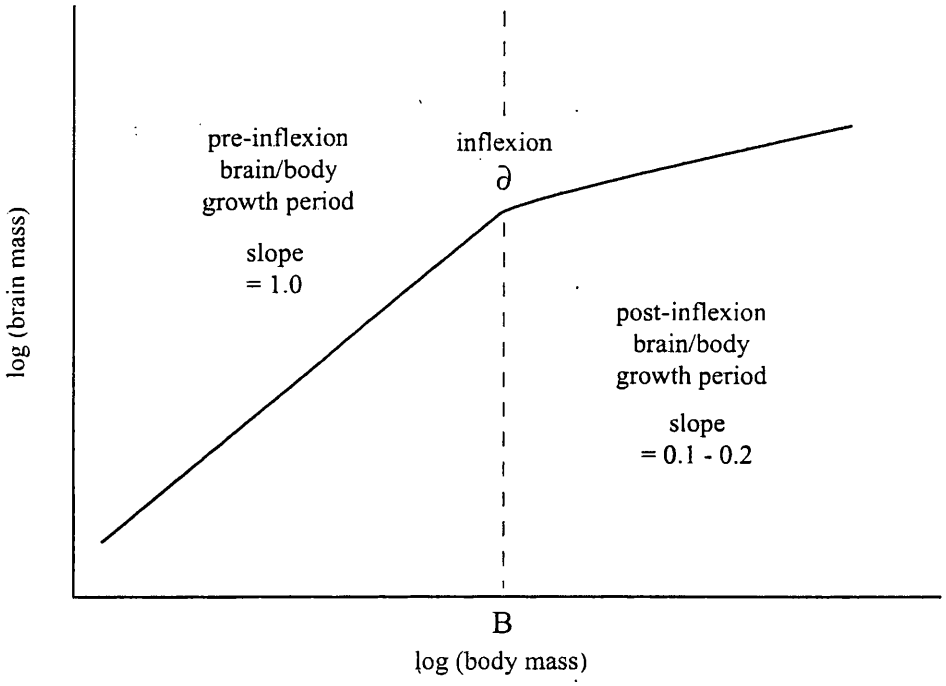


Figure 1.2. Modelling brain allometry over ontogeny. See text for explanation. B = birth in a typical mammal. Adapted from Deacon (1990). Prenatal brain growth is isometric with prenatal body growth. After birth, the slope of the curve decreases to between 0.1 and 0.2.

previous interspecific analyses (Count, 1947; Gould, 1975). This phase of brain growth is characterised by cell enlargement and gliogenesis (Smart, 1991). In Deacon's (1990) model, this phase includes within-species variation of adult brain and body size.

The point at which the slope changes from that of the pre-inflexion period to that of the post-inflexion period is marked δ in Figure 1.2: it represents the end of the phase of rapid brain growth. It should be noted that time is not represented on either axis: the model does not predict how long an individual will spend at any body or brain size. The slope, or 'rate', at which brain mass changes represents the amount by which brain mass increases in relation to body mass increase. Two of the model's predictions can be tested with postnatal growth data:

1] The curve describing primate postnatal brain and body growth is linear i.e. represents the post-inflexion phase of the mammalian growth model.

2] The slope of this postnatal, post-inflexion curve falls between 0.1 and 0.2.

If 1] is shown to be incorrect, a further prediction can be made:

3] The postnatal pre-inflexion curve represents an extension of the prenatal brain growth period, and the slope of this pre-inflexion curve equals 1.0.

1.3 Interspecific analysis of brain and body ontogeny: testing the brain allometry model

1.3.1 Materials*

In order to test the validity of the brain growth model, a dataset of non-human primate postnatal brain and body mass was collected. Data were of three types:

1] Endocranial volume

Endocranial volume (EV) is measured from wild-shot New World monkey crania housed in two Brazilian museums:

*All data analysed in this, and other chapters, are available from the author in electronic form. See Appendix 1 for details.

1] Museu Paraense Emilio Goeldi, Belem, Brasil (www.museu-goeldi.br/).

2] Museu de Zoologia, Universidade de Sao Paulo, Sao Paulo, Brasil (www.mz.usp.br).

Only intact crania for which field-recorded body masses at death were available were used. Loose debris was carefully removed from the endocranial cavity, and the orbits filled with cotton wool. Using a funnel, 1.00 millimetre diameter glass beads were introduced into the inverted skull via the foramen magnum. The beads were inserted in pre-measured increments of $50mm^3$ measured in a 50ml-capacity measuring tube. The skull was gently shaken and tapped after the addition of each increment to ensure even settling of the beads. The skull was filled to the level of the foramen magnum with the skull held in an inverted anatomical position. Volume was measured to the nearest $0.25mm^3$. Error was tested by remeasuring a random sample of 8 crania; the mean percent difference between the two measurements was $1.5 \pm 0.8\%$ (Table 1.1).

Some authors treat EV and brain mass as equivalent (e.g. Clutton-Brock & Harvey, 1980). Others use conversion factors to accommodate differences in the densities of water and neural tissue (Count, 1947; Stephan *et al.*, 1981; Smith *et al.*, 1995; Kappelman, 1996) and the percentage of the endocranial volume taken up by the brain (Tobias, 1971). For the sake of simplicity, and to avoid introducing further error into the analysis, EV was used here as a direct measure of brain mass (i.e. 1 mm^3 = 1 g).

2] Necropsy data

Necropsy data are useful for assessing organ mass immediately or soon after death, but like other tissues, the brain often becomes oedemic immediately before death (Macfarlane *et al.*, 2000), and necropsy data may not reflect the anatomy of healthy living subjects. Because of the problem of oedema, necropsy brain weights should be thought of as estimating the upper limit of living brain size, and those included here as broadly comparable across different studies. In all cases, data from stillborn infants were ~~also~~ excluded.

Table 1.1 Estimating endocranial volume measurement error

Cranium	Measurements		Difference	% Difference
	A	B	C = A – B	| C | / (A+B/2)
1	72.25	71.50	0.75	0.010
2	71.00	71.50	0.50	0.007
3	62.50	63.25	0.75	0.012
4	58.75	58.00	0.75	0.013
5	50.00	49.50	0.50	0.010
6	54.00	55.50	1.50	0.027
7	75.00	77.00	2.00	0.026
8	59.25	60.00	0.75	0.013
Mean ± sd				0.015 ± 0.008
se				0.003
95% confidence interval for mean				0.009 – 0.021

sd = standard deviation. se = standard error.

Necropsy brain masses were gathered primarily from the Yerkes Regional Primate Research Center (YRPRC). The YRPRC (www.emory.edu/WHSC/YERKES/yerkes.html) houses many different primate species, including chimpanzees (*Pan*), orangutans (*Pongo*), sooty mangabeys (*Cercocebus*), macaques (*Macaca*), and capuchins (*Cebus*). Body masses are routinely recorded at death, and autopsy is a standard procedure at the YRPRC (Herdon *et al.*, 1999). Ten years of necropsy records (1989 to 1999) were collected from the YRPRC computer database, and brain masses gathered for those individuals that were considered healthy at the time of sacrifice or that died from acute trauma or non-wasting disease. Animals noted to be emaciated or masses that were recorded more than two days after death were not included.

Necropsy data were also taken from five previously published reports (Crile & Quiring, 1940; Kennard & Willner, 1941a; Kennard & Willner, 1941b; Kennard & Willner, 1941c; Count, 1947). Not all these sources provide detailed necropsy protocols, and it is probable that the procedures for opening the skull, removing, fixing and weighing the brain vary considerably between different workers. On the other hand, Kennard & Wilner (1941a) find no difference in mass between similarly-aged formalin-fixed and unfixed *Macaca mulatta* brains (n = 118). Neither do they find a significant difference in brain masses between individuals that had and had not been anaesthetised prior to death. However, even if protocols do not differ, decisions such as how much brain stem to retain with the brain proper, or whether to remove meninges etc., can and often do vary from necropsy to necropsy. Neither do all published reports note whether the brain is oedemic or damaged, or whether the body is over- or underweight at death.

3] Mean reported neonatal and adult body masses from the literature

For some taxa neonatal brain and body data were scarce, and to ensure that the full range of postnatal body masses were encompassed by the analyses, mean neonatal and adult body and brain mass data were also gathered from the literature. Species-averages are sex-specific where possible, and are the mean of minimum and maximum reported mean values i.e. do not

indicate the full range of reported masses, but instead are an estimate of a mean value based on the highest and lowest mean reported in the literature. Details of these data are given in Appendix 1.

1.3.2 Methods

There is evidence for body and organ dimorphism in some primate species at birth and earlier, especially in the larger-bodied primate species (Smith & Leigh, 1998); where sample size allowed, male and female data were analysed separately. It should be remembered that most of the data presented in this section are cross-sectional and therefore do not represent one animal's lifetime ontogenetic curve, but rather a summary of different animals' body and brain proportions at different body sizes.

a] Non-brain body mass

The calculation of non-brain body mass (NBBM) is useful if brain and body masses are to be analysed independently. This is especially true for the smaller-bodied species and age groups, where brain tissue takes up a larger proportion of the total body mass. Non-brain body mass was calculated in one of two ways, as appropriate:

$$\text{NBBM} = \text{Whole body mass} - \text{brain mass} \qquad (1.1)$$

or

$$\text{NBBM} = \text{Whole body mass} - \text{EV} \qquad (1.2)$$

NBBM derived by these different methods are not distinguished in the analyses.

b] Non-linearity testing

The data were log-transformed, and the allometric relationship between brain mass and NBBM was tested for non-linearity by comparing linear and polynomial regressions of each sample. A curve was considered non-linear when the coefficient of determination of the quadratic regression was significantly larger than that of the linear regression (Sokal & Rohlf, 1995). Significance was assessed with the calculation of an F value such that:

$$F = [(R_2^2 - R_1^2) / k_2 - k_1] / [(1 - R_2^2) / (n - k_2 - 1)] \qquad (1.3)$$

R_1^2 and k_1 are the coefficient of determination and the number of power terms of the linear regression respectively; R_2^2 and k_2 are the coefficient of determination and the number of power terms of the quadratic regression respectively. The F value has $n - k_2 - 1$ degrees of freedom. Because $k_1 = 1$ and $k_2 = 2$ in each sample, the equation can be re-written:

$$F = (R_2^2 - R_1^2) / [(1 - R_2^2) / (n - 3)] \qquad (1.4)$$

This method compares the proportion of variation explained by the quadratic regression with the proportion of variation explained by the linear regression, in relation to the amount of residual (unexplained) variation, the number of power terms and the size of the sample (Sokal & Rohlf, 1995).

c] Estimation of inflexion

Where the allometric curve was shown to be non-linear, locally-weighted regression (loess; Cleveland & Devlin, 1988; Cleveland *et al.*, 1988) was used to estimate the position of the inflexion, δ. Whereas linear and other parametric line-fitting techniques presuppose the shape of a function, loess is a non-parametric smoothing technique that runs an iterative regression analysis along the x values, and finds a predicted curve from a weighted average of nearby y values. The resulting curve reflects the local parametric (least-squares) relationship between x

and y values. The statistical analysis package SYSTAT 9.0 (www.spss.com), which was used to fit the loess curves, requires that two loess parameters are set before running the analysis. The first of these is the degree of polynomial used to regress the data. In all analyses that follow this value is set to 1, in order to obtain a curve fitted by local linear regression. The second parameter is the percentage of neighbouring datapoints to be included in the least squares analysis. A high percentage specification results in a curve that reflects the trend of the majority of data. For example, a setting of 100% produces a curve that includes information from all data and results in a straight line i.e. a normal linear regression. A low percentage (e.g. 10%) results in a curve that includes only a small proportion of the data at any one point along the curve i.e. a very localised regression line. In all analyses that follow, the percentage of points to be included is set to 50%. This setting results in loess curves that reflect the general trend of the data and are sensitive to any large-scale changes in slope, but do not reflect small-scale inflexions caused by random scatter of the data.

d] Calculating pre- and post-inflexion RMAs, and δ

The loess estimate of the inflexion was used to divide each sample into pre- and post-inflexion subsets. Data which fell on the estimated inflexion were included in both subsets. The reduced major axis (RMA; Clarke, 1980) was then calculated for both the pre- and post-inflexion parts of the allometric curve. RMAs were used in preference to least squares regression because both variables included in the analysis are subject to error; calculation of the RMA standardises both variables such that each has a mean of zero and a standard deviation of one (Sokal & Rohlf, 1995). The two RMAs were solved as simultaneous equations, as follows. The allometric relationship is a function of x and y values such that:

$$\log (y) = a + b^{*}\log (x) \qquad (1.5)$$

If the pre-inflexion RMA equation is denoted by subscript 1 ($_1$) and the post-inflexion RMA is denoted by subscript 2 ($_2$), then:

$$Y = a_1 + b_1X \quad (1.6)$$

$$Y = a_2 + b_2X \quad (1.7)$$

Y and X represent log-transformed y and x respectively. Rearrangement of the terms produces the equation:

$$(b_1 - b_2)X - (a_1 - a_2) = 0 \quad (1.8)$$

Further rearrangement produces:

$$X = (a_1 - a_2) / (b_1 - b_2) \quad (1.9)$$

Y can be obtained from this derivation of X using either of the two original RMA equations. Here, the solution to the simultaneous equations represents the point along each axis where the two lines cross, i.e. the point of inflexion, δ. This method ensured that the inflexion was derived statistically, rather than visually from the loess estimate alone. Even when the inflexion of the loess was inconspicuous, for example in the male *Cercocebus* sample (see below), the calculation of RMAs meant the position of δ could be calculated. Where the allometric curve was shown to be linear, an RMA was calculated for the whole sample. The standard errors of all the RMAs were calculated from the standard error of the least squares regression, as recommended by Sokal & Rohlf (1995).

e] RMA 'lengths'

The 'lengths' of the RMAs were calculated as:

$$\text{RMA length} = \sqrt{(a^2 + b^2)} \quad (1.10)$$

a = difference in brain mass between 'endpoints' of the RMA, and b = difference in NBBM between the endpoints of the RMA. This method uses Pythagoras' theorem ($a^2 + b^2 = c^2$) to calculate the hypoteneuse of a right-angled triangle of which the differences in brain mass and NBBM are the two known sides.

f] Residual encephalisation

Residual encephalisation at adulthood and inflexion was calculated as:

Residual encephalisation = observed brain mass – predicted brain mass (1.11)

Observed brain mass was calculated from the post-inflexion RMA equation; predicted brain mass was calculated from the predicted RMA equation (see below).

g] Correlation analysis

As a statistical test of the relationship between the shape of the growth curve and encephalisation, the following variables were entered into a correlation analysis (Pearson's coefficient):

1] The slopes of the pre- and post-inflexion RMAs.

2] The lengths of the RMA between birth mass and δ, and between δ and adult NBBM.

3] Residual encephalisation at δ and adult mass,

1.3.3 Results

a] Data collection

Data for 9 taxa were available:

1] Squirrel monkey (*Saimiri sciureus*)

2] Tufted capuchin (*Cebus apella*)

3] Spider monkey (*Ateles* spp.)

4] Howler monkey (*Alouatta* spp.)

5] Sooty mangabey (*Cercocebus torquatus*)

6] Rhesus macaque (*Macaca mulatta*)

7] Common chimpanzee (*Pan troglodytes*)

8] Orangutan (*Pongo pygmaeus*)

9] Gorilla (*Gorilla gorilla*).

Detailed and numerous data were available for *Saimiri* (total n = 53), *Cercocebus* (n = 101), *Pan* (n = 133) and *Macaca* (n = 1088). It should be noted that:

1] Crile and Quiring (1940), from whom data for *Ateles*, *Alouatta* and *Pan* are taken, provide only sample size and mean in their organ mass compendium, not individual datapoints.

2] In order to increase the number of datapoints in the howler monkey sample, both *Alouatta belzebul* (47%) and *A. palliata* (53%) are included (n = 109).

3] The majority (72%) of the datapoints included in the spider monkey sample (n = 184) are *Ateles geoffroyi* specimens; the remaining are *A. fusciceps* (25%) and *A. paniscus* (3%).

4] The dataset mixes wild and captive data, as well as data from hand- and mother-reared infants.

A good mix of male and female data were available for four of the taxa (*Saimiri, Cercocebus, Macaca, Pan*), and in these groups the sexes are analysed separately. Fewer known-sex data were available for the remaining taxa, and data in these samples are sex- and source-combined. Some of the taxa included show a high level of sexual dimorphism as adults and over ontogeny (Leigh, 1992), but here are analysed as a sex-combined sample (e.g. *Gorilla*). This undoubtedly obscures some growth trends, but is unavoidable with the data available. A summary of the data included in the following analyses is given in Table 1.2.

Table 1.2 Sources and description of data used in the interspecific analyses

Common name	Genus	n of datapoints (m:f:?)[1]	Source[2]	Type	Data	Ages included (y, or based on source description)	Allometric curve calculated…
Squirrel monkey	*Saimiri*	53 (25:28:0)	7	Captive	Necropsy	0.00 – 3.00	By sex
Capuchin monkey	*Cebus*	28 (18:10:0)	2	Wild	EV	To complete eruption of permanent dentition	Sex and source combined
		16 (7:9:0)	6	Captive	Necropsy	Immatures and mature individuals	
		8 (3:5:0)	8	Captive	Necropsy	0.00 – 0.36	
Spider monkey	*Ateles*	3 (0:0:3)	2	Wild	EV	To complete eruption of permanent dentition	Sex and source combined
		171 (25:35:111)[3]	3	Wild	Necropsy	Immatures and adults	
		10 (4:5:1)	6	Captive	Necropsy	Immatures and adults	
Howler monkey	*Alouatta*	52 (28:24:0)	2	Wild	EV	To complete eruption of permanent dentition	Sex and source combined
		57 (2:4:51)[3]	3	Wild	Necropsy	Immatures and mature individuals	
Sooty mangabey	*Cercocebus*	88 (46:42:0)	8	Captive	Necropsy	0.00 – 30.08	By sex, sources combined
		13 (6:7:0)	6	Captive	Necropsy	Mature individuals	
Rhesus macaque	*Macaca*	1031 (552:479:0)	8	Captive	Necropsy	0.00 – 37.90	By sex, sources combined
		57 (27:30:0)	4	Captive	Necropsy	Immatures and mature individuals	
Chimpanzee	*Pan*	70 (30:40:0)	8	Captive	Necropsy	0.01 – 59.41	By sex, sources combined
		49 (18:31:0)	5	Captive	Necropsy	Immatures and adults	
		11 (8:3:0)	1	Wild	Necropsy	Immatures and adults	
		3 (2:1:0)	3	Wild	Necropsy	Immatures and adults	
Orangutan	*Pongo*	7 (4:3:0)	8	Captive	Necropsy	0.05 – 33.48	Sex and source combined
		4 (2:2:0)	5	Captive	Necropsy	Immatures and adults	
		29 (16:13:0)	1	Wild	Necropsy	Immatures and adults	
Gorilla	*Gorilla*	4 (3:1:0)	8	Captive	Necropsy	0.00 – 31.13	Sex and source combined
		10 (5:5:0)	1	Wild	Necropsy	Immatures and adults	
		2 (1:1:0)	5	Captive	Necropsy	Immatures and adults	

[1]? = sexes unknown or combined.

[2]1: Count (1945); 2: endocranial volume (see text for details); 3: Crile & Quiring (1940); 4: Kennard & Willner (1941a); 5: Kennard & Willner (1941b); 6: Kennard & Willner (1941c); 7: Manocha (1979); 8: Yerkes Regional Primate Research Center (YRPRC).

[3]Crile & Quiring (1940) provide sample size and mean rather than individual datapoints for all taxa except *Pan*.

Table 1.3 Testing the brain allometries for linearity

Taxon	Sex	n	R_1^2	R_2^2	df	F
Saimiri	F	28	0.820	0.917	25	2.303**
Saimiri	M	25	0.728	0.951	22	3.881***
Cebus	M&F	52	0.711	0.778	49	1.267**
Ateles	M&F	23	0.757	0.756	20	0.281
Alouatta	M&F	60	0.379	0.379	57	0.179
Cercocebus	F	50	0.740	0.822	47	1.328***
Cercocebus	M	53	0.794	0.847	47	1.283**
Macaca	F	507	0.703	0.752	504	0.790***
Macaca	M	580	0.759	0.801	573	0.699***
Pan	F	68	0.599	0.810	65	1.869***
Pan	M	68	0.618	0.775	65	1.564***
Pongo	M&F	40	0.369	0.415	37	0.648
Gorilla	M&F	15	0.776	0.798	12	0.497
		Mean ± sd	0.673 ± 0.147	0.754 ± 0.169		
		se	0.041	0.047		
		95% confidence interval	0.591 – 0.755	0.660 – 0.848		

* = P ≤ 0.05; ** = P ≤ 0.01; *** = P ≤ 0.001. R_1^2 and R_2^2 are the coefficients of determination of the linear and quadratic regressions respectively.

b] Statistical analyses: testing the three predictions

1] Is the curve describing primate postnatal brain allometry linear?

The amount of variation explained by linear regression is consistently high across the samples, with a mean R_1^2 of 0.673 ± 0.147 (Table 1.3). Mean R_2^2 across all samples is 0.754 ± 0.169. In 9 of the 13 data samples analysed, the proportion of variation explained by quadratic regression is significantly higher than the proportion of variation explained by linear regression. In these samples, postnatal brain allometry is non-linear. This indicates that there is a significant change in the rate of brain growth relative to body growth after birth in these primates that is not consistent with the model presented above.

The four taxa which do not depart from linearity in their brain allometries are *Ateles*, *Alouatta*, *Pongo* and *Gorilla*. The lowest R_1^2 of all samples is found in *Pongo* (= 0.369), although *Alouatta* shows similarly low and non-significantly different R^2 values ($R_1^2 = R_2^2 =$ 0.379). The trend to linearity observed in these species may have two explanations:

1] Postnatal brain growth in these four species is linear, i.e. no difference in brain growth rate relative to body mass is observed over ontogeny.

2] The small size of each of these four samples may obscure curvilinearity. *Gorilla* and *Ateles* in particular have a low number of datapoints in their samples (n = 15 and 22 respectively).

Of the four taxa which show linearity, it is likely that the *Pongo* sample is also biased by a relatively low number of datapoints, especially at the lower end of the body size range (Fig 1.3a). Only two datapoints represent very young individuals in this sample. The *Gorilla* sample also under-represents early postnatal growth (Fig 1.3b) but is similar in shape to the platyrrhine samples (*Ateles* and *Alouatta*) that show linearity (Fig 1.3c and d). There is no obvious inflexion of the curve in either of these two latter taxa. It is interesting to note that *Gorilla* and *Alouatta* are relatively non-encephalised as adults (see below). The possibility that this type of linear growth contributes to a low level of adult encephalisation is discussed below.

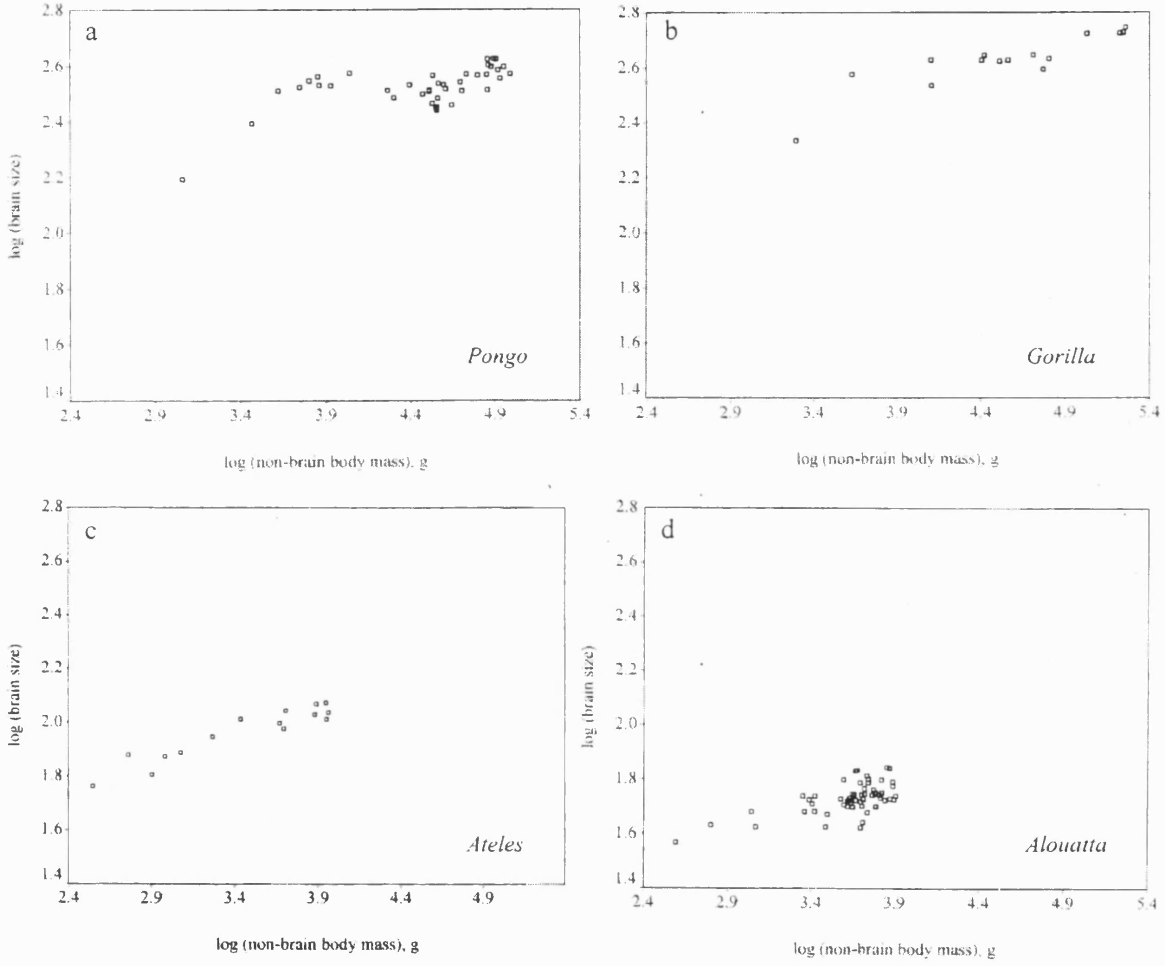


Figure 1.3. Brain allometries in the linear samples. (a) *Pongo;* (b) *Gorilla;* (c) *Ateles;* (d) *Alouatta.* Brain size is represented by brain mass or endocranial volume as appropriate. Non-brain body mass = whole body mass – brain size.

2] Is the slope of the postnatal post-inflexion curve approximately 0.1 or 0.2?

The pre-and post-inflexion slope values of the non-linear allometries are given in Table 1.4. Only in the *Cebus* (Fig 1.4) and *Cercocebus* (Fig 1.5) samples is a slope of 0.2 included in the 95% confidence interval of the post-inflexion slope (0.086 ± 0.059 for *Cebus*, 0.170 ± 0.029 and 0.141 ± 0.062 for *Cercocebus* females and males respectively). The other samples fall below the 0.2 prediction. Seven of the samples include either 0.2 or 0.1 in the slope 95% confidence interval. The only taxa not to include either 0.2 or 0.1 in the slope value estimate are the female *Macaca* sample and the male *Samiri* sample.

Both *Macaca* samples (Fig 1.6) have very low slope values for this part of the curve. The 95% confidence interval of the female *Macaca* slope only just excludes 0.1 (0.082 ± 0.008); the male *Macaca* slope's confidence interval includes 0.1 (0.095 ± 0.016). Although the slope is significantly lower than 0.2 in the male sample, their allometric curve is consistent with the model presented above. Male *Saimiri* also show a low post-inflexion slope that includes zero in its confidence interval (0.048 ± 0.024). On the other hand, the female *Saimiri* show a much higher slope than the males (0.128 ± 0.027), consistent with the predicted slope of 0.1 – 0.2. The male *Saimiri* sample contains only a few datapoints in the post-inflexion period, and this may account for the discrepancy between the sexes in their slope values (Fig 1.7).

The two chimpanzee samples (Fig 1.8) show slopes that are consistent with each other, both lower than 0.2, but including 0.1 in the slope confidence interval (0.149 ± 0.020 and 0.137 ± 0.020 for females and males respectively). The mean post-inflexion slope across all samples included in Table 1.4 is closer to 0.1 than 0.2 (0.115 ± 0.039). It appears that, in these primate species, the pattern of post-inflexion brain growth is consistent with that predicted for mammals in general. After the inflexion of the brain allometry, brain mass increases in relation to body mass at a rate that can be predicted from growth in non-primate mammals. Some taxa show very low slope values. In two samples (*Cebus*, *Saimiri* males), the slope cannot be statistically distinguished from zero. However, a visual inspection of the

Table 1.4 Pre- and post-inflexion RMAs of the non-linear samples

Taxon	Sex	Fig.	Pre-inflexion RMA slope ± se	Pre-inflexion RMA 95% confidence interval for slope	Pre-inflexion RMA intercept	Post-inflexion RMA slope ± se	Post-inflexion RMA 95% confidence interval for slope	Post-inflexion RMA intercept
Saimiri	F	1.7b	0.666 ± 0.068	0.530 – 0.802	-0.157	0.128 ± 0.027	0.074 – 0.182	1.024
Saimiri	M	1.7d	0.840 ± 0.042	0.756 – 0.924	-0.534	0.048 ± 0.024	0.000 – 0.096	1.333
Cebus	M&F	1.4b	0.422 ± 0.047	0.328 – 0.516	0.584	0.086 ± 0.059	-0.032 – 0.204	1.532
Cercocebus	F	1.5b	0.607 ± 0.060	0.487 – 0.727	0.190	0.170 ± 0.029	0.112 – 0.228	1.378
Cercocebus	M	1.5d	0.353 ± 0.035	0.283 – 0.423	0.868	0.141 ± 0.062	0.017 – 0.265	1.499
Macaca	F	1.6b	0.381 ± 0.022	0.337 – 0.425	0.756	0.082 ± 0.008	0.066 – 0.098	1.624
Macaca	M	1.6d	0.404 ± 0.020	0.364 – 0.444	0.713	0.095 ± 0.016	0.063 – 0.127	1.599
Pan	F	1.8b	0.522 ± 0.062	0.398 – 0.646	0.548	0.149 ± 0.020	0.109 – 0.189	1.886
Pan	M	1.8d	0.523 ± 0.062	0.399 – 0.647	0.576	0.137 ± 0.020	0.097 – 0.177	1.967
		Mean ± sd	0.524 ± 0.159			0.115 ± 0.039		
		se	0.053			0.013		
		95% confidence interval	0.418 – 0.630			0.089 – 0.141		

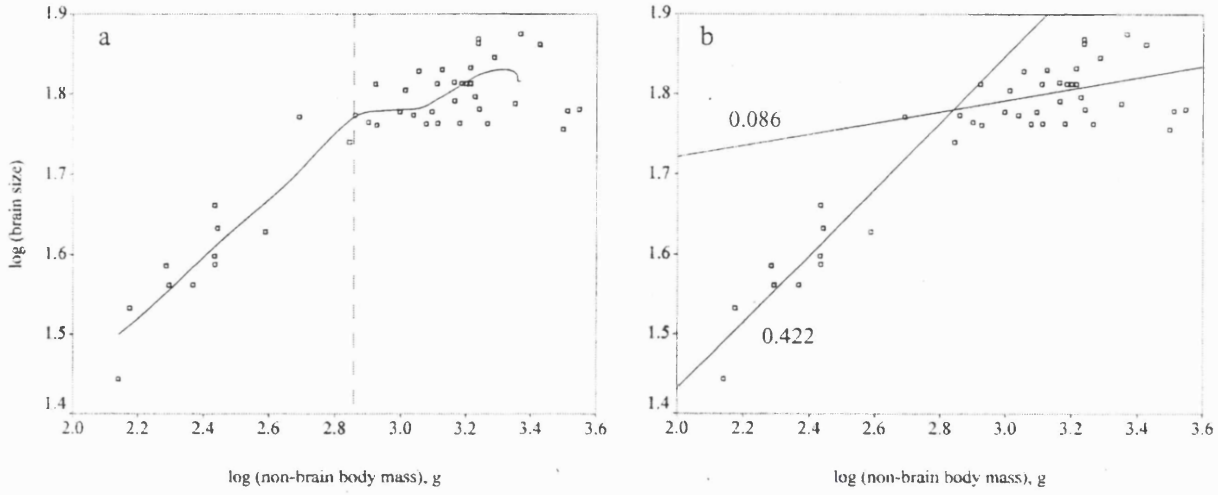


Figure 1.4. Brain allometry in *Cebus*. (a) Loess estimate of the inflexion, ∂. Hatched line represents the position of the inflexion as inferred from loess. (b) Pre- and post-inflexion RMAs (slope values shown). Details of RMAs given in Table 1.4.

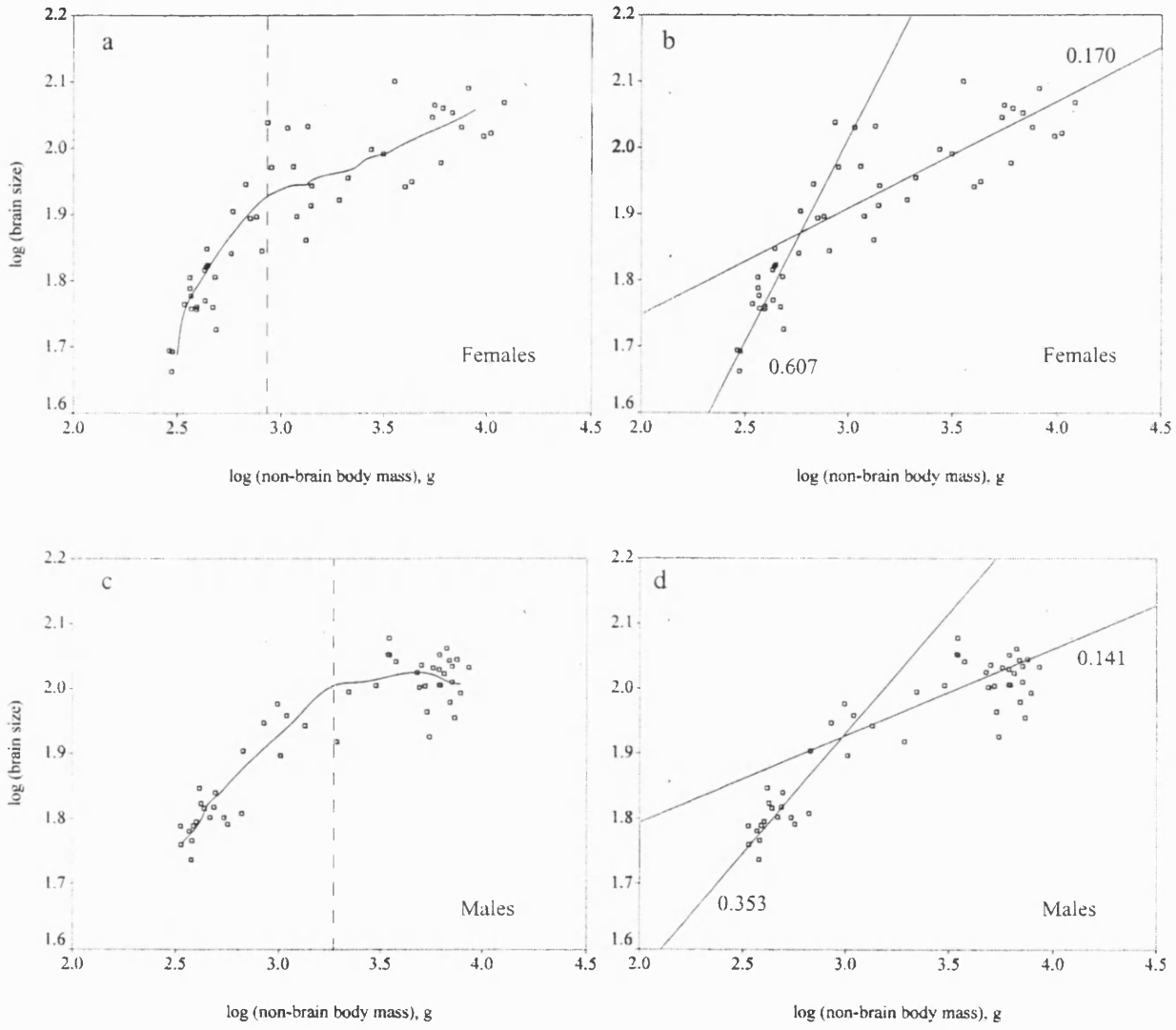


Figure 1.5. Brain allometry in *Cercocebus*. (a) Loess estimate of ∂ in females; (b) pre- and post-inflexion RMAs in females (slope values shown); (c) loess estimate of ∂ in males; (d) pre- and post-inflexion RMAs in males (slope values shown). Details of RMAs given in Table 1.4.

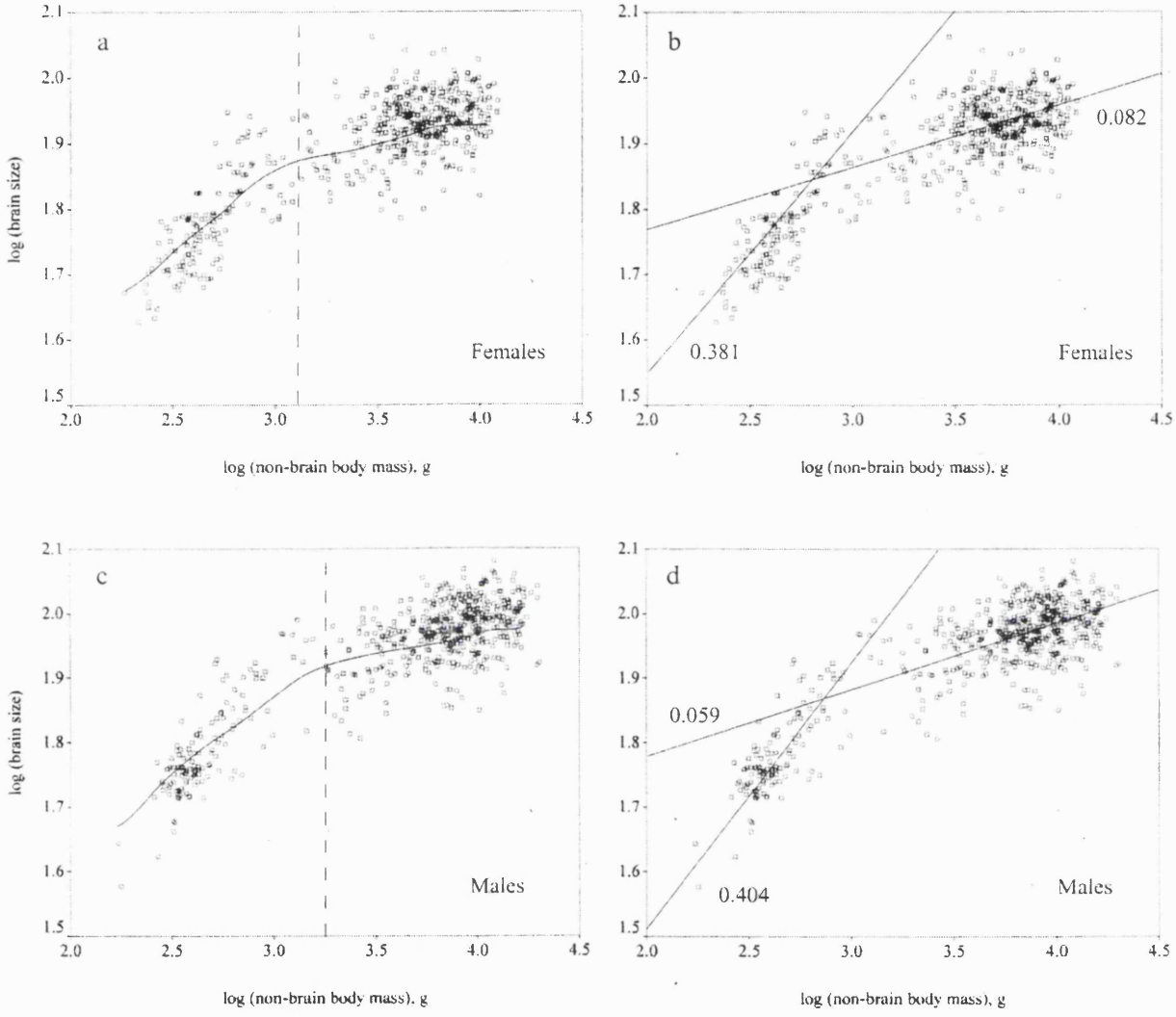


Figure 1.6. Brain allometry in *Macaca*. a] Loess estimate of ∂ in females b] Pre- and post-inflexion RMAs in females (slope values shown). c] Loess estimate of ∂ in males d] Pre- and post-inflexion RMAs in males (slope values shown). Details of RMAs given in Table 1.4.

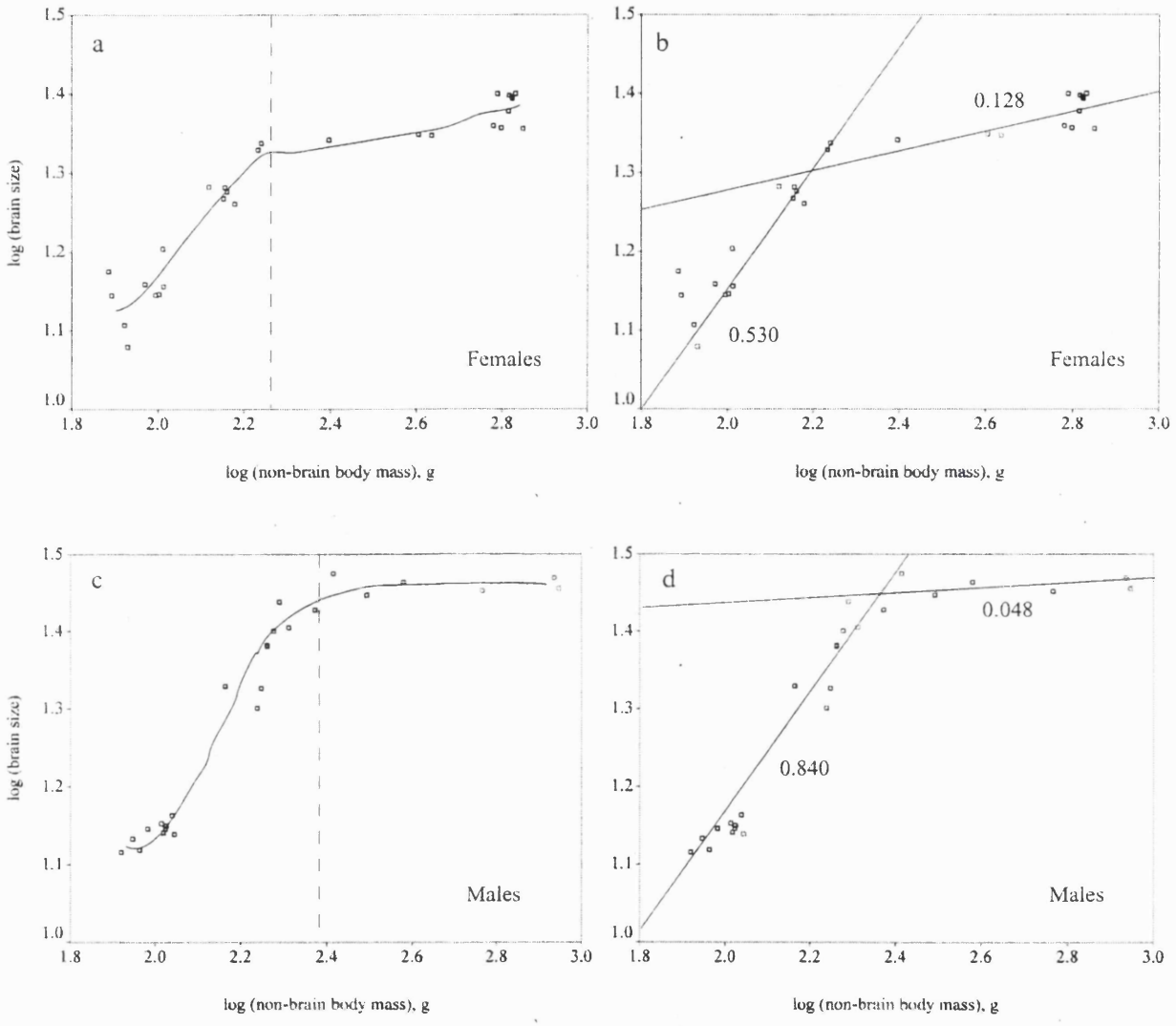


Figure 1.7. Brain allometry in *Saimiri.* a] Loess estimate of ∂ in females b] Pre- and post-inflexion RMAs in females (slope values shown). c] Loess estimate of ∂ in males d] Pre- and post-inflexion RMAs in males (slope values shown). Details of RMAs given in Table 1.4.

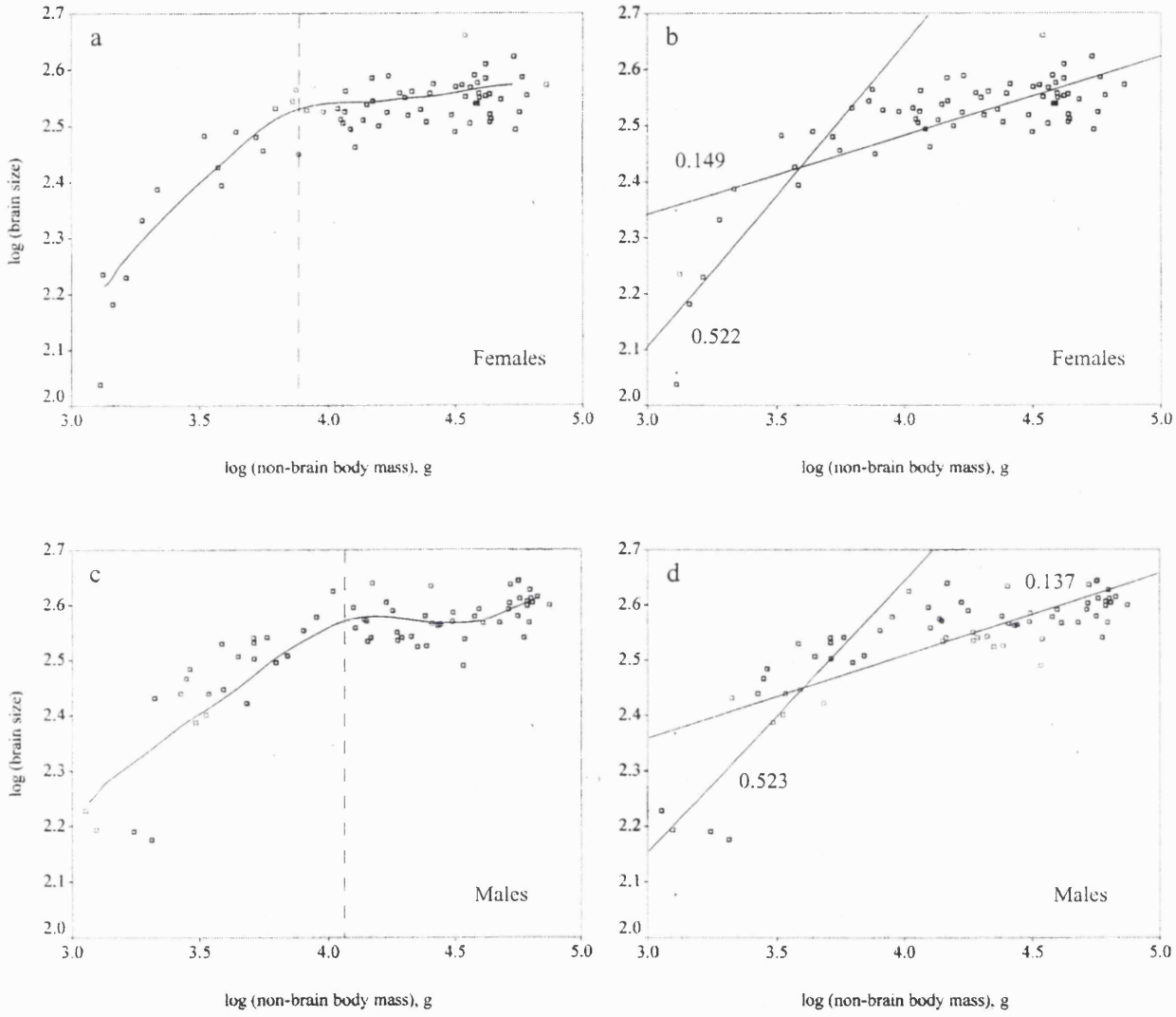


Figure 1.8. Brain allometry in *Pan*. (a) Loess estimate of ∂ in females; (b) pre- and post-inflexion RMAs in females (slope values shown); (c) loess estimate of ∂ in males; (d) pre- and post-inflexion RMAs in males (slope values shown). Details of RMAs given in Table 1.4.

Table 1.5 Whole-sample RMAs of the linear allometries

Taxon	Sex	Fig.	Whole-sample RMA slope ± s.e.	Whole-sample RMA 95% confidence interval for slope	Whole-sample RMA intercept
Pongo	M&F	1.4a	0.162 ± 0.021	0.120 – 0.204	1.802
Gorilla	M&F	1.4b	0.174 ± 0.023	0.128 – 0.220	1.839
Ateles	M&F	1.4c	0.215 ± 0.023	0.169 – 0.261	1.214
Alouatta	M&F	1.4d	0.233 ± 0.023	0.187 – 0.279	0.921
		Mean ± sd	0.196 ± 0.034		
		se	0.017		
		95% confidence interval	0.162 – 0.230		

Cebus data suggests that there is some increase in brain size after the inflexion (Fig 1.4), and the confidence interval for the *Cebus* slope includes 0.1 and 0.2.

Those samples which show linear postnatal brain allometries (*Alouatta, Ateles, Pongo, Gorilla*) conform closely to the model (Table 1.5). Postnatal brain growth in these taxa is similar to that predicted for other, non-primate mammals, i.e. linear and with a slope of 0.2 (mean slope 0.196 ± 0.034). All four of the linear allometry samples have 95% confidence intervals for the RMA slope that include 0.2; the two platyrrhine species show higher slopes than the two hominid species, but these differences are not statistically significant, as the confidence intervals for all four slopes overlap (Table 1.5).

3] Does the postnatal pre-inflexion curve represent an extension of the prenatal brain growth period, i.e. does the slope of the pre-inflexion curve equal 1.0?

None of the samples included in Table 1.4 have slopes that include 1.0 in the 95% confidence interval. The male *Saimiri* sample comes close, with a slope of 0.840 ± 0.042 (Fig 1.7). The slope values of the other samples range from 0.353 ± 0.035 in male *Cercocebus,* to 0.666 ± 0.068 in female *Saimiri*. The cross-sample mean is 0.524 ± 0.159, suggesting that brain mass increases at half the rate (relative to body mass) that it is predicted to before birth. The post-inflexion period does not, therefore, represent an extension of the prenatal growth phase as predicted by the model. The pace of primate brain mass increase in this period is slower than brain growth *in utero* but faster in relation to body mass than that seen after the inflexion of the allometric curve.

c] Modelling growth and encephalisation

How do these departures from the allometric model affect brain and body proportions (i.e. encephalisation) over ontogeny? The predicted RMAs shown in Figure 1.9 represent brain allometries as they would appear if each primate taxa conformed to a non-primate pattern of

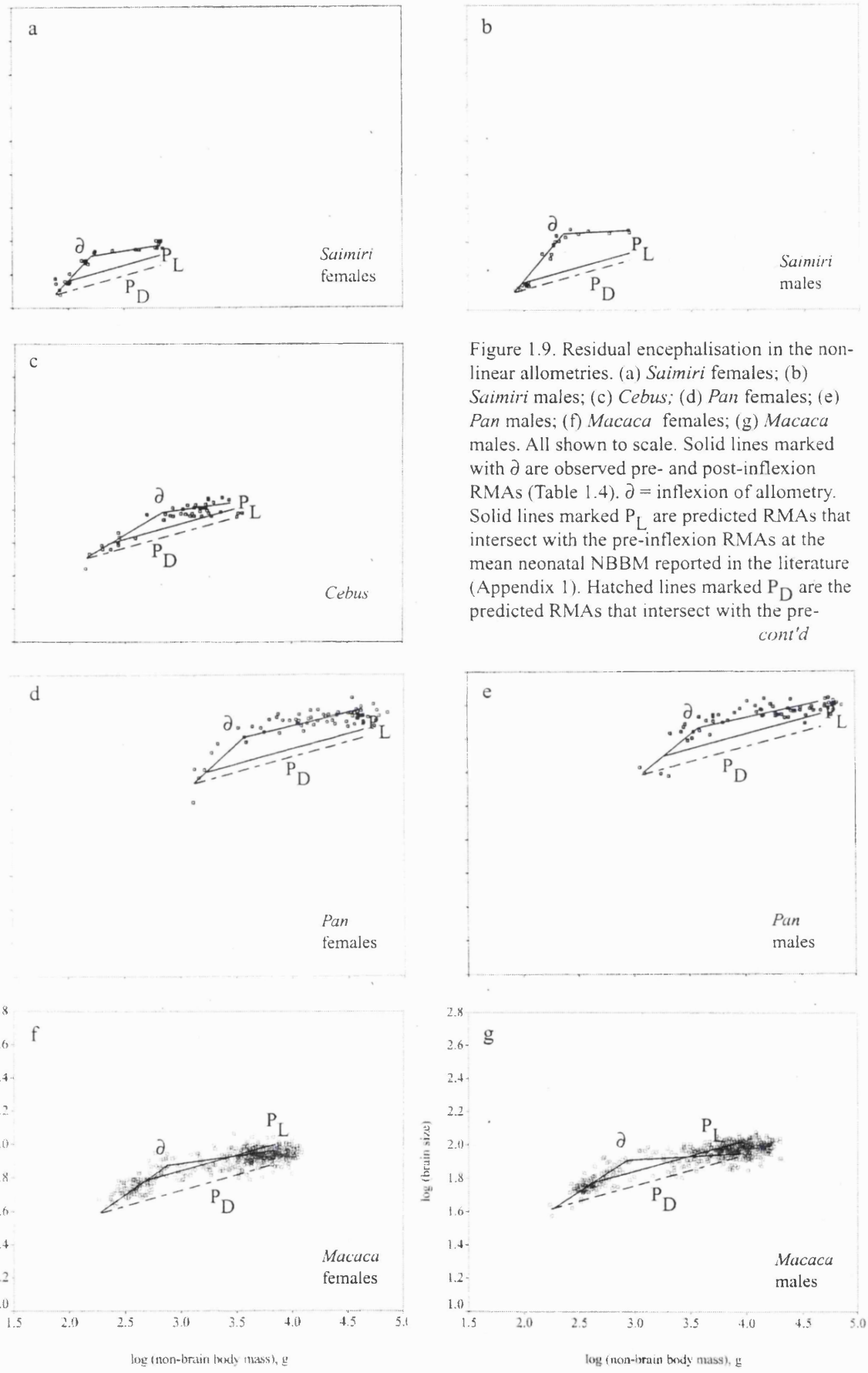


Figure 1.9. Residual encephalisation in the non-linear allometries. (a) *Saimiri* females; (b) *Saimiri* males; (c) *Cebus;* (d) *Pan* females; (e) *Pan* males; (f) *Macaca* females; (g) *Macaca* males. All shown to scale. Solid lines marked with ∂ are observed pre- and post-inflexion RMAs (Table 1.4). ∂ = inflexion of allometry. Solid lines marked P_L are predicted RMAs that intersect with the pre-inflexion RMAs at the mean neonatal NBBM reported in the literature (Appendix 1). Hatched lines marked P_D are the predicted RMAs that intersect with the pre-

cont'd

Fig 1.9 cont'd

inflexion RMAs at the minimum NBBM value for each sample. Minimum values were used because ages were not known for the majority of the data, and neonatal values could not be estimated. Mean reported adult NBBM (Appendix 1) was used as a measure of adult mass. The predicted RMAs impose a slope of 0.2 on the data. The intercepts of these predicted RMAs were calculated by substituting 0.2 for the appropriate slope value for each sample, and then re-arranging the terms of the equation (Table 1.6). The *Cercocebus* samples are not shown in this figure, or included in Table 1.6, because no estimates of neonatal brain mass for this species were available from the literature, and NBBM at birth could not be calculated.

postnatal growth. The shape of the observed allometric curve influences encephalisation during ontogeny and in adulthood (Fig 1.9). At the point of inflexion of the allometric curve (δ), all the samples have brains that are larger than would be predicted from the model. However, at adult NBBM, residual encephalisation varies between the different samples, with some taxa showing larger than, and other taxa showing smaller than, predicted relative brain size. Although the taxa are highly encephalised at δ (positive residual encephalisation), by adulthood residual encephalisation is generally low because NBBM increases more rapidly in relation to brain mass. This ties in well with Martin's (1983) observation that, in the later stages of growth, encephalisation is influenced by body, rather than brain, growth. However, the only significant association to emerge from the correlation analysis (see above) is between adult residual encephalisation and the pre-inflexion RMA slope ($r = 0.829$, $P = 0.010$, $n = 7$). Thus the only growth allometry variable that is a good predictor of species-level adult residual encephalisation is the rate at which the brain increases in mass relative to body mass, prior to the allometry inflexion. Over 68% of the variation in residual encephalisation seen in the different samples is explained by variation in the pre-inflexion slopes ($R^2 = 0.687$). Neither the rate of brain growth after the inflexion, nor the 'length' of each stage of growth, appears to be an important determinant of encephalisation later in life.

The small platyrrhines (*Saimiri, Cebus*) have a high level of residual encephalisation as adults, with brain masses that are at least 12% larger relative to body size than predicted (Fig 1.9a to c). This value increases if data-specific, rather than literature-derived, NBBM is used to predict brain mass (Table 1.6). The minimum NBBM datapoint was used as an estimate of neonatal mass because ages are not known, and actual neonatal masses could not be calculated. The same trend of increased residual encephalisation when minimum NBBM is used is seen in the *Pan* samples (Fig 1.9d, e). Female and male *Pan* brains are 17.4% and 13.8% heavier than predicted when mean reported neonatal NBBM is used as a predictor, but 23.7% and 25.1% larger when the minimum NBBM is used respectively (Table 1.6).

The adult macaques have negative residual encephalisation, i.e. smaller brains than predicted (88.3% and 84.8% of predicted size for females and males respectively). However,

Table 1.6 Predicting adult brain mass

			Predicting adult brain mass from mean reported neonatal NBBM				Predicting adult brain mass from minimum NBBM in sample			
		Adult brain mass (g)[1]	Predicted RMA[2,3] i.e. slope = 0.2	Predicted adult brain mass (g)	Difference	% Difference	Predicted RMA[2,4] i.e. slope = 0.2	Predicted adult brain mass (g)	Difference	% Difference
		A		B	C = A – B	| C | / (A+B/2)		D	E = A – D	| E | / (A+D/2)
Saimiri	F	24.04	y = 0.2x + 0.760	20.79	3.25	0.145	y = 0.2x + 0.719	18.91	5.13	0.239
Saimiri	M	29.80	y = 0.2x + 0.743	21.44	8.36	0.326	y = 0.2x + 0.695	19.20	10.6	0.433
Cebus	M&F	67.65	y = 0.2x +1.087	60.32	7.33	0.115	y = 0.2x +1.059	56.58	11.07	0.178
Ateles	M&F	110.66	y = 0.2x + 1.257	106.91	3.75	0.034	y = 0.2x + 1.252	105.74	4.92	0.045
Alouatta	M&F	58.08	y = 0.2x + 1.008	63.39	-5.31	0.087	y = 0.2x + 1.006	57.84	0.24	0.004
Macaca	F	85.55	y = 0.2x + 1.231	96.16	-10.61	0.117	y = 0.2x + 1.165	82.56	2.99	0.036
Macaca	M	94.59	y = 0.2x +1.248	110.10	-15.51	0.152	y = 0.2x +1.170	91.90	2.69	0.029
Pan	F	370.33	y = 0.2x +1.576	310.90	59.43	0.174	y = 0.2x +1.549	291.89	78.44	0.237
Pan	M	409.12	y = 0.2x +1.610	356.16	52.96	0.138	y = 0.2x +1.560	317.86	91.26	0.251
Pongo	M&F	374.11	y = 0.2x + 1.682	429.93	-55.82	0.139	y = 0.2x + 1.686	433.90	-59.79	0.148
Gorilla	M&F	596.49	y = 0.2x +1.755	533.33	63.16	0.112	y = 0.2x +1.753	593.20	3.29	0.006
		Mean ± sd				0.140 ± 0.072				0.146 ± 0.137
		se				0.021				0.041
		95% confidence interval of mean				0.098 – 0.182				0.064 – 0.228

[1]Calculated from the post-inflexion RMA or whole-sample RMA allometry as appropriate, using mean reported adult NBBM as an estimate of adult body size. Reported data from the literature given in Appendix 1. [2]Represents the postnatal brain allometry as predicted by the model discussed in the text. Slopes inferred as 0.2. [3]Intercept calculated from i = m – 0.2k, where i = intercept of predicted RMA, k = mean reported neonatal NBBM, and m = estimated neonatal brain mass (i.e. neonatal brain mass inferred from reported NBBM using the appropriate pre-inflexion or whole-sample RMA given in Tables 1.4 and 1.5). Reported data from the literature given in Appendix 1. [4]Intercept calculated from i = m – 0.2p, where i = intercept of predicted RMA, p = minimum NBBM in sample, and m = estimated neonatal brain mass (i.e. neonatal brain mass inferred from minimum NBBM using the appropriate pre-inflexion or whole-sample RMA given in Tables 1.4 and 1.5).

mean reported neonatal NBBM is considerably smaller than the minimum datapoint in both the *Macaca* samples (Fig 1.9f, g). When the minimum neonatal NBBM value is used to predict brain mass in adulthood, macaques have positive residual encephalisation, i.e. larger brains than expected (3.6% and 2.9% larger than predicted for females and males respectively). These old world monkeys are less encephalised as adults – i.e. have smaller brains relative to body mass – than the either the new world monkeys or chimpanzees included in the analysis.

The linear-allometry samples show either positive or negative adult residual encephalisation depending on whether the RMA slope is higher than, or lower than 0.2 (Fig 1.10). The *Ateles*' brains are on average 3.4% and 4.5% larger than predicted, depending on the source of the neonatal data (Fig 1.10c). *Alouatta* appears to have a larger brain than expected in adulthood: 8.7% larger if mean reported neonatal NBBM is used to predict brain mass, but only larger 0.4% if the sample-specific value is used (Fig 1.10d). Adult *Ateles* and *Alouatta* are reported to be relatively highly encephalised, and relatively non-encephalised, respectively (Harvey & Clutton-Brock, 1985).

The known pattern of great ape encephalisation (relatively non-encephalised adult gorillas and relatively encephalised adult orangutans; Harvey & Clutton-Brock, 1985) is reversed in this analysis (Fig 1.10a and b). This is likely to be the result of each samples' small number of datapoints; in *Pongo*, only one datapoint (indicated by an arrow in Figure 1.10a) represents NBBM smaller than 3kg, and this individual has a brain size much smaller than predicted by the whole-sample RMA. If this datapoint were taken as the origin of the postnatal growth allometries, *Pongo* would, in fact, have a brain size larger than predicted for its body size (shown by predicted line marked with an asterisk in Figure 1.10a). In a similar way, the gorilla data is scant, and it is possible that this lack of data obscures biological trends (Fig 1.10b). More information about brain size at birth in these species is needed to resolve this problem.

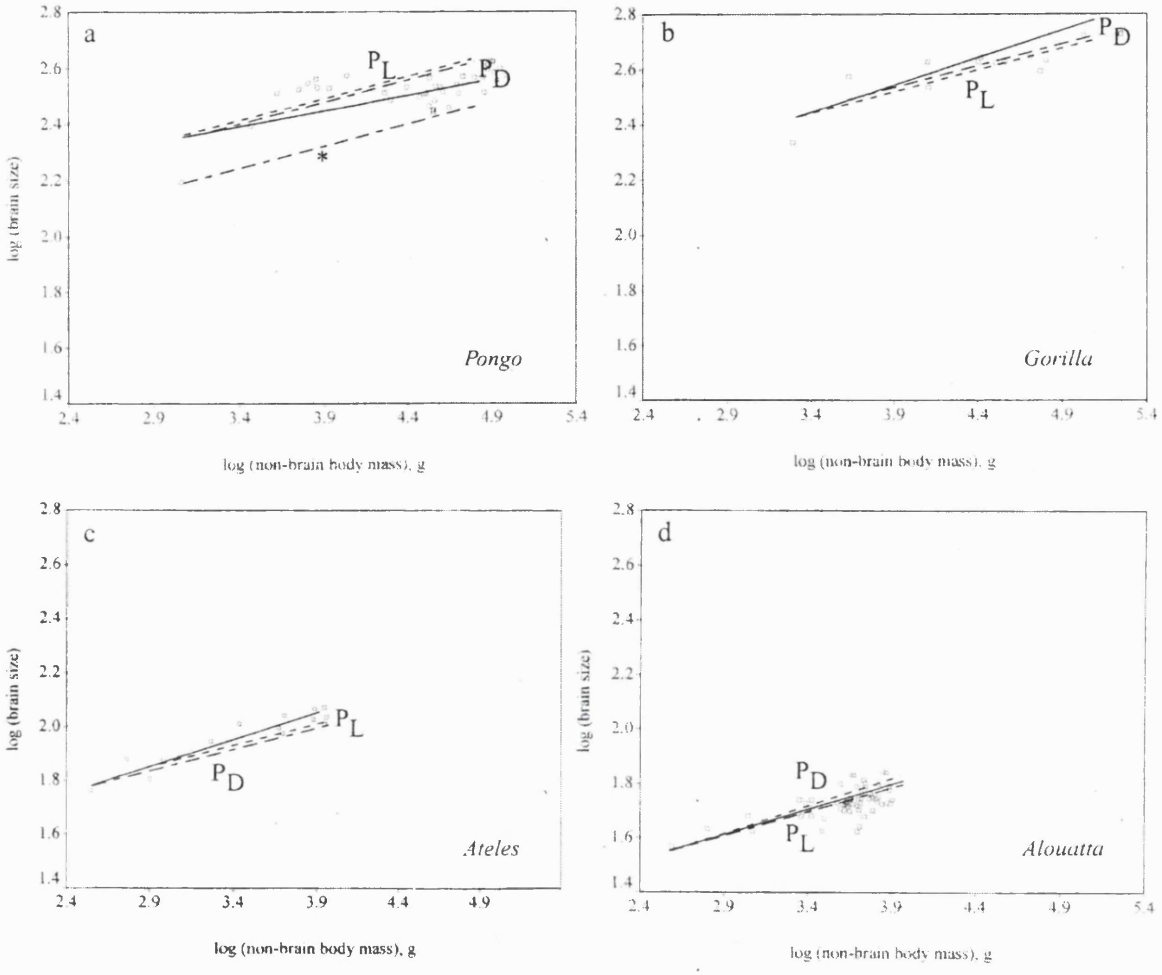


Figure 1.10. Residual encephalisation in the linear allometries. (a) *Pongo;* (b) *Gorilla;* (c) *Ateles;* (d) *Alouatta.* Solid lines are whole sample RMAs (Table 1.5). Dashed lines marked P_L are predicted RMAs that intersect with the pre-inflexion RMAs at the mean neonatal NBBM reported in the literature (Appendix 1). Hatched lines marked P_D are the predicted RMAs that intersect with the pre-inflexion RMAs at the minimum NBBM value for each sample. Minimum values were used because ages were not known for the majority of the data, and neonatal values could not be estimated. The hatched line marked by an asterisk in (a) is the predicted RMA when the arrowed datapoint is used as the origin (see text for explanation). Mean reported adult NBBM (Appendix 1) was used as a measure of adult mass. The predicted RMAs impose a slope of 0.2 on the data. The intercepts of these predicted RMAs were calculated by substituting 0.2 for the appropriate slope value for each sample, and then re-arranging the terms of the equation (Table 1.6).

