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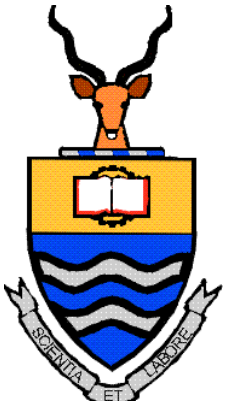
The Panglossian Paradigm Revisited: The role of non adaptive mechanisms in hominid brain and body size evolution

Muhammad Aadil Spocter

This research report is submitted in fulfillment of the requirements for the degree of Doctor of Philosophy



SCHOOL OF ANATOMICAL SCIENCES
UNIVERSITY OF THE WITWATERSRAND, MEDICAL SCHOOL
JOHANNESBURG

January, 2007

Declaration

I declare that this thesis is my own, unaided work. It is being submitted for the Degree of Doctor of Philosophy in the University of the Witwatersrand, Johannesburg. It has not been submitted before for any degree or examination in any other University.

_____

Muhammad Aadil Spocter

____ day of _____ 2007

Abstract

The largely dominant adaptationist argument is currently used as the framework within which hominid brain evolution is explained; however these adaptationist explanations are inherently problematic and only suffice to 'clutter' our knowledge of the possible causes of hominid brain evolution. This study addresses the caveats observed in the fossil record and aims to assess the relative influence of structural laws of form, phylogenetic constraints, and adaptive factors during the course of primate and hominid brain evolution. A combination of methods such as variance partitioning, phylogenetic regression procedures and path analysis indicate that constraints have played a critical role in the scaling attributes of the primate and hominid brain. In particular, developmental constraints governing the scaling attributes of the skull and body are shown to explain up to 50 % of the variation in body mass whereas phylogenetic constraints are purported to have played a lesser role (i.e. 0.8 -3.6 %). In addition, the scaling attributes of neural and non-neural components of the cranial vault suggest a highly constrained suite of traits and suggest that as much as 96 % of the variation in both brain mass and residual endocranial space may be explained by correlated scaling with the cranial vault. Constraints are observed to be far more pliable than traditionally thought – a feature highlighted by intraspecific analyses of scaling attributes in humans. Low regression coefficients typical reported for intraspecific curves are shown to arise during development as greater variation in body parameters is allowed with advancing age. Grade shifts in the scaling of brain and body size for primates and other mammalian orders is also emphasised by this current study and it is argued that correlated changes between the brain and body size may not necessarily impact upon the 'complexity' of

the neural system as the functional integrity may be maintained via higher output states initiated at certain levels of organisation such as at the level of the cortical area. Although constraints should rightfully be given greater coverage in explanations concerning hominid brain expansion, it is only through implementation of research protocols that take a pluralistic approach to an understanding of the role of both constraints and adaptation in the formation of the brain that our interpretation of the likely mechanism for hominid brain expansion may be understood.

To my family, Abeda, Naziem and Naim Spocter,

with love and appreciation.

Your constant confidence, love and dedication have always supported me through failure and success.

Acknowledgments

I wish to acknowledge my gratitude to the many people that have contributed towards the completion of this thesis. First, this thesis would not have been completed without the support of my supervisor, Professor Paul R. Manger. I thank him for his astute suggestions, discussions, unfailing support and patience. He has been an exemplary model for enquiry and an 'academic father' to whom I owe an ever growing penchant for challenging norms and for adhering ever closer to the edge of Ockham's Razor. His insistence on professionalism and sound reasoning has been matched only by his calming and friendly demeanor and has made this thesis even more so enjoyable during its inception and now a sad retrospect on what has been an absolute joy for the greater part of three years.

For the financial support which has made this research possible, I would like to extend my thanks to the National Research Fund (NRF), Paleontological Scientific Trust (PAST), J.J. Smiesjeck bursary, the Postgraduate Merit Award from University of the Witwatersrand, Johannesburg and the team of Paleotours (Marianne Robinson and Colin Mentor). Your efforts towards promoting evolutionary studies and the fostering of young minds especially those from previously disadvantaged backgrounds, has gone along way towards extending South Africa's rich fossil heritage and a culture of enquiry epitomized by iconic scholars such as Phillip V. Tobias and Sydney Brenner.

Special thanks must go to Professor Emeritus John Alan. I have always enjoyed our intriguing discussions and your cautious reminder for 'simplicity'. Your enthusiasm for anatomy, statistics and your love for teaching are shimmering beacons which I carry with me through life. Thanks must also go to Professor Emeritus Phillip

Tobias who always had a word of encouragement and whose door was always open to adding comment and guidance to certain aspects of this research. The value of having been in a Department where the likes of Prof. Tobias and Prof. Clarke and on any given day a myriad of other internationally renowned scholars could be seen walking the corridors and engaging in discussion with students and colleagues, has left a lasting impression on me.

A note of special thanks must go to the numerous technical and secretarial staff both internally from the School of Anatomical Sciences and the Sterkfontein Research Unit and the external assistance from various groups such as the Donald Gordon Medical Centre, Johannesburg Mortuary, Hamburg University Hospital, and the Northern Flagship Institute. Much of this work would not have been completed if it were not for your timely assistance during the data collection phase of the various projects presented here. To Elijah Mofokeng, the time weathered friend and curator of the Raymond Dart Collection of Skeletons, thank you sincerely for your assistance through the years. On behalf of all the countless students and established researchers whom you have helped, I wish you a speedy recovery and thank you for the great conversations, fleeting jazz and sharp humour which permeated from your office and made the data capturing procedure that little more enjoyable over the years.

I also owe thanks to the following people for their assistance during the completion of the following chapters.

Chapter 1: Elijah Mofokeng, Fred Ladner, Manoj Chiba, Kevin Kuykendall, Colin Menter, Manisha Dayal and Clarissa Snapper. Also to Francis Thackeray for permission to use the CT scanned images of the STS 5 specimen, Mike Raath for assisting with the acquisition of the digital scans for the remaining South African

specimens, and to Gerhard Weber and his team at the University of Vienna for providing us with CT scans of the South African Australopithecines.

Chapter 2: Dr. Mark Haagensen and the team from the MRI Unit at the Donald Gordon Medical Centre, Dr. Calle Undeman and Prof. Per Roland from the Karolinska Institute, Stockholm, Sweden (from work funded by grant EU-LSSM-CT-2003-504752), Dr James Rilling for giving permission for the use of a pre-published primate MRI dataset, Dr Colin Menter, Ms. Clarissa Snapper, Dr Yves Desdevises for his comments regarding the use of the technique of variance partitioning, and Prof Emeritus Phillip Tobias and Prof Emeritus John Allan for the numerous informative discussions entered into concerning aspects of this research.

Chapter 3: The staff from the Hamburg University Hospital, Federal Republic of Germany and Prof. Scholtz from the Johannesburg Government Mortuary, for there efforts in the procurement of the databases representing the geographic samples. In addition we also wish to thank Dr Sean Rice for making himself available for consultation on the methods of detecting heterochrony. Also to Prof Emeritus John Alan for the numerous informative discussions entered into concerning aspects of this research. I would also like to thank Dr William Divale from the Department of Social Sciences, York College, Jamaica New York for acquisition of the World Cultures Journal volume 1 -15.

Chapter 6: Dr Michael Hofman for making the micrencephalic data available for comparison in this final chapter.

A special note of thanks must also go to my girlfriend Clarissa Snapper and my friends in particular, Altaf Khumree, Olebogeng Moeng, Manoj Chiba, Brendon Billings, Jason Hemingway, Anthony Kegley, Justin Adams, Wanjeera Maina, Manisha Dayal, Salman Khan and Yin Hlaing. To the ‘usual suspects’ who ply their

trade outside the medical school entrance, consuming countless cups of coffee whilst puffing away at cigarettes and debating over the weekends activities, thank you for the much needed diversion during those periods when completing this contribution seemed all to distant.

Lastly, I wish to thank my family Abeda, Naziem and Naim Spocter for having given me the opportunity to further my studies and to pursue my dreams. Yours has been a contribution which I will carry with me to the end of my days. Thank you for the loving unconditional support I received from you over the years. As Albert Camus once wrote: "*A mans life is nothing but a slow trek to rediscover through the detours of art those one or two great and simple images in whose presence his heart was first opened*". Thank you for opening my eyes, heart and mind to the beauty of scientific enquiry. It is to you that I dedicate this contribution!

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Chapter 1

The Panglossian Paradigm Revisited: The role of non adaptive mechanisms in hominid brain and body size evolution

1.1 Introduction

Probably no branch of biology is more charged with immoderate, emotive and misleading statements than that which deals with man himself (Tobias, 1971).

Humans are a species obsessed with their own uniqueness. It is an obsession that has seen us devote an increasing amount of both rational and esoteric discussion towards understanding our place in nature. An obsession that has driven us towards the ends of enquiry, all to answer a deceptively difficult question: What is it that makes us human?

As an almost reflex like tendency, the relatively large human brain had been historically isolated as definitive of our cognitive and technical abilities; however, subsequent evidence would point towards the conclusion that the human brain is the serendipitous by-product of an evolutionary trajectory that started some 7-8 million years ago. Together with bipedality our hominid ancestors are surmised to be making use of rationalization and intelligence as an adaptive advantage. It is this cerebral adeptness and proficiency which has on the one extreme yielded the ilk of Einstein and Bach and yet on the same account has spawned racism, genocide, terrorism and a myopic potentially for self destruction. It is the intellect that sets humans apart from the other animals and that has fuelled our ongoing obsession with humanity. But a study of the human brain in isolation approximates only a limited set of answers and in fact says nothing about the evolutionary history of hominids, after all humans are primates and would be best investigated within the backdrop of this Order. But do we

stand to learn anything from analyses of the brain in non-human primates, and just what does it mean that gorillas and capuchin monkeys aren't asking questions as to their origins and how they came to be? What does it mean that despite the remarkable similarity between chimpanzees and humans, we are as yet to come across a chimp feverishly typing away on a laptop preparing a manuscript on the evolutionary trajectory of the chimpanzee brain?

If the evidence were clear-cut and the answers obvious, then there would be no need for any further discourse on the matter, but it is not. Indeed it is far from it as there remain few scientific questions as decorated with unproven scientific proclamations and held together by convenient omissions as that which attempts to explain the dichotomy between ape and human mental capabilities. With this in mind, it comes as no surprise that when a paleoanthropologist seeks to explore the possible mechanism by which the hominid brain emerged, a reckless path is tread. Departures from scientific rigour and a plethora of less parsimonious explanations has left the scholar palpably confused as to the most likely means by which the human brain evolved and thus an overall reassessment is necessary.

To date no completely satisfactory hypotheses have been proposed regarding the adaptive or other evolutionary forces resulting in the modern human brain – available hypotheses have met unresolved challenges or suffer from lack of substantiating evidence (e.g. concerning the evolutionary link between increases in brain size and language). Most scenarios present evolutionary increases in brain size in an adaptive context – large brains allowed early ancestors, to overcome challenges with cunning derived from a superior intellect. A popular notion in the 1950's was the hypothesis that the obvious differences between hominids and apes was the making and using of stone tools and that this was the most likely cause for brain expansion

(Wynn, 1991). This hypothesis, encapsulated by the 'Man the Tool-maker' slogan, explained the tripling of hominid brain size as being accompanied by the increasing complexity of the tool industry. A decade later, this hypothesis was replaced by the slogan 'Man the Hunter'; however, both hypotheses saw the engine of hominid brain expansion as being the mastery of practical affairs (Lewin, 1999). More recently, ideas have emerged which seek answers within the realm of primate social life (i.e. 'Man the Social Animal') and believe that the mastering of culture and language *via* the underlying complexity of primate social alliances and networks, had allowed hominids to remain the proverbial 'cognitive step' ahead of predators, prey and the environment (Dunbar, 1992, 1998; Seyfarth & Cheney, 1992); however, most discussions about hominid brain evolution do not give much consideration to primate evolutionary history, or to related evolutionary factors such as primate phylogenetic/developmental constraints.

For example, in Fleagle's 1999 textbook "Primate Adaptation and Evolution", there is only one paragraph and one figure (pg.307-308) out of the book's 596 pages discussing the issue of causal factors in brain evolution of primates. Fleagle cites the "gossip hypothesis" of Dunbar (1992, 1998) that relates primate brain size and relative neocortical size to average social group size, with the conclusion that increases in brain size are driven by increasing complexity in social interactions, rather than the need to remember food locales or to monitor the environment. First, the model presented in Fleagle's text is clearly adaptationist, suggesting that any increase in brain size must have an adaptive basis. Second, this model focuses on a primate adaptive trait – complex social behaviour- that is also invoked in hominid evolutionary/adaptive models. Fleagle's text may be considered to be somewhat conservative in order to appeal to a more general audience, but it is notable that there

is no consideration of alternate, non-adaptive models in primate brain evolution. What if we examine a less conservative approach to primate and human brain evolution? One of the more recent and bold attempts at synthesizing the various observations made on primate and hominid brain evolution is the book "Evolving Brains" (Allman, 1999). The final chapter of the book is an attempt to synthesize a broad range of observations and theories, including trade offs between gut and brain weight, developmental observations, dietary considerations, retinal evolution, longevity, parenting, metabolism, sociality, gender specific death rates, neural reactions to stress, climatic variations, paedomorphy and even dancing with wolves – in the end confusing rather than clarifying the evolution of the brain. Each of these factors is coupled to different adaptations determined from analysis of "brain residuals", however, no clear synthesis emerges, and there is no attempt to include phylogenetic contingencies, structural laws of form, or any other alternative factors.

Thus the adaptationist view, even at its most elaborate, fails to shed any distinct or potent concepts regarding primate and hominid brain evolution and generally fall into a chicken and egg argument. For example, in explaining the 'Expensive Tissue Hypothesis' Allman proposes that a higher quality diet with easier digestion of food will lead to a smaller gut allowing more energy to be channelled to the brain allowing it to become larger (Aiello & Wheeler, 1995). This in turn enhances the ability of the organism to find more and higher quality food, resulting in a higher quality diet and so on. But what came first, and what is the evolutionary or selective stimulus in such a network? This kind of explanation involves adaptationist theory, but is not causal, and therefore is not ultimately explanatory. In a somewhat clearer attempt at adaptationist explanation, McKee (2000) has proposed the "autocatalytic" feedback loop of human evolution, with such adaptations as dietary

niche, bipedalism, manual dexterity, material culture and language all interacting with each other and the brain to cause elaboration of the brain over time, a self-reinforcing system.

In stark contrast to these adaptationist views, although not directly applied to hominid brain evolution, is the recently proposed "developmental constraints hypothesis" of Finlay and co-workers (Clancy *et al.,* 2001; Finlay and Darlington, 1995; Finlay *et al.,* 2001). These studies of brain morphology in extant mammals have demonstrated those structural laws of forms in relation to brain evolution. These observations, coupled with observations on brain and body weight scaling in primates, demonstrate that the majority of brain evolution within the order primates might be explained by structural laws of form and phylogenetic constraints, as both the internal proportions and size of the brain are statistically significantly correlated to each other or to body size respectively, and the form of the brain is clearly homologous across primate species. The one clear exception to these laws of form is the relative brain size of modern humans, being far larger than predicted for body weight; however, the relative internal proportions of the human brain are exactly what would be expected for a primate with a brain size averaging around 1300 grams.

So what do we make of these disparate views? Evolutionary theory, as assessed in an open manner clearly indicates that there are three major influences on the genesis of form, these being adaptation, phylogenetic history and structural laws of form (Gould, 2000). The latter two are closely related, but the differences can be examined if comprehensive data is available. While these three factors are by no means mutually exclusive, explanations involving phylogenetic constraint or structural laws of form as primary influences usually do exclude adaptationist considerations since they are essentially non-selective mechanisms for change. It is

the determination of the relative balance of these three influences that ultimately provides the most informative evolutionary explanation for morphology. It is the aim of the current series of studies to provide data that can contribute to the assessment of the relative influences of the structural laws of form, phylogenetic constraints, and adaptive factors during the course of primate and hominid brain evolution.

1.2 The rise and fall of Dr Pangloss

The central pillar of the neo-Darwinian modern synthesis is that of adaptation by means of natural selection (Stebbins, 1966). This concept has served not only as a unifying theme to evolutionary biology but also as a framework within which differential survival may be viewed. Despite the undeniable success of the adaptationist paradigm in making sense of various morphologies, it becomes apparent to the biologist that its 'generous' usage to explain all evolutionary change must be fundamentally flawed; after all, the unwritten rule in biology is that nothing is as simple as we may come to expect.î"o emphasise this point Gould and Lewontin (1979) critiqued the reflex like tendency of evolutionary biologists to explain all morphologies in terms of inferred evolutionary adaptations. Under the influence of the adaptationist program, every organic feature had a well constructed adaptive reason for its occurrence, no matter how convoluted the justification. To Gould and Lewontin (1979), this type of reasoning was equivalent to that of Voltaire's Dr Pangloss, who claimed a noble purpose to any situation, no matter how ridiculous the reasoning:

"*Our noses were made to carry spectacles, so we have spectacles…Legs were clearly intended for breeches and so we wear them*" (Gould & Lewontin, 1979).

What Gould and Lewontin coined the 'Panglossian paradigm' (a euphemism for the adaptationist program), has also dramatically affected existing models of primate brain and body size evolution, in which adaptive explanations have been sought to explain the 'apparent' gradual expansion of the hominid brain. Many of these adaptive explanations have at their core, the basic pitfalls of the adaptationist arguments, which are excessive atomization, decoupling of biological elements or the reliance on trade-off relationships between structures (see Allman, 1999 for examples). As a result of this focus on adaptationist explanations, potentially important aspects of evolution relating to ontogenetic constraints, phylogenetic contingencies and structural laws of form have been marginalized (Gould & Lewontin, 1979; Gans, 1989; Lauder & Liem, 1989). It is imperative that evolutionary biologists heed a note of caution, so that adaptation and natural selection do not simply become 'evolutionary deities' upon which all questioning is explained away.

But what motivated Gould and Lewontin's (1979) searing attack on the Panglossian paradigm? When tracing the historical path up until the publication of this paper it is argued that a major contributing factor may have been the rise of sociobiology in a large part cemented by E.O. Wilson (1975) and also the celebrated popularity of Richard Dawkins' extreme reductionist, yet narrowly centred genetic approach, typified by his book "The selfish Gene" (Pigliucci and Kaplan, 2000). Critics to sociobiology in its old form and its more contemporary disguise as Evolutionary Psychology (e.g. Ramachandran, 1997) have often levelled concern at the less parsimonious and 'just so' approach of this discipline and Lewontin was no exception (Lewontin, 1992). In accordance with the point raised by Lewontin (1992), sociobiologists needlessly argue for the existence of adaptive reasons to explain human biology and behaviour (i.e. they regard the persistence of a trait as being

directly reflective of a hypothesis in favour of adaptation by means of natural selection and use this as prime evidence to argue for it being adaptive).

Subsequently, the writing of Dawkins that highlighted the agents of selection as being individual genes mediated by direct phenotypic responses became integral to the extremely dominant adaptationist notions. As a consequence, when applied to questions pertaining to human behaviour it opened itself to conclusions with contentious socio-political implications. One may argue that one prime example of this is the foil which it provided for the debate concerning the 'mismeasure of brain size' and the impetus for proponents of hierarchical views of intelligence (see Gould 1981 for review Rushton & Ankney, 1996 for an example of this line of argument). As sharply mentioned in jest by Pigliucci and Kaplan (2000), "*The attack on adaptationism…. would have been far less aggressive if the adaptive significance of the variation in the color of snail-shells were the only thing at stake*."

What is apparent from the above interpretation and the plethora of rebuttals from Gould and Lewontin following the "Spandrels" paper is that the authors had distinct foresight for what impact a strong unilateral approach such as that advocated by the sociobiologists would have on the public domain. However they (Gould and Lewontin, 1979) also highlighted an important theoretical point and that was the acknowledgement that alternate hypotheses be considered in order to understand the aetiology of traits and that organisms are not components of optimized parts forming a complete whole. This opened the door for the consideration of other factors in the modelling of traits (e.g. genetic drift; exaptation; indirect selection and the case for multiple adaptive peaks etc.) and an approach in which constraints upon optimization could play a more pivotal role in the evolutionary process. Despite what appeared to be a veritable *coup d'état* on what some would label a 'theoretical despot', Gould and

Lewontin repeatedly reinforced the idea that adaptation by means of natural selection has and continues to be an influential agent in the shaping of organisms. What they merely wished to emphasis was that evidence is necessary to support the notion that a trait is adaptive and a result of natural selection and that this may not be taken as an untestable null hypothesis.

Whether intended or not, what ensued was the championing of an alternate approach to account for the complexity of the evolutionary process (Gould, 2000). Unfortunately, although constraints were increasingly acknowledged as playing a key role in the evolutionary process, researchers were divided concerning the categories of constraints. This point is emphasized by Antonovics and Tienderen's (1991) plea for consensus in the use of terminology encapsulated by the title of their paper: "Ontoecogenophyloconstraints? The chaos of constraint terminology." As highlighted by Antonovics and Tienderen (1991) a survey of the literature revealed an enormous range of adjectives applied to constraints such as: developmental (Wagner, 1988); phylogenetic (Warburton, 1989); cytogeometric (Nanney *et al.,* 1980); morphological (Werdelin, 1987); physiological (Perrin and Rubin, 1989); ecological (Ziehe and Gregorius, 1988; Shine, 1989); pleiotropic (Johnson, 1987); environmental (Sjogren *et al.,* 1988); and mechanical (Carrier, 1987) to mention a few. What has been perhaps more disconcerting has been the early incongruent invocation of constraints as a non-specific label sometimes referring to traits, levels of variation and even processes. To lend clarity and uniformity to this problem, a number of suggestions have been made (e.g. Antonovics and Tienderen, 1991; Leroi, *et al.,* 1994; Burt, 2001). Although all of these are relevant, in this current study the approach outlined by Burt (2001) is used, which proposes a more unambiguous set of terminology to describe evolutionary stasis and constraints. These terms are preferred as they present a more concise set of

definitions that are free from the historical ambiguity associated with the traditional set of terminology. Burt (2001, pg. 515) defines evolutionary constraint as: "An evolutionary pattern in which a character fails to change in an adaptive manner due to preventative factors or mechanisms". If the various data points examined do change in manner that is not consistent with this definition of constraint, then it is possible that this or these changes represent an adaptive evolutionary scenario. In this study the use of variance partitioning methods with consideration of phylogenetic relationships aids in the disentanglement of the various adaptive and non-adaptive mechanisms of evolutionary change.

With this in mind, the following set of chapters designated by separate research projects, aims to re-examine the evolution of brain size and structure in primates, with a specific focus on hominids and the role of non-adaptive mechanisms on the genesis of form. A comprehensive comparative data set documenting brain and body size variation among living and fossil primates, including hominids is provided and the data is used to re-evaluate patterns of hominid brain evolution and to reconsider the role of both adaptive and non-adaptive evolutionary influences in hominid brain evolution. The subsequent chapters are divided as follows:

Chapter two addresses the question of body mass estimation from the cranium using an array of regression procedures. Whilst making up a substantial component of the data necessary for the final assessment of brain: body size allometry in fossil hominids, this chapter also serves to introduce the reader to the existence of strong correlative relationships between body mass and certain cranial variables. This scaling relationship is argued to be indicative of a set of developmental constraints whilst phylogenetic constraints are shown to play a lesser role in the channelling of brain and body dimensions.

In chapter three the relative occupancy of the skull is investigated by means of magnetic resonance imaging and a regression equation is derived from which the brain mass of fossil hominids may be accurately estimated from the available cranial capacities. Endocranial volume is shown to overestimate brain mass by about 20%, and *via* the use of variance partitioning procedures the case is made for the existence of developmental constraints governing the relative occupancy of the cranial vault by the brain.

Brain mass variation in *Homo sapiens* has long been the focus of attention for numerous researchers. In chapter four, the reason for population and inter sex differences in brain mass is investigated using bivariate and multivariate regression models and the argument is made for no significant differences in brain mass between the study groups. Changing scaling relationships during development (i.e. beyond the age of three years) are shown to facilitate the introduction of greater variation in brain mass and body size with age. It is believed that this 'relaxing'/diminishing of the brain: body constraint axis is responsible for the variation displayed in modern humans and is reflective of an initial critical period in development (i.e. prior to 3years of age) when the proportional integrity of the human body is maintained to prevent the influence of deleterious factors. These results indicate that constraints in the brain and body axes can been 'bent' or even 'broken' and that decoupling of these relationships may have been mediated by the developmental process to result in the scaling relationship displayed within our lineage.

Chapter five is concerned with investigating scaling relationships between organisms taken at different taxonomic levels. The case is put forth that at taxonomic levels lower than that of the Order, primate groupings show similar brain: body mass scaling relationships to that observed in certain Orders of mammals. The case is put

forth that there may be different constraints on the evolution of neural systems and that analyses must take phylogenetic effects into account as these may relate to the timing and tempo of change at various organizational levels within the brain.

The final chapter attempts to summate the significant results obtained from the previous chapters and to use this as a starting point from which a final assessment of the role of constraints in brain and body size may be elucidated. A test of this is made with reference to the newly discovered and contentious hominid fossil, *Homo floresiensis*. This chapter ends with a call for pluralism between the relative contributions of constraints and adaptations on the evolution of the hominid brain.

Chapter 2

The use of cranial variables for the estimation of body mass in fossil hominids

2.1 Introduction

Body mass remains a key aspect of species biology and has been shown to be intimately related to the energetic, ecological and physical properties of an organism (Calder, 1984; Damuth and MacFadden, 1990). In addition, body mass provides a measure with which to compare brain mass in organisms with very different body sizes (Jerison, 1973; Bauchot & Stephan, 1961; Manger, 2005, 2006). The correct determination of body mass is an essential precursor to comparisons of relative brain size in fossil primates (Jerison, 1973, 1979; Radinsky, 1977).

Estimating body mass of fragmentary and incomplete fossil remains poses serious challenges. In attempts to overcome these challenges, researchers have used variables such as femur length or cheek tooth area as surrogates for body mass (*e.g.*, Kay, 1975; Gingerich, 1977; Pirie, 1978; Gingerich & Schoeninger, 1979; Martin, 1979; Gould, 1975; Kay & Simons, 1980; Martin, 1980; Dechow, 1983; Jungers, 1985). Estimates of early hominid body mass have largely been based on postcranial material (McHenry, 1974, 1992, 1994; Steudel, 1980; Jungers, 1985; Ruff *et al.,* 1997) as it has been shown that limb bones are more strongly correlated with body mass than cranio-dental elements whose functional associations are not as direct (Aiello & Wood, 1994).

However, commonly used postcranial dimensions such as diaphyseal breadths have proved problematic in comparative settiV:s as they appear to be environmentally sensitive to responses in mechanical loading (Ruff *et al*., 1993). Attempts at overcoming this shortfall, has seen workers investigate the use of dimensions that are less sensitive to environmental perturbations (e.g. femoral head breadth) or are not

dependent on the assumptions of the mechanical association in the support of body mass (e.g. multivariate regressions using stature and bi-iliac breadth, Ruff *et al*., 1997). Whilst multiple regression using postcranial elements has proved an interesting and useful method for estimating body mass, its utility is limited by the availability of necessary skeletal elements to compute the equations. This has seen a continued reliance on the use of simple linear regression procedures using preferred postcranial elements to predict hominid body mass.

In this regard, McHenry's (1992) estimates of body mass for fossil hominids using either axial or appendicular material provide high correlation coefficients which range typically between r = 0.92 to r = 0.99. Further estimates of body mass yielded the following modal weights for male and female hominids of the genus *Australopithecus* and early *Homo*: *A. afarensis*, 45 kg and 29 kg; *A. africanus*, 41 kg and 30 kg; *A. robustus*, 40 kg and 32 kg; *A. boisei*, 49 kg and 34 kg; and *H. habilis*, 52 kg and 32 kg (McHenry, 1992). These estimates are consistent with those published in a later article by McHenry (1994) and are equivalent to estimates calculated by Steudel (1980).

However, while postcranial elements are still believed to provide the most reliable and consistent estimates of body mass, a number of factors combine to reduce the utility of these estimates. These include: a) the paucity of postcranial elements; and b) the uncertainty that exists in most cases when assigning a species designation to postcranial material (e.g. between early *Homo* and *Paranthropus* specimens) [McHenry, 1992b]. Furthermore, although there is a general consensus about limb proportions and locomotor strategies of early hominid species, there is also an acknowledgment of the problems related to the use of these inferences (Grauz, *et al*.,

1988), plus the fact that cranio-dental elements are usually the source of diagnostic features of early hominid taxa (Wood, 1992).

This study explores the utility of cranial fossil evidence for body mass predictions. While cranial variables have been used previously to predict hominid body mass (Kappelman, 1996; Aiello & Wood, 1994), and have yielded high correlation coefficients indicative of the strength of predictability, these studies have not taken into account the potential bias which may be introduced by the non-independence of species points. For instance, the study by Kappelman (1996) was concerned with investigating the predictive capability of orbital area on hominid body mass. This analysis was based on LSR and yielded an r value of 0.987 derived from 18 primate species. Aiello and Wood (1994) under took a similar study to predict hominid body mass from a sample of both Cattarrhini and Platyrrhini primates. Whilst this study also yielded high r values ($r > 0.96$ for best predictor variables) it too as with that of Kappelman (1996) had not taken phylogeny into account and thus the reported r values still contained a portion of variance which the species share in common as a result of phylogenetic autocorrelation.

Phylogenetic correction methods have grown in popularity in recent years as the pitfalls of traditional regression methods, such as the assumption of randomness and independent sampling, have been shown to be invalid for most biological enquiries (Sokal & Rohlf, 1995). In addition experimental models have shown that comparative analyses that do not take phylogeny into account incur higher type 1 error rates, sometimes as high as 44% as opposed to the usually assumed 5% in most analyses (Harvey & Rambaut, 2000). The rationale behind the use of phylogenetic methods is the empirical observation and theoretical prediction that closely related species are more likely to share characteristics in common than are more distantly

related species. This invalidates the use of traditional regression methods such as LSR. By accounting for phylogeny, phylogenetic methods may hope to reveal the 'true' extent of developmental association between structures in the absence of phylogenetic effects. These methods have proved, under various simulation studies, to outperform traditional methods even when their assumptions are violated. In particular, a simulation study by Martins *et al* (2002) has shown that Felsenstein's independent contrasts method gave the best performance when tested using computer simulated evolutionary scenarios, even when weak constraints had been acting throughout phenotypic evolution and that most phylogenetic methods yielded good statistical performance in comparison to traditional procedures, regardless of the details of the evolutionary models (Martins *et al*., 2002).

This study attempts to build on earlier studies by applying the more 'robust' technique of Phylogenetic Independent Contrast Analysis (Felsenstein, 1985) and investigating the strength of correlation between cranial dimensions and body mass and asks the following questions: a) are cranial variables useful for body mass prediction and if so which cranial variables offer greatest utility; b) what are the effects of controlling for phylogeny on the strength of relationships derived between body mass and cranial variables and what does quantifying the partitioned variation explained by each variable reveal about the influence of body mass on these cranial variables; and c) how do body mass estimates for fossil hominids derived from this method compare with those of previous studies using either cranial or postcranial variables.