1.3.4 Section summary

In summary, the taxa included in these analyses conform to the predicted model (Deacon, 1990) only in the post-inflexion phase of growth. Nine of the 13 samples show postnatal brain allometries that are non-linear. The slopes of the pre-inflexion curves in these samples are significantly lower than the predicted value of 1.0. This part of the brain allometry does not represent an extension of the prenatal growth curve. Similarly, the slopes of the post-inflexion curve are generally lower than predicted, although the 95% confidence interval of the mean in all but two samples includes 0.1 or 0.2. Primate growth is therefore unusual because most primate species display an elevated rate of relative brain mass increase after birth that is faster than predicted for other taxa but slower than brain growth *in utero*. When the curves are compared with the predicted allometries, all the taxa are more encephalised than expected during the pre-inflexion period and at δ. This is a result of elevated RMA slope values in the pre-inflexion period, compared to the model's predictions. Those samples that do not show curvilinearity in their allometries deviate only slightly from the predicted postnatal slope, and so show only slight deviation from predicted encephalisation as adults. Adult residual encephalisation, i.e. relative to the predicted level of encephalisation, is influenced by pre-inflexion growth. Growth after the rate of brain growth slows is not significantly associated with adult residual encephalisation in this species-level analysis.

1.4 Weaning and brain allometries

1.4.1 Definitions of weaning: theory...

Weaning is a process that involves many behavioural and physiological changes in both the mother and the infant. Metabolic and growth rates, milk composition and output, energy availability and juvenile mortality have all been linked to the timing and pattern of weaning (Oftedal, 1984; Nicoll & Thompson, 1987; Lee *et al.*, 1991; Janson & Van Schaik, 1993).

Milk production itself is regulated by the suckling arc reflex, a complex interaction of infant behaviour and physiology and the uterine/ovarian cycle regulating hormones (Fig 1.11). Weaning can be therefore defined in a variety of ways and with respect to several parameters. Dettwyler (1995) describes weaning as the complete cessation of breastfeeding (C in Figure 1.12), but where suckling provides non-nutritive support for the infant (as it does in primate infants, Lee *et al.* 1991), this definition may greatly exaggerate the period of nutritional/energetic dependence. It also ignores the transitional nature of weaning. Two definitions which define weaning more specifically and in terms of nutrient transfer are suggested by Martin (1984) and Lee *et al.* (1991). Martin (1984) describes weaning as shifts in the slope of the investment rate curve; Lee *et al.* (1991:101) refine this concept further, defining weaning as "a rapid decline in rate of [nutrient] transfer." They note that suckling terminates at a specific point in time, but determining this point in taxa which undergo slow rates of transfer (such as primates, Oftedal, 1984) is almost certainly impossible. More is to be gained, therefore, by focussing on rate changes than on the suckling behaviour itself (Martin, 1984). In this thesis, the term 'weaning' is used to denote the transition from nutritive and energetic dependence to independence, whereby the majority of the infant's needs are met by sources other than the mother (Fig 1.12). As noted above, psychological and behavioural development are also important aspects of weaning, and should be kept in mind in the following discussions of the weaning process.

1.4.2 Definitions of weaning: ...and practice

An empirical measure of weaning has proved as elusive as a theoretical one, especially when a comparative approach is required. Weaning age can be defined in relation to either the mother or infant, and usually refers to completion of weaning, or the age at which nutritional/energetic independence is achieved (see below). The age at which the weaning process begins is harder to estimate. Intake of solid food provides an indication that nutritive or energetic dependency is starting to shift away from the mother, but in practice

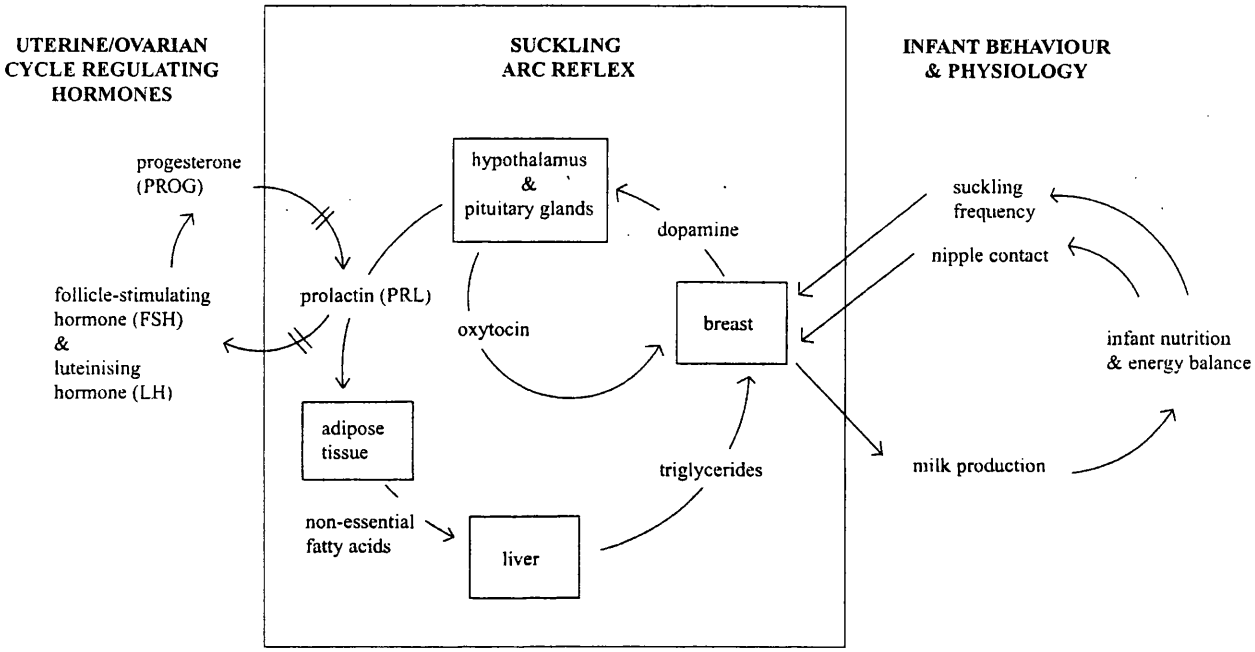


Figure 1.11. The suckling arc reflex. Hormones and metabolites in the breast and other female organs/tissues mediate the interaction of infant behaviour/physiology with the uterine/ovarian cycle regultaing hormones. Based on Lunn (1992) and Lincoln & Paisley (1982).

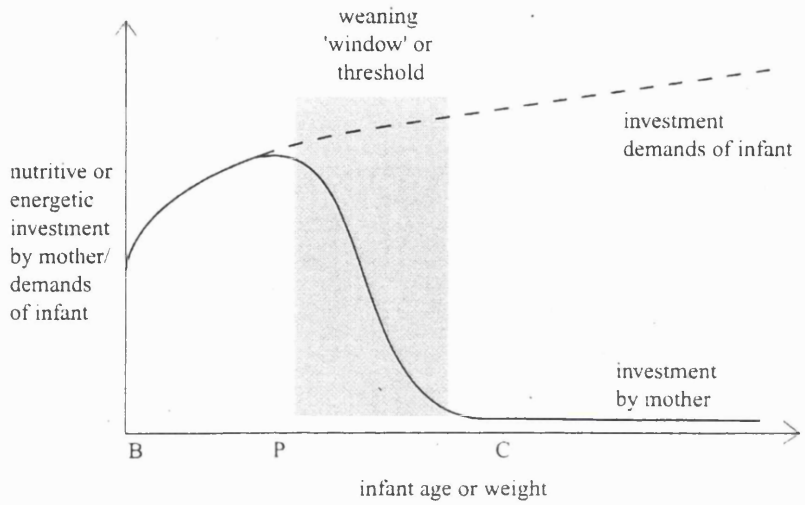


Figure 1.12. The weaning threshold in mammals. Based on Lee *et al.* (1991). B = birth; P = peak investment by mother; C = minimal/complete cessation of investment by mother. The shaded area represents the 'window' in which weaning occurs i.e. when the mother no longer meets infant demands.

the first occurrence of food intake is often difficult to observe. Many primate infants will eat small amounts of solid foods early in life – well before peak lactation yield occurs – as part of the ontogeny of foraging efficiency and gastrointestinal competence (Janson & Van Schaik, 1993). For example, Lee *et al.* (1991) find that first solid food intake tends to occur when the infant weighs just over twice as much as it did at birth. Whether this should be thought of as weaning behaviour is uncertain.

In taxa that experience postpartum anovulation or amenorrhea, resumption of sexual activity on the part of the mother provides a behavioural indication of hormone activity (Altmann *et al.* 1978). However, this definition will be less useful for describing weaning in taxa where mothers reconceive before their current offspring is fully independent (e.g., capuchins, Recabarren *et al.*, in prep.). Lee (1987) focuses on the weaning behaviour of the infant rather than the mother in her study of weaning in vervet monkeys (*Cercopithecus aethiops*). She describes weaning as a function of suckling frequency and nipple contact rate. Nipple stimulation forms part of the suckling reflex arc (Fig 1.11), and while Lee (1987) finds that contact frequency does provide a good indication of maternal hormone levels, it may be less useful in determining levels of energy transfer.

The most prevalent method of measuring weaning is to predict weaning age and mass from other size and life history variables. Weaning age is often derived from interspecific values of interbirth interval minus gestation length (Lee, 1996). This measure is likely to reflect the length of suckling that accounts for the majority of the infant's nutritional needs, and has the advantage of being calculable in species for which no direct evidence of weaning behaviour exists (Lee *et al.*, 1991). Such broad-scale calculations are subject to error, however, as interspecific averages cannot allow for intraspecific variation in behaviour or physiology. While gestation does not seem to vary by more than a few days in most taxa for which accurate information on conception date is available (Ardito, 1976, Lee 1996), interbirth interval shows marked variation at low taxonomic levels (Lee, 1999). Interbirth interval minus gestation length probably provides a generally accurate estimate

of age at nutritional independence, but should be used with caution when measurement and calculation error ranges are unknown.

Most studies infer weaning mass from growth curves at estimated weaning age, but weaning mass has also been predicted from various other developmental and life history parameters using the comparative method. Gestation length, for example, has been used as a predictor of weaning mass, as has adult mass (Harvey & Clutton-Brock, 1985; Charnov & Berrigan, 1991, 1993). Lee *et al.*'s (1991) observation that weaning occurs when the infant weighs four times as much as at birth appears to be the most robust predictor across different primate and non-primate taxa, and is used to predict weaning in the following analyses

1.4.3 Weaning in mammals and primates

Primates are unusual because, in comparison with most non-primate mammals, they are highly encephalised, grow slowly, and spend a relatively long time in maternal dependency i.e. have a late age at weaning (see Introduction). It is pertinent to ask: how does weaning fit into the pattern of brain and body growth outlined above? Is the tempo of brain growth associated with the lactation strategies, and if so, what role does brain and body growth play in the weaning process?

We have already seen that body size is linked to mortality during the juvenile period, and that body size at weaning is an important correlate of survivorship in the early stages of life (Janson & van Schaik, 1993; Lee, 1996). The broadest interspecific analysis of weaning body mass variation to date is that undertaken by Lee *et al.* (1991). They compare maternal, birth and weaning mass and duration of lactation across three different taxa: haplorhine primates, ungulates and pinnipeds. Many of their results confirm predictions of theoretical studies; for example, that birth and weaning masses are positively associated with maternal size across all taxa. Most interestingly, they find that neonatal mass scales to weaning mass by a factor of four, and that this trend is consistent across all the taxa examined, including primates. Whether the infant is a primate, pinniped or ungulate, "when a weight of around four

times birth [mass] is reached, infants are weaned, irrespective of the time taken to achieve weaning" (Lee *et al.*, 1991:104). This trend remains significant when the dataset is enlarged and refined (Lee, 1999). The authors go on to suggest that:

> "...mothers, from a range of body sizes and foraging modes, appear to be under a similar constraint, that of their infants achieving a threshold weaning weight, while duration of lactation varies both inter- and intraspecifically irrespective of maternal size... Maternal effort is constrained to ensure growth to a threshold weaning weight in order to maximise offspring survival."
>
> (Lee *et al.*, 1991:109)

Lee *et al.* (1991) suggest that this threshold is likely to be a metabolic one, in the sense that the mother is no longer capable of supplying the nutritional needs of the infant without causing detriment to herself and her future offspring. We saw in the introduction, however, that it might be related to other factors of growth: for example, that the infant achieves a certain level of gastrointestinal competence for weaning to solid food occur. Yet another threshold to be crossed might be a behavioural one, i.e. that the infant must have developed at least some adult behaviours before weaning, whether those behaviours concern foraging, social interaction, and/or predator avoidance (Boinksi & Fragaszy, 1989; Janson & van Schaik, 1993). This may be especially true of primates, which often live in complex foraging and social environments (Dunbar, 1998). In addition, the growth and development of a relatively large and energetically-expensive brain in primates might impinge further on the metabolic capacity of the mother.

Ages are not known for the majority of the individuals included in the previous analyses, and an examination of the timing of the weaning process is therefore problematic. However, an investigation of how weaning mass and brain allometries interact is possible. As we have seen, brain size can be used as a proxy for brain maturation and metabolic mass. By investigating the relationship between brain size, body size and weaning, we can ask: is brain growth associated with strategies of weaning and lactation in primates?

Weaning status was known for individuals in only two of the samples analysed in Section 1.3, those of the *Saimiri* males and females. Manocha (1979) notes that infants were weaned at 60 dpp. Although Manocha does not indicate whether the *Saimiri* were forcibly removed from their mothers or left to wean naturally, it can be assumed the weanlings were self-sufficient, at least nutritionally, after weaning. Their body mass growth curves (body mass over age) show no faltering after weaning (Manocha, 1979). In both the male and the female samples, the transition from infant to weanling status occurs at the inflexion of the brain allometry (Fig 1.13). This implies that the change from rapid to slow brain growth coincides with the process of weaning. Brain mass at δ is 20.18g and 27.93g for female and male *Saimiri* (Table 1.7). Adult brain masses are 24.04g and 19.80g respectively. Brain mass at δ is therefore 17.5% and 6.5% smaller than adult brain mass respectively (Table 1.6). In other words, when *Saimiri* infants' brains come within an average of 12% of adult size, two events happen: one, brain growth slows; and two, weaning occurs. We can therefore predict that these two trends will be true of the other samples in the analysis:

1] Body mass at weaning will coincide with body mass at δ.

2] Brain mass at inflexion will be between 10% and 20% smaller than adult brain mass.

1.4.4 Materials and methods

In order to test the prediction that body mass at weaning coincides with body mass at inflexion, and also that brain mass at weaning is approximately 80% of adult brain mass, the appropriate mass variables were calculated as follows:

Brain mass and NBBM at δ for each of the samples was calculated from the appropriate RMA equations (Table 1.7). These were then used to calculate whole body mass (i.e. brain mass + NBBM), and were compared with weaning mass. Weaning mass was calculated as:

Weaning mass = (minimum NBBM + brain mass at minimum NBBM)*4 (1.12)

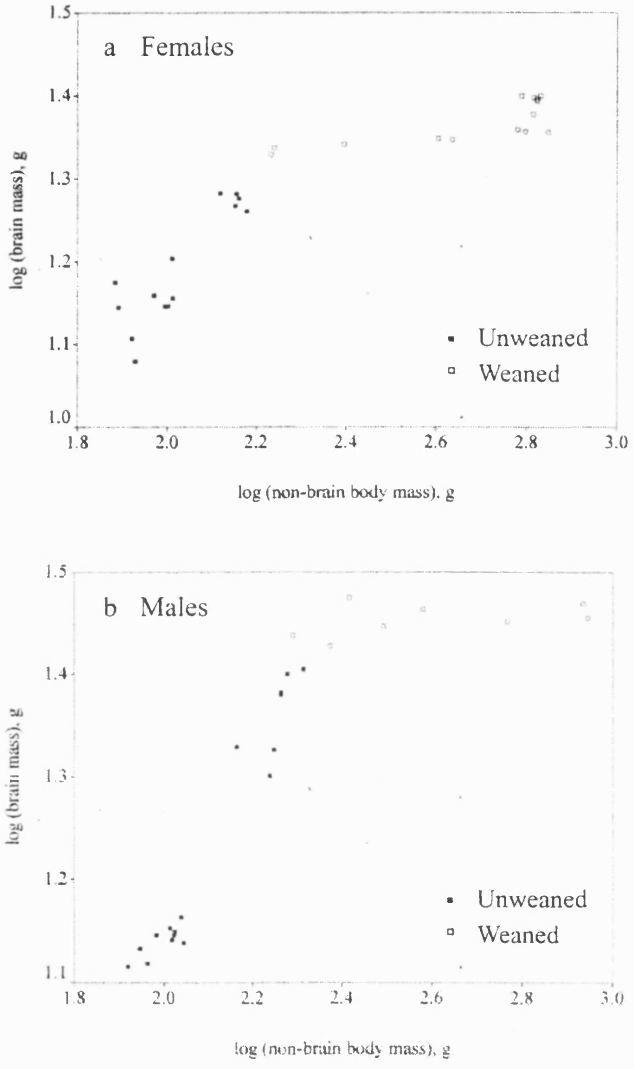


Figure 1.13. Weaning occurs at the inflexion of the brain allometry in *Saimiri*. (a) Females; (b) males. Solid squares = unweaned infants; open squares = weaned infants.

This is based on Lee *et al.*'s (1991) observation that weaning occurs when body mass is four times birth mass. Weaning mass was predicted for all samples except *Saimiri*, for which known weaning masses were available. Brain mass at δ was also compared with adult brain mass (Table 1.6).

1.4.5 Results

1] Mass at inflexion typically falls within 30% of predicted weaning mass.

When whole body mass at δ is compared with predicted weaning mass, the mean percent difference between the two is 31.0% ± 2.6 (Table 1.7). Some of the samples included in the analyses show very large differences in whole body mass at δ, compared to predicted weaning mass. For example, the *Cercocebus* samples are less than half as big at δ as they are predicted to be at weaning (the difference is 71.1% and 41.5% of mean in females and males respectively). *Cebus* and the female *Macaca* sample, on the other hand, have body masses at inflexion that are larger than predicted at weaning, although both by less than 10% (6.7% and 0.3% respectively). Surprisingly, the *Saimiri* samples also show a large difference between predicted weaning and inflexion masses (66.5% and 37.4% for females and males respectively). As described above, weaning status is known for these samples, and this difference highlights the fact that predicted weaning masses do not always reflect actual weaning mass in a particular population. Lee *et al.* (1991) find that the weaning to birth mass ratio varies from 2.3 to 9.4 in primates. In addition, Bowman & Lee (1995) tested for an interspecific weaning threshold with data from mother-reared captive rhesus macaques. They found that a threshold existed at the lower end of the range of birth-to-weaning mass multiples (3.2), and that when an infant's mass came within 15.04% of this threshold (1330g), it was weaned. Lee (1999) observes that "mothers were making behavioural decisions about the termination of investment in the current infant in the light of that infant's relative growth, the

Table 1.7 Comparing brain and body mass at inflexion with adult brain and weaning mass

		Brain mass at δ (g)	NBBM at δ (g)	Whole body mass (WBM) at δ	Predicted weaning mass[1]	Difference	% Difference	Adult brain mass (g)[2]	Difference	% Difference
		A	B	C = A+B	D	E = C - D	| E | / (C+D/2)	F	G = A - F	| G | / (A+F/2)
Saimiri	F	20.18	156.68	176.86	353.21	-176.35	0.665	24.04	-3.86	0.175
Saimiri	M	27.93	227.51	255.44	373.11	-117.67	0.374	29.80	-1.87	0.065
Cebus	M&F	59.57	662.22	721.79	674.91	46.88	0.067	67.65	-8.08	0.127
Cercocebus	F	72.95	570.16	643.11	1352.12	-709.01	0.711	107.93	-34.98	0.387
Cercocebus	M	82.99	946.24	1029.23	1568.72	-539.49	0.415	114.68	-31.69	0.321
Macaca	F	72.78	799.83	895.99	893.48	2.51	0.003	85.55	-12.77	0.161
Macaca	M	74.30	736.21	810.51	861.10	-50.59	0.061	94.59	-20.29	0.240
Pan	F	263.03	3863.67	4126.70	5746.01	-1619.31	0.328	370.33	-107.3	0.339
Pan	M	288.86	4017.91	4306.77	5081.08	-774.31	0.165	409.12	-120.26	0.345
				Mean ± sd			0.310 ± 0.260			0.240 ± 0.113
				se			0.086			0.038
				95% confidence interval			0.138 – 0.482			0.164 – 0.316

[1]Calculated as (whole body mass at minimum NBBM)*4, based on Lee *et al.* (1991).
[2]Calculated from the post-inflexion RMA using mean reported adult NBBM as an estimate of adult non-brain body mass. Reported data from the literature given in Appendix 1.
δ = inflexion.

social risks present in the environment, and the demands of the infant for nutrition and support. It would thus appear that, while a metabolic threshold is at least theoretically likely at the level of a species, individual growth and investment decisions may produce different values for [mass] at weaning around that threshold" (Lee, 1999:122). This may account for some of the variation in mass seen in the results presented here.

2] Brain mass at inflexion closely approximates adult brain size such that, when an infant reaches 80% of adult brain size, it is weaned.

The mean percent difference between adult brain mass and weaning brain mass is 24.0 ± 11.3% (Table 1.7). Typically, when an infant comes within 25% of its adult brain mass, it is weaned. *Cercocebus* females show the largest difference in brain size between δ and adulthood (38.7%), but as noted above, they also have small bodies at δ compared, to predicted weaning masses. The differences found in the *Pan* sample are also high, at 33.9% and 34.5% of mean size. However, brain mass at inflexion explains significantly more of the variation in brain mass at adulthood than does brain mass at birth ($F = 31.03$, $df = 7$, $P < 0.01$; Table 1.8). When brain mass at birth is held constant, brain mass at inflexion is still highly significantly associated with brain mass in adulthood ($r = 0.912$, $df = 6$, $P = 0.002$). When brain mass at inflexion was held constant, brain mass at birth and adulthood are no longer significantly correlated ($r = 0.463$, $df = 6$, $P = 0.249$). Brain mass at inflexion is therefore a better predictor of adult brain mass than is neonatal brain mass, and the association of brain mass at inflexion and in adulthood exists independently of the association between neonatal and adult brain mass.

How do the linear allometry samples (*Ateles, Alouatta, Pongo, Gorilla*), in which no clear inflexion exists, fit into this pattern? When whole body mass at which the brain is 80% of adult size is compared with predicted weaning mass, it can be seen that only *Alouatta* conforms to the prediction that body mass at weaning is similar to that at which the brain is 80% of adult size (Table 1.9). The difference between whole body mass when the brain is

TABLE 1.8 Brain mass comparisons in the non-linear samples

		Adult brain mass (g)[1]	Neonatal brain mass (g)[2]	Brain mass at δ (g)
		A	B	C
Saimiri	F	24.04	12.45	20.18
Saimiri	M	29.80	11.99	27.93
Cebus	M&F	67.65	30.69	59.57
Cercocebus	F	107.93	48.35	72.95
Cercocebus	M	114.68	57.44	82.99
Macaca	F	85.55	41.4	72.78
Macaca	M	94.59	41.5	74.30
Pan	F	370.33	148.25	263.03
Pan	M	409.12	148.25	288.86
Correlation between A and C:			$R^2 = 0.998$	$F = 31.03^{**}$ df = 7
Correlation between A and B:			$R^2 = 0.989$	
Partial correlation between A and C, controlling for B:			r = 0.912, df = 6, P = 0.002	
Partial correlation between A and B, controlling for C:			r = 0.463, df = 6, P = 0.249	

[1]Calculated from the post-inflexion RMA using mean reported adult NBBM as an estimate of adult NBBM. Reported data from the literature given in Appendix 1.
[2]Calculated from the pre-inflexion RMA using minimum NBBM as an estimate of neonatal NBBM.

Table 1.9 Brain mass comparisons in the linear samples

		80% of adult brain mass (g)	NBBM when brain is 80% of adult mass (g)	Whole body mass (WBM) when brain is 80% of adult size (g)	Predicted weaning mass[1]	Difference	% Difference
		A	B	C = A+B	D	E = C – D	| E | / (C+D/2)
Ateles	M&F	88.53	2566.27	2654.80	1626.71	1028.09	0.480
Alouatta	M&F	46.46	1592.21	1638.67	1689.88	-51.21	0.031
Pongo	M&F	299.00	14421.15	14720.15	5387.04	9333.11	0.928
Gorilla	M&F	477.19	67297.67	67774.86	8829.89	58944.97	1.539
	Mean ± sd						0.745 ± 0.644
	se						0.322
	95% confidence interval						0.101 – 1.389

[1]Calculated as (whole body mass [i.e. NBBM + brain mass] at minimum NBBM)*4, based on Lee *et al.* (1991).

80% of adult size and predicted weaning mass is 3.1% in *Alouatta* (Table 1.9). The three other linear-allometry taxa have body masses when the brain is 80% adult size that are larger than predicted weaning mass. The largest difference is seen in *Gorilla*: when a gorilla infant's brain is 80% of adult brain size, it has a whole body mass at that is one and a half times bigger (1.539) than predicted by weaning mass. This may contribute to the relatively low level of encephalisation seen in adult gorillas (see above). Whether the scarcity of the data in these plots has biased these results, or whether these taxa are showing a different trend over ontogeny is not clear.

1.4.6 Section summary

The change in postnatal brain growth rate observed earlier in this chapter occurs when the body is within 30% of the body mass at which weaning is predicted to occur. Furthermore, the brain first comes within 20% to 25% of its adult mass at the same body mass. Although variation in both brain and body mass parameters is large, the correlation between brain mass at inflexion and adult brain mass is highly significant, and remains so when neonatal brain mass is controlled for. This suggests that the weaning 'threshold' in primates might be associated with brain, as well as body, growth.

By definition, the data presented in this chapter are cross-sectional. Longitudinal data are needed to track changes in size during ontogeny, as cross-sectional samples tend to obscure growth spurts and other growth events (Bogin, 1999). The Chapter 2 re-examines the trends observed here in the context of longitudinal growth in one primate genus (*Cebus*), and investigates whether individual infants follow the patterns of growth presented here.

CHAPTER 2

BRAIN AND BODY MASS ONTOGENY IN *CEBUS*

Chapter 1 examined cross-sectional brain and body ontogeny across different haplorhine primate taxa at the species level, and found that body mass at the inflexion of the postnatal brain allometry approximated body mass at weaning, and brain size at 75 to 80% of adult size. The data presented in Chapter 1 are by necessity cross-sectional, and may obscure individual variation in ontogeny. This chapter examines whether the observed trends exist during an individual's ontogeny, or whether they are an artefact of a cross-sectional analysis. In order to examine the relationship of brain size and weaning over individual growth trajectories, longitudinal body and brain data were gathered. Data were collected for two species of capuchin monkeys, *Cebus apella* and *Cebus albifrons*. The data originate from a Harvard School of Public Health (HSPH) radiographic study that measured growth over the course of several years. The analysis presented here uses endocranial area (EA) measured in the mid-sagittal plane as a proxy for brain size. Section 1 of this chapter sets *Cebus* growth in context by introducing some themes of capuchin taxonomy and species-specific morphology. Section 2.2 presents the results of the longitudinal brain allometry analyses.

2.1 Introducing the genus *Cebus*

Capuchins form a diverse taxon, and are the most widely distributed of the neotropical primate genera (Nowak, 1999). Their range encompasses the northern Caribbean, Argentina, Peru, Ecuador and the Atlantic coasts of Brazil (Fig 2.1). The range of habitat-type they occupy is also wide (Fragaszy *et al.*, 1990). The success of capuchins in colonising these environments has been attributed in part to the variability of their behavioural and social repertoire, and also to the opportunistic and omnivorous nature of the their diet (Robinson & Janson, 1987).

Figure 2.1. Geographical distribution of, and species-specific facial pelage differences in, the four capuchin species.

Distinguished mainly by their facial pelage (Fig 2.1), the extant capuchin species are traditionally split into two groups:

1] The tufted capuchins, of which *C. apella* is the sole member. This group is named for the presence or absence of two tufts or ridges of black hairs on the crown of the adult *C. apella* pelage, which correspond to underlying temporal ridges on the skull (Napier & Napier, 1967).

2] The non-tufted capuchins, comprising the three smaller allopatric species (*C. albifrons*, the white-fronted or brown pale-fronted capuchin; *C. capucinus*, the white-faced or white-throated capuchin; *C. olivaceus* (= *nigrivittatus*), the wedge-capped or weeper capuchin).

The two most recently-recognised capuchin species, *C. kaapori* and *C. xanthosternos* (Queiroz, 1992; Rylands & Luna, 1993) are usually included within the *olivaceus* and *apella* species respectively (e.g. Wilson & Reeder, 1993). Because of the limited information available on *C. xanthosternos* and *C. kaapor*, they are not discussed further in this thesis.

The species show a moderate differentiation of body size, and species differences are present in the skeleton, especially in the postcranium (Jungers & Fleagle, 1980; Leutenegger & Larson, 1985). Capuchins appear to be either relatively 'robust' or 'gracile' in their limb proportions, and, as might be expected, this tends to correspond to body size (Ford & Hobbs, 1996). Capuchins are moderately sexually-dimorphic (Martin & MacLarnon, 1985; Masterson, 1997), and the level of sexual dimorphism present in each species is also positively correlated with body size (Rosenberger, 1992). Capuchin diets vary with body size, and with geographical location and habitat, but all capuchins consume insects on a regular basis (Janson & Boinksi, 1992). Most place a heavy reliance on energy-rich invertebrates and other protein-rich foods such as pith and palm nuts (Rosenberger, 1992). In fact, capuchins are expert manipulative foragers (Adams-Curtis, 1990), and profligate insect-extractors (Defler, 1979). The use of tools to aid extractive foraging is well-documented (Chevalier-Skolnikoff, 1989; Visalberghi & Fragaszy, 1995; Westergaard & Suomi, 1995; Phillips, 1998), although the cognitive implications of

capuchin tool use are debated (for example, see Chevalier-Skolnikov (1989) and the discussion following that paper). Perhaps the most distinctive aspect of capuchin morphology is the extremely large size of the brain compared to those of other primates of similar body size (Harvey & Clutton-Brock, 1985).

Capuchins show some neurological features that are distinct from both catarrhines and other platyrrhines. In general, the brains of the non-callitrichid platyrrhines show levels of cortical folding (relative to brain size) similar to those of catarrhine brains (Zilles *et al.*, 1989). However, a recent MRI study suggests that capuchins have a lower relative gyrification index than other platyrrhine species, indicating that less cortical folding is present (Rilling & Insel, 1999). This is especially true of the anterior half of the brain, and may be due to reduced growth of the outer cortical layers, or increased growth of the inner layers. Capuchins are also dissimilar to the other neotropical primates in the *patterning* of folding: *Cebus* is the most old-world-monkey-like of the platyrrhines, especially in its occipital sulcal pattern (Falk, 1989). In addition, capuchins are the only platyrrhine to show a well-defined arcuate sulcus that is separate from, and arches around, the caudal end of the rectus sulcus (Falk, 1989). In macaques this area of the frontal cortex receives fibres from the part of the parietal lobe which regulates complex somatosensory responses in the arm and face (Petrides & Panaya, 1984). Capuchins also show distinctive differences in the number of sensorimotor fibres emerging from the brain compared to other haplorhine species: Rilling & Insel (1999) find that *Cebus* have spinal cords that are large for their body size, although compared to brain size, the spinal cord is the size expected. In other words, both the brain and the motor fibres that connect to it are relatively large in capuchins. Rilling & Insel (1999) and Falk (1989) link these features with the extremely well-developed manipulation skills of capuchin monkeys. The corticospinal connections that are directly implicated in manual dexterity have been shown to be at least as dense in capuchins as in rhesus macaques, another highly manipulative species (Bortoff & Strick, 1993). Capuchin neocortices also have very dense neuronal populations; when brain

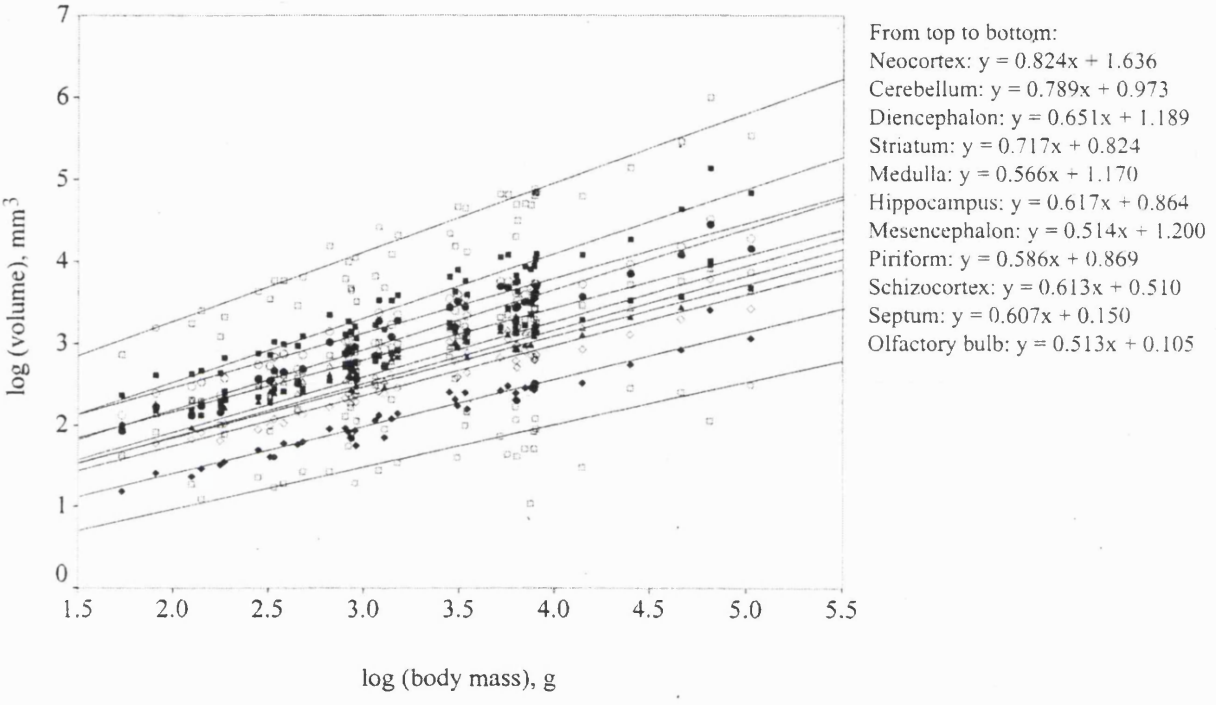


Figure 2.2. Calculating RMA residuals of brain structure size. Lines fitted are RMA residuals, haplorhine data only (n = 28).

volume is accounted for, they are at least as dense as the neocortices of humans (Haug, 1987).

In order to examine whether capuchins are unusual in any other aspect of gross brain morphology, a principal components analysis (PCA) was performed on brain size data from 45 species of primate, representing 12 families, including *Cebus* (Stephan *et al.*, 1981; *Cebus* species not given in the original source). The volume of each brain structure was calculated from stained and sectioned brains as described in Stephan *et al.* (1981). The log-transformed volumes of 11 brain structures were regressed against log-transformed body size, and RMA residuals calculated (RMAs of haplorhine data only; see Fig 2.2).

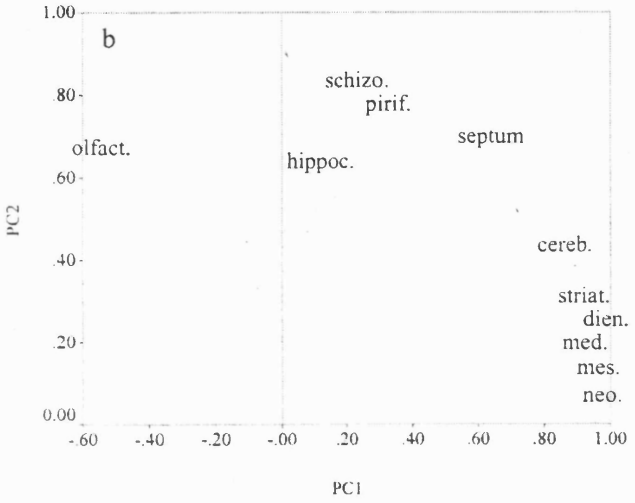
The size-corrected variables entered into the PCA were: neocortex, schizocortex, mesencephalon, diencephalon, cerebellum, hippocampus, medulla oblongata, olfactory bulb, piriform bulb, striatum and septum (Fig 2.3). The principal components were rotated using varimax rotation.

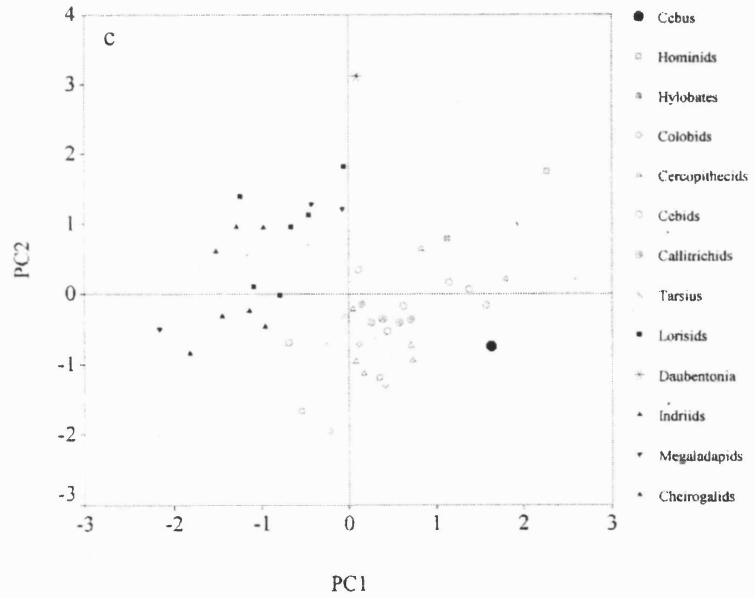
Seven of these 11 variables load strongly and positively (> 0.50) on the first PC extracted, which explains 52.13% of the total variation in the data set (Fig 2.3a). The neocortex shows the strongest loading of these variables (0.964). The olfactory bulb loads strongly and negatively on PC1 (-0.585). The schizocortex, hippocampus and piriform bulb do not load strongly on PC1 (0.195, 0.123 and 0.361 respectively). PC1 therefore distinguishes taxa whose brain structures (with the exception of the olfactory bulb) are relatively large from those whose brain structures (with the exception of the olfactory bulb) are relatively small (Fig 2.3b). This result supports Finlay & Darlington's (1985) hypothesis that variation in brain structure size is, for the most part explained by variation in whole-brain size. Their analysis was based on the same Stephan *et al.* (1981) dataset. The result also supports the observation that the first PC extracted usually reflects general (allometric) size variation (Shea, 1985).

We can predict that PC1 will distinguish relatively species with relatively large brains but small olfactory bulbs from those with relatively small brains but large olfactory bulbs. The residuals used in the analysis were calculated from a regression of the

a

Variable	Component loadings	
	PC1	PC2
Neocortex	0.964	0.078
Schizocortex	0.195	0.829
Mesencephalon	0.946	0.144
Diencephalon	0.943	0.238
Cerebellum	0.779	0.446
Hippocampus	0.123	0.624
Medulla	0.852	0.192
Olfactory bulb	-0.585	0.659
Piriform bulb	0.361	0.773
Striatum	0.887	0.307
Septum	0.612	0.682





n of species points = 45. It should be noted that a high ratio of variables to data points in statistical models artificially inflates the proportion of variation explained. However, similar results were obtained when this analysis was re-run using subsets of variables (data not shown)

Figure 2.3. Brain structure variation in 45 primate species. (a) Variable loadings on the two principal components. (b) Plot of PC1 vs. PC2, showing the variable loadings. (c) Primate families' distribution on PC1 and PC2. Hominids include *Homo*.

haplorhine species, which are large-brained but also tend to have relatively small olfactory bulbs. This is a result of their diurnality and the reliance haplorhines place on visual rather than olfactory foraging and communication (Smuts *et al.*, 1987). PC1 therefore distinguishes the haplorhines from the small-brained and predominantly nocturnal strepsirhines. PC2 explains just under 30% of variation in the dataset (27.16%). The schizocortex loads very strongly on PC2 (0.829); the hippocampus, septum, olfactory and piriform bulbs also load strongly on this PC (0.624, 0.682, 0.659 and 0.773 respectively). The second source of variation described by this PCA therefore arises largely from the relative size of the schizocortex and piriform bulb.

As predicted, the species positioned in the right hand quadrants of the plot (Fig 2.3c) are haplorhines. The strepsirhines fall exclusively in the left quadrants, with the exception of *Daubentonia*, which does not cluster with the other strepsirhines. *Daubentonia* is noted to be unusual in some aspects of its brain morphology (Matano & Ohta, 1999; Sterling & Povinelli, 1999). To the extreme right are the species that have relatively large neocortices (e.g. *Homo*, which is the most extreme of the datapoints along PC1). The capuchin's PC1 score is also high, (1.630) indicating that capuchins have a relatively large neocortex and small olfactory bulb compared to the rest of the brain. Unlike the strepsirhines (and *Aotus*), capuchins place little reliance on olfaction during foraging (Bolen, 1997). These results also support Fragaszy *et al.* 's (1990) observation that, compared to both the body and the brainstem, the cerebellum, neocortex is the most enlarged parts of the capuchin brain. *Cebus* does not score highly on PC2 (-0.750), and falls well within the range of other species on this component. Capuchins do not, therefore, appear to be unusual in any aspect of brain architecture other than the neocortex and olfactory bulb.

In summary, capuchins show an increased association of some of the integrative and visual systems, as well as of the sensorimotor system. These are most probably linked to the unusual propensity of *Cebus* to engage in behaviours that demand high levels of

manipulation and visual acuity, and may be associated both with the reduction in size of the olfactory bulb, and the relatively enlarged neocortex noted in the PCA results.

2.2 Analysis of brain and body ontogeny in capuchin individuals

This section examines data from two capuchin species, *C. apella* and *C. albifrons*. It asks: is the brain/body size relationship observed at the species level (Chapter 1) also seen during the ontogeny of an individual? And how do individuals vary from each other?

The three predictions outlined in Chapter 1 were tested, in order to assess the validity of the brain allometry model (see Chapter 1):

1] The curve describing individual primates' postnatal brain and body growth is linear i.e. represents the post-inflexion phase of the mammalian growth model.

2] The slope of this postnatal, post-inflexion curve falls between 0.1 and 0.2.

3] The postnatal pre-inflexion curve represents an extension of the prenatal brain growth period, and the slope of this pre-inflexion curve equals 1.0.

Weaning data were also integrated with these brain allometry data, and the two weaning hypotheses presented in Chapter 1 also tested:

4] Body mass at weaning will coincide with body mass at δ.

5] Brain mass at inflexion will be between 10% and 20% smaller than adult brain mass.

2.2.1 Materials and methods

The capuchin data presented here originate from a colony housed in the Department of Nutrition at the Harvard School of Public Health (HSPH), Boston, MA, USA (www.hsph.harvard.edu). The HSPH colony, now disbanded, was established in the late 1970s. It included both *C. apella* and *C. albifrons*, and was specifically set up for a radiographic study that investigated the effects of low-calorie and low-protein diets on body growth and skeletal maturation (Samonds & Hegsted, 1973; Thurm *et al.*, 1976;

Samonds & Hegsted, 1978; Ausman & Hegsted, 1980). The data used in this chapter are primarily taken from the dietary control group, although others were included to increase sample size (see below and Chapter 3 for details of other dietary groups). The HSPH project was designed to follow each animal from birth until the later stages of skeletal growth, and x-rays were recorded until the fifth year of life for some individuals.

The majority of work investigating *Cebus* growth and maturation published in the last twenty years is based on the morphometric and radiographic results of this study (Samonds & Hegsted, 1973; Samonds *et al.*, 1974; Fleagle & Samonds, 1975; Thurm *et al.*, 1975; Thurm *et al.*, 1976; Elias, 1977; Samonds & Hegsted, 1978; Wilen & Naftolin, 1978; Ausman & Hegsted, 1980; Ausman *et al.*, 1982; Fleagle & Schaffler, 1982; Ausman *et al.*, 1986; Ausman *et al.*, 1989; Watts, 1990; Accatino & Fleagle, in preparation). For example, some of the body mass data presented here have been previously described by Fleagle & Samonds (1975). Their analysis examines body shape (e.g. stature, chest circumference) in relation to age from birth until adulthood. Jungers & Fleagle (1980) present similar data, describing the results of an allometric investigation of how body proportions (specifically long bone lengths) change with body size. Samonds & Hegsted (1978) and Ausman *et al.* (1986; 1989) use the same body mass data as a control for the dietary-deficiency experiment described above. None of these analyses investigate any aspect of cranial shape or size.

a] Rearing conditions

All animals included in the HSPH experiment were laboratory conceived and delivered. The infants were removed from their dams soon after birth (Thurm *et al.*, 1975) and hand-reared in a lighting- (14h) and temperature- (28^0C) controlled nursery (Ausman *et al.*, 1970). They were fed a commercially prepared human infant formula (Similac, Ross Laboratories, www.ross.com/html/unifiedsite.cfm) for the first 8 weeks of life, after which the infants left the nursery and were allocated to different groups for the dietary deficiency study. The control group (15 males, 9 females) were subsequently fed an *ad libitum* liquid

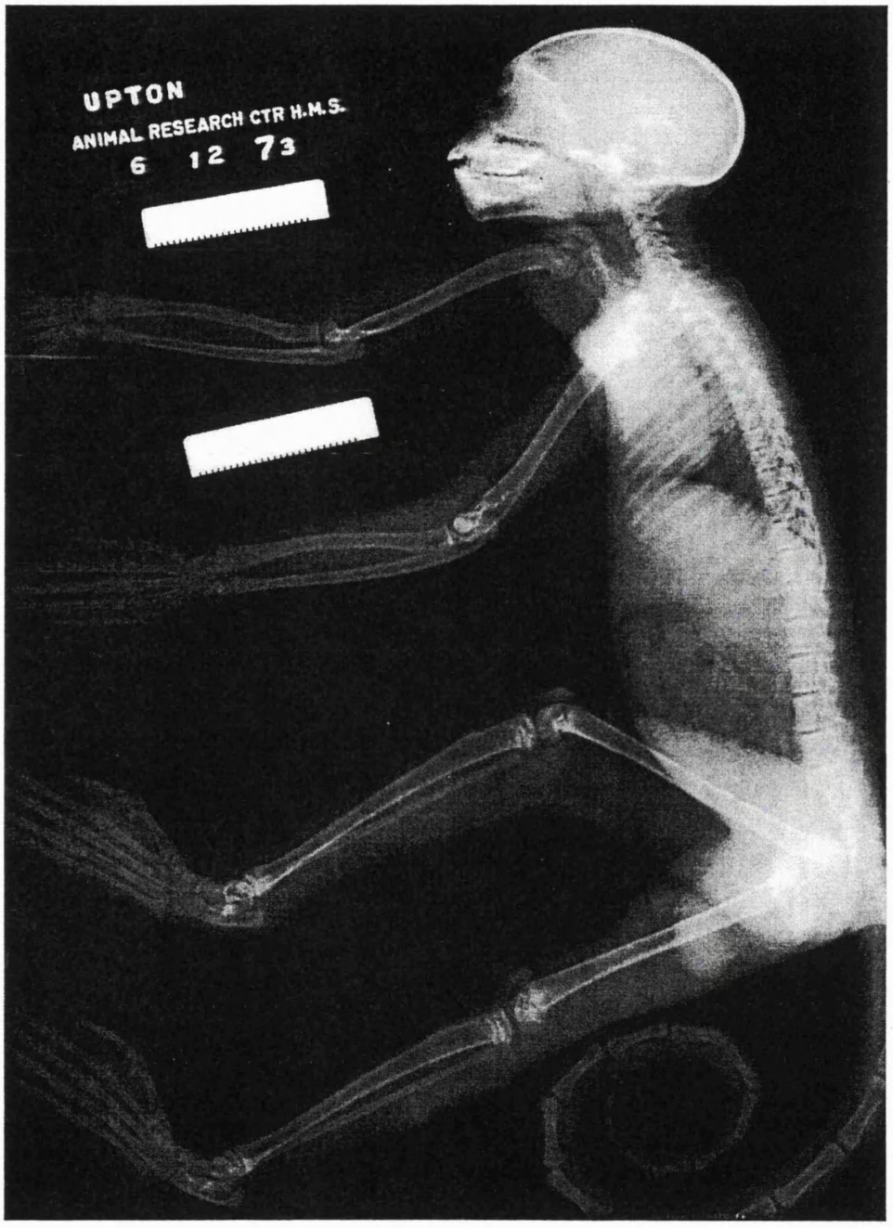


Figure 2.4. Example of the whole-body x-rays taken during the HSPH study. Scales shown are 5mm increments. See text for radiographic technique.

diet that provided 13% of calories as lactalbumin protein. This was approximately double the minimum protein level required for the attainment of the maximum rate of weight gain for animals of this species, age and size (Thurm *et al.*, 1975). More details of this control diet are given in Chapter 3. The feed was available from around 08h00 to 00h00 each day, and was supplied in modified plastic baby bottles in 100ml increments (Samonds & Hegsted, 1973; Samonds & Hegsted, 1978). Water was available on demand. The infants were housed separately in cages measuring 45cm x 45cm x 90cm (Ausman *et al.*, 1970) but allowed daily periods of exercise with the other members of the colony (Thurm *et al.*, 1975). Transferral of the HSPH infants from liquid to solid food was initiated between the ages of 7 and 9 months, when a standard adult agar-based diet was introduced (Corey *et al.*, 1970) and the number of bottles available to the monkeys during the day gradually reduced (Fleagle & Samonds, 1975).

b] Radiographic technique

Each monkey was radiographed over the course of several years, as often as once a week in the early stages of growth (0 to 10 weeks), then every fortnight until the end of the first year, and then every month. The animals were lightly anaesthetised and placed in a standardised position on the x-ray table (Fig 2.4; see Thurm *et al.*, 1975 for details of anaesthetic and x-ray model). This involved laying the monkey on its right side with right limbs extended and right hands and feet taped to the x-ray table. The left limbs were flexed slightly and taped to a 25mm-thick cardboard spacer. Calibrated brass bars were included in each x-ray at the level of both left and right limbs, along with the animal's name and the date of the examination. Tube to target distance was kept constant at 1.00m (Fleagle & Samonds, 1975). The resulting radiographic plates measured either 35cm x 40cm for the smaller monkeys, or 46cm x 64cm for the larger, older individuals. The error in positioning the animals was estimated by repositioning and x-raying one animal five consecutive times (Fleagle & Samonds, 1975). The results of this error estimation are not reported in Fleagle & Samonds (1975). Body mass and other morphometric variables were recorded at each x-

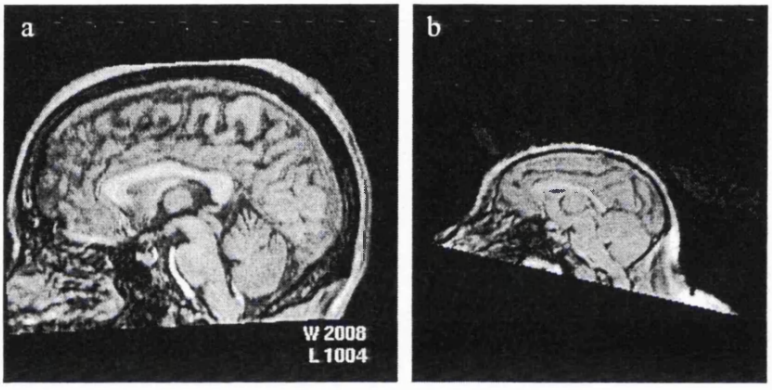


Figure 2.5. Examples of the mid-sagittal MRI images used in the comparison of endocranial area (EA) and brain volume. (a) *Homo;* (b) *Saimiri.*

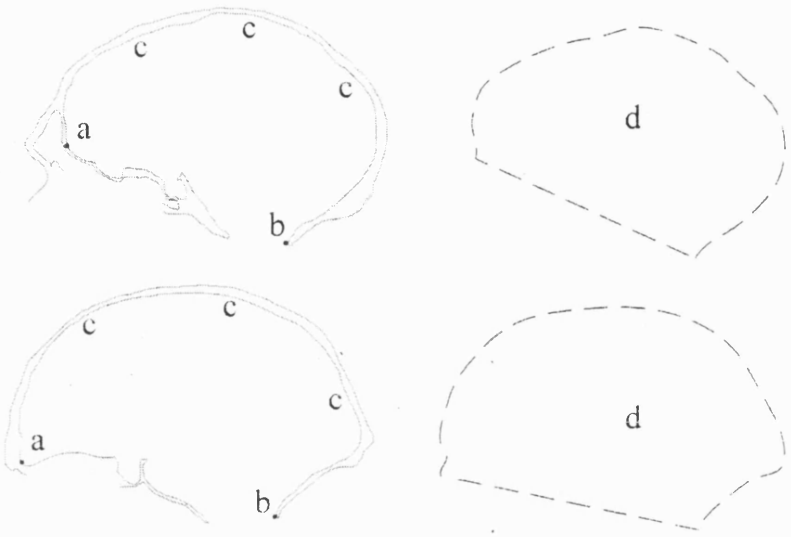


Figure 2.6. Landmarks used in the analysis of endocranial area (EA). (top) *Homo;* (bottom) *Saimiri.* Anterior is left, posterior is right. a = internal angle of the frontal bone; b = internal margin of the foramen magnum at opisthion; c = internal margin of the endocranial vault; d = endocranial area. Not shown to scale.

Table 2.1 Estimating measurement error in NIH Image 6.2

Shape	Area (mm^2)	Estimated area (mm^2)	Difference	% difference
	A	B	C = A-B	| C | / (A+B/2)
1	100	106.15	6.15	0.060
2	400	402.35	2.35	0.006
3	900	901.39	1.39	0.002
4	1600	1620.48	20.48	0.013
5	2500	2554.53	54.53	0.022
6	3500	3464.52	35.48	0.010
7	4900	4967.81	67.81	0.014
8	5600	5571.25	28.75	0.005
9	6000	5996.08	3.92	0.001
10	7500	7404.63	95.37	0.013
11	9000	8878.38	121.62	0.014
12	10000	10041.74	41.74	0.004
Mean ± sd				0.013 ± 0.016
se				0.005
95% confidence interval				0.003 – 0.023

ray examination. Intraobserver error (consistency of technique) for a suite of morphometric parameters including body mass measurement was estimated by the HSPH workers by measuring one monkey five times in one session. The largest coefficient of variation (CV) for the five sets of measurements was 1.49%; between observer differences were not estimated (Fleagle & Samonds, 1975).

c] MRI comparison of EA and whole-brain volume

Although brain mass itself cannot be ascertained from the x-rays, a very good approximation can be made by measuring the internal area of the cranial vault (endocranial area, EA). Using magnetic resonance images (MRIs) of primate crania (Rilling & Insel, 1999), it was possible to compare EA with brain volume *in vivo*. Forty-four adults or older subadults from 11 primate taxa (*Cebus*, *Cercopithecus*, *Gorilla*, *Homo*, *Hylobates*, *Macaca*, *Pan*, *Papio*, *Pongo*, *Saimiri*) were scanned to include the entire brain (see Rilling & Insel, 1999 for details of scanning techniques and sample composition). Whole brain volume was calculated, and error estimated, as described in Insel & Rilling (1999).

The MRI images were imported into an image analysis program (NIH Image 6.2; http://rsb.info.nih.gov/nih-image) on a PowerMacintosh (http://www.apple.com) by the author. The mid-sagittal MRI image of each individual was used where possible (Fig 2.5). In 18% of cases (n = 8) the most superior or posterior part of the cranial vault was missing from the MRI; the curve of the vault was inferred in these images. The program was calibrated and the measurement scale set. The Density Slice tool was used to highlight the contrast between bone and other tissues; the lower and upper bounds of the density selection were set to 230 and 250 respectively, at the upper (black) end of the look-up table (LUT) scale. This ensured that areas of both thick and thin bone were highlighted. To measure EA, only those endocranial landmarks which could be found in both MRI scans and x-rays were used (Fig 2.6). NIH Image's Freehand Selection tool was used to trace the internal margin of the endocranial vault, labelled C in Figure 2.6. The endocranium was traced from the internal angle of the frontal bone (where the frontal squama meets the

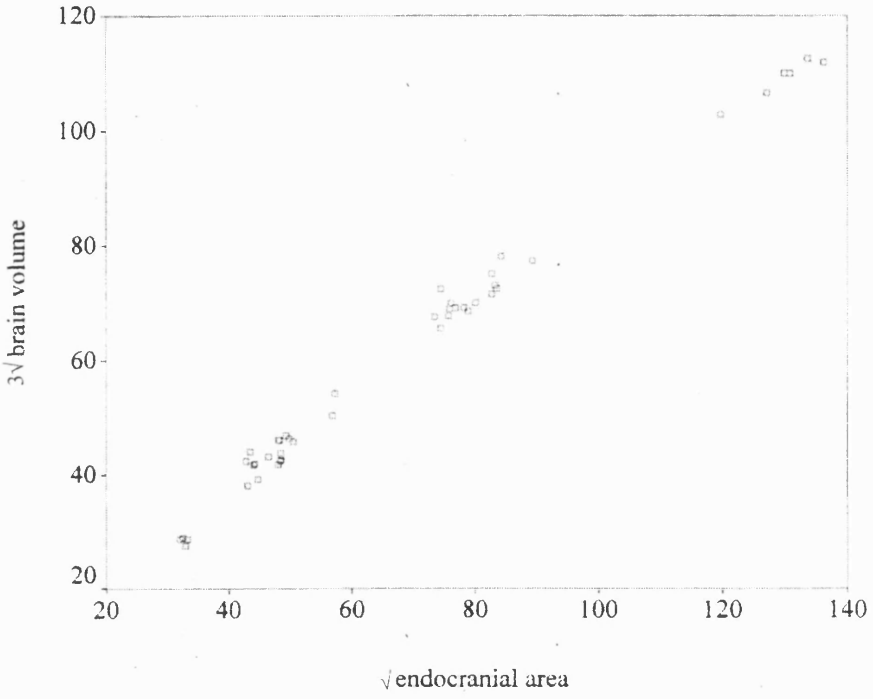


Figure 2.7. Endocranial area (EA) is a good predictor of brain volume. Both EA and brain volume measured from mid-sagittal MRI images (Rilling & Insel, 1999). 44 individuals represent 11 haplorhine species. $R^2 = 0.991$.

bones of the orbital roof, excluding any frontal sinuses; labelled A in Fig 2.6) to the internal margin of the foramen magnum at opisthion (labelled B in Fig 2.6). Endocranial area (labelled D in Fig 2.6) rather than external area was used as some of the species included in the analysis show extremely thick cranial vault bones (e.g. *Papio*, *Pan*).

The Freehand Selection tool automatically joins the endpoint of the selection to its origin with a straight line. This was preferred to tracing the internal features of the basicranium as many of these landmarks are obscured in the x-rays (by other bony features of the skull base such as the petrous temporal) and are difficult to locate accurately. The Measure tool was then used to quantify the selected area. The internal error of NIH 6.2's Measure tool was estimated by repeat analysis of various known-area polygons imported into NIH Image 6.2 (Table 2.1). The mean percentage difference between the known and estimated areas was 1.3 ± 1.6%. Observer error was estimated by repeat analysis of each MRI image; if the difference between the two values was greater than 2%, the measurement was repeated until the error fell below 2%.

Just under 100% of the total variation in brain volume is explained by variation in EA ($R^2 = 0.991$, n = 44; Fig 2.7). The relationship between the two variables is less than isometry when all 44 individuals are included in the regression (RMA slope for all species = 0.818; 95% confidence interval 0.806 – 0.830). With humans and apes excluded the 95% confidence interval for the slope includes isometry (RMA slope for all species except the hominids = 1.013; 95% confidence interval = 0.953 to 1.073, n = 18). An increase in EA is accompanied by a proportional increase in brain volume. Whichever taxonomic grouping is used, the proportion of variation explained is consistently high, despite small sample sizes (lowest $R^2 = 0.944$ for all species except the hominids, n = 18; highest $R^2 = 0.999$ for platyrrhines and humans only, n = 14). EA is therefore an excellent predictor of brain volume in primates, and can be used with confidence as a proxy for brain size. Martin (1990) found a similar result in his interspecific analysis of primate cranial capacity and brain mass. The exponent relating the two in that analysis (major axis slope = 1.018, n = 33) is similar to the slope describing EA and brain volume found here.

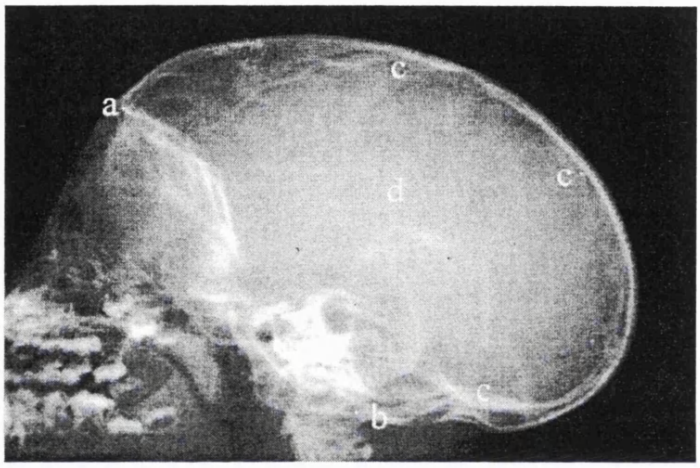


Figure 2.8. An example of the x-rays used in the EA analysis. See Fig 2.6 for explanation of landmarks.

d] Measuring EA from the HSPH radiographs

The HSPH x-rays were digitised (UMAX Mirage II High Resolution A3 scanner, G3 PowerMacintosh (http://www.umax.com, http//www.apple.com), using Adobe Photoshop 5.5 and UMAX MagicScan 4.3 software (http//:www.adobe.com; http://www.umax.com) at a resolution of 150dpi. Each file was archived on CD as a grayscale TIFF file (i.e. in tagged image file format, with LZW compression). NIH Image 6.2 cannot import LZW-compressed files, so each file was also saved as an uncompressed TIFF file. To minimise file size, each of these uncompressed images was cropped to include only the head and neck (Fig 2.8).

EA was measured in the same way as described in section c] above. In addition to the use of the Density Threshold tool, the contrast and brightness of the images were adjusted where appropriate. When an x-ray was not exactly in the mid-sagittal plane, the midpoint between left and right landmarks (such as the line of the superior orbital plates) was used. *Cebus* cranial bones are relatively thin, especially in infancy, and it proved easy to trace the inner tables of the bones from the x-rays. Magnification factors appropriate for different age groups were calculated from the calibration bars placed at the level of the spacer and the table (see Appendix 2). As in the MRI analysis above, each x-ray was measured at least twice to ensure the difference between each measurement was less than 2%.

EA was used as a proxy for brain size. Non-brain body mass was not calculated. Adult body mass was inferred from growth curves i.e. whole body mass over age. Body mass at weaning was calculated as neonatal body mass*4 (Lee *et al.*, 1991), using the HSPH sex- and species-specific mean neonatal birth masses described by Jungers & Fleagle (1980).

e] Statistical techniques

The statistical methods used in this chapter are described in detail in Chapter 1, and were performed in exactly the same way. Linearity was tested by a comparison of the

coefficients of determination (R^2) of linear and quadratic regressions. Loess was used to estimate the position of the inflexion, δ, and to divide the data into pre- and post-inflexion subsets. RMAs for datapoints before and after the inflexion were then calculated and compared, and solved as simultaneous equations to find the position of δ. The 'lengths' of the RMAs between birth and δ, and between δ and adulthood were calculated, as was residual encephalisation; all methods are described in Chapter 1.

2.2.2 Assumptions

Several assumptions have been made in this analysis which should be borne in mind during the following discussion.

1] The MRI analysis, which established the high correlation of EA to brain volume, is based on adult or older subadult individuals. It is likely that brain shape as well as size changes over ontogeny, and this may affect the relationship of mid-sagittal area to whole-brain volume. A decrease in cranial breadth over age has been reported for *C. capucinus* (Schultz, 1960) and *C. apella* (Corner & Richtsmeier, 1991), but there is little change in the width of the anterior cranium in the latter species, and the magnitude of size change is greater than that of shape change.

2] Males and female capuchins may show a different relationship of cranial width to cranial length and height as adults (Masterson & Hartwig, 1998). In the Masterson & Hartwig study, male crania are absolutely bigger than female crania in all three dimensions, but to differing degrees i.e. the shape of the male skull is different to that of the female skull. The disproportion of male skull dimensions is only in the order of 1 or 2%, and is not considered further here.

3] EA is a two-dimensional measure, and it was not possible to infer non-brain mass from the data (as in Chapter 1). This chapter assumes that the trends observed in Chapter 1 will be comparable with trends observed in an analysis of EA over whole body mass.

2.2.3 Results

a] Data collection

A total of 1038 radiographs were measured for this part of the analysis (Fig 2.9). The majority (72.3%) of the measurements come from the control diet group described above, but because individuals over 365 days postpartum (dpp) were scanned less frequently than those under one year old, there are relatively few x-rays representing ages older than one year in this group. EAs of 6 infants (4 males, 2 females) which were assigned at a younger age (8 to 28 weeks old) to the non-control calorie-deficient group are therefore included. Only data from ages older than 12 months were collected from the non-control animals in this analysis (Fig 2.9a). These infants are described in more detail in the next chapter. As Chapter 3 shows, by the end of 12 months postpartum there are no significant differences in EA or body mass between the control and non-control animals. More data were available for males than females (Fig 2.9b) and for more *C. albifrons* than *C. apella* (Fig 2.9c). This was due to the composition of the experimental groups. Although body masses were recorded and x-rays taken until six years of age in a few individuals, 36 months postpartum was the cut-off age at which sample sizes were maximised. Adult body size is not, therefore, represented in the brain allometries.

b] Statistical analyses: testing the three allometry predictions

1] Is the curve describing individual capuchins' postnatal brain allometry linear?

The data samples of eighteen individuals contained sufficient data to construct brain allometries (Table 2.2). No differences were observed between the species or the sexes in the coefficient of determination (R^2) for either the linear or the quadratic regression (data not shown). The level of variation explained by both regressions is high, with an average R^2 of 0.880 ± 0.071 and 0.950 ± 0.025 for the linear and quadratic regressions respectively.

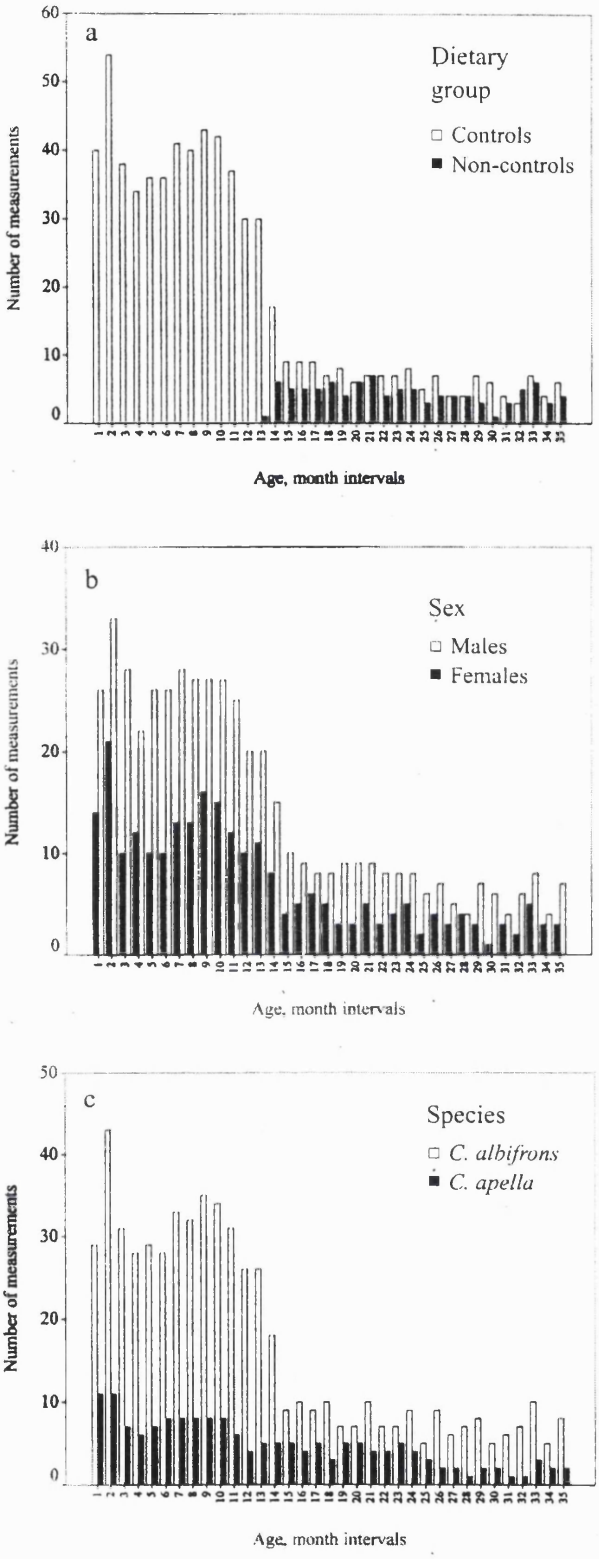


Figure 2.9. Composition of the EA dataset. (a) Dietary group; (b) sex; (c) species. Data are divided into month intervals.

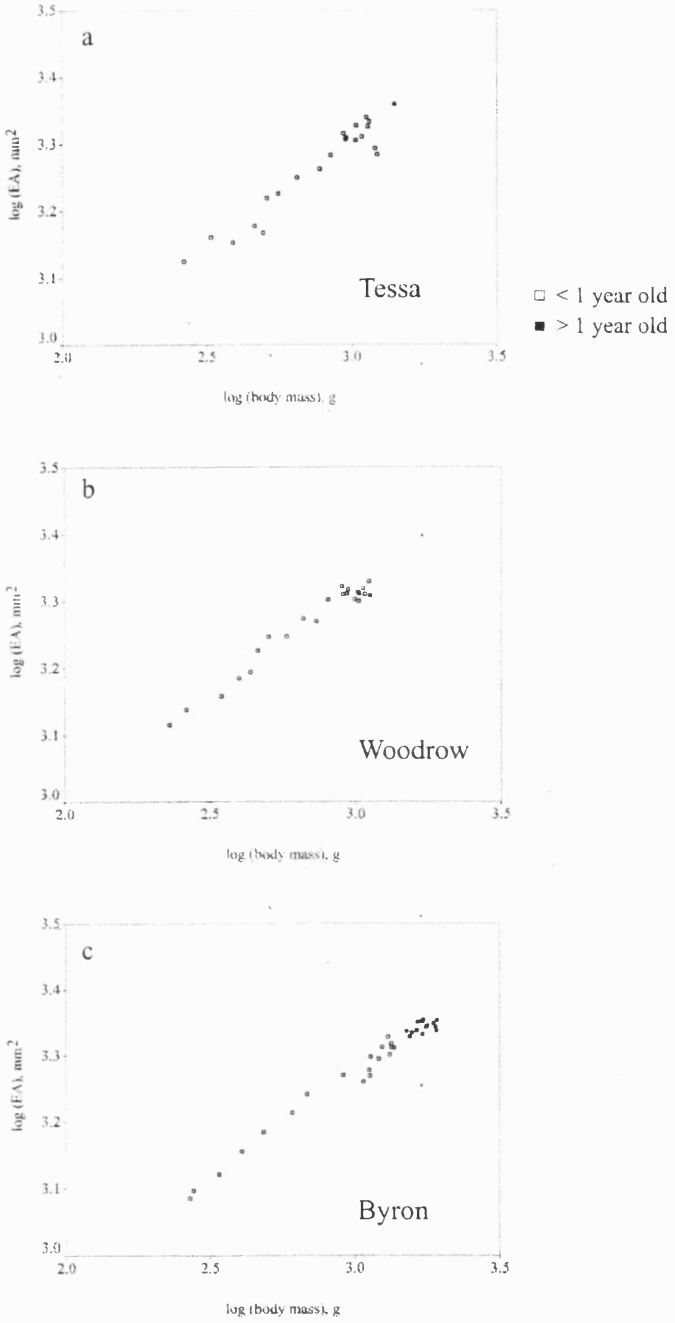


Figure 2.10. Brain allometries in the linear samples. (a) Female *C. albifrons* (Tessa); (b) male *C. albifrons* (Woodrow); (c) male *C. apella* (Byron).

Table 2.2 Testing the brain allometries for linearity

Name	Sex	n	R_1^2	R_2^2	df	F
			C. apella			
Xelysa	F	31	0.851	0.939	28	40.26***
Joshua	M	41	0.819	0.925	38	53.73***
Byron	M	33	0.980	0.983	30	4.83
Hamilton	M	38	0.871	0.950	35	54.94***
			C. albifrons			
Hecuba	F	17	0.746	0.902	15	22.48**
Roberta	F	35	0.861	0.916	32	20.69***
Sybil	F	20	0.902	0.956	17	21.53**
Tessa	F	22	0.925	0.925	19	0.00
Vixen	F	18	0.921	0.956	15	11.85**
Gordon	M	15	0.818	0.913	12	13.12**
Groove	M	27	0.939	0.988	24	98.62***
Kosta	M	25	0.924	0.958	22	18.00**
Mcgovern	M	20	0.713	0.958	18	111.99***
Olaf	M	24	0.905	0.973	21	53.32***
Quibble	M	26	0.873	0.941	23	26.51***
Ruka	M	22	0.952	0.967	19	6.83*
Uriah	M	34	0.879	0.973	31	107.47***
Woodrow	M	23	0.962	0.970	20	4.92
Mean ± sd			0.880 ± 0.071	0.950 ± 0.025		
se			0.017	0.006		
95% confidence interval			0.846 – 0.914	0.938 – 0.962		

Of the 18 individuals tested, three capuchins have brain allometries that are statistically indistinguishable from linearity. These individuals are one male *C. apella* (Byron), and one male and one female *C. albifrons* (Woodrow, Tessa; Fig 2.10). However, when the data samples for these capuchins are examined, it can be seen that two contain only one datapoint from ages above 365dpp (closed datapoints; Figs 2.10a, b). As will be seen below, it is only after one year of age that the brain finishes the majority of its growth, and these samples are therefore not representative of the complete brain allometry. The third sample that shows linearity (Byron, Fig 2.10c) includes data from over one year old, but these cluster closely to each other and indicate a period of growth in which body mass failed to increase significantly. Byron and Tessa are excluded from the following analyses; Woodrow is discussed separately.

The 15 other infants included in the analysis show allometric curves in which significantly more variation in EA and body size is explained by a quadratic, rather then linear, regression. Their brain allometries are therefore non-linear. As in Chapter 1, in the majority of samples included in the analysis postnatal growth does not conform to the predicted model.

2] Does the slope of the postnatal post-inflexion curve equal 0.2?

All the individuals for which RMAs were calculated are described in Table 2.3. No species or sexual differences were found in either the slopes or the intercepts of the pre- and post-inflexion RMAs (data not shown). The brain allometries shown in Figure 2.11 are those of four individuals (Joshua, Xelysa, Uriah, Roberta) which represent typical brain and body growth for a male and female *C. apella* and a male and female *C. albifrons* (in the case of Xelysa, the only female of the species included). As before, datapoints from ages older then 365 dpp are shown as closed symbols. Each capuchin shows a brain allometry that is similar to those observed in the cross-sectional analyses i.e. with a period of relatively

Table 2.3 Pre- and post-inflexion RMAs of the non-linear samples

Name	Sex	Pre-inflexion RMA slope ± s.e.	Pre-inflexion RMA 95% confidence interval for slope	Pre-inflexion RMA intercept	Post-inflexion RMA slope ± s.e.	Post-inflexion RMA 95% confidence interval for slope	Post-inflexion RMA intercept
				C. apella			
Xelysa	F	0.351 ± 0.024	0.303 – 0.399	2.285	0.166 ± 0.045	0.076 – 0.256	2.786
Joshua	M	0.368 ± 0.027	0.314 – 0.422	2.227	0.095 ± 0.020	0.055 – 0.135	3.015
Hamilton	M	0.386 ± 0.032	0.322 – 0.450	2.201	0.141 ± 0.022	0.097 – 0.185	2.893
				C. albifrons			
Hecuba	F	0.319 ± 0.032	0.255 – 0.383	2.383	0.098 ± 0.031	0.036 – 0.160	3.002
Roberta	F	0.381 ± 0.038	0.305 – 0.457	2.187	0.162 ± 0.023	0.116 – 0.208	2.779
Sybil	F	0.351 ± 0.037	0.277 – 0.425	2.313	0.126 ± 0.027	0.072 – 0.180	2.934
Vixen	F	0.418 ± 0.049	0.320 – 0.516	2.148	0.124 ± 0.026	0.072 – 0.176	2.951
Gordon	M	0.302 ± 0.046	0.210 – 0.394	2.465	0.136 ± 0.041	0.054 – 0.218	2.925
Groove	M	0.414 ± 0.015	0.384 – 0.444	2.124	0.163 ± 0.015	0.133 – 0.193	2.812
Kosta	M	0.339 ± 0.032	0.275 – 0.403	2.292	0.163 ± 0.022	0.199 – 0.207	2.784
Mcgovern	M	0.422 ± 0.038	0.346 – 0.498	2.111	0.173 ± 0.039	0.095 – 0.251	2.784
Olaf	M	0.330 ± 0.020	0.290 – 0.370	2.346	0.183 ± 0.050	0.083 – 0.283	2.743
Quibble	M	0.274 ± 0.021	0.232 – 0.316	2.491	0.089 ± 0.039	0.011 – 0.167	3.019
Ruka	M	0.424 ± 0.026	0.362 – 0.476	2.065	0.210 ± 0.017	0.005 – 0.123	2.659
Uriah	M	0.424 ± 0.023	0.378 – 0.470	2.117	0.112 ± 0.018	0.076 – 0.148	2.966
Mean ± sd		0.367 ± 0.048			0.143 ± 0.035		
se		0.012			0.009		
95% confidence interval		0.343 – 0.391			0.125 – 0.161		

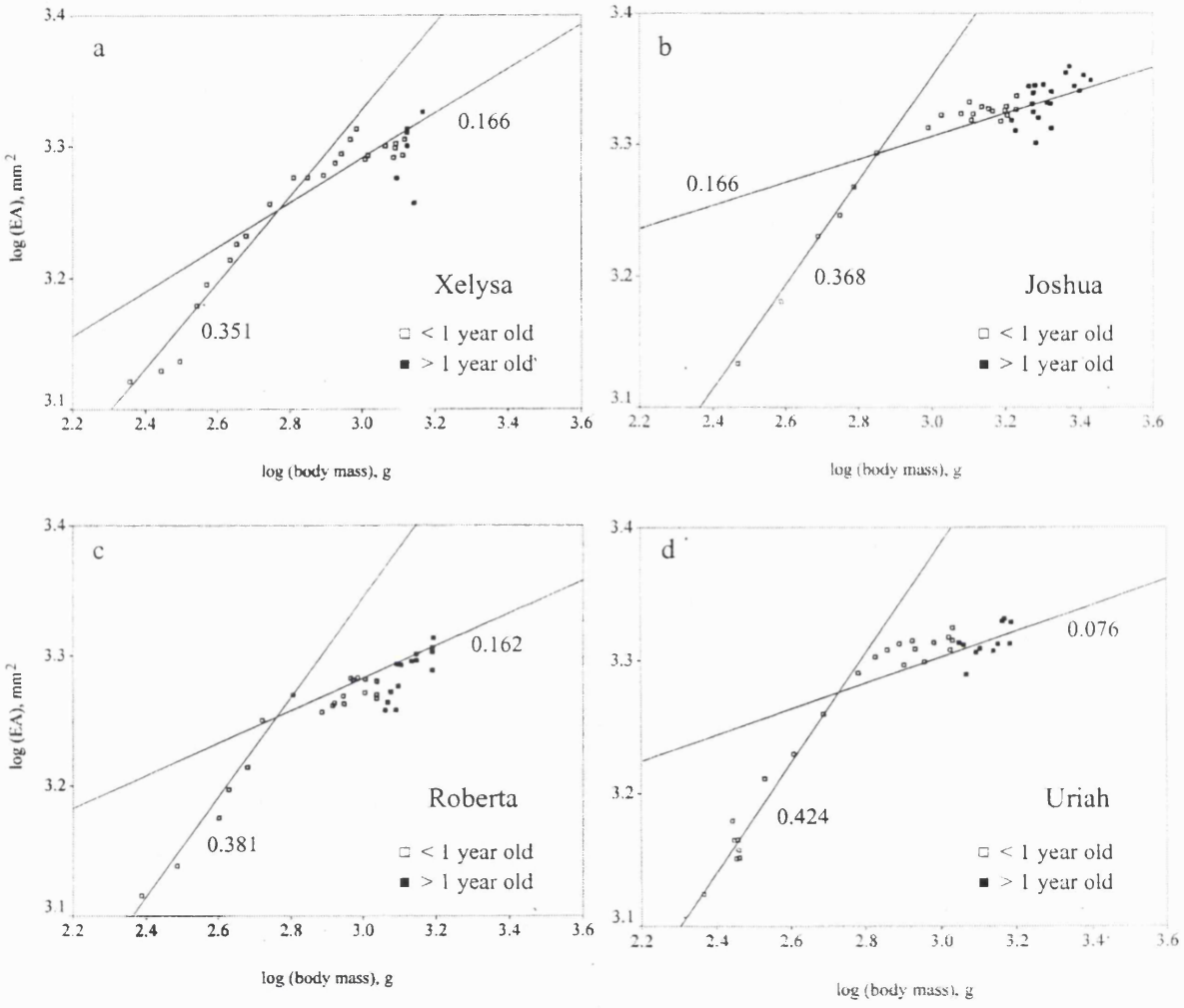


Figure 2.11. Non-linear brain allometries in four capuchins. (a) Female *C. apella* (Xelysa); (b) female *C. albifrons* (Roberta); (c) male *C. apella* (Joshua); (d) male *C. albifrons* (Uriah). Lines are pre- and post-inflexion RMAs (slope values shown). Details of RMAs given in Table 2.3.

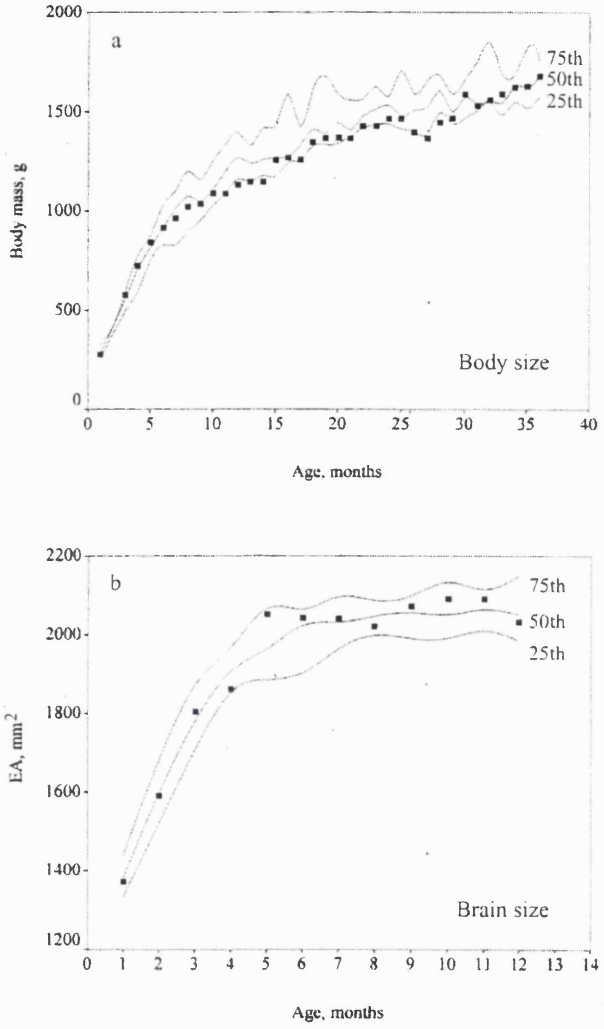


Figure 2.12. Brain and body growth in Woodrow, compared to growth percentiles. (a) Body mass; (b) EA. Percentiles are calculated from all individuals included in Table 2.2, with the exception of Woodrow. 25th, 50th and 75th percentile shown.

rapid brain growth in relation to body growth, and then a period when body size increases to a greater extent than brain size.

The mean post-inflexion slope falls between 0.1 and 0.2 (0.143 ± 0.009), but the 95% confidence interval includes neither 0.1 nor 0.2 (0.125 – 0.161, Table 2.3). However, all except one of the individuals shown in Table 2.3 (Groove) has post-inflexion slope confidence intervals that include either 0.1, or 0.2, or both. Groove's post-inflexion slope falls between 0.1 and 0.2, but neither 0.1 nor 0.2 are included in the confidence interval (slope = 0.163 ± 0.015). This part of the postnatal growth period does, therefore, conform to the model's prediction for this individual. In Chapter 1, the capuchin sample's post-inflexion slope was similar, i.e. 0.1 was included in the cross-sample mean confidence interval, and 0.1 and/or 0.2 were included in the majority of individual confidence intervals.

3] Does the postnatal pre-inflexion curve represent an extension of the rapid prenatal brain growth period, i.e. does the slope of the pre-inflexion curve equal 1.0?

The pre-inflexion slopes of the longitudinal allometries are all lower than 1.0, just as in the species-level analysis presented in Chapter 1. The mean pre-inflexion slope across all individuals included in Table 2.3 is 0.367 ± 0.048, and 1.0 is not included in the 95% confidence interval (0.343 – 0.391). This period of growth does not therefore represent an extension of the prenatal growth phase. Immediately after birth, capuchin brain growth proceeds at a rate that is rapid compared to the predicted model of mammalian growth, but not as rapid as before birth. The mean pre-inflexion slope value is significantly lower than was found in the analysis of Chapter 1 (0.367 ± 0.048 vs. 0.524 ± 0.159). This ties in with the observation that allometric slopes tend to decrease with decreasing taxonomic level (Pagel & Harvey, 1989).

The linear allometry of Woodrow (see above) has a slope of 0.292 ± 0.013. The 95% confidence interval does not include 0.1, 0.2 or 1.0 (0.266 – 0.318). As with the linear

samples in Chapter 1, Woodrow's allometry approximates that of the mammalian model, but with a slightly elevated slope value. Why does Woodrow not show an inflexion in his postnatal ontogeny? Compared to other infants in the population, Woodrow's body size is slightly smaller than average during the first three years of life, falling below the 50th body mass percentile in each month interval (Fig 2.12a). Woodrow's EA, on the other hand, falls above the 50th percentile for the majority of the first year of life (data for Woodrow's EA only available for the first year postpartum, Fig 2.12b). Only at the very end of the three-year period does Woodrow's body size fall on the 50th percentile (Fig 2.12a). However, Woodrow does not fall outside the 'normal' range of variation for either body or brain size, and it is unclear whether his unusual allometry is related to this postnatal body growth faltering.

c] Modelling growth and encephalisation

Because Woodrow's brain allometry has a slope above that predicted by mammalian growth, by adulthood Woodrow is more encephalised than predicted by the model (i.e. has positive residual encephalisation). In fact, he is the most encephalised of all the infants, having a brain that is 26% larger than predicted (Table 2.4). Of the fifteen capuchins shown in Table 2.4, 11 have larger than predicted brains (positive residual encephalisation) and 4 have smaller than predicted brains (negative residual encephalisation). The average absolute percentage difference between observed and predicted values of brain size is 7.6 ± 1.6%. Examples of differences in residual encephalisation are illustrated in Figure 2.13, which includes the same four individuals shown in Figure 2.11.

Both sex and species differences are observed in these four capuchins. Adult residual encephalisation is influenced by the amount of body growth undergone after the inflexion, δ. The two females (Figs 2.13a and b) are more encephalised than predicted by the model (have positive residual encephalisation) because their bodies stop growing before the allometric line has 'crossed' the predicted line. In the two males, body growth ceases after or just as the two lines cross (marked C in Figs 2.13c and d). Residual

Table 2.4 Predicting adult brain mass

		Adult whole body mass (WBM) (g)[1]	Adult EA (mm^2)[2]	Predicting adult brain mass from minimum whole body mass (WBM)			
				Predicted RMA[3] i.e. slope = 0.2	Predicted adult EA (mm^2)	Difference	% Difference
		A	B		C	D = B-C	| D | / (B+C/2)
Xelysa	F	2187.76	2189.97	y = 0.2x + 2.641	2037.04	152.93	0.072
Joshua	M	5248.07	2335.61	y = 0.2x + 2.641	2428.85	-93.24	0.039
Hamilton	M	5623.41	2640.89	y = 0.2x + 2.645	2483.13	157.76	0.062
Hecuba	F	3235.94	2218.09	y = 0.2x + 2.694	2488.86	-270.77	0.115
Roberta	F	1862.09	2035.82	y = 0.2x +2.620	1879.32	156.5	0.080
Sybil	F	2041.74	2244.19	y = 0.2x +2.657	2084.49	159.7	0.074
Vixen	F	1995.26	2291.92	y = 0.2x +2.688	2228.43	63.49	0.028
Gordon	M	4466.84	2638.76	y = 0.2x +2.739	2944.42	-305.66	0.109
Groove	M	4570.88	2562.01	y = 0.2x +2.632	2312.06	249.95	0.103
Kosta	M	4168.69	2366.35	y = 0.2x +2.628	2249.05	117.3	0.051
Mcgovern	M	3388.44	2481.36	y = 0.2x +2.658	2312.06	169.3	0.071
Olaf	M	4168.69	2571.94	y = 0.2x +2.663	2437.81	134.13	0.054
Quibble	M	3801.89	2501.15	y = 0.2x +2.683	2506.12	-4.97	0.002
Uriah	M	3388.44	2298.05	y = 0.2x + 2.647	2254.24	43.81	0.019
Woodrow	M	3981.07	2686.58	y = 0.2x +2.595	2065.38	621.2	0.261
			Mean ± sd				0.076 ± 0.061
			se				0.016
			95% confidence interval				0.044 – 0.108

[1]Adult body mass derived from individual growth curves of body mass over age. A growth curve for Ruka was not available, and he is not included in this table.

[2]Calculated from the post-inflexion RMA or whole-sample RMA allometry as appropriate, using adult body mass (column A).

[3]Represents the postnatal brain allometry as predicted by the model discussed in the text. Slopes inferred as 0.2. Intercept calculated from i = m – 0.2k, where i = intercept of predicted RMA, k = minimum whole body mass and m = neonatal brain mass (i.e. inferred from minimum WBM using the appropriate pre-inflexion or whole-sample RMA (Table 2.3).

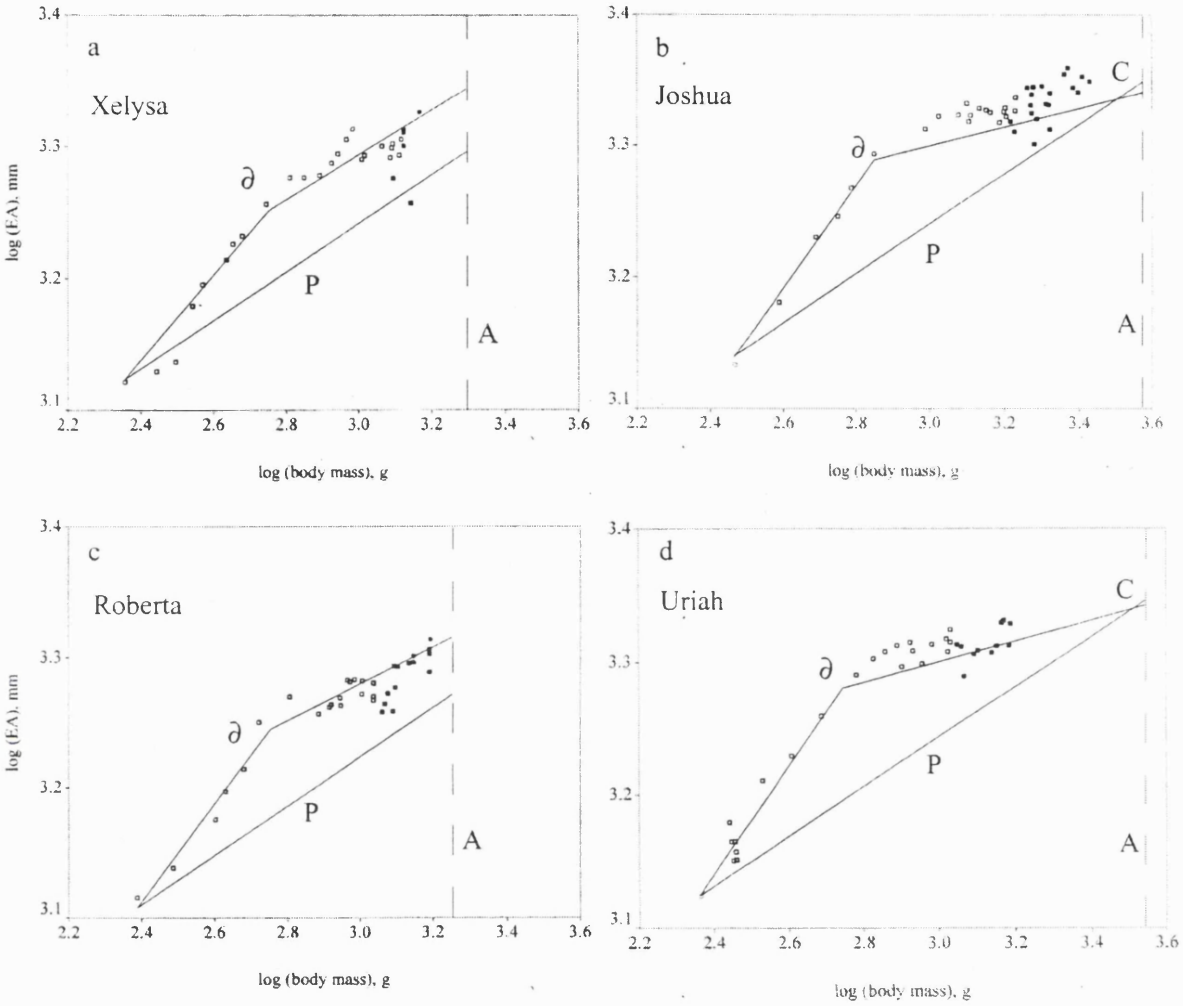


Figure 2.13. Residual encephalisation in four capuchins. (a) Female *C. apella* (Xelysa); (b) female *C. albifrons* (Roberta); (c) male *C. apella* (Joshua); (d) male *C. albifrons* (Uriah). Lines marked with ∂ are observed pre- and post-inflexion RMAs (Table 2.3). ∂ = inflexion of allometry. Lines marked P are predicted RMAs that intersect with the pre-inflexion RMAs at the youngest datapoint. The predicted RMAs impose a slope of 0.2 on the data. The intercepts of these predicted RMAs were calculated by substituting 0.2 for the appropriate slope value for each sample, and then re-arranging the terms of the equation (Table 2.4). Lines marked A represent adult body mass (Table 2.4). C = point at which observed post-inflexion RMA crosses the predicted RMA.

encephalisation is therefore smaller than in the two females. These four examples suggest patterns of growth are different in the two sexes. the 'length' of the post-inflexion slope is significantly shorter in females, i.e. male brain and body growth continues further along the allometry axes than female brain and body growth ($t = 5.651$, $df = 12$, $P = 0.000$; Fig 2.14a). However, no significant sexual differences were observed in the slopes of the pre- and post-inflexion RMAs, or in relative encephalisation at δ or adulthood (data not shown). Furthermore, a chi-square analysis of the distribution of the sexes into positive and negative residual categories is not significant ($\chi^2 = 0.171$, $df = 1$, $P>0.05$).

C. apella have significantly longer pre-inflexion slopes than *C. albifrons* ($t = 2.876$, $df = 12$, $P = 0.014$; Fig 2.14b). This indicates that *C. apella* are extending the pre-inflexion phase of growth into larger body sizes compared to *C. albifrons*. However, the difference between the species' mean relative encephalisation at δ is not significant, nor is it significant at adulthood (data not shown). This contrasts with the results of Chapter 1, in which the pre-inflexion slope, not length, correlated with species differences in adult encephalisation.

These results are summarised in Figure 2.14c. The predicted postnatal allometry (based on the model presented in Chapter 1) is denoted as P. All allometries converge at neonatal mass, marked B. The slopes of the pre-inflexion RMAs are indistinguishable between the species, but species differences are apparent in the length of the pre-inflexion RMA. This results in significant species dimorphism in body size at δ ($t = 3.940$, $df = 12$, $P = 0.002$), but not brain size (data not shown). The non-significant difference in species EA at δ is probably due to the measurement error associated with it (*C. apella* mean EA = 1874.27 ± 132.05 mm^2; *C. albifrons*: 1836.89 ± 106.14mm^2). Sexual differences are apparent in the post-inflexion part of the curve, when males tend to extend their allometries further along the growth trajectory.

The slopes and lengths of the pre-inflexion RMA can, to some extent, be predicted from the size of the body at birth (Fig 2.15). The size of the neonate influences the slope and the length independently, i.e. both relationships remain significant in a partial

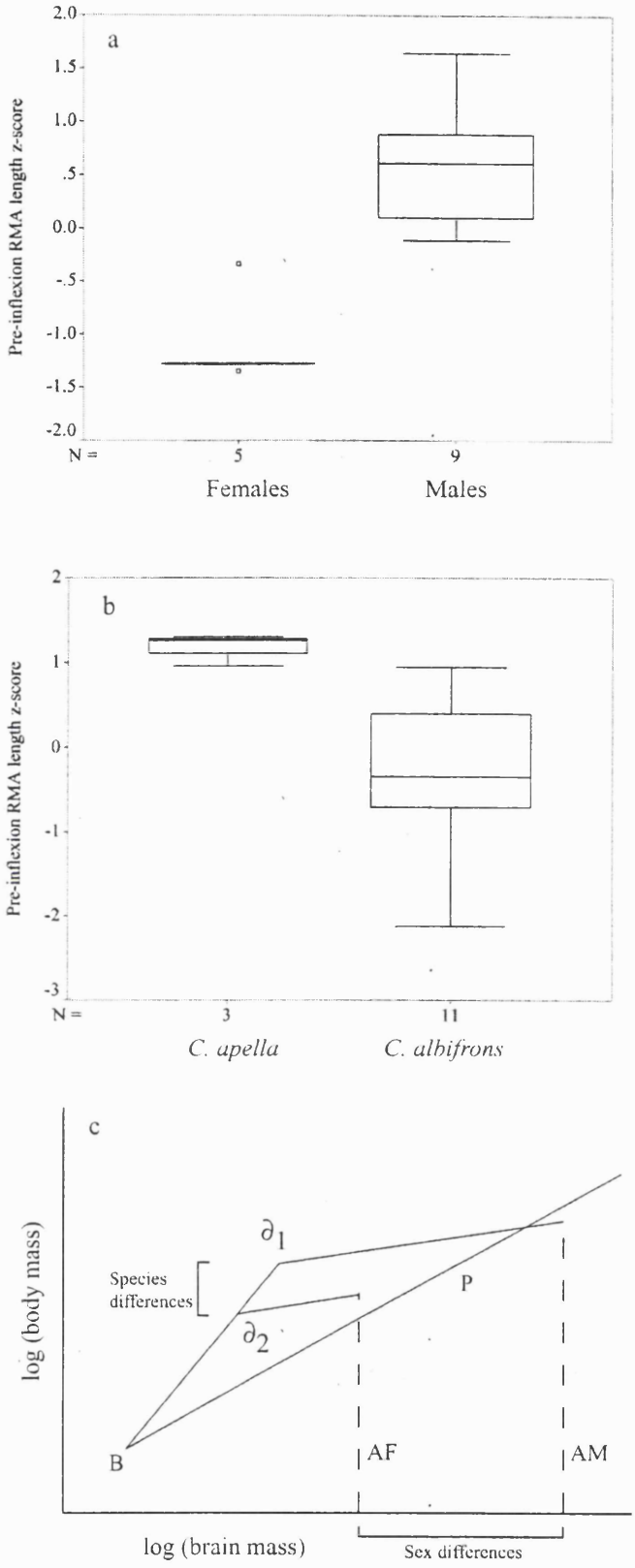


Figure 2.14. Pre- and post-inflexion RMA length associations. (a) Post-inflexion RMA length by sex. Individual datapoints are outliers. (b) Pre-inflexion RMA length by species. (c) Modelling the associations of pre- and post-inflexion RMA length. ∂_1 = *C. apella* inflexion; ∂_2 = *C. apella* inflexion; P = predicted RMA; B = birth; AF = adult body mass in females; AM = adult body mass in males.

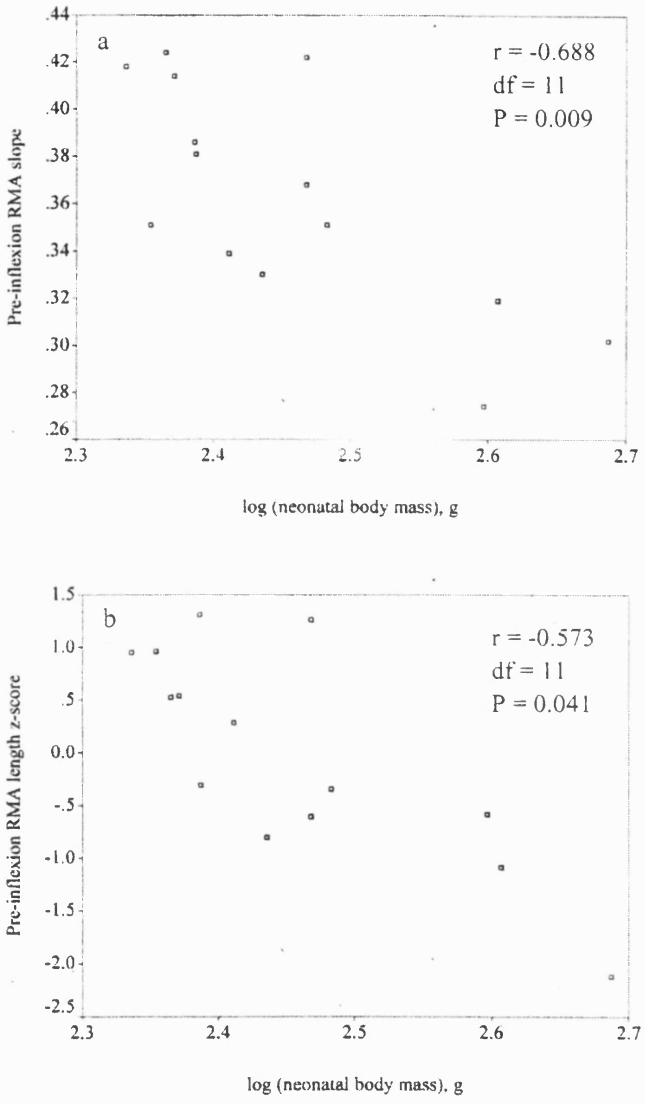


Figure 2.15. Neonatal body mass predicts the length and slope of the pre-inflexion RMA. (a) Pre-inflexion RMA length; (b) Pre-inflexion RMA slope. Partial correlation coefficients are shown in each plot.

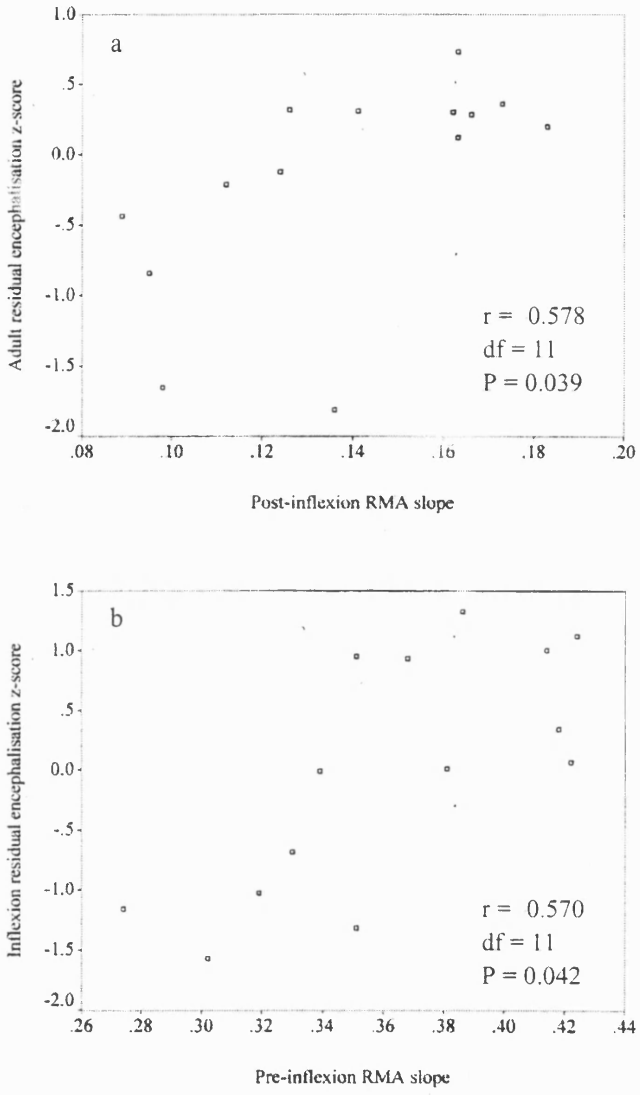


Figure 2.16. The RMA slopes predict residual encephalisation. (a) Adult residual encephalisation; (b) residual encephalisation at the inflexion. Partial correlation coefficients are shown in each plot.

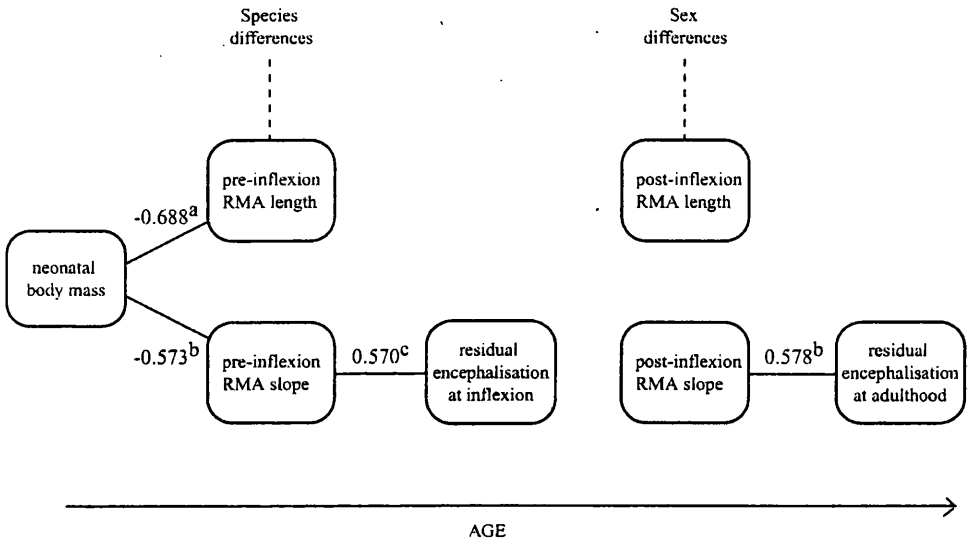


Figure 2.17. Modelling the interactions of pre- and post-inflexion RMA slopes and lengths, sex, species, and residual encephalisation. Values are partial correlation coefficients from Figs 2.15 and 2.16. a = Pre-inflexion RMA slope held constant. b = Pre-inflexion RMA length held constant. c = Post-inflexion RMA length held constant. Neonatal body mass is negatively correlated with both pre-inflexion RMA length and slope. Pre-inflexion RMA slope is positively associated with residual encephalisation at inflexion. Residual encephalisation in adulthood is significantly correlated with post-inflexion RMA slope.

correlation (Fig 2.15a and b). The larger the infant at birth, the lower the pre-inflexion slope, and the shorter its length i.e. in smaller neonates the allometry is extended further along the axes, and the rate at which the brain increases in mass relative to the body is greater. This indicates that brain size is targeted, and that catch-up growth of the brain (relative to body size) occurs in small infants. No body mass variables are significantly correlated with the post-inflexion slope, which suggests that this parameter varies independently of the size of the infant over ontogeny.

Adult residual encephalisation is significantly correlated with the both the slope and length of the post-inflexion RMA (data not shown). When these three variables are entered into a partial correlation, only the association of adult residual encephalisation and post-inflexion RMA slope remains significant (Fig 2.16a). The association of adult residual encephalisation and post-inflexion RMA length is explained by the latter's association with the post-inflexion RMA slope. Similarly, the correlation of inflexion residual encephalisation and pre-inflexion RMA length (data not shown) is explained by the latter's correlation with the pre-inflexion RMA slope (Fig 2.16b). The rate at which brain size increases in the post-inflexion part of the curve is not directly influenced by growth prior to the inflexion (data not shown).

In summary, neonatal body mass is negatively associated with both the length and slope of the pre-inflexion RMA (Fig 2.17). Species differences also influence the length of the pre-inflexion RMA. A high pre-inflexion RMA slope value is associated with positive inflexion residual encephalisation. The length of the post-inflexion RMA is influenced by sex differences. A high post-inflexion slope value is associated with positive adult residual encephalisation. In Chapter 1, we saw that species differences in adult residual encephalisation were associated with differences in the slope of the pre-inflexion curve. Pre-inflexion differences in RMA length were seen in the species in the present chapter (Fig 2.14), but adult residual encephalisation itself was not significantly different between the capuchin species (data not shown). As discussed above, this is probably because some measurement error is associated with EA. In the present chapter, however, adult residual

Table 2.5 Comparing brain and body mass at inflexion with adult brain and weaning mass

		EA at δ (mm^2)	Whole body mass (WBM) at δ (g)	Weaning mass (g)[1]	Difference	Difference	Adult EA (mm^2)[2]	Difference	Difference
		A	B	C	D = B – C	| D | / (B+C/2)	E	F = A – E	| F | / (A+E/2)
Xelysa	F	1721.87	779.00	856.00	-77.00	0.094	2189.97	468.10	0.239
Joshua	M	1946.31	769.90	972.80	-202.90	0.233	2335.61	389.30	0.182
Hamilton	M	1954.63	667.56	972.80	-305.24	0.372	2640.89	686.26	0.299
Hecuba	F	1890.25	632.27	903.60	-271.33	0.353	2218.09	327.84	0.160
Roberta	F	1647.57	504.89	903.60	-398.71	0.566	2035.82	388.25	0.211
Sybil	F	1913.20	575.44	903.60	-328.16	0.444	2244.19	330.99	0.159
Vixen	F	1948.41	538.63	903.60	-364.97	0.506	2291.92	343.51	0.162
Gordon	M	2003.83	590.32	990.40	-400.08	0.506	2638.76	634.93	0.274
Groove	M	1814.63	550.85	990.40	-439.55	0.570	2562.01	747.38	0.342
Kosta	M	1736.44	624.39	990.40	-366.01	0.453	2366.35	629.91	0.307
Mcgovern	M	1785.11	504.44	990.40	-485.96	0.650	2481.36	696.25	0.326
Olaf	M	1725.97	501.97	990.40	-488.43	0.655	2571.94	845.97	0.394
Quibble	M	1875.04	714.59	990.40	-275.81	0.324	2501.15	626.11	0.286
Uriah	M	1865.39	526.20	990.40	-464.20	0.612	2298.05	432.66	0.208
					Mean	0.453			0.254
					± sd	± 0.164			± 0.076
					se	0.044			0.020
					95% confidence interval	0.365 – 0.541			0.214 – 0.294

[1]Inferred from neonatal body mass *4, based on Lee *et al.* (1991). Sex- and species-specific HSPH neonatal body mass data taken from Jungers & Fleagle (1980).

[2]Calculated from the post-inflexion RMA using adult whole body mass (Table 2.4).

δ = inflexion of allometry.

encephalisation *is* associated with post-inflexion differences in growth, regardless of species (Fig 2.16a). At the intraspecific level, therefore, it appears that individual variation in post-inflexion brain and body growth (perhaps sex-related) is a more important determinant of adult residual encephalisation than is species variation.

d] Weaning and brain allometries

1] Does mass at inflexion typically fall within 25% of weaning mass?

Table 2.5 describes how inflexion mass relates to weaning brain and body size. It can be seen that the mean difference between body mass at inflexion and body mass at weaning is larger than was found in Chapter 1 (0.453% ± 0.164). The average difference between the two parameters is 45.3% of mean mass at δ and weaning. Body mass at inflexion underestimates weaning mass in each individual, which suggests that, at the inter-individual level, the change in the rate of brain growth occurs prior to the achievement of weaning mass. Weaning mass is calculated here as neonatal mass*4; this is based on Lee et al.'s (1991) interspecific observation that weaning occurs at four times birth mass. Their calculation of weaning mass is an estimate of size at the end of the weaning process, and it is likely that the inflexion occurs when these capuchin infants are beginning the weaning process. As Bowman & Lee (1985) show, there is considerable variation in the weaning:birth mass ratio at both the inter- and intra-specific level (see Chapter 3), and it is possible that neonatal mass*4 overestimates weaning mass in the HSPH capuchins. Unfortunately, individual weaning status is not known for these infants.

2] Does brain mass at inflexion approximate adult brain size such that, when an infant reaches 80% of adult brain size, it is weaned?

The average difference between EA at the inflexion and adulthood (25.4% ± 2.0) is comparable with that observed in Chapter 1 (25.4 ± 2.0%). The relationship between the size of the brain at these two ages is therefore similar whether data are analysed at the inter- or intraspecific level. The level of variation among the HSPH infants in this value is small (2.0%), suggesting that brain growth is more tightly constrained than is body growth. The 95% confidence interval of the difference does not include 0.20 (i.e. brain mass at δ is more than 20% smaller than adult brain mass) and the estimate of a brain size threshold should be revised downwards to 75%. In the HSPH capuchins, when an infant reaches 75% of adult brain size, it has begun the weaning process.

2.3 Summary

In summary, although variation between individual capuchins exists, for example between the sexes and species, all show similar patterns of brain and body growth. The inflexion of the brain/body allometry observed in Chapter 1 is also present in the longitudinal allometries, and the pre- and post-inflexion slopes are of similar value. Body mass at inflexion does not show as tight an association with weaning mass as was predicted. It is probable that the weaning:birth mass ratio of 4 used to predict weaning mass is less appropriate at the intra-specific level, as Lee & Bowman (1995) also found. However, the observation that weaning occurs at about the body size at which approximately 80% of adult brain size is first achieved holds true in the HSPH infants. The results of this and the previous chapter suggest that brain growth is associated with the process of weaning, and may in fact show a closer association than body mass and weaning. It is possible, however, that brain growth and weaning are linked by virtue of the fact that body growth and weaning are linked – in other words, that the trends observed in Chapters 1 and 2 are

artefacts of the close association of brain and body size during ontogeny. The next chapter goes on to test this artefact hypothesis with a larger set of data collected from the HSPH infants.

CHAPTER 3

BRAIN AND BODY GROWTH UNDER NUTRITIONAL STRESS: TESTING THE ARTEFACT HYPOTHESIS

We have seen that brain and body size increase in step with each other throughout lactation. As body mass approaches weaning size, the rapid rate of brain growth slows as body growth continues. These trends, and the values of the slopes that describe the different phases of growth, are consistent at the inter- and intra-specific level. Body size at weaning shows more variation than brain size at weaning, but it is possible that brain size and weaning are linked because body size and weaning are linked. This chapter asks: is the observation that weaning occurs when brain growth stops simply an artefact of the association of brain and body growth? If so, we should observe no disjunction in brain and body growth over lactation, even in the presence of growth-disrupting factors such as severe nutritional stress. This 'artefact' hypothesis is tested by integrating the body and brain size data presented in Chapter 2 with other data from the HSPH capuchin colony (Section 3.1). As described previously, these data are part of an investigation into the effects of under-and malnutrition on the skeleton. Section 3.2 examines size differences between various dietary-deficient and control infants, whilst Section 3.3 investigates mass- or size-specific growth rate differences. Section 3.4 discusses the implications these results have for the artefact hypothesis and the constraints brain growth places on strategies of lactation and weaning.

3.1 Materials and methods

The brain size (EA) and body mass data described in Chapter 2 were integrated with data from other *C. apella* and *C. albifrons* housed in the HPSH colony. These other infants were raised in exactly the same conditions as the control group (see Chapter 2 for details) with one important exception: at 8 weeks old they were separated into three groups, and each

Table 3.1 Dietary regimes followed by the HSPH capuchins

Dietary regime		n of animals (m:f)	% of calories as protein[1]	Access	Kcal per day per animal
Control	CONT	28 (18:10)	13.0	Free	Unlimited
Calorie-restricted	CAL	7 (5:2)	13.0	Limited	90
Protein-restricted	PROT	7 (4:3)	2.8	Free	Unlimited
Protein & calorie-restricted	PC	4 (2:2)	6.5	Limited	90

[1]21% of calories from fat (corn oil), 28% as sucrose; dry ingredients: sat (4.7%), vitamins (0.5%), inositol (0.1%), choline chlorides (0.28%), cellulose, (4.6%), solubilisers, stabilisers, and flavourings. Diluted with water to a concentration of 1 kcal/ml (Samonds & Hegsted, 1978).

fed a diet that was lacking in either calories, or in protein, or both (Table 3.1). The control (CONT) diet provided approximately twice the protein (expressed as % of caloric intake) necessary to promote growth (i.e. body mass increase). The protein-restricted diet (PROT) approximately half the protein needed for growth. The calorie-restricted diet (CAL) provided 90 kcals/day. The diet of the protein- and calorie-restricted group (PC) provided sufficient protein to permit growth, but restricted calorie intake to 90 kcals/day (Thurm *et al.*, 1975). Estimates of normal protein and calorie requirements were based on the results of a study of other HSPH adult capuchins (Ausman & Hegsted, 1980). These experimental dietary regimes continued for 20 weeks. All infants (including the controls) were transferred to a standard adult agar-based diet between the ages of 7 and 9 months (Corey *et al.*, 1970).

Body mass and endocranial area (EA) data were gathered as described in Chapter 2. In addition to body mass and EA, specific growth rates (g/d/g body mass, or $mm^2/d/mm^2$ EA) were calculated according to the formula:

$$\text{Specific rate} = ([S_{i+1} - S_i] / [t_{i+1} - t_i]) / S_{i+1} \qquad (3.1)$$

S_i and S_{i+1} represent size at successive examinations and t_i and t_{i+1} are the dates of the examinations, in days postpartum. The usual interval between examinations was 7 days in the first ten weeks of life and 14 days in the remainder of the first year. Rates were not calculated for examination intervals greater than 28 days. These measures of growth velocity are used in preference to absolute growth rates. Bigger animals tend to grow faster than smaller ones, and any significant differences that remain after size has been controlled for will reflect differences in rate, rather than differences in both size and rate (Leigh, 1992).

In order to assess the interspecific, sex and diet group differences in brain and body size over the first year of life, the data were divided into month intervals. Data were then compared across each age interval (independent sample t- tests, significance when $P \leq$

0.05, equal variances not assumed when F is significant). In the following figures, months in which the differences are significant are indicated by asterisks (*) unless otherwise specified. If significant sexual dimorphism was present in at least one age interval (i.e. when the difference between the sex averages was significant), the sexes were analysed separately within that dietary group. For example, sexual dimorphism in the control (CONT) group reaches significance only at the age of 7 months, but the sexes are analysed separately over all age intervals.

In addition to the t-test analysis, brain allometries were constructed and analysed as described in the previous two chapters. EA was used as a proxy for brain size. Non-brain body mass was not calculated. Linearity was tested by comparing the coefficients of determination (R^2) of linear and quadratic regressions of the data. Loess was used to estimate the position of the inflexion, δ, and to divide the data into pre- and post-inflexion subsets. RMAs for datapoints before and after the inflexion were then calculated and compared, and solved as simultaneous equations to find the position of δ. Body mass at weaning was calculated as neonatal body mass*4 (Lee *et al.*, 1991), using the HSPH sex- and species-specific mean neonatal birth masses described by Jungers & Fleagle (1980).

3.2 Results: I. Body mass

3.2.1 Sample size and composition

The numbers of individuals and datapoints included in the analysis are given in Table 3.2. Only *C. albifrons* were included in the protein- and calorie-deficient (PC) group in the original experiment. The datapoints were evenly distributed over the twelve month-long intervals (see figures following).

Table 3.2 Sample sizes and composition

	C. apella			*C. albifrons*		
	Males	Females	Total	Males	Females	Total
1] Individuals						
CONT	3	1	4	15	8	23
CAL	1	0	1	4	2	6
PROT	1	0	1	3	3	6
PC	0	0	0	2	2	4
2] Body mass datapoints						
CONT	152	31	183	494	233	727
CAL	28	0	28	175	101	276
PROT	31	0	31	105	116	221
PC	0	0	0	41	40	81
3] EA datapoints						
CONT	71	27	98	267	145	412
CAL	19	0	19	91	51	142
PROT	19	0	19	48	62	110
PC	0	0	0	33	20	53

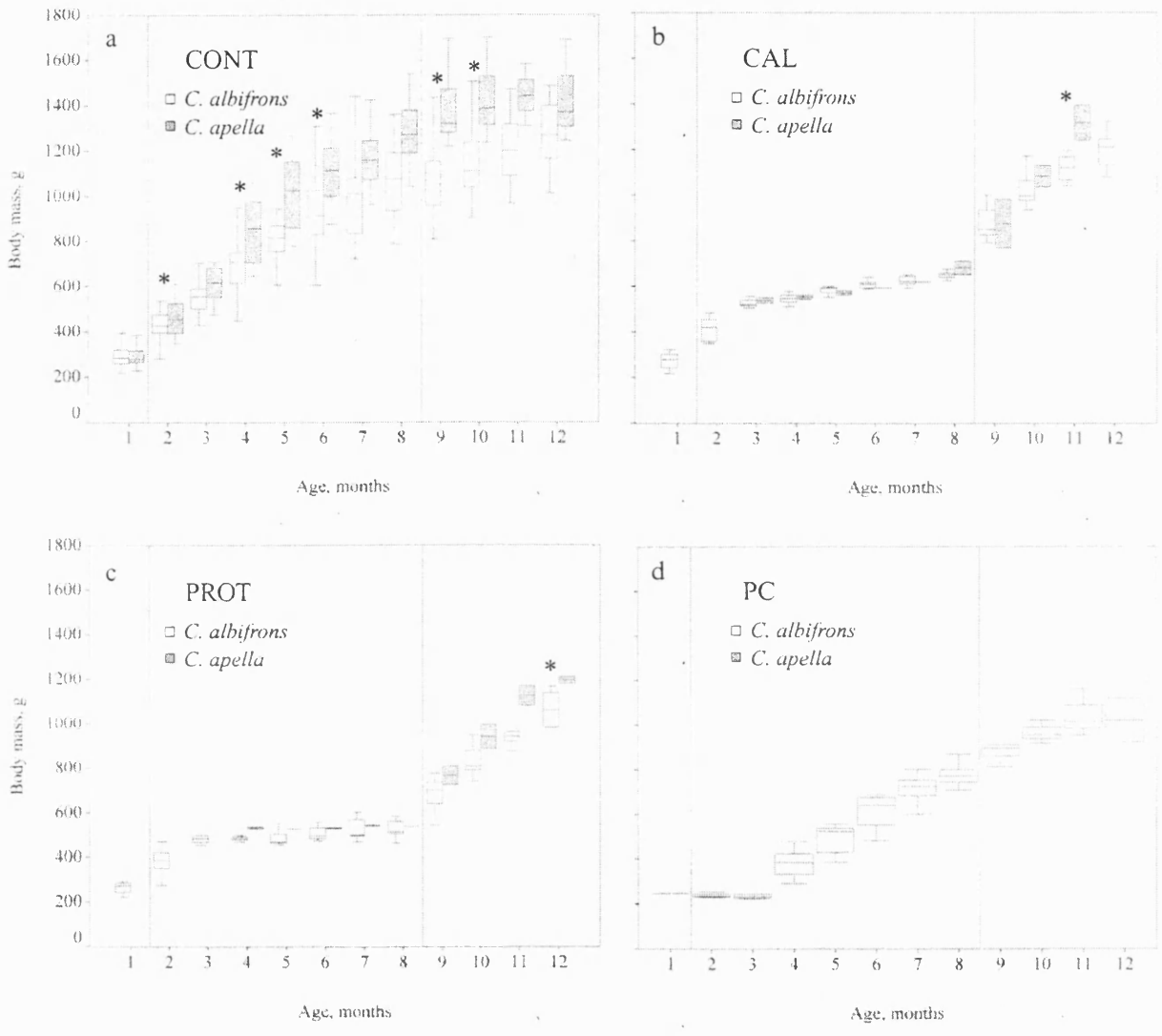


Figure 3.1. Body growth in the dietary groups: species dimorphism. (a) Controls (CONT); (b) calorie-deficient (CAL) infants; (c) protein-deficient (PROT) infants; (d) protein- and calorie-deficient (PC) infants. Vertical lines indicate the beginning and end of the experimental period. Asterisks indicate age intervals in which the differences between the species' means is significant (student's t-test, $P < 0.05$).

3.2.2 Species dimorphism and general trends

1] Normal body growth is rapid until 9 months postpartum.

Increase in control (CONT) body mass is rapid in the first part of the year, but slows after the end of the experiment (Fig 3.1a). This flattening of the body growth curve is more noticeable in the larger *C. apella*, where mean mass remains unchanged between months 9 and 12.

2] CONT C. apella *bodies are significantly heavier than* C. albifrons *from 2 months postpartum.*

Body size differences between the species are apparent from an early age. *C. apella* are significantly heavier than *C. albifrons* in the second month of life, and then intermittently throughout the first year (Fig 3.1a).

3] Body growth faltering is immediate and severe in all non-CONT groups.

CONT body size represents the 'target' body mass against which non-CONT body masses can be compared at each age interval. Severe growth faltering occurs in all the non-CONT groups from the beginning of the experiment (Figs 3.1b, c and d). The calorie-restricted (CAL) and protein-restricted (PROT) groups of both species appear to show similar responses to nutritional stress, and there are no significant differences in the species-average body masses in any of the experimental months. Thurm *et al.* (1976) find that skeletal maturation is also compromised in nutritionally-deprived capuchin infants. Their study analyses the same x-rays as the described here, to investigate ossification and epiphyseal formation in the hands and feet under restricted diets. The late-forming ossifications (e.g. sesamoids and tuberosities) are most severely affected, and the malnourished infants show an increase in bone abnormalities and scarring. Thurm *et al.* (1976) see this as evidence of preferential allocation of nutrients to the non-skeletal elements of the body, or of delayed cartilage formation.

4] CAL infants are able to increase body mass during the experiment, but PROT infants are not.

Infants in the CAL group show a slight increase in body mass over the 20-week low-calorie diet, but by 8 months old are still only half the weight of comparable CONT infants (651g ± 24.37 vs. 1120g ± 297.89; Fig 3.1b). Whilst the CAL infants are able to increase their mass by a minimal amount every month of the experiment, the PROT infants' body masses remain at approximately 500g for the entire period (Fig 3.1c). The level of protein in the PROT diet is indeed insufficient to promote growth, as intended by the experimenters. It is possible to increase body mass on a very calorie-deficient diet, but almost impossible on a very protein-deficient diet.

5] PC infants show a different pattern of body growth.

The PC infants show a different pattern of growth from either the CAL or PROT infants (Fig 3.1d). They are able to increase their body mass over the 20-week experiment, and at a rate comparable to that of the CONT group (30-35g per month in both groups). Even though these PC infants start the experiment at a much lower average body mass, their 'normal' rate of growth ensures that, by the end of the experiment, the PC capuchins are heavier than the other non-CONT groups.

6] Removal of the nutritional insult results in catch-up body mass growth in both the CAL and PROT infants, but to differing degrees.

Both the CAL and PROT capuchins show rapid mass increase after the nutritional insult is removed, and by 12 months old the CAL infants have reached 90.59% of their target mass (1197.88g ± 81.49 vs. CONT body mass of 1322.31g ± 287.30). The PROT group are 19.56% lighter than their CONT counterparts by the end of the first year (1063.67g ± 82.53; Fig 3.1b and c).

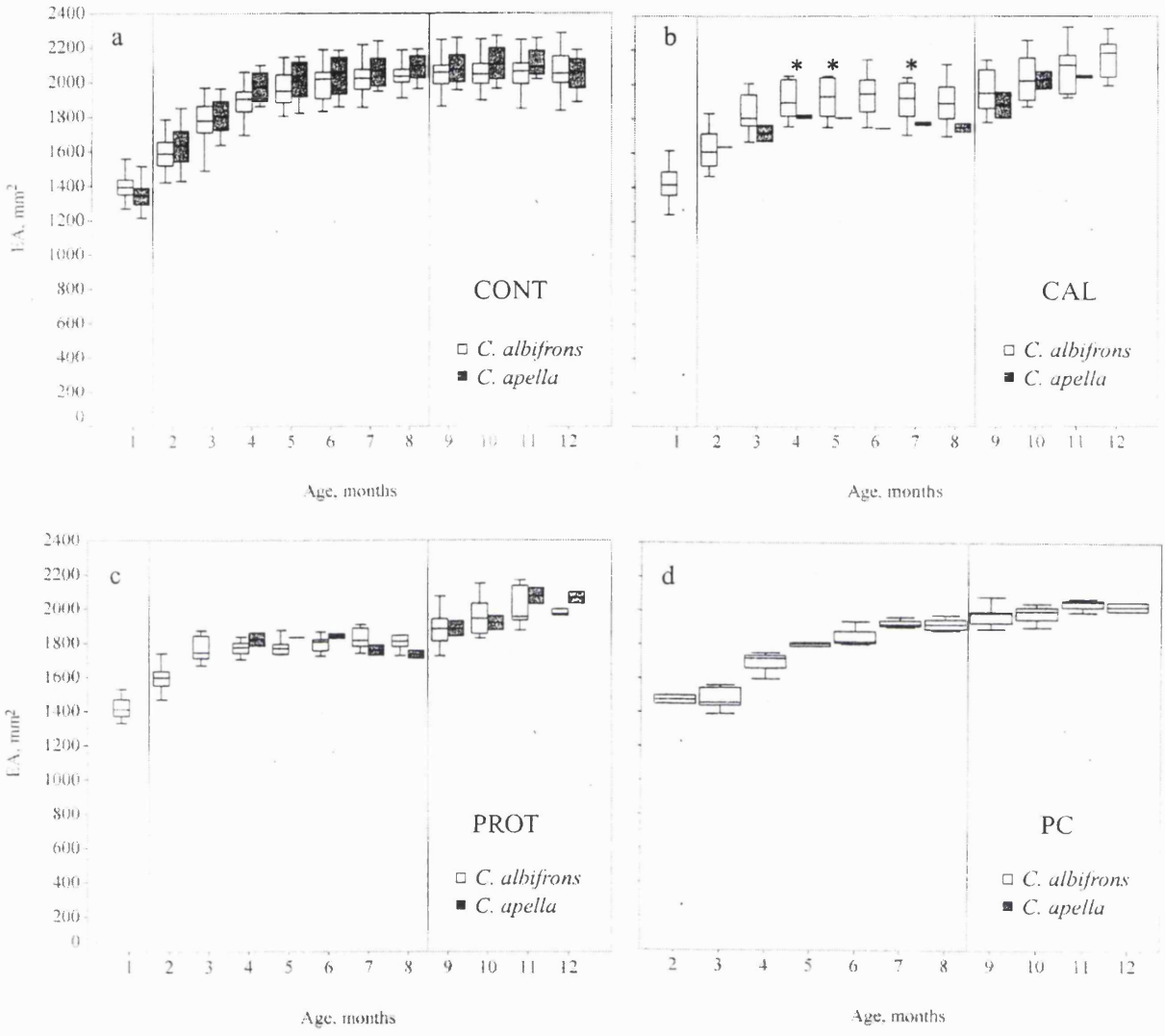


Figure 3.2. Brain growth in the dietary groups: species dimorphism. (a) Controls (CONT); (b) calorie-deficient (CAL) infants; (c) protein-deficient (PROT) infants; (d) protein- and calorie-deficient (PC) infants. No data available for PC infants in month 1. See Fig 3.1 for key.

7] PC infants show less catch-up body growth.

The PC infants are less able to recover from the nutritional insult. The rapid increase of body mass seen in CAL and PROT groups in the last quarter of the year is absent in the PC infants (Fig 3.1d). Body mass increases from 892.50g ± 58.34 to 1020.00g ± 137.18 between the 9th and the 12th month in the PC infants, with the result that they are 22.86% smaller than the CONT group at 12 months old.

8] C. apella *catch-up growth is more rapid than that of* C. albifrons.

Both the CAL and PROT *C. apella* appear to increase mass more rapidly in the post-experiment period, and are significantly heavier than the CAL *C. albifrons* during the 11th and 12th months (Fig 3.1b and c). *C. apella* are able to increase mass at a faster rate in this post-experiment period.

9] Few significant species differences exist in brain size.

Very few significant differences exist between the two species in terms of EA, although the CONT group *C. apella* show consistently larger average EA (Fig 3.2a). The only differences to reach statistical significance are in the CAL group, where *C. apella* show smaller brains over the experimental period (Fig 3.2b). The brains of the non-CONT groups remain virtually constant in size for the entire 20-week experiment, the CAL *C. albifrons* at approximately 1900mm^2, the PROT *C. albifrons* at approximately 1800mm^2. This equates to a difference of 10g, or 15% of a 65g brain, between these two non-CONT groups.

10] PC infants show elevated brain growth rates compared to the other non-CONT groups.

As noted above, the PC infants are relatively small when the experiment begins, but because they show an increase in brain size comparable in rate with that of the CONT group, their brain sizes are similar to the other non-CONT infants by 8 months old (Fig 3.2d).

11] No group differences in brain size exist by one year of age.

All four group mean EAs are indistinguishable by the 10th or 11th month, suggesting that brain size is largely protected during nutritional stress, and that higher proportions of target brain size are achieved relatively soon after the stress is removed.

3.2.3 Sexual dimorphism

As described above, no *C. apella* were placed in the PC group, and the only female *C. apella* included in the study was part of the CONT group. Sexual dimorphism could not, therefore, be assessed for the non-CONT *C. apella* groups.

1] Significant sexual body size dimorphism is present from an early age in the CONT infants.

The CONT males and females show statistically significant body mass dimorphism from an early age, in *C. albifrons* from the seventh month of life onwards (Fig 3.3b), in *C. apella* from the second (Fig 3.3b). By the age of one year, CONT males are approximately 25% larger than females in both capuchin species. No significant differences exist in the non-CONT groups, and male and female CAL, PROT and PC data are combined in the following analyses.