2.2 Materials and methods

The analysis is based on 259 primates representing 16 species (Table 1). Sample sizes for the hominoid species ranged from two individuals for *Pongo pygmaeus* to 180 individuals for *Homo sapiens*, whilst that for the non-hominoid primates varied between one and 30 individuals depending on availability. Sample sizes for each species are indicated in Table 1. Non-human primates were obtained from the J.C. Middleton-Shaw Collection (School of Anatomical Sciences, University of Witwatersrand, South Africa) and the comparative primate collection from the Transvaal Museum (Pretoria, South Africa), whilst modern human skeletal material was obtained from the Raymond A. Dart Human Skeletal Collection (School of Anatomical Sciences, University of Witwatersrand, South Africa). Average body masses were obtained for each species from the relevant sources (Table 1).

As an initial exploratory step eleven cranial variables that showed high correlations in previous studies (Radinsky, 1982; Aiello & Wood, 1994) were taken using conventional spreading and sliding callipers and were assessed using Lin's Concordance Correlation Coefficient (Lin, 1989). This method was used to assess repeatability and the measurement error for each variable. Not all variables proved useful in terms of their repeatability and only those with Pc values of 0.9 and above were selected for further analysis as this 'cut off' point suggests at least a 90% correlation between measurements taken at different time periods using the same variable, instrument and observer (*i.e*. only a 10% error in repeatability) (Lin, 1989). All variables were tested for normality prior to inclusion in the relevant analyses. From these variables a list of nine measurements were taken on the cranium of each specimen (Table 2) and two additional variables, namely foramen magnum area (Fma) and orbital area (Orba), were derived using the relevant lengths and breadths.

Martin (1990) maintains that this method remains efficient for an analysis of scaling relationships and is the argument used by Aiello and Wood (1994) for not deriving area estimates based on the area of an ellipse. In the current study area estimates based on the area of an ellipse were included, as the authors believe that their inclusion provides a more accurate reflection of the actual areas.

2.2.1 Traditional LSR and RMA regressions

All analyses were carried out on logarithmic (base 10) transformed mean body mass and cranial measurements for each species as has become 'standard' technique in most analyses, whilst the resulting regression equations for LSR and Reduced Major Axis (RMA) were computed using PAST (Version.1.18; PAST © Hammer & Harper, 1999-2005). As with Aiello and Wood (1994), two separate sets of z)alyses were undertaken using conventional regression techniques (*i.e*. LSR and RMA), the one based on a combined primate species sample (N=16 or N=12 where indicated) and the other on a smaller set of hominoid data (N=5). The purpose of these sets of analyses was to assess the possible differences in scaling in broader and narrower taxonomic groups. In accordance with Smith (1993) the Smearing and Ratio estimators were calculated for the LSR equations to allow for compensation of any bias introduced due to logarithmic transformation.

2.2.2 Control for phylogenetic effects

Because previous studies have calculated correlation coefficients for predicting body mass using individual species as data points and had not taken into account the potential for bias introduced by the non-independence of these species, the technique of independent contrast analysis (Felsenstein, 1985) was applied to the

dataset as implemented using COMPARE (Version. 2.0 – CONTRAST., © University of Oregon and E.P. Martins, 1997). This method compares sets of differences between different nodes in a known phylogeny and computes values known as 'contrasts' which represent the difference between a pair of species or nodes in a phylogeny and not individual species. Each contrast in the phylogeny is then 'weighted' by a factor proportional to the amount of evolutionary time estimated to separate nodes. This process adjusts for the different variances expected as the contrasts potentially represent accumulative differences over differing amounts of time. The assumptions of the 'comparative method' have been explored since the seminal publications of Cheverud *et al*., (1985) and Felsenstein (1985). The arguments in favour of its usage include the following points: a) the high type 1 errors incurred by traditional models that do no take phylogeny into account (Harvey & Rambaut, 2000); b) the robustness of the Brownian motion model even when violated (Garland *et al*., 1992; Garland & Ives, 2000); and c) traditional methods have reduced statistical power to detect associations and relationships among groups in data sets (Harvey & Rambaut, 1998; Nunn & Barton, 2001). Additionally, it is important to remember that even traditional comparative methods assume an underlying 'star' phylogeny (where each data point radiates from a central origin) and that replacing this with a 'hierarchical' model matches the way in which species are related and are compared.

The specific phylogenetic hypothesis used in this study is shown in Figure 1. It is based on a composite phylogeny for the Order Primates provided by Purvis (1995). The branch lengths for this figure were obtained from the scaled scanned image published in Purvis (1995) and using the program TreeThief version 1.0. TreeThief is an application for inputting phylogenetic trees, with branch lengths, into the computer for use in other programs like PAUP and COMPARE as used in this study. The input

method is essentially to draw the tree out by clicking on each node in turn on a template of the scanned image, and then to use the provided scale bar to calibrate the tree. In this method the branch lengths remain untransformed.Another consideration in favour of the use of independent contrasts especially when comparing correlations between structures within an organism is that associations with body mass are likely to be high whether or not they have any direct causal connection with body mass. This is true because any cross species variability of the subcomponents of mass is necessarily constrained by the variability of mass itself and thus these variables are not truly independent of one another which subsequently invalidate the use of the traditional statistical tests applied to correlations. In this current study correlation coefficients using phylogenetic correction were computed for comparison as well as the estimated regression slopes of the contrasts for the 12 species sample.

In addition, we analysed the body masses for the species used in this study by comparing their tip data (original data) and phylogenetic topology using a test for serial independence (von Neumann *et al.*, 1941). The test for serial independence (TFSI) provides a measure of the degree of non-randomness in a sequence of continuous characters. When observations are ordered sequentially along the tips of a phylogeny, the test for serial independence can be used to test for either positive or negative phylogenetic autocorrelation (Abouheif, 1999). Positive phylogenetic autocorrelation presents as similarities in adjacent observations due to phylogenetic descent whereas negative phylogenetic autocorrelation is a non-random pattern attributed to convergence (Abouheif, 1999). The test for serial independence was applied to both the original data (tip data) and to the absolute values of the standardized independent contrasts (Felsenstein, 1985) to assess the degree of non-randomness when using either traditional regression procedures or independent

contrasts. Subsequently, frequency distribution plots of the observed mean C-statistic for each technique relative to the distribution of randomized mean C-statistics were drawn to provide a visual perspective of the distribution of randomness in the sample and to indicate whether body mass is more suited to investigation using independent contrast analysis or traditional regression techniques.

2.2.3 Quantifying the partitioned variation

Comparative analyses that take phylogeny into account have become widely used in biology and usually consist of comparing either two or more traits across species, although it works equally as well when comparing a trait and an environmental variable (Harvey & Pagel, 1991). Several methods have been proposed for this type of analysis (e.g. Felsenstein, 1985; Grafen, 1989; Diniz-Filho *et al*., 1998). A controversy resulted from the discussion by Westoby *et al.,* (1995) who argued that comparative methods partition the explained variation of environmental/ecological data by "allocating the maximum possible variation in a trait to phylogeny, considering only the residual as potentially attributable to ecology" (Westoby *et al*., 1995). These authors noted that the phylogenetic portion of the total variance of a variable is not exclusive and may contain a phylogenetic component related to ecology, a term called "phylogenetic niche conservatism" by Harvey and Pagel (1991). This term also encompasses the shared attributes that related species display and have acquired due to the tendency to occupy similar niches during evolutionary history.

Westoby *et al.* (1995) proposed to partition the variance of the data into three portions *a*, *b* and *c* - where *a* is a part strictly due to environment, *b* is a part due to the common influence of environment and phylogeny, and *c* is a part strictly due to

phylogeny. Desdevises *et al*. (2003) proposed a method by which to calculate the partitioning of variation in a phylogenetic setting and is the technique used in this study as it not only partitions the variation into three components but also adds a further fourth component known as fraction *d* which is the unexplained part of the variation. The decomposition of the phylogenetic variation for the cranial variables under study was undertaken in accordance with the procedural steps outlined by Desdevises *et al*. (2003), where step 1 was the determination of the coefficient of determination from the computation of a regression of Y on X, thus giving fraction a + b; step 2 was the determination of fraction b+c by computing a multiple regression of Y on all principal coordinates; step 3 was the determination of fraction a+b+c by computing a multiple regression of Y on both X and the principal coordinates; and step 4 was the calculation of the individual values for a, b, c and d by subtraction from previous results, *e.g.* a = R^2 (step 3) - R^2 (step 2); c = R^2 (step 3) - R^2 (step 1). Principle coordinate analysis is an ordination method similar to that of principle component analysis and is aimed at finding the eigenvectors and eigenvalues of a matrix containing the distances between data points. Eigenvalues provide a measure of the variance accounted for by the corresponding eigenvectors/coordinates. All multiple regressions and eigenvalues were calculated using SPSS version 11.0. As suggested by Desdevises *et al*. (2003) the broken-stick model (Barton and David, 1956; Frontier, 1976) was abandoned for the use of only principal coordinates that were significantly contributing to the modelling of the phylogenetic distance matrix. Negative eigenvalues were sign corrected in accordance with the suggestions of Desdevises *et al*. (2003). In this present study, fraction a+b has been denoted as representing the amount of variation explained by a specific cranial variable, b+c the amount of

variation explained by phylogeny, whilst the subcomponents a and c represent the variation explained solely by a cranial variable and phylogeny respectively.

2.2.4 Assessment of cranial variables

The suitability of the eleven cranial variables as body mass predictors was primarily assessed on the basis of the resulting r values - the levels of transformation bias where useful equations should have relatively low and consistent bias estimates, approximately less than 10% (Smith, 1993), and the percentage prediction error (Smith, 1980, 1984) which should be relatively small (Aiello & Wood, 1994). The magnitude of the percentage prediction error was assessed by calculating the mean percentage prediction error (MPE) in accordance with the technique outlined by Aiello and Wood (1994).

2.2.5 Predicting Hominid body mass

On the basis of the above analyses, the most reliable body mass estimators in both the hominoid and primate models were used (where available) to give predictions of body mass for selected hominid species. Seven hominid specimens were included on the basis of availability and the potential to provide an insight into body mass for various species. These specimens are: STS 5, OH 5, MLD 37/38, STW 505, SK 48, La Ferrasie 1 and La Chappelle 1 which represent *Australopithecus africanus*, *Paranthropus boisei, Paranthropus robustus* and *Homo neanderthalensis*. High resolution plaster casts were used to measure the required intact cranial dimensions for the La Ferrasie 1, La Chappelle 1 and the OH5 specimens, whereas 3 Dimensional digital casts of the South African material were reconstructed and measured from Computer Topographical (CT) scans provided by the University of

Vienna. The CT scanned images were reconstructed and analysed using 3D Doctor (Version 4.0, © ABLE Software Corp. 1998-2006) which provides an array of advanced 3D image processing, visualization and rendering software for scientific applications. Measurements obtained using digital images were compared with those taken from plaster casts using a digital calliper in a sub-sample of 5 individuals. Comparable measurements proved to be only marginally different, with an error range of between 3% and 5% and argued in favour of the use of either technique as they were not significantly different from one another. Where needed, missing cranial fragments were reconstructed by 'mirroring' opposing sides, as is the case in STW 505. Figure 7 provides a view of the digitally reconstructed specimens from which measurements were subsequently taken.

Cranial measurements taken from hominid sources were then substituted into the derived LSR equations (Table 3 and Table 4) for the best predictor variables. Best predictor variables were selected on the basis of a combination of high predictability (high r values in both traditional regression techniques and independent contrasts analysis), low levels of transformation bias (useful equations displayed relatively low and consistent bias estimates, approximately less than 10%) and small mean percentage prediction errors. An estimate of species body mass was then computed by calculating the average body mass estimate derived from using each individual variable (*e.g*. as seen in Table 9, the estimated body mass using each separate best predictor variable on STS 5, in the whole primate sample is: 28.64 kg using orbital length; 25.54 kg using orbital area and 35.25 kg using upper facial breadth. Taking the average of these three body mass estimates provides an overall body mass estimate for *Australopithecus africanus* of 29.81 kg, as displayed in Table 10). In cases where certain best predictor variables are missing, the average species body

mass is calculated using the available body mass estimates calculated from the remaining variables (e.g. for SK48 average species body mass in the whole primate sample is calculated using orbital length and orbital area).

2.3 Results

2.3.1 Traditional LSR and RMA regressions

In the analysis of the whole primate sample (Table 3), the most reliable predictor variables as determined on the basis of the criteria stated in the methods section are upper facial breadth (Ufb), orbital area (Orba; OrbaElp), orbital height (Orbl), and bizygomatic breadth (Bizy). These variables have smearing estimates (SE) of 1.07 or lower (*i.e.*, 7% or lower underestimation), correlation coefficients of 0.96 or higher ($r^2 > 0.92$), and mean percentage prediction errors of 37% or less (See Figures 2a and 2b).

The results of the hominoid-based analysis are displayed in Table 4. According to this analysis, the best predictor variables are foramen magnum area (Fma & FmaElp); biorbital breadth (bpor); orbital height (Orbl); orbital area (Orba; OrbaElp) and biporionic breadth (Biorp). These variables display correlation coefficients of 0.98 or above ($r^2 > 0.96$); SE of less than 1.02 (*i.e.*, 2% or lower underestimation) and MPE of 10% to 16%.

2.3.2 Control for phylogenetic effects

The resultant correlation coefficients using independent contrast analysis are displayed in Table 5. Orbital height, orbital area (calculated as an ellipse) and upper facial breadth display on average the strongest correlations with body mass (*i.e.*, 0.99-0.91 for the whole primate sample and 0.96-0.94 for the hominoid sample). The

maximum length of the skull (Skul) displays the lowest correlation coefficient of between 0.53 and 0.77 for the whole primate sample and hominoid sample respectively. Estimated regression slopes using RMA and independent contrast analysis (in the 12 species sample) indicate that there exists no statistically significant difference ($P > 0.05$) between RMA and IC regression slopes for the variables understudy and thus common slopes were computed (Table 5).

Results from the test for serial independence are displayed in Figure 4a and 4b. Results indicate that the original body mass data as used in traditional regression procedures were significantly phylogenetically autocorrelated (Fig. 4a; $P = 0.01$; $\alpha = 0.05$) but when applying the technique of independent contrast, the TFSI detected no significant phylogenetic autocorrelation among the contrasts (Fig. 4b; $P = 0.51$; $\alpha = 0.05$).

2.3.3 Quantifying the partitioned variation

Using the partitioning method proposed by Desdevises *et al*. (2003), we attempted to quantify what proportion of body mass is correlated with a specific cranial variable, with phylogeny alone and jointly with a cranial variable and phylogeny. Using the phylogeny in Figure 1, a minimum of two and a maximum of three PCs were significant and retained in the 12 and 16 species models respectively. PC1 and PC2 explained 73% and 14% of the total variance and cumulatively explained 87% of the variation in the 12 species primate sample (See Tables 6). PC1, PC2 and PC3 were extracted to represent the matrix when using 16 species and explained 74%, 8% and 6% of the variation respectively, with a cumulative sum of 89% of the total variation being explained for the 16 species primate sample (See Table 7). Table 8 gives a breakdown of the components of body mass explained by

the various cranial variables. The range for the amount of variation in body mass explained by a cranial variable alone lies between 40% and 49%, with upper facial breadth (Ufb) explaining the highest percentage variation and maximum length of the skull (Skul) the lowest. Component *b* which represents the portion of variation explained by a combination of the cranial variable and phylogeny ranged between 43% and 46%, with orbital height and orbital area displaying the two highest values, even though a very narrow range of variation is displayed for *b*. The unexplained component of body mass variance included in the model ranges between 3% to 12% with the greatest uncertainty in body mass variation belonging to maximum length of the skull whilst orbital breadth and upper facial breadth showed the lowest values for the component of unexplained variance. Figures 5 and 6 provide a visual breakdown of the components as discussed above.

2.3.4 Predicting Hominid body mass

Table 9 gives the predicted masses derived using LSR from the whole primate and hominoid based samples and derived from the best predictor variables. LSR was used as no log transformation bias estimators exist for RMA. When using the criteria described earlier for selection of best predictor variables, the following variables were selected: upper facial breadth; orbital area and orbital length in the Whole primate sample and orbital area; orbital length; foramen magnum area; biporionic breadth and biorbital breadth in the Hominoid sample.

In accordance with these estimates, average body mass estimates for the hominid species are as follows: a body mass of approximately 30 kg and 47 kg for *Australopithecus africanus* based on STS 5 and STW 505 respectively; a body mass of 52 Kg and 48 kg for *Paranthropus robustus* and *Paranthropus boisei* based on SK

48 and OH 5 respectively; and a mass of approximately 75 kg for *Homo neanderthalensis* based on La Ferrasie 1and La Chapelle 1. In the absence of necessary best predictor variables for MLD 37/38, a hominoid model was used to derive an estimate of body mass for *Australopithecus africanus* which yielded a species body mass estimate of approximately 41 kg based on MLD37/38.

TABLE 1 List of specimens used in study and the average female body weights as obtained from relevantã]ources

Species	Average Body Weights (g)	N	Source
Petterus fulvus *	1200	1	Jerison, 1973
Varecia variegata	3850	1	Nowak, 1999
Daubentonia madagascariensis	2500	1	Nowak, 1999
Propithecus verrauxi *	3200	1	Jerison, 1973
Indri indri	6250	1	Nowak, 1999
Mircocebus murinus *	54	1	Jerison, 1973
Loris tardigradus *	195	1	Jerison, 1973
Callithrix jacchus	290	1	Aiello & Wood, 1994
Alouatta seniculus	6400	1	Aiello & Wood, 1994
Papio ursinus	27500	31	Nowak, 1999
Cercopithecus aethiops	5750	30	Nowak, 1999
Hylobates lar	5300	2	Aiello & Wood, 1994
Pongo pygmaeus	37000	2	Aiello & Wood, 1994
Pan troglodytes	31100	2	Aiello & Wood, 1994
Homo sapiens	49610.37	180	RDCS
Gorilla gorilla	93000	3	Aiello & Wood, 1994

(RDCS) Average body mass calculated from autopsy records from the Raymond Dart Collection of Skeletons; N = sample size.
Species marked with asterix (*) were included in an analysis of the correlation between body weight
and foramen magnum area (Fma) using 16 species.

TABLE 2 List of variables measured and the definitions (Singh & Bhasin, 1968).

Variable Name	Variable Abbreviation	Variable Description
Foramen magnum length	Fml	Maximum distance between basion and opisthion
Foramen magnum breadth	Fmb	Maximum distance in the coronal plane between the inner margins of the foramen magnum
Foramen magnum area	Fma	Product of foramen magnum length and breadth
Foramen magnum area (calculated as ellipse)	FmaElp	Product of foramen magnum length and breadth
Bizygomatic breadth	Bizy	Straight distance between two zygia *i.e.*, the most laterally placed points on the zygomatic bone
Max. Cranial length	Skul	Straight distance between glabella and opisthocranion
Upper facial breadth	Ufb	Straight distance between two frontomalare temporalia
Biorbital breadth	Bpor	Straight distance between two ectoconchion
Orbital height (length)	Orbl	Straight distance between upper and lower margins of the orbital cavity, taken at a right angle to orbital breadth
Orbital breadth	OrbB	Straight distance between maxillofrontale and ectoconchion
Orbital area	Orba	Product of orbital breadth and orbital length
Oribtal area (calculated as ellipse)	OrbaElp	Product of orbital breadth and orbital length
Biporionic breadth	Biorp	Straight distance from porion to porion

TABLE 3 Regression statistics to predict body weight from cranial variables (Whole primate sample).

	LSR									RMA		P Values
Variable	r	n	slope	intercept	error slope	error int.	SE	RE	MPE	slope	intercept	P (uncorr.)
Fml	0.94	12	2.63	0.67	0.30	0.38	1.13	1.05	47.99	2.79	0.46	$4.62*10^{-6}$
Fmb	0.94	12	3.30	-0.01	0.36	0.44	1.13	1.02	44.40	3.50	-0.25	$3.81*10^{-6}$
Fma	0.96	16	1.70	-0.25	0.13	0.30	1.17	0.90	51.09	1.77	-0.40	$3.56*10^{-6}$
FmaElp	0.95	12	2.89	-1.91	0.31	0.64	1.13	1.03	46.12	3.06	-2.25	$3.36*10^{-6}$
Bizy	0.97	12	2.93	-1.68	0.25	0.48	1.08	1.01	37.70	3.04	-1.88	$3.39*10^{-7}$
Skul	0.92	12	2.83	-1.38	0.38	0.73	1.19	0.99	53.78	3.08	-1.85	$2.43*10^{-5}$
Ufb	0.98	12	3.25	-1.99	0.22	0.41	1.05	1.03	27.28	3.32	-2.13	$4.00*10^{-8}$
Bpor	0.93	12	2.47	-0.47	0.31	0.56	1.17	1.06	51.88	2.65	-0.81	$1.23*10^{-5}$
Orbl	0.98	12	3.86	-1.62	0.25	0.36	1.06	0.96	29.58	3.77	-1.48	$7.34*10^{-8}$
OrbB	0.95	12	3.71	-1.18	0.37	0.52	1.11	1.07	39.71	3.89	-1.44	$1.56*10^{-6}$
Orba	0.97	12	1.89	-1.41	0.15	0.42	1.07	1.03	31.70	1.95	-1.57	$2.77*10^{-7}$
OrbaElp	0.97	12	3.79	-4.44	0.28	0.62	1.06	1.03	29.95	3.89	-4.66	$8.63*10^{-8}$
Biorp	0.96	12	3.57	-2.47	0.33	0.60	1.09	1.07	31.40	3.74	-2.74	$7.92*10^{-7}$

SE = Smearing estimate, RE = Ration estimate, MPE = Mean percentage error

TABLE 4 Regression statistics to predict body weight from cranial variables

(Hominoid sample)

	LSR									RMA		P Values
Variable	r	n	slope	intercept	error slope	error int.	SE	RE	MPE	slope	intercept	P (uncorr.)
Fml	0.98	5	3.86	-1.24	0.46	0.69	1.02	1.04	15.98	3.94	-1.37	0.004
Fmb	0.97	5	3.77	-0.73	0.52	0.72	1.02	1.03	19.40	3.88	-0.87	0.005
Fma	0.98	5	1.93	-1.03	0.22	0.64	1.02	1.04	14.13	1.96	-1.14	0.003
FmaElp	0.98	5	3.82	-4.06	0.39	0.88	1.01	1.03	12.80	3.88	-4.19	0.002
Bizy	0.92	5	3.08	-2.04	0.75	1.58	1.07	1.05	34.32	3.34	-2.59	0.026
Skul	0.76	5	2.75	-1.27	1.37	2.88	1.23	1.09	66.25	3.64	-3.13	0.14
Ufb	0.98	5	3.91	-3.38	0.51	1.02	1.02	1.02	16.77	1.01	-3.57	0.004
Bpor	0.98	5	3.81	-3.29	0.41	0.84	1.02	1.03	16.19	3.88	-3.43	0.002
Orbl	0.99	5	4.45	-2.64	0.30	0.49	1.01	0.99	10.32	4.48	-2.69	0.001
OrbB	0.93	5	3.78	-1.31	0.89	1.37	1.07	1.04	30.20	4.09	-1.77	0.024
Orba	0.98	5	2.16	-2.27	0.23	0.73	1.02	1.01	12.89	2.19	-2.39	0.003
OrbaElp	0.99	5	4.34	-5.79	0.40	0.95	1.01	1.01	11.07	4.39	-5.92	0.002
Biorp	0.98	5	4.82	-4.92	0.52	1.02	1.02	0.96	15.07	4.91	-5.09	0.003

SE = Smearing estimate, RE = Ration estimate, MPE = Mean percentage error

TABLE 5 Comparison of correlation coefficients and slopes obtained from independent contrasts analysis (IC) and traditional regression techniques (TRD) for the Hominoid and Whole primate sample.

	Whole sample				Hominoid sample			
	TRD		**IC**		**TRD**		**IC**	
Variable	**r**	**RMA slope**	**r**	**slope**	**r**	**RMA slope**	**r**	**slope**
Fml	0.94	2.79	0.88	2.91	0.98	3.94	0.95	3.79
Fmb	0.94	3.50	0.88	3.45	0.97	3.88	0.95	3.98
Fma	0.96	1.77	0.87	1.60	0.98	1.96	0.96	1.96
FmaElp	0.95	3.06	0.89	3.15	0.98	3.88	0.96	3.87
Bizy	0.97	3.04	0.92	2.96	0.92	3.34	0.84	2.72
Skul	0.92	3.08	0.77	2.79	0.76	3.64	0.53	1.79
Ufb	0.98	3.32	0.94	3.44	0.98	1.01	0.91	3.73
Bpor	0.93	2.65	0.89	2.96	0.98	3.88	0.96	3.87
Orbl	0.98	3.77	0.96	4.10	0.99	4.48	0.99	4.47
OrbB	0.95	3.89	0.90	3.54	0.93	4.09	0.89	3.46
Orba	0.97	1.95	0.95	0.95	0.98	2.19	0.96	2.08
OrbaElp	0.97	3.89	0.95	0.01	0.99	4.39	0.97	4.23
Biorp	0.96	3.74	0.94	3.71	0.98	4.91	0.97	4.50

TABLE 6 Total variance explained by PC analysis of the patristic distance matrix obtained from the 12 species phylogeny displayed in Figure 1. Only the first two PCs were selected as they cumulatively explained 87 % of the variance.

Total Variance Explained

	Initial Eigenvalues			Extraction Sums of Squared Loadings			Rotation Sums of Squared Loadings		
Component	Total	% of Variance	Cumulative %	Total	% of Variance	Cumulative %	Total	% of Variance	Cumulative %
1	8.775	73.124	73.124	8.775	73.124	73.124	7.301	60.845	60.845
2	1.686	14.050	87.174	1.686	14.050	87.174	3.160	26.330	87.174
3	.675	5.627	92.801						
4	.401	3.339	96.140						
5	.127	1.055	97.195						
6	.103	.861	98.056						
7	8.366E-02	.697	98.753						
8	6.392E-02	.533	99.286						
9	5.950E-02	.496	99.782						
10	1.648E-02	.137	99.919						
11	9.726E-03	8.105E-02	100.000						
12	-3.25E-16	-2.712E-15	100.000						

Extraction Method: Principal Component Analysis.

TABLE 7 Total variance explained by PC analysis of the patristic distance matrix obtained from the 16 species phylogeny displayed in Figure 1. Only the first three PCs were selected as they cumulatively explained 89 % of the variance.

Total Variance Explained

	Initial Eigenvalues			Extraction Sums of Squared Loadings			Rotation Sums of Squared Loadings		
Component	Total	% of Variance	Cumulative %	Total	% of Variance	Cumulative %	Total	% of Variance	Cumulative %
1	11.848	74.048	74.048	11.848	74.048	74.048	6.590	41.190	41.190
2	1.385	8.659	82.707	1.385	8.659	82.707	5.326	33.285	74.475
3	1.007	6.294	89.000	1.007	6.294	89.000	2.324	14.525	89.000
4	.579	3.618	92.618						
5	.452	2.822	95.441						
6	.295	1.842	97.283						
7	.112	.702	97.984						
8	7.922E-02	.495	98.480						
9	6.710E-02	.419	98.899						
10	4.908E-02	.307	99.206						
11	4.105E-02	.257	99.462						
12	3.706E-02	.232	99.694						
13	3.269E-02	.204	99.898						
14	1.050E-02	6.560E-02	99.964						
15	5.798E-03	3.624E-02	100.000						
16	1.273E-16	7.957E-16	100.000						

Extraction Method: Principal Component Analysis.