2] Under stress, the direction of body mass dimorphism is reversed.

None of the male and female CAL, PROT or PC *C. albifrons* show statistically significant differences in body mass during the first year of life. However, the direction of dimorphism is changed in the CAL group, i.e. female CAL infants have slightly heavier bodies than males during the experimental period (Fig 3.3c). It is not clear if the same is true of the PROT group. Although both sexes continue to grow at a fairly rapid rate in the PC group, females are again larger than males at each month interval of the experiment. However, the

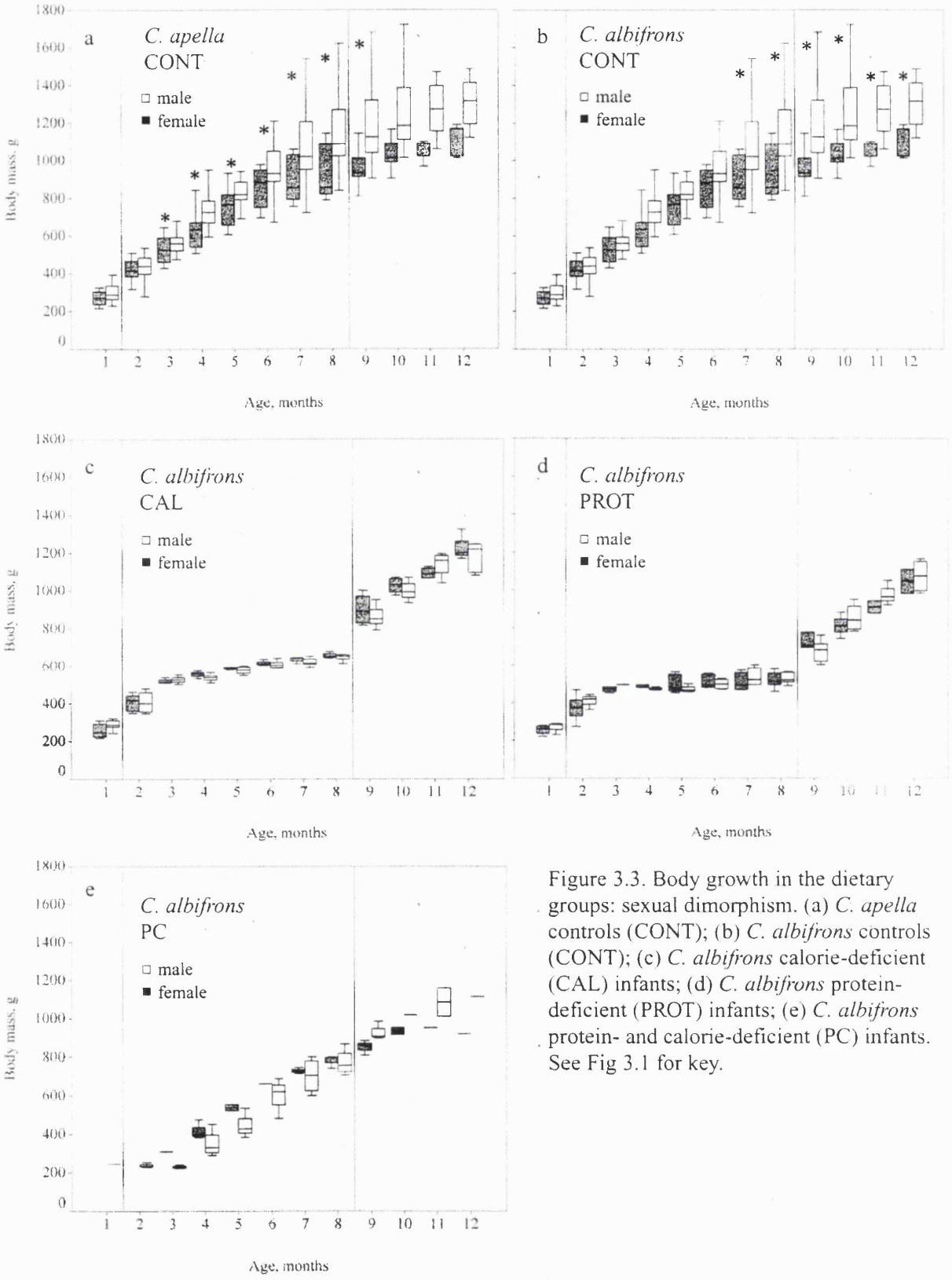


Figure 3.3. Body growth in the dietary groups: sexual dimorphism. (a) *C. apella* controls (CONT); (b) *C. albifrons* controls (CONT); (c) *C. albifrons* calorie-deficient (CAL) infants; (d) *C. albifrons* protein-deficient (PROT) infants; (e) *C. albifrons* protein- and calorie-deficient (PC) infants. See Fig 3.1 for key.

males show an elevated body growth rate after the experiment finishes, and soon outweigh the females. Males therefore seem able to respond more effectively to the removal of the nutritional stress.

3] Few significant sex differences exist in brain size.

Although male brains are consistently larger than female brains in the CONT groups, for the most part there are few statistically-significant sex differences, and these all occur after 8 months postpartum when female, but not male, brain growth slows (Fig 3.4).

4] Direction-reversal in brain size dimorphism exists in some, but not all, groups.

The trend for a direction-reversal in dimorphism (for bigger females and smaller males), seen for the body masses of the CAL and PC infants (and possibly the PROT *C. albifrons*), is also present in PROT and PC brain sizes, as might be expected (Fig 3.4d, e). In these groups, the females show larger brains until the end of the experimental period, after which male growth soon outstrips female growth. By the 12th month, the sexes are similar in brain size. The CAL capuchins are an interesting exception to this non-CONT trend: male CAL EA is consistently larger than female EA throughout the experiment (Fig 3.4c). These results suggest that, in the presence of undernutrition (calorie deficiency), the brain is protected to the extent that normal patterns of male/female brain growth differences are preserved, even when body growth patterns are not maintained. On the other hand, malnutrition (protein deficiency) produces a different response: the patterns of both brain and body growth are different from those of the CONT groups, and because of their larger target sizes, males appear to be worse affected than females.

When the minimum sufficient protein for growth is available, as in the PC group, the growth patterns are similar to those of the PROT infants, rather than the CAL infants, i.e. males fare worst (Fig 3.4e). It is probable that the impact of both mal- and undernutrition in these individuals disrupts growth in the same way as more severe protein-deficiency.

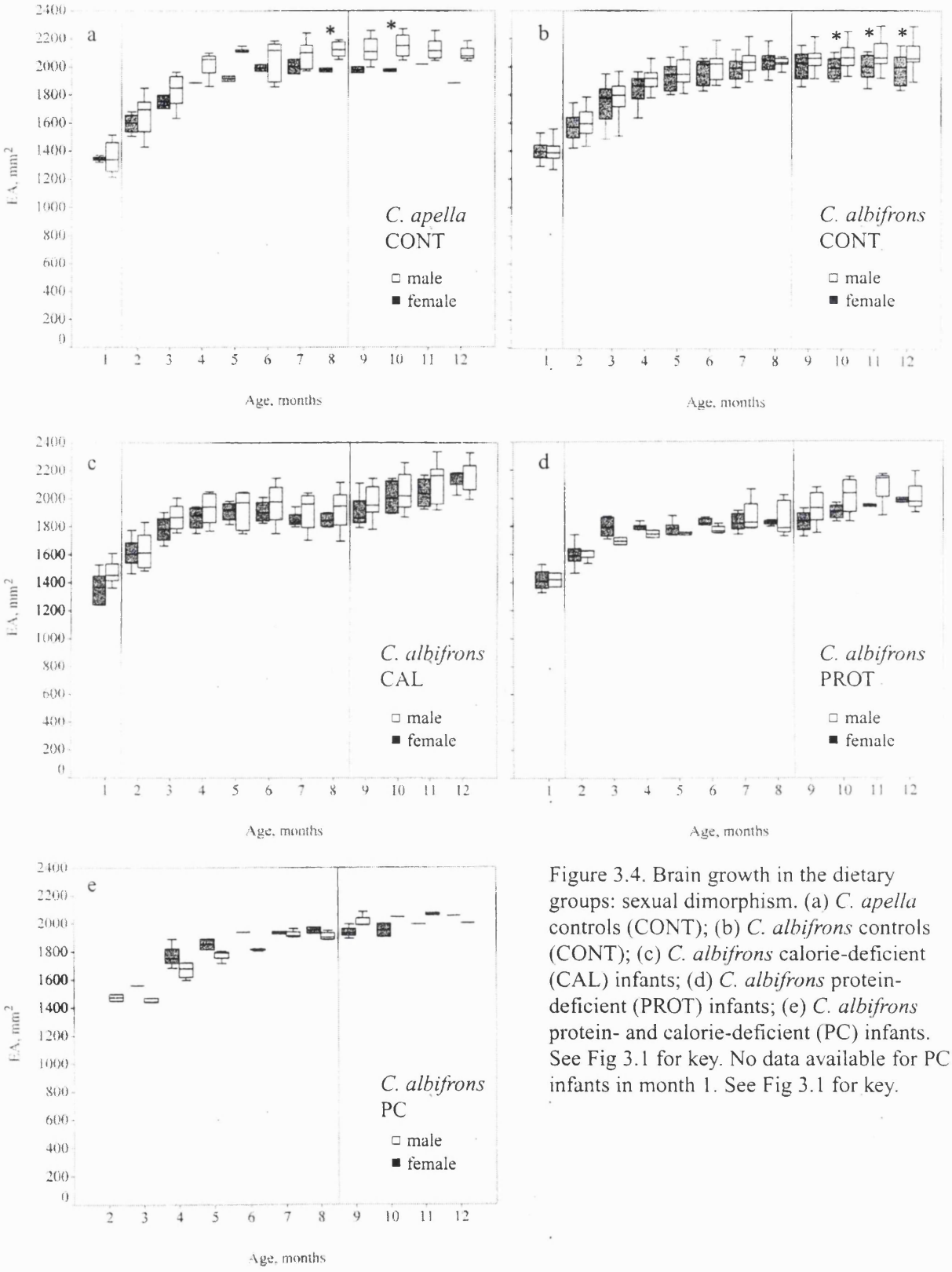


Figure 3.4. Brain growth in the dietary groups: sexual dimorphism. (a) *C. apella* controls (CONT); (b) *C. albifrons* controls (CONT); (c) *C. albifrons* calorie-deficient (CAL) infants; (d) *C. albifrons* protein-deficient (PROT) infants; (e) *C. albifrons* protein- and calorie-deficient (PC) infants. See Fig 3.1 for key. No data available for PC infants in month 1. See Fig 3.1 for key.

3.2.4 CONT vs. non-CONT: is brain growth protected?

Figures 3.5 and 3.6 compare body mass and EA between the CONT and non-CONT infants at each age interval and within each species. Mean non-CONT values are expressed as percentages of mean CONT values i.e. are relative masses. CONT infants are divided into male and female groups (where possible), but are compared with combined-sex averages from the non-CONT groups as appropriate.

a] *C. apella*

The non-CONT *C. apella* infants appear to fare the worst of all groups (Fig 3.5). Compared to *C. albifrons*, dietary-deficient *C. apella* infants achieve smaller proportions of their target CONT body mass during the experiment. Non-CONT body masses are significantly smaller than CONT mass throughout the experimental period, and for at least two months following the resumption of a normal diet. Relative mass reduction (relative to CONT mass) begins in the very first month of the experiment, the lowest mass (40% of CONT mass) occurring at or towards the end of the experiment.

The CAL infants stabilise their relative mass at 50% of CONT mass, and maintain this over three or four months (Fig 3.5a). In fact, absolute body mass is increasing in the CAL group (Fig 3.1b); the fact that relative mass is constant across the experiment suggests that the amount of energy invested in growth by these infants is the minimum amount necessary to prevent further loss of mass. PROT relative body mass, on the other hand, continues to drop over each successive month of the experiment, to a low of 40% of CONT mass (Fig 3.5b). This trend shows no sign of diminishing by the end of the experiment, and it is probable that relative mass loss would have continued had a normal diet not been resumed. This continued drop in PROT relative mass is the result of a stabilisation of body mass over the entire 20-week diet (Fig 3.1c) such that, relative to the CONT infants, the percent of target size achieved by the PROT infants decreases each month.

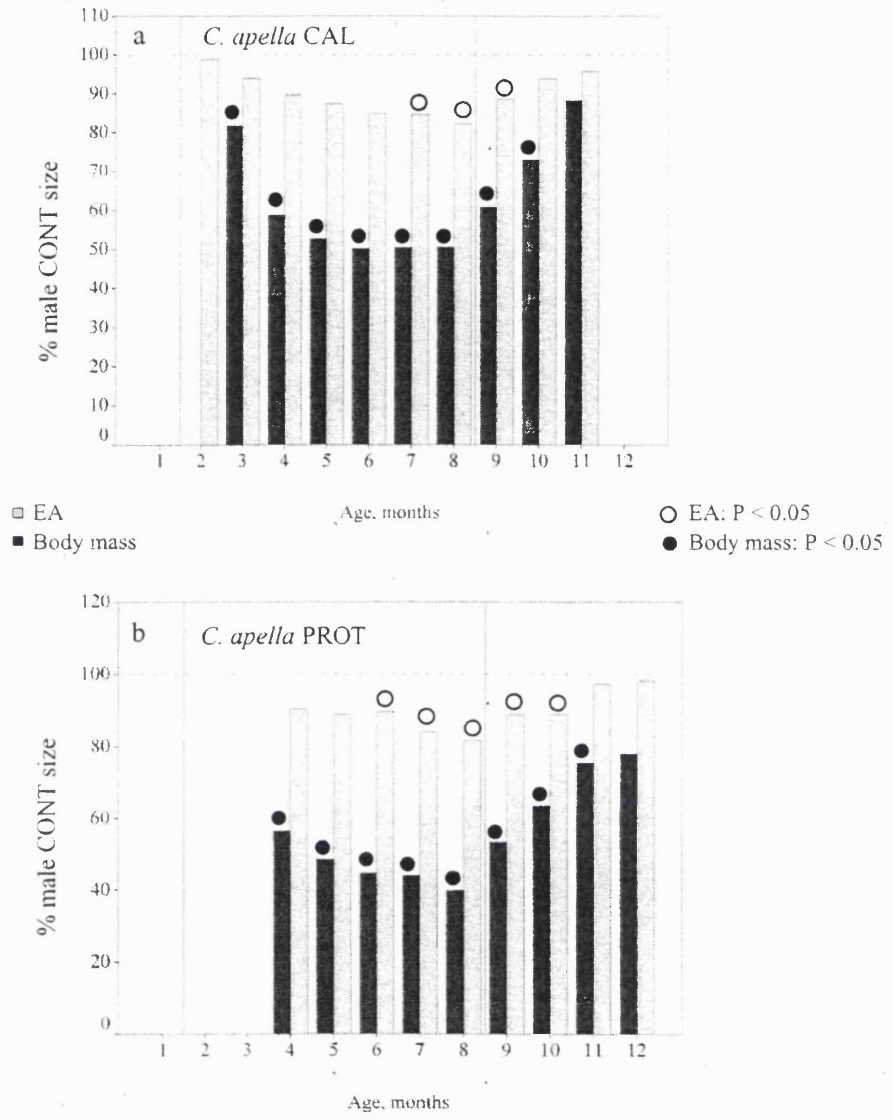


Figure 3.5. Brain and body size differences in the *C. apella* CONT and *C. apella* non-CONT groups. (a) Male CONT and male calorie-deficient (CAL) infant comparisons; (b) male CONT and male protein-deficient (PROT) infant comparisons. The mean non-CONT value for each month is expressed as a percent of the mean value for the CONT group in that month. Error bars could not be calculated, but significant differences between the group mean values are shown: solid circles indicate that body mass is significantly different in a month, open circles indicate that EA is significantly different in a month (student's t-test, $P < 0.05$).

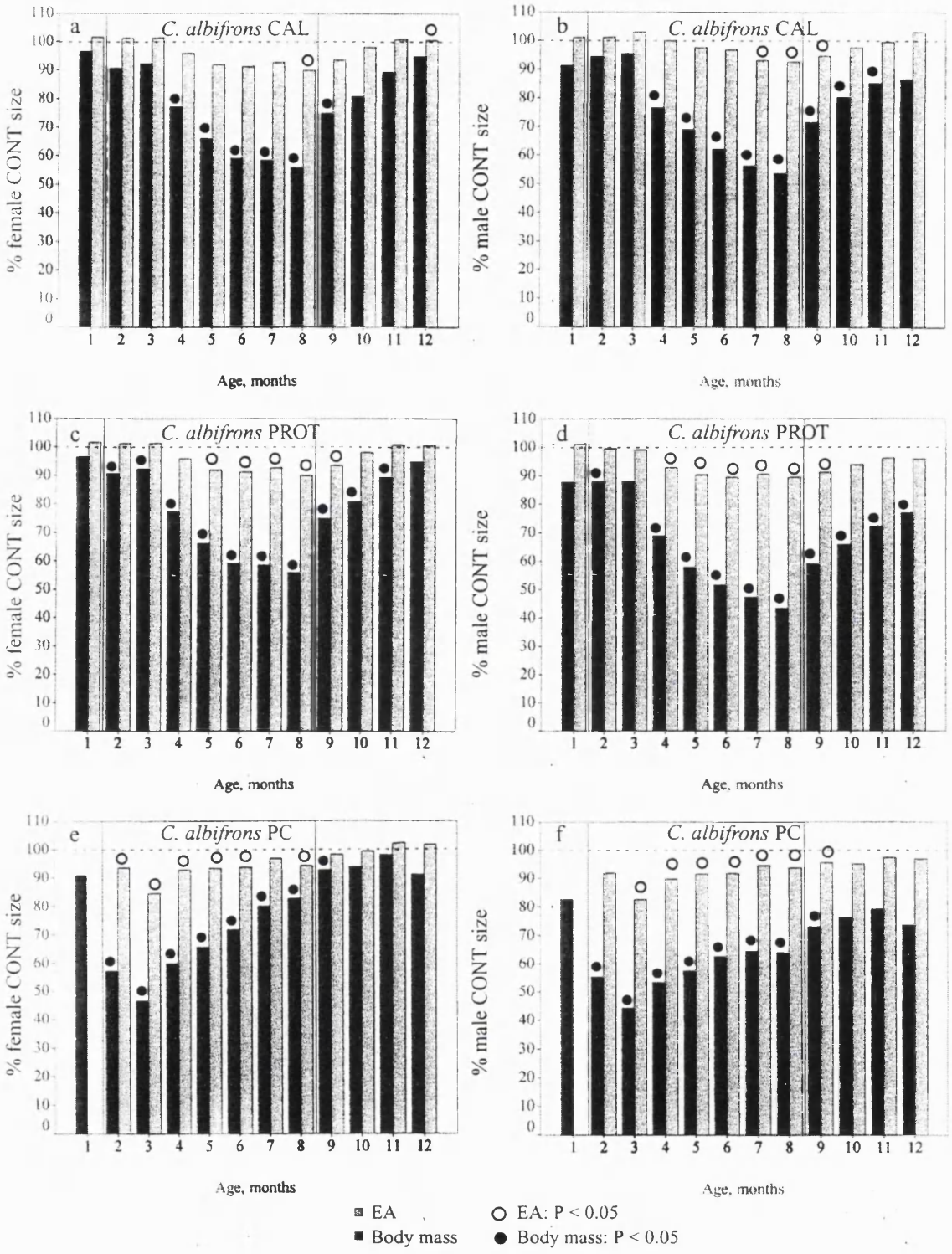


Figure 3.6. Brain and body size differences in the *C. albifrons* CONT and *C. albifrons* non-CONT groups.
(a) Female CONT and sex-combined calorie-deficient (CAL) infant comparisons.
(b) Male CONT and sex-combined calorie-deficient (CAL) infant comparisons.
(c) Female CONT and sex-combined protein-deficient (PROT) infant comparisons.
(d) Male CONT and sex-combined protein-deficient (PROT) infant comparisons.
(e) Female CONT and sex-combined protein- and calorie-deficient (PC) infant comparisons.
(f) Male CONT and sex-combined protein- and calorie-deficient (PC) infant comparisons.
See Fig 3.5 for key.

Brain size in *C. apella* is also diminished in response to the CAL and PROT regimes, but is not affected to the same extent as body size. Even at its smallest percentage size (during the 8th month of the experiment in both groups), EA never drops below 80% of target size. EA is significantly different from CONT size in only the last two (in the CAL group, Fig 3.5a) or three (in the PROT group, Fig 3.5b) months of the experimental period.

b] *C. albifrons*

Just as the larger *C. apella* suffer the effects of mal- and undernutrition more than the smaller-bodied *C. albifrons*, when the non-CONT *C. albifrons* are compared with the different sex CONT groups, it is against the larger male target size that they fare the worst (Fig 3.6). The CAL group weighs 69.45% of female CONT body mass by 8 months old (Fig 3.6a), but as low as 53.8% of the male CONT body mass in the 8th month postpartum (Fig 3.6b). It is interesting to speculate whether their mass would have stabilised, had the experiment continued. PROT body masses drop as low as 56.0% of female CONT size (Fig 3.6c) and to 43.4% of male CONT size (Fig 3.6d). Brain size does not drop below 90% of target size in either the CAL or PROT groups.

The PC infants again present a distinctive pattern of growth (Fig 3.6e and f). All other groups show a decline in proportion of target body mass achieved as the experiment continues, but the PC group shows the opposite: they achieve progressively higher proportions of target body mass, whether compared against male or female CONT infants. Although they are only 46.5% of female and male target body mass during month 3, by month 8 the PC infants have recovered to 8.7% of female and male target body mass. Similarly, brain size drops to 84.6% of target size in the first month of the experiment, but by the last month have brains that are 94.3% of target size.

3.3 Results: II. Brain and body growth rates

3.3.1 Species dimorphism and general trends

1] Normal body growth rates decline smoothly over 12 months, but C. apella *grow faster than* C. albifrons.

The CONT groups show a smooth decline of body growth rates from the first to the 12th month of life (Fig 3.7a). Both species increase their body mass by 0.02g per day per gram body mass (approximately 40g a week for a 250g infant) in the first month. *C. apella* grow consistently faster than *C. albifrons* for the next four months. The larger species grows faster, even after size has been controlled for, although the differences are not significant, and by the middle of the year both species grow at a quarter of the rate of month 1(0.005g/d/g body mass, or 30g a week for an 800g infant). *C. apella* shows a slight increase in rate during month 9, but by month 12 are actually showing a decrease in size compared to the previous months. Body growth rates of *C. albifrons*, on the other hand, remain low but positive until the end of the year, and only in the twelfth month is the difference between the species significant.

2] Few significant species differences exist in the body growth rates of the non-CONT groups.

There are similarly few significant differences between species' body growth rates in the non-CONT groups. The larger *C. apella* tend to fare worse than the smaller *C. albifrons*, growing more slowly per unit body mass. This is especially apparent in the CAL group (Fig 3.7c). Here, body growth rates drop to a minimum over the experimental period in both species, but *C. apella* CAL rates are lower than *C. albifrons* CAL rates, even though they are quicker to recover high rates of growth on resumption of a normal diet. The same is true of the PROT group. The PROT growth rates hover at or below zero for the entire 20-week experiment. This difference in the two groups – CAL infants showing minimal

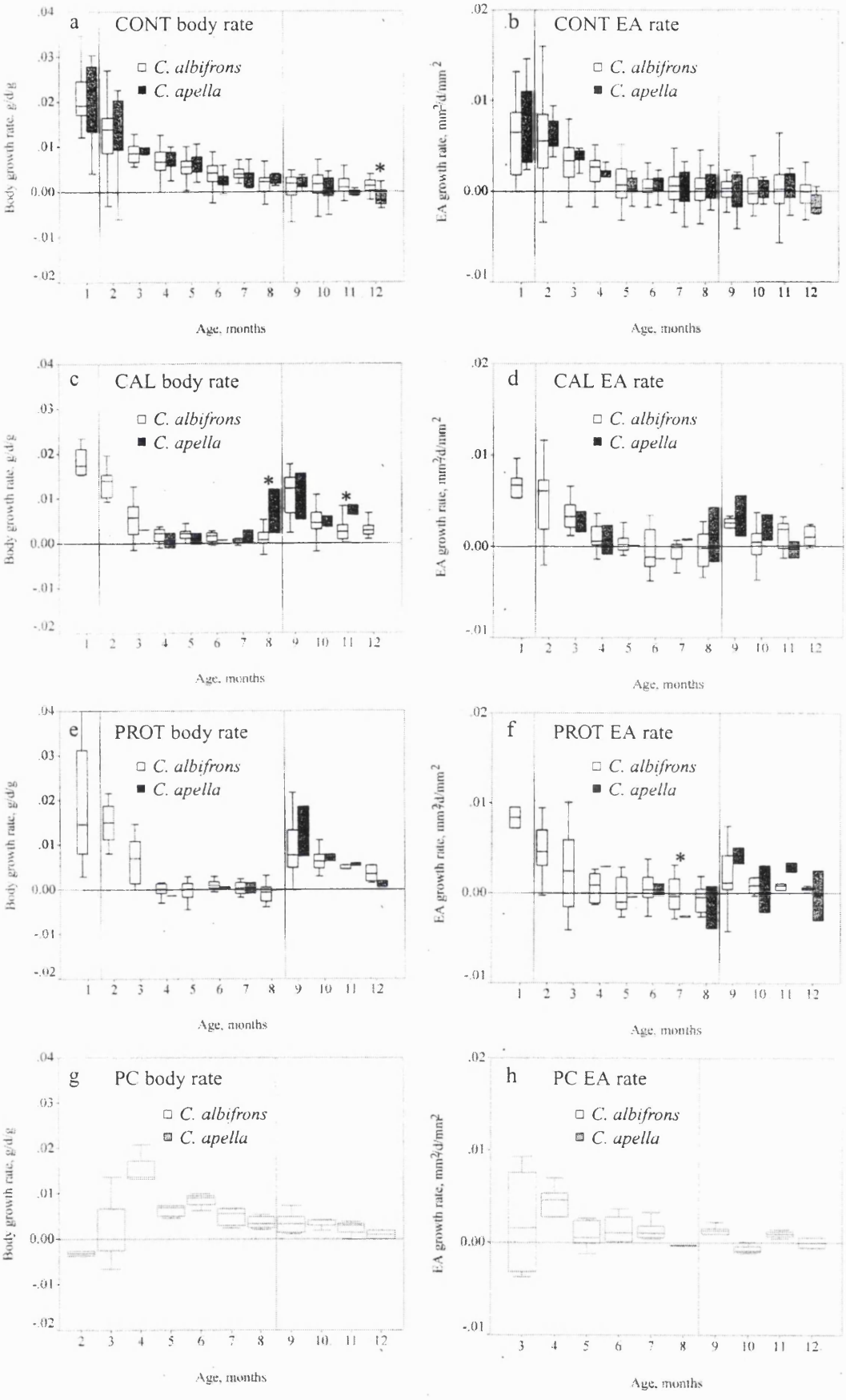


Figure 3.7. Body and brain growth rates in the dietary groups: species dimorphism.
(a) Control (CONT) body growth rate.
(b) Control (CONT) EA growth rate. *cont'd*

Fig 3.7 cont'd

(c) Calorie-deficient (CAL) body growth rate.
(d) Calorie-deficient (CAL) EA growth rate.
(e) Protein-deficient (PROT) body growth rate.
(f) Protein-deficient (PROT) EA growth rate.
(g) Calorie- and protein-deficient (PC) body growth rate.
(h) Calorie- and protein-deficient (PC) EA growth rate
No body mass data available for PC infants in month 1; no EA data available for PC infants in months 1 or 2. See Fig 3.1 for key.

growth, PROT infants showing a size decrease – is consistent with the absolute mass trends seen in Section 3.2 (Fig 3.1b and c).

3] When the nutritional stress is removed, CAL and PROT body growth rates return to pre-experiment levels.

CAL and PROT rates increase in the last quarter of the year to levels comparable with the CONT group at about three months old. These rates then decline again as the end of the year is reached (Fig 3.7c and e).

4] PC infants follow a different pattern of growth.

Once again, the PC group follows a different pattern of growth (Fig 3.7g). Initial growth rates are lower than those of any other group, suggesting that the sudden removal of both protein and calories from the diet produces an acute and severe body mass loss. However, by 4 months old, rates are in fact higher than those of the CONT infants at a similar age (0.015g/d/g vs. 0.007g/d/g). The PC infants' body masses are increasing at normal rates even though they are only achieving a small part of target size (Fig 3.1d). Neither do they show elevated growth rates in the post-experiment period; body mass increases at an ever-diminishing rate, as it does in the CONT groups.

5] Variation in EA is high, but growth is consistent over the first six months postpartum.

Variation in EA growth rates is much higher than in body rates, and only one difference between the species is significant (Fig 3.7f). However, consistent trends do emerge from the data even though statistical significance cannot be attached to them. As with body mass, brain size increases at a faster rate in CONT *C. apella* compared with CONT *C. albifrons* throughout the first year of life. Both species show a size decrease in the twelfth month. Unlike body mass, the drop in EA growth rate is sharp over the first four months and reaches a plateau at about 5 or 6 months old, suggesting that the majority of postnatal brain growth occurs in the first half of the year. The non-CONT groups provide further

indication that brain growth is protected at the expense of body growth. In all three, EA growth rates are very similar to those of the CONT group up until the age of about 5 or 6 months, the age at which growth is essentially completed in the CONT group. As discussed above, body growth rates in the non-CONT groups are severely compromised in this same period. However, after month 6 EA mass decreases, in the *C. albifrons* CAL group for the remainder of the experiment, in the *C. apella* CAL group for one month only. On removal of the nutritional stress, *C. apella* show a sharp rise in rate, followed by a swift decline; as before, *C. albifrons* show a more subtle decline in rates in the last quarter of the year.

The same trends are observed in the PROT group, with the exception that the peaks and troughs of the EA growth rate are more exaggerated than those of the CAL infants. Clearly, dietary deficiency does affect the rates at which brain size changes during development. As with body mass, brain size is a labile characteristic that does not always follow a strict pattern of development. As we saw in Section 3.2, however, brain size is less plastic than body size, and from the shapes of the non-CONT curves below 6 months old, it seems that certain stages in postnatal brain development are not compromised, even under severe nutritional stress. This result has been found in other taxa reared under experimental nutritional regimes, e.g. rats (Smart, 1990; Royland *et al.*, 1992; Bedi, 1994).

3.3.2 Sexual dimorphism

1] Males generally grow faster than females, both in terms of brain and body size.

Although female growth rates are higher than those of males in the first one or two months of life, CONT males grow consistently (but not significantly) faster than CONT females in both species over the next four months (Fig 3.8a and b). Growth rates are similar in the second half of the year, although females show a slight peak in rates (at month 8 or 9 in *C. apella*, at month 10 in *C. albifrons*). In this period they are, in fact, increasing mass at a greater rate than their male counterparts. There is no consistent pattern of rate dimorphism

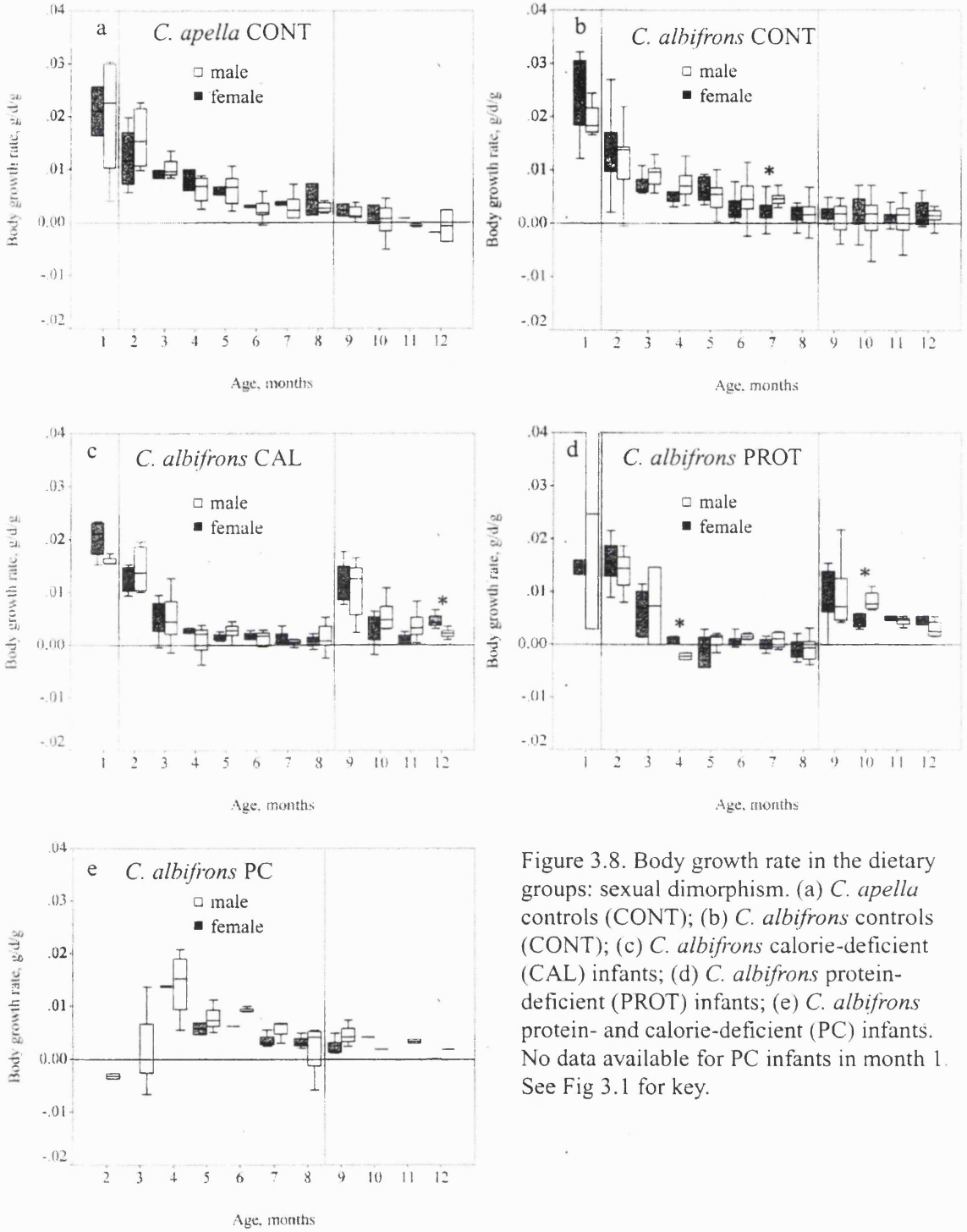


Figure 3.8. Body growth rate in the dietary groups: sexual dimorphism. (a) *C. apella* controls (CONT); (b) *C. albifrons* controls (CONT); (c) *C. albifrons* calorie-deficient (CAL) infants; (d) *C. albifrons* protein-deficient (PROT) infants; (e) *C. albifrons* protein- and calorie-deficient (PC) infants. No data available for PC infants in month 1. See Fig 3.1 for key.

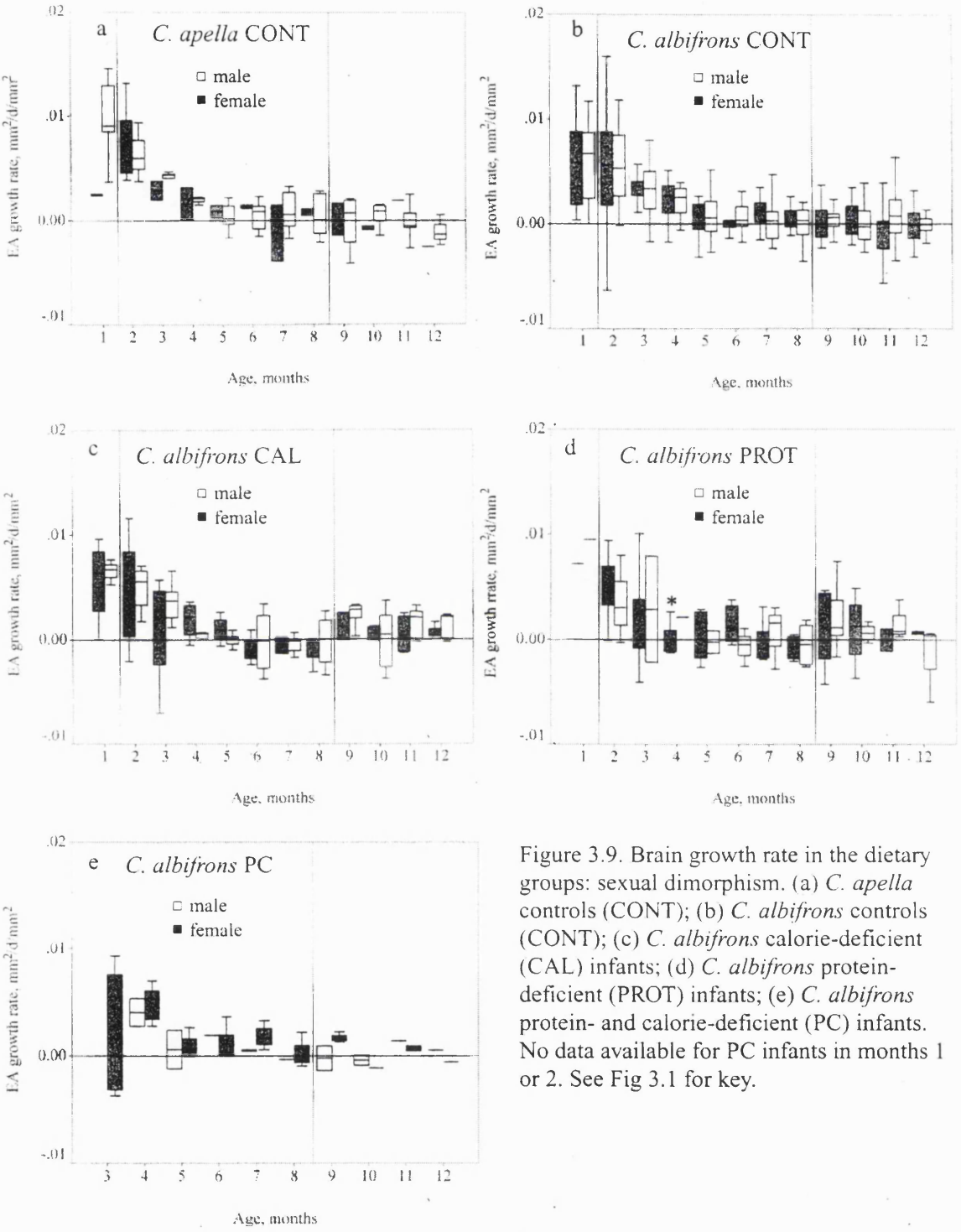


Figure 3.9. Brain growth rate in the dietary groups: sexual dimorphism. (a) *C. apella* controls (CONT); (b) *C. albifrons* controls (CONT); (c) *C. albifrons* calorie-deficient (CAL) infants; (d) *C. albifrons* protein-deficient (PROT) infants; (e) *C. albifrons* protein- and calorie-deficient (PC) infants. No data available for PC infants in months 1 or 2. See Fig 3.1 for key.

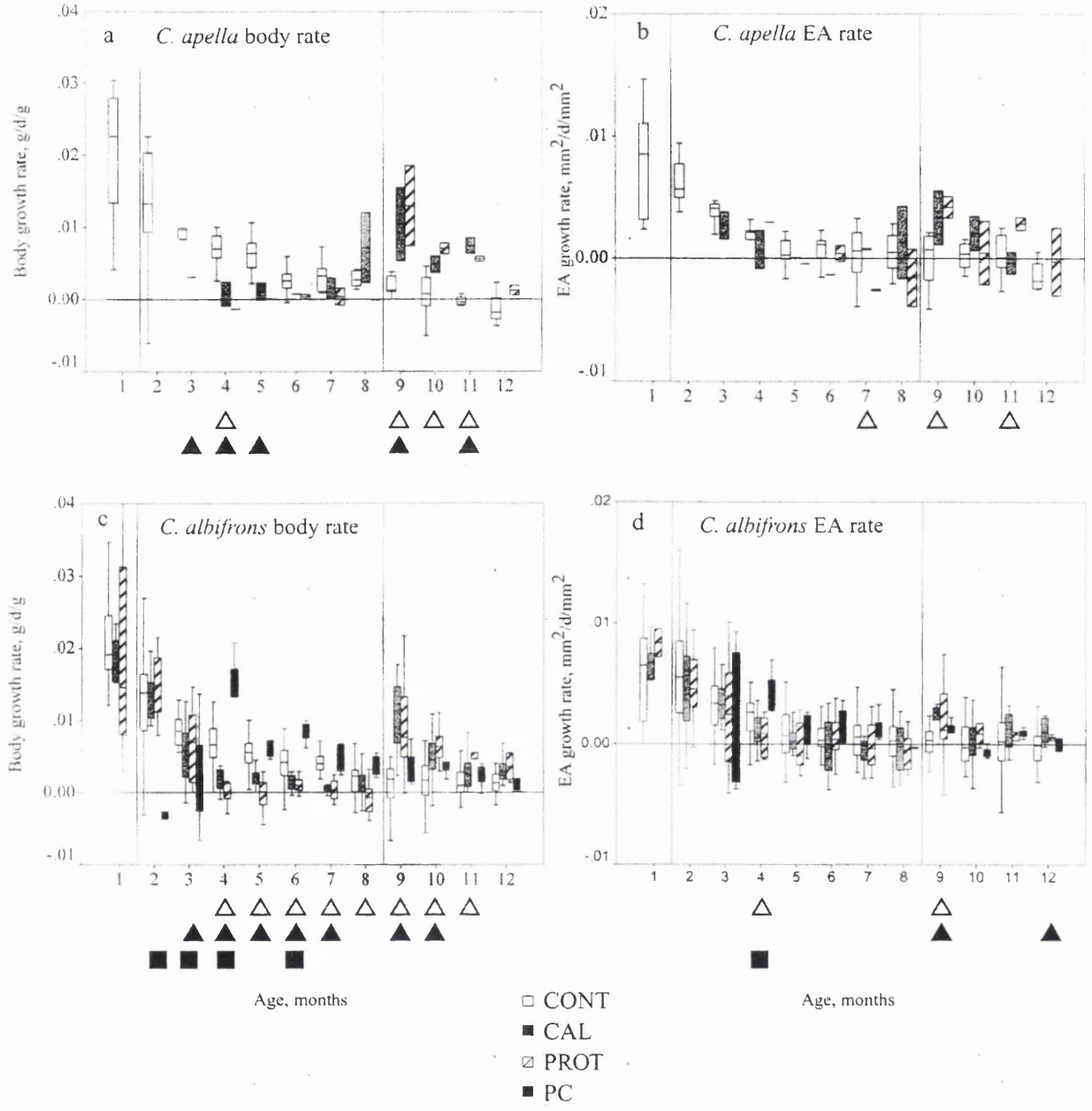


Figure 3.10. Brain and body growth rate differences in the CONT and non-CONT groups. (a) Body growth rates in *C. apella infants*; (b) Body growth rates in *C. albifrons* infants; (c) EA growth rates in *C. apella* infants; (d) EA growth rates in *C. albifrons* infants. Significant differences between the mean values of the CONT and the CAL infants indicated by a closed triangle; between the CONT and PROT infants, by an open triangle, and between the CONT and the PC infants, by a closed square.

in the CAL or PROT groups (Fig 3.8c, d, e), but the PC infants show a pattern of rate change that is similar to that of the CONT infants.

As with body mass, males generally increase in brain size faster than do females, at least in the early part of the first year. After six months, when brain growth is more or less complete, no clear differences between the sexes are apparent in Figure 3.9. This is true for both the CONT and non-CONT groups. All non-CONT infants show a relative increase in brain growth rate for at least one month after the experiment ends.

3.3.3 CONT vs. non-CONT: are brain growth rates protected?

CAL body growth rates are significantly lower than CONT rates from the beginning of the experiment, in *C. apella* remaining so until month 5 (Fig 3.10a), in *C. albifrons* until month 7 (Fig 3.10c). EA rates, on the other hand, are not significantly different in these two groups in any month during the experiment (Figs 3.10b, d). Only when a normal diet is resumed do CAL EA growth rates become significantly higher than CONT EA rates, and this only occurs in *C. albifrons* (Fig 3.10d). Similarly, PROT brain and body growth rates are significantly lower during the experiment, and significantly higher afterwards (Fig 3.10). The PC infants show a different pattern. They increase body mass at a faster rate than the CONT infants (Fig 3.10c), both during and after the experiment. EA growth rates are again elevated compared to CONT rates from months 3 to 7 (Fig 3.10d), perhaps compensating for their relatively small brain size at 3 months old (Fig 3.2d). Many more significant differences are found in the body mass comparisons than are found in the EA comparisons. This suggests that brain growth rate in the deficient-diet groups is protected at the expense of the body growth rate. It is, however, worth noting that the EA sample sizes are small, and show a larger range of variation than the body mass data.

Table 3.3 Testing the dietary groups' brain allometries for linearity

Group	Sex	n	R_1^2	R_2^2	df	F
			C. apella			
CONT	F	31	0.851	0.939	28	40.26***
CONT	M	65	0.872	0.906	62	22.25***
CAL	M	18	0.865	0.865	15	0.00
PROT	M	19	0.863	0.879	16	2.20
			C. albifrons			
CONT	F	149	0.739	0.789	146	34.70***
CONT	M	289	0.828	0.866	286	80.65***
CAL	F	51	0.807	0.863	48	19.37***
CAL	M	91	0.581	0.632	88	12.15**
PROT	F	61	0.688	0.829	58	47.61***
PROT	M	48	0.685	0.790	45	22.41***
PC	M&F	53	0.893	0.922	50	18.42***
Mean ± sd			0.788 ± 0.101	0.844 ± 0.085		
se			0.030	0.026		
95% confidence interval			0.728 – 0.848	0.792 – 0.896		

Table 3.4 Pre- and post-inflexion RMAs of the *C. albifrons* samples

Group	Sex	Pre-inflexion RMA slope ± s.e.	Pre-inflexion RMA 95% confidence interval for slope	Pre-inflexion RMA intercept	Post-inflexion RMA slope ± s.e.	Post-inflexion RMA 95% confidence interval for slope	Post-inflexion RMA intercept
			C. albifrons				
CONT	F	0.403 ± 0.047	0.309 – 0.497	2.155	0.179 ± 0.019	0.141 – 0.217	2.754
CONT	M	0.384 ± 0.024	0.336 – 0.432	2.201	0.193 ± 0.010	0.173 – 0.213	2.711
CAL	F	0.447 ± 0.052	0.343 – 0.551	2.059	0.165 ± 0.027	0.111 – 0.219	2.782
CAL	M	0.505 ± 0.084	0.337 – 0.673	1.909	0.192 ± 0.025	0.142 – 0.242	2.714
PROT	F	0.410 ± 0.064	0.282 – 0.538	2.162	0.096 ± 0.016	0.064 – 0.128	2.979
PROT	M	0.352 ± 0.042	0.268 – 0.436	2.300	0.174 ± 0.029	0.116 – 0.232	2.754
PC	M&F	0.431 ± 0.050	0.331 – 0.531	2.145	0.151 ± 0.014	0.123 – 0.179	2.838

3.4 Results: III. Brain allometries in the non-CONT infants

All except two non-CONT groups have postnatal brain growth allometries that are significantly non-linear (Table 3.3). These two exceptions are the CAL and PROT *C. apella* groups, which each contain data from one individual. They are discussed separately below. As in previous chapters, the amount of variation explained by both the linear and quadratic regressions is high across all groups ($R_L^2 = 0.788 \pm 0.031$; $R_Q^2 = 0.844 \pm 0.026$).

The slopes of the pre- and post-inflexion curves of the *C. albifrons* non-CONT groups are similar to those of the *C. albifrons* CONT groups (Table 3.4). All non-CONT slopes fall within the 95% confidence intervals of the CONT groups, with the exceptions of the pre-inflexion slope in the male CAL sample, and the post-inflexion slope in the female PROT sample. The male CAL slope is higher than the other groups' slopes (0.505 ± 0.084), but the 95% confidence interval is comparable with those obtained for the CONT individuals described in Chapter 2, e.g. Vixen (0.418 ± 0.049, Table 2.3). The female PROT confidence interval falls below the female CONT range for the post-inflexion slope (0.096 ± 0.016), but again, this slope is comparable with those of other individuals in the CONT group (Chapter 2, Table 2.3). The differences in mean slopes between the CONT and non-CONT *C. albifrons* groups is not significant (data not shown).

Although the majority of non-CONT groups in Table 3.4 fall within the confidence intervals of the CONT groups, they tend to show consistently higher pre-inflexion slopes than the CONT groups (Fig 3.11a). For clarity, only female *C. albifrons* data are shown in Figure 3.11a; male *C. albifrons* trends are similar. The CONT brain allometry is shown with a solid line. The other dashed lines represent the brain allometries of the non-CONT groups. Each of these latter groups has pre-inflexion slopes that are elevated compared to the non-CONT groups. The difference in mean pre-inflexion RMA intercept between the CONT and non-CONT groups is not significant (data not shown), but the figure shows that the non-CONT animals are more encephalised than the CONT animals. As the analyses in the previous sections of this chapter have shown, this difference has arisen because, for

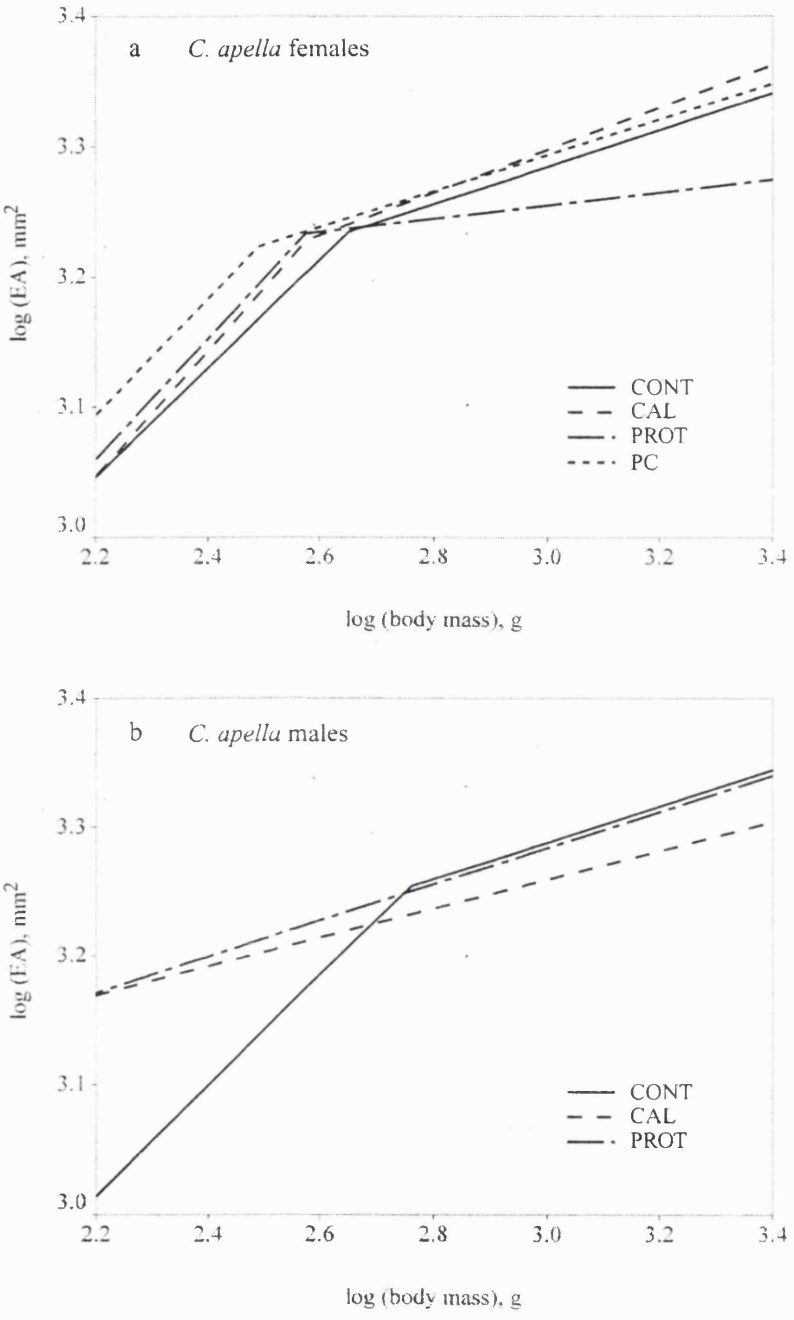


Figure 3.11. Brain allometries in the CONT and non-CONT infants. (a) Female *C. albifrons*; (b) male *C. apella*.

Table 3.5 Pre- , post-inflexion and whole sample RMAs of the *C. apella* samples

Group	Sex	Pre-inflexion RMA slope ± s.e.	Pre-inflexion RMA 95% confidence interval for slope	Pre-inflexion RMA intercept	Post-inflexion RMA slope ± s.e.	Post-inflexion RMA 95% confidence interval for slope	Post-inflexion RMA intercept
				C. apella			
CONT	F	0.351 ± 0.024	0.303 – 0.399	2.285	0.166 ± 0.045	0.076 – 0.256	2.786
CONT	M	0.418 ± 0.033	0.352 – 0.484	2.088	0.154 ± 0.043	0.068 – 0.240	2.821

		Whole-sample RMA slope ± s.e.	Whole-sample RMA 95% confidence interval for slope	Whole-sample RMA intercept
			C. apella	
CAL	M	0.140 ± 0.018	0.104 – 0.176	2.843
PROT	M	0.145 ± 0.017	0.111 – 0.179	2.842

their brain size, the non-CONT groups have small bodies. The most elevated pre-inflexion slope in Figure 3.11a is that of the PC infants; the next is that of the PROT infants; the slope of the CAL infants most closely approximates that of the CONT group. This arrangement ties in well with the trend to progressively smaller relative body masses (relative to CONT mass) across the dietary-deficient groups (Fig3.6). The position of the inflexion along the body mass axis also corresponds to the relative severity of the diet. The difference in mean body mass at inflexion between the CONT and non-CONT groups is significant ($t = 3.979$, $df = 5$, $P = 0.011$), but the difference in EA at inflexion is not (data not shown).

The female PROT infants (Fig 3.11a) have a low slope value in the post-inflexion period, whilst the CAL and PC groups' slopes are much more similar to the CONT slope than to that of the PROT group (Table 3.4). However, Table 3.4 shows that the male PROT infants have a slope that is comparable with the other non-CONT groups, and it is probable that the female PROT trajectory is unusual i.e. that growth after the inflexion is in fact similar in all dietary groups. This ties in well with the observation in Chapter 2 that post-inflexion growth is not significantly influenced by pre-inflexion growth, and that the slope of the post-inflexion RMA is relatively constant across different individuals.

A similar trend is seen in the male *C. apella* groups (Fig 3.11b) Growth in the linear-allometry non-CONT *C. apella* groups is similar to the post-inflexion growth of the *C. apella* CONT group (Table 3.5). As a result of body growth faltering early in growth, the non-CONT infants are highly encephalised at smaller body sizes. At a larger body size, on the other hand, the relative brain size of the non-CONT groups is similar to the relative brain size of the CONT infants.

3.5 Summary

In summary, it appears that brain size is protected in the non-CONT groups such that it rarely drops below 80% to 90% of target size. In general, the CAL infants are able to

stabilise their body mass at 50% of male target size, or 70% of female target size. The PROT infants cannot protect relative body mass, and the percent of target size diminishes as the experiment progresses. PC infants show a different trend, in which the lowest relative body mass (45% of target size) is achieved in the first, not the last, month of the experiment i.e. PC infants suffer acute but not chronic mass loss. Larger infants fare worse on limited calories or protein than do smaller infants, but when the nutritional stress is removed, catch-up growth occurs to differing degrees in different groups.

Whilst capuchin body growth is significantly diminished under both calorie- and protein-deficient diets, brain growth is protected to the extent that very few significant differences in brain size are found between the deficient and non-deficient dietary groups during the experimental period. Furthermore, the differential response of brain and body size to nutritional stress underlines the importance of maintaining normal patterns of brain growth during development. In this analysis the most nutritionally-deprived infants, the PC animals, show a minimal disruption of brain growth (attainment of 80% of target size for age), even though their bodies are less than half the normal size for their age in the first month of the experiment. The relative severity of the diet is therefore associated with relative encephalisation, at least in the early part of postnatal growth.

The position of the inflexion is influenced by brain rather than body size: infants that are small-bodied before the inflexion will be small-bodied after the inflexion (Fig 3.11). If weaning coincides with the position of the inflexion in the non-CONT groups, these infants will be small-bodied as weanlings. This will have important consequences for survival in more naturalistic environments (Janson & van Schaik, 1993; Lee, 1996). The problems of being a small-bodied weanling will be further compounded by the higher energetic demands of a relatively large brain. Unfortunately, specific weaning data for the HSPH infants does not exist, and it is impossible to assess whether the inflexion of the bran allometry and weaning mass have also become dissociated in the non-CONT infants.

After the inflexion of the allometric curve, growth trajectories are similar in all groups, supporting the observation of earlier chapters that post-inflexion rates of brain

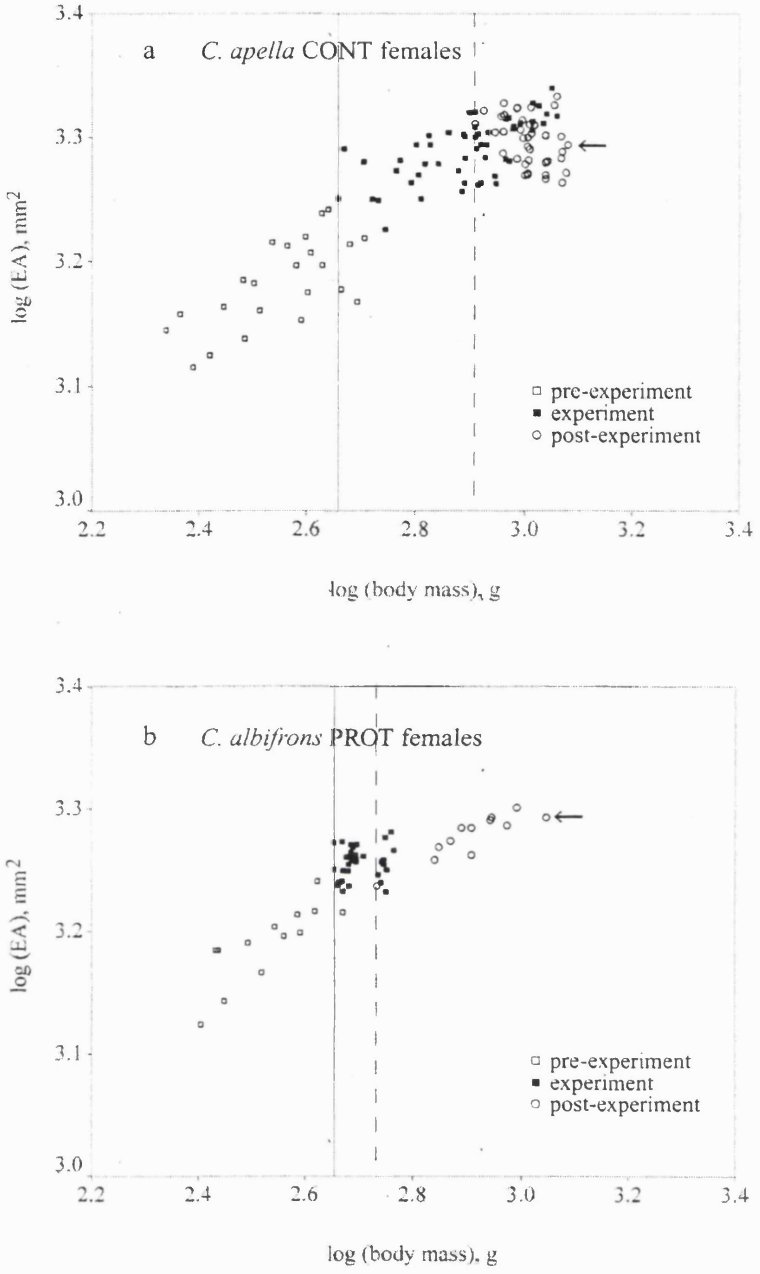


Figure 3.12. Non-CONT infants show catch-up growth in the post-experiment period. Age information superimposed on the brain allometries of female from (a) the CONT and (b) PROT groups. Open squares represent pre-experiment data (i.e. younger than 2 months postpartum), closed squares represent data from the experimental period (2 – 8 months postpartum), and open circles represent data taken from the end of the experiment until the end of the first year of life. The beginning of the experimental period is marked with a solid vertical line, the first datapoint of the post-experimental period is marked with a dashed vertical line. The largest body mass achieved by one year postpartum is arrowed.

growth are relatively invariable across individuals and species. Although infants from the non-CONT groups appear to follow the same allometry as the CONT groups, they have achieved different body masses by the end of the experimental period (Fig 3.12). The first experimental datapoint occurs at a similar body mass in both groups, but the position of the first datapoint of the post-experimental period is very different across the two groups. Once the nutritional stress has been removed, the PROT infants are able to catch up with the CONT group, and by the end of the first year of life, body mass is again similar in the two groups (largest body mass attained by 12 months indicated by arrows in Figures 3.12a and b).*

In conclusion, the results of this chapter suggest that brain and body growth can indeed be dissociated during ontogeny. The artefact hypothesis, which stated that brain growth and weaning are associated only because both brain growth and body growth are associated, is unlikely to be true. Brain growth is protected in the face of nutritional stress, and the position of the allometry inflexion is associated with brain, rather than body, growth. The degree to which undernutrition is present in wild primate populations is uncertain (Altmann & Alberts, 1987; Leigh, 1994). It is possible that the under- and malnutrition of the HSPH non-CONT infants has disrupted the normal pattern of brain and body growth, and that growth in these infants is unrepresentative of growth in healthy individuals. However, the results of these analyses are likely to represent extremes of growth that would be observed in wild-living capuchins, and illustrate the plasticity of ontogenetic processes under stressful growth conditions.

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*The concept of nutritional programming, whereby nutrition early in life influences later size and disease status (Waterland & Garza, 1999) is of relevance here. The effects of uterine and early post-natal nutritional stress on later development and health has been noted in humans, non-human primates, and non-primates (see Lucas, 1991). The effect has been noted in both brain growth/size and body composition, and suggests that over- or underfeeding in a "critical period" (Lorenz, 1970) may have non-immediate consequences for growth. The mechanism by which such metabolic imprinting occurs is debated (Waterland & Garza, 1999). The capuchins in this study appear to show catch-up growth in the early stages of the post-experimental period. How their long term size or health was affected by the nutritional deficiency is unclear from the data available.

CHAPTER 4

BEHAVIOURAL AND MORPHOLOGICAL CORRELATES OF BRAIN GROWTH AND WEANING

In Chapter 3 it was shown that brain and body growth can be dissociated under nutritional stress. This suggests that brain size and weaning are not linked simply because body size and weaning are linked; rather, brain growth follows its own relatively fixed course over development, and body growth is more labile. This chapter presents the results of an analysis that examines the interrelationships of brain growth, body growth, and the maternal and infant behaviours that comprise the weaning repertoire. The analysis tracks the development of specific weaning behaviours, integrating the control group (CONT) growth curves described in Chapters 2 and 3 with behavioural, morphological and other data taken from the literature. In their discussion of capuchin foraging efficiency, Fragaszy & Adams-Curtis (1997) neatly summarise the equal importance of both behavioural and morphological development in determining the timing and pattern of weaning:

> "It is difficult to disentangle the contributions of neural maturation, sensorimotor development, cognitive development, and physical growth to increasing foraging efficiency in natural settings... Moreover, they affect one another reciprocally and dynamically."
>
> Fragaszy & Adams-Curtis (1997:202)

The same is likely to be true of the weaning process. This chapter seeks to disentangle the different behavioural elements of weaning, and asks the question: *why* are brain growth and weaning linked, and are there aspects of weaning that are specifically associated with brain, rather than body, growth? In the light of Chapter 2's results, we might predict that parameters directly associated with nutritional independence (such as nursing/feeding behaviours) might be closely associated with brain growth. Other parameters that measure

more general morphological maturity (e.g. locomotor independence) might be closely associated with body growth. This chapter therefore tests four hypotheses:

1] Changes in infant nursing/feeding behaviours (e.g. suckling frequency) are associated with the pattern and tempo of brain, rather than body, growth.

2] Changes in infant behavioural independence (e.g. level of maternal contact) are associated with body, rather than brain, growth.

3] Changes in infant locomotor independence (e.g. frequency of leaves and approaches from/to the mother) are associated with body, rather than brain, growth.

4] Changes in morphological development (e.g. body proportions) are associated with body, rather than brain, growth.

4.1 Behavioural ontogeny in the context of brain and body growth

4.1.1 Materials and methods

The brain size (endocranial area, EA) and body mass data presented in Chapter 2 were integrated with data taken from the literature, described below. Body mass is inclusive of brain mass. Data were gathered on a variety of behaviours, and comprise quantitative and qualitative descriptions of behavioural ontogeny in capuchins (Table 4.1). In most sources, estimates of behavioural development are presented in terms of activity budgets (i.e. as percent of time observed). Measures of variation in these estimates (e.g. standard deviation) are rarely indicated in the original sources, and it was not possible to report error in the following analyses.

The behavioural dataset used in this analysis describes the behaviours of both male and female *C. apella* and *C. albifrons*. Sex- and species-combined averages were therefore used in constructing brain and body growth curves. As in Chapter 3, data are divided into month or, for two variables (time spent nursing (NUR) and duration of nursing bout (DNB)), into week intervals. Brain and body size are described in terms of percent of adult

TABLE 4.1 Data used in the behaviour analysis: summary of data sources and type

Source	Species	No. of social groups & ~~individuals~~ infants (m:f)	Housing	Sampling procedure	Interobserver reliability
Byrne & Suomi (1995)	*C. apella*	7 (13:4)	1 group housed in an indoor/outdoor run (5 x 6 x 5m); 1 group in similar run in winter, and in an outdoor 'crib' (4.2 x 5m) in summer; 5 groups in indoor cages (0.9 x 1.8 x 1.7m)	Data recorded on 13 separate days; 3 hours of 1-min scan samples performed each day at 11h00, 13h00 and 15h00. Also, 10-min videos recorded 3 times per week for 13 infants up to the age of 13 months plus one other infant up to 11 months.	0.88 – 0.90
Elias (1977)	*C. albifrons*	1 (3:5)	As described in Chapter 2	10-min sessions once weekly.	0.8 – 1.00
Fleagle dataset, e.g. Jungers & Fleagle (1980)	*C. albifrons & C. apella*	26 (18:8)	As described in Chapter 2	Data recorded from x-rays, and from live animals	
Fragaszy (1989)	*C. apella*	2 (2:0)	Two-room pens (8-38 m^3 each)	*Ad hoc* recording of behaviour	Data not shown
Fragaszy (1990)	*C. apella*	1 (various)	Two-room pens (8-38 m^3 each)	Focal animal sampling in 15-min sessions between 07h00 and 18h00.	0.85 – 0.90
Fragaszy et al. (1991)	*C. apella*	1 (various)	Two-room pens (8-38 m^3 each)	Focal animal sampling in 15-min sessions between 07h00 and 18h00.	0.85 – 0.90
Smith et al. (1994)	*C. apella*	Data compendium			

size achieved i.e. are relative rather than absolute measures; this method was chosen to produce a suitable scale against which the growth and behaviour data could be compared. The species- and sex-combined mean EA at 3 years old was used as a measure of adult brain size (2175.59mm^2, n = 5). Adult body mass was calculated as the sex-combined average of mean values given in Smith & Jungers (1997) and in Table 2.2 of Chapter 2 (= 2190.00g).

In the figures that follow, the vertical 'size' and behavioural development axis runs from zero to one hundred percent (Fig 4.1). The solid line represents the percent of adult brain size achieved in each month of the first year postpartum, and the dashed line indicates the percent of adult body size achieved in each month of the first year postpartum. The brain and body curves are similar in shape to the ones seen in previous chapters. It can be seen from Figure 4.1 that the 'average' capuchin has a body size that is 10% of adult size in month 1, and a brain that is 60% of adult size in month 1.

Twenty-two behavioural and physiological variables were investigated, which are divided into 5 groups:

a] Nursing/feeding behaviour

1] NURTime spent nursing.

> Data taken from Fragaszy (1989) and Byrne & Suomi (1995); expressed as % of month 1or week 1 value. Original data are percentages of time observed. 'Nursing' defined as "[infant] on mother's ventrum, with head orientated towards nipple. Scored in favour of 'sleep' [defined as "eyes closed for ≤ 1 minute, body generally still, although twitching of the limbs and tail may occur"] when ambiguous... minimum duration at 3 seconds" (Fragaszy, 1989:143).

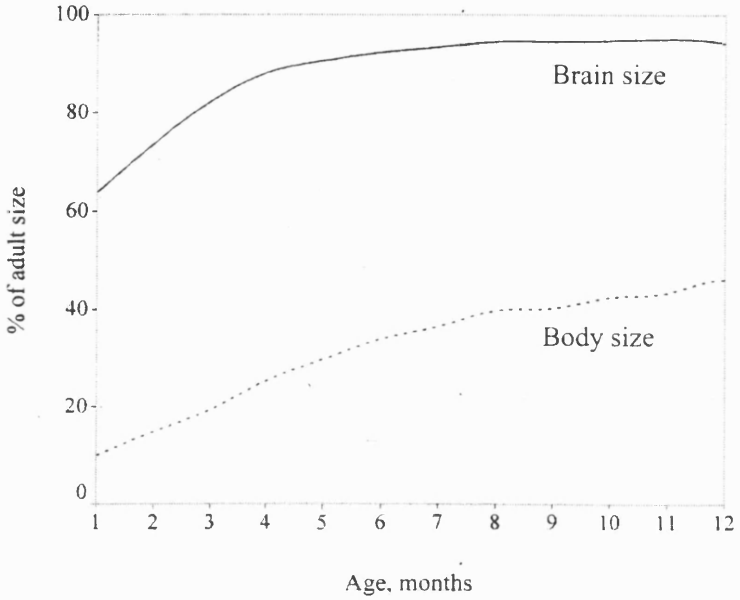


Figure 4.1. Normative capuchin brain and body curves over the first year of life. Data expressed as percentage of adult body size; solid line indicates brain growth, dashed line indicates body growth.

2] DNB Duration of nursing bout.

Data taken from Fragaszy (1989) and Byrne & Suomi (1995); expressed as % of week 1 value. Original data are mean duration of bout in minutes; bout estimated from scan sample data as "number of consecutive 1-minute scans scored in the same state [i.e. scored as 'nursing']." (Byrne & Suomi 1995:257).

3] IBF1 Investigatory bouts towards food 1.

Data taken from Byrne & Suomi (1995); expressed as % of adult value. Original data are percentages of time observed. 'Investigatory bout' defined in Fragaszy (1989): "a 3-second rule was used to determine the termination of bouts: acts occurring within 3 sec of each other were considered to have occurred in the same bout" (Fragaszy 1989:144). 'Food' defined as monkey chow (Byrne & Suomi 1995). Adult value calculated from Visalberghi & Fragaszy (1995), who record investigatory behaviour in adults presented with novel foods: they report 10 acts of sniffing/licking/holding an item of food in 5 minutes of observation. They do not report actual duration of bouts, but if it is assumed each act lasts about 5 seconds, then approximately one sixth (15%) of observed time is spent investigating food. However, this is likely to be an overestimation of adult values, as novel foods are not as frequently encountered in non-experimental situations.

4] IBF2 Investigatory bouts towards food 2 (IBF2).

Data taken from Byrne & Suomi (1995); expressed as % of month 12 value. Original data are percentages of time observed. 'Investigatory bout' defined as above.

b] Behavioural Independence

1] VC Ventral contact.

Data taken from Byrne & Suomi (1995); expressed as % of month 1 value. Original data are percentages of time observed. 'Ventral contact' defined as "[infant] ventral on mother" (Byrne & Suomi 1995:257).

2] DC Dorsal contact.

Data taken from Byrne & Suomi (1995); expressed as % of month 1 value. Original data are percentages of time observed. 'Dorsal contact' defined as "[infant] dorsal on mother" (Byrne & Suomi 1995:257).

3] TSM Time spent interacting with mother.

Data taken from Byrne & Suomi (1995); original data are percentages of time observed, as presented here. Defined as time spent in dorsal, ventral or other contact with mother, plus time spent grooming, nursing or in proximity with mother; not mutually exclusive with TSO below.

4] TSO Time spent in contact with others (i.e. not mother).

Data taken from Byrne & Suomi (1995); original data are percentages of time observed, as presented here. Defined as time spent in proximity to or in contact with others, plus time spent grooming or playing with others; not mutually exclusive with TSM above.

5] GRM Time spent grooming.

Data taken from Byrne & Suomi (1995); original data are percentages of time observed. Grooming in capuchins is associated with the redirection of aggression, usually between individuals of differing rank, and the reinforcement of affiliative interactions and social bonds. Grooming itself is sometimes used as a pretext for other females to investigate new infants (see O'Brien & Robinson, 1991 for discussion). The percent of time devoted to grooming was only around 1% of observed time, and was not found to vary with age. This variable is not, therefore, considered further in the analysis.

c] Locomotor independence

1] IL Initiation of leaves.

Data taken from Byrne & Suomi (1995); expressed as percentage of infant's month 12 value. Original data are mean frequencies per 10 minutes. Represents the number of leaves (breaks of contact) away from the other instigated by either the mother or the infant, relative to the number of leaves instigated by the infant at age 12 months.

2] IA Initiation of approaches.

Data taken from Byrne & Suomi (1995); expressed as percentage of infant's month 12 value. Original data are mean frequencies per 10 minutes. Represents the number of approaches (initiation of contact) towards the other instigated by either the mother or the infant, relative to the number of approaches instigated by the infant at age 12 months.

3] AI Approach index.

Values of the approach index are taken from Byrne & Suomi (1995) and Fragaszy *et al.* (1991), and the methodology of both sources is based on Hinde (1974). The index measures the shift in responsibility for proximity from mother to infant. It ranges from 100 to –100, and is calculated by subtracting the percentage of all leaves initiated by the infant from the percentage of all approaches initiated by the infant. The index is therefore positive if the infant is responsible for maintaining proximity to the mother, and negative if the mother is responsible.

d] Limb/body proportions

Variables 1] to 6] in this group are measured from the same Harvard School of Public Health (HSPH) x-rays described in Chapters 2 and 3. Variables 7] and 8] originate from data collected from the same animals at each x-ray examination. Unlike the other variables in this analysis (with the exception of the skeletal and manipulation scores, below), the parameters included in this group are therefore taken from a subset of the same individuals from which the brain and body growth data are taken. Data come from a total of 26 *C. albifrons* and *C. apella* individuals, all belonging to the CONT diet group (see Chapter 3

for details). All length measurements were taken on the right side of the body. See Jungers & Fleagle (1980) for details of measurement error. Species- and sex-combined means were calculated for each variable for each month of the first year postpartum, and expressed as a percentage of adult value (species- and sex-combined means of all ages older than 1500dpp).

1] FOOT Foot length.

Maximum length from distal end of the calcaneus to tip of third digit along the main axis of the foot.

2] TIB Tibia length.

Maximum length from proximal epiphysis to distal tip of medial malleolus.

3] FEM Femur length.

Maximum length from most proximal point on head to distal condyle along the main axis of the femur.

4] HAND Hand length.

Maximum length from proximal point of scaphoid to distal tip of third digit.

5] RAD Radius length.

Maximum length from most proximal point on the head to tip of styloid process.

6] HUM Humerus length.

Maximum length from top of the proximal epiphysis to the end of the distal epiphysis.

7] CRL Crown-rump length.

Vertex to the most caudal point on the buttocks over the ischial tuberosities (Fleagle & Samonds, 1975).

8] CHC Chest circumference.

Measured just below the nipples (Fleagle & Samonds, 1975).

e] Maturity scores

As a more general test of the association of maturity and growth, four maturity scores that measure different aspects of development were developed from data in the literature. These summarise the ontogeny of behavioural and locomotor independence, the maturation of the skeleton, the development of manipulation, and dental eruption. Significant landmarks in the development of each system were arranged in order of average age at first appearance and assigned a score of 10 points per event (except in the case of the skeletal score, where one point was awarded for each landmark achieved). Landmarks were not weighted. Each month interval was assessed for number of landmarks achieved, and an appropriate score awarded (Table 4.2). For example, in the independence score below, infants aged two months old have been off their mother for the first time (landmark 1), been separated from her (landmark 2) and also spent some time alone (landmark 3); they therefore score 30 in this particular system. Finally, each score was converted to a percentage of the total score (100% = full independence or full maturation of the system in question).

1] IS Independence score.

Data taken from Byrne & Suomi (1995) and Fragaszy *et al.* (1991). Based on Martin's (1984) observation that weaning represents a transition from complete dependence to independence. A score of 100% indicates locomotor independence from the mother, assuming an infant that is away from the mother more than 70% of the time is independent in this respect. Other landmarks were chosen to represent the development of independent locomotor behaviour: off mother for first time, physical separation from mother, infant away from mother for up to 50% of time, infant away from mother between 50 and 70% of time, infant away from mother more than 70% of time. Total score: 50.

2] SS Skeletal score.

Data taken from the skeletal dataset described in section d] above. Scored according to the skeletal maturation scale devised by Accatino & Fleagle (Accatino & Fleagle, in preparation). Scores are awarded for appearance and growth of 51 centres of ossification in the limbs, trunk, neck and tail, but not the cranium. Scores not weighted in the original atlas. Total score: 172.

3] MS Manipulation score.

Data taken from Elias (1977) and Fragaszy (Fragaszy, 1990). Landmarks chosen to represent the development of individual exploration and manipulation of objects, from independent approach, touch, grasp, swing object to the appearance of a precision grip and the use of strength in manipulation (i.e.

TABLE 4.2 Maturity scores and their landmarks[1]

Maturation score	Landmarks	Score	Mean average age of first appearance (months)
1] Independence score (IS)			
1	Off mother for first time	10	1.65
2	Physical separation from mother	20	1.85
3	Infant away from mother up to 50% of time	30	1.88
4	Infant away from mother between 50% and 70% of time	40	5.53
5	Infant away from mother more than 70% of time	50	5.75
	Total	50	
2] Skeletal score (SS)			
	See Accatino & Fleagle (in prep.) for scoring system.		
	Total	172	
3] Manipulation score (MS)			
1	Independent approach to object	10	2.63
2	Touch object	20	3.03
3	Grasp object	30	3.25
4	Swing object	40	3.50
5	Precision grip	50	3.75
6	Strength and precision e.g. smacking, banging objects	60	6.25
	Total	60	
4] Dental score (DS)			
1	di1 eruption	10	0.00
2	di2 eruption	20	0.00
3	dp2 eruption	30	2.60
4	dc eruption	40	2.92
5	dp3 eruption	50	3.49
6	dp4 eruption	60	5.12
7	M1 eruption	70	16.68
8	I1 eruption	80	18.25
9	I2 eruption	90	20.35
10	M2 eruption	100	29.72
11	M3 eruption	110	-
12	P2 eruption	120	-
13	P3 eruption	130	-
14	P4 eruption	140	-
15	C eruption	150	-
	Total	150	

[1]See text for data origin.

smacking, banging objects, both of which require force to achieve). Total score: 60.

4] DS Dental score

Data taken from the dental compendium of Smith *et al.* (1994). A dental score of 100% indicates eruption of complete adult dentition. The species for which the most data are available in this compendium is *C. apella*, and these are the ages used to construct the dental score; unfortunately, no data are available for the eruption of the third molar, the permanent premolars or the permanent canine. When compared with *C. apella*, the *C. albifrons* data in Smith *et al.* (1994) indicate delayed eruption of the deciduous premolars (in the order of 10 days or so) and permanent molars (around 33 days and 73 days for lower M1 and M2 eruption). The eruption of each tooth is considered a separate landmark. Total score: 150.

These variables are summarised in Table 4.3. The brain and body size trajectories described in Figure 4.1were superimposed on the various behaviour curves, and visually compared. In addition, correlations between mean brain or body size (month interval means) and the behaviour variables were assessed statistically, holding either brain or body size constant for each analysis as appropriate (partial correlation, 2-tail significance, $P \leq 0.05$).

4.1.2 Results

Compared with other primate taxa, capuchins are behaviourally and morphologically altricial at birth (Fragaszy *et al.*, 1991; Byrne & Suomi, 1995). As we saw in Figure 4.1,

TABLE 4.3 Variables used in the analysis[1]

	Variable	Abbr.	Original data scored as...	Expressed as...
a] Nursing/feeding behaviours				
1	Time spent nursing	NUR	% of time observed	% of month 1 value
2	Duration of nursing bout	DNB	Mean bout duration in minutes	% of month 1 value
3	Investigatory bouts towards food 1	IBF1	% of time observed	% of adult value
4	Investigatory bouts towards food 2	IBF2	% of time observed	% of month 12 value
b] Maternal contact				
1	Ventral contact	VC	% of time observed	% of month 1 value
2	Dorsal contact	DC	% of time observed	% of month 1 value
3	Time spent in contact with mother	TSM	% of time observed	% of time observed
4	Time spent in contact with others (not mother)	TSO	% of time observed	% of time observed
c] Infant locomotor independence				
1	Initiation of leaves	IL	Mean frequency per 10 minutes	% of infant's month 12 value
2	Initiation of approaches	IA	Mean frequency per 10 minutes	% of infant's month 12 value
3	Approach index	AI	(% approaches made by infant) – (% leaves made by infant)	Age at which index changes from negative to positive
d] Limb/body proportions				
1	Foot length	FOOT	Millimetres	% of adult value
2	Tibia length	TIB	Millimetres	% of adult value
3	Femur length	FEM	Millimetres	% of adult value
4	Hand length	HAND	Millimetres	% of adult value
5	Radius length	RAD	Millimetres	% of adult value
6	Humerus length	HUM	Millimetres	% of adult value
7	Crown-rump length	CRL	Millimetres	% of adult value
8	Chest circumference	CHC	Millimetres	% of adult value
e] Maturation scores				
1	Independence score	IS		See text for explanation
2	Skeletal score	SS		See text for explanation
3	Manipulation score	MS		See text for explanation
4	Dental score	DS		See text for explanation

[1]See text for explanation.

only 60% of adult brain size has been achieved by birth in the HSPH infants, and some authors (e.g. Elias, 1977) place this figure as low as 37%. *Cebus* infants develop more slowly than other taxa such as *Saimiri* and *Macaca*, with a prolonged and gradual transition to independent foraging (Boinksi & Fragaszy, 1989). They also show an extended period of maternal dependency, and delayed attainment of other life history variables, e.g. age at first reproduction (see Chapter 6).

a] Nursing/feeding behaviour

Most of a capuchin infant's early months are spent sleeping, drowsing or nursing (Byrne & Suomi, 1998). Infants are in contact with, or in proximity to, their mothers for the majority of the first year. Capuchin mothers also maintain physical intimacy with their infants for a far longer period than do squirrel monkey mothers (Boinksi & Fragaszy, 1989). Capuchin mothers are observed to ventrally cradle their infant for extended periods of time well after the infant has become able to support itself posturally (Fragaszy *et al.*, 1991). This intimacy is no doubt an important factor in extending lactation; Fragszy *et al.* (1991:391) observe that, "for capuchins, intimate affiliative behaviours (grooming, resting together) throughout infancy provide a social context in which nursing can take place regularly, albeit infrequently."

1] Time spent nursing (NUR)

The inflexion of the NUR curve occurs between second and fourth month postpartum (Fig 4.2a, marked N). The inflexion of the brain growth curve occurs at approximately the same time, i.e. in the fourth month (Fig 4.2a, marked B). At this age, brain size has reached approximately 80% of adult size, and the time spent nursing has dropped to 32.1% of the month 1 value (suckling for 1.8% of observed time). As the infant's brain becomes even larger, the mother suckles the infant less frequently, and by one year of age, the infants

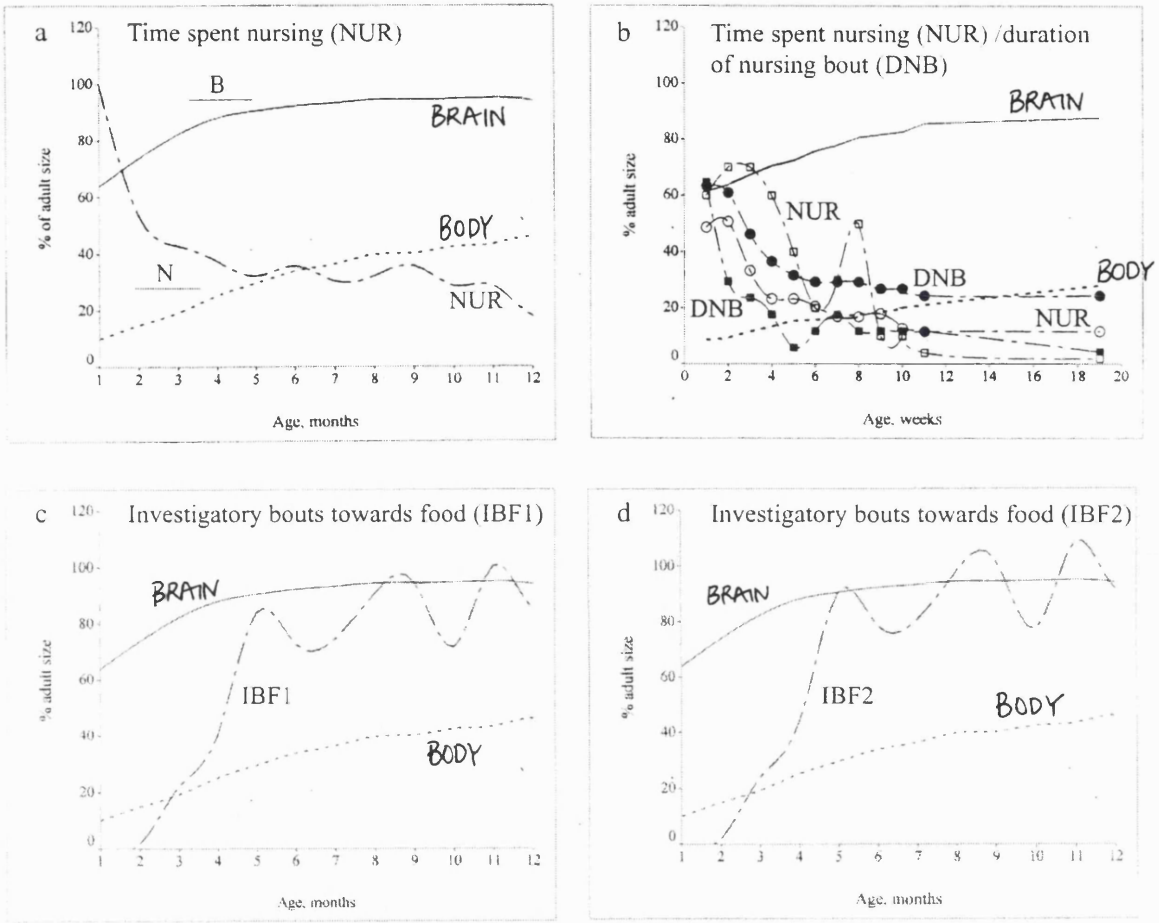


Figure 4.2. Nursing/feeding behavioural ontogeny. (a) Time spent nursing (NUR) during months 1 to 12. N = inflexion of NUR curve; B = inflexion of brain growth curve. (b) Time spent nursing (NUR) and duration of nursing bout (DNB) during weeks 1 to 20. Square datapoints are taken from Fragaszy (1989; n = 2 infants); round datapoints are taken from Byrne & Suomi (1995; n > 14 infants). In both cases, open symbols represent time spent nursing (NUR), and closed symbols represent duration of nursing bout (DNB). (c) Investigatory bouts towards food 1 (IBF1). (d) Investigatory bouts towards food 2 (IBF2). See Fig 4.1 for key.

only suckles for around 1% of observed time (17.8% of the month 1 value). At this age, the infant's body is approximately 40% of adult size.

2] Duration of nursing bout (DNB)

Figure 4.2b also shows how mean time spent nursing (NUR) declines with age, but in this graph, NUR is scaled against weeks. Alongside are data for duration of nursing bout (DNB). Brain and body sizes are re-scaled accordingly. The data are data taken from two sources: Fragaszy (1989), and Byrne & Suomi (1995). All four curves show a steep decline over the first two months postpartum (Fig 4.2b). Fragaszy's (1989) data are more erratic, but by 20 weeks (5 months) postpartum, both NUR samples, and Fragaszy's (1989) DNB sample, have declined to less than 10% of their week 1 value. Byrne & Suomi's (1995) DNB data show a plateau after three months old similar to that of NUR in Figure 4.2a. By 20 weeks postpartum, the typical duration of a suckling bout in this sample is 24.4% of the week 1 value, or 1.00 min in length.

Why is the slowing of brain growth and the reduction of suckling frequency and bout duration so strongly associated? The hypothesis that the energy in milk is important in regulating brain growth is tested in Chapter 5. Other neural growth-promoting factors are also found in milk (e.g. thyroxine: Hartmann *et al.*, 1984; taurine: Sturman *et al.*, 1985). Most interesting and well-studied are the essential fatty acids (EFAs) linoleic acid (LA) and α-linolenic acid (LNA), and their long-chain polyunsaturated fatty acids (LCPUFAs) derivatives, docosahexanoic (or docohexanoic) acid (DHA) and arachidonic acid (AA). Both have been shown to be important in promoting brain growth, although AA appears to be more closely associated with the regulation of whole brain mass (as measured by head circumference; Crawford, 1993; Lanting & Boersma, 1996; Clandinin & Jumpsen, 1997; Brody, 1999; Clandinin, 1999). Both are essential to the structural integrity of lipid-rich neuronal membranes (Birch *et al.*, 1997), as well as to the growth and function of neural connections, especially synaptogenesis and myelination (Crawford, 1993). The

mechanisms by which these structural lipids modify neural development and function are still largely unclear (Burdge, 1998 but see; Carlson & Neuringer, 1999; Fernstrom, 1999). Human infants are only able to synthesis their own DHA and AA late in lactation (Henderson *et al.*, 1996), and all LCPUFA needs are supplied at first via the placenta, then from milk (Birch *et al.*, 1997).

In addition to the association of AA levels and prenatal brain growth, a highly significant correlation exists between the AA:DHA ratio in milk and change in head circumference between birth and 3 months postpartum, Xiang *et al.* (2000). Human infants fed a diet rich in vegetable oils (i.e. lacking LCPUFAs) have been shown to have lower levels of omega-3 and omega-6 metabolites in the brain, and impaired CNS development (Heird & Gomez, 1996). Research in macaques suggests that infants born to mothers deprived of DHA in pregnancy and lactation suffer brain dysfunction during development (Neuringer *et al.*, 1996). On the other hand, human and non-human studies find that even severe, long term omega-3 deficiency does not cause deficits in general learning or memory (Carlson & Neuringer, 1999). This evidence points to the fact that LCPUFAs are integral to normal patterns of CNS development, at least in the short term. Milk provides EFAs, the building blocks of the LCPUFAs as the infant continues to increase in brain mass after birth. In those species that are relatively altricial (e.g. *Cebus*, see above), the milk must supply large amounts of EFAs to support continued brain growth (Hall & Oxberry, 1977). In *Cebus*, the cessation of milk fatty acid transfer coincides with the achievement of 75% of adult brain size, and with the physiological competence needed to metabolise fatty acids from the weanling diet. The implications of this association for humans and capuchins are discussed in Chapter 7.

3] Investigatory bouts towards food (1BF1 and IBF2)

How important to infant capuchin development are food-exploring behaviours? It has been suggested that infant neophobia towards food, coupled with a readier acceptance of food

previously tested by a conspecific, is a mechanism by which exposure to dangerous foods is limited and a homogenous diet established (Galef, 1993). Observations of capuchins, however, show that many infants investigate (and eat) novel foods in preference to familiar ones (Fragaszy *et al.*, 1997b). This is also true of adults (Visalberghi & Fragaszy, 1995), whose diets are extremely varied. Investigatory behaviour by infants in fact plays an important part in developing dietary competence and foraging efficiency (Fragaszy *et al.*, 1997b). This contrasts with *Saimiri* infants, which are self-sufficient foragers within 8 to 10 weeks of the onset of independent locomotion (Boinksi & Fragaszy, 1989).

Individual exploration of food is vital for capuchin infants, as social transferral of information about food from other members of the group (Fragaszy *et al.*, 1997b) does not appear to be as widespread in capuchins as it is in some other taxa, e.g. gorillas (Watts, 1985). Food sharing (transferral) between non-infants and infants is also tolerated by capuchins, and frequently observed in captivity (de Waal *et al.*, 1993; Fragaszy *et al.*, 1997a). Whilst food transferral fulfils both nutritive and social functions in other wild primates, and is largely kin-based (e.g. callitrichids and chimpanzees, Feistner & McGrew, 1989), capuchin food transferral appears to be related to the age of the infant and the difficulty with which a particular food is processed. In their study of captive *C. apella*, Fragaszy *et al.* (1997a) found that attempts at food acquisition by capuchin infants were always directed at adults, never to other infants. Furthermore, older infants (18 months or older) were less likely to attempt to acquire food than were younger infants. Fragaszy *et al.* (Fragaszy *et al.*, 1997a) suggest that this is because smaller infants are less successful in dealing with difficult foods than older, larger ones. Capuchin food transferrals are sometimes voluntary and unsolicited (de Waal *et al.*, 1993), and capuchins are not observed to 'beg' for food as chimpanzees do (Goodall, 1986). In the study of infant reactions to novel foods described above (Fragaszy *et al.*, 1997b), none of the novel foods presented in that study were difficult to extract, and no preference was shown towards adults (compared to efficient-extractor juveniles) in terms of attempted acquisition. In both

studies, the infants appeared more concerned with the food – and with the difficulty in extraction – rather than with whom was eating it.

Investigatory behaviour towards, and sharing of, food is clearly part of the infant capuchin's path towards efficient foraging and acquisition of an adult diet. In light of this, we can predict that these other sources of nutrition will be more frequently investigated as suckling rates decline. Figures 4.2c and d plot the frequency with which food is investigated in relation to adult behaviour and to the behaviour of one-year-old infants. Whether we compare the data against the adult mean for investigating food (IBF1), or the infants' month 12 value (IBF2), a plateau in food investigation occurs at 5 months. One explanation for this increase in investigatory acts towards food may simply be that, as infants get older, they get more curious, and as the brain matures, all sorts of investigative behaviours increase in frequency or consistency. However, Fragaszy & Adams-Curtis (1997) note that capuchin infants (n = 8) "devoted proportionally significantly less time to social activity (manual contact with other animals), and more time to investigating food and browse in the second [six months] than in the first..." (p205). Of all the various exploratory acts scored in their study, only those involving direct manipulation of objects with the mouth (lick/sniff/mouth and bite/chew, out of a possible 13 acts) show a significant increase over all four 6-month intervals in the two-year period of development investigated (with the exception of bite/chew, which showed a significant decline between the third and fourth 6-month interval. Fragaszy & Adams-Curtis (1997) suggest the eruption of permanent teeth in this period (see below) as a factor in this decline. In both these variables, the greatest change in frequency occurs between the first and second six-month intervals: it is at these ages that infants are 'testing' the edibility of all objects, not just food. Food remains the major targets of investigative activity in the months following, as Fragaszy & Adams-Curtis' (1997) data describe: by the end of the first year, infants are directing 35% of all activities towards food or browse; by the end of the second year, this figure has risen to 47%.

Although 'investigatory bout' is not synonymous with food consumption, this result may indicate that the rate at which solid food is eaten also increases significantly at this time, even if efficiency is low. The drop in milk supply seen in Figure 4.2a and b suggests that nutrition must play a part in the infant's growing need for solid food. Wright & Bush (1977) report that solid food was first eaten by their captive capuchins at 100dpp, or about 3.5 months. Lee *et al.* (1991) predict that the ingestion of solid food will first occur when body mass is 2.1 times as heavy as at birth. In this sample of infants, this mass is achieved between 3 and 4 months old (i.e. when body mass reaches 21% of adult size, Fig 4.1). Food investigation begins early in life, at about 3 or 4 months, and assumes increasing importance as the infant grows, reaching a peak of investigatory behaviour at about 5 or 6 months old.

Data describing actual food consumption during lactation period of infant guenons confirms the growing importance of food even in this early stage: as suckling frequencies drop away, food consumption increases (Queirouil & Blois-Heulin, 1998a). As in capuchins, weaning in the similarly-sized guenon is an extended process, complete by about 6 months (Harvey & Clutton-Brock, 1985). Even after six months, infants are both suckling and eating solid food, though the frequency of suckling is low (Fig 4.3). In Querouil & Blois-Heulin's (1998b; 1998a) study, the guenon infants were observed to learn some information about food from other members of the social group (especially their mothers), but much of food choice was based on trial-and-error experience (1998b; 1998a), as it is in capuchins (Fragaszy *et al.*, 1997a). This guenon pattern equates well with the increase in exploration of food seen in the capuchins at similar stages of lactation. This evidence suggests that Fragaszy *et al.*'s (1991:390) view that "capuchin infants clearly do not obtain much nutrition from foraging activities well into the second year of life" may underestimate the importance of testing and tasting new foods in these early stages of postnatal brain growth.

Of all the variables in this group of nursing/feeding behaviours, none are significantly correlated with body size when the effects of brain size are held constant

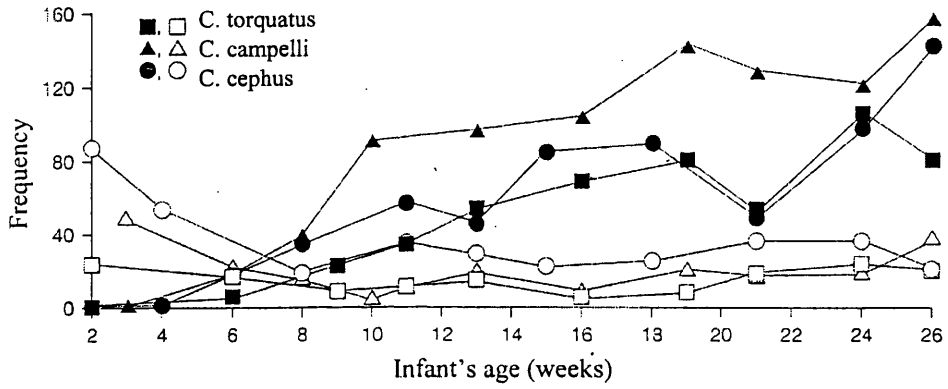


Figure 4.3. Infant guenon food consumption during lactation *(Cercopithecus* spp.). Based on figure in Queirouil & Blois-Heulin (1998a). Open symbols represent suckling, closed symbols represent eating (see Queirouil & Blois-Heulin, 1998a for definition of terms).

TABLE 4.4 Partial correlations between brain size, body size and behavioural /physiological variables

Variable	Correlation with brain size, when body size held constant df	R	P	Correlation with body size, when brain size held constant df	R	P
a] Nursing behaviours						
NUR*	9	-0.772	**0.005**	9	0.257	0.445
NUR**	9	-0.707	**0.015**	9	0.225	0.507
DNB	9	-0.670	**0.017**	9	0.263	0.434
IBF	9	0.415	0.205	9	0.546	0.082
b] Maternal contact						
VC	9	-0.929	**0.000**	9	0.593	0.055
DC	9	0.405	0.216	9	-0.906	**0.000**
TSM	9	-0.546	0.082	9	-0.892	**0.000**
TSO	9	-0.433	0.183	9	0.238	0.482
c] Locomotor independence						
IA_{infant}	9	0.543	0.084	9	0.667	**0.025**
IA_{mother}	9	0.932	**0.000**	9	-0.858	**0.001**
IL_{infant}	9	0.745	**0.009**	9	-0.202	0.551
IL_{mother}	9	0.731	**0.011**	9	0.323	0.333
d] Limb/body proportions						
FOOT	9	0.976	**0.000**	9	0.994	**0.000**
TIB	9	0.536	0.089	9	0.9923	**0.000**
FEM	9	0.432	0.184	9	0.987	**0.000**
HAND	9	0.973	**0.000**	9	0.993	**0.000**
RAD	9	0.153	0.654	9	0.988	**0.000**
HUM	9	0.678	**0.022**	9	0.990	**0.000**
CHC	9	0.618	**0.043**	9	0.854	**0.001**
CRL	9	0.938	**0.000**	9	0.989	**0.000**
e] Maturation scores						
IS	9	0.310	0.355	9	0.421	0.197
MS	9	0.585	0.059	9	0.441	0.175
SS	9	0.597	0.053	9	0.986	**0.000**
SS***	32	0.534	**0.001**	9	0.836	**0.000**
DS	9	0.793	**0.004**	9	0.340	0.306
DS***	32	-0.004	0.983	9	0.755	**0.000**

Data grouped into month intervals, mean values for each month used in the partial correlation. All correlations are for the first 12 months postpartum, unless otherwise indicated. Significant correlations are shown in bold type.

*Data from Byrne & Suomi (1995), for months 1-12

**Data from for Byrne & Suomi (1995), for weeks 1-19

***Months 1- 36

(Table 4.4), although IBF almost reaches significance. On the other hand, all nursing variables except IBF are strongly and negatively correlated with brain size over 12 months, when body size is controlled for (Table 4.4). Over both the long (one year) and the short (19 weeks) term, increases in brain size are significantly associated with a drop-off in suckling frequency and duration, and strongly (if not quite significantly) associated with an increase in investigatory acts towards food.

Of relevance here is the report of allonursing (where an infant nurses from a female other than its own mother) in wild capuchins. In her study of white-faced capuchins (*C. capucinus*), Perry (1996) observes that "the period of 3 – 6 months of age – the period during which infants were first beginning to explore extensively away from their mothers, yet remained dependent on milk for most of their nutritional needs – was the period during which the most allonursing occurred" (p170). Female wedge-capped capuchins (*C. olivaceus*) in Venezuela (O'Brien, 1988) were also observed to suckle from unrelated individuals, but in all of the reported suckler-nurser dyads, the suckler was older than 2 years old and (presumably) weaned. Unlike the suckling bouts described for *C. capucinus*, these were not co-operative, and the nurser usually showed distress during a bout. O'Brien describes these instances of allonursing as "parasitic" and exploitative (1988).

Perry (1996) reports a dominance effect in the *C. capucinus* group, such that the nursing-suckling dyads were arranged around relative rank of the nursing female rather than that of the suckler, or of the suckler's mother. Higher-ranking females nursed less frequently than lower-ranking females. O'Brien (1988) found that the 'exploitative' behaviours in the wedge-capped capuchins were also rank-associated, but in this population sucklers were (in all but one case, total n = 8) dominant to the nurser. O'Brien & Robinson (1991) found the most consistent influence on the pattern of allonursing in their study of *C. olivaceus* was relatedness. These contradictory results suggest that allonursing might fulfil different functions within different capuchin social groups, and these are only partly associated with the nutritive needs of the infant. Perry does, however, note that:

> "because *C. capucinus* infants are often widely separated from their foraging mothers, even in the first few months of life, it is often difficult for infants to locate their mothers when they need to nurse. Therefore, there could be a great advantage to infants in being able to nurse from the closest lactating female."
>
> Perry 1996:179

Allonursing may contribute to the relatively elevated rates of postnatal body growth shown by capuchins, in a manner similar to the smaller callitrichids, where allomaternal care is common and infant growth is relatively rapid (Ross, 1991).

b] Behavioural Independence

1] Ventral and dorsal contact (VC, DC)

Capuchin infants maintain almost constant contact with their mothers until the second or third month, when they begin to explore independently. As we might expect, the sum of the initial values of time spent in ventral (VC) and dorsal (DC) contact is close to 100% (17.9% for VC plus 81.2% for DC = 99.1%). Both show a drop off in frequency similar to that of nursing above (Fig 4.4a and b). It should also be remembered that 'nursing' and 'ventral contact' are likely to have been hard to distinguish in the observational studies, and may not be mutually exclusive. Infants are spending as little time in ventral contact at 4 or 5 months old (around 3%) as when they are one year old (Fig 4.4a). Wright & Bush (1977) also report that infants are carried by their mothers until about 140dpp, or 5 months old. Data from a Venezuelan population of *C. olivaceus* (O'Brien & Robinson, 1991) confirm that the development of independent locomotion follows a similar timescale in the wild. This pattern of VC decline is the inverse of brain growth, and the two are significantly correlated when body size is controlled for ($R = -0.929$, $P = 0.000$).

Time spent in dorsal contact (Fig 4.4b) shows a slower rate of change than ventral contact; after the first month, there is a more or less linear drop off in DC until 11 or 12 months old, when dorsal contact is virtually nil. Change in body size has an effect on both ventral and dorsal contact (Table 4.4). DC is highly significantly correlated with body size when brain size is held constant ($R = -0.906$, $P = 0.000$), and VC only just misses the significance threshold ($R = 0.593$, $P = 0.055$). Unlike VC, DC is not significantly associated with brain size ($R = 0.405$, $P = 0.216$). Infants are usually carried dorsally (Byrne & Suomi, 1998), and as the infant becomes heavier it presumably becomes more of a burden to the mother.

2] Time spent with mother (TSM)

Time spent with mother (TSM, Fig4.4c) includes all interactions with the mother i.e. ventral and dorsal contact, grooming, nursing etc. As might be expected, it shows a curve very similar to that of dorsal contact, but it is clear that, by one year of age, infants are still spending around 20% of their time interacting with their mothers, though not necessarily in direct physical contact. Time spent with mother is significantly and negatively correlated with body size ($R = -0.892$, $P = 0.000$). The correlation of TSM and brain size almost reaches the significance threshold, also with a negative association ($R = -0.546$, $P = 0.086$). As responsibility for maintaining contact shifts quite soon in development to the infant (see approach index, below), this implies that the mother's response to the infant's attempts at contact is influenced by changing size of the whole infant, not just its brain.

3] Time spent with others (TSO)

In comparison with TSM, time spent in contact with others (TSO) does not vary much over the first year of age; it remains at around the 20% level over all age intervals, with perhaps a little more interest shown in the infant in the first couple of months (Figure 4.4d). Neither

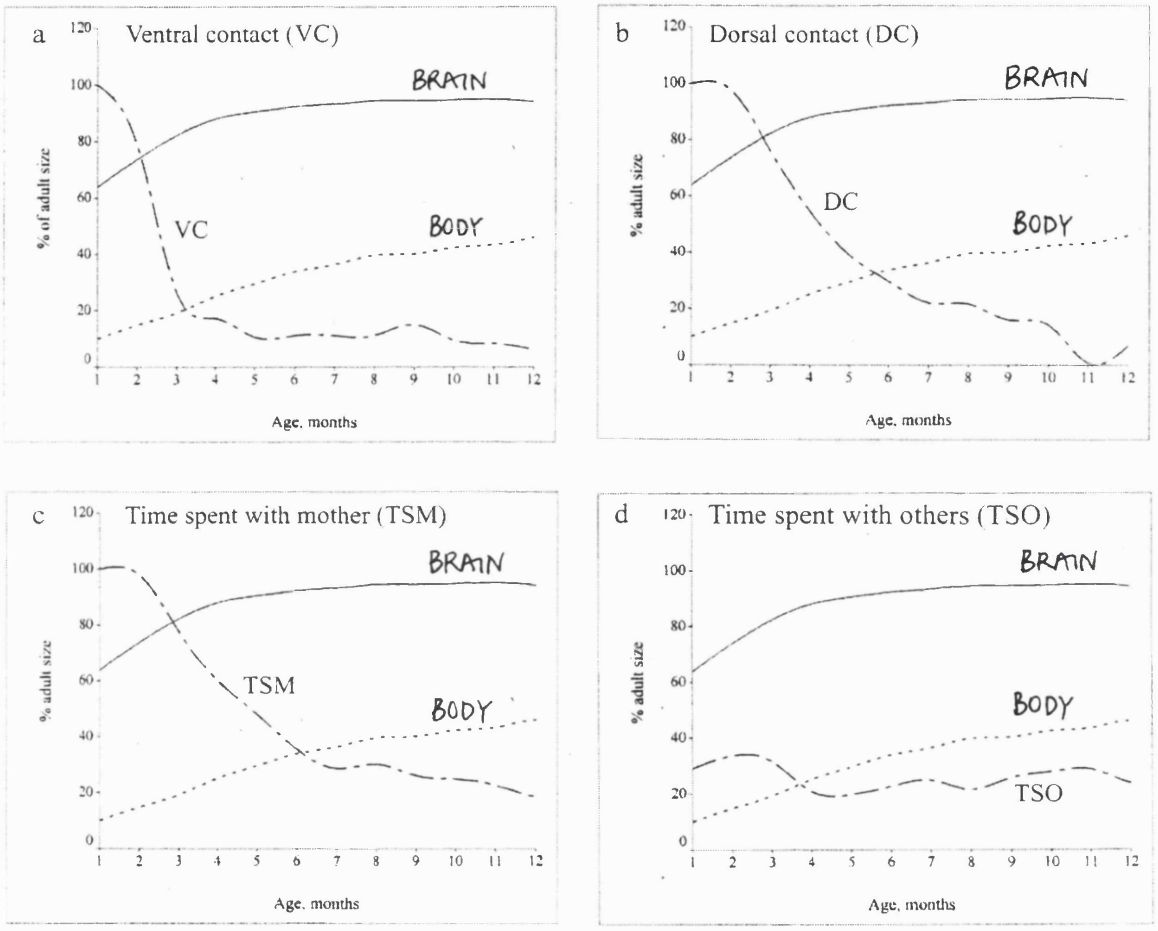


Figure 4.4. The ontogeny of behavioural independence. (a) Ventral contact (VC); (b) dorsal contact (DC); (c) time spent with mother (TSM); (d) time spent with others (TSO). See Fig 4.1 for key.

brain size nor body size reach a significance level of association with TSO (R = -0.433, P = 0.183; R = 0.238, P = 0.482 respectively). As Fragaszy (1989) notes in her report of one young male capuchin:

> The capuchin infant was a frequent recipient of affiliative contacts from individuals other than the mother from the day of birth. All other animals in the group, including the adult male, frequently investigated, groomed and made face-to-face contact (which we described as being very like kissing) with the infant from birth onward.
>
> Fragaszy 1989:149

It appears that capuchin infants are always of interest to other members of the social group. Infant handling is also common in wild capuchins, though it remains relatively unstudied (see Manson, 1999 for recent review). As with the nursing variables above, the greatest change in most of the behaviours observed here occur, for the most part, at the time that infants have essentially achieved adult brain size. Ventral contact, in particular, shows a very fast decline in the first few months postpartum. Other forms of contact with the mother (such as grooming) are slower to decline, and reflect the non-nutritive function of many of the mother-infant interactions over the first year postpartum.

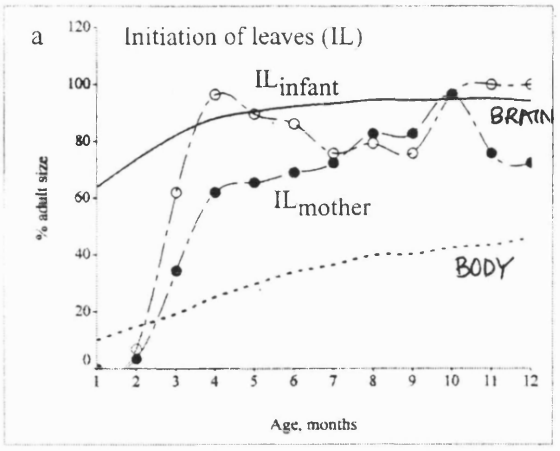
c] Infant locomotor independence

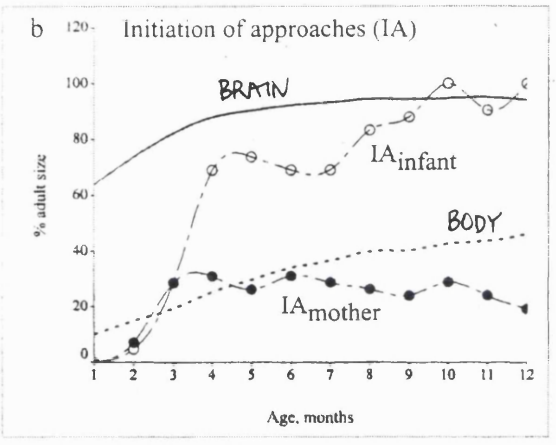
The contact variables above indicate that capuchin infants are moving independently of their mothers by about 4 or 5 months old, but retaining a level of interaction with her throughout the first year. Locomotor independence is achieved by about 3 months old, by which age capuchins have progressed from early crawling and unsteady standing, to walking quadrupedally, standing unsupported, and playing rough-and-tumble with siblings and peers (Elias, 1977; Fragaszy, 1989). Vision has also improved, from being relatively

undeveloped at birth, to accommodating rapid visual movement by about 11 weeks old (Fragaszy, 1990).

1] Initiation of leaves (IL) and approaches (IA)

As the infant begins to explore independently, responsibility for contact shifts from the mother to the infant (Fig 4.5). Both mother (IL_{mother}, IA_{mother}) and infant (IL_{infant}, IA_{infant}) values for leaving and approaching are low in the first few months, because the infant is rarely (if ever) apart from the mother at this stage (Figs 4.4a and b). The frequency with which leaves and approaches occur rises rapidly between 2 and 5 months. Both infant and mother leave each other at about the same rate over the first year such that, by 12 months, both leave the other at the rate of about 2 to 3 times per 10 minutes (maternal initiation is measured here in comparison with infant initiation at 12 months). On the other hand, the frequency with which the mother *approaches* the infant after the third month is much lower in comparison with the infant data. Infants approach their mother at the same rate at which they leave them (i.e. about 2 times per 10 minutes) until 7 months old, after which the rate of approaches increases to about 4 times per 10 minutes by the age of 12 months. In comparison, mothers approach their infants only once every ten minutes, on average, by one year postpartum. This is still a fairly high rate of contact, and contrasts with the results of Fragaszy *et al.*'s (1991) study which found a similar pattern but lower rate of interactions. In that study, the frequency of approaches and leaves initiated by the infant peaked at around 3.5 and 2.5 months old respectively, after which they remained constant at about 3 or 4 times *per hour*. Approaches and leave made by the mother remained a higher proportion of the infant values throughout the study, at around 50% (~2 times per hour). The capuchins observed by Byrne & Suomi (1995) were housed not only in larger cages than those studied by Fragaszy *et al.* (1991), but also in larger groups; this may explain the discrepancy in figures seen here. This possibility is examined in more detail below.





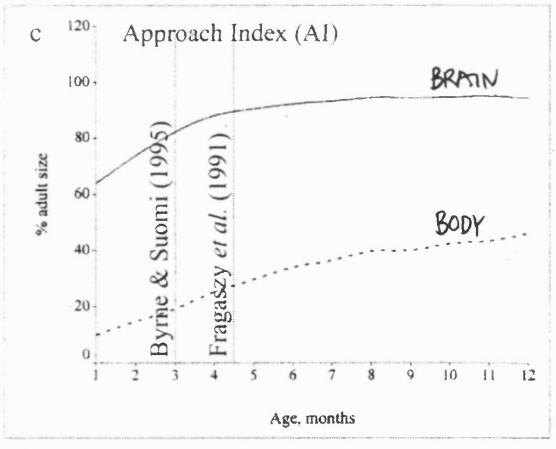


Figure 4.5. The ontogeny of locomotor independence. (a) Initiation of approaches (IA); (b) inititation of leaves (IL); (c) Approach index (AI). In (a) and (b) infant data is indicated by open symbols, maternal data indicated by closed symbols. In (c), the age at which the AI becomes positive (i.e. the infant assumes responsibility for contact; see text) is marked with a vertical line; this event occurs earlier in the Byrne & Suomi (1995) data than in the Fragaszy *et al.* (1991) data. See Fig 4.1 for key.

The initiation of leaves by both the mother and the infant are significantly correlated with brain size when body size is held constant, but not with body size (Table 4.4). For both partners of the mother-infant dyad, the frequency with which physical contact is broken appears to be modulated by the growing brain, rather than change in body size. The initiation of approaches, however, is associated with both brain and body size independently of each other: IA_{mother} is significantly associated with both the brain (P = 0.000) and the body (P = 0.001); IA_{infant} reaches the significance threshold with body size (P = 0.025) but not brain size (P = 0.084; Table 4.4).

2] Approach index (AI)

It is clear that, whatever the frequency of contact, the responsibility for maintenance shifts to the infant fairly early in life. This is shown even more emphatically in Figure 4.5c. Here, the age at which the approach index (AI) switches from negative to positive – i.e., the age at which the percentage of approaches made by the infant is greater than the percentage of leaves it makes – is shown. Both data sources agree that this shift occurs early in lactation, between 3 and 4.5 months postpartum. Once more, the period of rapid change in all these measures of 'independence' coincides with the age at which the majority of adult brain size is achieved (5 months), in synchrony with the achievement of locomotor skills sufficient to support independent movement.

d] Limb/body proportions

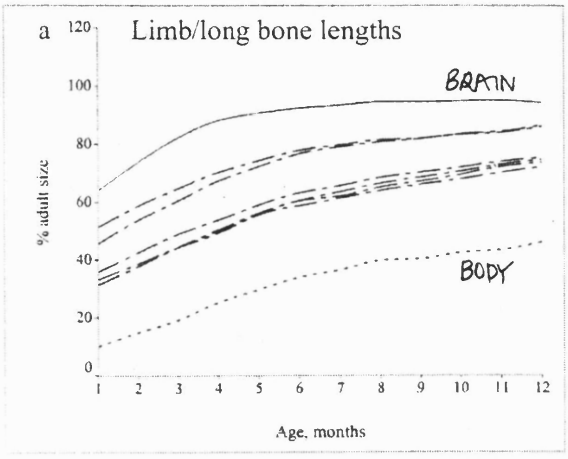
All four limbs are important weight-bearing appendages in quadrupedal animals, and we can predict that in capuchins, both limb and other body segment proportions will track the growth of body mass, rather than bran size. Chest circumference, and to some extent crown-rump length, should be associated with the growth of the internal organs (e.g. the

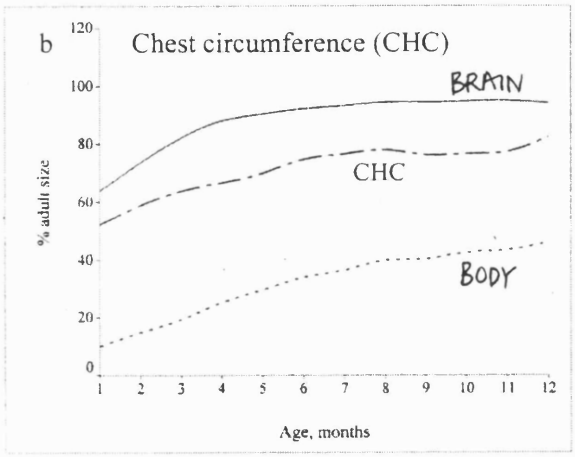
lungs and heart); in other species these are known to follow growth curves similar to the other non-brain tissues (see Chapter 1).

The data presented here shows that limb and body proportions are relatively advanced (compared to adult size) than is body size over all month intervals, but not as advanced as brain size. Furthermore, they show relative maturity that is somewhere between the level of maturity seen in brains and bodies (Fig 4.6). It is interesting to note that, of all the limb segments, it is the distal elements that achieve the greatest proportion of adult size first. Feet (FOOT) and hands (HAND) are (relative to adult size) the biggest at birth, and at around 50% are only slightly smaller than the brain in terms of relative development. This contrasts with Watts' (1990) analysis of wrist bone development in *Cebus*, which finds that capuchin ossification at birth is delayed relative to that of *Macaca* in a pattern more similar to *Homo* and *Pan* development. It must be assumed that digit length and wrist ossification reflect two different aspects of appendage maturity.

In the first five months postpartum, the infants' hand lengths are relatively more developed than their foot lengths; it is during this time that infants spend much of their time clinging in either ventral or dorsal contact (Figs 4.4a and b). This is also the age at which manipulative behaviours develop which involve dexterity and, later, strength, to perform (see below). The humerus (HUM) is also relatively advances compared to the other, more proximal, parts of the fore- and hindlimbs in these months, presumably for much the same reasons. After 5 months, there is little or no difference between the relative size of hands and feet, and this catch-up in maturity of the feet occurs at about the age when the infants are spending much less time clinging, and when quadrupedal locomotion becomes the norm for the infant. By the age at which independent locomotion is achieved, the other limb elements have largely caught up with the humerus in terms of percent of adult size achieved.

As predicted, all limb elements investigated here show a highly significant correlation with body size, once brain size has been controlled for (Table 4.4). Most also show a significant relationship with brain size too; only TIB, RAD and FEM show non-





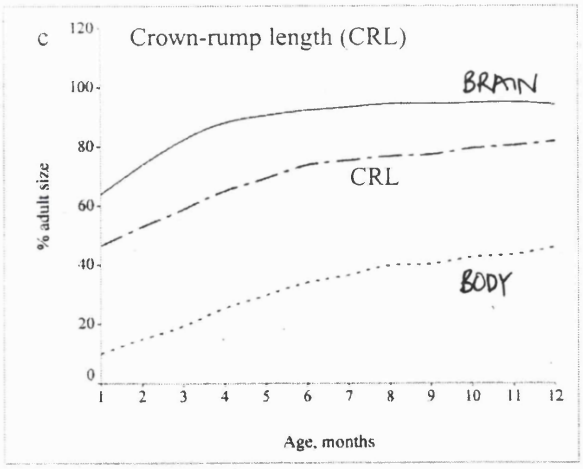


Figure 4.6. The ontogeny of limb and body proportions. (a) Limb/long bone lengths. From top to bottom, hatched data lines represent:
hand length (HAND),
foot length (FOOT),
humerus length (HUM),
tibia length (TIB),
femur length (FEM) and
radius length (RAD).
(b) Chest circumference (CHC). (c) Crown-rump length (CRL). See Fig 4.1 for key.

significant results, and TIB is very close to being significant. In view of the close association between all parts of the body during growth, it is not surprising that brain and body size are linked with almost all other aspects of size change in the growing infant. Both chest circumference and crown-rump length are also more relatively advanced compared to body size, and like the other morphological features of the body described above, show a strong significant correlation with both brain and body size (Table 4.4).

e] Maturity scores

1] Independence score (IS)

The attainment of independence (IS) is marked by significant and rapid changes in behaviour at 2 and at 6 months old (Fig 4.7a). After two months, having previously spent virtually all its time with its mother, the infant is spending up to 50% of its time away from her. After 6 months, it is spending the majority of its time (>70%) away. As we saw earlier, this does not mean the infant and mother no longer interact: at one year postpartum, as the infant still spends around 20% of its time in maternal contact (Fig 4.4c). It does reflect the fact that even by six months old capuchins are interacting with other members of their social group far more than with their mother alone. Interestingly, the correlation of IS with brain and body size is not significant in either case (Table 4.4); the relative simplicity of this score (with only 5 landmarks) may obscure the relationship.

2] Manipulation score (MS)

The development of manipulative behaviours also undergoes rapid change in the third month postpartum (Fig 4.7b). Towards the end of the second month the infant approaches objects independently for the first time. By four months old, it is touching, grasping and swinging objects, and a precision grip involving the pseudo-opposition of the thumb (i.e.

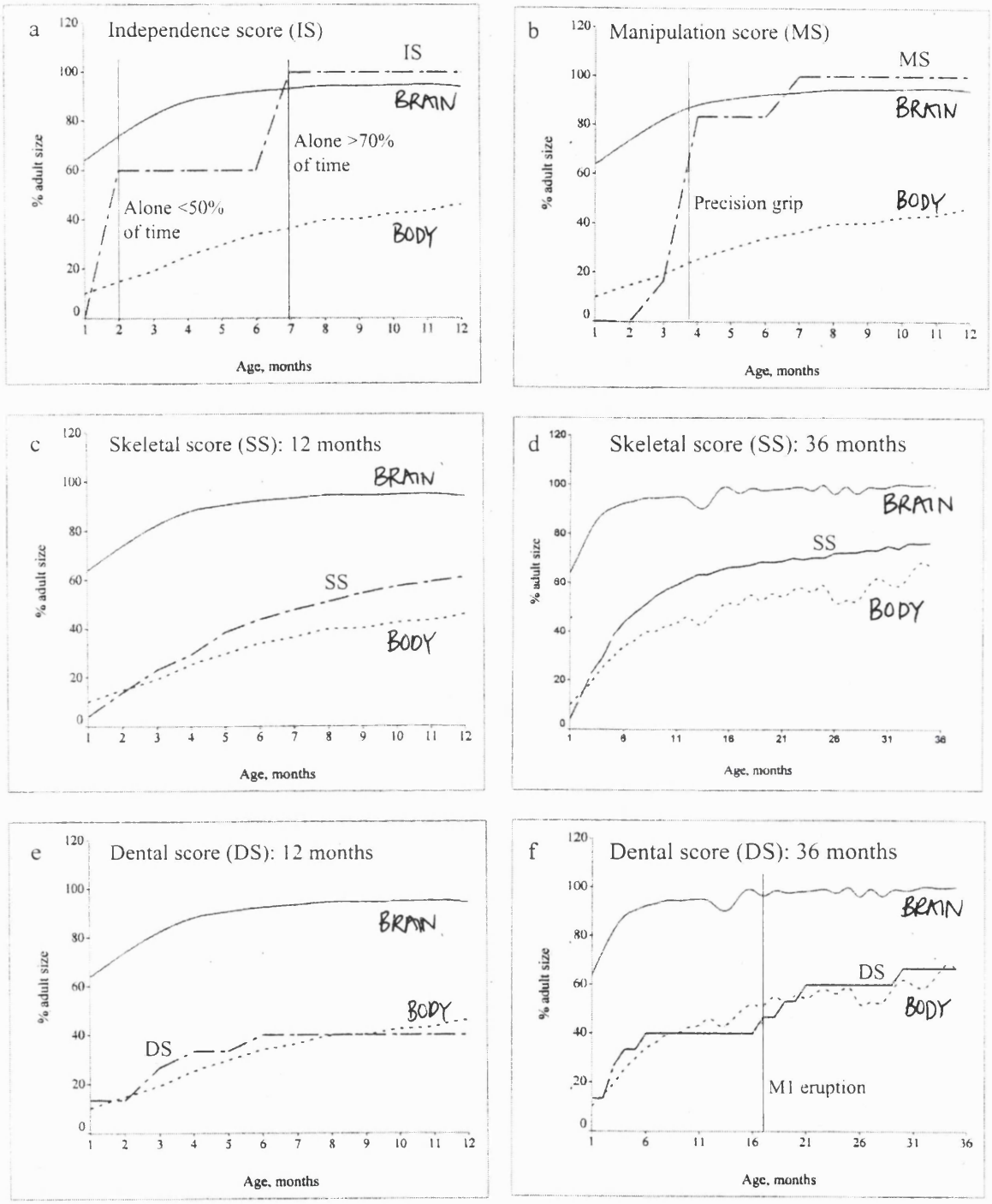


Figure 4.7. Maturity score development. (a) Independence score (IS). (b) Manipulation score (MS). (c) Skeletal score (SS) over the first 12 months postpartum. (d) Skeletal score (SS) over the first 36 months postpartum. (e) Dental score (DS) over the first 12 months postpartum. (f) Dental score (DS) over the first 36 months postpartum. Vertical line marks the age at which M1 erupts.

with the side of the first finger rather than the tip) emerges. Adams-Curtis & Fragaszy (1994) note that the emergence of the precision grip in capuchins "reflects a maturational process relatively independent of immediate environmental constraints and supports" (p135), and is therefore a good maturity indicator to extrapolate across infants. In the next few months after the appearance of the precision grip, enough strength has been gained to move objects with force, and perform behaviours that demand a coordination of strength and precision. The adult repertoire of food extraction relies on both power and precision, as Costello & Fragaszy's (1988) study of grip preference in 7 adult capuchins shows. 70% of grips used to extract a variety of items in an experimental set-up (from live crickets and coconuts to syrup) were power grips i.e. did not involve opposition of the thumb. All remaining grips were precision grips, of which most (75%) involved the thumb and first finger, the rest involving the thumb, first and second fingers (Costello & Fragaszy, 1988).

Based on evidence from rhesus macaques (Bortoff & Strick, 1993), Fragaszy & Adams-Curtis (1997) estimate that the corticospinal connections involved in manual dexterity do not mature until well after one year of age. As with all the parameters included here, it should be remembered that these data do not capture achievement of adult capabilities; rather, they chart the route along which the infants develop whilst still nursing, and the influence brain size change has on these behaviours. Fragaszy & Adams-Curtis (1997) do note, however, that "species-normal sensorimotor development must be largely achieved, if not mature, by one year, as subsequent changes in manipulative activity are minor indeed" (p208).

How does the ontogeny of manipulation influence foraging efficiency? Clearly, as infants become more adept at manipulation, the efficiency with which they are able to select, extract and process food sources will increase. In the wild, *C. olivaceus* infants younger than 18 months old engage in a repertoire of manipulative behaviours that is similar to that seen in adults (Fragaszy & Boinksi, 1995). However, they show less proficiency; in addition, the success rate at which they are able to extract food items – their foraging efficiency – is low, as might be expected (Fragaszy & Boinksi, 1995). Infants

ingested items in only 21% of foraging attempts, compared with a score of 80% for adult males and 57% for adult females; they also spent less time than adults engaging in these behaviours. Fragaszy & Boinksi describe infants at this age as "incidental foragers" (1995:246). Capuchins older than two years (i.e. older weanlings) are observed to spend *more* time than adults foraging, again because their foraging efficiency is relatively low and more time and effort must be expended to extract sufficient nutrition (Fragaszy & Boinksi, 1995). Foraging efficiency is not just determined by manipulation, however: size, strength (e.g. bite force), postural control emergence and of the dentition affect the capuchin's ability to exploit food resources (Fragaszy & Adams-Curtis, 1997), and these are qualities that develop in tandem with, and often much later than, manipulative ability. The association of brain size and MS only just fails to reach significance ($R = 0.585$, $P = 0.059$), while that between body size and MS is firmly in the non-significant range ($R = 0.441$, $P = 0.175$). As for the IS scores above, the simplicity of this score may obscure relationships between the variables.

3] Skeletal score (SS)

The skeletal altriciality of infant capuchins noted by other authors studying the HSPH population (Fleagle & Samonds, 1975; Watts, 1990) is illustrated in Figure 4.7c. The infants have actually achieved less of their adult skeletal maturity in the first month than they have of their body size maturity, but this soon changes as the high rate at which the centres of ossification emerge and develop continues (Accatino & Fleagle, in preparation). By one year of age SS is 60% (Fig 4.7c), and this level of maturity remains largely unchanged at 3 years old (Fig 4.7d). Capuchin skeletons are clearly not fully grown even by this age, and, as for body mass, still have approximately 25% of maturation to complete. Jungers & Fleagle (1980) note that skeletal maturation in capuchins is not achieved until six years of age. All the partial correlations of SS with brain size and with body size either

attain significance, or only just fall below the significance level. As noted above, this is as one would expect for systems that are so intimately linked.

4] Dental score (DS)

It is well established that there is a strong correlation between relative brain size and the age at which the first molar (M1) erupts in primates (Smith, 1991; Smith, 1992; Smith *et al.*, 1995). A similar association of deciduous tooth eruption and brain size is observed here (Fig 4.7e). By 12 months old, the dentition is about as relatively developed as is body mass, having achieved 40% of adult maturity in both, but shape of the dental curve is much more similar to that which describes brain growth (Fig 4.7e). The inflexions of both brain growth and dental development occur at the same age, i.e. at 6 months old, when the deciduous dentition is fully erupted. This similarity is statistically robust: the correlation of DS and brain size over these first 12 months is highly significant, whilst that of DS and body size is not ($R = 0.793$, $P = 0.004$; $R = 0.340$, $P = 0.306$ respectively). The deciduous teeth appear to follow a pattern of eruption that is closely linked to the growth of the brain.

This pattern changes after brain growth is complete. M1 eruption occurs in the 17th month postpartum in *C. apella*, and after this age, the DS curve loses its similarity to the brain growth curve (Fig 4.7f). Instead, it tracks the body growth curve. By three years old body size and dental maturity are at exactly the same percent of adult values (70%). The significance of the partial correlation between body size and DS over 36 months confirms this trend ($R = 0.755$, $P = 0.000$). As we might predict, the correlation of brain size and DS over 36 months is not significant ($R = -0.004$, $P = 0.983$). It appears that, with the attainment of adult brain size, dental development is arrested and the emergence of the permanent dentition delayed by about a year. M1 eruption occurs only after or at the time that the infants are fully weaned. Lee (1999) comments on Smith's (1991; 1994) observed correlation of brain size and M1 eruption:

> "The strong association between relative M1 eruption and relative brain growth... suggests that... when maternal size is removed, brain and tooth growth appear to covary. I would suggest that one expense of growth is that of maintaining a suite of rate-limited traits that all contribute to the energy burden on the mother."
>
> Lee (1999:127)

The results presented here suggest that the permanent dentition erupt only when proto-adult food-processing capabilities are required, i.e. at the completion of weaning. However, it is worth noting that even by this late age (17 months potspartum), the permanent dentition has a long way to go before it is fully erupted. This may be one reason why the transferral of 'difficult' foods, i.e. ones that require at permanent dentition to extract, is so common between adults and weanling capuchins (see above).

4.1.3 The influence of rearing conditions

The influence of social and other environmental factors on primate behavioural ontogeny is well recognised (Falkner & Tanner, 1986; Hofer, 1987; Spijkerman *et al.*, 1995; Queirouil & Blois-Heulin, 1998b). Differences in sensorimotor maturity have been reported between capuchin infants that are hand-reared and those that are mother-reared (with the former being more advanced relative to the latter; Adams-Curtis & Fragaszy, 1994). Furthermore, social deprivation and enrichment have been shown to affect the ontogeny of feeding, exploratory, manipulative and many other aspects of capuchin behaviour (Elias & Samonds, 1973; Fragaszy, 1989; Visalberghi & Fragaszy, 1995; Fragaszy & Adams-Curtis, 1997; Fragaszy *et al.*, 1997b). For instance, Elias & Samonds (1973) found that 3 HSPH monkeys, reared in partial isolation for the first 6 months postpartum, exhibited different exploratory behaviours from those reared in a group setting. The average age of first appearance of approaching/touching objects was delayed by a few weeks in the deprived animals, and they exhibited more stereotypical behaviours. Although no retardation of

perceptual or locomotor development was noted over the course of the six months, at the end of the experimental period these animals were more reluctant to explore and habituate to novel environments.

The different housing conditions (Table 4.1) of the capuchin colony studied by Byrne & Suomi (1995; 1998) provide an opportunity to investigate the influence of social and physical environment on behavioural development. Those authors note that because of the group and housing size differences in the runs (larger) and cages (smaller), the run environment was both physically and socially more complex than the cage environment (Byrne & Suomi, 1995). Although infants in both conditions show similar levels of ventral contact with their mother at 12 months of age, those living in runs are quicker to achieve this 12 month value, and have much less ventral contact during earlier months than cage infants (Fig 4.8a). The run infants also show a much lower frequency of approaches and leaves towards or away from their mothers in the second half of the year (Fig 4.8b). The extra space associated with the runs (and the greater complexity of interactions the infants experience) may have a positive effect on the weaning process, accelerating infant independence.