TABLE 8 Decomposition matrix of the percentage body weight variance explained by each cranial variable

		% Explained by cranial variable			%Unexplained
			% Explained by phylogeny		
	Cranial variable	a	b	c	d
Body weight					
	Biorp	46.70	45.38	1.72	6.20
	Bizy	49.00	44.31	2.79	3.90
	Blop	41.40	44.97	2.13	11.50
	FmaElp	43.50	45.96	1.14	9.40
	Fmb	43.50	45.69	1.41	9.40
	Fml	42.80	45.97	1.13	10.10
	Orba	48.10	46.17	0.93	4.80
	OrbaElp	48.60	46.30	0.80	4.30
	OrbB	45.30	45.64	1.46	7.60
	Orbl	48.80	46.27	0.83	4.10
	Skul	40.90	43.50	3.60	12.00
	Ufb	49.70	45.93	1.17	3.20
	Fma16sp	32.10	60.50	0.90	6.50

TABLE 9 Body weight estimates for selected hominids

		Hominoid sample	Whole primate sample
Specimen	**Variable**	**Body weight (kg)**	**Body weight (kg)**
STS 5	Fma	26.19	-
	Blop	28.50	-
	Orbl	21.74	28.64
	Orba	20.95	25.54
	Biorp	28.51	-
	Ufb	-	35.25
STW 505	Fma	-	-
	Blop	-	-
	Orbl	38.22	46.75
	Orba	35.36	-
	Biorp	-	-
	Ufb	-	-
MLD 37/38	Fma	36.57	-
	Blop	-	-
	Orbl	-	-
	Orba	-	-
	Biorp	47.15	-
	Ufb	-	-
SK 48	Fma	15.14	-
	Blop	24.05	-
	Orbl	33.77	41.98
	Orba	44.07	62.62
	Biorp	-	-
	Ufb	-	-
OH 5	Fma	26.43	-
	Blop	68.02	-
	Orbl	36.89	45.34
	Orba	38.50	43.58
	Biorp	58.41	55.38
	Ufb	-	-
La Ferrasie 1	Fma	114.97	-
	Blop	76.50	-
	Orbl	59.25	68.41
	Orba	46.38	51.32
	Biorp	78.16	-
	Ufb	-	63.42
La Chapelle 1	Fma	97.01	-
	Blop	63.18	-
	Orbl	69.19	78.27
	Orba	54.71	59.34
	Biorp	93.61	-
	Ufb	-	71.72

TABLE 10 Average body mass estimates using least square regression for selected hominids

Specimen	Average Body weight (kg) using LSR
Whole Primate Model	
STS 5	29.81
STW 505	46.75
SK 48	52.3
OH 5	48.10
Hominoid Model	
La Chapelle 1	75.54
La Ferrasie 1	75.05
MLD 37/38	41.86

TABLE 11 Comparative body weight estimates (kg) for *A. africanus*, *P. boisei* and *P. robustus*

Author	Anatomical Region	Variables	Regression type	*A. africanus*	*P. boisei*	*P. robustus*
McHenry,1988	Postcranial	Femur shaft	Inter-hominoid/intra-human	45.50	46.10	47.70
McHenry,1991	Postcranial	Hip Joint size	Human	-	-	35.30
		Hip Joint size	Ape	-	-	43.70
McHenry,1992	Postcranial	Hind Limb Joint size	Intra-human	35.50	41.30	-
		Hind Limb Joint size	Inter-hominoid	44.80	59.00	-
Aiello & Wood, 1994	Cranial	orba	Hominoid	33.35	53.37	-
		horb	Hominoid	26.45	41.70	-
		bpor	Hominoid	36.95	92.40	-
		orba	Simian	34.00	50.70	-
		horb	Simian	28.45	43.70	-
		bpor	Simian	36.50	81.53	-
Current Study	Cranial	Fma	Hominoid	31.38	26.43	15.14
		Blop	Hominoid	28.50	68.02	24.05
		Orbl	Hominoid	29.98	36.89	33.77
		Orba	Hominoid	28.16	38.50	44.07
		Biorp	Hominoid	37.83	58.41	-
		Orbl	Whole primate (simian)	37.70	45.34	41.98
		Orba	Whole primate (simian)	25.54	43.58	62.62
		Biorp	Whole primate (simian)	-	55.38	-
		Ufb	Whole primate (simian)	35.25		-

Figure 1 Phylogeny used for determining independent contrasts. Species marked with an asterix (*) were omitted from analyses conducted on the 12 species sample. Diagram modified from Purvis (1995). MaBp – Million years before present

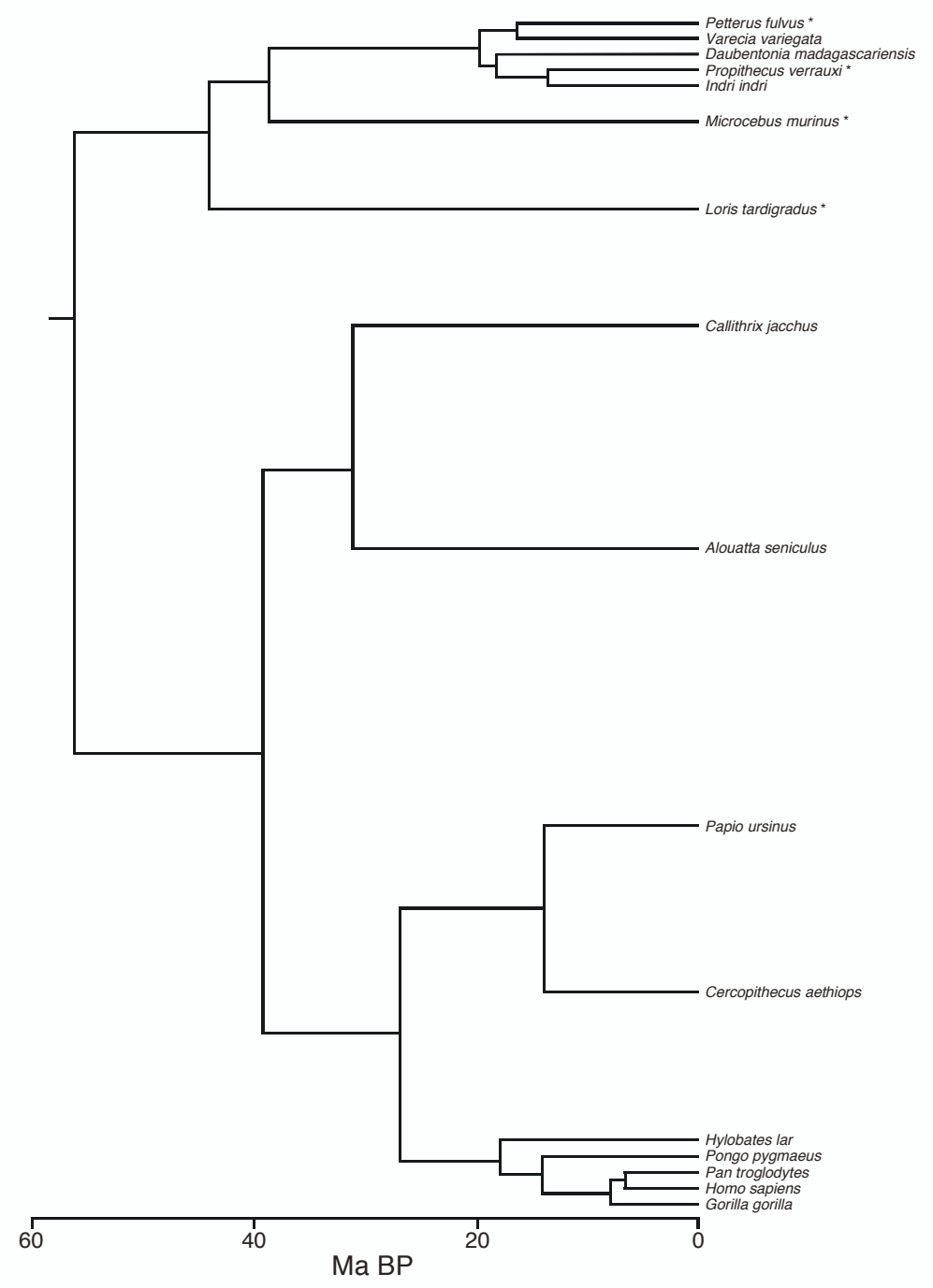

Petterus fulvus *
Varecia variegata
Daubentonia madagascariensis
Propithecus verrauxi *
Indri indri
Microcebus murinus *
Loris tardigradus *
Callithrix jacchus
Alouatta seniculus
Papio ursinus
Cercopithecus aethiops
Hylobates lar
Pongo pygmaeus
Pan troglodytes
Homo sapiens
Gorilla gorilla
60
40
20
0
Ma BP

Figure 1

Figure 2 **A**. Plot of the mean percentage prediction error (MPE%) for the cranial variables, based on whole primate sample. **B**. Plot of the Smearing Estimate expressed as a percentage for the cranial variables, based on the whole primate sample. Biorp – Biporionic breadth; Bizy – Bizygomatic breadth; Bpor – Biorbital breadth; Fma – Foramen magnum area; FmaElp – Foramen magnum area calculated as an ellipse; Fmb – Foramen magnum breadth; Fml – Foramen magnum length; Orba – Orbital area; OrbaElp – Orbital area calculated as an ellipse; OrbB – Orbital breadth; Orbl – Orbital height; SE% – Smearing estimate expressed as a percentage; Skul – Maximum cranial length; Ufb – Upper facial breadth.

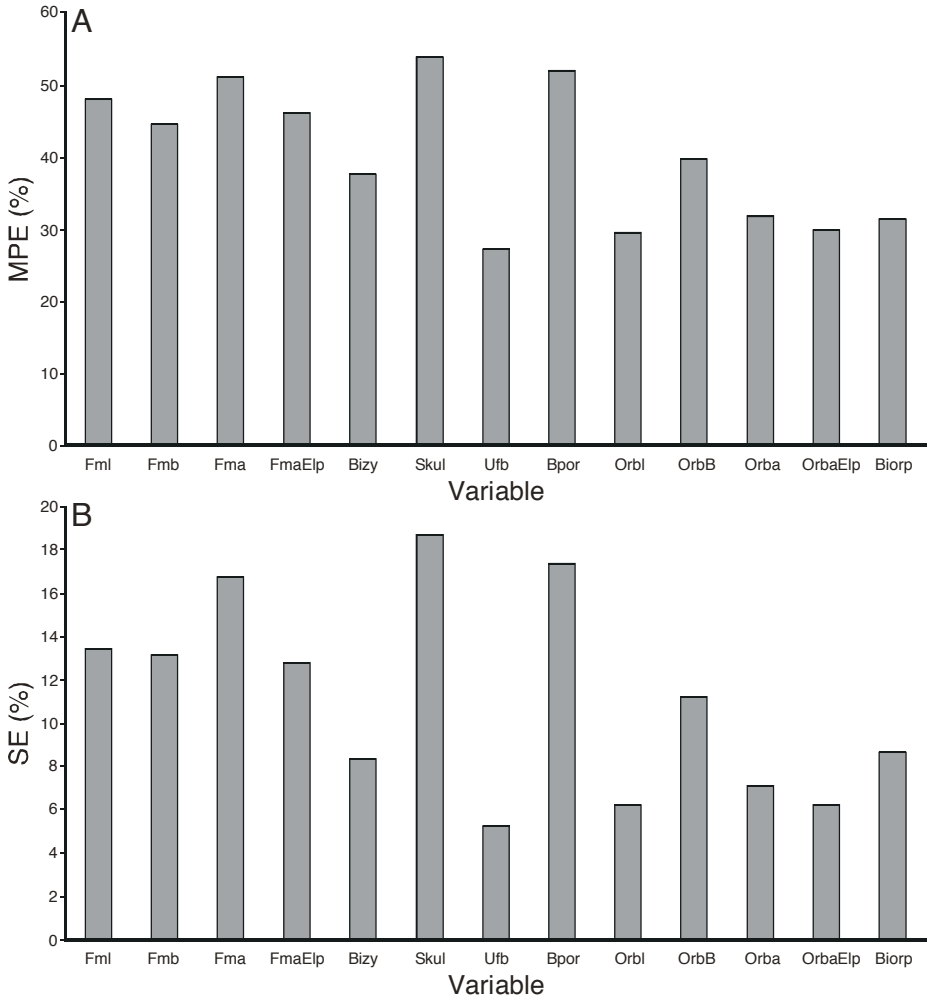

A
MPE (%)
60
50
40
30
20
10
0
Fml
Fmb
Fma
FmaElp
Bizy
Skul
Ufb
Bpor
Orbl
OrbB
Orba
OrbaElp
Biorp
Variable
B
SE (%)
20
18
16
14
12
10
8
6
4
2
0
Fml
Fmb
Fma
FmaElp
Bizy
Skul
Ufb
Bpor
Orbl
OrbB
Orba
OrbaElp
Biorp
Variable

Figure 3 **A**. Plot of the mean percentage prediction error (MPE%) for the cranial variables, based on hominoid sample. **B**. Plot of the Smearing Estimate expressed as a percentage for the cranial variables, based on the Hominoid sample. Abbreviations as in Figure 2.

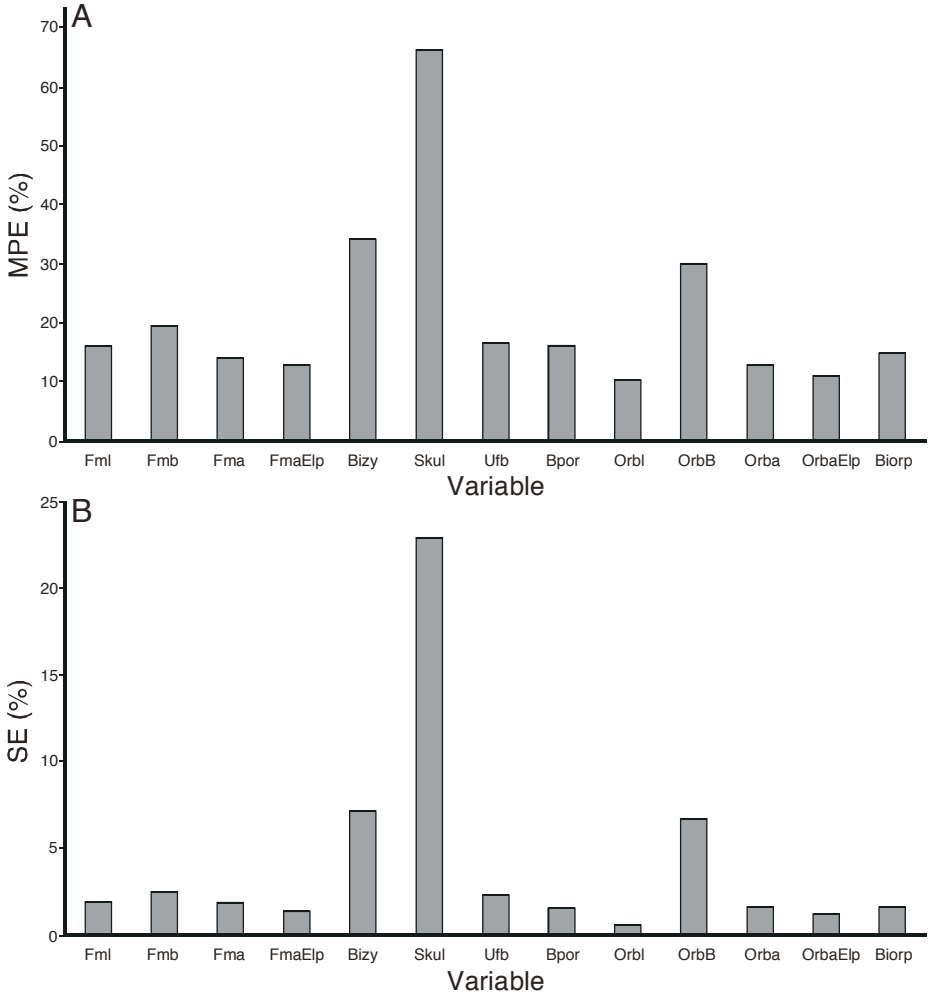

A
MPE (%)
70
60
50
40
30
20
10
0
Fml
Fmb
Fma
FmaElp
Bizy
Skul
Ufb
Bpor
Orbl
OrbB
Orba
OrbaElp
Biorp
Variable
B
SE (%)
25
20
15
10
5
0
Fml
Fmb
Fma
FmaElp
Bizy
Skul
Ufb
Bpor
Orbl
OrbB
Orba
OrbaElp
Biorp
Variable

Figure 4 **A**. The test for serial independence applied to the data set of body masses for the 12 primate species. The arrows indicate the position of the observed mean C-statistic relative to the sampling distribution of randomized mean C-statistics. The frequency distribution in (a) represents the mean C-statistics calculated from the body mass data along the tips of the phylogeny (original logged data used in traditional regression procedures). **B**. The test for serial independence applied to the data set of body masses for the 12 primate species. The arrows indicate the position of the observed mean C-statistic relative to the sampling distribution of randomized mean C-statistics. The frequency distribution in (b) represents the mean C-statistics calculated from independent contrasts.

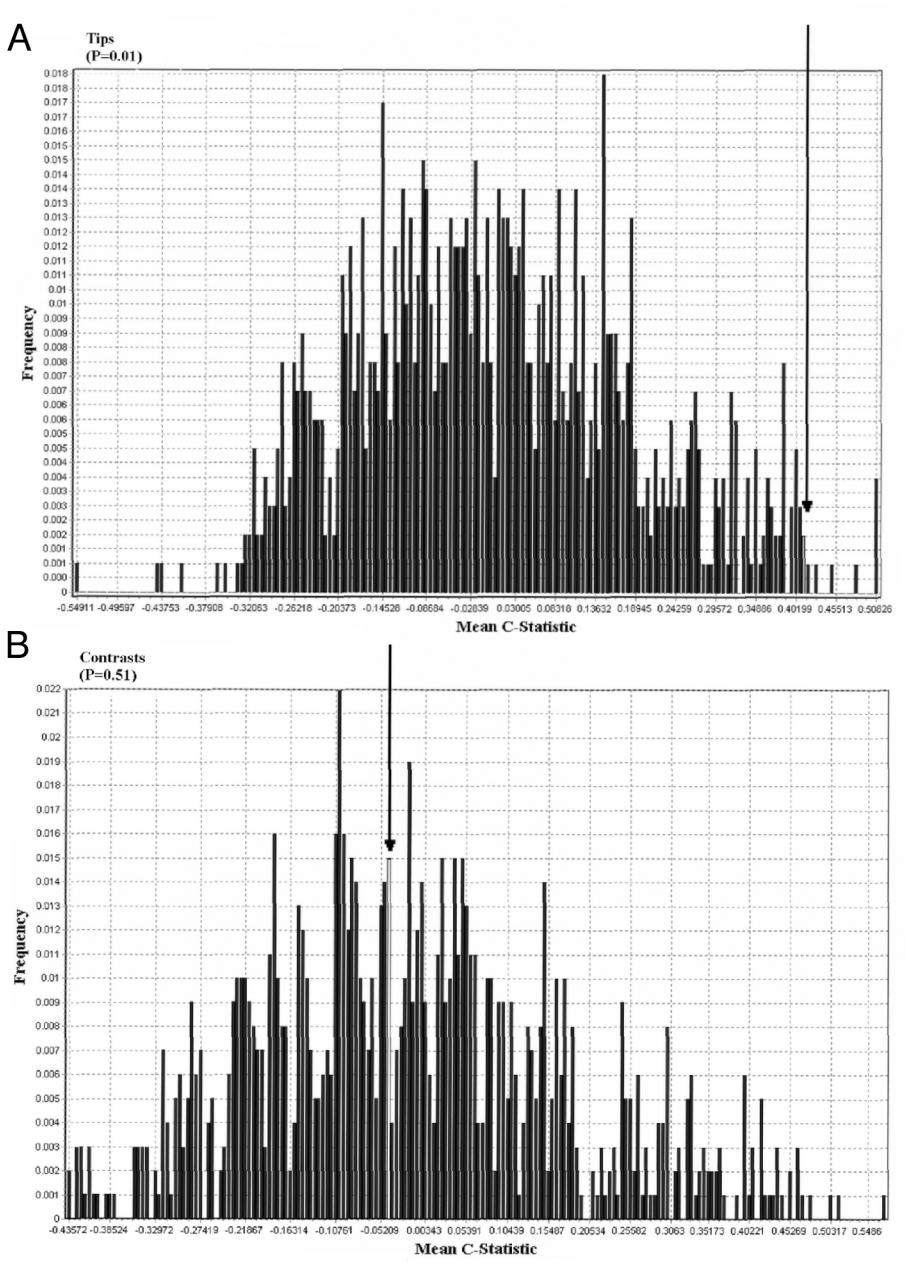

A
Tips
(P=0.01)
Frequency
Mean C-Statistic
B
Contrasts
(P=0.51)
Frequency
Mean C-Statistic

Figure 5 Plot of the percentage variance for the decomposition components obtained from the cranial variables displayed in Table 8. (a+b) = Percentage variance explained by the cranial variable; (b+c) = Percentage variance explained by the phylogeny; (d) = Percentage variance which is unexplained. Abbreviations as in Figure 2.

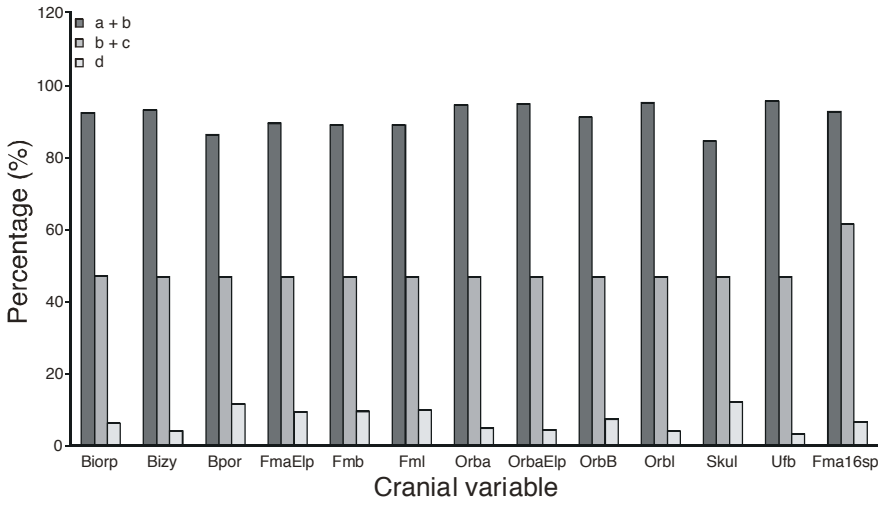

a + b
b + c
d
120
100
80
60
40
20
0
Percentage (%)
Biorp
Bizy
Bpor
FmaElp
Fmb
Fml
Orba
OrbaElp
OrbB
Orbl
Skul
Ufb
Fma16sp
Cranial variable

Figure 6 Plot of the percentage variance for the decomposition components obtained from the cranial variables displayed in Table 8.The letters a, b, c and d are as described by Westoby *et a*l 1995. a – Percentage variance explained by cranial variable alone; b – Percentage variance explained by both the phylogeny and the cranial variable; c – Percentage variance explained by phylogeny alone; d – Percentage variance which is unexplained; other abbreviations as in Figure 2.

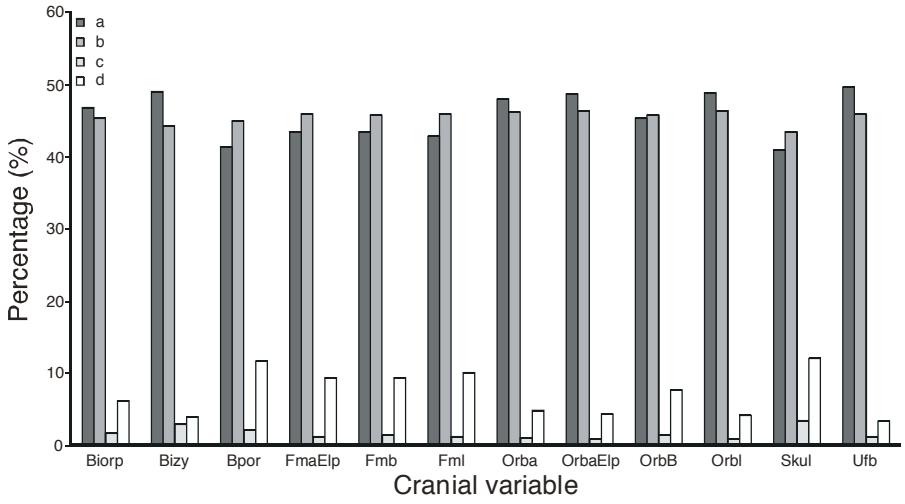
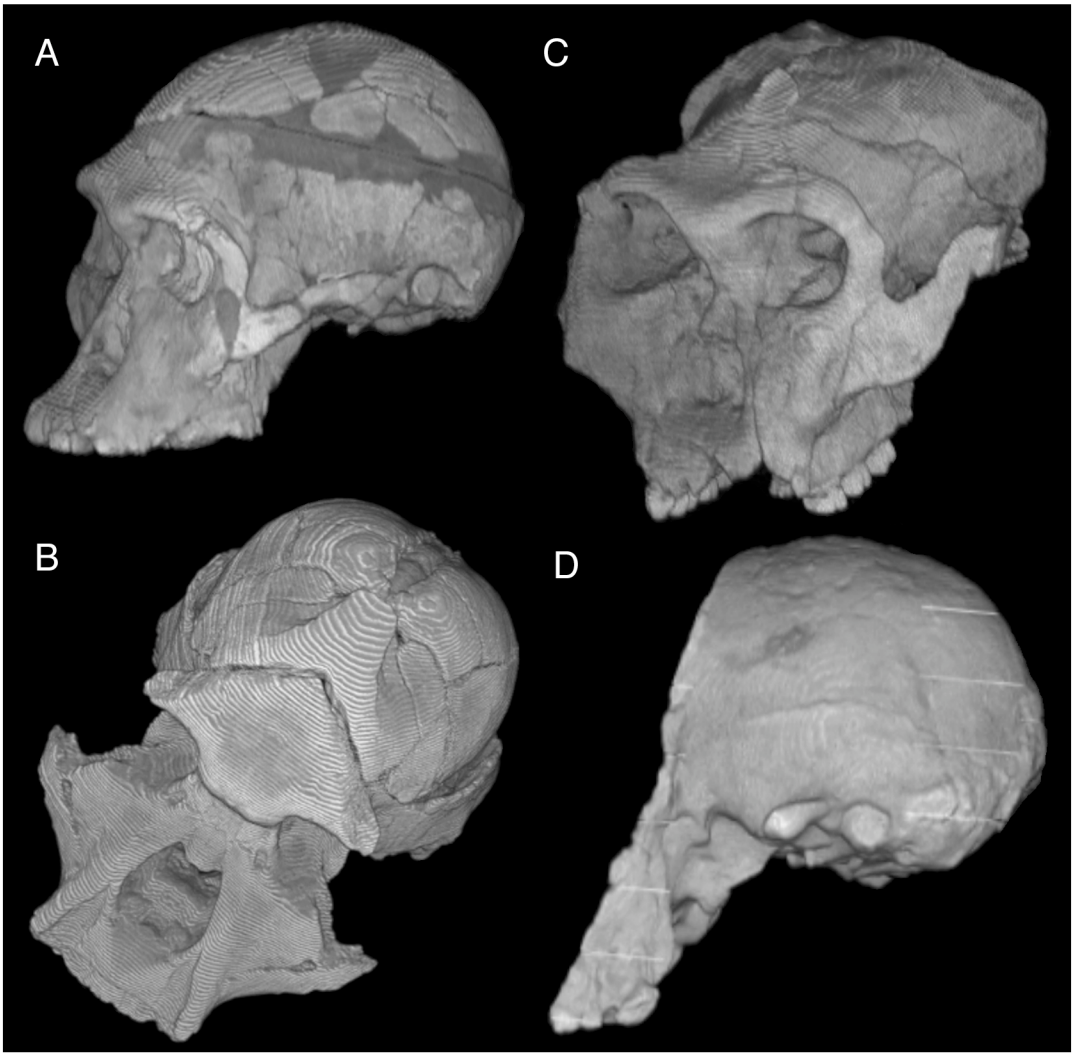

a
b
c
d
60
50
40
30
20
10
0
Percentage (%)
Biorp
Bizy
Bpor
FmaElp
Fmb
Fml
Orba
OrbaElp
OrbB
Orbl
Skul
Ufb
Cranial variable

Figure 7 Selected South African hominid specimens a) STS 5; b) STW 505; c) SK 48; and d) MLD 37/38 reconstructed using 3 D Doctor (Version 4.0, © ABLE Software Corp. 1998-2006).


A
C
B
D

2.4 Discussion

2.4.1 Traditional LSR and RMA regressions

In the analysis of the whole primate sample the most reliable predictor variables were upper facial breadth (Ufb), orbital area (Orba; OrbaElp), orbital height (Orbl), and bizygomatic breadth (Bizy), whilst in the hominoid sample the best predictor variables were foramen magnum area (Fma & FmaElp); biorbital breadth (bpor); orbital height (Orbl); orbital area (Orba; OrbaElp) and biporionic breadth (Biorp). From the analyses it is apparent that these variables are the best predictor variables for the estimation of body mass as they present with high correlation coefficients ($r^2 > 0.92$), low mean percentage prediction errors (in the range of 10% to 37%) and smearing estimates of less than 7%. Orbital area and orbital height are selected in both the hominoid and whole primate analyses as one of the better predictor variables for the estimation of body mass. This is not the first time that orbital dimensions have proved useful in the estimation of body mass. Shultz (1940) examined the relationship between body mass and both eye and orbital volume and showed a general trend for the largest primates to have both the largest orbits and eyes although both indicated negative allometry relative to body mass. Shultz's (1940) sample study consisted of both diurnal and nocturnal Cattarrhini and Platyrrhini primates, a feature which would invariably affect an analysis of the orbit and its contents. A subsequent reanalysis of Shultz's data revealed a correlation coefficient of 0.96 between orbital volume and body mass, a result which has been confirmed by further regression analysis performed by Kappelman (1996) indicating a correlation coefficient of 0.98 between body mass and orbital area. In contrast, Delson *et al* (2000) found orbital dimensions to be poor predictors of body mass in an analysis of cercopithecoid postcranial and cranio-dental dimensions.

Plavcan (2003) also showed that primates exhibit general patterns of greater facial versus neurocranial and orbital dimorphism and greater length as opposed to breadth dimorphism. These findings add credence to developmental studies which have demonstrated that dimorphic growth tends to be stronger in the anterior-posterior components of the skull as opposed to the medio-lateral components of the skull (Leutennegger & Masterson, 1989; Leigh & Cheverud, 1991; Ravosa & Ross, 1994) a feature which possibly underlies the utility of orbital dimensions for body mass estimation.

2.4.2 Control for phylogenetic effects

The resultant correlation coefficients using independent contrast analysis reveal an overall adjustment in r values for the two samples. Results prove overall consistency in the strength of relationship between body mass and cranial variables, with most remaining highly correlated with body mass even after phylogeny is taken into account. Once again, orbital dimensions (orbital height and orbital area) prove to be highly correlated with body mass, with r values ranging between 0.95-0.99. As is the case when using the traditional regression techniques, the maximum length of the skull (Skul) displays the lowest correlation coefficient of between 0.53 and 0.77 for the whole primate sample and hominoid sample respectively. The relatively low r values, together with the high mean percentage prediction errors of 54% and 66% for the whole primate and hominoid samples respectively, strongly argue against the use of the maximum length of the skull to predict body mass for primate species. The poor predictive statistics reported for this variable is indicative of the relatively large amount of variation in skull length between primate species a point which is reiterated by the analysis of the decomposed variance (Table 8).

Using the test for serial independence, it is apparent that the original body mass data as used in traditional regression procedures displays significant phylogenetic autocorrelation (Fig. 4a; $P = 0.01$; $\alpha = 0.05$) whereas the use of 'contrasts' sufficiently adjusts the data to remove any significant phylogenetic autocorrelation (Fig. 4b; $P = 0.51$; $\alpha = 0.05$). As shown in this analysis and previous studies (Gittelman *et al*., 1996; Abouheif and Fairbairn, 1997), body mass is strongly correlated with phylogeny and is ideally suited to analysis by means of independent contrasts to reduce correlation due to common descent. Thus the technique of independent contrast analysis is advocated as a preferred method of investigating body mass regressions, but not for predicting body mass due to the lack of transformation bias error estimates.

2.4.3 Quantifying the partitioned variation

The amount of variation in body mass explained by phylogeny alone from the list of cranial variables is small and argues in favour of the use of cranial variables for the estimation of body mass as only a small component of variance in body mass is attributed to phylogeny alone (range between 0.80% to 3.6%). Once again orbital breadth and orbital area together with upper facial breadth and foramen magnum dimensions are most reflective of this diminished component of variance explained by phylogeny alone.

It is interesting to note the changes in the various components when variance partitioning is applied to the phylogeny in Figure 1 containing 16 species of primates and tested for the variable foramen magnum area (Fma). What is noticeable is the change in the amount of variance explained by foramen magnum area (only) in the 12 species sample as compared to the 16 species sample, and the paralleled change in the

component of variance explained by a combination of foramen magnum area and phylogeny. For the 16 species sample 60% of the variance in body mass is explained by a combination of foramen magnum area and phylogeny as compared to 45% displayed in the 12 species sample. This difference may be largely due to a reduction in the amount of variation explained by the cranial variable alone as component *c*, for the 16 species sample reveals that the amount of variance explained by phylogeny alone has not changed as markedly and is still reflective of the usefulness of cranial variables for body mass estimation.

2.4.4 Predicting Hominid body mass

Differences exist between the body masses derived from the hominoid based equations and those using the whole primate sample, with certain variables showing considerable variability. Larger bodied hominids display a greater range in predicted body mass than that of smaller bodied hominids, *e.g*., *Homo* vs. *Australopithecus*. The degree of variation in predicted body mass when utilising different variables raises the question as to which mass estimates are most representative of the fossil hominids. Differential scaling of fossil crania negates the option of simply choosing variables with the highest predictive capability (Aiello & Wood, 1994). McHenry (1994) proposes guiding the choice of appropriate predictive variable by comparing the average mass predicted from cranial and post-cranial variables for each fossil taxon. As this study wished to examine the utility of cranial elements solely without drawing upon the somewhat problematic post-cranial material, this procedure was not undertaken, and body mass averages based solely on the cranial elements were calculated by averaging the estimates obtained from the best predictor variables (See Table 10). The choice of equation when deriving estimates of fossil hominid body

mass is often guided by phylogenetic closeness. In addition, our understanding of the likely body proportions for the various fossil hominids may also guide our choice of model. In this particular study it is proposed that equations derived from the whole primate sample be used to model body mass for the australopithecines, whilst hominoid based regressions are used when dealing with body mass estimates for *Homo*. This is recommended as body mass estimates based on the whole primate/simian model are largely consistent with those reported in the literature using postcranial elements (See Table 11).

Resultant body mass estimates for the fossil hominids are comparable with that obtained in previous studies. As seen in Table 11, estimates of species body mass for *Australopithecus africanus* have been in the range of 35kg to 45kg as based on postcranial dimensions derived from inter-hominoid/intra human regressions (McHenry, 1988, 1992). Later estimates of body mass of *Australopithecus (A.) africanus* using cranial dimensions, revealed that average body mass for the species ranged between 26kg- 37kg (hominoid regressions) and 28kg-36kg (simian regressions) [Aiello & Wood, 1994]. The body mass estimates for *A. africanus* derived from this current study lie between 28kg-37 kg depending on the variables and regression sample used. This is directly comparable with that observed in a previous study by Aiello & Wood (1994) which also showed a similar body mass range for *A. africanus*. Our average estimate of body mass for *A. africanus* based on the preferred whole primate sample is approximately 30 kg as based on STS 5, whilst MLD 37/38 and STW 505 produce average body mass estimates of 41kg and 47kg respectively. This variation in species body mass when using different fossil specimens may be indicative of sex differences within the species, a point which has seen certain researchers assign two body mass estimates to the taxon, one based on a

small morph of approximately 30kg and that for the larger morph of 41 kg (McHenry, 1992). For *Paranthropus (P.) boisei* comparative body mass estimates based on the postcranial skeleton, typically range between 41kg and 59kg (McHenry, 1988, 1992). Aiello and Wood (1994) had obtained average body mass estimates for *P. boisei* of between 41kg-92kg (hominoid regression) and 43kg-81kg (simian regression) using cranial dimensions. The range in average body mass for *P. boisei* in this current study is 26kg-68kg based on the hominoid regressions and 43kg-55kg based on the preferred whole primate regressions. The average body mass estimate derived from this current study for *P. bosei* is approximately 48 kg. Postcranial elements have yielded average body mass estimates for *P. robustus* within the range of 35kg-47kg (McHenry, 1988, 1991). Cranial dimensions have also yielded similar average body mass estimates using the preferred whole primate regressions, with body mass for *P. robustus* ranging between 41kg-62kg and having a mean species body mass of 52kg as based on SK 48. Body mass estimates for *Homo neanderthalensis* based on La Chapelle 1 and La Ferrassie 1, both approach a species average of 75kg, which is comparable to that observed in the literature for this hominid species and reinforces the utility of the hominoid based regressions in this current study to predict species body mass for later hominids.

2.5 Conclusion

These analyses have supported the usefulness of using cranial variables for the estimation of average body mass in fossil hominids. The advantages of using cranial variables are numerous but of primary utility is the fact that it provides (when reasonably intact) both a measure of cranial capacity and average species body mass from a single specimen. The use of both independent contrasts analysis and a method

of partitioning the variance of body mass explained by each cranial variable reinforces the idea that cranial variables are strongly correlated with body mass even when taking phylogeny into account. Body mass estimates derived for fossil hominids using certain cranial variables display consistency and are similar to those obtained from post-cranial elements.

Chapter 3

Quantitative magnetic resonance imaging of the endocranial volume in humans and other primates: Predicting fossil hominid brain weights

3.1 Introduction

MRI based brain volumetrics is concerned with the analysis of relationships between volumes and / or structural components of the brain (Caviness *et al*., 1999). The versatility and reliability of this technique has seen its increasing application to comparative neuroanatomy and the elucidation of evolutionary similarities or dissimilarities between species (*e.g*. Rilling & Seligman, 2002; Semendeferi & Damasio, 2000; Hopkins *et al*., 1998). In this regard, the endocranial volume serves as a prime candidate for investigation as it makes-up a substantial amount of the baseline data surrounding hominid brain evolutionary studies. The argument in favour of the use of endocranial volumes as a source of data is the seemingly close association between the brain and the skull, a point which has seen numerous researchers use endocranial capacity as a proxy for brain size in both extant and extinct species (*e.g*. Lee & Wolpoff, 2003; Elton *et al*'; 2001; Falk *et al.,* 2000; Leigh, 1992; Hennenberg, 1987; Beals *et al.,* 1984; Lestrel & Read, 1973; Tobias, 1971).

However there are numerous neural and non-neural structures found within the endocranium. Apart from the brain, these include the cerebrospinal fluid, meninges, subarachnoid cisterns, cerebral arteries and veins, cranial venous sinuses and the roots and trunks of the cranial nerves (Romanes, 1996). Estimates of this non-neural component of the endocranial volume have varied with some workers reporting a 10 % discrepancy between neural and non-neural structures (Brandes, 1937) and others reporting values as high as 33.5 % (Mettler, 1955). When one looks at a human

developmental series and ranks it according to age, the non-neural component is shown to range from 6 % at birth to 20 % into early adulthood (Blinkov & Glezer, 1968; Tobias, 1994). This suggests that the amount of neural tissue expressed as a percentage of endocranial volume is inversely correlated with age up to adulthood (Tobias, 1994).

Despite the usefulness of these studies in providing us with an understanding of the variation in the components of the developing human endocranium, several unanswered questions remain. Of particular interest is the investigation of whether endocranial volume is strongly and predictably correlated to brain weight after accounting for the 'residual' non-neural component. If so, we believe that this may render a reliable estimate of brain weight for fossil hominid specimens, thereby facilitating a more accurate depiction of hominid brain evolution.

The present study will address the following questions concerning the proportional occupation of the endocranium by utilizing magnetic resonance imaging techniques: 1) What is the average measure of the non-neural relative to neural component in the endocranium of humans and other primates; 2) Can endocranial volume be used as an intermediate character for the estimation of hominid brain weight; and 3) What does a quantitative assessment using variance partitioning reveal about the strength of relationship between brain weight and endocranial volume.

3.2 Materials and methods

3.2.1 Subjects and MRI acquisition

For the human dataset, a total of 47 adult individuals were obtained from three sources; a student volunteer group of 18 individuals between the ages of 18-25 (6 males and 12 females); 13 normal patients between the ages of 29-77 (1 male and 12

females) scanned on request by neurologists within the Johannesburg region; and a supplementary dataset of 16 normal male subjects between the ages of 21-28 scanned and obtained from the Karolinska Institute, Stockholm, Sweden (Roland *et al.,* 2001, EU-LSSM-CT-2003-504752). The sample was deliberately skewed towards individuals between the age range of 20-35 years of age (See Figure 1) as previous studies have shown that brain growth ceases in the early 20's and that the human brain may undergo atrophy and subsequent reduction in weight in advanced age (Pakkenberg & Voigt, 1964; Chrzanowska *et al*., 1973; Dekaban & Sadowsky, 1978; Hartmann, *et al.,* 1994), though this doesn't appear to be the case for all primates (Herndon *et al*., 1998).Thus this age range was focused on as it represented individuals that were ideally suited for the purposes of this investigation. All subjects consented to making their images available for this study. MRI scans covering the whole brain were performed on a 1.5 Tesla whole body imager (Gyroscan Intera, Phillips Medical Systems). The subjects were imaged in the supine position using a routine protocol. A head coil was employed and a T1 3D acquisition series was obtained to cover the entire head. Acquisition parameters were as follows: voxel spacing [1.04/1.04/3]; dimensions [512, 512, 65] with a TR = 25ms; TE = 4.6ms; fov = 30 with a 0 gap; a matrix of 288 with a reconstruction matrix of 512; a flip angle of 30 and a slice thickness of 3mm.

The non-human primate dataset was obtained from the published data of Rilling and Insel (1998) released under permission from the authors to the fMRI Data Center and obtainable on request from www.fmridc.org. Scan acquisition parameters are as indicated in the original article (Rilling & Insel, 1998) with a total sample size of 29 specimens used in this current study, representing 11 anthropoid species. The included species were: *Saimiri sciureus* (N = 2), *Cebus apella* (N = 2), *Cercocebus*

atys (N = 3), *Macaca mulatta* (N = 3), *Papio cynocephalus* (N = 2), *Hylobates lar* (N = 4), *Pongo pygmaeus* (N = 3), *Gorilla gorilla* (N = 2), *Pan troglodytes* (N = 4) and *Pan pansicus* (N = 4).

3.2.2 Volumetric image analysis

Coronal T1 weighted images were pre-processed using MRIcro Version.1.39 (Rorden & Brett, 2000) whereby each image sequence was manually outlined for the region of interest (ROI), in this case being the endocranial volume. The resultant endocranial volume sequences were then saved in Analyze format and then imported into ITK-SNAP Version. 1.4. RCI (Yushkevich *et al.,* 2006), an open source application which provides semi-automated segmentation of anatomical structures and allows for the reconstruction of three dimensional volumes. Brain volumes were determined for the specimens of interest by using a skull-stripping procedure (BSE) which accompanies the neuron-imaging package Brainsuite2.01 (Shattuck & Leahy, 2001). This skull-stripping procedure was preferred to the built-in Brain extraction Tool (Smith, 2000) which accompanies MRIcro on the basis that it has been shown to yield better results for brain size (Fennema-Notestine *et al*., 2005).

Pre-processing facilitated the removal of any non-brain tissue (*i.e*. endocranial space, meninges etc.) and provided images which were free of extra-neural tissue and were exclusively representative of the brain volume. Both endocranial volume sequences and brain volume sequences were then semi-automatically segmented in ITK-SNAP to produce 3-D reconstructions of the endocranial volumes and brain volumes. The difference between endocranial volume and brain volume, termed endocranial space (ECS) and representing the non-neural component of the endocranial volume was calculated by simple subtraction and used in subsequent

statistical analyses along with the already determined values for endocranial volume and brain volume for each individual. Brain volumes were converted to weight by multiplying the volumes by the specific gravity of brain tissue (Stephan *et al*., 1981). Weighted means for each variable were calculated per species in order to account for the sometimes uneven representation of males and females for the samples (Sokal & Rolf, 1969). This allowed for the calculation of mean values for each species which were representative of the number of males and females which made up each sample (See Table 12 and Table 13). Figures 9 and 10 provides an example of the procedural outline used during image preparation and analysis.

3.2.3 Statistical analysis

3.2.3.1 Repeatability

Absolute volumes were calculated for each image sequence using the volume and statistics function provided in ITK-SNAP. As a means of ensuring that accurate volumes were being obtained, 10 image sequences were manually outlined using ImageJ Version.1.3 (Abramoff *et al.,* 2004). From the calibrated images, area dimensions were calculated on each slice and these areas were summed and multiplied by the slice thickness to provide a volumetric measure of the combined regions of interest in the image sequence. Repeated measures of volume were also taken on selected image sequences using both ITK-SNAP and ImageJ and these were tested using Lin's concordance correlation coefficient (Lin, 1989). This method was used to assess the level of repeatability and the measurement error (intra-observer) for each variable.

3.2.3.2 Regression analysis

All variables were tested for normality prior to inclusion in any subsequent analyses whereafter the data was logarithmically transformed (base 10) and 'traditional' regression equations using Reduced Major Axis analysis (RMA) were computed using PAST (Version.1.18; PAST © Hammer & Harper, 1999-2005).

In order to take into account the potential bias introduced by treating species asZon-independent data points, the technique of independent contrast analysis (Felsenstein, 1985) was also applied to the dataset using COMPARE (Version. 2.0 – CONTRAST © University of Oregon and E.P. Martins, 1997). As a method, independent contrasts analysis operates by comparing sets of differences between different nodes in a known phylogeny and computing 'contrasts' which represent the difference between pairs of species/nodes in a phylogeny. Contrasts are then 'weighted' in proportion to the amount of time estimated to separate nodes. Numerous arguments have been raised in favour of the use of independent contrasts but some of the more pertinent points are as follows: a) lower type 1 errors compared to those incurred by traditional regression techniques (Harvey & Rambaut, 2000); b) the greater robustness of the Brownian motion model even when violated (Garland *et al*., 1992; Garland & Ives, 2000); and c) the increased statistical power to detect associations and relationships among groups (Nunn & Barton, 2001). The specific phylogenetic hypothesis used in this study is shown in Figure 4 and is based on a composite phylogeny for the Order Primates (Purvis, 1995).

3.2.3.3 Variance partitioning

The advent of the 'Comparative method' has brought to prominence a number of techniques that not only take phylogeny into account but are also widely applicable to differing datasets. These techniques have facilitated the study of both 'trait on trait' comparisons and that of the effects of environmental variables on traits. (Harvey & Pagel, 1991).However, an interesting point was raised by Westoby *et al.,* (1995) who argued that comparative methods investigating the interaction between a trait and an environmental variable, may have an intrinsic bias by allocating the maximal amount of variation in the trait as due to phylogeny and only considering the ecological effects as being residual (Westoby *et al*., 1995). Accordingly it was argued that the phylogenetic portion of the total variance of a variable is not exclusive and may also contain a phylogenetic component related to ecology, called "phylogenetic niche conservatism" (Harvey and Pagel, 1991). The mixed interaction between adaptation and phylogenetic constraint poses a challenge to comparative techniques as it represents the portion of variation simultaneously explained by both ancestry and adaptation, an area which is largely not accounted for in comparative settings.

Westoby *et al.,* (1995) proposed to partition the variance of data into three portions *a*, *b* and *c* - where *a* is a part strictly due to environment, *b* is a part due to the common influence of environment and phylogeny, and *c* is a part strictly due to phylogeny. Subsequently, a method was proposed to quantify and calculate the partitioning of variation in a phylogenetic setting whilst also taking into account the unexplained part of variation (Desdevises *et al.*, 2003). This method is applied in the current study and follows the procedural steps outlined by Desdevises *et al*., (2003) and as described in a previous article by some of the current authors (Spocter & Manger, 2006). Although the use of variance partitioning has been advocated for the

investigation and quantification of the relative effects of adaptation and constraints on the formation of traits, this method is not strictly devoted to this study. In fact when using non-ecological variables the method may be used successfully to study the effects of phylogeny and developmental/structural or functional components making up the variables of interest. In this particular study it was decided to investigate the strength of relationship existing between endocranial volume and its sub-components, with the aim of quantifying the effects of developmental and phylogenetic effects. This facilitated the dissection of the variance into four major components, namely: the variance solely due to endocranial volume (developmental constraints); variance due to phylogeny alone (phylogenetic constraints); variance due to a combination of phylogeny and the effects of endocranial volume; and the variance designated as unexplained by the model.

3.3 Results and Discussion

Endocranial volume inflates brain size by approximately 21% in both adult humans and anthropoid primates. Endocranial volume is highly correlated with brain weight and this relationship is used to derive an equation for the calculation of brain weights for fossil hominid species (Brain weight (g) = 0.8177 * Endocranial volume (cc) – 14.138). Analysis using variance partitioning reveals that endocranial volume explains 96 % of the variation in both brain weight and endocranial space and is most likely to be the result of developmental constraints acting upon the internal components of the endocranium.

3.3.1 Repeatability

A test of the degree of intra observer error using Lin's Concordance Correlation Coefficient revealed a Pc value of between 0.90 and 0.98, indicating a percentage accuracy of 90 % for outlines of endocranial volume and 98 % for the designation of brain volume. Cross validation results of endocranial volume calculated by using a combination of regions of interest (ROI) outlined in MRIcro and then segmented in ITKSNAP were highly confluent with those obtained using ImageJ. The combination of the results summarised above and the accuracy provided by the technique of magnetic resonance imaging, highlights the confidence which the authors have in the techniques and the subsequent results obtained in this study.

3.3.2 Human dataset

The analysis of the human dataset shows an interesting range of variation (See Table12). In particular, endocranial volume ranges between 1316 cc to 1862 cc, whilst brain volume ranges between 991cc to 1476cc. Associated with this variation in both endocranial volume and brain volume is an evident variable measure of body weight which has a minimum value of 44 kg and a maximum value 121 kg for the subjects studied. Although computation of brain weights for the sample is also indicative of the variation highlighted above, what is most apparent and surprising is the relatively conservative measure of endocranial space. This ranges between 14 % and 27 %, with a mean of 21 %; a median of 20.9 % and a standard deviation of 3.2. In order to account for the uneven representation of males and females in the sample, weighted means were calculated. This provided a weighted mean of 21.1 % for endocranial space, a value consistent with previous studies (e.g. Blinkoff & Glezer,

1968; Tobias, 1994) and reiterates the percentage overestimate of brain volume when using endocranial volume/cranial capacity as a 'stand alone' estimate.

3.3.3 Non-human primate dataset

Volumes for the endocranial contents of the non-human primate dataset were calculated with the weighted means for the species (Table 13). This computation revealed that for both endocranial volume and brain volume/mass the human sample displayed the largest overall values in comparison to the other species. Average endocranial volume for the human sample was approximately 1575 cubic centimetres (cc). This is approximately 2.5 times greater than in *Pongo pygmaeus,* which had the second largest endocranial volume (614 cc), followed by *Gorilla gorilla*, *Pan troglodytes* and *Pan paniscus* which have endocranial volumes of 558 cc, 535 cc and 534 cc respectively. Species with larger body weights also present with larger endocranial volumes and overall larger amounts of endocranial space. But the percentage endocranial space (ECS/ECV *100) seems more consistent across species, ranging between 18% for the 0.7kg *Saimiri sciureus* to 31 % for the 45kg *Pan paniscus.* Although the human sample has an overall larger endocranial space totaling approximately 328 cc, the percentage endocranial space is only 21 % which is comparable to that observed in the other non-human primate species, where 90 % of the species points lie within 5 percentage points of the median. This preliminary analysis would seem to suggest that endocranial volume over- estimates brain volume/size across the species represented in this study, by approximately 25 %.

3.3.4 Regression analysis

Following this preliminary investigation, further analyses were performed on the raw data to investigate the relationships between the residual endocranial space, endocranial volume, brain size/weight and body size. Results obtained from this set of regression analyses are presented in Table 14 which gives *r* –values obtained using both reduced major axis (RMA) and independent contrast analysis (IC). All *r* – values were high and were associated with other good indicators reflective of the strength of relationship, such as low error terms, high p-values and fairly normally distributed residuals. Notable was the exceptionally strong correlations between brain volume and endocranial space and brain volume and endocranial volume. This result is hardly surprising considering the close association as indicated in both this study and previous studies between the endocranial vault and the underlying brain, especially when considering the cross-species 'conservation' of the relative amount of residual endocranial space forming the interface between the brain and the endocranium. Just as interesting is the strongly almost isometric, relationship existing between brain weight and endocranial volume ($R^2 = 0.996$, slope=1.001, y –intercept= -0.116) a result which proved just as strongly correlated even when adjusting for phylogeny.

It is apparent that the expressed relationship between brain weight and endocranial volume may be employed with accuracy to estimate brain weights for fossil species. Most comparative neuroanatomical studies in extant species use brain weight as an integral part of their study, as a means of extracting necessary data, but cranial capacity has been used successfully as a proxy for brain size in fossil hominid species (e.g. Lee & Wolpoff, 2003). Despite this success it is apparent that if a reliable estimate of brain weight were to be extracted from the existing data, this may facilitate a more refined investigation of brain size changes for fossil species. As

shown in this current study, endocranial volume/cranial capacity may overestimate brain size by approximately 25 %. Thus an accurate estimate of brain weight computed by using the underlying biological relationship, especially a strong isometric relationship as that between brain weight and endocranial volume, may add greater value and 'sensitivity' to an analysis of brain and body size changes in hominid evolution.

In order to test this assertion we compiled a dataset of pre-published cranial capacities for various fossil hominids for whom there is relatively little debate concerning the validity of the reconstructions (Table 15). Following this, the equation derived for the relationship between brain weight and endocranial volume was used to compute brain weights. To avoid the bias associated with the de-transformation of data as is the case when aiming to predict variables from a log transformed dataset (Smith, 1993), it was decided to use the 'raw' /non-log transformed data to derive the equation representing the relationship between endocranial volume and brain weight. This relationship as when using the log transformed data, shows strong predictive capability with an r value = 0.998; slope = 0.8177 and a Y-intercept = -14.138 [*i.e.* **Brain weight (g) = 0.8177 * Endocranial volume (cc) – 14.138**] (See Figure 12a; 12b; 12c and 12d). The probability of the two variables being uncorrelated is P (uncorr) = $9.706 * 10^{-13}$ whilst the probability of the slope being equal to one is P (a=1) = $2.39 * 10^{-7}$, both supporting statistics reiterating the general results of isometry and strong predictive capability as seen in the log transformed data. The resultant brain weight estimates for the fossil specimens is displayed in Table 15.

3.3.5 Variance partitioning

In order to investigate the relative contributions of developmental constraints, phylogenetic constraints, and the 'mixed' effects of phylogeny and developmental constraints, we decided to employ a method of variance partitioning as implemented by Desdevises *et al.,* (2003). In the absence of an 'ecological' variable, endocranial volume was used as a primary *X* variable to investigate the portion of variance in brain size and endocranial space explained by endocranial volume attributed to developmental constraint, after having accounted for phylogeny. When utilising this approach as a means of discerning between partition categories, the following results were obtained (See Figure 13). Approximately 96% of the variation in either endocranial space or brain weight can be attributed to endocranial volume and thus argued to be a result of developmental constraints existing between the structures. Both niche conservatism and phylogenetic constraint appeared to play a substantially lesser role in explaining the variation existing in the models.

3.4 Conclusion

Cranial capacity/endocranial volume has long been used as a proxy for brain size in hominid evolutionary studies and has in the past provided a suitable measure for comparison in the absence of neural tissue. However, the current study has re-emphasised the magnitude of the over-estimation of brain volume by endocranial volume. If future studies are to improve upon their quantitative description of brain evolution for the hominid lineage, then a reassessment of brain size by reference to weights calculated from the strong isometric relationship existing between endocranial volume and brain weight is essential and will allow an extension and refinement of the available data. We believe that the contribution provided in this

study may serve as an adequate starting point from which future paleoneurological investigators may approach questions of size and relative size for changes in the hominid brain. Whilst this study has tried at best to provide a mean measure for the relatv_e over-estimate of brain size in adult primate species, there still remain a number of questions concerning the change in endocranial space with age in other non-human primates. This investigation of a developmental series for non-human primates may prove to be an apt extension of the results provided in this current study.

Table 12 Mean age (Age), body weight (BW), endocranial volume (ECV), brain volume (BrnV), brain weight (BrnW), residual endocranial space (ECS) and percentage endocranial space (ECS/ECV*100) in 47 Human subjects. Average values are weighted means calculated in order to account for uneven distribution of males and females in the sample.

Specimen	Age(years)	BW (kg)	ECV(cc)	BrV(cc)	BrW(g)	ECS(cc)	ECS/ECV*100
Homo sapiens 1	43.00	44.00	1562.93	1298.19	1344.92	264.74	16.94
Homo sapiens 2	33.00	60.00	1574.21	1253.42	1298.55	320.78	20.38
Homo sapiens 3	49.00	75.00	1579.43	1268.62	1314.29	310.81	19.68
Homo sapiens 4	40.00	73.00	1542.82	1248.69	1293.64	294.13	19.06
Homo sapiens 5	35.00	65.00	1629.67	1307.86	1354.95	321.81	19.75
Homo sapiens 6	49.00	82.00	1720.94	1346.89	1395.37	374.05	21.74
Homo sapiens 7	36.00	65.00	1861.35	1385.24	1435.11	476.11	25.58
Homo sapiens 8	29.00	62.00	1602.14	1319.52	1367.02	282.62	17.64
Homo sapiens 9	56.00	66.00	1794.96	1366.87	1416.07	428.09	23.85
Homo sapiens 10	56.00	53.00	1543.11	1183.58	1226.19	359.53	23.30
Homo sapiens 11	23.00	121.30	1711.86	1354.70	1403.47	357.16	20.86
Homo sapiens 12	22.00	47.00	1582.75	1221.47	1265.44	361.28	22.83
Homo sapiens 13	18.00	61.00	1452.34	1163.08	1204.95	289.26	19.92
Homo sapiens 14	18.00	51.10	1568.21	1275.37	1321.29	292.84	18.67
Homo sapiens 15	22.00	45.00	1612.35	1318.50	1365.97	293.85	18.22
Homo sapiens 16	20.00	59.90	1862.34	1475.82	1528.95	386.52	20.75
Homo sapiens 17	20.00	44.50	1512.60	1259.67	1305.02	252.92	16.72
Homo sapiens 18	20.00	49.00	1689.58	1383.57	1433.38	306.01	18.11
Homo sapiens 19	22.00	48.00	1459.68	1196.92	1240.01	262.76	18.00
Homo sapiens 20	21.00	54.00	1446.13	1184.52	1227.16	261.61	18.09
Homo sapiens 21	23.00	58.00	1493.26	1221.10	1265.06	272.16	18.23
Homo sapiens 22	20.00	52.90	1638.72	1327.61	1375.40	311.11	18.98
Homo sapiens 23	77.00	75.00	1551.20	1327.04	1374.81	224.16	14.45
Homo sapiens 24	70.00	80.00	1481.21	1142.23	1183.35	338.98	22.89
Homo sapiens 25	60.00	115.00	1768.59	1440.96	1492.83	327.63	18.53
Homo sapiens 26	22.00	79.00	1675.45	1302.88	1349.78	372.58	22.24
Homo sapiens 27	22.00	62.00	1626.68	1307.49	1354.56	319.19	19.62
Homo sapiens 28	20.00	72.00	1574.68	1295.53	1342.17	279.14	17.73
Homo sapiens 29	21.00	52.00	1550.57	1222.35	1266.36	328.22	21.17
Homo sapiens 30	23.00	82.30	1511.41	1169.14	1211.23	342.27	22.65
Homo sapiens 31	21.00	83.20	1613.06	1270.54	1316.27	342.52	21.23
Homo sapiens 32	23.00	N/S	1506.00	1119.17	1159.46	386.83	25.69
Homo sapiens 33	30.00	N/S	1481.66	1136.02	1176.91	345.65	23.33
Homo sapiens 34	22.00	N/S	1458.25	1156.86	1198.51	301.39	20.67
Homo sapiens 35	28.00	N/S	1615.54	1386.34	1436.25	229.20	14.19
Homo sapiens 36	22.00	N/S	1548.08	1194.20	1237.19	353.88	22.86
Homo sapiens 37	27.00	N/S	1558.71	1139.49	1180.51	419.22	26.90
Homo sapiens 38	25.00	N/S	1513.18	1178.61	1221.04	334.57	22.11
Homo sapiens 39	23.00	N/S	1520.15	1166.52	1208.52	353.62	23.26
Homo sapiens 40	28.00	N/S	1467.55	1183.24	1225.83	284.31	19.37
Homo sapiens 41	24.00	N/S	1628.08	1186.05	1228.75	442.03	27.15
Homo sapiens 42	24.00	N/S	1316.03	990.96	1026.63	325.07	24.70
Homo sapiens 43	26.00	N/S	1358.24	988.39	1023.97	369.85	27.23
Homo sapiens 44	23.00	N/S	1382.20	1006.62	1042.86	375.58	27.17
Homo sapiens 45	24.00	N/S	1616.79	1207.43	1250.90	409.35	25.32
Homo sapiens 46	21.00	N/S	1644.18	1276.24	1322.18	367.95	22.38
Homo sapiens 47	23.00	N/S	1639.38	1275.83	1321.76	363.55	22.18
Average	**29.87**	**43.34**	**1575.49**	**1243.22**	**1287.98**	**332.27**	**21.11**

Table 13 Mean age (Age), body weight (BW), endocranial volume (ECV), brain volume (BrnV), brain weight (BrnW), residual endocranial space (ECS) and percentage endocranial space (ECS/ECV*100) for subjects from 11 anthropoid species. Five anthropoid families are represented namely: Hominidae, Pongidae, Hylobatidae, Cercopithecidae and Cebidae. Intraspecific sample sizes range from between 2-50 individuals. Average values are weighted means calculated in order to account for uneven distribution of males and females in the sample.

Specimen	Age(years)	BW (kg)	ECV(cc)	BrV(cc)	BrW(g)	ECS(cc)	ECS/ECV*100
Pan paniscus 1	25.00	68.50	570.48	370.11	383.43	200.37	35.12
Pan paniscus 2	8.00	35.00	474.37	353.55	366.28	120.82	25.47
Pan paniscus 3	11.50	36.70	557.07	376.58	390.13	180.49	32.40
Pan paniscus 4	28.00	41.50	535.46	357.05	369.90	178.41	33.32
Average	**18.13**	**45.43**	**534.34**	**364.32**	**377.44**	**170.02**	**31.58**
Pan troglodytes 1	8.00	43.00	584.39	397.43	411.74	186.96	31.99
Pan troglodytes 2	39.00	41.50	500.18	334.31	346.35	165.86	33.16
Pan troglodytes 3	36.00	44.00	464.88	348.95	361.51	115.93	24.94
Pan troglodytes 4	20.50	62.50	592.46	424.79	440.09	167.67	28.30
Average	**25.88**	**47.75**	**535.48**	**376.37**	**389.92**	**159.10**	**29.60**
Gorilla gorilla 1	8.00	68.10	625.54	522.29	541.09	103.26	16.51
Gorilla gorilla 2	13.50	55.40	490.56	364.67	377.79	125.89	25.66
Average	**10.75**	**61.75**	**558.05**	**443.48**	**459.44**	**114.58**	**21.08**
Pongo pygmaeus 1	8.50	52.50	608.49	428.85	444.28	179.65	29.52
Pongo pygmaeus 2	15.00	87.00	629.89	467.83	484.68	162.06	25.73
Pongo pygmaeus 3	18.00	100.00	604.32	503.74	521.88	100.58	16.64
Average	**13.83**	**79.83**	**614.24**	**466.81**	**483.61**	**147.43**	**23.96**
Hylobates lar 1	26.00	5.48	196.88	119.50	123.80	77.38	39.30
Hylobates lar 2	23.50	5.35	127.14	84.33	87.36	42.81	33.67
Hylobates lar 3	18.50	6.70	137.24	90.91	94.18	46.33	33.76
Hylobates lar 4	5.50	4.20	110.52	98.35	101.89	12.17	11.01
Average	**18.38**	**5.43**	**142.94**	**98.27**	**101.81**	**44.67**	**29.44**
Papio ursinus 1	4.50	20.60	184.93	136.87	141.79	48.06	25.99
Papio ursinus 2	6.50	23.15	242.73	188.73	195.52	54.00	22.25
Average	**5.50**	**21.88**	**213.83**	**162.80**	**168.66**	**51.03**	**24.12**
Cercocebus atys 1	9.00	6.30	130.31	102.21	105.89	28.10	21.56
Cercocebus atys 2	5.00	8.20	135.00	103.12	106.83	31.88	23.62
Cercocebus atys 3	8.00	9.40	146.31	108.70	112.62	37.60	25.70
Average	**7.33**	**7.97**	**137.21**	**104.68**	**108.45**	**32.53**	**23.63**
Macaca mulata 1	14.50	12.50	113.97	80.69	83.59	33.29	29.21
Macaca mulata 3	11.00	9.70	122.98	91.46	94.75	31.52	25.63
Average	**10.33**	**11.07**	**120.38**	**84.50**	**87.54**	**35.88**	**29.78**
Saimiri sciureus 1	2.50	0.80	34.12	28.15	29.16	5.98	17.52
Saimiri sciureus 2	2.50	0.60	33.53	27.18	28.15	6.36	18.96
Average	**2.50**	**0.70**	**33.83**	**27.66**	**28.66**	**6.17**	**18.24**
Cebus apella 1	10.50	4.30	109.61	76.94	79.71	32.67	29.80
Cebus apella 2	4.50	2.30	87.95	65.47	67.83	22.48	25.56
Average	**7.50**	**3.30**	**98.78**	**71.21**	**73.77**	**27.58**	**27.68**
Homo sapiens average	**29.87**	**43.34**	**1575.49**	**1243.22**	**1287.98**	**332.27**	**21.11**

Table 14 Regression statistics summarising the strength of relationship existing between body weight (BW) and brain weight (BrnW), body weight and endocranial volume (ECV), body weight and endocranial space (ECS), body weight and brain volume (BrnV), brain volume and endocranial volume and brain weight and endocranial volume using both Reduced Major Axis (RMA) and Independent Contrasts Analysis (IC).All results are computed using log transformed data. a = slope; b = Y intercept; r = Pearsons correlation coefficient; P(uncorr)= probability of being uncorrelated; P(a=1) = Probability of the slope being 1.