4.2 Summary

This chapter asks the question: *why* are brain growth and weaning linked, and are there aspects of weaning that are specifically associated with brain, rather than body, growth? It describes various behaviours and physiological features that are components of weaning, and links their ontogenetic trajectories with those of brain and body size.

The first hypothesis suggested that nursing behaviours are associated with brain, not body, growth. Suckling behaviours are indeed associated with brain growth. Time spent nursing and the duration of each nursing bout declines rapidly with age, and both plateau at the same age (5 months) at which the rapid rate of brain growth slows. This

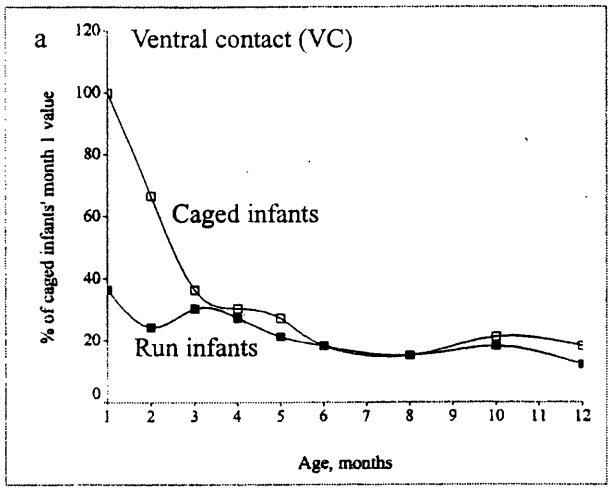


Figure 4.8. The influence of rearing conditions on behavioural and locomotor independence. (a) Ventral contact (VC). Expressed as a percentage of the caged infants' mean month 1 value. (b) Sum of leaves (IL) and approaches (IA) made by the infant. Expressed as a percentage of the caged infants' mean month 12 value. In both plots, open squares are datapoints from caged infants, closed squares are datapoints from run infants.

specific association of nursing and brain growth supports theories that link the delayed attainment of infant independence with brain development (see Ross & Jones, 1999).

The second hypothesis suggested that behavioural independence is associated with body growth. The correlation results show that time spent in ventral contact with the mother in fact shows a pattern of decline similar to the nursing variables, and is also strongly associated with brain growth. This is logical when one considers that nursing occurs with the infant in position on the mother's ventrum. Brain growth is therefore associated not just with direct measures of weaning such as suckling behaviour, but also with other aspects, e.g. maintenance of maternal contact.

Body growth is also an important predictor of weaning behaviour. The third hypothesis predicted that locomotor independence and body growth are associated. Infants do approach their mother more frequently as they increase in body mass, possibly to attempt suckle and support the growing needs of their bodies. However, the shift in responsibility for the maintenance of contact from mother to infant occurs between the ages of 3 and 5 months old, when brain growth begins to plateau. The incidence of investigatory behaviour towards food is more strongly correlated with body growth than brain growth, but this relationship does not reach significance. Approaches by the mother are much less frequent as the infant grows, and are correlated with increases in both brain and body size. Two other measures of maternal contact, time spent in dorsal contact (i.e. being carried) and time spent with mother (i.e. in direct physical contact), correlate very strongly with body size. This indicates that the mothers' decision to carry and support their infant depends to a large extent on how big the infants' bodies have become. Whether brain and/or body growth are limited by maternal metabolism (i.e. size) is not clear from this analysis, but is investigated in more detail in Chapter 5.

The fourth hypothesis suggested that body growth is also associated with general morphological maturity. Whilst other aspects of morphology (e.g. long bone lengths) mature at rates intermediate to brain and body growth, and overall skeletal maturation is significantly correlated with both brain and body size increase, dental maturation is

strongly associated with brain growth in the first year postpartum. There is a developmental hiatus between the eruption of the deciduous teeth and the eruption of M1 that appears to last until brain growth is completed at 12 to 18 months postpartum. After this age, dental eruption tracks body mass growth until the end of the study period.

The development of independent locomotion and manipulative ability is not significantly correlated with brain or body size, but these behaviours become extremely well developed after the 4th or 5th month postpartum. The explosion of manipulative, locomotor and other activities in the middle of the first year postpartum coincides with the period of maximum postnatal brain growth, and with a concurrent drop in the nutritional support given to the infant by the mother. As was noted in the introduction to this chapter, these aspects of growth appear to "affect one another reciprocally and dynamically" (Fragaszy & Adams-Curtis, 1997:202). Fragaszy & Adams-Curtis go on to speculate that "neural development, sensorimotor development, and cognitive reorganisation may pace the development of foraging competence and the termination of nursing" (Fragaszy & Adams-Curtis, 1997:202). The analysis presented here supports their argument, and highlights those elements of weaning that are specifically associated with the maturation of the brain.

CHAPTER 5

MODELLING ENERGETICS IN LACTATION AND WEANING

The results of the previous chapter suggest that weaning is associated with a repertoire of behaviours that emerge and develop as the brain achieves its adult size and brain growth slows. This chapter examines a mechanism by which the slowing of brain growth might be linked to the termination of lactation: the high energy demands of the brain and the provision of energy by the milk. It investigates growth and lactation strategies in the context of brain and body energetics. Data from the Harvard School of Public Health (HSPH) colony described in Chapters 2 and 3, and data from another *Cebus apella* colony housed in the Chilean Primate Centre, are used to model the energy costs of body and brain maintenance. These costs are analysed in relation to encephalisation, maternal capacity and lactation.

5.1 Modelling the energetics of lactation

The composition of the non-brain tissues is relatively heterogeneous, comprising both energetically-expensive organs (such as the liver) and non-expensive structural tissue (e.g. the skeleton) (Sinclair & Dangerfield, 1998). The energy cost of maintaining the non-brain tissues is therefore a function of the costs of different tissues. In comparison, brain tissue is homogeneous and its maintenance costs are uniformly high (Aiello & Wheeler, 1985). The energy cost of the brain and non-brain components of the body can be estimated from the amount of oxygen consumed over a unit of time, ~~[i.e. as a metabolic rate (MR)]~~. The rate of oxygen consumed by, and therefore energy needed for maintenance of, tissues is directly proportional to size, and in general, larger bodies require less energy relative to their size (Kleiber, 1947; Heusner, 1985; Ross, 1992a). Adult bodies are generally more 'efficient' when it comes to energy consumption; infants, because of their small size, are relatively less efficient at utilising energy than adults (Janson & van Schaik, 1993). Substrate utilisation mediates the relationship between metabolic rates and the volume of oxygen consumed (Brody, 1999), and only the volume of oxygen (VO_2) consumed is discussed here.

Lactating mothers must supply all of their infants' energetic demands, at least early in development. If the energetics of growth are important in determining lactation strategies, we can predict that the relative sizes of the brain and the body at birth will impact upon weaning size. This will be especially true if the energy investment in brain growth is obligatory and protected at the expense of body growth. The larger the brain relative to body size at birth, the less 'spare' energy available to fuel body growth. We can predict that variation in separation or weaning size will be explained by variation in encephalisation.

We might also predict that mothers are able to regulate lactation such that infant energy demands do not outstrip maternal energy supply. Mothers might regulate the size of their infant at birth, or at weaning. Mothers might restrict the length of time infants spend dependent on maternal resources; alternatively, mothers might spread the high energy cost of lactation over a long period of maternal dependency (Lee, 1987). With these predictions in mind, several questions can be formulated which investigate the energetics of growth:

1] Is maternal mass associated with infant brain and/or body mass at birth?

Previous research has highlighted the role of maternal body mass in influencing neonatal body size such that larger mothers produce larger infants (Leutenegger, 1979; Martin & MacLarnon, 1985; Ross, 1988; Lee *et al.*, 1991; Lee, 1999). Maternal mass and, by inference, metabolic capacity, has also been implicated in the regulation of neonatal brain size (Martin, 1983; Martin, 1996). It is of interest to establish whether neonatal brain size or body size is more tightly linked to maternal capacity.

2] Is maternal mass associated with infant brain and/or body mass at separation?

As an extension of Question 1, it is predicted that maternal reserves will also influence the size of the infant at separation.

3] Is maternal mass associated with infant ~~metabolic rate~~ oxygen consumption during ontogeny?

The rate at which the infant brain and non-brain tissues consume energy is likely to be regulated by maternal mass such that infant demands do not outrun maternal reserves.

4] Is maternal mass associated with age at separation?

Mothers may spread the cost of investment in expensive offspring over a long lactation period (Lee, 1987). On the other hand, larger mothers may be able to supply more energy to the infant, reducing the length of the current reproductive event by growing their infants to a large size more quickly (Boinksi & Fragaszy, 1989).

5] Is encephalisation at birth a good predictor of brain and body size at separation?

As mentioned above, the relative sizes of the brain and the non-brain tissues are likely to be important in predicting separation size. In particular, infants that are relatively encephalised as neonates are predicted to have less heavy non-brain tissues at separation, independent of brain size variation. This is because the demands of brain maintenance are largely obligate; the size of the other body tissues at separation will therefore depend on the 'spare' energy supplied by the milk over lactation.

6] ~~Are metabolic rates~~ Is oxygen consumption associated with growth variables?

The rates at which the tissues consume ~~energy~~ oxygen are predicted to correlate with other aspects of growth such as the age at separation, and the amount of growth undergone by separation.

5.1.1 Materials

The data on growth and lactation used in this chapter are taken from two sources:

1] The HSPH capuchin colony described in detail in Chapter 2, and

2] A colony of *C. apella* housed in the Chilean Primate Centre (CPC), Universidade Catolica de Chile, Santiago, Chile.

The CPC colony was established in 1989 and currently houses around 80 breeding animals. Research on this colony is carried out by the Catholic University of Chile's Unit of Reproductive and Developmental Biology, and has focused mainly on the hormonal aspects of female reproduction and infant perinatal development (Diaz *et al.*, 1995; Recabarren *et al.*, in preparation). All animals in the colony are second-generation laboratory-conceived and – delivered, and exact age is known for all individuals, both in terms of gestational age (via radioimmunoassays) and postnatal chronological age (days postpartum).

The CPC infants are reared by their mothers, remaining with them until separation at 6 or 7 months postpartum. Separation occurs when members of the CPC staff judge that the infant is old enough to support itself nutritionally, and is evaluated on an individual basis. Size of the infant is taken into consideration, but the decision is primarily based on observations of feeding ability (pers. obs). The infants has access to all food provided for the dam, but are reliant on milk for nutritional support for the majority of the period until separation (M. Serron-Ferre, pers. comm.). The infants' body mass at separation approximates weaning weights observed in the wild (see below), but separation should not be thought of as 'weaning' in the behavioural sense discussed in Chapter 4. In the analyses that follow, separation age represents the attainment of a developmental landmark that is analogous to weaning i.e. the achievement of nutritional independence. Infants that are advanced in their ability to support themselves are separated earlier than others who are less able.

Once removed from their mothers, the weanlings are housed in cages with other infants from their age cohort, typically three or more individuals (pers. obs.). Juveniles older

than one year old are separated and kept in individual cages. The cages are similar in size to those of the HSPH colony (70cm x 60cm x 80cm), and also temperature- and light-controlled (14h, 28°C). After separation, the infants' diet is the same as that of the adults, consisting of chow pellets (Quimagro S. A., Santiago, Chile), whole-wheat bread, fruit and vegetables (pear, kiwi, orange, banana, apple, carrot), peanuts and other mixed nuts, and polenta, polenta cake and fruit cake (which includes dried figs, raisins eggs and honey in its ingredients). Food is provided twice daily, at 12h00 and 18h00. The macronutrient composition of the diet is shown in Table 5.1.

Data were gathered from the CPC colony records, and include information from the date of the colony's first birth, in September 1991, until the end of 1999. The sex of each infant born in the colony and the identity of its mother were noted. Body and head size data were also collected.

Measurement of body mass in the CPC colony was on an *ad hoc* basis. The masses of both the infant and mother were recorded as soon after birth as possible, always within 24 hours. Head breadth, defined as the widest distance across the parietal bones perpendicular to the mid-sagittal plane, was measured at the same time as birth weight, and then periodically after birth. Sliding calipers were used, and head breadth recorded to the nearest millimetre. Nursing mothers were subject to experimental or health check procedures approximately once a week following birth. During these checks females and their infants were weighed. After separation from their mothers, infants were weighed less often until they come to reproductive maturity. Females of reproductive age were given regular smear tests to monitor their menstrual cycle, and were weighed at the same time. In most circumstances, animals were sedated before measurement, and weighed on a balance scale. All masses noted in the lab records, and the date on which they were taken, were gathered, except where an animal was reported to be emaciated or unhealthy. In general, infant health in the colony was excellent: of a total of 82 infants born in the colony between 1991 and 1999, none died from disease or ill-health, only by experimental sacrifice.

Table 5.1 Dietary intake in the CPC colony *Cebus apella*[1]

	Total	Protein[2]	Fat	Carbohydrate*	Fibre
Daily intake (g/day):	2282.00	396.00	23.48	109.31	82.22
% of daily intake		17.37	1.03	4.79	3.60

[1]Calculated from Bender & Bender (1999) food values and analysis of monkey chow by Quimagro S. A., Santiago, Chile.
[2]Calculated from total nitrogen values, as described in McCance & Widdowson (1991).
*Monosaccharides

5.1.2 Methods

a] Head breadth and brain size

Data on head breadth at separation were relatively scarce. In order to maximise the dataset sample size, average head breadth growth rate (mm/d over 30 day intervals) was used to calculate head breadth at the end of each successive month for those individuals for which empirical data were not available (n = 12). Head breadth at separation was then inferred from the projected growth curve for each individual, at the known separation age. Brain size at birth and separation were estimated from head breadth in the following way:

1] The mean and standard deviation of head breadth at birth and separation were calculated, based on the CPC colony records (Fig 5.1a and b). The distribution of head breadth at birth has a standard deviation that is approximately 10% of the mean value (38.7 ± 4.55mm; Fig 5.1a). The distribution of head breadth at separation has a standard deviation that is approximately 5% of the mean value (55.1 ± 3.18mm; Fig 5.1b).

2] Z-scores were calculated for each individual in the population using the formula:

$$z_c = (a_c - mean_c) / sd_c \qquad (5.1)$$

where a_c is the head breadth value of each CPC infant, $mean_c$ is the population mean head breadth, sd_c is the population standard deviation, and z_c is the infant's head breadth z-score.

3] To convert each head breadth size to estimated brain size (BR), z-scores were transferred from the real to a hypothetical brain size distribution by rearrangement of the previous equation:

$$BR = (z_c * sd_h) + mean_h \qquad (5.2)$$

where sd_h is the standard deviation of the hypothetical population and $mean_h$ is the mean value of the hypothetical population. Estimated brain mass at birth and separation (BR_b and BR_s) are

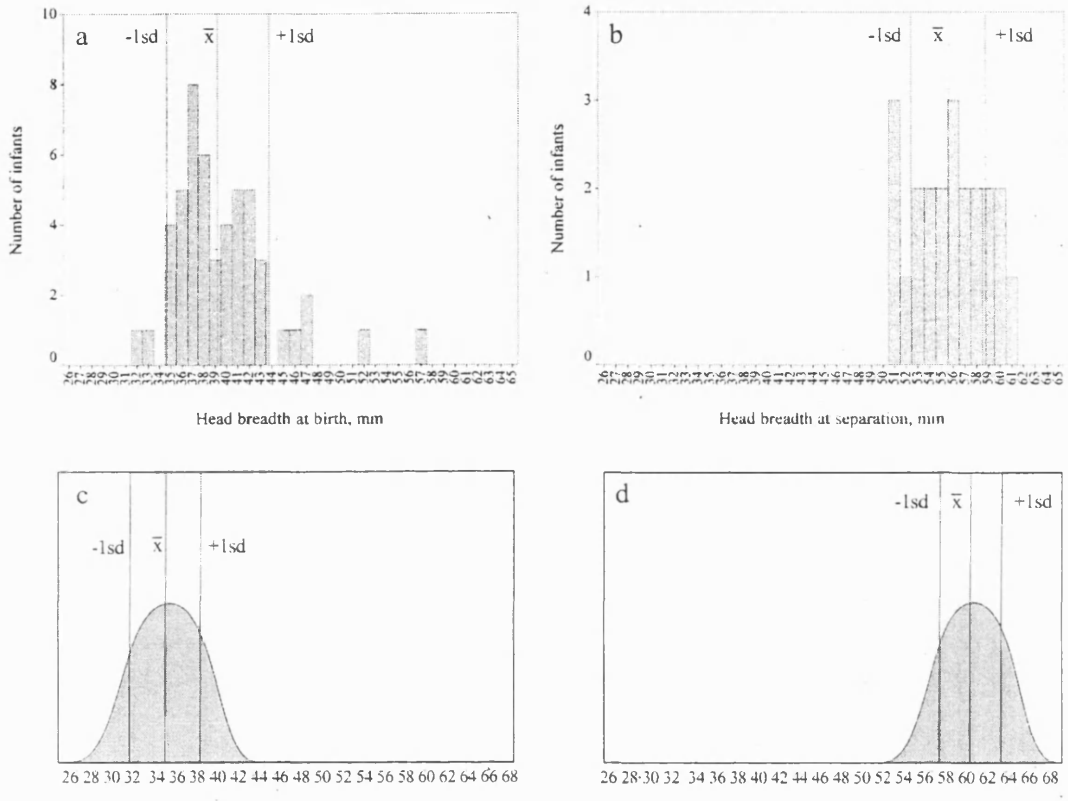


Figure 5.1. Estimating brain size on the CPC infants: hypothetical and real population distributions. (a) Head breadth at birth; (b) head breadth at separation; (c) brain mass at birth (BR_b); (d) brain mass at separation (BR_s). Vertical lines mark (from left to right in each plot) the mean minus one standard deviation; the mean; and the mean plus one standard deviation respectively. (c) and (d) are hypothetical distributions based on brain size data discussed in text and summarised in Appendix 1.

measured in grams. The hypothetical brain size distributions (Fig 5.1c and d) assume a mean brain size at birth of 35 ± 3.5g (i.e. standard deviation = 10% of mean) and a mean brain size at separation of 60 ± 3.0g (standard deviation = 5% of mean). These figures are based on reported mean brain sizes from the literature, assuming brain size is 90% of adult size at separation (Appendix 1).

b] Non-brain body mass

Non-brain body mass was calculated as:

$$\text{NBBM} = \text{body mass} - \text{BR} \qquad (5.3)$$

Body mass that includes brain mass is designated 'whole body mass' (WBM) in the following analyses.

c] Tissue maintenance costs

Kuzawa (1998) and Schmidt *et al.* (1945) report the oxygen consumption rate of brain tissue for a variety of primate and non-primate taxa (n = 12 species). Both sources report the results of experiments that use empirical measurements of cerebral oxygen uptake (direct flow analysis of the cerebral bloodflow) to estimate the oxygen consumption of the brain. From these data (Table 5.2), the ~~metabolic rate~~ volume of oxygen consumption of the brain (brain $\dot{V}O_2$) was calculated as:

$$\text{Brain } \dot{V}O_2 = \text{total } \dot{V}O_2 * \text{percentage of total } \dot{V}O_2 \text{ consumed by the brain} \qquad (5.4)$$

Kuzawa (1998) and Schmidt *et al.*'s (1945) data are presented in Table 5.2. It can be seen that the percent of total oxygen (total $\dot{V}O_2$) that is required by the brain varies hugely, from 1% of total MR for *Sus* to 60% of total $\dot{V}O_2$ for a human neonate. However, when brain size is taken into account, all species show a similar relationship between brain size and brain $\dot{V}O_2$. An

TABLE 5.2 **Calculating ~~metabolic rates~~ oxygen consumption of brain and body tissue in mammals**[1]

Taxon	Age	Brain mass (BR) (g)	Non-brain body mass (NBBM) (g)	Total ~~MR~~ VO (ml/min)	% of total ~~MR~~ VO to brain	Brain ~~MR~~ VO (ml/min)	% of total ~~MR~~ VO to NBBM	Body ~~MR~~ VO (ml/min)
Homo	Neonate	350.00	3150.00	20.00	60.00	12.00	40.00	**
Ovis	Neonate	53.80	3446.20	21.00	10.00	2.10	90.00	18.90
Homo	Adult	1400.00	68600.00	250.00	19.60	49.00	80.40	201.00
Pan	Adult	430.00	37570.00	158.00	8.60	13.59	91.40	144.41
Macaca	Adult	88.00*	3412.00	29.00	9.00	2.61	91.00	26.39
Macaca	Adult	114.00*	3386.00	30.00	11.70	3.51	88.30	26.49
Macaca	Adult	89.00*	4011.00	40.00	6.90	2.76	93.31	37.24
Ateles	Adult	110.00	3690.00	36.00	8.80	3.17	91.20	32.83
Loxodonta	Adult	5712.00	6644288.00	7800.00	2.50	195.00	97.50	7605.00
Equus	Adult	662.00	599338.00	1296.00	1.80	23.33	98.20	1272.67
Ovis	Adult	106.5	51893.50	215.00	1.70	3.66	98.30	211.34
Sus	Adult	123.90	124876.10	400.00	1.00	4.00	99.00	396
Canis	Adult	80.00	16320.00	83.40	2.90	2.42	97.10	80.98
Lepus	Adult	10.14	2589.86	21.70	3.10	0.67	96.90	21.03
Cavia	Adult	4.23	795.77	10.3	1.60	0.16	98.40	10.14
Rattus	Adult	2.30	247.70	3.40	2.10	0.07	97.90	3.33

[1]All data taken from Kuzawa (1998) except those for *Macaca*, which come from Schmidt et al. (1945).

**Human neonate not included in the calculation of body ~~MR~~ VO.

*Brain masses given in source. All other brain masses estimated from Crile & Quiring (1940).

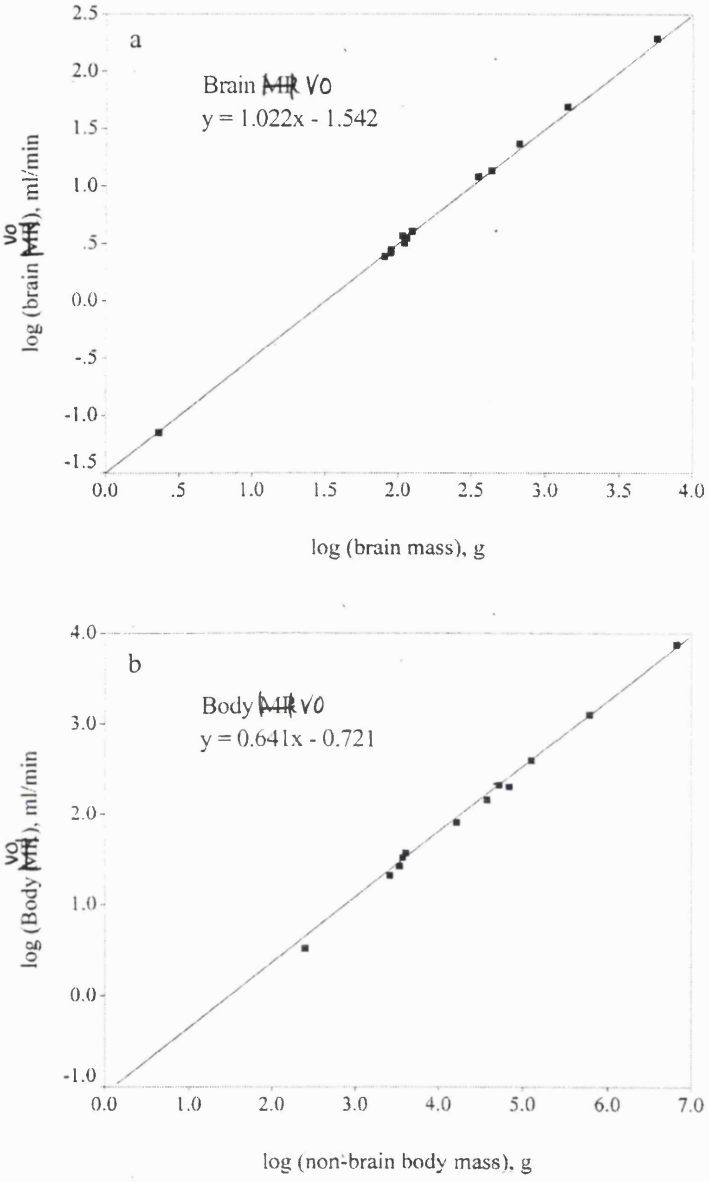


Figure 5.2. Predicting metabolic rates from brain and body mass. (a) Brain metabolic rate (Brain $\dot{V}O_2$) vs. brain mass (BR); (b) non-brain tissue metabolic rate (Body $\dot{V}O_2$) vs. non-brain body mass (NBBM). Data are from Table 5.2. Lines fitted are RMAs.

increase in one unit of brain size incurs an increase in ~~metabolic rate~~ oxygen consumption of approximately one unit (Fig 5.2a) although the 95% confidence interval for the slope does not include isometry (1.013 to 1.031):

$$\text{Brain } \dot{V}O_2 = 1.542*(\text{brain mass})^{1.022} \quad (5.5)$$

The metabolic rate of the non-brain tissues (body $\dot{V}O_2$) was calculated as:

$$\text{Body } \dot{V}O_2 = \text{total } \dot{V}O_2 - \text{brain } \dot{V}O_2 \quad (5.6)$$

The rate at which body $\dot{V}O_2$ increases in relation to body size is lower than isometry: the RMA slope describing the relationship is 0.641 (confidence interval 0.607 to 0.675; Fig 5.2b):

$$\text{Body } \dot{V}O_2 = 0.721*(\text{non-brain body mass})^{0.641} \quad (5.7)$$

These equations describing the relationship of rate to size were used to calculate brain and body $\dot{V}O_2$ at birth and separation for each infant. These variables quantify $\dot{V}O_2$ at two specific points in ontogeny: birth and separation. In order to examine how $\dot{V}O_2$ changes between and after these points, brain $\dot{V}O_2$, body $\dot{V}O_2$ and total $\dot{V}O_2$ (brain $\dot{V}O_2$ + body $\dot{V}O_2$) were calculated for a 'typical' capuchin, based on the growth curves presented in Chapter 4.

The variables used to model energetics are given in full below and listed in Table 5.3. The data did not reveal any significant sexual dimorphism in any of the variables (data not shown), and all analyses are performed on sex-combined data. Descriptive statistics of the colony are given in Table 5.4.

1] WBM_b Whole body mass at birth (grams).

2] WBM_s Whole body mass at separation (grams).

3] BR_b Brain mass at birth (grams).

4] BR_s Brain mass at separation (grams).

5] $NBBM_b$ Non-brain body mass at birth (grams).

6] $NBBM_s$ Non-brain body mass at separation (grams)

7] WBM ratio Whole body mass at separation:whole body mass at birth ratio.

8] BR ratio Brain mass at separation:brain mass at birth.

9] NBBM ratio Non-brain body mass at separation:non-brain body mass at birth.

10] MM_b Maternal mass at birth of infant (grams).

Taken at the same time as the infant's birth mass i.e. within 24h of the birth. For 7 individuals, maternal mass at birth was approximated by the most recent previous body mass measurement.

11] MM_c Maternal mass at conception (grams).

Inferred from the body growth curve of the mother at conception date.

12] MM_n Maternal mass, non-pregnant and non-lactating (grams).

Calculated as the mean mass of the mother when neither pregnant nor lactating, excluding data prior to first pregnancy where possible.

13] S_{age} Age at separation (days).

14] Pre-separation WBM rate Pre-separation whole body mass growth rate, (grams/day). Pre-separation WBM rate = [whole body mass at separation – whole body mass at birth] / age at separation

15] Pre-separation BR rate Pre-separation brain mass growth rate (grams/day). Pre-separation BR rate = [brain mass at separation – brain mass at birth] / age at separation

16] Pre-separation NBBM rate Pre-separation non-brain body mass growth rate, (grams/day). Pre-separation NBBM rate = [non-brain body mass at separation – non-brain body mass at birth] / age at separation

17] ENC_b Encephalisation at birth. ENC_b = brain mass at birth / non-brain body mass at birth

18] ENC_s Encephalisation at separation. ENC_s = brain mass at separation / non-brain body mass at separation

19] Brain VO_b ~~MR_b~~ Oxygen consumption ~~Metabolic rate~~ of the brain at birth (millilitres oxygen/minute).

20] Brain VO_s ~~MR_s~~ Oxygen consumption ~~Metabolic rate~~ of the brain at separation (millilitres oxygen/minute).

21] Body VO_b ~~MR_b~~ oxygen consumption ~~Metabolic rate~~ of the non-brain body tissues at birth (millilitres oxygen/minute).

22] Body VO_s ~~MR_s~~ Oxygen consumption ~~Metabolic rate~~ of the non-brain body tissues at separation (millilitres oxygen/minute)

23] Total VO_b ~~MR_b~~ Oxygen consumption ~~Metabolic rate~~ of the brain and body at birth (millilitres oxygen/minute)

24] Total VO_s ~~MR_s~~ Oxygen consumption ~~Metabolic rate~~ of the brain and body at separation (millilitres oxygen/minute).

d] Statistical analysis

Correlations, partial correlations and linear regressions were performed on pairs and groups of variables (Pearson's correlation, significance when $P \leq 0.05$).

5.1.3 Limitations

1] Although the CPC infants are mother-reared, the environment in which they live is not naturalistic. They are not left to wean naturally, instead being removed from their mothers when judged to be self-sufficient. As noted above, although the infants' masses at weaning approximate those of wild weanling capuchins, separation is not weaning in the behavioural

TABLE 5.3 Variables used in the energetics modelling

Variable	Abbr.
Age at separation	S_{age}
Brain mass at birth	BR_b
Brain mass at separation	BR_s
Brain mass ratio	BR ratio
Encephalisation at birth	ENC_b
Encephalisation at separation	ENC_s
Maternal mass at birth	MM_b
Maternal mass at conception	MM_c
Maternal mass non-pregnant, non-lactating	MM_n
Metabolic rate of the brain at birth	Brain MR_b
Metabolic rate of the brain at separation	Brain MR_s
Metabolic rate of the NBBM at birth	Body MR_b
Metabolic rate of the NBBM at separation	Body MR_s
Non-brain body mass at birth	$NBBM_b$
Non-brain body mass at separation	$NBBM_s$
Non-brain body mass ratio	NBBM ratio
Pre-separation NBBM rate	
Pre-separation BR rate	
Pre-separation WBM rate	
Total MR at birth	Total MR_b
Total MR at separation	Total MR_s
Whole body mass at birth	WBM_b
Whole body mass at separation	WBM_s
Whole body mass ratio	WBM ratio

ERRATUM: FOR 'METABOLIC RATE' AND 'MR' READ 'OXYGEN CONSUMPTION' AND 'VO' RESPECTIVELY

Table 5.4 Descriptive statistics from the CPC colony records

Species		*C. apella*				
Total n of infants for which data are available (m:f)		65 (36:29)				
Variable	Abbr. (see text)	n	Units	Mean	Standard error	Standard deviation
HB at birth		51	mm	38.73	0.64	4.55
HB at separation		22	mm	55.09	0.68	3.18
Whole body mass at birth	WBM_b	65	g	197.38	3.35	27.02
Whole body mass at separation	WBM_s	31	g	777.48	18.11	100.83
Maternal mass at birth	MM_b	65	g	2489.85	46.34	373.62
Maternal mass at conception	MM_c	64	g	21825.81	30.91	257.30
Maternal mass non-pregnant, non-lactating	MM_n	65	g	2221.95	31.62	254.91
Age at separation	S_{age}	31	d	196.55	6.48	36.07
Whole body mass ratio	WBM ratio	31	-	3.974	0.08	0.43

sense of the word. Inevitably, brain and body growth in the CPC infants will differ from other, non-captive populations.

2] Activity budgets and growth costs in these infants are unknown. The interaction of growth, activity and maintenance costs cannot, therefore, be examined. The maintenance costs of tissues are estimated from their masses. The actual mass of the brain is not known and has been inferred from other animals from different taxa. Capuchin brains might be exceptional in their metabolic capacity; until experimental and empirical data is gathered, this cannot be known.

3] The dataset used to derive ~~metabolic rates~~ oxygen consumption includes mostly adult data, whereas this analysis is primarily of infant growth and energetics. Infants differ from adults in typical body composition, and the metabolic demands of tissues are unlikely to be equivalent across different ages.

4] Because the CPC data was gathered retrospectively by the author, and over the course of different years and by different observers, it was not possible to estimate measurement errors in any of the mass or head breadth parameters. Error is inevitable, especially when the object being measured is relatively small (e.g. head breadth in neonates). The methods used in measuring head breadth, inferring brain mass and calculating brain metabolic rate will also compound the errors inherent in each stage of inference.

5.1.4 Results

1] Is maternal mass associated with infant brain and/or body mass at birth?

The only maternal mass variable to correlate significantly with any measure of infant size at birth is maternal mass when non-pregnant and non-lactating (MM_n; Table 5.5). Maternal mass when non-pregnant and non-lactating is positively correlated with brain size at birth (BR_b, Table 5.5.1), and also at separation (BR_s, Table 5.5.2). MM_n correlates with the BR ratio (Table 5.5.3), but this relationship disappears when brain mass at birth is held constant (Table

Table 5.5 Infant mass and age correlates of maternal mass

	Correlation between...	...and...	...controlling for...	r	r^2	P	n
5.5.1	Maternal mass non-pregnant and non-lactating (MM_n)	Brain mass at birth (BR_b)	-	**0.388**	**0.114**	**0.005**	**51**
5.5.2	Maternal mass non-pregnant and non-lactating (MM_n)	Brain mass at separation (BR_s)	-	**0.433**	**0.187**	**0.044**	**22**
5.5.3	Maternal mass non-pregnant and non-lactating (MM_n)	BR ratio	-	**-0.431**	**0.186**	**0.045**	**22**
5.5.4	Maternal mass non-pregnant and non-lactating (MM_n)	BR ratio	Brain mass at birth (BR_b)	-0.398	0.158	0.865	19
5.5.5	Maternal mass at birth (MM_n)	Non-brain body mass at separation ($NBBM_s$)	-	**0.428**	**0.183**	**0.047**	**22**
5.5.6	Maternal mass non-pregnant and non-lactating (MM_n)	Brain mass at separation (BR_s)	Brain mass at birth (BR_b)	0.157	0.025	0.498	19

See text for discussion.

5.5.4). MM_n does not correlate with any parameters that measure infant body size at birth (data not shown).

Figure 5.3a shows brain mass at birth plotted against maternal mass when non-pregnant and non-lactating. One infant has a very heavy mother compared to the rest of the sample (marked A). When this infant is excluded from the analysis, the correlation remains significant ($r = 0.322$, $P = 0.023$, $df = 50$). Figure 5.3b shows the same data; infants with the same mothers are indicated by similar symbols. One mother in particular gave birth to siblings with a wide range of brain masses. Her offspring are marked by closed diamonds (Fig 5.3b). When her infants are removed from the analysis, the significance of and amount of variation explained by the association increases ($r = 0.471$, $P = 0.001$, $df = 45$). When both her infants, and the infant marked A, are removed from the analysis, the relationship remains highly significant ($r = 0.355$, $P = 0.018$, $df = 44$). Although the datapoints shown in Figure 5.3 are not independent (most infants are related to at least one other infant included in the analysis), the general trend is for larger mothers to give birth to larger-brained infants. When brain size at birth is averaged over all births for each female, mean brain size is highly significantly correlated with maternal mass when non-pregnant and non-lactating ($r = 0.569$, $P = 0.000$, $n = 51$).

It is interesting to note that of the three maternal parameters, it is the mass that represents maternal size when non-pregnant and non-lactating that best explains variation in brain size at birth. This may be because maternal mass when non-pregnant and non-lactating reflects maternal size generally, rather than specifically to any one reproductive event, and general maternal size is a better indicator of overall maternal metabolic capacity.

2] Is maternal mass associated with infant brain and/or body mass at separation?

Maternal mass at birth (MM_b) is significantly and positively correlated with the size of the non-brain tissues at separation ($NBBM_s$), although the R^2 value is low (0.183), and the correlation only just reaches significance (Table 5.5.5). In other words, there is a loose but

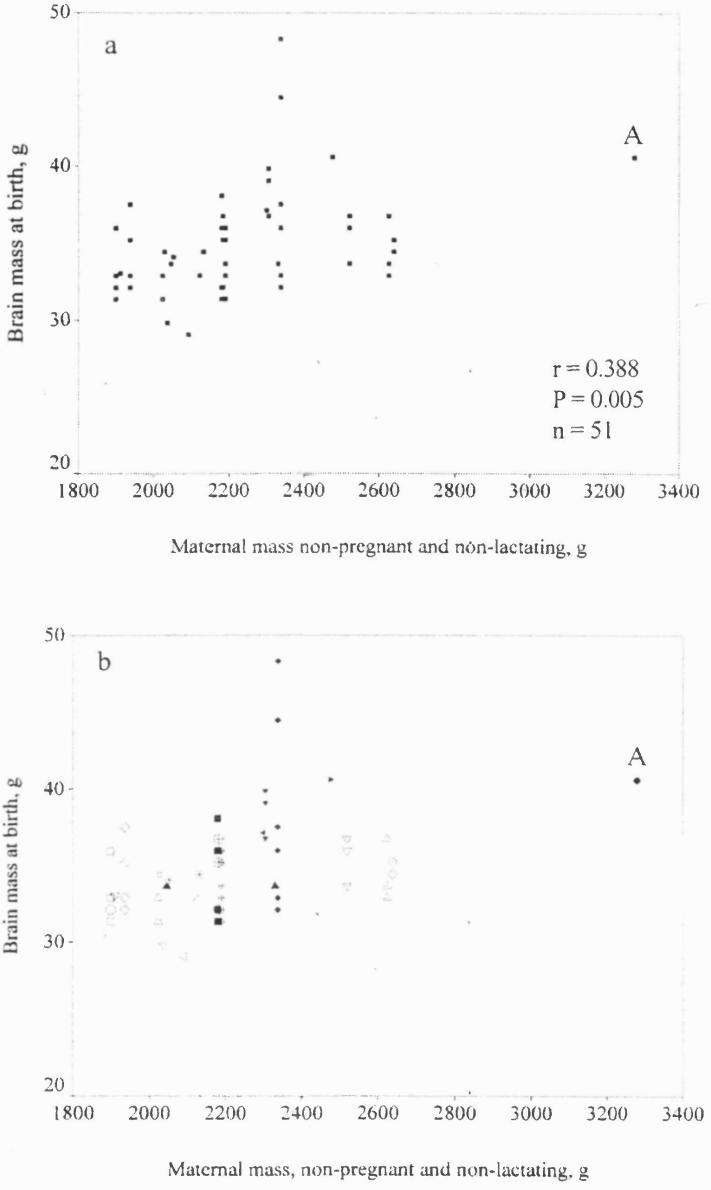


Figure 5.3. Maternal mass when non-pregnant and non-lactating predicts brain mass at birth. (a) maternal mass when non-pregnant and non-lactating (MM_n) vs. brain mass at birth (BR_b). Infant marked A is discussed in the text. (b) maternal mass when non-pregnant and non-lactating (MM_n) vs. brain mass at birth (BR_b); infants marked with the same symbols are siblings. Infant marked A is discussed in the text.

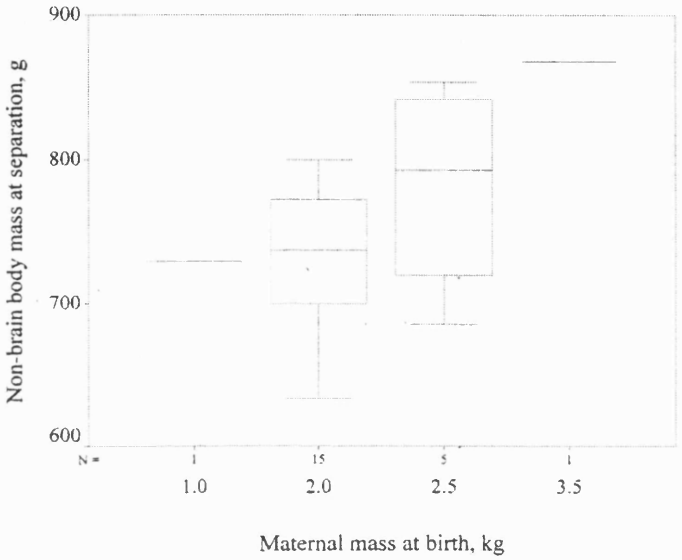


Figure 5.4. Maternal mass at birth (MM_b) predicts infant non-brain body mass at separation ($NBBM_s$). Maternal masses at birth are grouped into half-kilogram intervals.

significant association between the size of the mother immediately after birth and the non-brain body mass achieved at the infant by the end of lactation (Fig 5.4). This result supports other research that links maternal condition or body fat reserves (here represented by mass) during reproduction with investment in infant growth over lactation (see Lee et al. 1991).

The correlation between maternal mass when non-pregnant and non-lactating with brain mass at separation (Table 5.5.2) only just passes the significance threshold (P = 0.044), and we might predict that brain size at separation is associated with maternal mass primarily because it is associated with brain size at birth. Indeed, when these three variables are entered into a partial correlation, the significance of both correlations disappears (Table 5.5.6). It appears that target brain size, at least at birth, is 'set' by the general size of the mother over her entire reproductive career. The amount of non-brain body growth achieved by each infant is influenced by maternal mass at the beginning of the specific lactation period.

3] Is maternal mass associated with infant ~~metabolic rate~~ oxygen consumption during ontogeny?

As we might predict from the results presented in answer to Question 2, maternal mass when non-pregnant and non-lactating (MM_n) is correlated with ~~metabolic~~ brain oxygen consumption rate, but only at separation (Brain MR_s; Table 5.6.1). This is interesting because, in the correlations above, maternal mass was much more strongly associated with brain mass at birth. This result might indicate that a secondary association of maternal mass exists with the rate of ~~energy~~ oxygen consumption by the infant brain at the time of separation. The sample size for this correlation is, however, small, and the result only just significant. When brain mass at separation (BR_s) is held constant, the significance of the correlation disappears, as might be expected, as brain ~~MR~~ $\dot{V}O_2$ is calculated from brain mass (Table 5.6.2). Maternal mass at birth (MM_b) is significantly correlated with the total ~~metabolic rate~~ oxygen consumption rate of the infant at separation (Total ~~MR_s~~ $\dot{V}O_2$; Table 5.6.3). This association also disappears when non-brain body mass at separation ($NBBM_s$) is held constant (Table 5.6.4), but the correlation suggests that brain and body size are modulated

Table 5.6 Metabolic rate correlates of maternal mass

	Correlation between...	...and...	...controlling for...	r	r^2	P	n
5.6.1	Maternal mass non-pregnant and non-lactating (MM_n)	Brain metabolic rate at separation (Brain ~~MR_s~~ VO_2)	-	**0.432**	**0.187**	**0.045**	**22**
5.6.2	Maternal mass non-pregnant and non-lactating (MM_n)	Brain metabolic rate at separation (Brain ~~MR_s~~ VO_2)	Brain mass at separation (BR_s)	-0.119	0.014	0.609	19
5.6.3	Maternal mass at birth (MM_b)	Total metabolic rate at separation (Total ~~MR_s~~ VO_2)	-	**0.443**	**0.196**	**0.039**	**22**
5.6.4	Maternal mass at birth (MM_b)	Total metabolic rate at separation (Total ~~MR_s~~ VO_2)	Non-brain body mass at separation ($NBBM_s$)	0.159	0.025	0.551	19

See text for discussion.

ERRATUM: FOR 'METABOLIC RATE' READ 'OXYGEN CONSUMPTION'

such that the total infant ~~metabolic~~ oxygen consumption rate at separation is determined in part ($R^2 = 0.196$) by maternal mass at birth.

4] Is maternal mass associated with age at separation?

Maternal mass at conception (MM_c) is the only maternal mass parameter to correlate significantly with age at separation (S_{age}; Table 5.7.1), explaining approximately 25% of variation in this latter variable. Maternal mass at the beginning of each reproductive event is therefore important in determining the rate at which nutrients are transferred and target size attained, even in the postnatal period. The larger the mother, the shorter the time to separation (Figure 5.5). This contrasts with the interspecific results of Lee *et al.* (1991), which found that maternal mass and lactation length are positively correlated in primates.

Maternal mass at conception also correlates significantly with the rates at which the infant brain, non-brain, and whole body increase in mass before separation (Table 5.7.2, 5.7.3, 5.7.4). The other two maternal mass parameters (MM_b, MM_c) correlate only with the pre-separation WBM rate (Table 5.7.5, 5.7.6). However, when age at separation is held constant, all these latter associations become non-significant, as might be expected (i.e. because all rates are calculated from age at separation; data not shown).

5] Is encephalisation at birth a good predictor of brain and body size at separation?

When compared to the other variables in the analysis that are significantly correlated with $NBBM_s$, encephalisation at birth (ENC_b) is the best predictor of non-brain body mass at separation ($NBBM_s$) ($R^2 = 0.211$; Table 5.8.1). This relationship is negative i.e. the larger the brain relative to body mass at birth, the smaller the mass of the non-brain tissues at separation (Fig 5.6). This relationship is not significant when the outlier infant (marked A in Figure 5.6) is removed from the analysis, but the P value is very close to the significance threshold ($r = -0.416$, $P = 0.06$, $df = 21$). The next best predictor of non-brain body mass at separation is non-

Table 5.7 Growth rate correlates of maternal mass

	Correlation between...	...and...	...controlling for...	r	r^2	P	n
5.7.1	Maternal mass at conception (MM_c)	Age at separation (S_{age})	-	**-0.496**	**0.246**	**0.005**	**31**
5.7.2	Maternal mass at conception (MM_c)	Pre-separation BR rate	-	**0.519**	**0.270**	**0.013**	**22**
5.7.3	Maternal mass at conception (MM_c)	Pre-separation NBBM rate	-	**0.604**	**0.365**	**0.003**	**22**
5.7.4	Maternal mass at conception (MM_c)	Pre-separation WBM rate	-	**0.314**	**0.099**	**0.035**	**45**
5.7.5	Maternal mass at birth (MM_b)	Pre-separation WBM rate	-	**0.295**	**0.087**	**0.049**	**45**
5.7.6	Maternal mass non-pregnant and non-lactating (MM_n)	Pre-separation WBM rate	-	**0.361**	**0.130**	**0.015**	**45**

See text for discussion.

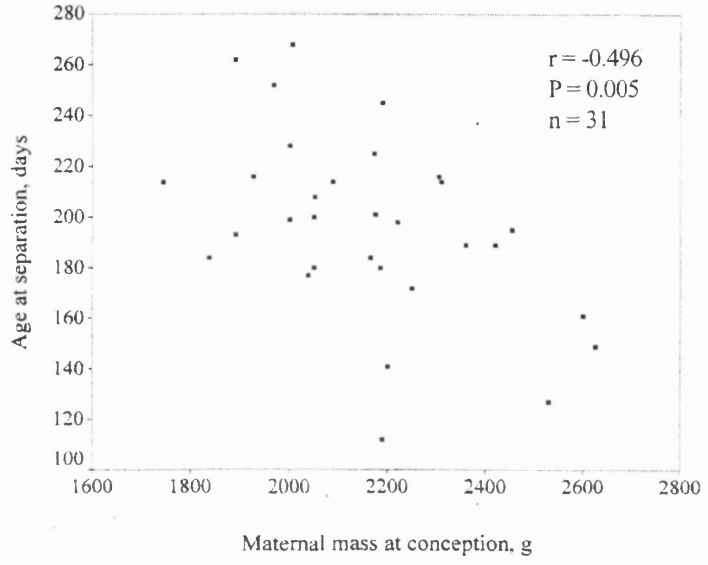


Figure 5.5. Maternal mass at conception (MM_c) correlates with age at separation (S_{age}).

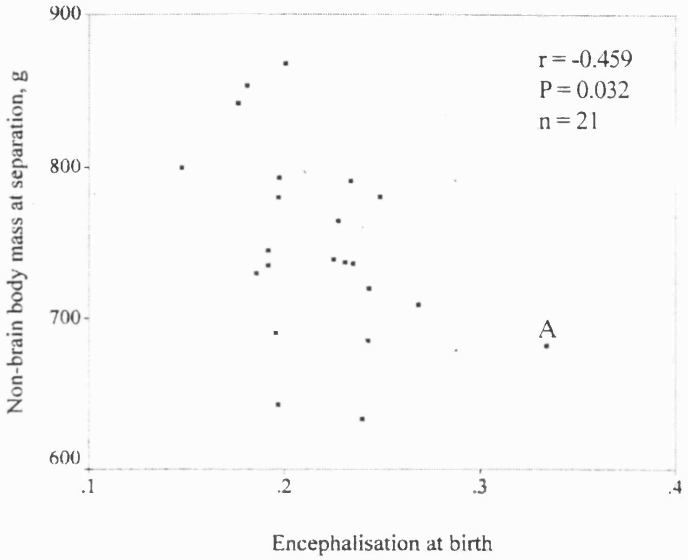


Figure 5.6. Encephalisation at birth (ENC_b) predicts non-brain body mass at separation ($NBBM_s$). Infant marked A is discussed in the text.

Table 5.8 Encephalisation correlates in comparison with other correlations

	Correlation between...	...and...	...controlling for...	r	r^2	P	n
5.8.1	Encephalisation at birth (ENC_b)	Non-brain body mass at separation ($NBBM_s$)	-	**-0.459**	**0.211**	**0.032**	**21**
5.8.2	Non-brain body mass at birth ($NBBM_b$)	Non-brain body mass at separation ($NBBM_s$)	-	**0.457**	**0.209**	**0.032**	**22**
5.8.3	Encephalisation at birth (ENC_b)	Whole body mass at separation (WBM_s)	-	**-0.431**	**0.247**	**0.045**	**21**
5.8.4	Encephalisation at birth (ENC_b)	Encephalisation at separation (ENC_s)	Brain mass at separation (BR_s)	0.376	0.141	0.093	19
5.8.5	Encephalisation at birth (ENC_b)	Encephalisation at separation (ENC_s)	Non-brain body mass at separation ($NBBM_s$)	**0.623**	**0.388**	**0.003**	**19**

See text for discussion.

brain body mass at birth ($NBBM_b$), which explains only slightly less variation ($R^2 = 0.209$; Table 5.8.2). This is to be expected, when $NBBM_b$ is one of the components of encephalisation at birth. The more encephalised the infant at birth, the smaller the whole infant will be at separation, as reflected by the negative (but not highly significant; $P = 0.045$) correlation of encephalisation at birth and whole body mass at separation (Table 5.8.3).

These correlations are to be expected, as encephalisation is calculated from the ratio of brain to body size. Many are unlikely to be independent correlations. When brain mass at separation is held constant, for example, the association of encephalisation at birth and separation becomes non-significant (Table 5.8.4). On the other hand, when non-brain body mass at separation is held constant, the relationship between encephalisation at birth and at separation remains strongly significant (Table 5.8.5). This suggests that brain size at birth and separation covary independently of body size.

The only significant correlation between encephalisation and the rate of growth is between encephalisation at birth and whole body mass growth rate (Table 5.9.1). However, when non-brain body mass at birth is held constant, this relationship disappears (Table 5.9.2), suggesting that the rate of body growth is influenced by the absolute size of the non-brain tissues more than by encephalisation (i.e. relative size).

6] ~~Are metabolic rates~~ Is oxygen consumption associated with growth variables?

None of the ~~MR~~ $\dot{V}O$ variables show a significant relationship with age at separation (data not shown). Total ~~MR~~ $\dot{V}O$ at separation is highly correlated with whole body mass at separation, and this relationship remains highly significant when non-brain body mass at separation is held constant (Table 5.10.1, 5.10.2). The ~~metabolic~~ oxygen consumption rates of the brain at birth and separation are correlated with the BR ratio (Table 5.10.3, 5.10.4), but this association disappears when brain mass is taken into account (Table 5.10.5, 5.10.6). The ~~metabolic~~ oxygen consumption rates used in this analysis are not associated with any growth variables except the masses from which they were calculated.

Table 5.9 Growth rate correlates of encephalisation

	Correlation between...	...and...	...controlling for...	r	r^2	P	n
5.9.1	Encephalisation at birth (ENC_b)	Pre-separation WBM rate	-	**-0.532**	**0.283**	**0.001**	**35**
5.9.2	Encephalisation at birth (ENC_b)	Pre-separation WBM rate	Non-brain body mass at birth ($NBBM_b$)	0.112	0.013	0.562	27

See text for discussion.

Table 5.10 ~~Metabolic rate~~ Oxygen consumption **correlates**

	Correlation between...	...and...	...controlling for...	r	r^2	P	n
5.10.1	Total MR at separation (Total MR_s)	Whole body mass at separation (WBM_s)	-	**0.996**	**0.992**	**0.000**	**22**
5.10.2	Total MR at separation (Total MR_s)	Whole body mass at separation (WBM_s)	Non-brain body mass at separation ($NBBM_s$)	**0.990**	**0.980**	**0.000**	**19**
5.10.3	BR ratio	Brain MR at birth (BR_b)	-	**-0.737**	**0.543**	**0.000**	**22**
5.10.4	BR ratio	Brain MR at separation (BR_s)	-	**-0.834**	**0.700**	**0.000**	**22**
5.10.5	BR ratio	Brain MR at birth (BR_b)	Brain mass at birth (BR_b)	0.321	0.103	0.156	19
5.10.6	BR ratio	Brain MR at separation (BR_s)	Brain mass at separation (BR_s)	-0.257	0.066	0.912	19

See text for discussion.

ERRATUM: FOR 'MR' READ 'VO'.

5.2 Summary

Maternal mass when non-pregnant and non-lactating is associated with the size of the infant brain at birth, but not with the size of the body. Larger mothers tend to give birth to larger-brained infants. This supports earlier work that suggests that the size or metabolic capacity of the mother that determines the size of the infant brain *in utero* (Martin, 1983; Martin, 1996). Maternal mass when non-pregnant and non-lactating also correlates with the ~~metabolic~~ oxygen consumption rate of the infant's brain at separation. Although not statistically robust, this association might indicate a secondary relationship between maternal mass and the metabolic demands of the infant brain at separation. Infants with larger mothers have larger brains with absolutely and relatively (i.e. with infant non-brain body mass held constant) higher ~~energy~~ oxygen demands at separation. On the other hand, maternal mass at birth – i.e. at the end of gestation – is an important indicator of maternal investment over lactation, notably in influencing the size of the non-brain tissues at separation. The size of the brain at birth, and hence at separation, is largely set by the general size of the mother, whilst the amount of body growth attained by the infant depends on maternal size or condition at the beginning of lactation. This ties in with the result that encephalisation is the best predictor of non-brain body mass at separation. The size of the brain relative to the rest of the body is important in determining how much of the energy supply goes to fuelling the non-brain tissues. The high energy demand of large infant bodies does not appear to compromise maternal energy supplies: larger mothers are capable of supplying their infants' higher energy needs over a shorter time period. Approximately 25% of variation in age at separation is explained by variation in maternal mass and larger mothers wean their infants at an earlier age.

~~Metabolic~~ Oxygen consumption rates do not appear to be significantly associated with any growth variables except size, as might be expected as the ~~metabolic~~ rates are calculated from the size variables. It is likely that the non-maintenance costs associated with growth (i.e. the energy costs of producing and laying down new tissue (Davies *et al.*, 1997) – which, by necessity are not included in this chapter's modelling – play an important role in determining maternal

investment. However, this analysis shows that the relationship of total ~~metabolic~~ oxygen consumption rate at separation and whole body mass at separation remains significant even when the size of the non-brain tissues at separation are held constant. This suggests that the total ~~metabolic cost~~ oxygen consumption of the infant at separation is influenced not just by the size of the non-brain tissues, but also independently by the size of the brain itself. This ties in with the idea, presented in earlier chapters, that brain mass, as well as body mass, is an important determinant of the weaning threshold in primates. The next chapter models the interactions of the growth variables outlined here in the more general context of capuchin life histories.

The observation, noted in this chapter, that maternal size and oxygen consumption are associated in this group of primates does not in itself provide evidence that the maternal energy hypothesis is the appropriate metabolic model to use. Alternative explanations might be invoked to explain the association; for example, that size and therefore metabolic rate, and its proxy oxygen consumption, are highly heritable from mother to infant (Sinclair & Daingerfield, 1998).

CHAPTER 6

CAPUCHIN GROWTH AND MATURATION IN THE CONTEXT OF LIFE HISTORY

This chapter integrates the Chilean Primate Centre (CPC) brain and body size data presented in the previous chapter with other life history and lactation parameters from the same *C. apella* colony. Firstly, capuchin life histories are introduced (Section 6.1). Secondly, the results of a principal components analysis, and of correlation and partial correlation analyses, are used to model the interaction between growth and other variables (Section 6.2). Finally, the limitations of the model are outlined (Section 6.3), and some of the implications the results have for life history variation are discussed (Section 6.4).

6.1 Life histories: body size, achievement of maturity and rates of increase

As we saw in the Introduction, life history theory predicts that the size of an animal, the time it takes to mature, and the frequency with which it reproduces are interrelated (Charnov, 1991; Stearns, 1992). The interaction of these three parameters has implications for the speed at which a population grows. The maximum intrinsic rate of natural increase (r_{max} or r_m) is the rate at which a population is predicted to grow in a hypothetical environment of unlimited resources, and is calculated as a function of three variables that summarise the reproductive capacity of individuals within a population (Ross & Jones, 1999):

1] The rate at which a typical female produces offspring.

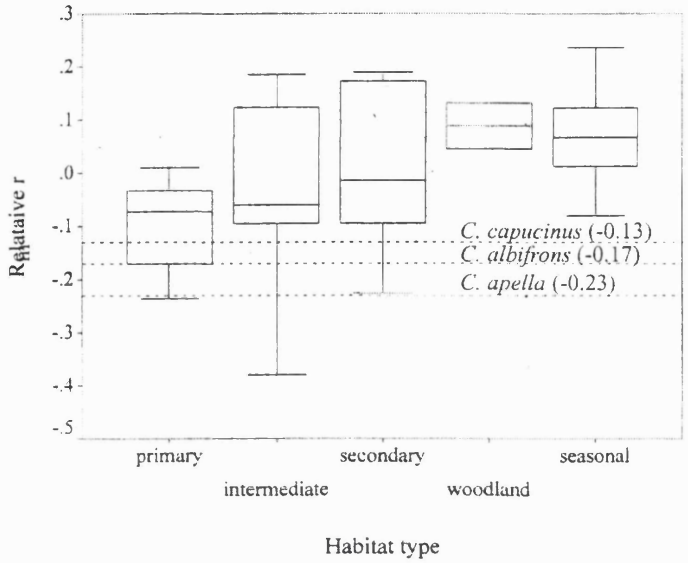
2] The length of time in a female's lifespan in which she is able to reproduce.

3] Average female body mass.

These three traits tend to covary. Animals can show a mixture of fast and slow life history strategies, but in general the 'speed' at which a taxon's life history runs is predicted to correlate with the environment in which it finds itself (Stearns, 1992). Animals in stable, predictable, resource-rich and low-mortality environments are usually large, slow-growing,

slow-reproducers (low r_m) who are able to offset the costs of extended development with the benefits of reproduction later in the lifespan (Pagel & Harvey, 1993). Animals that live in low-quality, resource-poor and high-mortality habitats will be small, fast-growing, fast-reproducers (high r_m) who reap the rewards of early and frequent reproduction but who are more susceptible to mortality pressures (Charnov, 1991; Charnov & Berrigan, 1993). This association of habitat type and r_m has been observed in primates (Rasmussen, 1985; Ross, 1991; Ross, 1992; Pagel & Harvey, 1993), although a more recent phylogenetically-controlled analysis (Ross & Jones, 1999) has failed to find robust statistical support for some of Charnov's (1991) predictions. However, on the whole, when body size is controlled for, taxa living in woodland or highly seasonal habitats tend to have higher r_m values than those living in primary, secondary or intermediate forests (Fig 6.1). Capuchins have low r_m values when compared with the majority of other primates that live in secondary or intermediary habitats. The pace of capuchin life histories is therefore slow even when body mass at habitat are controlled for (Fig 6.1).

What factors contribute to the low r_m seen in *Cebus*? Charnov & Berrigan (1993) suggest that "the key to slow primate life histories lies in the slow growth of juveniles [i.e. after infancy has ended]"(p 191). Ross (1991) also notes that "the late maturity of [these] species [i.e. the cebids] is not due to a relatively low infant growth rate but, instead, is linked to a prolonged time between weaning age and sexual maturity [i.e. low growth rates in juveniles]" (Ross 1991: 492). Capuchins do reach reproductive maturity at a later age than predicted for a primate of their body size (Fig 6.2a). Age at first reproduction for a 2.5kg primate is typically three years and three months old, but mean age of first reproduction is four years in *C. albifrons* and *C. capucinus*, five and a half in *C. apella* (Ross, 1992). Capuchins also have a relatively long lifespan for their body size; maximum recorded longevity is at least 40 years, comparable with the lifespan of much larger taxa such as *Papio*, *Mandrillus*, and even *Gorilla* (Ross, 1992). In other words, the whole schedule of capuchin life history is extended.



Habitat type:	Taxa included (see Ross 1992):
Primary	*Ateles geoffroyi, A. fusciceps; A. paniscus, Lagothrix lagotricha, Cercopithecus diana, Macaca silenus, Pongo pygmaeus*
Intermediate	*Callithrix argentata, Saguinus fuscicollis, Leontopithecus rosalia, Callicebus moloch, Pithecia pithecia, Alouatta palliata, Cercopithecus ascanius, C. mitis, C. neglectus, Cercocebus albigena, C. atys, C. torquatus, C. niger, Macaca arctoides, M nemestrina, Mandrillus leucophaeus, M. sphinx, Colobus badius, C. guereza, C. polykomos, Hylobates lar, Symphalangus syndactylus.*
Secondary	*Callithrix jacchus, Cebuella pygmaeus, Saguinus midas, Callimico goeldii, Aotus trivirgatus, Alouatta seniculus, Saimiri sciureus, Miopithecus talapoin, Macaca sinicus, Gorilla gorilla, Pan troglodytes*
Woodland	*Saguinus oedipus, Macaca fascicularis*
Seasonal	*Cercopithecus aethiops, Erythrocebus patas, Macaca fuscata, M. mulatta, M. sylvanus, Papio cynocephalus, P. hamadryas, T. gelada, P. entellus.*

Figure 6.1. Relative r_m is associated with habitat type in primates. r_m is expressed relative to body mass. Based on a figure in, and data taken from, Ross (1992).

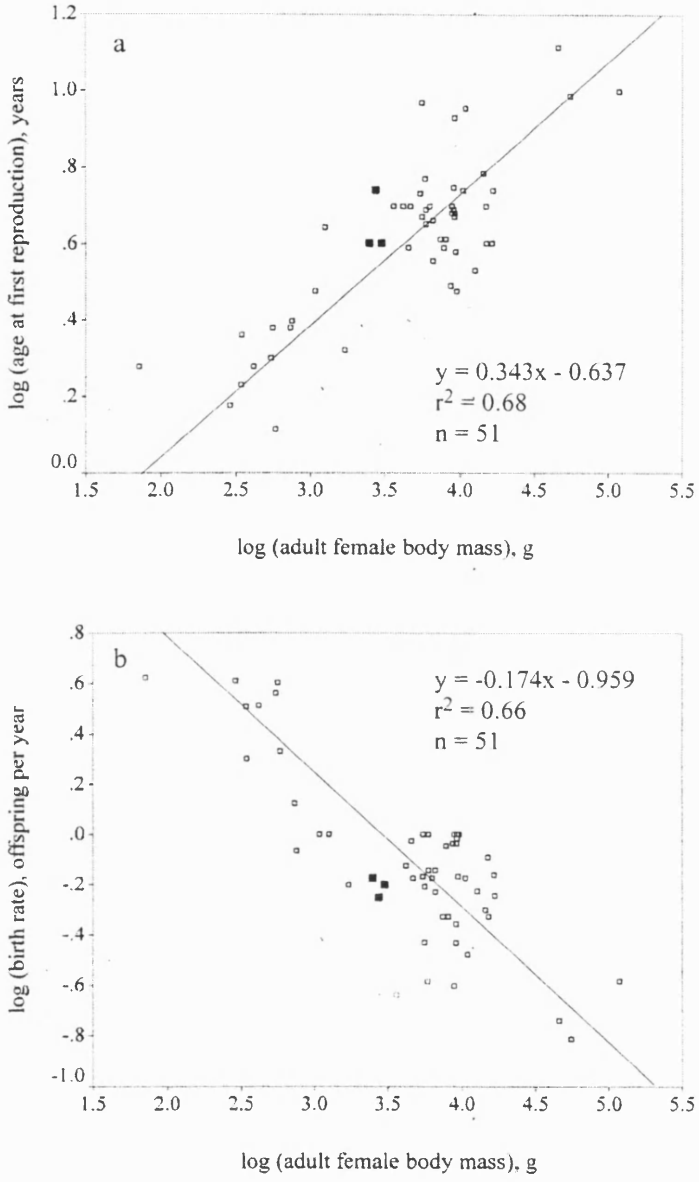


Figure 6.2. Age at first reproduction and birth rate in capuchins compared to other primate taxa. (a) Age at first reproduction; (b) birth rate. Capuchins data indicated by closed symbols. All data taken from Ross (1992). Lines fitted are RMAs. Only haplorhines are included in each plot.

Capuchin birth rates are also low; reported birth rate varies from 0.56 births per year for *C. apella* to 0.67 for *C. albifrons* (Ross, 1992). This is equivalent to an interbirth interval of 1.49 years for *C. apella* and 1.78 years for *C. albifrons*. In this respect, capuchins are similar to the much larger platyrrhines (e.g. *Alouatta*, *Ateles*, *Lagothrix*) who display interbirth intervals of well over one year (Fragaszy *et al.*, 1991), and to many of the large cercopithecines (Smuts *et al.*, 1987). Capuchin birth rates fall below the RMA fitted to other haplorhine species (Fig 6.2b).

How does infant growth fit into this life history picture? The analysis presented below uses maternal, paternal and infant growth data from the Chilean Primate Centre (CPC) to investigate the interaction of growth rates, time to weaning and other life history parameters. It asks two questions:

1] Which factors influence the timing and pattern of infant postnatal growth before and after weaning?

2] How does infant growth contribute to the pattern of delayed life histories seen in capuchins?

6.2 Modelling life history variation

Some of the data used in this chapter was presented in Chapter 5. All data are taken from a colony of captive *Cebus apella* housed in the Chilean Primate Centre, Chile (CPC). The capuchins are second-generation laboratory-conceived and –delivered, and exact age is known for all individuals, both in terms of gestational age (via hormone radioimmunoassays, (Recabarren *et al.*, in preparation)) and postnatal chronological age. Colony females of reproductive age do not get to choose their mates, nor the timing of conception, as the laboratory requires planned pregnancies for experimental procedures. This analysis is not intended to be a study of naturalistic female/male interaction or female choice; rather, it examines the effect of variation in maternal and paternal parameters, e.g.

body mass, on infant growth and development. Details of rearing conditions and diet are outlined in Chapter 5.

6.2.1 Materials

Data were gathered from 10 years of CPC colony records, as described in Chapter 5. In addition to the variables outlined in the previous chapter, five other parameters were collected from the records:

1] GEST Gestation length, (days).

Gestation length was not known for 4 infants, and in these individuals, mean gestation length (156.68d) was substituted.

2] $NBBM_t$ Non-brain body mass at 1000dpp, (grams).