		RMA	IC									
X	**Y**	**r**	**r**	**N**	***a***	***b***	**error *a***	**error *b***	**P (uncorr)**	**P (*a*=1)**	**95 % on *a***	**95 % on *b***
BW	BrnW	0.938	0.52	11	0.728	-1.815	0.076	0.745	$1.97*10^{-5}$	0.006	0.59 - 1.08	-5.41 - -0.62
BW	ECV	0.944	0.52	11	0.728	-1.510	0.073	0.710	$1.28*10^{-5}$	0.004	0.60 - 0.99	-4.17 - -0.32
BW	ECS	0.946	0.504	11	0.742	-3.035	0.073	0.713	$1.10*10^{-5}$	0.006	0.58 - 0.95	-5.24 - -1.48
BW	BrnV	0.938	0.52	11	0.728	-1.815	0.076	0.745	$1.97*10^{-5}$	0.006	0.59 - 1.06	-5.25 - -0.61
BrnV	ECS	0.975	0.955	11	1.019	-1.185	0.068	0.362	$3.21*10^{-7}$	0.784	0.84 - 1.19	-2.01 - -0.16
BrnV	ECV	0.998	0.998	11	0.999	0.303	0.017	0.091	$1.46*10^{-12}$	0.942	0.96 - 1.04	0.09 - 0.55
BrnW	ECV	0.998	0.998	11	1.001	-0.116	0.017	0.042	$1.46*10^{-12}$	0.942	0.96 - 1.05	-0.24 - -0.03

Table 15 Fossil hominid data collated from the literature with recorded chronological age (MyBp) and cranial capacity (cc). The estimated brain weight (BrnW) for each specimen as calculated using the equation Brain weight (g) = 0.8177 * Endocranial volume (cc) – 14.138 is also presented. Note the average difference of approximately 20 grams between reported cranial capacity and estimated brain weight. MyBp = Millions of years before present.

Specimen	Taxon	Age	CC	BrnW
TM 266-01-60-1	*Sahelanthropus tchadensis*	6	365	312.6
A.L. 333-45	*Australopithecus afarensis*	3.4	500	423
A.L. 162-28	*Australopithecus afarensis*	3.4	400	341.2
A.L. 444-2	*Australopithecus afarensis*	2.9	500	423
ARA-VP-12/130	*Australopithecus garhi*	2.6	400	341.2
KNM-WT 17000	*Paranthropus aethiopicus*	2.5	410	349.4
MLD 1	*Australopithecus africanus*	2.6	500	423
MLD 37/38	*Australopithecus africanus*	2.6	435	369.8
Sts 5	*Australopithecus africanus*	2.25	485	410.7
Sts 19/58	*Australopithecus africanus*	2.25	436	370.7
Sts 60	*Australopithecus africanus*	2.25	428	364.1
Sts 71	*Australopithecus africanus*	2.25	428	364.1
Stw 505	*Australopithecus africanus*	2.25	585	492.5
KNM-ER 406	*Paranthropus boisei*	1.8	510	431.2
KNM-ER 732	*Paranthropus boisei*	1.8	500	423
KNM-ER 13750	*Paranthropus boisei*	1.8	475	402.5
KNM-ER 407	*Paranthropus boisei*	1.8	506	427.9
OH 5	*Paranthropus boisei*	1.81	530	447.5
SK 1585	*Paranthropus robustus*	1.81	530	447.5
OH 7	*Homo habilis*	1.81	674	565.3
OH 13	*Homo habilis*	1.81	673	564.5
OH 16	*Homo habilis*	1.81	638	535.8

Specimen	Taxon	Age	CC	BrnW
La Quina 5	*Homo neanderthalensis*	0.05	1350	1118
La Quina 18	*Homo neanderthalensis*	0.05	1310	1085.3
Spy 1	*Homo neanderthalensis*	0.05	1305	1081.2
Spy 2	*Homo neanderthalensis*	0.05	1553	1284
Shanidar 1	*Homo neanderthalensis*	0.05	1600	1322.5
Shanidar 5	*Homo neanderthalensis*	0.05	1550	1281.6
Teshik-Tash 1	*Homo neanderthalensis*	0.05	1578	1304.5
Qafzeh 6	*Homo sapiens*	0.09	1535	1269.3
Qafzeh 9	*Homo sapiens*	0.09	1531	1266
Qafzeh 11	*Homo sapiens*	0.09	1280	1060.8
Skhul 4	*Homo sapiens*	0.09	1554	1284.8
Skhul 5	*Homo sapiens*	0.09	1518	1255.4
Skhul 9	*Homo sapiens*	0.09	1587	1311.8
Cro-Magnon 1	*Homo sapiens*	0.03	1600	1322.5
Dolni Vestonice 3	*Homo sapiens*	0.026	1322	1095.1
Grotte des Enfants 4	*Homo sapiens*	0.028	1775	1465.6
Grotte des Enfants 5	*Homo sapiens*	0.028	1375	1138.5
Grotte des Enfants 6	*Homo sapiens*	0.028	1580	1306.1
Mladec 1	*Homo sapiens*	0.035	1620	1338.8
Mladec 5	*Homo sapiens*	0.035	1500	1240.7
Paderbourne	*Homo sapiens*	0.027	1531	1266
Pataud 1	*Homo sapiens*	0.021	1380	1142.6

Specimen	Taxon	Age	CC	BrnW
OH 24	*Homo habilis*	1.81	594	499.9
KNM-ER 1805	*Homo habilis*	1.81	582	490
KNM-ER 1813	*Homo habilis*	1.81	509	430.3
KNM-ER 1470	*Homo rudolfensis*	1.81	752	629
D2700	*Homo georgicus*	1.7	600	504.8
D2280	*Homo georgicus*	1.7	775	647.9
D2282	*Homo georgicus*	1.7	655	549.7
KNM-ER 3733	*Homo erectus*	1.78	804	671.6
KNM-ER 3883	*Homo erectus*	1.58	848	707.5
OH 9	*Homo erectus*	1.2	1067	886.6
UA 31	*Homo erectus*	1	775	647.9
BOU-VP-2/66	*Homo erectus*	1	995	827.7
Gongwangling 1	*Homo erectus*	1.15	780	651.9
Sangiran 2	*Homo erectus*	1	813	678.9
Sangiran 10	*Homo erectus*	0.8	700	586.5
Sangiran 12	*Homo erectus*	0.8	1059	880.1
Sangiran 17	*Homo erectus*	0.8	1004	835.1
Zhoukoudian D1	*Homo erectus*	0.44	1030	856.4
Zhoukoudian E1	*Homo erectus*	0.44	915	762.3
Zhoukoudian H3	*Homo erectus*	0.44	1140	946.3
Zhoukoudian L1	*Homo erectus*	0.44	1225	1016
Zhoukoudian L2	*Homo erectus*	0.44	1015	844.1
Zhoukoudian L3	*Homo erectus*	0.44	1030	856.4
Sambungmacan 1	*Homo erectus*	0.4	1035	860.5
Hexian 1	*Homo erectus*	0.2	1025	852.3
Ngandong 1	*Homo erectus*	0.2	1172	972.5
Ngandong 5	*Homo erectus*	0.2	1251	1037
Ngandong 6	*Homo erectus*	0.2	1013	842.5
Ngandong 9	*Homo erectus*	0.2	1135	942.2

Specimen	Taxon	Age	CC	BrnW
Pavlov 1	*Homo sapiens*	0.026	1522	1258.7
Predmosti 3	*Homo sapiens*	0.027	1608	1329
Predmosti 4	*Homo sapiens*	0.027	1518	1255.4
Predmosti 9	*Homo sapiens*	0.027	1555	1285.7
Predmosti 10	*Homo sapiens*	0.027	1452	1201.4
Nazlet Khater 1	*Homo sapiens*	0.033	1420	1175.3
Minatogawa 1	*Homo sapiens*	0.018	1390	1150.7
Minatogawa 2	*Homo sapiens*	0.018	1170	970.85
Minatogawa 4	*Homo sapiens*	0.018	1090	905.43
Zhoukoudian Up. Cave 1	*Homo sapiens*	0.018	1500	1240.7
Zhoukoudian Up. Cave 2	*Homo sapiens*	0.018	1380	1142.6
Zhoukoudian Up. Cave 3	*Homo sapiens*	0.018	1290	1069
Arene Candide 1-IP	*Homo sapiens*	0.018	1490	1232.5
Arene Candide 1	*Homo sapiens*	0.011	1414	1170.4
Arene Candide 2	*Homo sapiens*	0.011	1424	1178.5
Arene Candide 4	*Homo sapiens*	0.011	1520	1257
Arene Candide 5	*Homo sapiens*	0.011	1661	1372.3
Barma Grande 2	*Homo sapiens*	0.019	1880	1551.4
Bruniquel 2	*Homo sapiens*	0.012	1555	1285.7
Cap Blanc 1	*Homo sapiens*	0.012	1434	1186.7
Chancelade 1	*Homo sapiens*	0.012	1700	1404.2
Oberkassel 1	*Homo sapiens*	0.012	1500	1240.7
Oberkassel 2	*Homo sapiens*	0.012	1370	1134.4
Saint Germain-la-Riviere 1	*Homo sapiens*	0.015	1354	1121.3
San Teodoro 1	*Homo sapiens*	0.011	1565	1293.8
San Teodoro 2	*Homo sapiens*	0.011	1569	1297.1
San Teodoro 3	*Homo sapiens*	0.011	1560	1289.8
San Teodoro 5	*Homo sapiens*	0.011	1484	1227.6
Veryier 1	*Homo sapiens*	0.01	1430	1183.4

Specimen	*Taxon*	Age	CC	BrnW
Ngandong 11	*Homo erectus*	0.2	1090	905.4
Hexian 1	*Homo erectus*	0.2	1025	852.3
Saldanha 1	*Homo heidelbergensis*	0.5	1225	1016
Swanscombe 1	*Homo heidelbergensis*	0.4	1325	1098
Arago 21	*Homo heidelbergensis*	0.4	1166	967.6
Steinheim 1	*Homo heidelbergensis*	0.3	950	791
Petralona 1	*Homo heidelbergensis*	0.3	1230	1020
Ndutu 1	*Homo heidelbergensis*	0.4	1100	913.6
Atapuerca 4	*Homo heidelbergensis*	0.3	1390	1151
Atapuerca 5	*Homo heidelbergensis*	0.3	1125	934.1
Atapuerca 6	*Homo heidelbergensis*	0.3	1140	946.3
Broken Hill 1	*Homo heidelbergensis*	0.3	1280	1061
Dali 1	*Homo heidelbergensis*	0.3	1120	930
Ehringsdorf 9	*Homo heidelbergensis*	0.2	1450	1200
Jinnu Shan 1	*Archaic Homo*	0.2	1300	1077
Narmada 1	*Archaic Homo*	0.3	1260	1044
Singa 1	*Archaic Homo*	0.15	1550	1282
Laetoli 18	*Archaic Homo*	0.13	1367	1132
Omo-Kibish 2	*Archaic Homo*	0.13	1435	1188
Krapina 3	*Homo neanderthalensis*	0.13	1200	995.4
Saccopastore 1	*Homo neanderthalensis*	0.1	1258	1043
Tabun C1	*Homo neanderthalensis*	0.15	1271	1053
Amud 1	*Homo neanderthalensis*	0.045	1750	1445
La Chapelle-aux-Saints	*Homo neanderthalensis*	0.052	1626	1344
La Ferrassie 1	*Homo neanderthalensis*	0.072	1681	1389
Forbes' Quarry	*Homo neanderthalensis*	0.05	1200	995.4
Ganovce 1	*Homo neanderthalensis*	0.05	1320	1094
Guattari 1	*Homo neanderthalensis*	0.057	1550	1282
Le Moustier 1	*Homo neanderthalensis*	0.04	1600	1322

Specimen	*Taxon*	Age	CC	BrnW
Pecos	*Homo sapiens*	0.001	1030	856.37
Pecos	*Homo sapiens*	0.001	1275	1056.7
Pecos	*Homo sapiens*	0.001	1300	1077.1
Pecos	*Homo sapiens*	0.001	1120	929.96
Pecos	*Homo sapiens*	0.001	1380	1142.6
Pecos	*Homo sapiens*	0.001	1380	1142.6
Pecos	*Homo sapiens*	0.001	1270	1052.6
Pecos	*Homo sapiens*	0.001	1100	91U^61
Pecos	*Homo sapiens*	0.001	1465	1212.1
Pecos	*Homo sapiens*	0.001	1320	1093.5
Pecos	*Homo sapiens*	0.001	1285	1064.9
Pecos	*Homo sapiens*	0.001	1350	1118
Pecos	*Homo sapiens*	0.001	1440	1191.6
Pecos	*Homo sapiens*	0.001	1410	1167.1
Pecos	*Homo sapiens*	0.001	1350	1118
Pecos	*Homo sapiens*	0.001	1190	987.2
Pecos	*Homo sapiens*	0.001	1300	1077.1
Pecos	*Homo sapiens*	0.001	1390	1150.7
Pecos	*Homo sapiens*	0.001	1350	1118
Pecos	*Homo sapiens*	0.001	1140	946.32
Pecos	*Homo sapiens*	0.001	1155	958.58
Pecos	*Homo sapiens*	0.001	1178	977.39
Pecos	*Homo sapiens*	0.001	1340	1109.9
Pecos	*Homo sapiens*	0.001	1500	1240.7
Pecos	*Homo sapiens*	0.001	1550	1281.6
Pecos	*Homo sapiens*	0.001	1400	1158.9
Pecos	*Homo sapiens*	0.001	1350	1118
Pecos	*Homo sapiens*	0.001	1325	1097.6

Figure 8 A plot of the frequency distribution for the age ranges used in the human dataset. Note the skewing of the distribution towards individuals between the ages of 20-35 years of age.

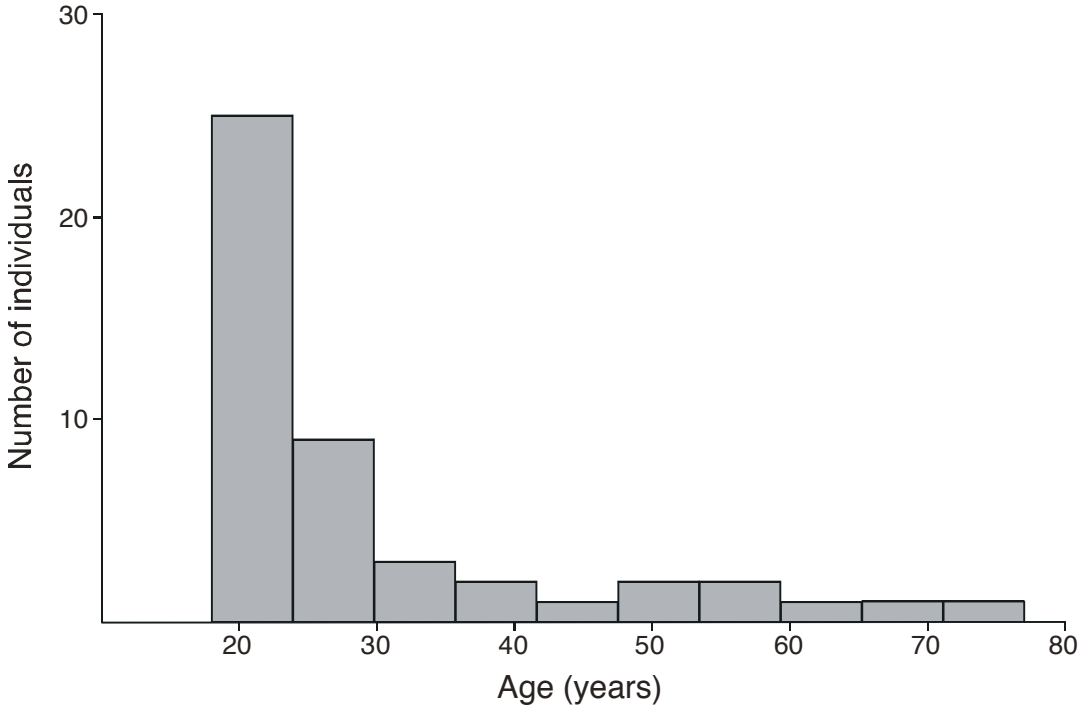

30
20
10
Number of individuals
20
30
40
50
60
70
80
Age (years)

Figure 9 Procedural outline used for the extraction of brain and endocranial volumes. **A**- Represents the 'Raw' magnetic resonance images prior to outlining and isolating the volumes of interest. **B**- Represents the outlined endocranial volume using a region of interest tool provided in MRIcro. The isolated endocranial volume is shown in **C** where after the brain volume is extracted using the Brain Surface Extraction tool (BSE) as provided in Brainsuite 2.01 and as shown in **D** and **E**. The final endocranial vol_qe and brain volumes are then 3-dimensionally reconstructed using ITKSnap examples of which are shown in **F** and **G**.

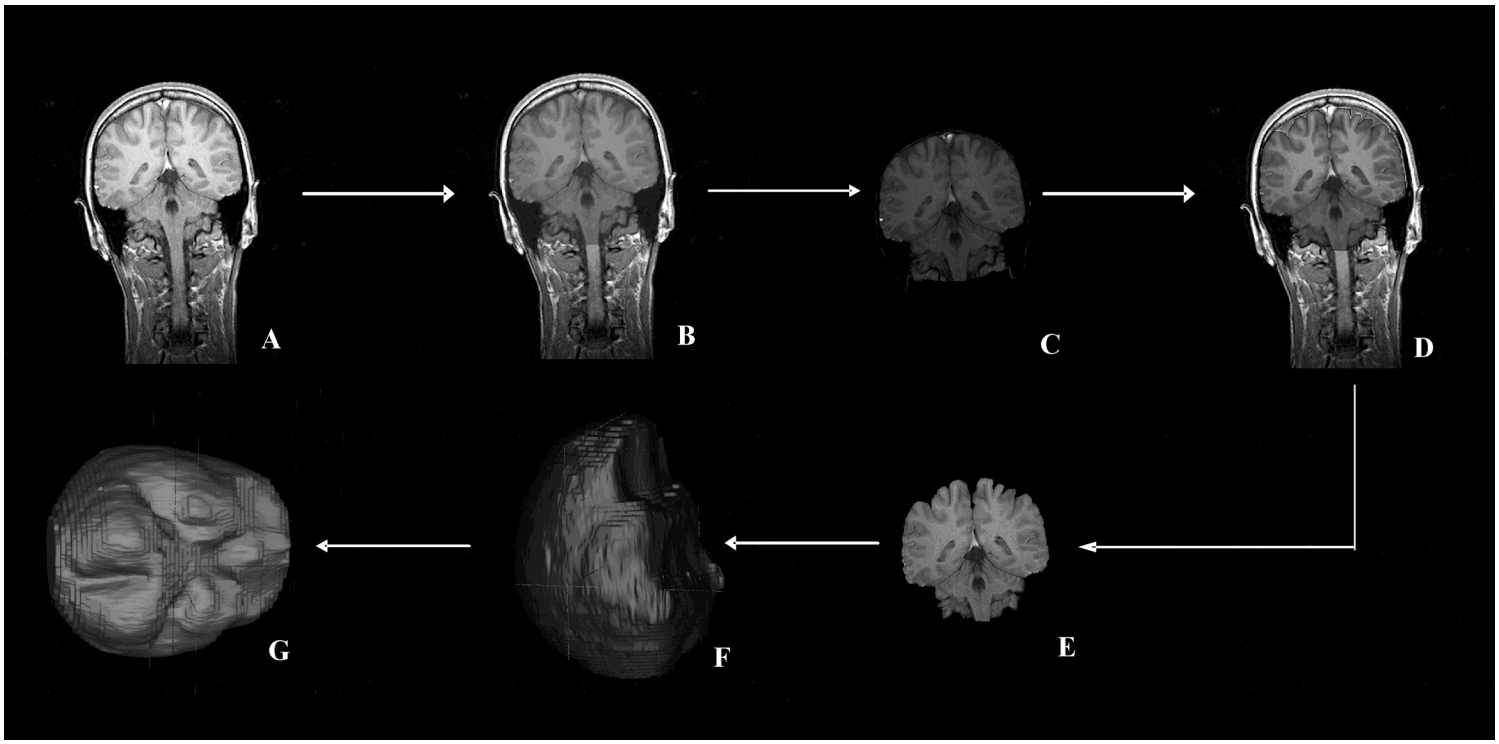

A
B
C
D
E
F
G

Figure 10 An example of the reconstructed brain volume superimposed upon the endocranial volume and skull of one of the non-human primate specimens. Note that despite the close association between the endocranial volume and brain volume, there still remains a cumulatively significant amount of 'residual' endocranial space in certain regions.

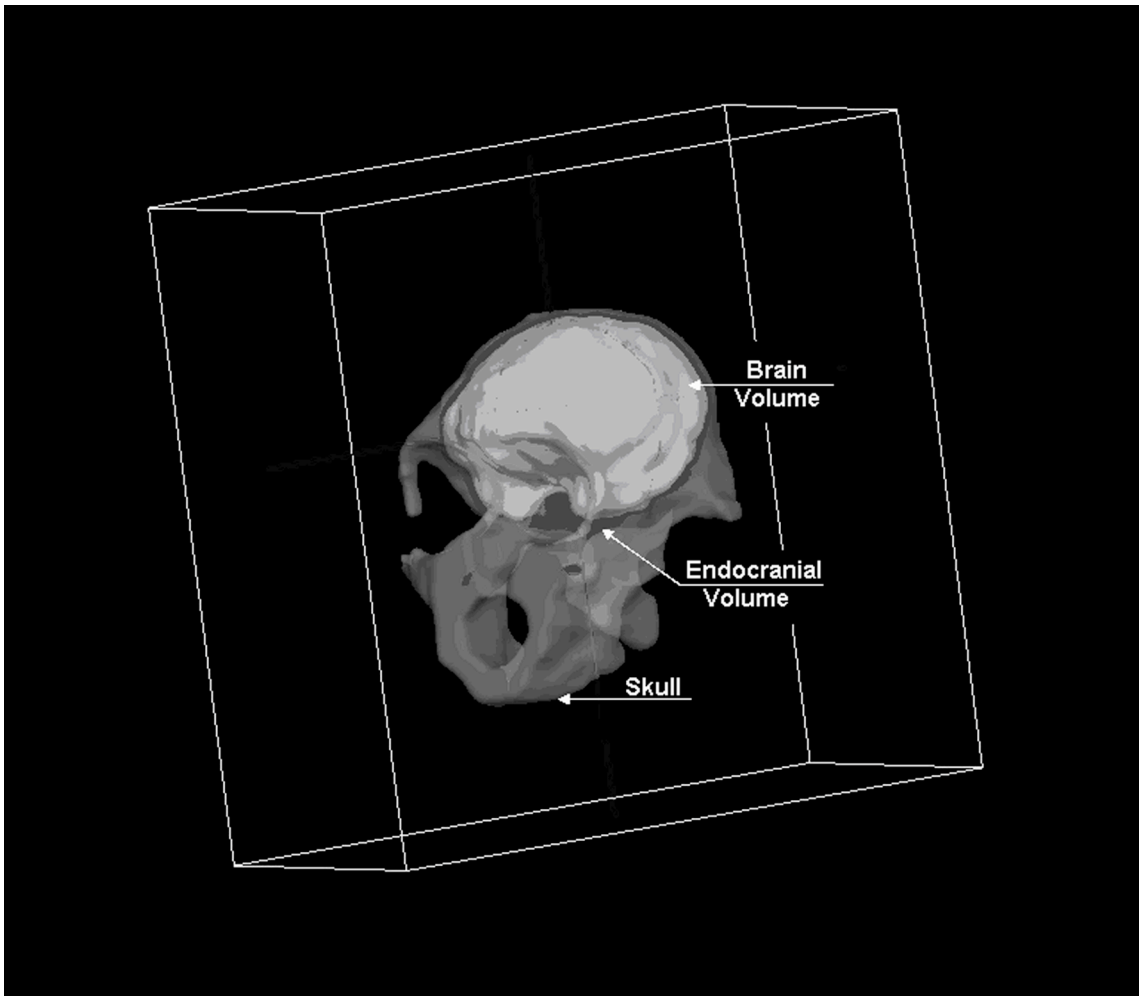

Brain
Volume
Endocranial
Volume
Skull

Figure 11 Phylogeny used for the calculation of independent contrasts. A scaled tree provided by Purvis (1995) was used as a graphic template and then redrawn to highlight the species of interest. Nodes and branch lengths were then 'captured' from the scanned and calibrated tree using TreeThief Version 1.01 where after the tree statistics were used to compute contrasts in Compare version 2.0.The final tree for publication was then reconstructed using the Drawtree program accompanying Joseph Felsenstein's Phyllip 3.65 ©1999-2004 University of Washington.

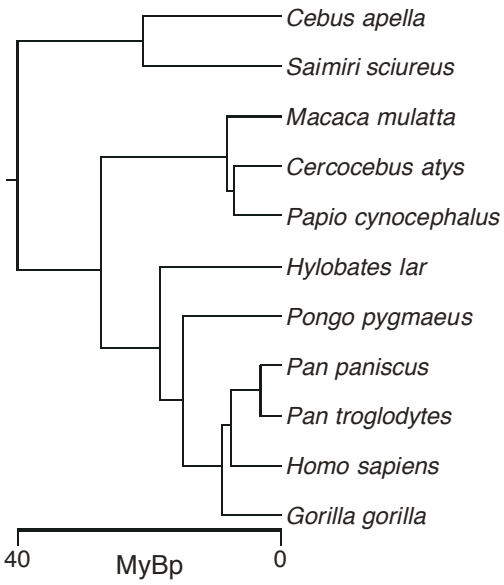

Cebus apella
Saimiri sciureus
Macaca mulatta
Cercocebus atys
Papio cynocephalus
Hylobates lar
Pongo pygmaeus
Pan paniscus
Pan troglodytes
Homo sapiens
Gorilla gorilla
40
MyBp
0

Figure 12 **A**. Regression analysis plot (reduced major axis) of log endocranial volume against log brain weight for the human dataset using reduced major axis. Note the strength of correlation r = 0.88; P (uncorr) = $1.66*10^{-16}$ and the slope statistics which are suggestive of isometry. **B**. Regression analysis plot (reduced major axis) of endocranial volume against brain weight for all primate species. **C**. Regression analysis plot (reduced major axis) of endocranial volume against brain weight for all primate species using the weighted means of the variables under study. **D**. Least square regression analysis plot of the 'contrasts' computed using phylogenetic independent contrast analysis for endocranial volume against brain weight. Once again note the strong correlation and indications of isometry.

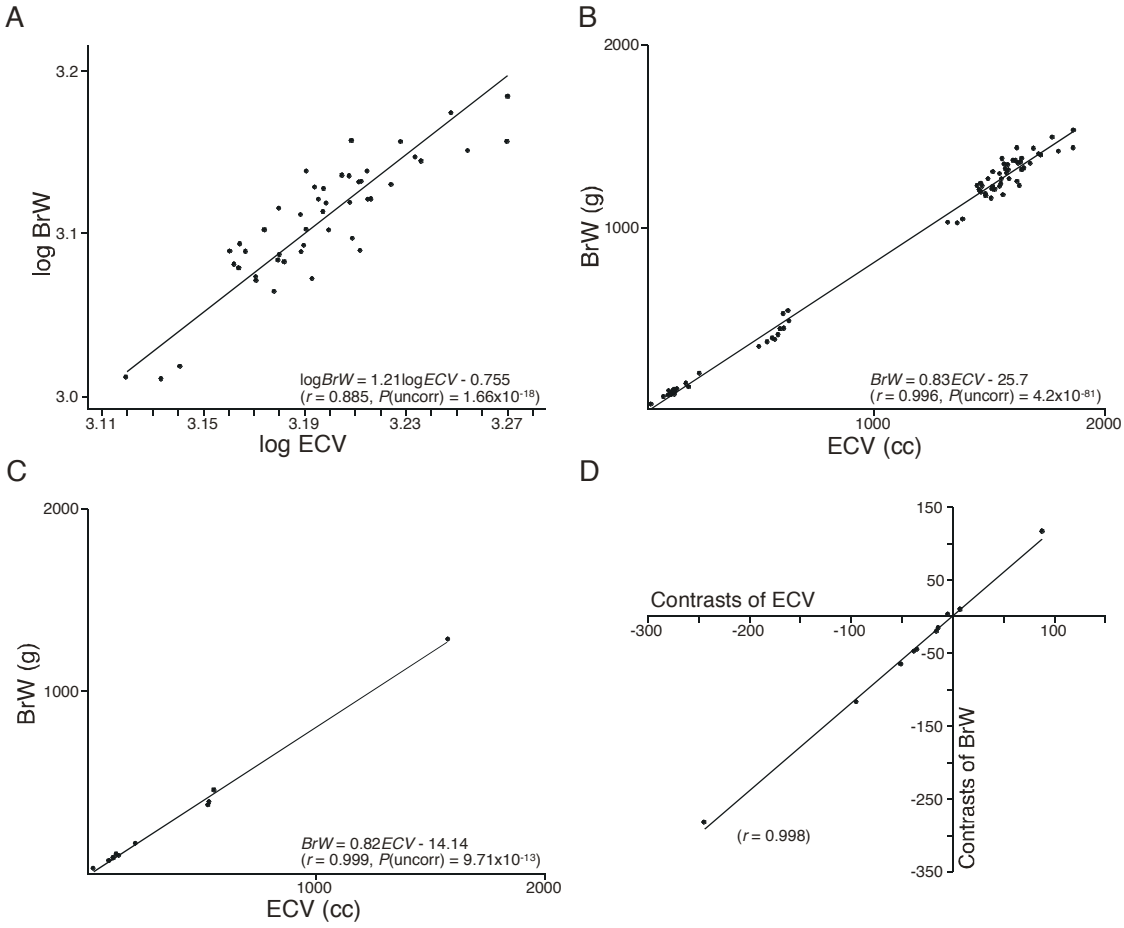
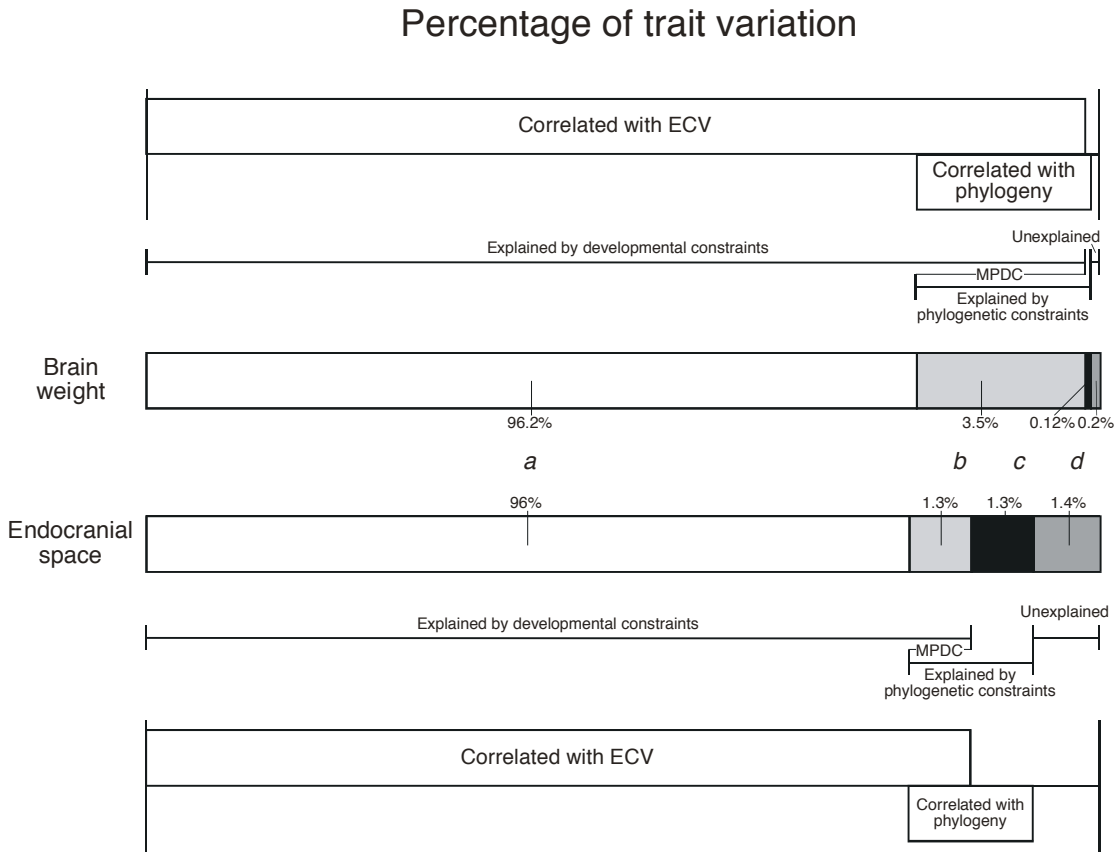

A
3.2
3.1
3.0
log BrW
3.11
3.15
3.19
3.23
3.27
log ECV
logBrW = 1.21logECV - 0.755
(r = 0.885, P(uncorr) = 1.66x10-18)
B
2000
1000
BrW (g)
1000
2000
ECV (cc)
BrW = 0.83ECV - 25.7
(r = 0.996, P(uncorr) = 4.2x10-81)
C
2000
1000
BrW (g)
1000
2000
ECV (cc)
BrW = 0.82ECV - 14.14
(r = 0.999, P(uncorr) = 9.71x10-13)
D
150
50
-50
-150
-250
-350
Contrasts of ECV
-300
-200
-100
100
Contrasts of BrW
(r = 0.998)

Figure 13 Summary of the results from the variance partitioning method aimed at exploring the percentage trait variation in brain weight and endocranial space explained by endocranial volume (**ECV**). As mentioned in the text, when endocranial volume is used as a primary X variable, then the results from the variance partitioning method may be argued to render the relative effects of developmental constraint, phylogenetic constraints and the mixed effects of phylogeny and developmental constraints (**MPDC**). Accordingly it is shown that the greatest amount of the variation in brain weight and the absolute amount of endocranial space can be explained by developmental constraints whilst the mixed combinatory effects of developmental and phylogenetic constraints appear to have played a substantially lesser role in the molding of the displayed variance for the species understudy.


Percentage of trait variation
Correlated with ECV
Correlated with phylogeny
Unexplained
Explained by developmental constraints
MPDC
Explained by phylogenetic constraints
Brain weight
96.2%
3.5%
0.12%
0.2%
a
b
c
d
Endocranial space
96%
1.3%
1.3%
1.4%
Explained by developmental constraints
Unexplained
MPDC
Explained by phylogenetic constraints
Correlated with ECV
Correlated with phylogeny

Chapter 4

Intra specific variation in human brain mass

4.1 Introduction

Amongst the numerous questions pertaining to the structure and evolution of the human brain, brain mass both absolute and relative stands out as an obvious feature for comparison and is often argued to underscore much of humankind's uniqueness in terms of cognition and problem-solving abilities (e.g. Jerison, 1973). This line of reasoning is apparent in hominid evolutionary studies where the size of the hominid brain remains an integral character for investigating evolutionary trends and for assigning taxonomic categories (e.g. Lee & Wolpoff, 2003; Elton *et al.,* 2001; Falk et al, 2000; Leigh, 1992; Henneberg, 1987; Tobias, 1985; Holloway, 1983), and also in comparative studies of extant primate brains (e.g. Elston *et al.,* 2006). An extension from these type of studies is the close association between absolute brain size and absolute cerebral cortical volume (Finlay *et al*., 2001), that indicates that absolute brain size is a good indicator of the cognitive complexity that can be found in the cerebral cortex of humans and other mammals.

Variable estimates for brain size in fossil specimens coupled with considerable overlap for certain taxa, raise serious questions concerning the validity of brain size estimates and the variation in estimates due to differing techniques (Henneberg & De Miguel, 2001). Perhaps more disconcerting is the obvious existence and acknowledgment of individual variation in brain size (e.g. Tobias, 1968, 1971; Olivier & Tissier, 1975; Holloway, 1980; Henneberg *et al.,* 1985; Blumenberg, 1985; Henneberg, 1990). This evident variation signals a note of caution towards the use of brain mass as a taxonomically diagnostic feature or cognitive indicator and warrants an enquiry as to the range and nature of variation in brain mass for extant species.

In this regard several attempts have been made to document the range of inter-specific variability in brain size for *Homo sapiens*, however despite the success of these studies, many fall short on one of three grounds: 1) the use of cranial capacity as a proxy for brain mass despite the fact that this has been shown to overestimate brain mass by approximately 20% (Brandes, 1936; Metteler, 1955; Tobias, 1994; Spocter, *et al.,* 2007); 2) the use of relatively small sample sizes to represent population means; and 3) the failure to recognise covariation in parameters such as height, weight and age and its effects on brain mass.

Hierarchical views of inter and intra-population differences in brain mass for *Homo sapiens* and the possible existence of links to intelligence as first argued by early 19th Century researchers (reviewed by Gould, 1981), have once again surfaced in the writings of a sector of present day evolutionary and behavioural psychologists (Rushton, 1988, 1991, 1992, 1994, 1997, 1998; Rushton & Ankney, 1996; Rushton & Osborne, 1995; Ankney, 1992; Rushton, 1991). Despite contemporary reports refuting these claims (e.g. Gould, 1978, 1981; Gross, 1990; Peters, 1993) it appears appropriate to rebut proponents of hierarchical models of intelligence by drawing upon large, reliable datasets and to investigate the various biological factors that may exist and contribute in a 'causal' manner towards inter and intra- population brain mass variation.

This study asks the following specific questions concerning brain mass variation for *Homo sapiens* using a dataset of over 18 500 individual records of brain mass, body mass, height, sex, population affinity, and age: 1) What is the range of variation and the level of sexual dimorphism in brain mass (within and between population groups); 2) Is there any evidence for stratification in brain mass along the lines of population affinity and sex; 3) What is the strength of correlation between

brain mass, body mass, height and age; and 4) Using various factors simultaneously can a hypothesised 'causal' model be established to explain the variation existing in human brain mass.

4.2 Materials and methods

4.2.1 Sample and measurement acquisition

The database accumulated consisted of a total of 18 612 autopsy records (12 560 males; 6 152 females) of individuals aged from 1 month to 100 years obtained from two sources: 1) the Hamburg University Hospital, Federal Republic of Germany (17 860 individuals) and 2) the Johannesburg Government Mortuary, South Africa (752 individuals). All individuals were autopsied within three days of death and individuals with emaciated bodies or head injury were excluded from the sample. Measurements of body mass and brain mass were taken using autopsy scales with the brain masses representing 'wet' weights with the dura mater removed and the brain stem clipped at the level of the foramen magnum. Measurements of height were taken using a tape measure (centimetres) while age, sex and population affinity were confirmed using the death certificates. The German sample represented a population of 'European descent' collected from autopsy records over a period of 20 years in and around the Hamburg area, while the South African sample was a mixed sample of South Africans of both 'European' and 'African' descent having died within the Johannesburg region between January 2005 to January 2006. In the current study, most of the statistical analyses and results are based upon a sub-sample of 11 000 individuals drawn from the original database and aged between 18 years and 60 years of age. This age cohort is used as it eliminates the effects of individuals undergoing significant changes in growth as well as the degenerative effects of aging.

4.2.2 Statistical analysis

4.2.2.1 Brain mass, body mass and height variation for males, females and population groups

In order to determine the range of variability in brain mass, body mass, height and age for males, females and between and within the various population groups, univariate descriptive statistics (i.e. means, standard deviation, variances etc.) were computed for each sex and population group (Table 16). In addition, coefficients of variation (COV) and significance tests such as the Student's t-test and the F-test were used to test for significant differences between the means and variances (Table 17).

4.2.2.2 Regressional permutations between brain mass, body mass, height and age

Regression analyses (reduced major axis) were performed on the data, to investigate the permutational relationships between brain mass, body mass, height, and age, between and within the sexes and population groups. The resulting regression equations were computed using PAST (Version.1.18; PAST © Hammer & Harper, 1999-2005) while tests to determine if there exists any significant difference between male versus female and African versus European regression slopes were implemented using (S)MATR: Standardised Major Axis Tests & Routines (Version 2.0; © Falster, 2003-2005) (Table 20).

4.2.2.3 Sexual dimorphism in brain mass, body mass and height

Sexual dimorphism is an important component of the morphological variation for a species (e.g. Oxnard, 1987; Kelley, 1993) and is related to various aspects of social organisation and behaviour (e.g. Alexander *et al.,* 1979; Clutton-Brock, 1985;

Plavcan & van Shaick, 1992; Lovejoy, 1981; Borgognini Tarli & Repetto, 1986; Kappelman, 1996). A number of different approaches are used to measure sexual dimorphism. In this current study estimates of the amount of sexual dimorphism in brain mass, body mass and height were calculated per population group by assessing the magnitude of the difference between mean male and mean female measures for the traits in question. This difference represented the absolute difference in size between the sexes. Subsequently, a further measure derived from the more widely used quotient approach of Borgognini et al. (1986) representing the percentage sexual dimorphism in size was computed as follows:

Percentage sexual dimorphism = [(Mean male measure – mean female measure) / mean female measure] * 100

To investigate whether the amount of sexual dimorphism varies with age, it was decided to partition the samples into age cohorts. These cohorts represent: a developmental phase (Phases 1 to 3, which is characterised by pronounced growth in body dimensions); an adult phase (Phase 4, characterised by relatively little growth in the body dimensions); and a degenerative phase (Phase 5, characterised by a reduction in size with advanced age). The results where calculated for cohorts with large enough sample sizes are tabulated in Table 18.

4.2.2.4 Testing heterochrony as an explanation for why females have smaller mean brain and body masses than males

Using an ontogenetic series drawn from the European (Hamburg) sample a test for heterochrony was performed on the brain and body mass growth curves for males

and females. In this particular study, we have adopted a definition for heterochrony which is similar to that proposed by Rice (1997, 2002). We thus define heterochrony as a uniform change in the rate or timing of a developmental process. Subsequently, growth curves were analysed by transforming female curves using sequential hypermorphosis (Rice, 2002). In this particular study the phenotype axes for brain mass and body mass was multiplied by the factors 1.12 and 1.19 respectively (calculated from the mean brain mass and body mass ratios). A line of the form $Y = ab^x x^c$ was fit through the male data points ($r > 0.97$) and then the distribution of the transformed female data points were investigated for evidence of even distribution about this line (Rice, 2002). The runs test was then applied in accordance with the procedures outlined by Rice (2002) by finding the longest run of transformed female data points on the same side of the male line and calculating the probability of this occurrence. Although the female growth curves were transformed and male growth curves were used as reference points, transformation of either sex would provide equivalent results. To accommodate for reservations concerning the use of a 'runs test' on a potentially non-symmetrical dist~öbution of brain masses, the more robust Kolmogorov-Smirnov test was applied to the dataset to test if there exists any significant difference between male and female transformed and untransformed data points.

4.2.2.5 Causal modelling of human brain mass variation

Having analysed the separate sources of variation in brain mass due to age, body mass and height, it is apparent that perhaps a simultaneous analysis of these traits and their 'joint' effect may provide greater explanatory power and provide insight into modelling the causes of variation in human brain mass. To test this, both

multiple regression and path analysis was applied to the various age cohorts within the dataset. The underlying reason for partitioning the data into age cohorts is the possibility that the strength of relationship between variables may vary at different developmental periods. Multiple regression analysis was thus applied to the following age cohorts: phase 1 (0-3yrs); phase 2 (0-5yrs); phase 3 (6 -17yrs); phase 4 (18-60yrs) and phase 5 (61-100yrs), with the included variables being age, body mass, height and brain mass. Cohorts were then selected for path analysis depending on the strength of the multiple regression coefficients (i.e. the cohort with the largest coefficient of multiple determination was selected for subsequent path analysis). Derived 'causal hypotheses' were then tested using the available data and were offered up as potential theoretical frameworks within which the variables are related. The procedures of path analysis and multiple regression analysis were performed using AMOS 5 (Version 5. © Arbuckle, 2005, Statistical Product and Service Solutions).

4.2.2.6 The effects of climate on human brain mass

A reanalysis of the Beals *et al* (1984) dataset (CRANDAT) was performed in this study by omitting populations for which cranial capacity or body mass had not been recorded. The original sample from Beals *et al* (1984) consisted of cranial capacity data from 122 human populations and body mass data obtained from 52 human populations worldwide, and had been published in a dataset available for study from the World Cultures Journal. An updated version of the file may be obtained directly from the Department of Anthropology, York College, CUNY Jamaica, NY USA. In this current study the original dataset had been reduced to 34 populations so as to include only populations for which body and brain size estimates had been recorded. Measures of cranial capacity were converted to brain mass by using the

equation derived by Spocter *et al*, (2007). In addition the two samples used in the population comparison from this study (i.e. African and European samples) were added to the Beals *et al*. (1984) dataset and correlation coefficients were computed for brain mass against body mass, height and climatic zone.

4.3 Results

While several findings of interest emerged from the current study, those most noteworthy include our findings that: (1) the average human brain mass is 1412 g; (2) there are no differences in brain mass between population groups investigated; and (3) once body mass is corrected for, providing an encephalization quotient (EQ), there are no apparent differences between the sexes.

A study using a database such as the one used herein may produce numerous results however in the current study only those results which have direct implications towards the understanding of brain and body size variability in *Homo sapiens* are reported and discussed here. For a more detailed outline of the results obtained from this investigation the accompanying Tables can be consulted.

4.3.1 Brain mass, body mass and height variation for males, females and population groups

4.3.1.1 Brain mass descriptives

When looking at the 18-60 year cohort, the mass of the average human brain was found to be approximately 1412 g and ranged between 960 g to 2033 g (Table 16). The standard error in brain mass for_Ôhis sample was 1.46 g and was associated with a standard deviation (SD) of 153.31 g. When we compared the sexes of the 18-60 year cohort we found that males had on average larger brain masses than females,

with the mean male brain mass being approximately 1447.95 g while the comparable female average was 1302.14 g. Within the population groups investigated in the present study similar trends were observed. African males had a mean brain mass of 1376.23 g while their female counterparts had a mean brain mass of 1273.42 g. Males from the Hamburg European sample also had larger mean brain masses than females drawn from the same population (i.e. 1452.12 g as compared to 1302.63 g), while results from the South African European sample indicated an average male brain mass of 1425.63 g and an average female brain mass of 1346.1 g. Mean brain mass also varied significantly between the population groups (See Table 17) a result that on its own would erroneously argue in favour of the existence of sex and population differences in brain mass. However on investigation of the 95th percentile ellipses of body mass against brain mass, it became apparent that both sex and population differences were small, if any, for the sample under study (Figs. 14 – 16).

4.3.1.2 Body mass descriptives

Across the 18-60 year cohort mean human body mass was approximately 73.46 kilograms (kg) and ranged between 25 kg to 208 kg. This was accompanied by a standard error of 0.169 kg and a standard deviation of 17.75 kg. By and large as with brain mass there existed significant differences in the means and variances of body mass for the individual sex and population specific sub-samples (Table 17). Males were on average heavier than females, with the mean male body mass for the 18-60 year cohort being 76 kg whereas that for females was 65.67 kg. Within the population groups similar results were obtained, for example for the two European sub-samples males from Hamburg and South Africa had average body masses of 76.23 kg and 83.97 kg respectively whilst females from the two regions had average

body masses of 65.39 kg and 74.90 kg respectively. An exception to this trend was observed in the body mass of Africans where females were marginally heavier than their male counterparts (72 versus 70.30 kg).

4.3.1.3 Body height descriptives

The height of the average human as obtained from the 18-60 year cohort was 173.29 cm. Mean height ranges between 101-250 cm and was associated with a standard error and standard deviation of 0.09 cm and 9.19 cm respectively. Sexual dimorphism in height was apparent as revealed by a mean male body height of 175.97 cm as compared to 165.01 cm for females. Within the population groups, similar trends were observed as those displayed for the full 18-60 year cohort. African males were taller than African females (172.94 cm as compared to 164.78 cm for females) and European males from both Hamburg and South Africa were taller than their female counterparts (176.10 cm for males versus 165.04 cm for females; and 178.90 cm for males versus 172.05 cm for females from Europe and Africa respectively). Significant differences in the means and variances were found between the sexes for each population group, and between the populations groups when comparing the same sex (Table 17).

4.3.2 Regressional permutations between brain mass, body mass, height and age

4.3.2.1 Body mass versus brain mass regressions

The equation that defined the relationship between body mass and brain mass across the entire 18-60 year cohort was found to be:

$$\text{Brain mass (g)} = 8.63 \times \text{Body mass (kg)} + 777.76$$

$$(r = 0.28;\ P\ (uncorr) = 6.54\times10^{-194}). \quad (1)$$

The derived coefficient of determination demonstrates that only 7.84 % of the variation in brain mass can be accounted for by variation in body mass. For males and females from the 18-60 year cohort the equations that defined the relationships between body mass and brain mass were, for males:

$$\text{Brain mass (g)} = 8.50 \times \text{Body mass (kg)} + 801.96$$

$$(r = 0.22;\ P\ (uncorr) = 2.93\times10^{-91}). \quad (2)$$

and, for females:

$$\text{Brain mass (g)} = 7.03 \times \text{Body mass (kg)} + 840.35$$

$$(r = 0.13;\ P\ (uncorr) = 3.11\times10^{-11}). \quad (3)$$

Coefficients of determination were 4.84 % for males and 1.69 % for females. Similar results are observed for males and females of the individual population groups with *r* values ranging between 0.05, for the African female sample, and 0.33 for the South African European male sample. Despite the low *r* values across and within the sexes and population groups, all equations defining the relationship between body mass and brain mass, were associated with low probabilities of the two variables being uncorrelated. Results for the individual equations defining the relationship

between body mass and brain mass for the population groups are tabulated in Table 20.

When regression analyses are performed on the different age cohorts a clearer picture of the establishment of the correlation coefficients presented in Table 20 emerged (See Table 21). For phase 1 (0- 2.5 yrs of age) correlation coefficients between body mass and brain mass are high (r ~ 0.86) with approximately 74 % of the variability in brain mass being explained by the variability in body mass. The high regression statistics are maintained across the sexes and are also associated with low probabilities of the two variables being uncorrelated [P (uncorr) = 2.42×10^{-85} – 4.25×10^{-163}]. By phase 2, (3-5 yrs) the r values are of the order of 0.23-0.32 and are largely maintained into advanced ages beyond phase 2 (Table 20).

4.3.2.2 Height versus brain mass regressions

When regression analysis is performed to investigate the relationship between body height and brain mass across the entire sample of the 18-60 year cohort, the following equation is obtained:

$$\text{Brain mass (g)} = 16.68 \times \text{Height (cm)} + -1478$$

(r = 0.40; P *(uncorr)* = 0). (4)

As is evident from the above equation, the strength of relationship between height and brain mass is 0.40 and thus only 16 % of the variability in brain mass can be accounted for by the variability in height. For males and females of the 18-60 year cohort the respective equations defining the relationship between height and brain mass are:

Brain mass (g) = 18.00 × Height (cm) -1719.90

(r = 0.24; *P (uncorr)* = 1.45×10^{-105}). (5)

and

Brain mass (g) = 16.57 × Height (cm) -1433.60

(r = 0.29; *P (uncorr)* = 6.96×10^{-53}). (6)

Similar results are observed for males and females from the individual population groups with relatively low r values reported (Table 20). Despite the low r values most of the equations defining the relationship between height and brain mass, are associated with low probabilities of the two variables being uncorrelated. As with body mass, height is also strongly correlated with brain mass up until 2.5 years of age (r = 0.88) [Table 21]. The sex combined equation defining the relationship between height and brain mass in phase 1 for the European (Hamburg) sample is:

Brain mass (g) = 21.34 × Height (cm) - 653.80

(r = 0.88; *P (uncorr)* = 3.29×10^{-181}). (7)

By phase 3 the relationship between height and brain mass decreases in strength to an r value of 0.36 with a P value of 6.50×10^{-10} of the two variables being uncorrelated. Although differences exist for the individual regression statistics of males and females, both r values and probabilities are comparable and indicate the same trends between the age cohorts observed across the sexes.

4.3.2.3 Age versus brain mass regressions

When regression statistics are computed for age (years) against brain mass (See Table 22), low *r* values and negative slopes are observed across almost the entire 18-60 year cohort, though the probability of the two variables being uncorrelated still remains fairly low, with the only exception being that observed for the South African European male sample. The equation defining the relationship between age and brain mass for the sex and population combined 18-60 year cohort is:

$$\text{Brain mass (g)} = -12.98 \times \text{Age (Yrs)} + 1953$$

$$(r = 0.11;\ P\ (uncorr) = 2.07\times10^{-28}). \quad (8)$$

Age also displays low correlation coefficients with both body mass and height across the entire 18-60 year cohort (Table 22) with the equations defining the relationships between age and body mass and age and height for the sex and population combined sample being:

$$\text{Body mass (kg)} = 1.50 \times \text{Age (Yrs)} + 10.80$$

$$(r = 0.06;\ P\ (uncorr) = 1.87\times10^{-9}). \quad (9)$$

and

$$\text{Body height (cm)} = -0.78 \times \text{Age (Yrs)} + 205.72$$

$$(r = 0.16;\ P\ (uncorr) = 2.72\times10^{-66}). \quad (19¢$$

4.3.2.4 Slope comparisons and tests for heterogeneity in slope

A comparison of the slopes as defined by the regression equations for body mass versus brain mass and height versus brain mass revealed the following results (Table 19). Male and female slopes of brain mass versus body mass were significantly homogenous [*P (heterogeneity)* >0.05] in the South African European sample whereas all other sub-samples showed significant differences in slope between males and females [*P (hetero.) < 0.05*]. Further analysis also revealed the existence of significant similarities in male and female height versus brain mass regression slopes for the South African European and African sub-samples. Africans and Europeans displayed similarity in slope for body versus brain mass regression equations however significant heterogeneity was observed in the slopes defining height versus brain mass and body mass versus height regressions.

4.3.2.5 Sexual dimorphism in brain mass, body mass and height

When sexual dimorphism in brain mass is examined at different age cohorts within the population groups an interesting pattern becomes apparent (Table 18). In the first 2.5 years of life the amount of sexual dimorphism in brain mass is approximately 51 grams (i.e. a percentage sexual dimorphism of 6.7 %). This measure is then markedly increased during the next phases of life to reach the levels of between 102-150 grams (i.e. a percentage sexual dimorphism of between 8-12%) as displayed within the 18-60 age cohort.

Sexual dimorphism in body mass and height as with brain mass, also varies with age. In both body mass and height sexual dimorphism is low during the first 2.5 years of life and then increases to the levels observed in Phase 4. In the early phases sexual dimorphism in body mass is approximately 0.58 kg (8.8%), whilst for height a

sex difference of 1.13 cm (1.8%) is observed. By phase 4 these measures have increased to a 10 kg (16.6%) difference in body mass and an 11 cm (6.7%) difference between the sexes (See Table 18).

4.3.2.6 Lack of Sexual Dimorphism in Encephalization Quotient

Given the above described differences in the absolute mass of the brain between the two sexes, it was of interest to calculate the encephalization quotient (EQ) for each sex as described by Jerison (1973). To calculate EQ we used the following equations based on a generalized mammalian regression (not including primates and cetaceans) from Manger (2006):

$$(1)\ M_{br} = 0.069 M_b^{0.718}$$

and that from an unpublished equation derived from additional data encompassing approximately 800 mammalian species (Spocter and Manger, unpublished observations):

$$(2)\ M_{br} = 0.054 M_b^{0.741}$$

The resulting encephalization quotients for humans as a whole was 6.564 and 6.482 for both equations (1) and (2) respectively. For equation (1), the average male EQ was 6.569 and the average female EQ was 6.561, and for equation (2) the average male EQ was 6.482 and the average female EQ was 6.495. Despite the absolute differences in mass of both the brain and the body for the sexes, there was no discernable difference in the EQ of the sexes.

4.3.2.7 Testing heterochrony as an explanation for why females have smaller mean brain and body masses than males

In order to test for the effects of heterochrony on the sex difference exhibited between male and female brain and body masses, the use of mathematical transformation was performed on the growth curves for the female ontogenetic dataset using the procedures outlined by Rice (2002). This analysis involves a comparison of two growth curves using plots of residuals from the transformed and untransformed data points. In accordance with Rice (2002) two growth curves would be considered commensurate if the distributions of the residuals against age from each curve (i.e. the male growth curve and the transformed female growth curve for brain mass and body mass in this case) are shown to 'overlap' and are uniformally distributed about 0. This may also be stated as: the two untransformed growth curves and derived residual plots would display bimodality prior to transformation, but on transformation the growth curves and subsequent residual plots should overlap and prove positive using the runs test. The resultant plots from the current study are presented in Figure 19 A – H.

For brain mass, Figure 19A and 19B are indicative of the trends observed in the male and female growth curves prior to transformation. Note the bimodal distribution indicated by the separation of the curves where male data points lie above those of females. However, on transformation (as shown in Figure 19C and 19D) female data points are adjusted and overlap with their male counterparts.

For body mass similar results are found. Note once again the existence of a bimodal distribution in body mass prior to transformation (Figs. 19E,F) and subsequent overlap of male and female body masses on transformation (Figs. 19G,H).

Testing via the use of the runs test revealed that out of 134 points, the longest run of points on the same side of the regression line is 33 points for brain mass and 36 points for body mass. Since the probability of getting a run of 33 or more if the transformed female points were randomly drawn from the same distribution as the male points is < 0.05, we conclude that the difference between the male and female brain mass and body mass trajectories when investigated using the runs tests, is unlikely to be explained by sequential hypermorphosis of the male data points. Thus we can say that using the runs test, males do not appear to undergo a period of prolonged growth in brain mass or body mass relative to females.

However as is evident from the residual plots presented in Figures 19D and 19H, the male and female residuals are not symmetrically distributed about 0, thus violating an assumption of the technique used here. As an extension of this analysis, the Kolmogorov-Smirnov test was implemented to test whether there is any significant similarity between the untransformed male and transformed female data points. Results indicated that when the untransformed male and female brain and body mass growth curves are compared to one another, there exists a small probability of the male and female curves being commensurate [i.e. P (same) = 6.19×10^{-23} & 1.03×10^{-13}, for brain and body mass respectively] (See Table 23). However, when transformation of the female brain and body mass growth curves was performed, a significant similarity was achieved between it and the untransformed male growth curves (i.e. $P > 0.05$). Thus, in this case using the more appropriate Kolmogorov-Smirnov technique we can conclude that by transformation of the female growth curves to match that of males, significant similarity in growth trajectories is achieved and thus the size dimorphism exhibited between male and female brain and body masses may be attributed to an extended period of growth in males relative to females.

4.3.2.8 Causal modelling of human brain mass variation

4.3.2.8.1 Multiple regression analysis on the 18-60 year age cohort

When the simultaneous effects of age, body mass and height are used to explain brain mass variation for the sex and population combined sample (18-60 year age cohort), only 12% of the variation in brain mass is explained by the combinatory effects of these variables. Standardised regression coefficients indicate that body mass is the strongest contributor towards explaining this joint variation (i.e. when body mass goes up by one standard deviation brain mass goes up by 0.27 standard deviations, as compared to height which only results in a 0.16 increase in brain mass with every one increment in standard deviation. An increase in age by one standard deviation results in a decrease of brain mass by 0.11 standard deviations) (See Figure 20A). These results clearly indicate that a substantial amount of variation in brain mass still remains unaccounted for. Proponents of hierarchical models of intelligence would argue that sex and population affinity when factored in would dramatically increase the explanatory power and will assist in explaining a substantial component of the unexplained variation. However, when factored into the model, the combined effect of sex and population affinity, along with the remaining variables, only help to increase the explanatory power of the regression analysis by a further 6% (i.e. only 24% of the variation in brain mass is explained by the combined effects of body mass, age, height, sex, and population affinity) (Fig. 20B). This once again, argues against the utility of either sex or population affinity as having significant statistical power to explain a large component of brain mass variability. The low multiple correlation

coefficients for the 18-60 year cohort, lend themselves to the argument that a change may have been effected during development (i.e. a decoupling or relaxing of constraints on brain: body size), which resulted in a loss of explanatory power and a reduction in the amount of variation in brain mass explained by the combinatory effects of body mass, height and age.

4.3.2.8.2 Multiple regression analysis on the various age cohorts

To investigate the possibility of a change in explanatory power due to developmental changes, multiple regression analyses were performed on five age cohorts, the results of which are presented in Figure 21. Multiple regression analyses revealed, that the largest amount of brain mass variation (i.e. 84%) is explained by the combined effects of body mass, height and age during the first three years of life and that this explanatory power rapidly drops to below 20% between the ages of 6- 60 years only to rise marginally to a level of 24% between the ages of 61-100 years. During both the developmental phase (0-17years) and degenerative phase of life (61-100 years) height remains the major contributor towards explaining brain mass variation. Within the first five years of life an increase in height by one standard deviation results in a concomitant increase in brain mass. During the same time period however, body mass and age exert a negative effect on brain mass (i.e. with every increase in body mass or age, brain mass decreases). During the age cohort 6-17 years of age the relationship between body mass and brain mass changes. Body mass is now no longer inversely related to brain mass but rather an increase in body mass results in a subsequent increase in brain mass, a trend which continues into the final phase of

life (61- 100 age cohort). Age however almost always exerts a negative effect on brain mass, with brain mass decreasing with increases in age.

4.3.2.8.3 Potential 'causal' models

The use of multiple regression analysis revealed that the highest multiple coefficient of determination (i.e. 84%) is observed early during development (i.e. 0-3 years of age). Using this observation path analysis was used to derive potential 'causal' models for brain mass variation during the first 3 years of life. Numerous causal models were computed, many of which proved 'untestable' and were subsequently rejected. However, four models were both successfully fit and tested on the 0- 3 year age cohort. In all of the presented models $P > 0.05$, thus all four models significantly agree with the 0-3 year age cohort, but when tested on other age cohorts such as the 18-60 year cohort these models were rejected and deemed to depart significantly from the data, reiterating the effect of a change in the brain and body size scaling relationships. On the basis of the accompanying P values, the two best models as tested on the 0-3 year age cohort, were those with $P = 0.938$ as presented in Figure 22. In model 1, height has both direct and indirect effects on brain mass. The direct effect of height on brain mass indicates that when height goes up by one standard deviation, brain mass goes up by 1.045 standard deviations (See Table 23). The indirect effect of height on brain mass is affected *via* body mass and results in a decrease in brain mass by 0.134 standard deviations for every increment in height (Table 23). Body mass has a direct, inverse effect on brain mass of 0.14 standard deviations, whilst the indirect effects of age results in an increment of 0.771 standard deviations for every increment in age. Height and age jointly help to explain 83% of variation in body mass, whilst age explains 71% of the variation in height. These

results provide one of potentially numerous theoretical frameworks governing the control of brain mass during the first 3 years of life when maximum variation in brain mass can be accounted for by the body parameters of age, height and body mass.