Adult body masses were available for a minority of individuals. In order to maximise the dataset mass at 1000dpp (2.7y) was used as a measure of post-separation body mass. Head breadth after separation was not known for any infant and brain mass at separation could not be directly inferred (see Chapter 5). For the purpose of the life history model it was assumed that brain mass at 1000dpp could be estimated from brain mass at separation (BR_s):

$$\text{Brain mass at 1000dpp} = BR_s * 1.10 \qquad (6.1)$$

This equation is based on the assumption that brain mass at separation is 90% of brain mass at 1000dpp (Chapter 2). Non-brain body mass at 1000dpp was then calculated as:

Non-brain body mass at 1000dpp = whole body mass at 1000dpp –

brain mass at 1000dpp

(6.2)

3] SIBS Number of older siblings.

This variable includes all live and dead siblings of the present infant, i.e. all previous pregnancies of the mother. Thus an infant born to a primiparous mother was scored as zero. This measure is in most cases an indicator of minimum maternal investment because 1] the parity of the females imported into the original breeding colony between 1989 and 1991 was not always known and 2] one quarter of the infants born in the colony (27% of all births) were removed some time after birth, either to be donated to other colonies, or to encourage the mothers to begin cycling again as soon as possible. Maternal investment was therefore curtailed in the mothers of these infants. The results of the following analyses were not affected when the number of live siblings was substituted for number of live and dead siblings (data not shown).

4] MA_c Maternal age at conception (months).

5] PM_c Paternal mass at conception (grams).

Extrapolated from growth curves at date of conception. Male body masses were gathered by the CPC staff on an *ad hoc* basis, usually when semen was collected for sperm count analysis.

The variables analysed in this chapter are summarised in Table 6.1.

6.2.2 Methods

The model was constructed in three stages:

1] A PCA analysis was performed on twelve variables that represent maternal mass and previous investment, paternal mass, and infant mass and age variables (Table 6.2). Unlike in Chapter 5, whole body masses were not entered into the analysis, because they were found to correlate highly with non-brain body masses. Similarly, maternal age at conception (MA_c) was not entered into the PCA because it was found to correlate highly with number of older siblings (SIBS, see below). The axes were rotated using the varimax rotation method.

2] The results of the PCA were used to assign variables to groups that describe distinct sources of variation in the dataset. Correlations between variables were calculated (Pearson's correlations, two-tailed significance, $P \leq 0.05$). Partial correlation analyses were performed on certain pairs of variables (discussed in the text) holding others in the group constant. This identified those variables that were related independently of other parameters.

3] The results of these partial correlations formed the basis of a model that describes the interaction of growth and other variables.

6.2.3 Results: I. Principal Components Analysis

Four principal components (PCs) were extracted (Table 6.2) which together explain 77.52% of variation in the CPC dataset.

1] PC 1

PC 1 explains 22.82% of the variation in the dataset (Table 6.2). The first component in a PCA is usually associated with variation in size (Shea, 1985). In this analysis it is associated with brain and body size at the beginning and end of lactation, i.e. with brain

TABLE 6.1 Variables used in the life history modelling

Variable	Abbr.
Age at separation	S_{age}
Brain mass at birth	BR_b
Brain mass at separation	BR_s
Gestation length	GEST
Maternal age at conception	MA_c
Maternal mass at birth	MM_b
Maternal mass at conception	MM_c
Maternal mass non-pregnant, non-lactating	MM_n
Non-brain mass at 1000dpp	$NBBM_t$
Number of siblings	SIBS
Paternal mass at conception	PM_c
Non-brain body mass at birth	$NBBM_b$
Non-brain body mass at separation	$NBBM_s$

TABLE 6.2 PCA results: variable loadings

		Loadings			
Variable	Abbr.	PC 1	PC 2	PC 3	PC 4
Non-brain body mass at birth	$NBBM_b$	0.802	0.223	0.026	-0.100
Paternal mass at conception	PM_c	0.759	0.102	-0.021	0.008
Brain mass at separation	BR_s	-0.746	0.495	-0.010	0.183
Brain mass at birth	BR_b	-0.669	0.618	-0.048	0.167
Age at separation	S_{age}	-0.113	-0.829	0.190	0.0802
Maternal mass at conception	MM_c	0.138	0.734	0.411	-0.313
Number of siblings	SIBS	-0.161	-0.336	0.780	-0.139
Maternal mass at birth	MM_b	0.088	0.251	0.771	0.382
Non-brain body mass at separation	$NBBM_s$	0.497	-0.014	0.637	-0.043
Maternal mass when non-pregnant and non-lactating	MM_n	-0.378	0.543	0.588	-0.179
Gestation length	GEST	-0.046	-0.019	0.201	0.892
Non-brain body mass at 1000dpp	$NBBM_t$	0.239	0.287	0.373	-0.765

It should be noted that a high ratio of variables to data points in statistical models artificially inflates the proportion of variance explained. However, similar results were obtained when this analysis was re-run using subsets of variables (data not shown).

size at birth (BR_b) and at separation (BR_s), and with non-brain body mass at birth ($NBBM_b$) and separation ($NBBM_s$). Brain and body masses are opposed on PC 1: infants with large brains tend to have small bodies at both birth and separation. Neither brain nor body mass at 1000dpp load strongly on PC 1 (Table 6.2). This indicates that the most important source of variation among the CPC infants relates to the infant brain and body proportions (i.e. encephalisation) prior to separation. The other variable that loads strongly on PC 1 is paternal mass at conception (PM_c) (Fig 6.3). PC 1 distinguishes infants that are encephalised as neonates and at separation, with small fathers, from those that are non-encephalised at birth, with large fathers. None of the maternal mass variables loads strongly on this component (Table 6.2).

2] PC 2

PC 2 explains almost as much of total variation as PC1 (20.55%, Table 6.2). Maternal mass at conception (MM_c) and when non-pregnant and non-lactating (MM_n) load strongly on this PC, as does brain mass at birth and brain mass at separation (BR_b, BR_s). This second significant source of variation therefore arises from the covariance of maternal mass and infant brain mass. Infants with larger mothers tend to have larger brains independently of body size (Fig 6.3). In addition, age at separation loads strongly and negatively on PC 2 (Table 6.2): infants that have large mothers and large brains tend to wean earlier (Fig 6.3).

3] PC 3

PC 3 also accounts for just under 20% (19.53%) of total variation in the dataset (Table 6.2). Body size at separation ($NBBM_s$) loads strongly on this PC, and it therefore distinguishes infants that are large at separation from those that are small at separation (Fig 6.4). It also shows that maternal mass at birth (MM_b), maternal mass when non-pregnant and non-lactating (MM_n) and previous maternal investment (measured as number of siblings, SIBS) are all associated with the infant body mass at separation.

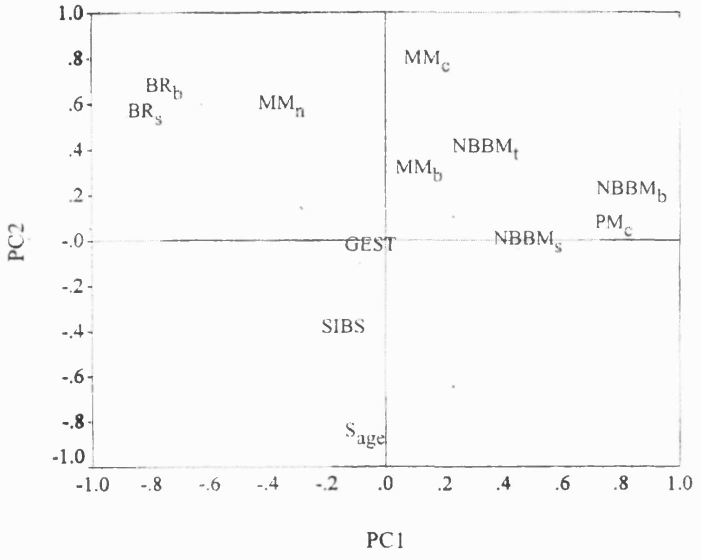


Figure 6.3. PCA results: variable loadings on PC1 and PC2.
See Table 6.2 for data and abbreviations.

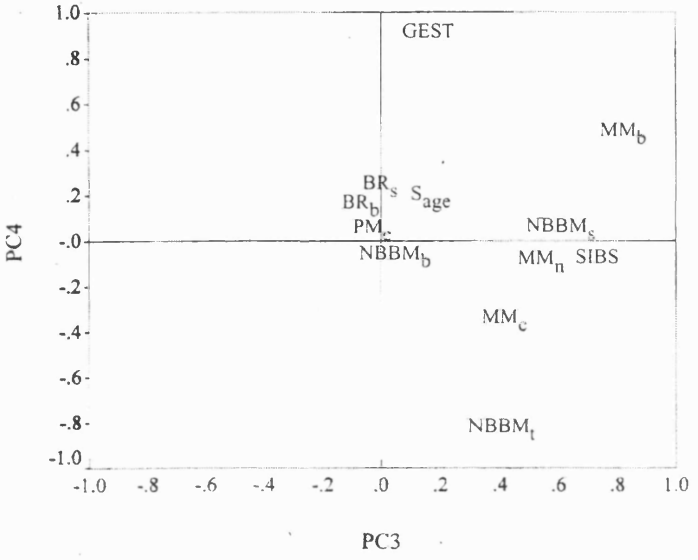


Figure 6.4. PCA results: variable loadings on PC3 and PC4.
See Table 6.2 for data and abbreviations.

4] PC 4

PC4 indicates that the last significant source of variation (14.63%) in the CPC dataset comes from the association of gestation (GEST) and body mass at 1000dpp ($NBBM_t$). Infants that are gestated for a relatively short period go on to be relatively large at 1000dpp, and vice versa (Fig 6.4).

6.2.4 Results: II. Investigating the PCA groups

1] Group 1: brain and non-brain body mass at birth and separation (BR_b, $NBBM_b$, BR_s, $NBBM_s$), paternal mass at conception (PM_c).

PC 1 separates infants that are relatively encephalised (large brain masses, small non-brain body masses) from those that are relatively non-encephalised (small brain masses, large body masses), both at birth (Fig 6.5a) and at separation (Fig 6.5b). There is a tighter association between PC 1 score and encephalisation at birth ($r^2 = 0.736$; Fig 6.5a) than between PC 1 and encephalisation at separation ($r^2 = 0.517$; Fig 6.5) that is statistically significant ($F = 17.82$, $P < 0.001$, $df = 21$). When encephalisation at birth is held constant, the relationship between PC 1 and encephalisation at separation is not significant (Table 6.3.1). PC 1 remains significantly correlated with encephalisation at birth when encephalisation at separation is held constant (Table 6.3.2), suggesting that the most important source of variation identified by PC1 is, in fact, variation in neonatal brain and body mass.

Paternal mass at conception (PM_c) is positively correlated with infant non-brain body mass at birth ($NBBM_b$; Table 6.3.3). Paternal mass at conception is also significantly correlated with brain mass at separation (BR_s), but the association is weak ($P = 0.048$), and when non-brain body mass at birth ($NBBM_b$) is held constant, the correlation disappears (Table 6.3.4). Larger males tend to father infants with heavier non-brain tissues at birth, and infants which have relatively large bodies at birth have relatively small brains at birth. Paternal mass appears to influence the size of the non-brain tissues at birth, with brain mass

TABLE 6.3 PC1 and Group 1 correlations

	Correlation between...	...and...	...controlling for...	r	r^2	P	n
6.3.1	PC1	Encephalisation at separation	Encephalisation at birth	-0.400	0.160	0.072	19
6.3.2	PC1	Encephalisation at birth	Encephalisation at separation	**-0.735**	**0.540**	**0.000**	**19**
6.3.3	Paternal mass at conception (PM_c)	Non-brain body mass at birth ($NBBM_b$)	-	**0.315**	**0.010**	**0.026**	**50**
6.3.4	Paternal mass at conception (PM_c)	Brain mass at separation (BR_s)	Non-brain body mass at separation ($NBBM_b$)	-0.282	0.080	0.215	19

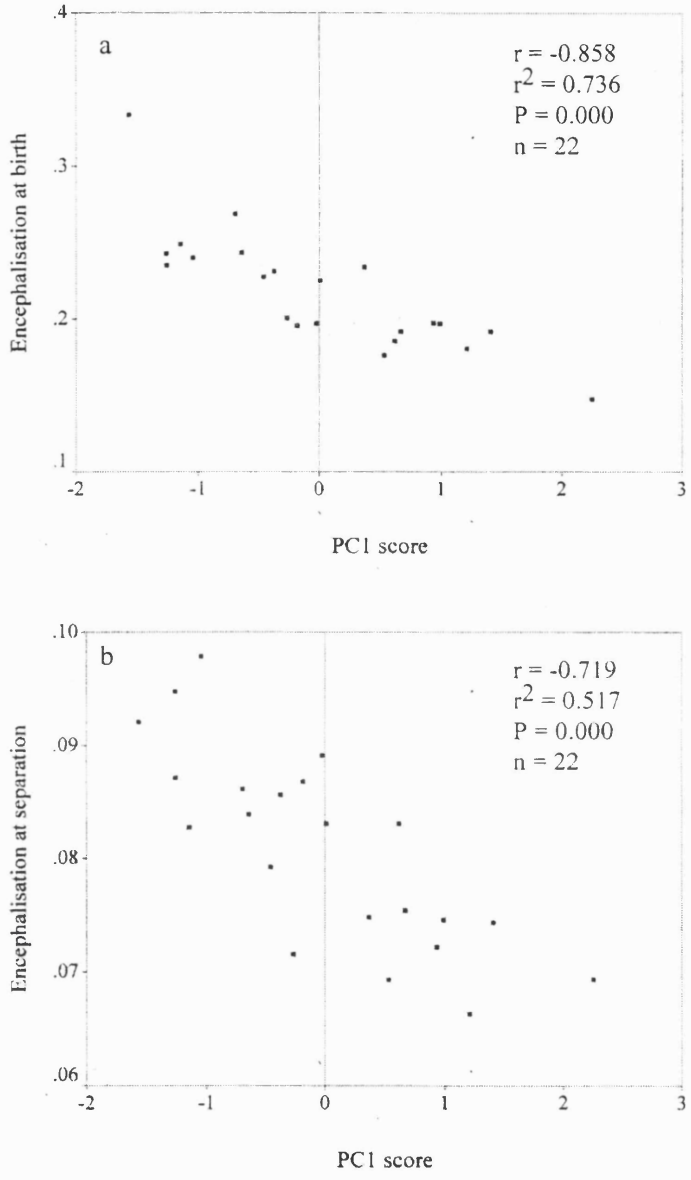


Figure 6.5. PC1 describes encephalisation during ontogeny. Encephalisation at (a) birth (ENC_b) and (b) separation (ENC_s) plotted against PC1 score.

at birth covarying accordingly; paternal mass also influences infant brain mass at the end of lactation, primarily through the association of infant brain mass at separation with body mass at birth.

This correlation of paternal and infant mass may be biased by a few large or small males fathering a disproportionate number of infants in the colony. An examination of the individual datapoints for non-brain body mass at birth ($NBBM_b$) regressed against paternal mass at conception (PM_c; Fig 6.6) suggests that this might be the case. Paternity is known for 62 infants, and 13 males fathered all of the infants in the colony. The modal number of offspring per father is 1 (n = 7), but 3 males fathered 63% of the infants in the colony, with an average of 13 infants each. Infants with the same fathers are marked with similar symbols in Figure 6.6b. Mass at conception for one of the males (indicated by open triangles in Figure 6.6b) is extrapolated for each infant from only two mass records; the relationship between his mass at conception and his infants' masses at birth does not, therefore show much variation. However, when the data for this particular male's infants are excluded, the amount of explained variation (R^2) increases from 10.0% to 12.7%, and the association remains significant (Fig 6.6b). The correlation between paternal mass at conception and mean $NBBM_b$ (i.e. averaged over all siblings for each male) is highly significant ($r = 0.645$, $P = 0.000$, $df = 51$).

2] Group 2: Maternal mass at conception (MM_c) and when non-pregnant and non-lactating (MM_n), brain mass at birth and separation (BR_b, BR_s), and age at separation (S_{age}).
In addition to their association with paternal mass, brain mass at birth and separation (BR_b, BR_s) show a secondary association with two measures of maternal mass: maternal mass at conception (MM_c) and maternal mass when non-pregnant and non-lactating (MM_n; Fig 6.3). However, when maternal mass when non-pregnant and non-lactating (MM_n) is held constant, the association of maternal mass at conception and brain mass at birth disappears (Table 6.4.1). This suggests that brain mass at birth and maternal mass at conception are correlated because both are correlated with maternal mass when non-pregnant and non-

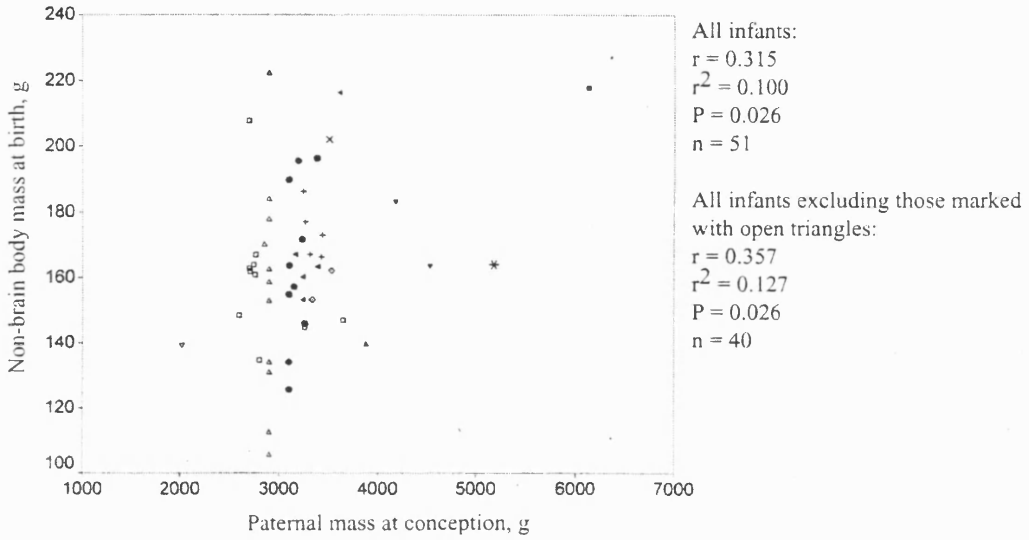


Figure 6.6. Paternal mass at conception (PM_c) correlates with the size of the non-brain tissues at birth ($NBBM_b$). Infants marked with the same symbols are siblings. See text for discussion.

TABLE 6.4 Group 2 correlations

	Correlation between...	...and...	...controlling for...	r	r^2	P	n
6.4.1	Brain mass at birth (BR_b)	Maternal mass at conception (MM_c)	Maternal mass when non-pregnant and non-lactating (MM_n)	-0.227	0.052	0.116	47
6.4.2	Brain mass at birth (BR_b)	Maternal mass when non-pregnant and non-lactating (MM_n)	Maternal mass at conception (MM_c)	**0.494**	**0.244**	**0.002**	**47**
6.4.3	Maternal mass when non-pregnant and non-lactating (MM_n)	Brain mass at birth (BR_b)	Age at separation (S_{age})	0.409	0.167	0.066	19
6.4.4	Maternal mass when non-pregnant and non-lactating (MM_n)	Age at separation (S_{age})	Brain mass at birth (BR_b)	-0.076	0.006	0.744	19
6.4.5	Maternal mass when non-pregnant and non-lactating (MM_n)	Age at separation (S_{age})	Brain mass at separation (BR_s)	-0.149	0.022	0.519	19
6.4.6	Maternal mass when non-pregnant and non-lactating (MM_n)	Brain mass at separation (BR_s)	Age at separation (S_{age})	0.405	0.164	0.069	19
6.4.7	Brain mass at separation (BR_s)	Age at separation (S_{age})	Brain mass at birth (BR_b)	**0.759**	**0.576**	**0.000**	**19**
6.4.8	Brain mass at birth (BR_b)	Age at separation (S_{age})	Brain mass at separation (BR_s)	**-0.782**	**0.612**	**0.000**	**19**

lactating. When MM_c is held constant, the relationship between MM_n and BR_b remains significant (Table 6.4.2). Maternal mass when non-pregnant and non-lactating explains 24.4% of variation in brain mass at birth when MM_c is held constant (Table 6.4.2). This result was also found in the analyses presented in Chapter 5, and is not discussed further here, except to note that whilst body mass at birth is correlated with paternal size, brain mass at birth is associated with maternal size (see Discussion for further comment).

Age at separation (S_{age}) also falls in this PC 2 group. Infants that have large mothers tend to have large brains (Fig 6.7a); they also tend to wean at an earlier age, although this relationship is not significant (Fig 6.7b). The associations of age at separation, and brain mass at birth, with maternal mass when non-pregnant and non-lactating are weak, and both are non-significant when either age at separation or brain mass at birth is held constant as appropriate (Table 6.4.3, 6.4.4). The same is true when brain size at separation, rather than at birth, is substituted into these two partial correlations (Table 6.4.5, 6.4.6). Maternal mass and age at separation appear to be correlated because both correlate with brain mass at birth.

Although age at separation (S_{age}) is not significantly correlated with either brain mass at birth (BR_b) or at separation (BR_s; data not shown), S_{age} *is* strongly associated with both brain size at birth (BR_b) and at separation (BR_s) when the other brain mass is held constant as appropriate (Table 6.4.7, 6.4.8). The amount of variation explained by these partial correlations is very high: 57.6% for BR_s and 61.2% for BR_b (Table 6.4.7, 6.4.8). In addition, age at separation is positively correlated with brain size at separation (independent of brain size at birth), but negatively correlated with brain size at birth (independent of brain size at separation). Infants with small brains at birth are separated later than those with large brains at birth, regardless of their eventual brain mass at separation; infants that have large brains at separation are separated later than those with small brains at separation, regardless of their brain mass at birth. Age at separation is therefore strongly related to relative brain mass at separation i.e. the amount of brain growth undergone since birth, irrespective of the size of the brain either at birth or

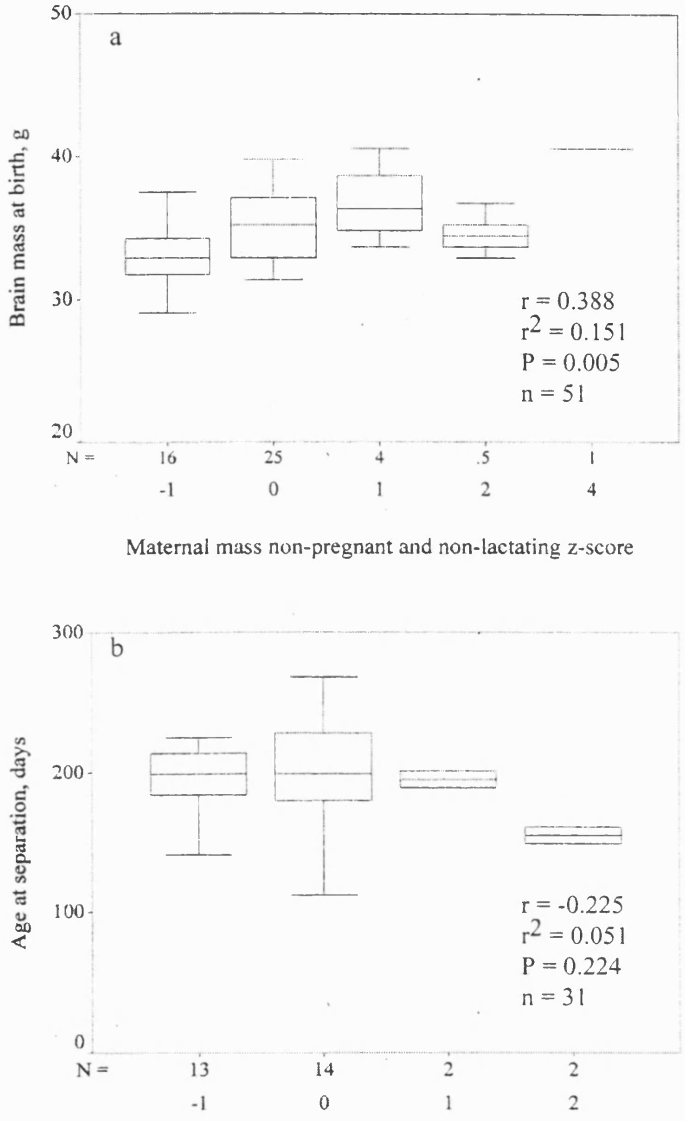


Figure 6.7. Maternal mass when non-pregnant and non-lactating (MM_n) correlates significantly with brain mass at birth (BR_b) , and non-significantly with age at separation (S_{age}). (a) Brain mass at birth; (b) age at separation. Maternal masses are expressed in standard deviation intervals.

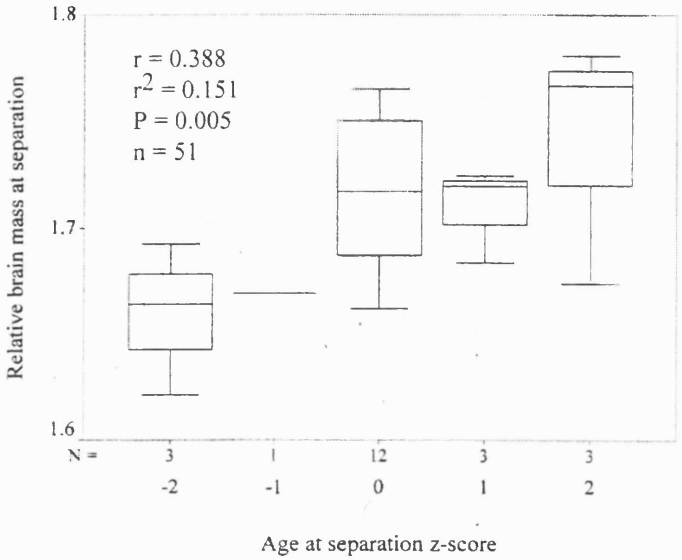


Figure 6.8. Age at separation (S_{age}) predicts relative brain size at separation. Relative brain size at separation = brain mass at separation / brain mass at birth, i.e. describes how much brain growth is undergone postnatally. Age at separation is expressed in standard deviation intervals.

separation (Fig 6.8). Age at separation is not significantly correlated with any measure of infant non-brain body mass, either at birth or at separation (data not shown).

3] Group 3: Maternal mass at birth (MM_b) and when non-pregnant and non-lactating (MM_n), number of siblings (SIBS) and non-brain body mass at separation ($NBBM_s$). Non-brain body mass at separation ($NBBM_s$) correlates significantly and positively with maternal mass at birth (MM_b) when maternal mass when non-pregnant and non-lactating (MM_n) is held constant (Table 6.5.1) but the significance of the correlation between MM_n and $NBBM_s$ disappears when MM_b is held constant (Table 6.5.2). Non-brain body mass at separation therefore appears to be associated with maternal mass when non-pregnant and non-lactating primarily because maternal mass when non-pregnant and non-lactating is correlated with maternal mass at birth. Mothers that are large at birth produce infants that have larger non-brain body tissues at separation. This implies that it is the maternal body reserves at the end of gestation (measured as body mass at birth) that are important in determining how large the infant non-brain body mass will be at separation. It should be noted that only two infants have mothers that are relatively large at birth (i.e. that fall more than 1 standard deviation above the mean for MM_b; Fig 6.9); however, these two heavy mothers do produce heavy infants at separation (>800g; Fig 6.9).

The PCA results indicate that number of older siblings (SIBS) is also associated with the size of the non-brain body mass at separation ($NBBM_s$; Fig 6.4). An ANOVA of $NBBM_s$ and SIBS is not significant, but firstborn infants (SIBS = 0) tend to have heavier bodies at separation than infants with one elder sibling (SIBS = 1; Fig 6.10). In those infants which are not firstborn (SIBS > 0), average non-brain body mass at separation increases with increasing number of older siblings (Fig 6.10), and an ANOVA between infants with one, two or three older siblings is only just non-significant ($P = 0.061$; Fig 6.10).

This result may be explained by the correlation of number of siblings (SIBS) with maternal mass at birth (MM_b; Fig 6.11a). The CPC females have not stopped growing

TABLE 6.5 Group 3 correlations

	Correlation between...	...and...	...controlling for...	r	r^2	P	n
6.5.1	Non-brain body mass at separation ($NBBM_s$)	Maternal mass at birth (MM_b)	Maternal mass non-pregnant and non-lactating (MM_n)	**0.440**	**0.194**	**0.046**	**19**
6.5.2	Non-brain body mass at separation ($NBBM_s$)	Maternal mass non-pregnant and non-lactating (MM_n)	Maternal mass at birth (MM_b)	-0.145	0.013	0.531	19
6.5.3	Number of siblings (SIBS)	Maternal mass at birth (MM_b)	Maternal age at conception (MA_c)	**0.318**	**0.101**	**0.011**	**62**
6.5.4	Maternal mass at birth (MM_b)	Maternal age at conception (MA_c)	Number of siblings (SIBS)	-0.012	0.000	0.928	62
6.5.5	Non-brain body mass at separation ($NBBM_s$)	Maternal mass at birth (MM_b)	Number of siblings (SIBS)	0.341	0.116	0.130	19
6.5.6	Non-brain body mass at separation ($NBBM_s$)	Number of siblings (SIBS)	Maternal mass at birth (MM_b)	0.221	0.049	0.337	19
6.5.7	Brain mass at birth (BR_b)	Number of siblings (SIBS)	Maternal mass at birth (MM_b)	**-0.386**	**0.149**	**0.006**	**48**
6.5.8	Brain mass at birth (BR_b)	Number of siblings (SIBS)	Maternal mass non-pregnant and non-lactating (MM_n)	**-0.341**	**0.116**	**0.016**	**48**
6.5.9	Number of siblings (SIBS)	Maternal mass at birth (MM_b)	Brain mass at birth (BR_b)	**-0.414**	**0.168**	**0.003**	**48**

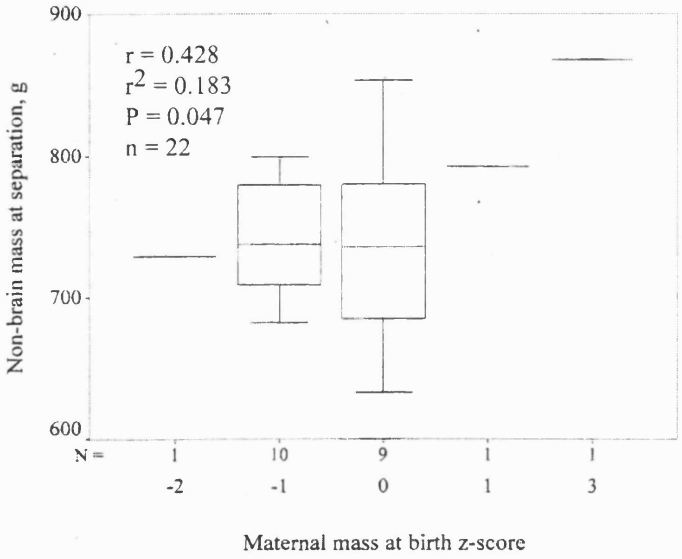


Figure 6.9. Maternal mass at birth (MM_b) predicts the size of the non-brain tissues at separation ($NBBM_s$). Maternal masses are expressed in standard deviation intervals.

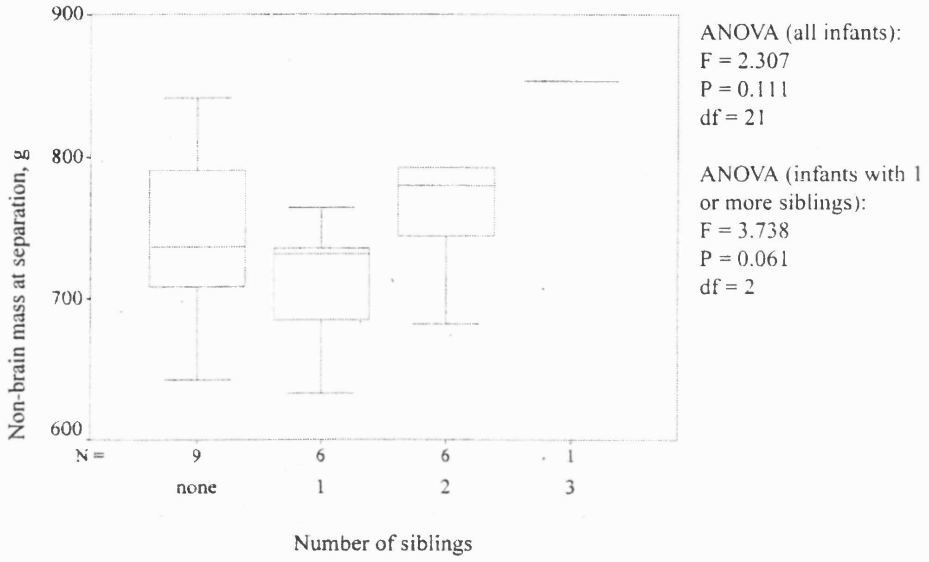
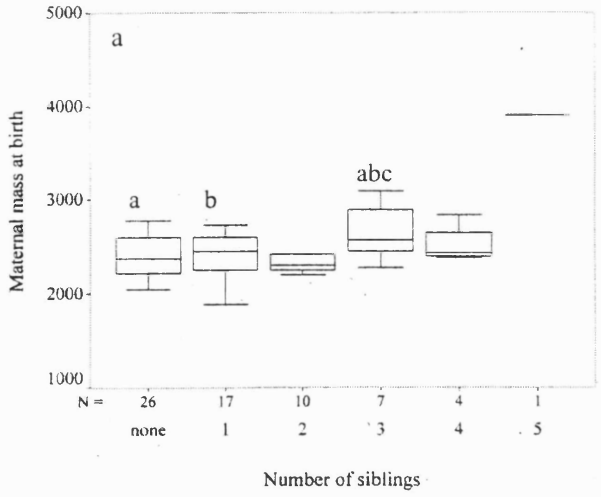


Figure 6.10. Number of siblings (SIBS) is associated with the size of the non-brain tissues at separation ($NBBM_S$). Maternal masses are expressed in standard deviation intervals.

when they begin their reproductive careers (see below), and both maternal mass and the number of offspring a female has increase with age (Fig 6.11). However, maternal mass at birth (MM_b) is significantly and positively correlated with the number of older siblings (SIBS) even when maternal age at conception (MA_c) is held constant (Table 6.5.3). Females are therefore heavier at birth with each successive pregnancy, independent of the effects of increasing mass with age. When the number of older siblings (SIBS) is accounted for, there is no residual covariance between maternal mass at birth (MM_b) and maternal age at conception (MA_c; Table 6.5.4). Non-brain body mass at separation ($NBBM_s$) is not correlated with maternal mass at birth (MM_b) when the number of siblings (SIBS) is held constant (Table 6.5.5); neither does the size of the non-brain tissues at separation correlate with the number of siblings when maternal mass at birth is accounted for (Table 6.5.6).

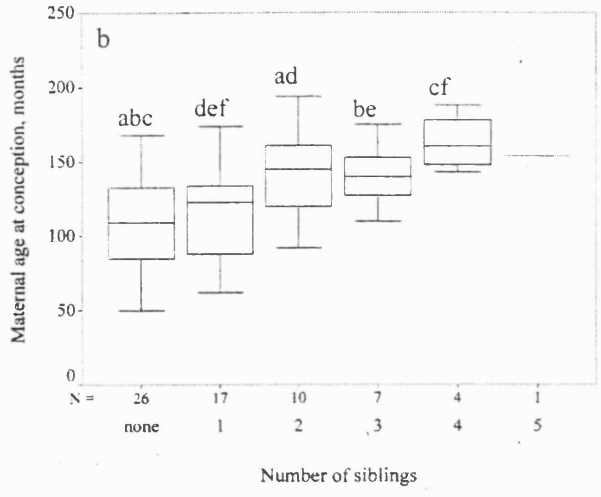
It should be noted that the relationship between number of siblings and maternal mass at birth is not significant when the infant with five siblings is removed from the analysis (Fig 6.11a). However, if the correlation between number of siblings and maternal mass at birth is robust, it implies that older females are able to allocate more resources 1] to themselves during gestation (i.e. resulting in heavier masses at birth) and 2] to their infants during lactation (i.e. resulting in heavier non-brain body masses at separation). This is probably because younger females are themselves still growing. The results presented here suggest that only after the birth of their second infant can females convert investment in own body growth into that of their infants i.e. produce infant with heavier non-brain body masses at separation compared to infants with no older siblings (Fig 6.10).

In summary, it appears that maternal mass at birth is associated with number of older siblings regardless of the age of the mother at conception; furthermore, it is an important mediator of the relationship between maternal mass at birth and the size of the non-brain body tissues at separation. The number of older siblings is also associated with brain mass at birth (BR_b), even when maternal mass at birth and maternal mass when non-pregnant and non-lactating are controlled for (Table 6.5.7, 6.5.8). The amount of variation

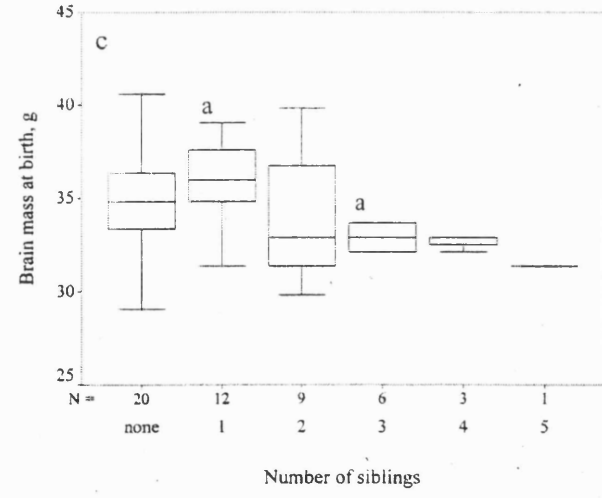


ANOVA (all infants):
F = 5.259
P = 0.001
df = 5

ANOVA (excluding infant with 5 siblings):
F = 1.719
P = 0.158
df = 4



ANOVA (all infants):
F = 4.297
P = 0.002
df = 64



ANOVA (all infants):
F = 1.670
P = 0.161
df = 50

Figure 6.11. Other correlates of number of siblings (SIBS). (a) Maternal mass at birth (MM_b); (b) maternal age at conception (MA_c); (c) brain mass at birth (BR_b). Number of siblings is expressed in standard deviation intervals. Pairs of letters indicate significant differences between groups ($P < 0.05$, least-significant differences (post hoc) method).

in neonatal brain mass explained by number of siblings is small (14.9% when maternal mass at birth is held constant, Table 6.5.7), but it is statistically significant. There is a decline in brain mass at birth in infants with two or more older siblings (Fig 6.11c). Many of the CPC females that have more than one offspring breed every year (pers. obs.), and the brain mass of the current infant may compromised after three successive successful pregnancies and lactations, despite the continued adequacy of maternal nutrition. The association of maternal mass at birth (MM_b) and number of siblings (SIBS) remains significant when brain mass at birth (BR_b) is held constant (Table 6.5.9).

4] Group 4: Gestation length (GEST) and non-brain body mass at 1000dpp ($NBBM_t$). Gestation length has been shown to be positively correlated with maternal mass in many interspecific analyses (e.g. Martin & MacLarnon, 1985; Harvey *et al.*, 1987; Ross, 1988; Hartwig, 1996; Lee, 1999). Although the PCA does not indicate that gestation length and maternal mass are associated in the CPC capuchins, a correlation analysis reveals that, gestation length (GEST) is positively correlated with maternal mass at birth (MM_b; Table 6.6.1) and maternal mass when non-pregnant and non-lactating (MM_n; Table 6.6.2), as well as with non-brain body mass at 1000dpp ($NBBM_t$; Table 6.6.3). The association of GEST and MM_n disappears when non-brain body mass at 1000dpp is held constant ($NBBM_t$; Table 6.6.4), but GEST and MM_b remain significantly associated when $NBBM_s$ is accounted for (Table 6.6.5). Larger mothers therefore tend to gestate infants for a longer period independent of the correlation between long gestations and large non-brain body masses at 1000dpp. The correlation of gestation length (GEST) and non-brain body mass at 1000dpp ($NBBM_t$) also remains significant when maternal mass at birth is controlled for (Table 6.6.6).

This positive relationship between maternal mass and gestation length appears to hold true below a certain maternal mass: above 3kg, gestation length appears to be relatively invariable (although females heavier than 3kg are represented in the dataset by only 5 datapoints; Fig 6.12a). Length of gestation does not, in fact, vary much between the

TABLE 6.6 Group 4 correlations

	Correlation between...	...and...	...controlling for...	r	r^2	P	n
6.6.1	Gestation length (GEST)	Non-brain body mass at 1000dpp ($NBBM_t$)	-	**-0.538**	**0.289**	**0.010**	**22**
6.6.2	Gestation length (GEST)	Maternal mass at birth (MM_b)	-	**0.253**	**0.640**	**0.042**	**65**
6.6.3	Gestation length (GEST)	Maternal mass non-pregnant and non-lactating (MM_n)	-	**0.300**	**0.090**	**0.015**	**65**
6.5.4	Gestation length (GEST)	Maternal mass non-pregnant and non-lactating (MM_n)	Non-brain body mass at 1000dpp ($NBBM_t$)	0.271	0.073	0.235	19
6.6.5	Gestation length (GEST)	Maternal mass at birth (MM_b)	Non-brain body mass at 1000dpp ($NBBM_t$)	**0.505**	**0.255**	**0.020**	**19**
6.6.6	Gestation length (GEST)	Non-brain body mass at 1000dpp ($NBBM_t$)	Maternal mass at birth (MM_b)	**-0.616**	**0.380**	**0.003**	**19**

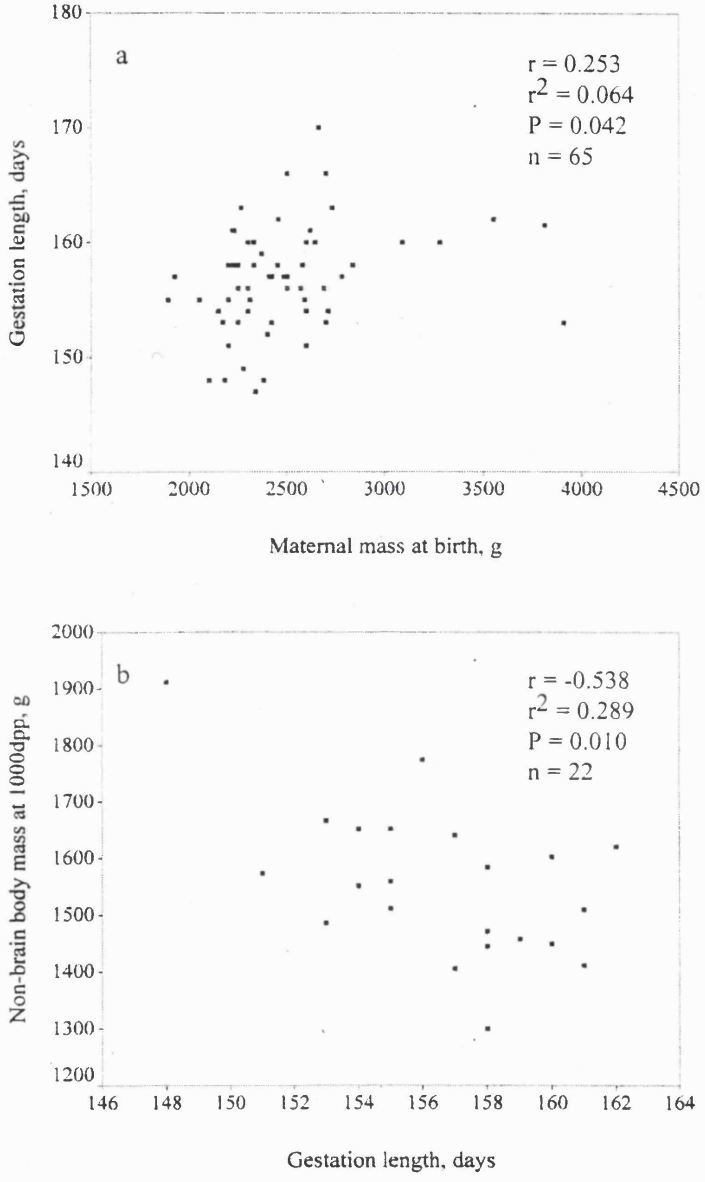


Figure 6.12. Gestation length (GEST) is significantly correlated with maternal mass at birth (MM_b) and non-brain body mass at 1000dpp ($NBBM_t$). (a) Maternal mass at birth; (b) non-brain body mass at 1000dpp.

CPC infants: the standard deviation from the mean is four and a half days, or about 3% of average gestation length (mean = 156.68 days). The amount of variation in non-brain body mass at 1000dpp ($NBBM_t$) explained by variation in gestation length (GEST) increases when maternal mass at birth (MM_b) is held constant, from 28.9% to 38.0% (Table 6.6). Furthermore, infants that have long gestation periods tend to be small at 1000dpp (Fig 6.12b). Infants that are small at 1000dpp, and have long gestation lengths, also tend to be female (Fig 6.13). The effects of sex differences on growth are examined in further detail below.

Lee (1999) finds that gestation length and neonatal brain mass are highly significantly correlated in an interspecific study of 41 primate species ($R^2 = 0.75$). The correlation of brain mass at birth and gestation length in the CPC dataset is not significant (data not shown); nor does gestation length show a significant association with non-brain body mass at birth or separation (data not shown).

5] The role of sex in determining growth.

The only infant size variable to show statistically significant sexual dimorphism is non-brain body mass at 1000dpp (Table 6.7). Both male and female infants weigh just less than 200g at birth (Table 6.7), and no sexual differences are observed in gestation length (GEST), brain mass at birth (BR_b) or non-brain body mass at birth ($NBBM_b$; Table 6.7). This contrasts with reports of sexual dimorphism in other captive *Cebus* neonates: Nagle & & Denari (1982), for instance, report a mean female neonate mass of 212.2g ± 10.1, and a mean male neonate mass of 243.9g ±15.8 (n not given).

Neither brain mass at separation (BR_s) nor non-brain body mass at separation ($NBBM_s$), is significantly sexually dimorphic (Table 6.7). Dimorphism becomes significant only after separation occurs: at the age of 1000dpp males have non-brain body masses that are, on average, 8.8% bigger than those of females (1641.31g ± 142.33 vs. 1507.47g ± 68.59; Table 6.7). Whole body mass at 1000dpp is 25.1% bigger in males than females (1729.38g ± 145.43 vs. 1537.05g ± 109.81; Table 6.7). Average adult mass in the

TABLE 6.7 Sex differences in the CPC dataset

Variable	Sex	n	Mean ± s.d.,	F	P	t	df	P
Gestation length (GEST), days	F	29	157.38 ±3.75	1.320	0.255	1.130	63	0.263
	M	36	156.13 ± 4.94					
Brain mass at birth (BR_b), g	F	23	34.69 ± 2.84	0.283	0.597	-0.569	49	0.572
	M	28	35.25 ± 4.00					
Non-brain body mass at birth ($NBBM_b$), g	F	23	161.83 ± 23.47	0.775	0.383	-0.411	49	0.683
	M	28	164.78 ± 27.09					
Whole body mass at birth*, g	F	29	195.52 ± 24.80	0.504	0.480	-0.497	63	0.621
	M	36	198.89 ± 28.94					
Brain mass at separation (BR_s), g	F	14	60.29 ± 3.43	2.423	0.135	0.589	20	0.562
	M	8	35.25 ± 4.00					
Non-brain mass at separation ($NBBM_s$), g	F	14	738.64 ± 68.59	0.548	0.468	-0.091	20	0.373
	M	8	764.01 ± 50.47					
Whole body mass at separation**, g	F	17	781.00 ± 75.44	3.544	0.069	0.211	29	0.835
	M	14	773.21 ± 128.14					
Non-brain body mass at 1000dpp ($NBBM_t$), g	F	14	1507.47 ± 105.65	**0.393**	**0.538**	**-2.251**	**20**	**0.020**
	M	8	1641.31 ± 142.33					
Whole body mass at 1000dpp****, g	F	19	1537.05 ± 109.81	**1.562**	**0.220**	**-4.454**	**33**	**0.000**
	M	16	1729.38 ± 145.43					
Adult whole body mass***, g	F	65	2221.95 ± 254.91	**10.582**	**0.001**	**-11.067**	**76^**	**0.000**
	M	61	3212.49 ± 653.94					

*Whole body mass at birth = brain mass at birth + non-brain body mass at birth.

***Adult whole body mass inferred from maternal mass when non-pregnant and non-lactating (MM_n) for females, and paternal mass at conception (PM_c) for males.

^Equal variance not assumed.

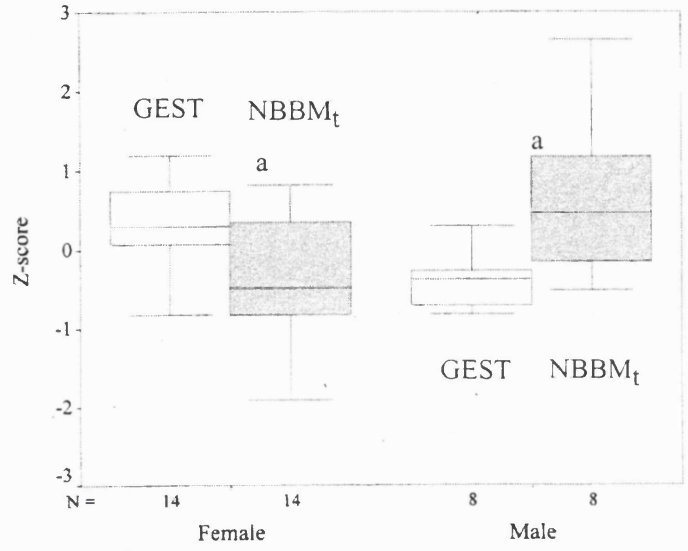


Figure 6.13. Sex differences in gestation length (GEST) and non-brain body mass at 1000dpp (NBBM$_t$). See Fig 6.11 for key.

colony can be inferred from maternal mass when non-pregnant and non-lactating (MM_n) for females, and from paternal mass at conception (PM_c) for males; these adult masses are also significantly dimorphic (Table 6.7). On average, adult females weigh 2221.95g ± 254.91, whilst adult males weigh approximately one kilogram heavier (3212.49g ± 653.94; Table 6.7). By the age of 1000dpp, therefore, females have attained approximately 70% of the average adult female body mass; males, on the other hand, have attained only 54 % of the average adult male body mass. This implies that males will either continue to grow faster than females after 1000dpp, and/or for longer, if they are to achieve target adult body mass.

Although gestation length (GEST) is not significantly different between the sexes (Table 6.7), female infants tend to have been gestated for a long period compared to males (Fig 6.13). When sex is controlled for, the correlation between gestation length (GEST) and non-brain body mass at 1000dpp ($NBBM_t$) is not significant (data not shown). In the CPC capuchins, the association of gestation length and body mass is only significant when the effects of sex are included in the analysis.

6] Correlations between infant brain and body masses over development.

The results of the correlation analyses indicate that maternal and paternal masses, and other parameters such as number of older siblings, are significantly associated with infant mass over development. Infant masses later in ontogeny can also be predicted from infant masses earlier in ontogeny (Fig 6.14). Brain mass at birth (BR_b), for example, accounts for 96.4% of the variation in brain mass at separation (BR_s; Fig 6.14). The association of body masses at the beginning and end of lactation is less strong: only 20.9% of variation in non-bran body mass at separation ($NBBM_s$) is explained by variation in non-brain body mass at birth (Fig 6.14). This suggests that brain growth is more constrained than non-brain body growth, as was noted in Chapter 3. The relationship between non-brain body mass at separation and non-brain body mass at 1000dpp ($NBBM_t$) is not significant: it is not possible to predict body mass at 1000dpp from body mass at separation.

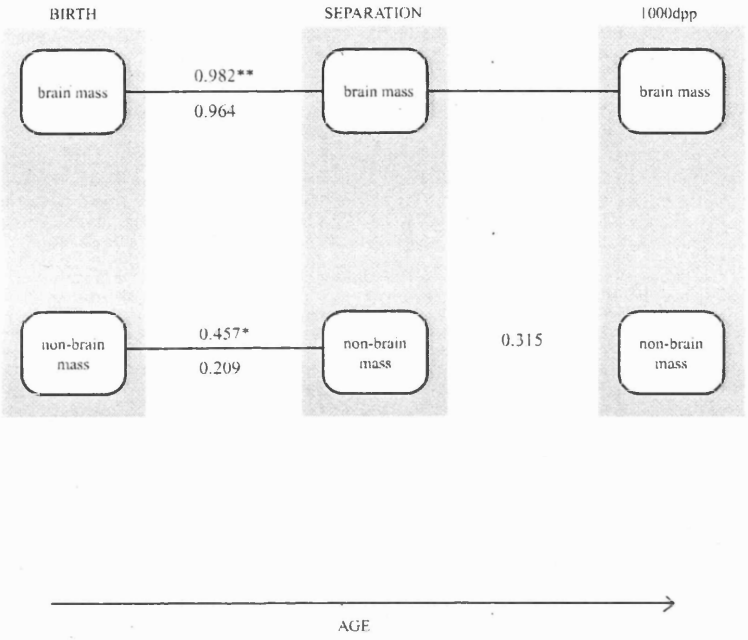


Figure 6.14. Modelling infant mass correlations in the CPC dataset. Top figure in each pair is r, bottom figure is r^2, where correlation is significant (indicated by asterisks). Where correlation is not significant, only r is shown.

6.2.5 Constructing a life history model

The model presented here (Fig 6.15) summarises the results of the correlation and partial correlation analyses. As in Figure 6.14, infant brain and body masses are represented at three ages (birth, separation and 1000dpp) and change in the age of the infant is represented as a horizontal axis. Two time variables (separation age and gestation length) are represented by circles. Other variables, e.g. maternal and paternal parameters, are represented by boxes. Lines joining variables indicate that a significant correlation exists between them, and the values shown are the appropriate correlation coefficients. No correlation is indicated between brain mass at separation and 1000dpp because brain mass at 1000dpp is inferred from brain mass at separation.

The variables positioned to the left of the neonatal mass parameters are those that influence either brain or body mass at birth. Maternal mass when non-pregnant and non-lactating is positively correlated with brain mass at birth when maternal mass at conception is held constant (Table 6.4.1). Brain size at birth is also inversely correlated with previous maternal investment, measured as number of older siblings (again with maternal mass held constant; Table 6.5.8). Mothers who have invested in previous offspring tend to produce infants with smaller brains. Non-brain body mass at birth is positively correlated with paternal mass at conception: bigger males tend to father larger offspring (Table 6.3.3).

Separation age is negatively correlated with brain mass at birth when brain mass at separation is held constant (Table 6.4.8), but positively with brain mass at separation when brain mass at birth is held constant (Table 6.4.7). The size of the brain at separation is influenced to a very large degree by the size of the brain at birth (Fig 6.14). Non-brain body mass at separation is significantly correlated with both infant non-brain body mass at birth (Fig 6.14) and maternal mass at birth (with maternal mass when non-pregnant and non-lactating is held constant; Table 6.5.1). The correlation coefficients of both these relationships are similar; maternal mass at birth influences the size of the non-brain tissues

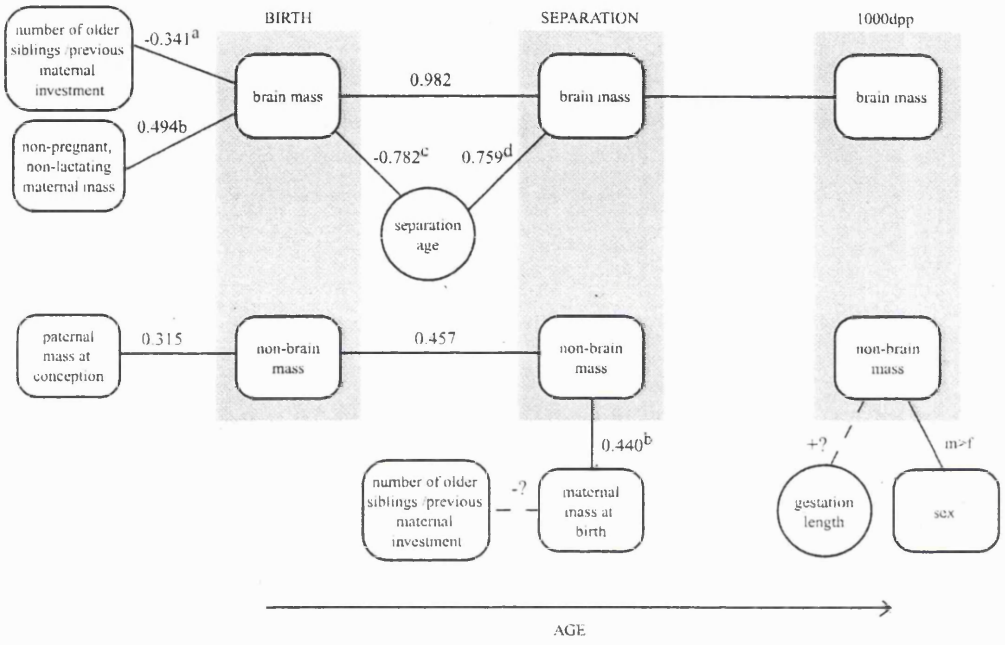


Figure 6.15. Modelling life history interactions in the CPC dataset. Figures shown are r values. a = maternal mass when non-pregnant and non-lactating held constant (Table 6.5.8). b = maternal mass at conception held constant (Table 6.4.2). c = brain mass at separation held constant (Table 6.4.8). d = brain mass at birth held constant (Table 6.4.7). +? and -? indicate that the relationship is not statistically robust.

at weaning to the same extent as the infant's own body mass at birth (Fig 6.15). Maternal mass at birth might also be correlated with the number of older siblings, independent of maternal mass when non-pregnant and non-lactating, although this result is influenced by data from one very heavy female (Fig 6.11).

Finally, on the extreme right of the age axis, non-brain body mass at 1000dpp is significantly influenced by the sex of the infant (Table 6.7). Gestation length might also show some association with non-brain body mass at 1000dpp independently of maternal mass at birth (Table 6.6.6), but the relationship between these three parameters – sex, body mass at 1000dpp and gestation length – is ambiguous (Table 6.6).

6.3 Limitations of the model and the CPC dataset

1] The analyses presented above treat the CPC infants as independent individuals, but in fact many are siblings. In addition, the relatedness of the members of the original breeding group is not known, and as we saw earlier, paternity of the infants was shared between a limited number of males. These data are unlikely, therefore, be independent. Non-independence will tend to over-emphasise the role of genetic factors in determining phenotypic variation (Harvey & Pagel, 1993).

2] As discussed in Chapter 5, separation is not weaning in its behavioural sense. Inferring strategies of lactation and other weaning behaviours from these infants might underestimate the importance of behavioural development in determining weaning masses and ages. However, mean whole body mass at separation in the CPC infants (777.48g ±100.83) approximates weaning mass in other capuchin infants (see Chapters 1 and 5).

6.4 Discussion

1] Which factors influence the timing and pattern of infant postnatal growth before and after weaning?

The results of the PCA and correlation analyses suggest that the size of the infant brain at birth is determined to some extent by maternal mass. Neonatal brain size is associated with maternal mass when non-pregnant and non-lactating, and also that it is associated with the level of previous investment on the part of the mother (measured as number of older siblings), suggesting that this relationship is based on maternal resources or capacity (measured as maternal mass; Martin, 1983; Martin, 1996). Paternal mass is associated with size of the non-brain tissues at birth. The differential effects of the maternal and paternal genome on brain and body size have been noted in transgenic mice (Keverne *et al.*, 1996). It is interesting to speculate whether the differences in maternal and paternal associations seen in capuchin infant brain and body growth are also the result of each parent's genes determining different aspects of growth before birth.

Postnatal brain growth does not appear to be influenced by any other variable included in the analysis other than brain size at birth. This suggests that the trajectory of brain growth is largely set before birth, as was observed in Chapter 3. However, maternal resources (measured as maternal mass at birth) do appear to be important predictors of infant postnatal body growth. The effects of maternal condition on infant growth have been described in other species (e.g. maternal adiposity and infant mass in rhesus macaques, (Johnson & Kapsalis, 1995); maternal weight for height and infant mass gain in Bolivian children, (Novotny & Haas, 1987), and the capuchin results suggest that a similar positive association of maternal condition and infant body growth can exist in females that are well-nourished. This is probably because the CPC females are themselves still increasing in body mass when they start reproducing.

When the period of maternal dependency has ended, the size of the non-brain tissues is influenced by the sex of the infant. This presumably occurs via the action of the sex hormones associated with the pubertal growth spurt which male capuchins show at 1000dpp (Jungers & Fleagle, 1980; Leigh, 1992). Although these capuchins have not yet achieved their adult mass by 1000dpp (see above), sexual differences in mass are apparent. The role of gestation length in determining body mass at 1000dpp is unclear but, as adult body stature and weight are highly heritable in humans (Bouchard *et al.*, 1990), it is possible that this association reflects the prenatal (i.e. genetic) control of adult size.

2] How does infant growth contribute to the pattern of delayed life histories seen in capuchins?

The capuchin model suggests that age at separation is directly modulated by the growth of the brain. This evidence supports theories that relate the evolution of delayed life histories in primates (in particular, a later age at weaning and a low birth rate) to the large size of the primate brain (see Introduction). In Chapter 4 it was shown that the development of specific weaning behaviours is significantly associated with increasing brain size during lactation. It was noted in that chapter that the direct association supports theories that link slow infant growth with the development of appropriate social/foraging behaviours (see Ross & Jones, 1999). The results described in the present chapter also support the idea that brain growth is limited by the need to attain a certain size and, by inference, maturity. Infants that wean late have achieved a large brain size, regardless of brain size at birth (Fig 6.15); infants which undergo more brain growth after birth wean later than those which undergo less brain growth (Fig 6.8). Postnatal brain growth in capuchins therefore appears to be target seeking, and able to show catch-up growth. This suggests that it is advantageous for a certain brain size to have been achieved by separation, and that age at separation is delayed until it is reached. In Chapter 5, it was seen that maternal mass appears to be primarily associated with postnatal body, not brain, growth. This result was

also found in the present chapter. The length of the weaning period is associated with target-seeking brain growth, whereas the mass that the non-brain tissue has reached by weaning is dependent on maternal resources.

The timing of separation or weaning also directly impacts upon other aspects of life history e.g. interbirth interval. Capuchins, like humans, apes and some other catarrhines, can display an extended period of lactational infertility (Altmann *et al.*, 1978; Tutin, 1980; Diaz *et al.*, 1995). Recabarren *et al.* (in press) present data collected from some of the same CPC females and infants included in the present dataset. They use endocrine radioimmunoassays to establish reproductive status in the females. In nursing females (n = 15), the mean period of amenorrhea lasted 155 days ± 13.0 (5 months), i.e. ceased prior to separation which, in this set of infants, occurred at 260 days ± 16.7 (over 9 months old). Females were mated with males as soon as menses resumed, but the females became pregnant again only after a further period of residual fertility that lasted, on average, 10 months. Mean interbirth interval in these CPC females is thus 613.4 days ± 30.8 (1.68 years), assuming gestation length is 155d (Recabarren et al. in press). In comparison, non-nursing females in the CPC colony (i.e. mothers whose infants died or were removed at birth) showed truncated interbirth intervals when time from first postpartum mating was considered (349.5 ± 11.8 vs. 613.4 ± 30.8; Recabarren et al., in press). When the period after the removal of the infant was compared, both groups of females showed similar time to subsequent pregnancy (195.1 ± 15.8 vs. 199.0 ±33.7 respectively). Thus it appears to be the presence of the unweaned infant that prolongs the length of both anoestrus and residual infertility in these capuchins, i.e. extends the interbirth interval (Altmann *et al.*, 1978; Zeigler *et al.*, 1990).

Can any of the variables studied here predict whether a CPC mother will go on to reconceive in the same year? The number of infants for which it was possible to calculate the length of time until subsequent conception of the mother was small (n = 14), and of these only two infants had mothers who went on to conceive immediately after separation ('conceivers' as defined in Bowman & Lee, 1995), rather than in the next breeding season

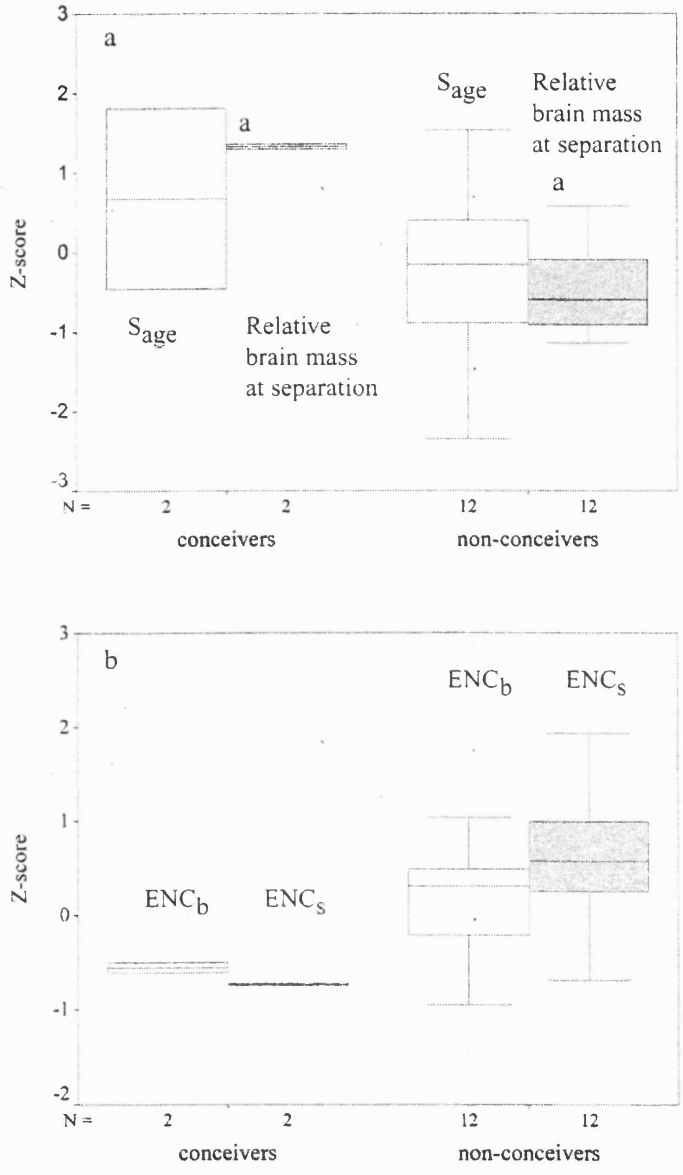


Figure 6.16. Differences between the infants of 'conceivers' and 'non-conceivers'. (a) Age at separation (S_{age}) and relative brain mass at separation; (b) encephalisation at birth (ENC_b) and separation (ENC_s). Relative brain mass at separation = brain mass at separation / brain mass at birth. See Fig 6.11 for key.

i.e. one year later ('non-conceivers' as defined in Bowman & Lee, 1995). It should be remembered that the CPC breeding regime is not naturalistic, but the data give a good indication of how reproductive strategies differ between conceivers and non-conceivers. Brain mass at separation relative to the size of the brain at birth (Fig 6.16a), and the sex of the infant, were the only variables to show a significant association with conception status (more infants of non-conceiving mothers are female than can be expected by chance, and more of the conceivers' infants are male ($\chi^2 = 4.592$, df = 1, P = 0.032).

Each of the two conceivers' infants has a large brain mass at separation relative to brain mass at birth, and a late age at separation (Figure 6.16a). These two mothers are therefore investing highly in their offspring; the level of maternal investment in the infant can be gauged from the large size of the brain at separation relative to the size of the brain at birth. However, both infants have small brain masses relative to non-brain body masses - i.e. are relatively non-encephalised – at both birth and separation (Fig 6.16b). A neonate with a small brain at birth relative to body mass will need more investment in brain growth during lactation than one with a relatively large brain at birth, if a target brain size is to be achieved by separation. It is possible that these two mothers are investing heavily in their non-encephalised infants, but then re-conceiving in the same year in order to increase reproductive fitness. This would require that infants with relatively small brains at birth have higher mortality than those with relatively large brains at birth, an assumption for which no evidence yet exists. It does appear, however, that these mothers are modulating their lactation and weaning strategies according to the size and relative development of their infants, and that these lactation strategies have an important impact on reproduction and life history.

CHAPTER 7

GROWTH AND MATURATION IN CAPUCHINS AND HUMANS: A COMPARATIVE DISCUSSION

This thesis investigated the interrelationship between primate encephalisation, growth and lactation strategies. It examined the ways in which primate mothers invest in their relatively 'expensive' offspring, and how infants grow within the constraints of that investment. This chapter discusses the implications of the results presented in Chapters 1 to 6, and compares capuchin growth strategies with those of another encephalised primate species, *Homo sapiens*. Finally, the aims outlined at the beginning of the thesis are assessed:

- Do different primate species share a common pattern of postnatal brain and body ontogeny, and how does this pattern compare with that seen in other mammals?
- How do ontogenetic changes in brain and body size relate to lactation and weaning strategies?
- How do ontogenetic changes in brain and body size relate to other aspects of primate lif history?

7.1 Primate encephalisation, growth strategies and life histories, with special reference to *Cebus*

The Introduction describes how the 'slow' life histories of primates are associated with the relatively large size of the primate brain. For example, for their size, primates wean later than other taxa, grow more slowly during the lactation period and mature at a later age. Encephalisation is predicted to influence maternal investment via lactation and infant growth. Two hypotheses link brain size with growth:

1] The first predicts that, because brain tissue is relatively expensive to maintain, the large brains of primate infants place an increased energetic burden on mothers and constrain growth during lactation (Martin, 1996; Lee, 1999).

2] Secondly, large brains are predicted to take a longer time to mature than small ones, especially in the complex social world of primates, and large brains are hypothesised to be associated with a long period of behavioural development (Dunbar, 1992).
Alternatively, the slow growth rates seen in primates might be unrelated to brain size. Rather, slow growth rates may reduce juvenile risk by avoiding the need for extensive foraging at a time when individuals are relatively inexperienced foragers, and are prone to predation (Janson & van Schaik, 1993).

The analyses presented in this thesis have examined encephalisation, growth and lactation strategies in primates in the context of life history. The next eight sections discuss the main results of the analyses.

7.1.1 The primate pattern of brain and body growth is unusual

This thesis shows that, in addition to possessing unusually large brains, primates follow an unusual pattern of growth. Chapter 1 examined interspecific postnatal brain and body growth variation in a selection of haplorhine primate taxa. It compared observed brain allometry patterns with those predicted by a general mammalian model. In the majority of species investigated, the postnatal brain allometry is not linear, as it is in non-primate species (Deacon, 1990). These primate taxa undergo a period of postnatal brain growth that is rapid (in relation to body growth) compared to the model's predictions. Only after the rapid phase of growth has ended is the exponent describing brain and body growth similar to that predicted in non-primate taxa from birth onwards. These trends are also found at the intraspecific level (Chapter 2). Capuchin infants undergo a period of rapid brain growth in the first half of the first year postpartum, only after which do they conform to the model's slope predictions. The departure from a non-primate growth pattern contributes to the relative encephalisation seen in primates in two ways. Firstly, interspecific differences in adult residual encephalisation are significantly correlated with the pre-inflexion RMA exponent. Secondly, at the intraspecific level, inter-individual differences in post-inflexion growth are important determinants of adult

brain and body proportions. For example, significant sexual differences appear in the amount of body growth (RMA length) undergone in this later post-inflexion period, although adult encephalisation is not significantly different between the sexes. Although both sexes are of similar brain and body size at the inflexion of the brain allometry, males undergo more mass increase by adulthood, and the male post-inflexion trajectory is extended into a larger body size range (Fig 2.14c). Thus growth that occurs early in postnatal life has an important influence on adult morphology.

7.1.2 Primate strategies of growth are riskier than those of non-primates

In non-primates, the majority of brain growth is prenatal (Martin, 1990). The mammalian pattern of ontogeny therefore ensures that almost all of brain growth occurs in the protected, constantly-nourished uterine environment (Pond, 1984). By continuing significant brain growth after parturition, primates are adopting a risky strategy of growth. Why do primates adopt a strategy that is expensive both in energetic terms (because of the increased costs of lactation compared to gestation) and in fitness terms (high risk investments often do not pay off)?

A crucial difference between non-primates and the majority of primate taxa is the complexity of the social world into which they are born (Dunbar, 1988). Not only must infant primates learn to forage effectively, they must also develop those behaviours that will ensure their successful integration into a social network of older and younger siblings, parents, other kin, unrelated males and females, strangers and non-conspecifics. We have already seen that, in primates, neocortex size correlates with both group size and the length of the juvenile period (Joffe, 1997; Dunbar, 1998). In primates, postnatal brain growth – with all the 'hardwiring' of experience it involves – goes hand in hand with living in a complex social environment. Neonatal capuchins are relatively altricial at birth (Watts, 1990), and the results of Chapter 4 show that brain growth is directly associated with the emergence of behavioural and physiological independence (e.g. decline in nursing frequency, increase in number of

times an infant leaves its mother, and in the number of times a mother leaves its infant) independently of body growth. In addition, elements of the capuchin foraging repertoire (such as a precision grip) are not present until later in the period of rapid brain growth, i.e. after the age of three or four months old. The suite of behaviours that characterise weaning and independence are therefore directly correlated with postnatal brain growth.

7.1.3 Brain growth and lactation/weaning strategies are linked

Weaning coincides with the inflexion of the brain allometry in primates, i.e. with the body mass at which the rapid rate of postnatal brain growth slows. On average, the brain is 75% of adult size when weaning occurs. Postnatal brain growth is therefore associated with the pattern of lactation such that infants are weaned only once the majority of brain growth is completed (Chapters 1, 2), and appropriate weaning behaviours have emerged (Chapter 4). As mentioned in the previous section, behaviours that indicate behavioural as well as energetic independence – for example, frequency of leaves to/approaches from mother – are correlated with the pattern of brain growth rather than body growth. This suggests that sensorimotor, and possibly cognitive, development is an important part of locomotor maturity. It also suggests that mothers alter their behaviour in response to the increasing maturity of the infant brain, which feeds back into the regulation of lactation (Lee, 1987). Body growth correlates with other aspects of weaning and behavioural independence. Those variables that are related to the size of the infant, for example time spent carried on the mother's back, decrease as the infant grows. Foot and hand maturity (e.g. size, manipulative ability) are attained relatively soon after birth, and probably reflect the need for the infant to cling from an early age (pers. obs.). It is interesting to note, however, that capuchins are similar to humans and chimpanzees in the level of skeletal (specifically wrist bone ossification) altriciality they display at birth (Watts, 1990).

Separation age is correlated not with the absolute size of the brain, but with the relative amount of postnatal brain growth undergone by the infant. It does not appear to be

associated with body size or body growth. When brain size at birth is controlled for, later weaning ages are associated with bigger brains. In Chapter 2 it was shown that neonatal size is a good predictor of pre-inflexion growth: the brains of small-brained infants grow faster than do those of large-brained infants, again suggesting that a certain brain size must be achieved before the rapid rate of brain growth slows. In the later stage of brain growth the majority of brain mass increase occurs through myelination, i.e. is associated with the canalisation of neural pathways and the 'fixing' of the spectrum of behavioural responses to stimuli (Nolte, 1999). Although the size of the neuronal population is set early in development, and tends to be relatively invariable within species (Williams & Herrup, 1988), it can be hypothesised that brains which fail to grow in the hyperplasic phase (increase in cell number) of growth are characterised by a smaller scope for interconnectivity between neurons, as well as a by a smaller neuronal population (Barton, 1999). As Horrobin (1998) notes, "the richness and specificity of the fine connections within the neural network determine the complexity of the information processing which can occur" (p281). Whether such differentiation between 'large' and 'small' brains translates into functional or behavioural differences is unclear ~~[refs.]~~, but the target-seeking pattern of brain growth, and the extent to which brain growth is protected in the face of nutritional stress (Chapter 3) suggests that developmental mechanisms operate to ensure that brain growth is maximised. This ties in with the 'experience-limited' nature of brain ontogeny in primates (Altmann & Alberts, 1987). In *Cebus*, the relationship between weaning age and postnatal brain growth appears to be influenced both by the metabolic costs of the infant brain (as well as of the infant body; Chapter 5) and by the need for behavioural maturity (Chapter 6). Thus it appears that the weaning 'threshold' identified by Lee *et al.* (1991) is associated with brain and body mass in primates, and is both metabolic and behavioural in nature.

7.1.4 The importance of fatty acids

The period of rapid brain growth exactly matches the period in which the infant is dependent on milk for its nutritive requirements. The tempo of brain growth significantly correlates with two direct measures of milk transferral, the frequency of suckling, and the duration of suckling bouts. This implies that some of components of milk are important promoters of brain growth, and that when their effects are removed, brain growth slows. It was suggested in Chapter 4 that long-chain polyunsaturated fatty acids (LCPUFAs) are an important regulator of brain growth, and the inability of the infant to manufacture LCPUFAs – and its reliance on those present in milk – provides a mechanism by which the two might be linked. Human infants fed LCPUFA-poor formula show some brain growth deficiencies in comparison with breast-fed infants (Lanting & Boersma, 1996; Carlson & Neuringer, 1999), and the need for including LCPUFAs and their precursors in human infant formula has been frequently stated (Clandinin, 1999). Unfortunately, no data exist concerning fatty acid synthesis in capuchins. Human infants are able to manufacture LCPUFAs by the age of six months old (Yehud & Motofsky, 1997), suggesting that competence develops at about the same time as peak lactation ends, and other foods become an important part of the diet. The implications of this timing for humans, where brain growth continues long after weaning is completed, are discussed below.

7.1.5 Brain and body growth constraints: implications for life histories

The association of brain growth and weaning has important consequences for capuchin life histories. Compared to other primates, capuchins tend to show a late age at first reproduction and a birth rate more similar to that seen in apes than in other new and old world monkeys; however, for a given body mass, capuchin growth rates during the lactation period fall between those of the callitrichids and the cercopithecines and apes (Ross, 1991). This study found that capuchin brains impose behavioural and metabolic constraints on infant growth during lactation. Whether infant growth is relatively fast or slow compared to other taxa

depends on the amount to which these constraints can be circumvented. For example, the presence of allonursing in capuchins may be an important factor in increasing energy transfer to infants, and therefore in elevating the infant growth rate (Perry, 1996). Similarly, the explosion of food-exploratory behaviours that occurs at about the time that weaning begins might help to increase the breadth of the weanling capuchin diet (Fragaszy & Boinksi, 1995; Fragaszy *et al.*, 1997). It is probable that food-sharing in *Cebus* (de Waal *et al.*, 1993) is an important mechanism for ensuring weanling access to tough but high-energy foodstuffs. In addition, in the present study, large mothers are able to wean their infants at an early age; presumably these females are capable of transferring nutrients to their offspring at a relatively fast rate. This result contrasts with that of Lee *et al.* (1991), who found that lactation length and maternal mass are positively correlated in primates at the interspecific level. The good nutritional status of the CPC females may contribute to this reversal, but it should be noted that Lee *et al.*'s (1991) analysis includes data from captive and provisioned, as well as wild, populations. How this 'shortening' of the lactation period by larger mothers affects behavioural development is unclear, and it underlines the equal importance of energetics and behaviour in influencing the timing of weaning.

The late maturity seen in capuchins is likely to be influenced by a prolonged period between weaning age (i.e. end of infancy) and sexual maturity, rather than by slow infant growth (Ross, 1991). The results of Chapter 6 indicate that inter-individual variation in post-weaning growth is associated with the development of sexual dimorphism. It was not possible in this thesis to investigate post-weaning growth in relation to any other variables except maternal mass, paternal mass, number of siblings, and others discussed in Chapters 5 and 6.

7.1.6 Maternal mass, metabolism and investment

Maternal mass when non-pregnant and non-lactating is likely to be a good proxy for metabolic capacity, reflecting as it does the general size of the mother over her reproductive career. That it is found to influence neonatal brain size provides support for Martin's hypothesis that

maternal metabolic capacity, limits brain growth *in utero* (Martin, 1983; Martin, 1990; Martin, 1996). Maternal body mass was not found to correlate with infant mass at birth, at least over the first two pregnancies (see below). This is in contrast to the results of other studies (Leutenegger, 1979; Harvey *et al.*, 1987; Ross, 1988; Lee *et al.*, 1991; Lee, 1999). Maternal mass at birth is, however, positively associated with the size of the non-brain tissues at separation even though the mothers themselves are adequately nourished. Thus maternal resources at the beginning lactation dictate investment in the non-brain tissues during infant growth. During calorie- or protein-deprivation, body mass increase is checked in favour of brain growth (Chapter 3). This suggests that body growth is far more labile than brain growth, and is sensitive to the amount of energy (over and above that which must be devoted to brain growth and maintenance) the infant receives. Body size has been linked to a primate infant's chances of survival after weaning, both in terms of susceptibility to predation and in success in competition (e.g. foraging ability, social dominance etc.; Janson & Van Schaik, 1993). Maternal investment in infant body growth during lactation therefore represents an optimisation of maternal resources (Charnov & Berrigan, 1993).

The CPC mothers have not finished growing by the age of first reproduction, and face a trade-off between investing in their own growth and that of their infant. The results of Chapter 6 suggest that females giving birth to their second infant can afford to invest less than they could in their first: second infants tend to have smaller non-brain tissues at separation than first infants. However, by the third infant, maternal and infant masses become positively correlated. Because the capuchin mothers studied here have themselves not achieved adult body mass when they begin reproducing, infants that are born later in a female's reproductive career (i.e. that have more older siblings) also tend to have larger brains at birth (because mothers get bigger as they grow older). This interrelationship of maternal age, mass, and investment in previous and current infants is likely to be an important factor in determining reproductive strategies. For example, the presence of the infant prolongs the length of both anoestrus and residual infertility in females. However, two mothers who have offspring that are relatively non-encephalised at birth went on to reconceive in the same year. Their infants

underwent more brain growth than those of non-conceivers, and weaned later, but were still non-encephalised at separation, compared to other infants. Both mothers invest heavily in infant brain growth (both in terms of resources and time), but go on to reconceive very soon after separation. Birth rate is increased despite later weaning, and a high birth rate is associated with a small, rather than a large brain size. These may be extreme cases. More generally, weaning age is positively associated with the amount of brain growth undergone after birth (Chapter 6).

These findings confirm Lee's (1999) prediction that "whilst the mother's body size and condition determines some of her ability to allocate milk resources for growth [during lactation], the growth requirements are twofold: firstly, that of somatic growth to a metabolic weaning weight... and secondly, that of brain growth during gestation and lactation" (p126). Furthermore, the infant body mass at 1000dpp correlates with maternal mass at conception (Chapter 6). Infant size at 1000dpp gives an indication of post-weaning growth. As body size and stature are highly heritable (Ulijaszek & Macie-Taylor, 1994), this association probably reflects a genetic control of ultimate body size. The effects of maternal mass are therefore threefold: on the size of the brain before birth, on body mass during lactation and also after weaning is completed.

7.1.7 Paternal mass associations

The association of paternal mass with prenatal infant body growth is a very interesting result. Larger males tend to father larger-bodied infants. Paternal mass was not correlated with brain growth. It was noted in Chapter 6 that the datapoints in this correlation are not independent, coming as they do from related individuals, and reflecting the size effects of only a relatively small number of males. It is possible that the inter-relatedness of infants in the CPC colony amplifies genetic trends that would, in other situations, be swamped by epigenetic variation (Little, 1989). It is difficult to establish whether this result reflects a spurious connection

between paternal mass and neonatal body size, or whether it identifies the differential effects of the maternal and paternal genomes (Keverne *et al.*, 1996).

7.1.8 Encephalisation and undernutrition

We have seen that nutritional stress can uncouple brain and body growth, and that brain growth is highly protected (Chapter 3). Furthermore, the position of the brain allometry inflexion in these nutritionally-stressed groups occurs at a constant brain size, not body size. For nutritionally-stressed capuchin infants, the long-term effects of undernutrition are two-fold. Although even the most severely deprived infants show catch-up growth after the end of the nutritional stress, they are small compared to their better-fed peers in the post-experiment period. Small weight-for-age infants are more likely to suffer mortality, and are physiologically immature (Lee, 1987). Weanling capuchins are relatively inefficient foragers, and small infants are unable to exploit many of the tougher substrates that adults regularly consume (Fragaszy & Boinksi, 1995). Coupled with a brain that is relatively large in relation to the body, the energetic challenge facing these undernourished infants in a naturalistic environment is large.

7.1.9 Section summary

The main results of this thesis can be summarised as follows:

- Primate postnatal brain and body growth follow a different pattern from that of non-primates: the brain grows rapidly in relation to body size for a period after birth, after which the rate of brain growth slows and is similar to that seen post parturition in non-primate taxa.
- Brain growth is highly protected in nutritionally-stressed capuchin infants.

- The rapid phase of postnatal brain growth ends when the brain has attained 75% of adult brain size. It also coincides with the period of lactation, and the attainment of weaning body mass is associated with the attainment of the majority of adult brain size.
- The rapid phase of postnatal brain growth is also associated with the development of various behaviours that are important in weaning and the achievement of independence.
- Separation age is associated with the amount of brain growth that occurs in the rapid phase of postnatal brain growth, rather than the absolute size of the brain.
- Maternal mass is significantly associated with prenatal neonatal brain growth, and also with postnatal non-brain body tissue growth. Paternal mass is associated with prenatal body growth in this capuchin population.

7.2 Human growth strategies and life histories

This final section compares capuchin and human adaptation and seeks to answer several questions:

1] How useful is *Cebus* as an analogy for human adaptation? How similar are the energetic problems they face as a result of encephalisation, and are there other aspects of capuchin morphology or life history that make them a good model for human evolution?
2] What can the patterns of capuchin growth and maturation described here tell us about human evolution?
3] Are humans unique in the ways they afford the high energetic burden of large brains?

7.2.1 Capuchin and human morphological convergence

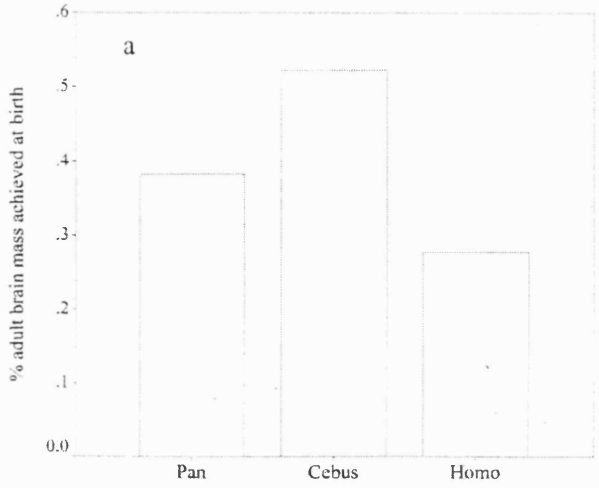
Capuchins show many behavioural adaptations that are convergent with great ape, particularly chimpanzee, evolution (Fragaszy & Bard, 1997; Visalberghi & McGrew, 1997). For example, the problem-solving and tool-using behaviours observed in capuchins, and the assumed

sensorimotor skills that underpin them, are often likened to those seen in common chimpanzees (Visalberghi, 1997), although differences have also been noted (Anderson, 1996; McGrew & Marchant, 1997). Capuchins and chimps are both highly explorative and dextrous species, often employing extractive techniques when foraging (Fragaszy & Adams-Curtis, 1997; Fragaszy & Bard, 1997). Other complex social behaviours such as cooperation and coalitions occur in both taxa (de Waal *et al.*, 1993; Rose, 1997), and both have a propensity to hunt and consume invertebrate and vertebrate prey (Rose, 1997).

Capuchins differ from the non-human apes in certain respects. They are more similar to *Homo* in some features of morphology e.g. the morphology of the digestive tract (discussed below). In particular, capuchins and humans are similarly encephalised as neonates; chimpanzee neonates are relatively non-encephalised in comparison (Watts, 1990). By adulthood, however, the ratio of brain to non-brain body mass has increased in humans to the extent that *Homo* has by far the largest brains relative to body mass of all the primates (Martin, 1990). Capuchins are the next most encephalised primate taxon (Martin, 1990). This implies that postnatal, rather than prenatal, brain and body growth variation contributes to differences in encephalisation between humans and capuchins.

What are these postnatal growth differences? Capuchin and chimpanzee brains have achieved a larger proportion of adult size by birth, compared to humans (Fig 7.1a). Humans are therefore unusual in the extreme amount of postnatal brain growth (approximately 75%) they must undergo to reach adult brain size. In terms of the brain allometry model described in Chapter 1, humans are born at a lower percentage of adult brain size achieved. If the allometry trajectory followed by humans is similar to that observed in capuchins, the position of human birth is shifted left, towards the lower body mass range (Fig 7.1b). Relative to adult body size, therefore, humans are predicted to have attained a smaller size at birth than capuchins. A 210g capuchin neonate weighs approximately 10% of its 2.2kg mother (Chapter 6); in comparison, a 3kg human neonate weighs 6% of its 50kg mother (Key, in preparation). It has been hypothesised that the size of the birth canal is a limiting factor in human prenatal brain growth (Martin, 1990). Birth in humans is relatively early compared to other taxa (i.e. human infants

; data summarised from 1° literature



Taxon	Neonatal brain mass (g)	Adult brain mass (g)	% adult brain mass at birth
Pan	128*	335**	38.21
Cebus	35*	67**	52.24
Homo	360^	1300^†	27.69

Female data only
*See Appendix 1.3
**See Appendix 1.2
^ Dekaban & Sadowsky (1978)
†mean of ages 19 to 40 years old

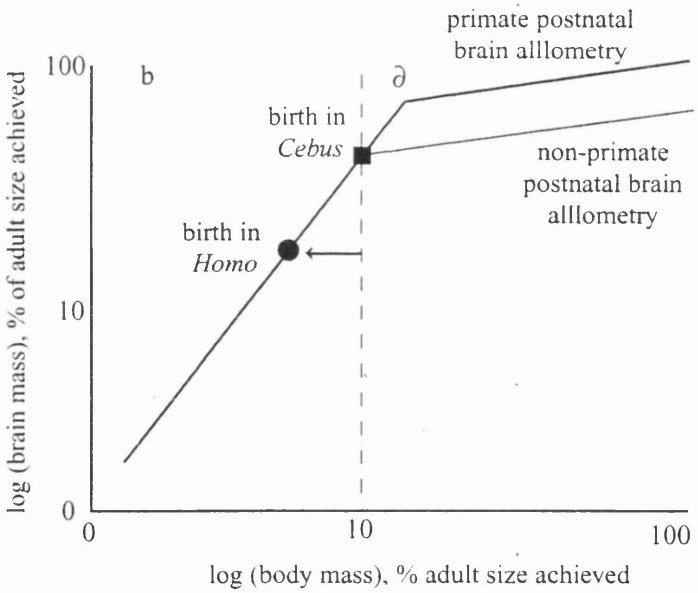


Figure 7.1. Comparing human and capuchin encephalisation at birth, and brain allometries. (a) Humans have achieved a smaller percentage of adult brain size at birth than either capuchins or chimpanzees. (b) Capuchins are born further along their brain allometry compared to humans.

are small-bodied in relation to adult size, Martin, 1990) and probably occurs at a body size at which the head is able to pass through the canal.

It should be noted that humans do not have short gestation lengths relative to adult body mass, when compared with other primates (Kihlstrom, 1972); rather, it is the position of birth relative to brain and body growth that is 'early' in humans. Human gestation length is long compared to neonatal body mass, but is as long as expected when neonatal brain size is used to predict gestation length (Little, 1989). Gestation length in humans is therefore associated with brain, rather than body, growth.

7.2.2 Comparing brain allometries

The following analyses uses the brain allometry model introduced in Chapter 1 to compare capuchin and human postnatal growth. Differences in temporal rates of growth are discussed separately (see below).

Three alternative explanations for the increase in encephalisation seen in *Homo* during postnatal development can be tested:

1] The exponents of the human brain allometry differ such that brain growth in *Homo* is more rapid than that of capuchins in either:

a] the pre-inflexion period,

b] the post-inflexion period, or

c] both (Fig 7.2a).

2] Alternatively, human and capuchin allometry curves follow similar exponents, but the 'shape' of the human allometry (i.e. RMA length) is different (Deacon, 1990). To achieve a higher level of encephalisation in adulthood, humans would have to display either:

a] a shorter post-inflexion RMA, or

b] a longer pre-inflexion RMA i.e. extend the period of rapid brain growth in relation to body growth, or c] a mixture of the two (Fig 7.2b).

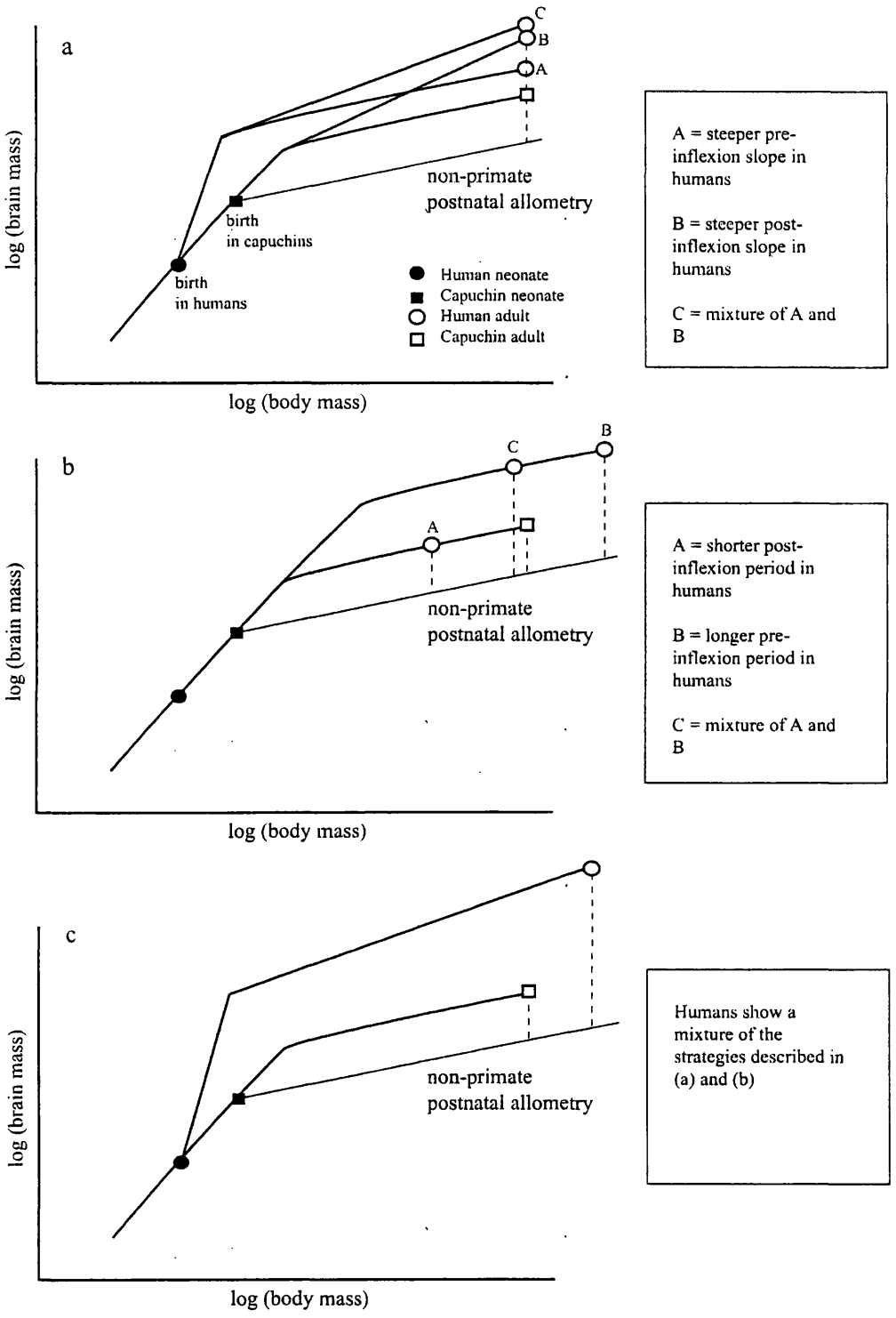


Figure 7.2. Modelling human brain allometries. (a) Predicted effects of slope differences in humans and capuchins; (b) predicted effects of length of pre- and post-inflexion period differences in humans and capuchins; (c) predicted effects of a mixture of slope and length differences in humans and capuchins. Each scenario is a possible explanation of the differences in encephalisation at birth seen between capuchins and humans, and are tested in the text.

3] Human brain allometries are different from capuchin allometries in both the shape (relative lengths of the RMAs) and exponents (Fig 7.2c).

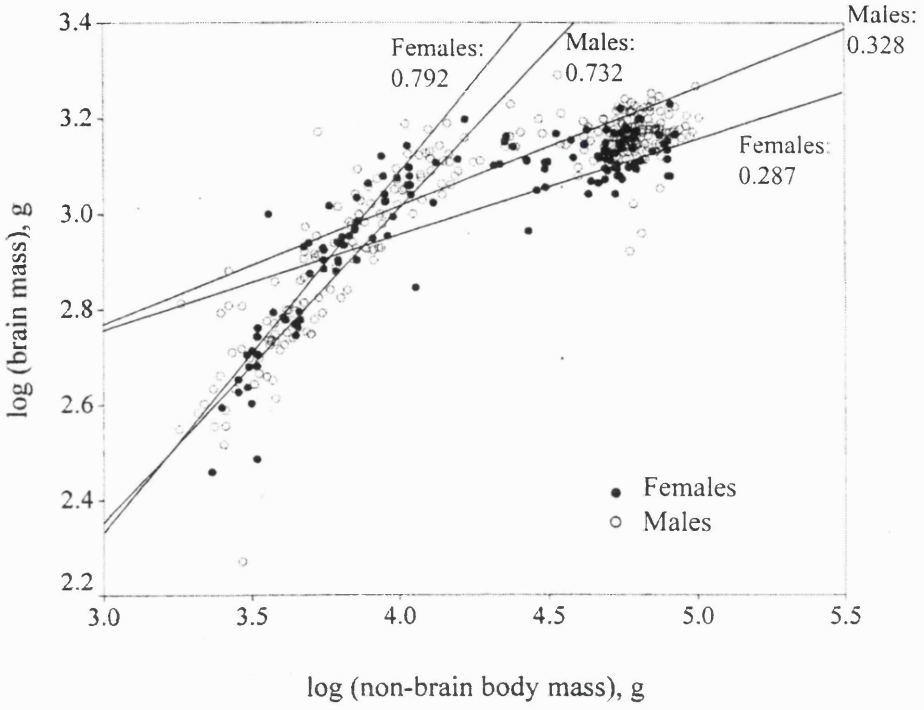
Previous authors have suggested that:

1] the rate of human postnatal brain growth relative to body growth is elevated when compared to other primate taxa (Deacon, 1990; Dienske, 1993), and

2] that the period of rapid brain growth (i.e. the pre-inflexion period in this model) is extended in humans (Martin, 1990). If both suggestions are correct, it would indicate that explanation 3 above is correct.

An analysis of human necropsy data shows prediction 3 to be true (Fig 7.3). In an analysis of necropsy data from 258 males and 137 females, the brain allometries for both sexes are significantly curvilinear (females: $F = 146.72$, $df = 134$, $P<0.001$; males: $F = 204.38$, $df = 255$, $P<0.001$), as they are in the majority of other primate taxa examined in Chapter 1. However, both the male and female human pre-inflexion exponents fall outside the 95% confidence interval for non-human primates in general and capuchins in particular (Fig 7.3). In Chapter 1, it was hypothesised that the pre-inflexion period represented an extension of the hypothesised prenatal growth curve, in which brain and body growth is isometric (see Chapter 1). Isometry is not included in the 95% confidence interval for the male and female human samples (Fig 7.3). This pre-inflexion period is not, therefore an extension of the prenatal growth period. However, human brains do grow at a faster rate (relative to body mass) after birth than all other primate brains, including those of capuchins (Fig 7.3). After the inflexion, the brain continues to grow in relation to body growth at a rate that is approximately twice as rapid as post-inflexion growth rate in capuchins and other taxa (Fig 7.3).

No significant differences between human males and females were found in any of the allometric slopes or intercepts (data not shown), and, because the RMAs describing the capuchin brain allometry in Chapter 1 are based on a sex-combined sample, sex-combined human RMAs were calculated for comparison (Fig 7.3). When the sex-combined capuchin allometry (see Fig 1.4, Chapter 1) and the sex-combined human allometry are compared, the ratio of post-inflexion RMA length to pre-inflexion RMA length is different in the two taxa



Taxon	Pre-inflexion RMA slope ± se	Post-inflexion RMA slope ± se
Non-human primates (Table 1.4)	0.524 ± 0.053	0.115 ± 0.013
Cebus (Table 1.4)	0.422 ± 0.047	0.086 ± 0.035
Cebus (Table 2.3)	0.367 ± 0.012	0.143 ± 0.090
*Homo** (females)	0.792 ± 0.047	0.287 ± 0.033
*Homo** (males)	0.732 ± 0.032	0.328 ± 0.028
*Homo** (sex-combined)	0.749 ± 0.775	0.329 ± 0.011

*Human data from necropsy records of the Sheffield Coroner's Office, c/o Forensic Pathology Department, Sheffield University (www.shef.ac.uk~dgm/op). See Chapter 1 for details of methods used to calculate brain mass and non-brain body mass.

Figure 7.3. Human brain allometry exponents are higher than those of non-human primates. Values shown in the plot are exponents.

(Fig 7.4). Figure 7.4 scales the capuchin and humans postnatal brain allometries so that the axes describe brain and body size in terms of percent of postnatal growth achieved. It should be noted that this is a different scaling from Fig 7.1, where brain and body masses are described in terms of percent of adult mass achieved. The scaling used in Figure 7.4 does not, therefore, include differences in prenatal growth, but compares the postnatal period of growth only.

In humans, the post-inflexion RMA is over twice the length of the pre-inflexion RMA (Fig 7.4a). In capuchins, the post-inflexion RMA is approximately half as long as the pre-inflexion RMA (Fig 7.4b). A greater proportion of human postnatal brain growth (approximately 75%) occurs at the slower, post-inflexion rate in humans compared to capuchins. In *Cebus*, a greater percentage of postnatal brain and body growth is achieved before the rapid rate of brain growth slows. It should be remembered that Figure 7.4 compares percentages of postnatal growth attained, rather than absolute brain and body masses. As we shall see below, human brain growth continues late into the juvenile period. Humans are therefore extending the amount of brain growth that occurs in the post-inflexion period, compared to capuchins.

In summary, prediction 3 (above) is correct. Humans have both higher pre- and post-inflexion allometry exponents, and a differently-shaped brain allometry (more brain growth occurs in the post-inflexion than in the pre-inflexion period). A greater proportion of postnatal brain and body growth occurs when the rapid rate of brain growth has ended. Capuchins are useful models for human adaptation primarily because both species face a similar ontogenetic problem: how to balance the costs of an altricial, relatively very large-brained neonate. However, human brains grow differently in the postnatal period, and compared to capuchins, humans have relatively larger brains during ontogeny and as adults. The following sections expand on this analogy, and compare the responses of both taxa to the challenge of growing expensive offspring.

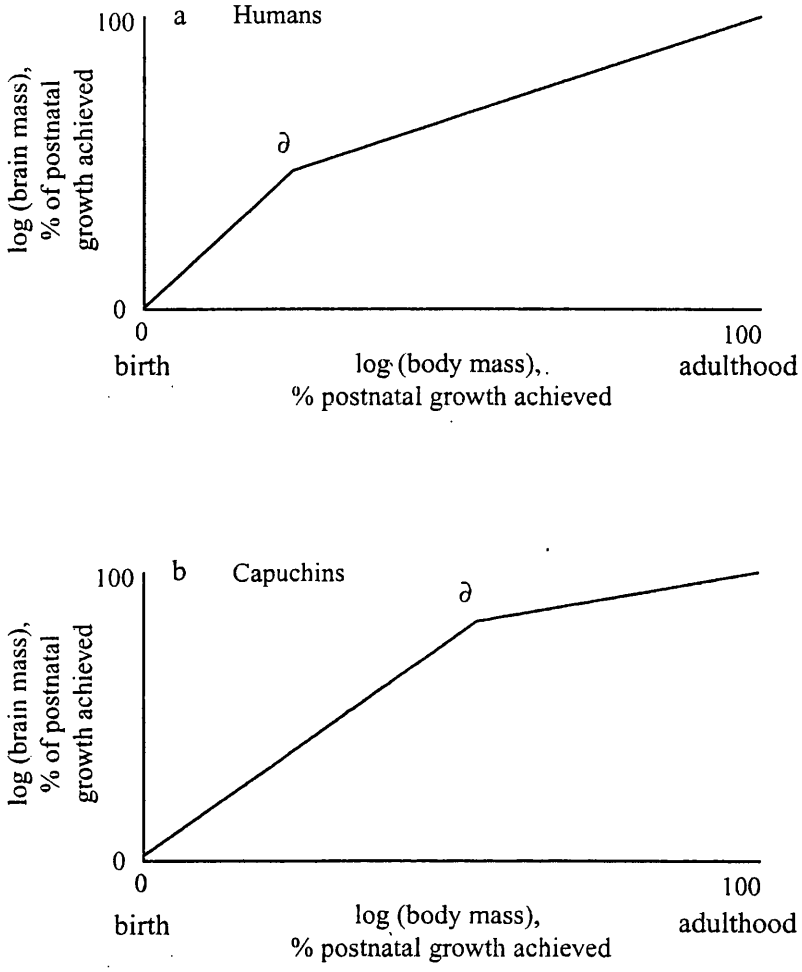


Figure 7.4. The shape of the human brain allometry is different from that of capuchins. (a) Human brain allometry; (b) capuchin brain allometry. A greater part of human brain and body growth occurs in the post-inflexion period compared to capuchins. Body mass in both cases is non-brain body mass. Inflexion in human sample calculated as described in Chapter 1. Human adult non-brain body mass calculated from the mean adult non-brain body mass (ages 19-40) in the Sheffield necropsy sample (see Fig 7.3).

7.2.3 Weaning mass in humans

We saw in Chapters 1 and 2 that brain and body masses at the inflexion of the brain allometry coincide with brain and body masses at weaning. The inflexion of the brain allometry in humans occurs at a lower percentage of postnatal body growth compared to capuchins (Fig 7.4). We can therefore predict that humans wean at a relatively smaller body mass compared to capuchins, i.e. that weaning mass and mass at inflexion both occur at a smaller relative size in humans.

In Lee *et al.'s* (1991) review of weaning masses, infants in both 'food enhanced' and 'food limited' human societies weaned at approximately 9kg. Lee *et al.* (1991) assume a human neonatal mass of 3.4kg; humans therefore wean when they weigh 2.7 times as much as they did at birth. The mean sex-combined birth mass in the human sample shown in Figure 7.3 is 3366.13g ± 507.44 (n = 8). If this figure is multiplied by 2.7, mean weaning mass in this sample of humans is also approximately 9kg (9088.55g).

If the typical weaning mass in the human necropsy sample is 9kg, it is 31.21% larger than whole body mass at inflexion for the combined-sex sample (6191.40kg; calculated from solving the pre- and post-inflexion RMAs shown in Fig 7.3, as described in Chapter 1). This is a similar percentage difference to those seen in other primate taxa (mean percentage difference for non-human primates: 31.0 ± 0.26, Table 1.7). Actual weaning mass is not know in the human necropsy sample, but it can be inferred that the inflexion of the brain allometry coincides with the end of lactation. This speculation requires a more detailed dataset for confirmation, but if correct, it has several implications for human lactation, which are discussed below.

It should be remembered that one of the assumptions made in earlier chapters was that capuchin weaning masses were approximated using the 4*birth mass ratio. It was noted then that considerable variation in this ratio (called the whole body mass ratio in Chapter 5) occurs across all primate taxa. However, mean whole body mass ratio in the CPC infants is exactly four (3.97 ± 0.43, n = 31; Table 5.4), and this assumption is likely to be valid. In summary:

humans differ from capuchins in the value of the whole body mass ratio, but the inflexion of the human brain allometry coincides with weaning mass, as it does in capuchins.

Interestingly, the first occurrence of infant food intake in humans tends to occur at the birth mass multiple seen in other primates (i.e. 2.1*birth mass; Bogin, 1999; Lee *et al.*, 1991). Thus humans begin the weaning process at the 'correct' body size, but complete it much earlier in ontogeny.

7.2.4 Long-chain fatty acids in human evolution

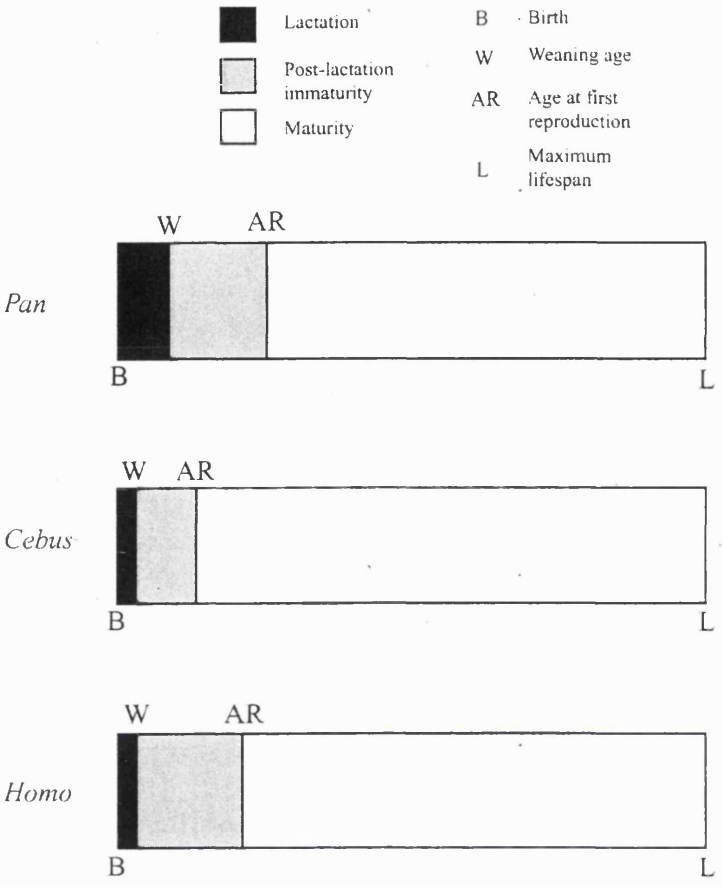
If the pre-inflexion period of rapid brain growth coincides with the period of lactation in humans, as inferred above, it can be hypothesised that the long-chain polyunsaturated fatty acids (LCPUFAs) found in milk are important in regulating human postnatal brain growth, as they are in capuchins (Chapter 4). Human infants are capable of synthesising LCPUFAs at around six months old, the same age as milk production begins to decline (Yehud & Motofsky, 1997). Before this time they are dependent on the placenta and then milk to supply their fatty acid needs. Even after this age, synthesis is relatively inefficient, and the majority of LCPUFAs must be taken directly from the diet (see below). The voracity of the fetal/infant brain for these pre-formed LCPUFAs is probably responsible for the 3% to 5% decrease in maternal brain size in the last trimester of pregnancy (Holdcraft *et al.*, 1997). A mother's brain is her largest store of AA/DHA, and it is likely that the fetal brain represents a 'sink' into which her own LCPUFA store must be donated (Horrobin, 1998).

If LCPUFAs are important in brain growth, the need for increased levels in humans (compared to other primates) is likely to continue after lactation. This is because the human brain continues to grow at a relatively fast rate after weaning, and because human brains are relatively large in comparison to body mass during ontogeny and as adults. Synthesis, incorporation of AA/DHA are relatively inefficient processes in humans, however, and it is likely that the explosion of brain size seen in human evolution 3-4 million years ago, and also in the last 50-200,000 years, could only be accommodated by long-term dietary changes that

dramatically increased LCPUFA uptake (Horrobin, 1998). AA and DHA are found predominantly in egg yolks, meat and organs of animals, and in the aquatic food chain: algae, molluscs, crustacea, fish, and aquatic birds and mammals all contain increased levels in their tissues (Broadhurst *et al.*, 1998). Encephalisation has increased dramatically over the course of human evolution, which has been characterised by long periods of brain size stasis followed by rapid change (Martin, 1990). The incorporation of these foods into the diet is likely to have occurred coincidentally within brain size increase i.e. in the diets of early *Homo* (2-3 mya) and of late Pleistocene *Homo* (Foley & Lee, 1991; Broadhurst *et al.*, 1998). The 'agricultural' diets of many extant human populations (i.e. those in which meat and/or shellfish and fish do not play a large part) provide saturated fats in large quantities which, when present in excess, displace EFAs from membrane phospholipids. This deficiency probably underlies the increased severity and incidence of diseases such as schizophrenia (which is aggravated by lack of LCPUFAs) in typically agrarian modern human populations (Horrobin, 1998; Peet *et al.*, 1999). As well as determining the level of LCPUFAs found in the body, diet also influences the LCPUFA and FFA composition of milk (Crawford, 1993), and incorporating meat, organs and foods from aquatic sources into their diets has probably helped humans cope with the increased metabolic demands of their infants' brains, as well as of their own. These foods also tend to be energy-rich and high in protein, two other important regulators of brain and body growth (Chapter 3). This is probably also the case in capuchins, which regularly consume faunivorous material (Janson & Boinski, 1992). Furthermore, this adaptation probably arose early in the cebine lineage, as *Saimiri* and *Cebus* (both relatively encephalised) consume meat (Fragaszy *et al.*, 1990). It is interesting to note that island-living capuchins have been observed to include oysters, a rich source of LCPUFAs, in their diet (Hernandez-Camacho & Cooper, 1976).

7.2.5 Weaning age in humans

The problems of defining weaning age in humans are well-acknowledged (Dettwyler, 1995). As well as weaning at a small body mass, human lactation also terminates at a relatively early age, both absolutely and in relation to the maximum lifespan (Fig 7.5). Humans and capuchins wean at roughly the same age, around one and a half years old. Although variation in human weaning age is large, a survey of non-bottle-feeding populations suggests that one and a half to two years is the age at which most humans are weaned (Dettwyler, 1995; Key, in preparation). When one considers the huge difference in body size between capuchins and humans, this is a striking phenomenon. This period occupies approximately the same percentage of the lifespan in the two taxa (~2.7%; Fig 7.5) primarily because capuchins have long lifespans for their body size, comparable with the chimpanzee lifespan in absolute terms (i.e. 40 to 50 years; refs.). The necropsy data presented above are scarce in the post-weaning period, but other published data show that brain size does indeed continue to grow after weaning, as the allometric model predicted (Fig 7.6). Only by six or seven years of age are human brains the same percentage of adult size (75%) as they are in non-human primates at weaning (Bogin, 1999). During this period, humans are relatively altricial (Martin, 1990). Adult dentition does not begin to erupt until late in this second phase of postnatal brain growth (6.25 years, Smith *et al.*, 1994). Other maturational markers do not occur until relatively late in adolescence, for example skeletal and reproductive maturity (Sinclair & Dangerfield, 1998). In this respect humans are more similar to chimpanzees, which are also weaned without their adult dentition and at a smaller percentage of body mass than capuchins (Fragaszy & Bard, 1997). In capuchins, the first permanent tooth has erupted by the age at which weaning is completed (Smith *et al.*, 1994; Fragaszy & Bard, 1997). Thus, even though the period of primary maternal dependency has ended, human juveniles are still immature with respect to both brain growth and other developmental systems during the post-infancy period.



Taxon	Maximum lifespan (y)	Weaning age (y)	Age at first reproduction (y)
Pan	53^	4.0*	13.0^
Cebus	44^	1.2**	5.5^
Homo	65†	1.7*	14.0^

Female data only where possible
*Key (in preparation)
**Fragaszy & Bard (1997)
^Ross & Jones (1999)
†Smith (1992)

Figure 7.5. Relative lactation length in *Cebus*, *Homo* and *Pan*.
Humans and capuchins show similar lactation lengths compared to total lifespan (L), even though they have very different ~~body~~ sizes.

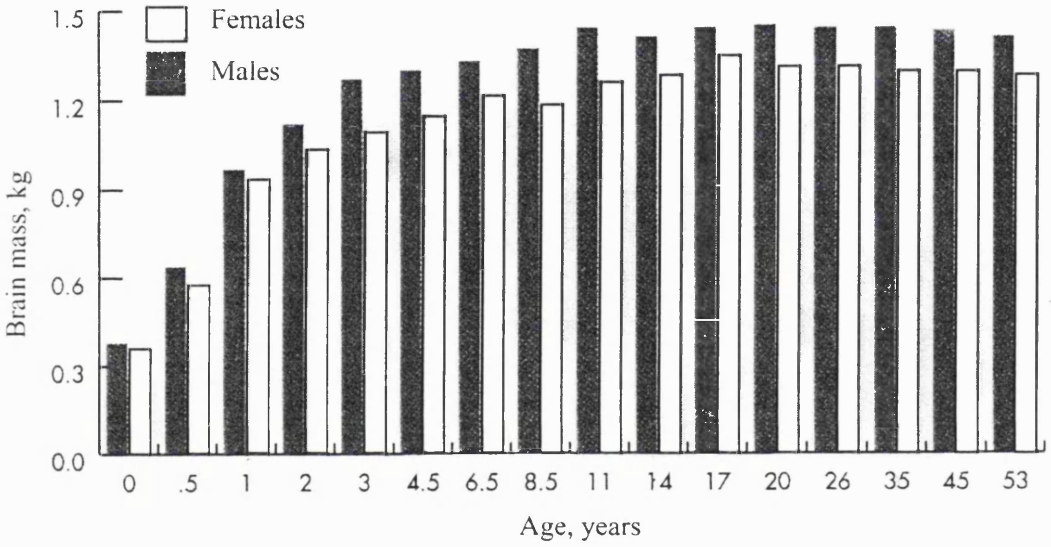


Figure 7.6. The tempo of human postnatal brain growth. Taken from Nolte (1999).
Only by the age of 6 or 7 years have both sexes achieved more than 75% of adult brain mass.

7.2.6 Energetics and the weaning threshold in humans

Why should human mothers wean their infants when a significant amount of brain growth has yet to occur? One possibility is that infant brain and body growth are regulated such that the total metabolic cost of the infant is sustainable by the mother during lactation. None of the metabolic rate variables analysed in Chapter 5 correlated with maternal mass independently of infant size, but total metabolic rate at separation did correlate with the metabolic rate of the brain and the metabolic rate of the body independently of each other (Table 5.10.2). This indicates that total metabolic rate at separation is determined by both brain and body mass independently. The weaning threshold observed by Lee *et al.* (1991) would therefore appear be dependent on both brain and body metabolic costs, as the results of previous chapters suggested. Human mothers are likely to wean their infants at an early stage of both brain and body growth because to prolong lactation would be to go beyond the threshold set by maternal metabolic capacity. Infants that are exclusively breastfed late into childhood (i.e. longer than two years postpartum) tend to increase in mass more slowly than infants that are weaned to solid foods early (Prentice & Prentice, 1990). This suggests that maternal energetic capacity cannot support prolonged infant growth.

7.2.7 Energy-sparing mechanisms in capuchin and human evolution

Humans may have coped with the increased energy demands of encephalisation by reducing the size (and therefore the metabolic costs) of other 'expensive' tissues. For example, the relatively small size of the human gastrointestinal tract represents a cut in tissue maintenance costs that balances those associated with increased brain size (Aiello & Wheeler, 1995). Such a change in morphology is associated with the shift towards high-energy food (e.g. meat) consumption noted above. As with meat-eating, capuchins are convergent with humans in the gastrointestinal tract morphology: as Fragaszy *et al.* (1990) observe, "the length of the large intestine relative to the length of the small intestine, and to body size, is among the shortest in

primates, and is similar in this respect to humans" (p115). Such an energy-sparing mechanism would be advantageous soon after weaning, providing weanlings had access to sufficiently energy-rich sources of food. Weanling capuchins are relatively inefficient foragers (Fragaszy & Boinksi, 1995), and (as noted above) the occurrence of food-sharing between adults and older infants may serve to ensure that weanlings have access to 'difficult' high-energy foods such as palm nuts (Defler, 1979; de Waal *et al.*, 1993). Food-processing and food-sharing activities in humans enable human infants to access a nutrient-rich diet soon after weaning (Foley & Lee, 1991), and are probably important in alleviating the reproductive costs of females by sharing the increased costs of foraging and other activities associated with infant investment between individuals other than the mother. As with allonursing in capuchins, and with the other allomaternal care strategies seen in the smaller neotropical primates (Tardif, 1994; Garber & Leigh, 1997), humans have evolved a way of sharing female reproductive costs, and increasing inclusive fitness, across the group.

Human infants also appear to offset the energy costs of growth with those of activity in the first year of life, when postnatal brain growth is at its most rapid (Wells & Davies, 1998; Fig 7.7). Although the activity patterns of the HSPH and CPC capuchins are unknown, the results of earlier chapters suggest that a similar trade-off might occur in capuchins i.e. that activity (measured as locomotor activity) increases as the rapid phase of brain growth is completed (Chapter 4). Body growth, as well as behaviour, is of importance in determining patterns of energy expenditure; for example, the best predictors of total energy expenditure in 9 and 12 month old human infants are fat-free mass, time spent feeding, and time spent upset (i.e. in an agitated state; ~~Davies *et al.*, 1997~~ Wells *et al.*, 1997). In younger infants (12 weeks old), the main aspect of behaviour to influence energy expenditure is time spent sleeping, which is also inversely correlated with activity energy costs (Wells & Davies, 1996). Capuchin infants also spend much of their early infancy sleeping or drowsing (Fragaszy, 1989), and this may help to regulate energy expenditure during the early phase of growth.

Early infant activity patterns, and gastrointestinal proportions, appear to be similar in humans and capuchins. Both probably represent energy-sparing mechanisms evolved to cope

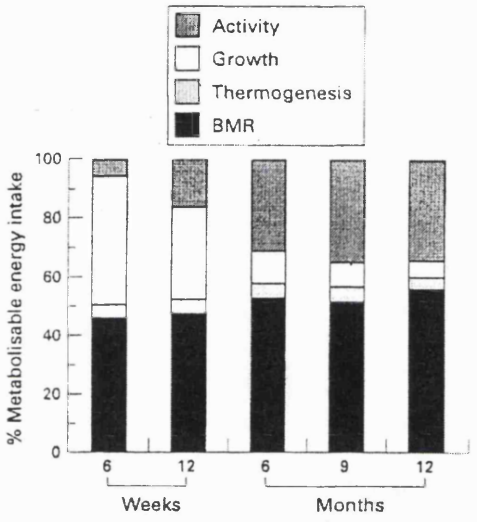


Figure 7.7. Components of energy expenditure in human infants. Taken from Wells & Davies (1998). Note that, whilst BMR and thermogenetic costs are similar over all ages, the proportion of metabolisable energy that is devoted to growth shows an inverse relationship to the proportion of energy devoted to activity. See Wells & Davies (1998) for data details and definition of terms.

with the increased costs of encephalisation. However, in comparison with capuchins, and other primates and non-primates, human infants are born with increased levels of white adipose tissue (WAT; Kuzawa, 1998). WAT may help to insulate neonates, thereby reducing the energy costs of thermogenesis, but its main metabolic role is likely to be as a store for large amounts of energy (Kuzawa, 1998). As noted in Chapter 1, infant metabolism switches from glucose to lipids soon after birth, and an infant's carbohydrate reserves are very soon exhausted (Pond, 1984). Thus WAT serves to ensure a constant supply of energy is available to the infant in the early stages of lactation (Kuzawa, 1998). When the human neonatal brain consumes as much as 60% of the total metabolic expenditure (Aiello & Wheeler, 1995), WAT is likely to be an important energetic buffer. Capuchin infants appear to be similar to other mammals in terms of neonatal fatness: fat constitutes approximately 2% of the neonatal body (total carcass mass; Ausman *et al.*, 1982) compared to 15% in humans. A typical mammal neonate (including non-human primates) is approximately 2-3% fat (Kuzawa, 1998).

7.2.8 Section summary

Capuchins and humans show some convergent adaptations to the costs of raising expensive offspring. Although the weaning process begins (i.e. first food intake) at the same relative body size in both taxa, human infants are weaned at a smaller body mass. This may be because the elevated rate of brain growth seen in humans means that the metabolic weaning threshold is reached at a smaller infant body mass. Weaning also occurs at an early point in ontogeny: human weaning age is similar to that of the much smaller capuchin, both absolutely and in relation to the length of the lifespan. Relative to the pre-inflexion period, the post-inflexion period is extended in humans. Humans also show an elevated rate of brain growth, compared to other primates, in the post-inflexion period.

The increased LCPUFA intake associated with meat-eating is likely to have played a key part in both capuchin and human evolution. Both have gut morphologies that are specifically adapted to high-energy food consumption. Humans and capuchins have also

evolved energy-sparing mechanisms to cope with expensive offspring. Food-sharing exists in both taxa, but is more prevalent in humans, who also have the ability to process food before consumption. This was probably important in ensuring the weanling human had access to high-energy foods. Human and capuchin activity costs appear to be inversely correlated with growth costs during the brain growth period. This may also fulfil an energy-sparing role in growth. The fatness of human neonates appears to be a unique adaptation among the primates to fuelling the high energy requirements of the brain immediately after birth.

7.3 Conclusion

- Do different primate species share a common pattern of postnatal brain ontogeny, and how does this pattern compare with that seen in other mammals?

The primate postnatal growth period is characterised by two phases of brain growth. In the first, the brain grows in relation to the body at a rate that is faster than predicted for other mammals. In the second, the rate of brain growth in relation to body growth is comparable with that seen in other mammals. Humans are the exceptions to this trend: the exponents describing brain growth in both growth periods are elevated compared to non-human primates and to non-primate mammals. Humans are also unusual in the shape of their postnatal brain allometry compared to another encephalised taxon, *Cebus*.

- How do ontogenetic changes in brain and body size relate to lactation and weaning strategies?

This thesis confirms the hypothesis that brain growth and lactation strategies are associated. The end of the maternal investment period (weaning) coincides with the attainment of 75% of adult brain size. Infant growth is limited by maternal investment i.e. by the costs associated with growing and maintaining a large brain. Infants have also achieved a certain level of

behavioural and physiological maturity by the time lactation ends. The weaning threshold is therefore both metabolic and behavioural in nature, and is associated with brain, as well as body, growth. Weaning age itself is directly correlated with the amount of brain growth undergone in the postnatal period in one encephalised taxa, *Cebus*. These results also underline the importance of maternal mass, and probably paternal mass, as determinants of infant growth.

- How do ontogenetic changes in brain and body size relate to other aspects of primate life history?

The weaning threshold is influenced by both brain and body size in primates. In addition, brain growth is directly associated with the length of the infancy period, which impacts upon reproduction in females and growth in infants. Other developmental markers, for example, foraging behaviours and dental eruption, are delayed until brain growth and weaning is completed. These metabolic and behavioural associations of relative brain growth and weaning have important consequences for other aspects of life history: they lend support to the models of life history that predict brain growth constraints on life history are both metabolic and behavioural in nature.

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Appendix 1.1 Contact details for dataset

The necropsy and EA data presented in Chapters 1 to 7 are available from the author in electronic form (SPSS 7.5 for Windows 95). The whole body x-rays measured as part of this thesis are also available from the author (created in Photoshop 5.5 using UMAX MagicScan 4.3 on a PowerMacintosh; 150dpi; files are .tiff (LZW compressed). Archived as a set of 3 CD-Rs). Please contact:

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For more information on the HSPH x-ray collection, contact:

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Appendix 1.2 Neonatal brain and body masses gathered from the literature[1]

Taxon	Neonatal body data: Minimum reported mean body mass (g) A		Maximum reported mean body mass (g) B		Average mean body mass (g) C = [A+B]/2		Neonatal brain data: Minimum reported mean brain mass (g) D		Maximum reported mean brain mass (g) E		Average mean brain mass (g) F = [D+E]/2		Neonatal non-brain (NNB) body mass (g) G = C – F	
Saimiri	M	113	M	114	M	114	M	14	M	16	M	15	M	99
	F	105	F	110	F	108	F	14	F	15	F	15	F	93
Cebus	M	221	M	243	M	232	M	35**	M	35**	M	35	M	197
	F	197	F	212	F	205	F	33**	F	33**	F	33	F	172
Ateles	M&F	426	M	480	M	453	M&F	64	M&F	64	M&F	64	M	389
	F	425	M&F	512	F	469							F	405
Alouatta	M&F	440	M&F	480	M&F	460	M&F	31	M&F	31^	M&F	31	M&F	429
Cercocebus	M	517	M	517	M	517	-	-	-	-	-	-	M	-
	M&F	473*	F	530***	F	502							F	-
Macaca	M	453	M	490	M	472	M&F	52	M&F	58	M&F	55	M	417
	F	473	M&F	481	F	477							F	422
Pan	M&F	1560	M	1877	M	1724	M&F	128	M&F	128	M&F	128	M	1596
			F	1814	F	1687							F	1559
Pongo	M&F	1500	M	1965	M	1733	M&F	129	M&F	170	M&F	150	M	1583
			F	1653	F	1577							F	1427
Gorilla	M&F	1750	M	2251	M	2001	M&F	227	M&F	227	M&F	227	M	1774
			F	1996	F	1873							F	1646

[1]M = male; F = female; M&F = sex combined data. See Appendix 1.4 for references.

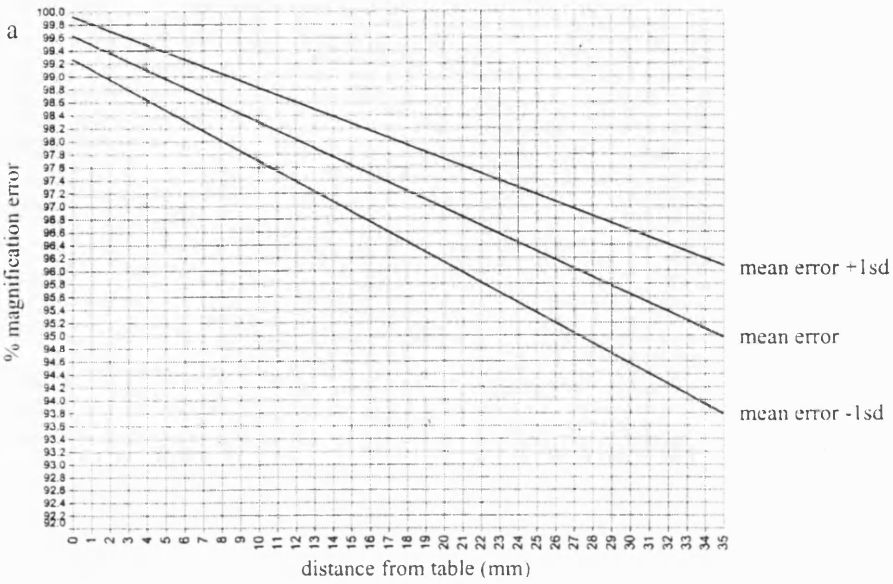
*Data from *Cercocebus agilis*.

***Data from *Cercocebus atys*.

**Data from *Cebus albifrons*.

^Data from *Alouatta palliata*.

a



b

	Table	Table +25mm
Distance from table (mm)	00.00	25.00
Mean increment length (mm)	50.22	51.88
Standard deviation	0.140	0.453
Magnification error	99.60	96.34

Figure A2.1. Calculation of magnification error. (a) Magnification error plotted by distance from table, based on data in (b). Standard deviations of mean shown. (b) Magnification error data.

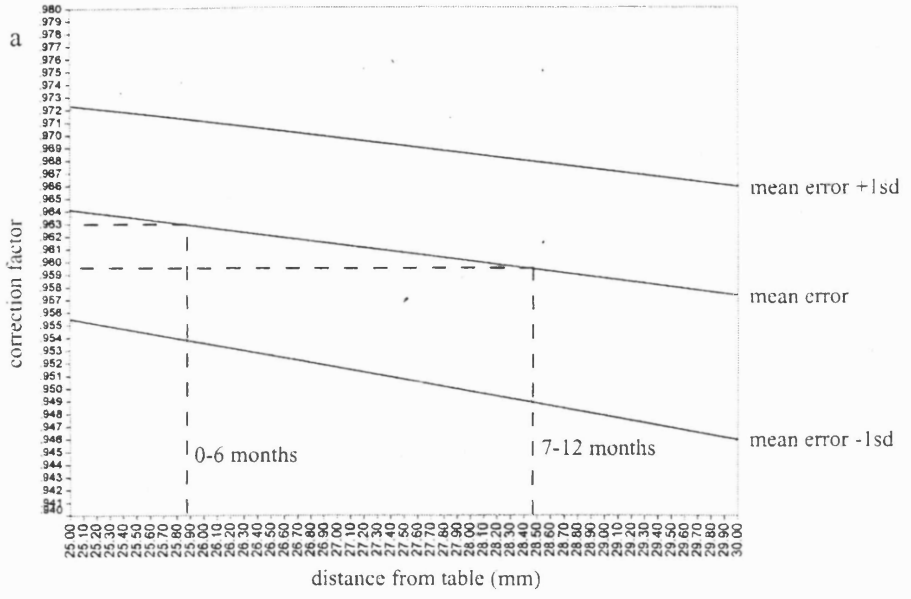
mean [head breadth]/2 in the older age group was 28.47mm ± 1.07 (Fig A2.2). These figures represent the mean distance from the x-ray table at which EA was measured in the two groups.

From Fig A2.2, it can be seen that the appropriate magnification factors for the 0-6 month age group and the 7-12 month age group were 0.963 ± 0.008 and 0.959 ± 0.009 respectively. The latter factor was applied to EA measurement of infants older than 12 months old.

3] Error estimation

The calculation of magnification error in the x-rays includes error that arises from variation in infant head breath observer measurement error. The effect of measurement error was investigated as follows: if a 5-month-old infant's raw EA measurement was 1720.00mm^2, multiplying this measurement by the appropriate magnification factor (0.963) results in a 'real' EA measurement of 1656.36mm^2. Multiplying the raw measurement by the magnification error minus one standard deviation results in a lower estimate of the real EA of 1640.88mm^2. Multiplying the raw measurement by the magnification error plus one standard deviation results in an upper estimate of the real EA of 1670.12mm^2. The difference between this lower and upper estimate only represents 1.76% of 1656.36mm^2, the 'real' EA measurement. Similar levels of error are found in individuals belonging to the older age group (data not shown). It should be remembered that this error will compound other sources of error in the EA calculation (see Chapter 2).

a



b

Age (mo)	N	[head breadth]/2	s.d.
0-6	273	25.88	2.14
7-12	225	28.47	1.07
13-18	111	28.88	1.01
19-24	85	29.14	1.26
25-30	74	29.47	1.05
31-36	7	29.82	1.59
37-42	44	29.73	1.25
43-48	70	29.94	1.26
49-54	53	30.23	1.29
55-60	38	30.66	2.02
61-66	24	30.95	2.20
67-72	13	30.60	1.90

Figure A2.2. Magnification correction factor (a) Correction factor plotted by distance from table. This figure is a a higher resolution version of the one shown in Fig A2.1. Correction factor for infants aged 0-6 months: 0.963 ± 0.008; correction factor for infants aged 7 months and older: 0.959 ± 0.009. See text for discussion. (b) Capuchin head breadth data.