4.3.3 The effects of climate on human brain mass

Climate has been invoked by numerous researchers as the prime causative agent in the expansion of the hominid brain and the variation displayed in human brain mass (e.g. Beals et al 1984). To retest this assertion a modified version of the Beals et al. (1984) dataset was combined with the data from this current study and correlation coefficients were computed to test which of climatic zone or body parameters explain more of the variation in brain mass. The Beals et al. (1984) dataset was modified by excluding all populations for which either cranial capacity or body mass were not available and this meant that in total data from 34 human populations were used in this analysis. In order to facilitate direct comparison all cranial capacities were converted to brain mass, whereafter the African and European (Hamburg) samples were included in the analyses.

Results indicate that although climatic zone is relatively highly correlated with brain mass across the human populations, the highest correlation is still between brain mass and body mass ($r = 0.67$; [p (uncorr) = 8.74×10^{-6}] while height [$r = 0.50$; p (uncorr) = 0.0025] is also more strongly correlated with brain mass than is climatic zone ($r = 0.43$; [p (uncorr) = 0.0093]). Climatic zone also shows relatively small correlation coefficients with body mass and height, though still significant (See Table 25). These results argue in favour of the consideration of body size parameters into explanations of human brain mass variation. Inclusion of climatic factors at the cost of

body parameters, may thus result in omission of the significant role played by body size constraints in our evolutionary history.

Table 16 Descriptive statistics for brain mass, body mass and height in three human population groups. N = sample size; Min = minimum value; Max = maximum value; Std. error = standard error; Stand. Dev = Standard deviation; COV = Coefficient of variation.

Parameter	Sample	Sex	N	Min	Max	Mean	Median	Std. error	Variance	Stand. Dev	COV
Brain mass	African	m	426	1022	1988	1376.23	1376.5	6.41765	17545.3	132.459	9.62
	African	f	77	1025	1868	1273.42	1252	17.0593	22408.5	149.695	11.76
	European	m	7786	960	2000	1452.12	1455	1.6294	20671.3	143.775	9.9
	European	f	2619	1000	1880	1302.63	1300	2.42846	15445.3	124.279	9.54
	SA. European	m	87	1037	2033	1425.63	1426	15.7996	21717.6	147.369	10.34
	SA. European	f	21	1105	1872	1346.1	1346	34.6602	25227.9	158.833	11.8
	Whole sample	m+f	11016	960	2033	1411.99	1410	1.46067	23503.1	153.307	10.86
Body mass	African	m	426	25	179	70.3404	68	0.723472	222.973	14.9323	21.23
	African	f	77	42	173	72.6494	68	2.63064	532.862	23.0838	31.77
	European	m	7786	40	208	76.2301	75	0.192756	289.287	17.0084	22.31
	European	f	2619	24.5	200	65.3862	63	0.34387	309.688	17.5979	26.91
	SA. European	m	87	48	140	83.9655	84	1.77236	273.289	16.5315	19.69
	SA. European	f	21	53	137	74.9048	73	4.18552	367.89	19.1805	25.61
	Whole sample	m+f	11016	24.5	208	73.4578	72	0.169187	315.324	17.7574	24.17
Height	African	m	426	101	196	172.944	173.5	0.442674	83.479	9.13668	5.28
	African	f	77	149	185	164.779	165	0.935117	67.3322	8.20562	4.98
	European	m	7785	107	250	176.103	176	0.089548	62.4273	7.9011	4.49
	European	f	2619	113	195	165.042	165	0.146971	56.5714	7.5214	4.56
	SA. European	m	87	145	198	178.897	180	0.926608	74.6985	8.64283	4.83
	SA. European	f	21	160	189	172.048	169	1.83935	71.0476	8.42897	4.9
	Whole sample	m+f	11015	101	250	173.286	174	0.087585	84.4968	9.19222	5.3

Table 17 Results from the significance tests of means and variances for male versus female and African versus European population groups. f = female; m = male

Parameter	Sample	Sex	F Test	t Test
Brain mass	European	m vs f	p(equal) = 3.01*10 -19	p(equal) = 0.0
	African	m vs f	p(equal) = 0.142	p(equal) = 1.62*10-7
	SA.European	m vs f	p(equal) = 6.14*10-1	p(equal) = 0.046
	African vs European	Comb.	p(equal) = 0.001	p(equal) = 0.001
Body mass	European	m vs f	p(equal) = 3.13*10-2	p(equal) = 0.0
	African	m vs f	p(equal) = 4.34*10-8	p(equal) = 4.00*10 -1
	SA.European	m vs f	p(equal) = 3.47*10-1	p(equal) = 0.056
	African vs European	Comb.	p(equal) = 0.74	p(equal) = 0.05
Height	European	m vs f	p(equal) = 2.27*10-3	p(equal) = 0.0
	African	m vs f	p(equal) = 2.51*10-1	p(equal) = 0.0
	SA.European	m vs f	p(equal) = 9.46*10-1	p(equal) = 0.0023
	African vs European	Comb.	p(equal) = 0.0	p(equal) = 0.40

Table 18 Absolute sexual dimorphism in brain mass, body mass and height for the African and European (Hamburg) sample taken at various age cohorts where available. Note that values in parentheses () are the calculated percentage sexual dimorphism calculated as: Percentage seã·al dimorphism = [(Mean male measure – mean female measure) / mean female measure] * 100.

		Sexual Dimorphism				
		Phase 1	**Phase 2**	**Phase 3**	**Phase 4**	**Phase 5**
Parameter	**Sample**	**(0y-2.5y)**	**(3y-5y)**	**(6y-17y)**	**(18 y- 60 y)**	**(61 y-100 y)**
Brain mass (g)	African	-	-	-	102.81 (8.07%)	-
	European	51.06 (6.69%)	44.77 (3.42%)	143.45 (10.97%)	149.49 (11.48%)	148.43 (12.08%)
Body mass (kg)	African	-	-	-	2.31 (3.18%)	-
	European	0.58 (8.75%)	2.45 (13.06%)	8.47 (17.31%)	10.84 (16.58%)	15.6 (38.71%)
Height (cm)	African	-	-	-	8.16 (4.96%)	-
	European	1.13 (1.77%)	0.49 (0.45%)	9.31 (6.04%)	11.06 (6.7%)	15.25 (10.15%)

Table 19 Slope comparisons between male and female and African versus European population groups. Euro = European (Hamburg) sample; Afric = African population group; m = male; f = female; P = probability; LowCI = lower confidence interval; UppCI = upper confidence interval; Com slope = common slope; P (Hetero) = probability of the slopes being heterogeneous.

						X			Y		Slope test	
Parameter	**Sample**	**Sex**	**slope**	**R 2**	**P**	**LowCI**	**UppCI**	**Inter.**	**LowCI**	**UppCI**	**Com.slope**	**P (Hetero)**
Body vs Brain Mass	Euro.	m	8.45	0.05	0	8.27	8.64	807.7	793.2	822.3	8.09	*P=0.001*
		f	7.06	0.02	0	6.8	7.34	840.9	822.2	859.5		
	SA Euro.	m	8.28	0.06	0.3	5.28	13	725.8	423.1	1028.6	8.79	*P=0.762*
		f	8.91	0	0.11	7.28	10.91	677.1	520.3	833.9		
	Afric. vs Euro.	m	-6.49	0	0.68	-8.14	-5.16	1744.5	1626.6	1862.5	8.47	*P=0.018*
		f	8.87	0.03	0	8.08	9.74	752.3	691.4	813.1		
	Afric. vs Euro.	Afric.	8.52	0.01	0.02	7.81	9.3	758	702.9	813.1	8.62	*P=0.794*
		Euro.	8.63	0.08	0	8.47	8.79	780.3	768.1	792.5		
Height vs Brain Mass	Euro.	m	18.2	0.06	0	17.81	18.59	-1752	-1822	-1683	17.75	*P=0.001*
		f	16.52	0.08	0	15.93	17.14	-1424	-1525	-1324		
	SA Euro.	m	17.05	0	0.56	13.77	21.11	-1625	-2283	-967	17.44	*P=0.665*
		f	18.84	0.17	0.06	12.33	28.79	-1896	-3314	-478		
	Afric. vs Euro.	m	14.51	0.03	0	13.21	15.94	-1133	-1370	-896	15.03	*P=0.068*
		f	18.24	0.03	0.12	14.58	22.83	-1733	-2414	-1051		
	Afric. vs Euro.	Afric.	14.8	0.06	0	13.59	16.12	-1181	-1399	-964	16.67	*P=0.005*
		Euro.	16.75	0.17	0	16.46	17.05	-1489	-1540	-1438		
Body mass vs Height	Euro.	m	0.46	0.11	0	0.45	0.47	140.7	139.9	141.5	0.46	*P=0.001*
		f	0.43	0.07	0	0.41	0.44	137.1	136	138.2		
	SA Euro.	m	0.52	0.15	0	0.43	0.64	135	126.1	143.9	0.49	*P=0.309*
		f	0.44	0.57	0	0.32	0.6	139.1	128.4	149.8		
	Afric.	m	1.63	0.1	0	1.49	1.79	-212.3	-238	-186.6	1.77	*P=0.001*
		f	2.81	0.1	0.01	2.27	3.49	-390.9	-492.4	-289.5		
	Afric. vs Euro.	Afric.	0.58	0.07	0	0.53	0.63	131	127.4	134.6	0.52	*P=0.009*
		Euro.	0.52	0.16	0	0.51	0.52	135.5	134.8	136.2		

Table 20 Results from reduced major axis analysis (RMA) of body mass versus brain mass; height versus brain mass; body mass versus height and lean body mass versus brain mass. Lean body mass = [1] was calculated using the equation derived by Hume (1966). According to Hume (1966): For men over the age of 16: lean body mass in kilograms = (0.32810 * (body weight in kilograms)) + (0.33929 * (height in centimeters)) - 29.5336, whereas for women over the age of 30: lean body mass in kilograms = (0.29569 * (body weight in kilograms)) + (0.41813 * (height in centimeters)) - 43.2933.

X	Y	Population	Sex	*r* - value	Slope	Y-int.	95% on slope	95% on Int.	P(uncorr)
Body mass	Brain mass	European	f	0.14	7.06	840.86	[6.59; 7.59]	[808.3; 868.5]	3.11*10-12
Body mass	Brain mass	European	m	0.21	8.45	807.72	[8.16; 8.77]	[784.8; 829]	1.14*10-81
Body mass	Brain mass	African	f	-0.05	-6.48	1744.5	[-9.411; 7.81]	[722.6; 1949]	0.68
Body mass	Brain mass	African	m	0.17	8.87	752.27	[7.25; 10.84]	[622.1; 857]	0
Body mass	Brain mass	SA European	f	0.24	8.28	725.81	[2.93; 18.02]	[74.34; 1100]	0.3
Body mass	Brain mass	SA European	m	0.33	8.91	677.13	[6.28; 11.99]	[425.2; 887.6]	0
Body mass	Brain mass	Whole sample	f	0.13	7.03	840.35	[6.55; 7.57]	[808.5; 868.2]	3.11*10-11
Body mass	Brain mass	Whole sample	m	0.22	8.5	801.96	[8.19; 8.81]	[779.8; 823.5]	2.93*10-91
Body mass	Brain mass	Whole sample	m&f	0.28	8.63	777.76	[8.39; 8.89]	[759.8; 795]	6.54*10-194
Height	Brain mass	European	f	0.29	16.52	-1424.4	[15.75; 17.32]	[-1556; -1298]	1.67*10-51
Height	Brain mass	European	m	0.24	18.2	-1752.5	[17.58; 18.82]	[-1863; -1643]	1.73*10-98
Height	Brain mass	African	f	0.18	18.24	-1732.6	[-22.9; 22.99]	[-2504; 5066]	0.12
Height	Brain mass	African	m	0.17	14.5	-1131	[11.81; 17.76]	[-1705; -669.4]	0
Height	Brain mass	SA European	f	0.42	18.84	-1895.9	[9.12; 32.65]	[-4240; -241.3]	0.06
Height	Brain mass	SA European	m	0.06	17.05	-1624.7	[-19.34; 22.52]	[-2631; 4882]	0.56
Height	Brain mass	Whole sample	f	0.29	16.57	-1433.6	[15.8; 17.37]	[-1566; -1306]	6.96*10-53
Height	Brain mass	Whole sample	m	0.24	18	-1719.9	[17.37; 18.63]	[-1831; -1609]	1.45*10-105
Height	Brain mass	Whole sample	m&f	0.4	16.68	-1478.2	[16.27; 17.07]	[-1548; -1409]	0
Body mass	Height	European	f	0.27	0.43	137.1	[0.3978;0.4593]	[135; 138.9]	1.4712*10-43
Body mass	Height	European	m	0.33	0.46	140.69	[0.4461;0.4848]	[139.2; 142.1]	2.4668*10-200
Body mass	Height	African	f	0.31	2.81	-390.9	[2.062;3.76]	[-5.46.2; -271.9]	0.01
Body mass	Height	African	m	0.31	0.61	129.9	[0.48866; 0.774]	[118.8; 138.6]	7.6477*10-11
Body mass	Height	SA European	f	0.76	0.44	139.13	[0.327;0.7159]	[120.4;147]	7.3939*10-5
Body mass	Height	SA European	m	0.39	0.52	135	[0.4071;0.6708]	[122.3;145.2]	0
Body mass	Height	Whole sample	f	0.27	0.42	137.22	[0.3966;0.4555]	[135.3;139]	9.8943*10-48
Body mass	Height	Whole sample	m	0.38	0.47	140.08	[0.4532;0.49]	[138.7; 141.5]	2.3361*10-219
Body mass	Height	Whole sample	m&f	0.4	0.53	135.26	[0.5024; 0.5336]	[134.1; 136.3]	0
Lean Body mass[1]	Brain mass	European	f	0.24	18.39	474.1	[17.37; 19.42]	[428.5;518.1]	2.5869*10-35
Lean Body mass[1]	Brain mass	European	m	0.26	20.69	309.29	[20.1; 21.31]	[276.2; 341,6]	2.2024*10-123
Lean Body mass[1]	Brain mass	African	f	0.03	17.52	448.6	[-23.55; 21.54]	[277.4; 2386]	0.77
Lean Body mass[1]	Brain mass	African	m	0.21	20.2	321.12	[17.11; 24.23]	[108.3; 479.9]	1.4258*10-5
Lean Body mass[1]	Brain mass	SA European	f	0.33	18.36	413.31	[7.858;36.74]	[-433.4;916.7]	0.01
Lean Body mass[1]	Brain mass	SA European	m	0.28	20.75	207.54	[14.61; 27.85]	[-202.8; 558.9]	0.01
Lean Body mass[1]	Brain mass	Whole sample	f	0.23	18.31	475.63	[17.22; 19.27]	[433.8; 521.9]	4.216*10-34
Lean Body mass[1]	Brain mass	Whole sample	m	0.27	20.69	307.66	[20.13; 21.27]	[276.8; 338]	4.4877*10-136
Lean Body mass[1]	Brain mass	Whole sample	m&f	0.42	18.79	422.92	[18.43; 19.16]	[403.2; 442]	0

Table 21 Results from reduced major axis analysis (RMA) of body mass versus brain mass and height versus brain mass for the European (Hamburg) sample taken at various age cohorts. Note the high correlation coefficients in the first 2.5 years of life followed by a rapid reduction to the levels observed in the adult population.

Age cohort	X	Y	Sex	*r* - value	Slope	Y-int.	95% on slope	95% on Int.	*P* (uncorr)
Phase 1 (0-2.5yrs)	log Body mass	log Brain mass	f	0.91	0.78	2.22	[0.73; 0.83]	[2.18; 2.26]	2.42*10-85
	log Body mass	log Brain mass	m	0.83	0.83	2.17	[0.76; 0.90]	[2.11; 2.22]	3.44*10-85
	log Body mass	log Brain mass	m&f	0.86	0.81	2.19	[0.76; 0.86]	[2.16; 2.23]	4.25*10-163
	Height	Brain mass	f	0.93	20.93	-635.1	[19.67; 22.36]	[-718.7; -558.8]	4.46*10-92
	Height	Brain mass	m	0.86	21.54	-661.44	[19.81; 23.05]	[-760.9; -546.3]	3.79*10-98
	Height	Brain mass	m&f	0.88	21.34	-653.8	[20.17; 22.47]	[-727.3; -576.2]	3.29*10-181
Phase 2 (3-5yrs)	log Body mass	log Brain mass	f	0.32	0.32	2.69	[-0.19; 0.64]	[2.29; 3.34]	0.18
	log Body mass	log Brain mass	m	0.23	0.58	2.36	[-0.46; 1.22]	[1.56; 3.70]	0.13
	log Body mass	log Brain mass	m&f	0.25	0.51	2.45	[0.18; 0.89]	[1.97; 2.83]	0.05
	Height	Brain mass	f	0.16	9.68	174.73	[-10.33; 20]	[-865.1; 2384]	0.5
	Height	Brain mass	m	0.25	12.12	-10.28	[-11.18; 27.15]	[-1555; 2536]	0.1
	Height	Brain mass	m&f	0.23	11.73	8.21	[-10.47; 21.05]	[-948.7; 2454]	0.07
Phase 3 (6-17yrs)	Body mass	Brain mass	f	0.26	7.63	935.31	[6.09; 9.19]	[852.1; 1021]	0.01
	Body mass	Brain mass	m	0.34	7.4	1045.6	[5.86; 9.16]	[948.2; 1129]	6.12*10-6
	Body mass	Brain mass	m&f	0.37	8.23	961.71	[6.97; 9.59]	[889.2; 1029]	2.59*10-10
	Height	Brain mass	f	0.25	7.19	200.33	[5.86; 9.02]	[-99.1; 419.6]	0.01
	Height	Brain mass	m	0.33	7.19	295.84	[5.82; 8.82]	[20.32; 521.3]	1.54*10-5
	Height	Brain mass	m&f	0.36	7.9	144.17	[6.91; 9.11]	[-63.79; 307.3]	6.50*10-10
Phase 4 (18-60yrs)	Body mass	Brain mass	f	0.13	7.03	840.35	[6.55; 7.58]	[807.8; 868.3]	3.11*10-11
	Body mass	Brain mass	m	0.22	8.5	801.96	[8.209; 8.79]	[780.4; 822.5]	2.93*10-91
	Body mass	Brain mass	m&f	0.28	8.63	777.76	[8.38; 8.89]	[760.3; 795.4]	6.54*10-194
	Height	Brain mass	f	0.29	16.52	-1424.4	[15.75; 17.32]	[-1556; -1298]	1.67*10-51
	Height	Brain mass	m	0.24	18.2	-1752.5	[17.58; 18.82]	[-1863; -1643]	1.73*10-98
	Height	Brain mass	m&f	0.41	16.75	-1488.6	[16.37; 17.14]	[-1557; -1423]	0
Phase 5 (61-100yrs)	Body mass	Brain mass	f	0.17	7.11	811.72	[6.78; 7.44]	[793.2; 831.1]	5.84*10-23
	Body mass	Brain mass	m	0.2	7.57	847	[7.19; 7.96]	[819.3; 873.3]	3.09*10-35
	Body mass	Brain mass	m&f	0.32	8.18	776.1	[7.91; 8.44]	[759; 793.3]	1.94*10-169
	Height	Brain mass	f	0.23	13.27	-859.95	[11.87; 14.8]	[-111; -637.1]	4.53*10-41
	Height	Brain mass	m	0.19	12.55	-744.14	[11.32; 14]	[-997.6; -533.5]	2.83*10-34
	Height	Brain mass	m&f	0.43	12.75	-778.03	[12.05; 13.49]	[-901.4; -662.1]	1.02*10-311

Table 22 Results from reduced major axis analysis (RMA) of age versus brain mass, age versus body mass and age versus height for the three populations taken in the 18-60 age cohort. Note the overall low correlation coefficients across the sub-samples.

X	Y	Population	Sex	*r* - value	Slope	Y-int.	95% on slope	95% on Int.	*P* (uncorr)
Age	Brain mass	European	f	-0.14	-10.18	1730.7	[-10.55; -9,83]	[1713; 1749]	3.91*10-12
Age	Brain mass	European	m	-0.13	-12.38	1973.3	[-12.62; -12.13]	[1962; 1984]	4.06*10-31
Age	Brain mass	African	f	-0.17	-12.5	1710.1	[-16.19; 10.85]	[880; 1850]	0.13
Age	Brain mass	African	m	-0.12	-14.33	1854.3	[-16.06; -12.52]	[1789; 1913]	0.01
Age	Brain mass	SA European	f	0.18	13.55	813.79	[-13.83; 20.28]	[554.2; 1887]	0.43
Age	Brain mass	SA European	m	-0.03	-12.99	1942.2	[-16.37; 16.04]	[796.5; 2093]	0.77
Age	Brain mass	Whole sample	f	-0.13	-10.24	1730.4	[-10.62; -9.88]	[1713; 1750]	1.28*10-11
Age	Brain mass	Whole sample	m	-0.11	-12.36	1962.7	[-12.60; -12.13]	[1951; 1973]	1.20*10-22
Age	Brain mass	Whole sample	m&f	-0.11	-12.98	1953	[-13.19; -12.78]	[1943; 1963]	2.07*10-28
Age	Body mass	European	f	0.06	1.44	4.78	[1.35; 1.54]	[0.84; 8.40]	0
Age	Body mass	European	m	0.05	1.46	14.58	[1.41; 1.51]	[12.72; 16.68]	4.27*10-5
Age	Body mass	African	f	0.29	1.93	5.31	[1.41; 2.51]	[-12.75; 20.83]	0.01
Age	Body mass	African	m	0.11	1.62	16.44	[1.27; 1.96]	[5.74; 26.89]	0.03
Age	Body mass	SA European	f	0.04	1.64	10.62	[-2.66; 2.22]	[-7.73; 180.2]	0.87
Age	Body mass	SA European	m	0.06	1.46	26.02	[-1.69; 1.76]	[13.97; 152.4]	0.58
Age	Body mass	Whole sample	f	0.06	1.46	4.77	[1.36; 1.55]	[1.13; 8.54]	0
Age	Body mass	Whole sample	m	0.06	1.45	15.44	[1.41; 1.50]	[13.49; 17.3]	5.20*10-8
Age	Body mass	Whole sample	m&f	0.06	1.5	10.8	[1.46; 1.55]	[9.15; 12.47]	1.87*10-9
Age	Height	European	f	-0.22	-0.62	190.95	[-0.64; -0.59]	[189.8; 192.2]	2.01*10-29
Age	Height	European	m	-0.2	-0.68	204.74	[-0.70; -0.66]	[203.8; 205.7]	2.50*10-73
Age	Height	African	f	0.08	0.68	140.84	[-0.76; 0.79]	[136.9; 191.7]	0.47
Age	Height	African	m	-0.07	-0.99	205.92	[-1.20; 0.92]	[142.5; 212.4]	0.14
Age	Height	SA European	f	-0.11	-0.72	200.3	[-1.049; 0.77]	[140.6; 213]	0.63
Age	Height	SA European	m	-0.14	-0.76	209.19	[-0.96; 0.73]	[150.7; 217.2]	0.2
Age	Height	Whole sample	f	-0.21	-0.62	190.93	[-0.64; -0.59]	[189.8; 192.1]	1.18*10-27
Age	Height	Whole sample	m	-0.18	-0.69	204.57	[-0.71; -0.67]	[203.6; 205.5]	3.47*10-60
Age	Height	Whole sample	m&f	-0.16	-0.78	205.72	[-0.79; -0.76]	[205; 206.4]	2.72*10-66

Table 23 Results from the Kolmogorov Smirnov test of the untransformed and transformed male and female growth curves. Prior to transformation the untransformed male and female brain mass and body mass growth curves display a small probability of being equal (i.e. $P<0.05$) . However on transformation of the female dataset, both brain mass and body mass distributions display significant similarity, as is indicated by $P>0.05$.

These results indicate that sequential hypermorphosis of the female growth curves and a prolonging of male growth is responsible for the sexual size dimorphism observed between human males and females. Utr. Male Brm = untransformed male brain mass; Utr. Fem. Brm = untransformed female brain mass; Tr. Male Brm = transformed male brain mass; Tr. Fem Brm = transformed female brain mass; Utr. Male BM = untransformed male body mass; Utr. Fem. BM = untransformed female body mass; Tr. Male BM = transformed male body mass; Tr. Fem Bm = transformed female body mass.

Male Curve	**Female Curve**	**D statistic**	***P (same)***
Utr. Male Brm	Utr. Fem. Brm	0.61194	6.19?10-23
Utr. Male Brm	Tr. Fem. Brm	0.13433	0.16451
Utr. Male BM	Utr. Fem. BM	0.47015	1.03?10-13
Utr. Male BM	Tr. Fem. BM	0.14925	0.091617

Table 24 Summation of path analysis results for Model 1 used to explain the variation in human brain mass in the 0-3 year age cohort. RW = regression weights; Std RW = standardised regression weights; Imp Cor = implied correlations.

Regression Wts.		RW	Std.RW	Imp.Cor.
Height	Age	15.425	0.843	0.8
Body mass	Height	0.288	0.928	0.91
Body mass	Age	-0.125	-0.022	0.8
Brain mass	Height	20.301	1.045	0.913
Brain mass	Body mass	-9.052	-0.145	0.806
Total Effects		**Age**	**Height**	**Body mass**
	Height	15.425	0	0
	Body mass	4.317	0.288	0
	Brain mass	274.08	17.695	-9.052
Direct Effects	**Height**	15.425	0	0
	Body mass	-0.125	0.288	0
	Brain mass	0	20.301	-9.052
Indirect Effects	**Height**	0	0	0
	Body mass	4.441	0	0
	Brain mass	274.08	-2.606	0
Std.Total Effects	**Height**	0.843	0	0
	Body mass	0.761	0.928	0
	Brain mass	0.771	0.911	-0.145
Std.Direct Effects	**Height**	0.843	0	0
	Body mass	-0.022	0.928	0
	Brain mass	0	1.045	-0.145
Std.Indirect Effects	**Height**	0	0	0
	Body mass	0.783	0	0
	Brain mass	0.771	-0.134	0

Table 25 Matrix of correlation coefficients and probabilities comparing the strength of relationship between brain mass, body mass, height and climatic zone. Note that although climatic zone is relatively highly correlated with brain mass across the human populations, the highest correlation is still between brain mass and body mass ($r = 0.67$; [p (uncorr) = 8.74×10^{-6}] while height [$r = 0.50$; p (uncorr) = 0.0025] is also more strongly correlated with brain mass than is climatic zone ($r = 0.43$; [p (uncorr) = 0.0093]). P (uncorr) = probability of being uncorrelated; r = Pearsons correlation coefficient.

***P* (uncorr)**

r **Variable**	Brain mass	Body mass	Height	Climatic Zone
Brain mass	**0**	0.000006	0.002067	0.008119
Body mass	0.67	**0**	0.000002	0.034435
Height	0.49	0.69	**0**	0.662266
Climatic Zone	0.43	0.35	-0.07	**0**

Table 26 Comparison of average brain sizes reported in the literature from human populations. The 'weighted' mean for human brain size as calculated from the literature is 1413.18 g and is obtained by using the means and sample sizes reported in the following studies: Borowska & Golachowska, 1935; Milicer, 1955; Strzalko, 1974; Holloway, 1980 and Henneberg *et al.,* 1985. When the 'weighted' mean is recalculated using the dataset presented in this study, the average brain size for humans is 1412.14 g. CC = cranial capacity; Std. Dev = standard deviation.

Study	Measure	Method	Sample/Population size	Mean	Variance	Std.Dev
Borowska & Golachowska, 1935	Cranial capacity	Individual	268	1481.9	17822	133.5
Milicer, 1955	Cranial capacity	Individual	63	1169.3	85661	92.6
Strzalko,1974	Brain mass	Individual	254	1367.1	19500.4	139.6
Holloway, 1980	Brain mass	Individual	667	1387.6	17916.4	133.9
Beals et al., 1984	Cranial capacity	mean of means	122 populations	1349.3	12681.7	112.6
Henneberg et al., 1985	Cranial capacity	Individual	302	1498.3	23478.7	153.2
Henneberg, 1990	CC & Brain Mass	mean of means	6 previous studies	1350	24550	157
This current study	Brain mass	Individual	11000	1411.99	23503.1	153.31

Figure 14 **A**. Scatter plot of brain mass (g) versus body mass (g) for the whole male sample. **B**. Scatter plot of brain mass (g) versus body mass (g) for the whole female sample. **C**. 95th percentile ellipses of the male and female samples drawn from the 18-60 year age cohort and constructed from the bivalent plot of brain mass (g) against body mass (g). Note the pronounced overlap between males and females with the male ellipse only marginally above that of the females.

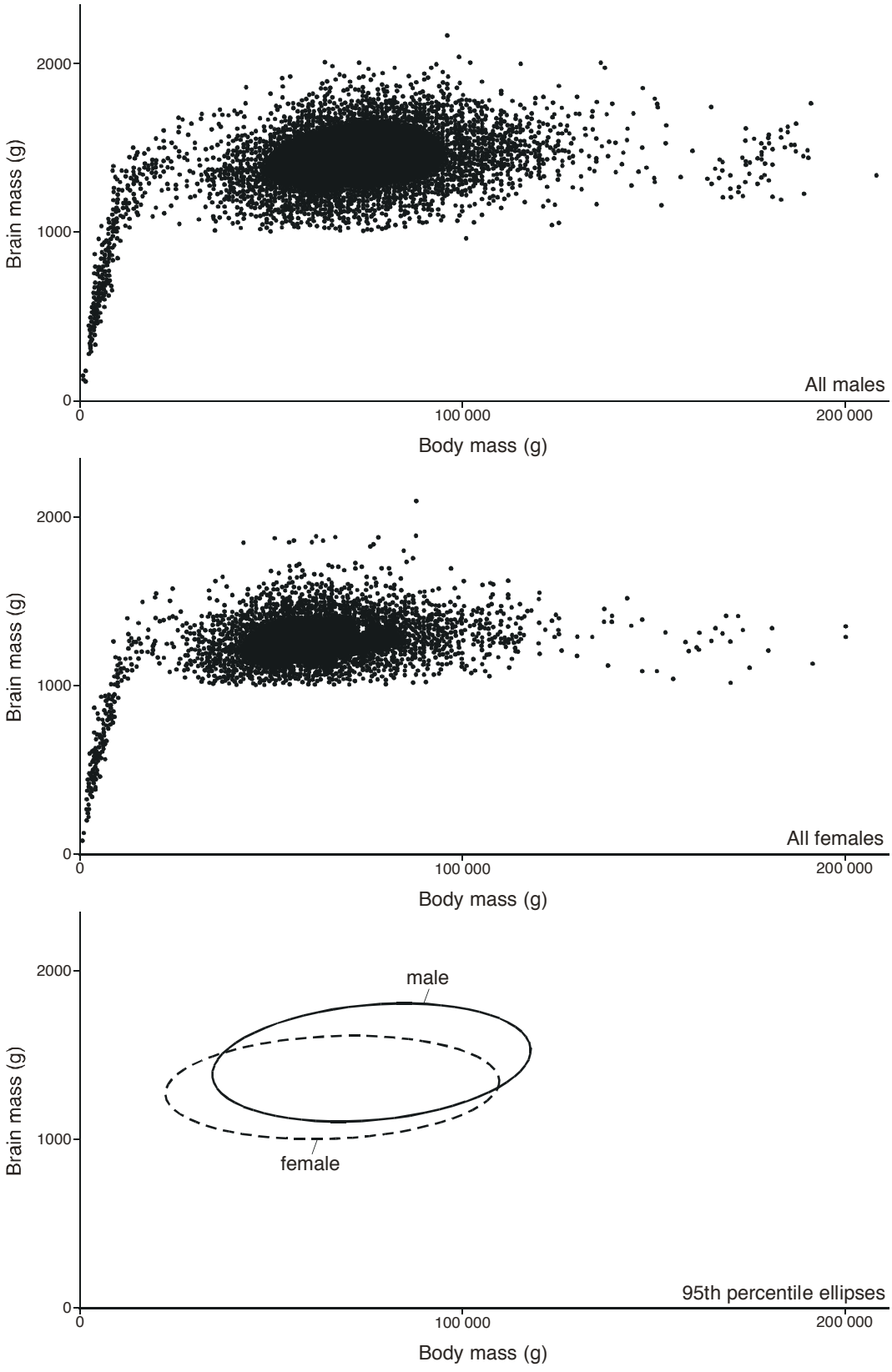
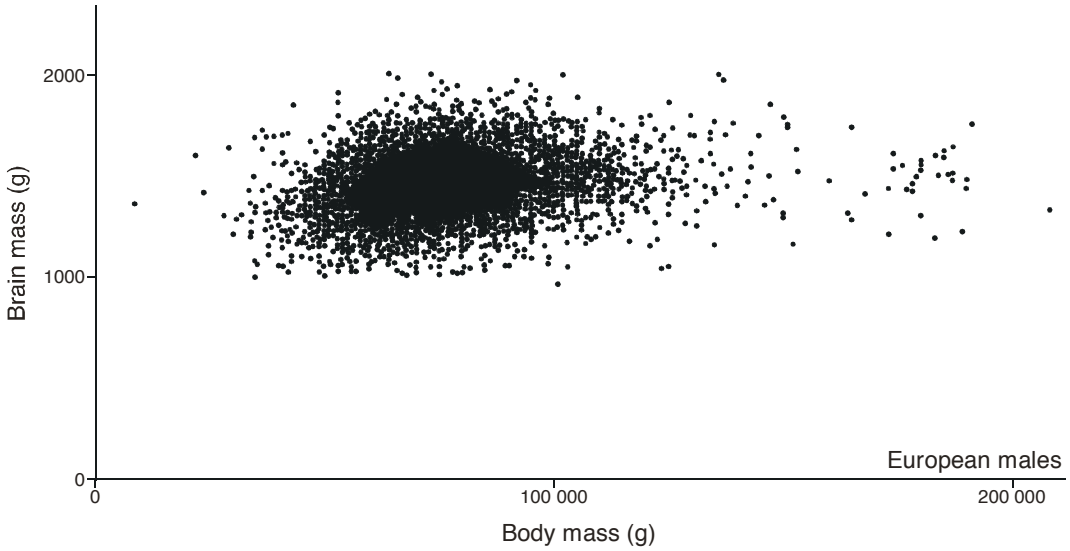
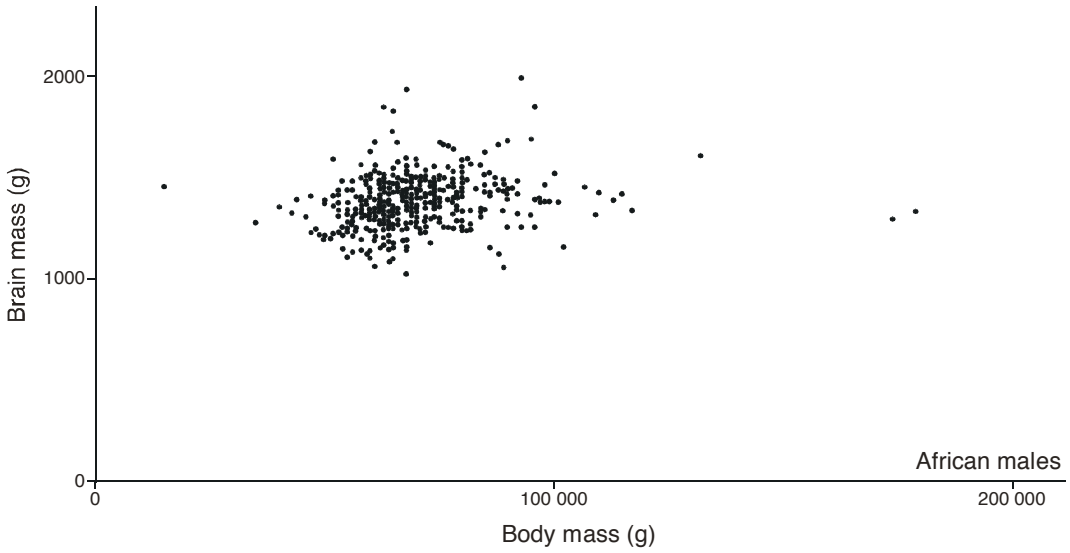

Brain mass (g)
2000
1000
0
All males
0
100 000
200 000
Body mass (g)
Brain mass (g)
2000
1000
0
All females
0
100 000
200 000
Body mass (g)
Brain mass (g)
2000
1000
0
male
female
95th percentile ellipses
0
100 000
200 000
Body mass (g)

Figure 15 **A**. Scatter plot of brain mass (g) versus body mass (g) for the whole European (Hamburg) male sample. **B**. Scatter plot of brain mass (g) versus body mass (g) for the whole African male sample. **C**. 95th percentile ellipses of the European (Hamburg) male and African male samples drawn from the 18-60 year age cohort and constructed from the plot of brain mass (g) against body mass (g). Note the pronounced overlap between the population groups with the European ellipse only marginally above that of the African sample.


2000
1000
0
Brain mass (g)
European males
0
100 000
200 000
Body mass (g)


2000
1000
0
Brain mass (g)
African males
0
100 000
200 000
Body mass (g)

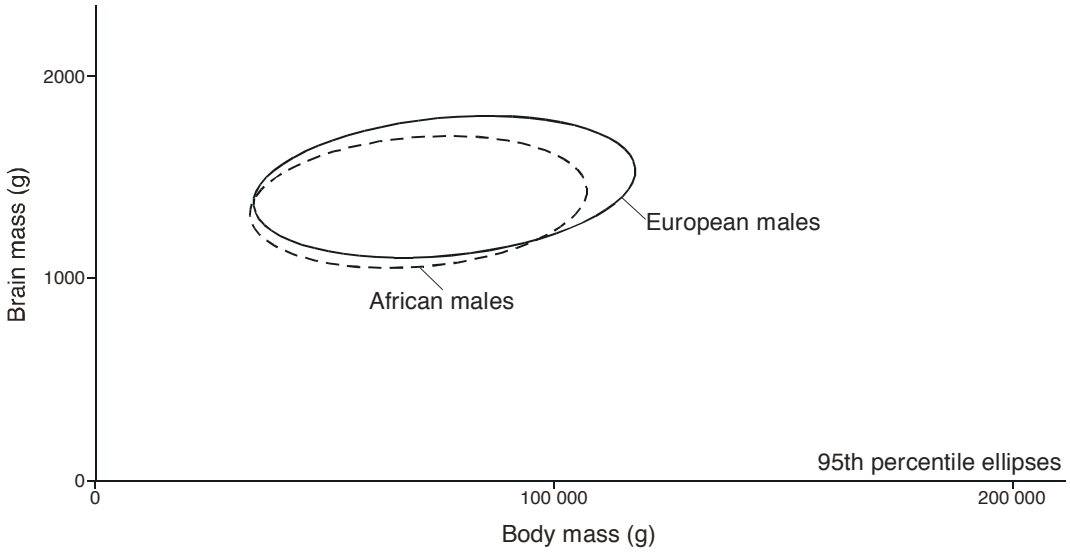
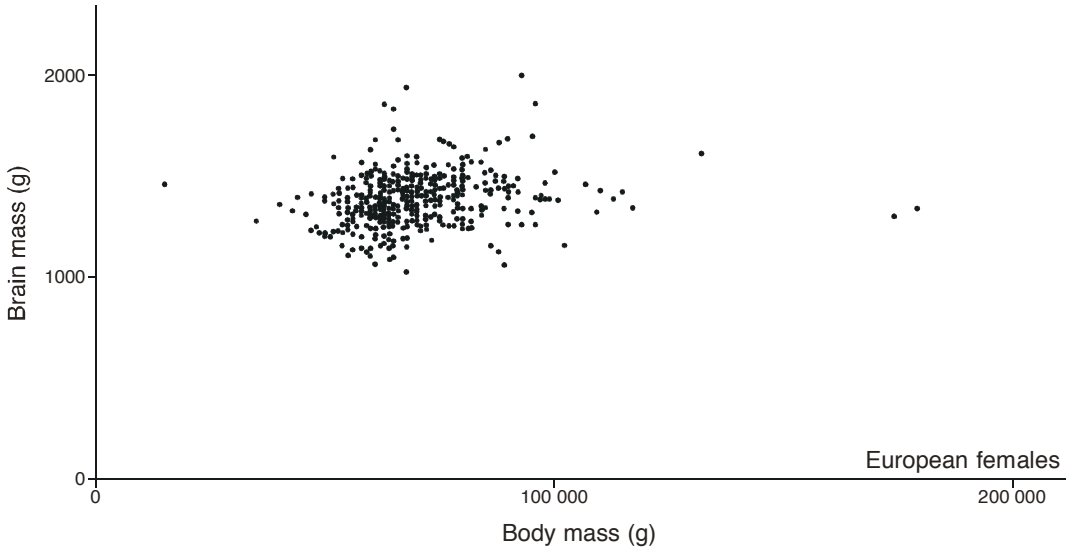
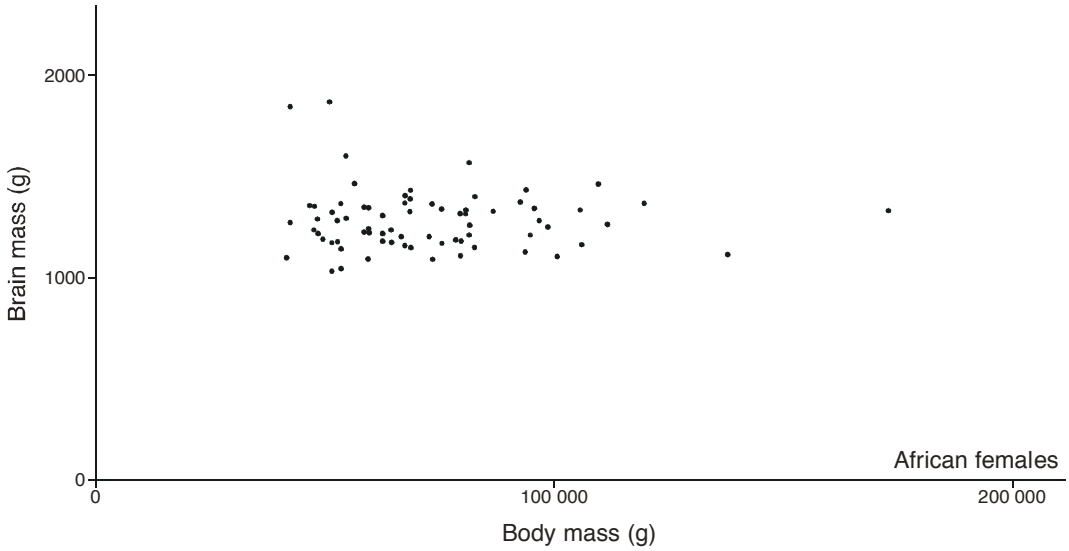

2000
1000
0
Brain mass (g)
European males
African males
95th percentile ellipses
0
100 000
200 000
Body mass (g)

Figure 16 **A**. Scatter plot of brain mass (g) versus body mass (g) for the whole European (Hamburg) female sample. **B**. Scatter plot of brain mass (g) versus body mass (g) for the whole African female sample. **C**. 95th percentile ellipses of the European (Hamburg) female and African female samples drawn from the 18-60 year age cohort and constructed from the bivalent plot of brain mass (g) against body mass (g). Note the overlap between the population groups with the European ellipse only marginally above that of the African sample.


Brain mass (g)
2000
1000
0
European females
0
100 000
200 000
Body mass (g)


Brain mass (g)
2000
1000
0
African females
0
100 000
200 000
Body mass (g)

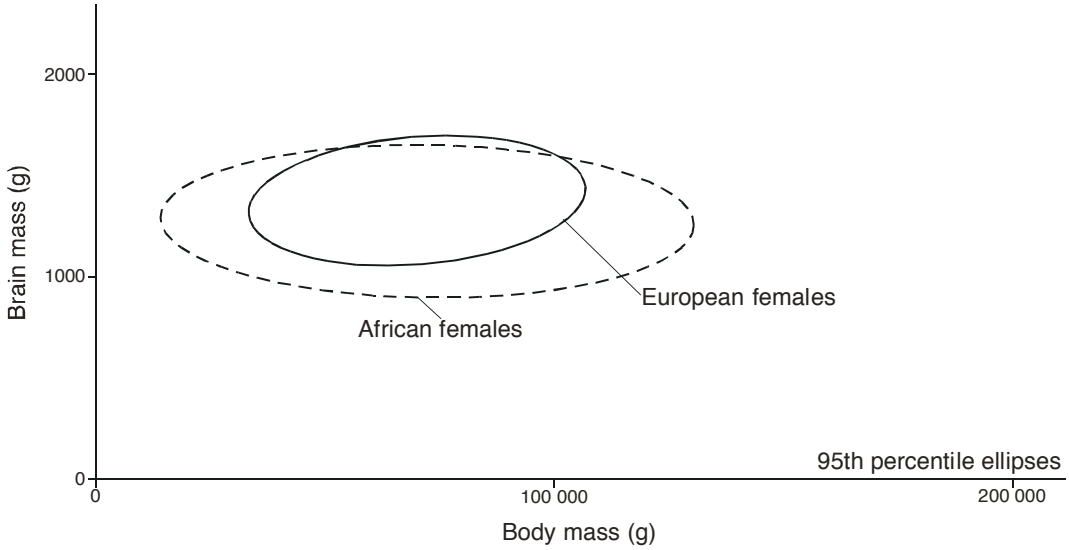
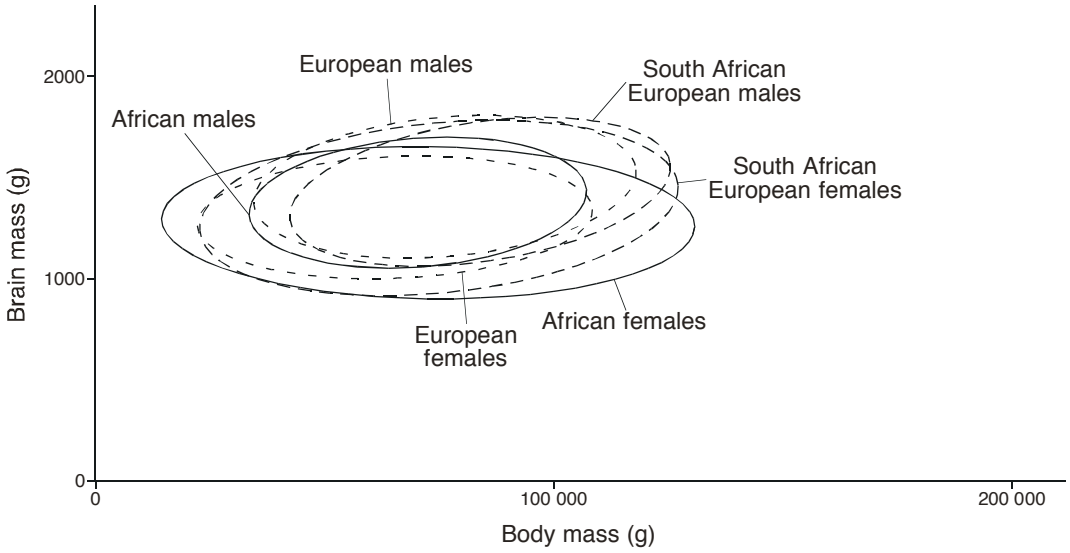

Brain mass (g)
2000
1000
0
European females
African females
95th percentile ellipses
0
100 000
200 000
Body mass (g)

Figure 17 **A**. 95th percentile ellipses of all the sub-samples used in this study for the 18-60 year age cohort. Ellipses were constructed from the brain mass versus body mass scatter plots. **B**. 95th percentile ellipses of all the sub-samples used in this study for the 18-60 year age cohort. Ellipses were constructed from the brain mass versus height scatter plots. Once again substantial overlap between the sub-samples is observed.


European males
South African
European males
African males
South African
European females
African females
European
females
2000
1000
0
Brain mass (g)
0
100 000
200 000
Body mass (g)

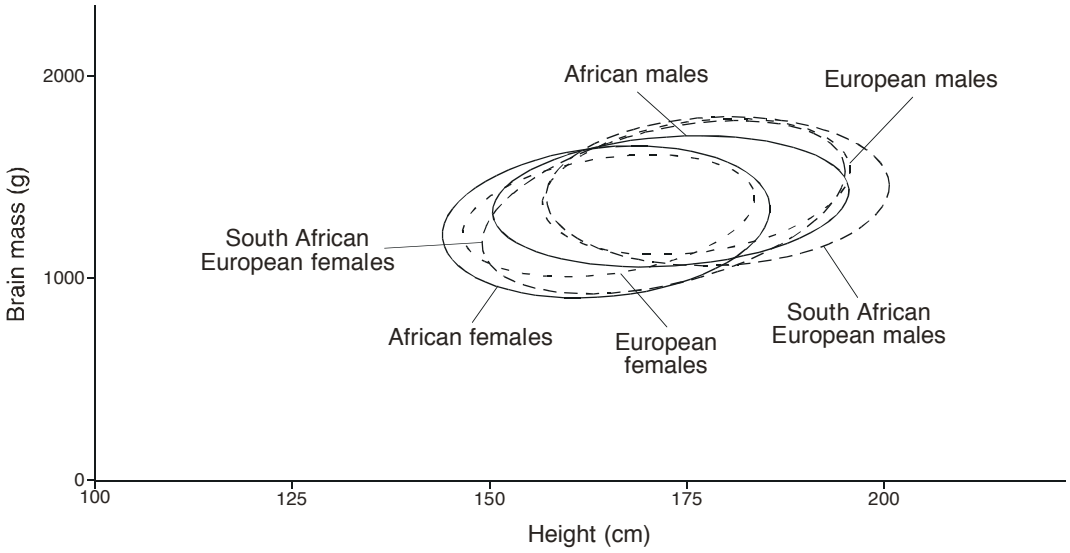

African males
European males
South African
European females
African females
European
females
South African
European males
2000
1000
0
Brain mass (g)
100
125
150
175
200
Height (cm)

Figure 18 Box and whisker plot for the six sub-samples used in this study. Despite differences in mean brain mass for the samples, what is most apparent is the range of variation in brain mass which reiterates the overlap between the samples.

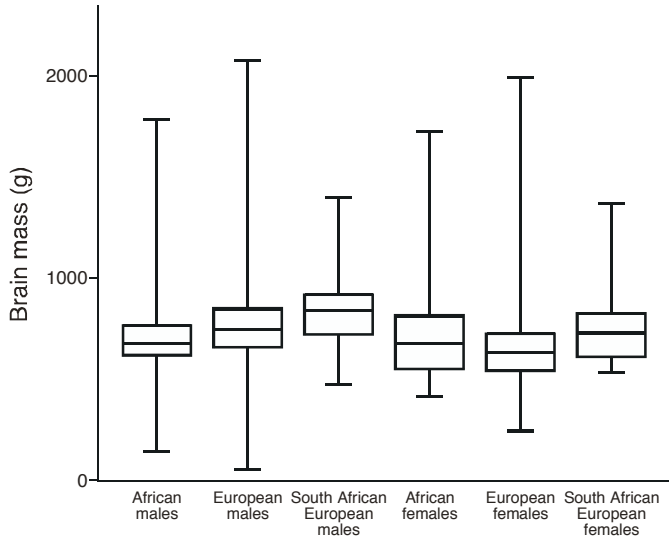
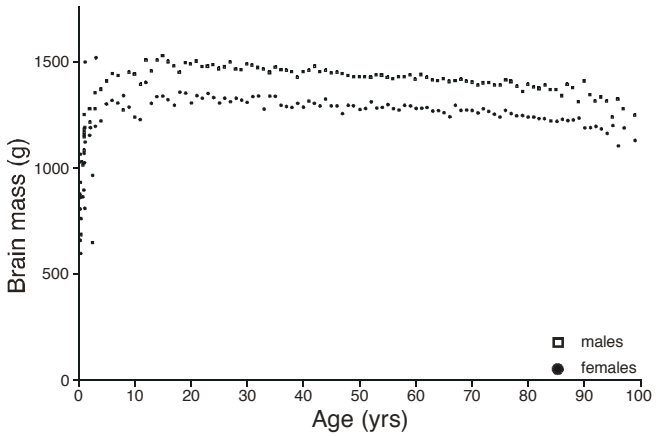
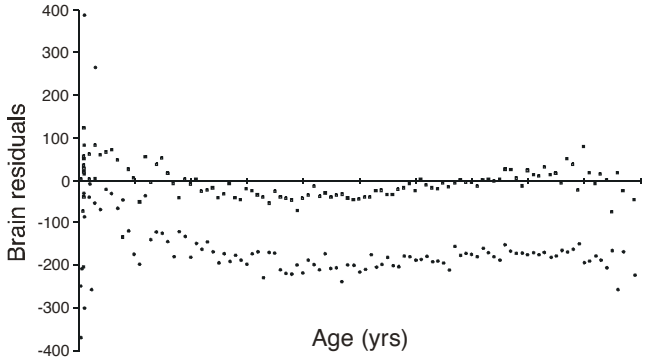
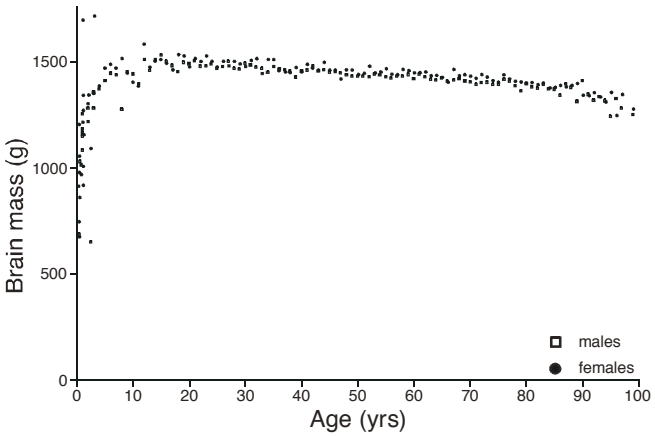
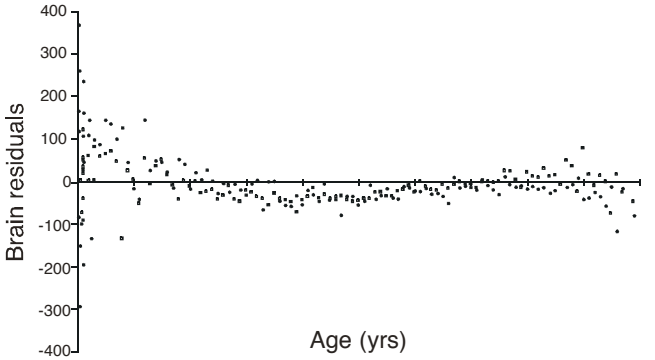

Brain mass (g)
2000
1000
0
African males
European males
South African European males
African females
European females
South African European females

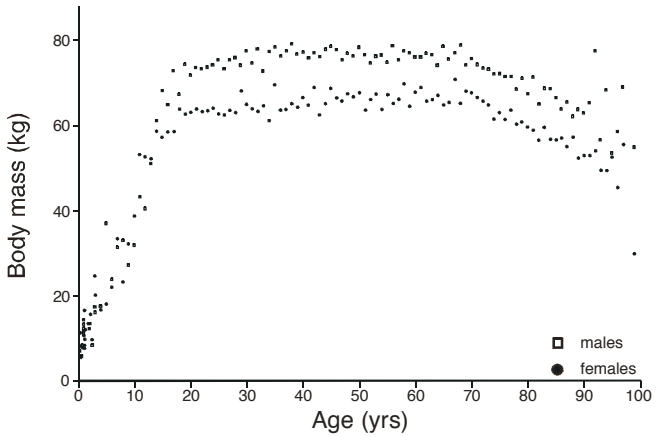
Figure 19 **A**. Comparison of male and female untransformed brain mass growth trajectories. Note the bimodal brain mass distributions with the male growth curve lying above that of the female sample prior to transformation of the female curve. **B**. Plot showing the distribution of brain mass residuals representing the distance between each data point on the female curve and the local best-fit regression line through the male data. **C**. Comparison of growth trajectories after transformation of the female brain mass growth curve by sequential hypermorphosis (i.e. phenotype axis was multiplied by 1.12). **D**. After transformation the female brain mass residuals come to be evenly distributed around the male datapoints. There are 134 residual points and the longest run of female brain mass residuals on the same side of the male curve is 33. **E**. Comparison of male and female untransformed body mass growth trajectories. Once again a bimodal distribution is observed with the male growth curve lying above that of the female sample. **F**. Plot showing the distribution of body mass residuals representing the distance between each data point on the female curve and the local best-fit regression line through the male data. **G**. Comparison of growth trajectories after transformation of the female body mass growth curve by sequential hypermorphosis (i.e. phenotype axis was multiplied by 1.19). **H**. After transformation the female body mass residuals are evenly distributed around the male datapoints. There are 134 residual points and the longest run of female body mass residuals on the same side of the male curve is 36

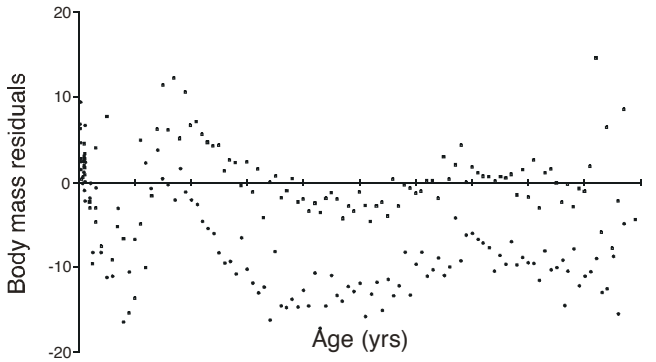

Brain mass (g)
1500
1000
500
0
0 10 20 30 40 50 60 70 80 90 100
Age (yrs)
males
females

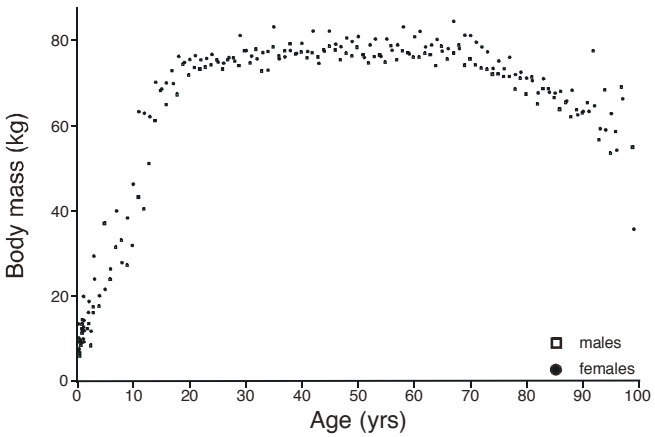

Brain residuals
400
300
200
100
0
-100
-200
-300
-400
Age (yrs)


Brain mass (g)
1500
1000
500
0
0 10 20 30 40 50 60 70 80 90 100
Age (yrs)
males
females


Brain residuals
400
300
200
100
0
-100
-200
-300
-400
Age (yrs)


Body mass (kg)
80
60
40
20
0
0 10 20 30 40 50 60 70 80 90 100
Age (yrs)
males
females


Body mass residuals
20
10
0
-10
-20
Age (yrs)


Body mass (kg)
80
60
40
20
0
0 10 20 30 40 50 60 70 80 90 100
Age (yrs)
males
females

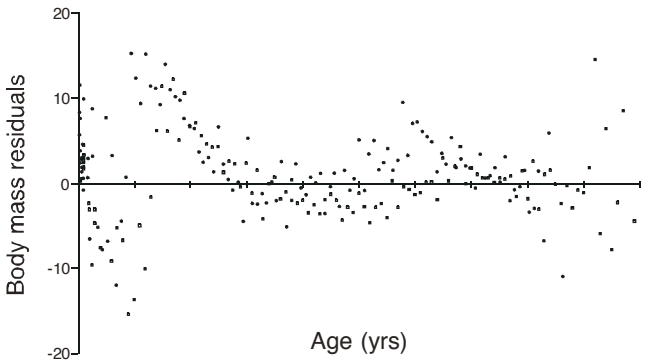
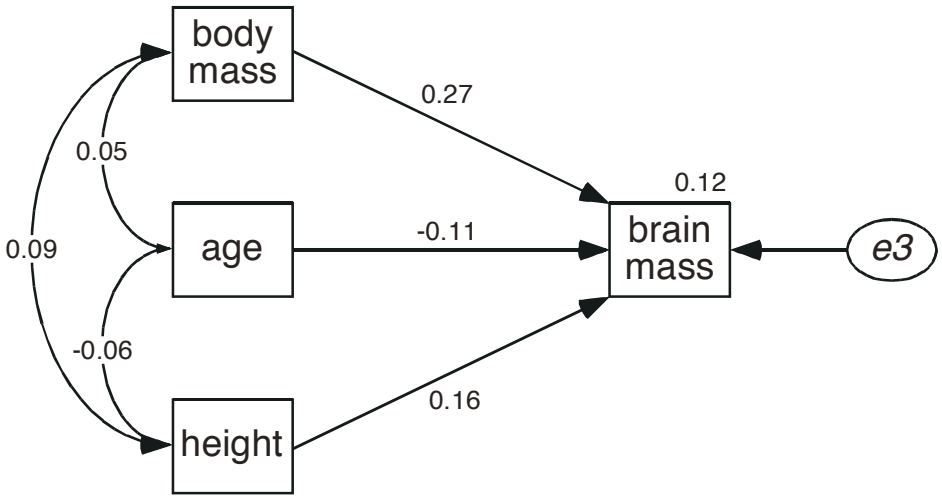

Body mass residuals
20
10
0
-10
-20
Age (yrs)

Figure 20 **A**. Diagram summating the results of multiple regression analysis for brain mass variation in the 18-60 age cohort. Explanatory variables were body mass, age and height. Based on the coefficient of multiple correlation 12 % of the variation in brain mass is explained by the combined effects of age, height and body mass. **B**. A diagrammatic summation of the extended multiple regression analysis for brain mass variation in the 18-60 age cohort. This analysis indicates that even after factoring in the effects of sex and population affinity, the coefficient of multiple correlation is only marginally increased to explain 24% of the variation in brain mass.


body mass
age
height
brain mass
e3
0.27
0.12
-0.11
0.16
0.05
0.09
-0.06

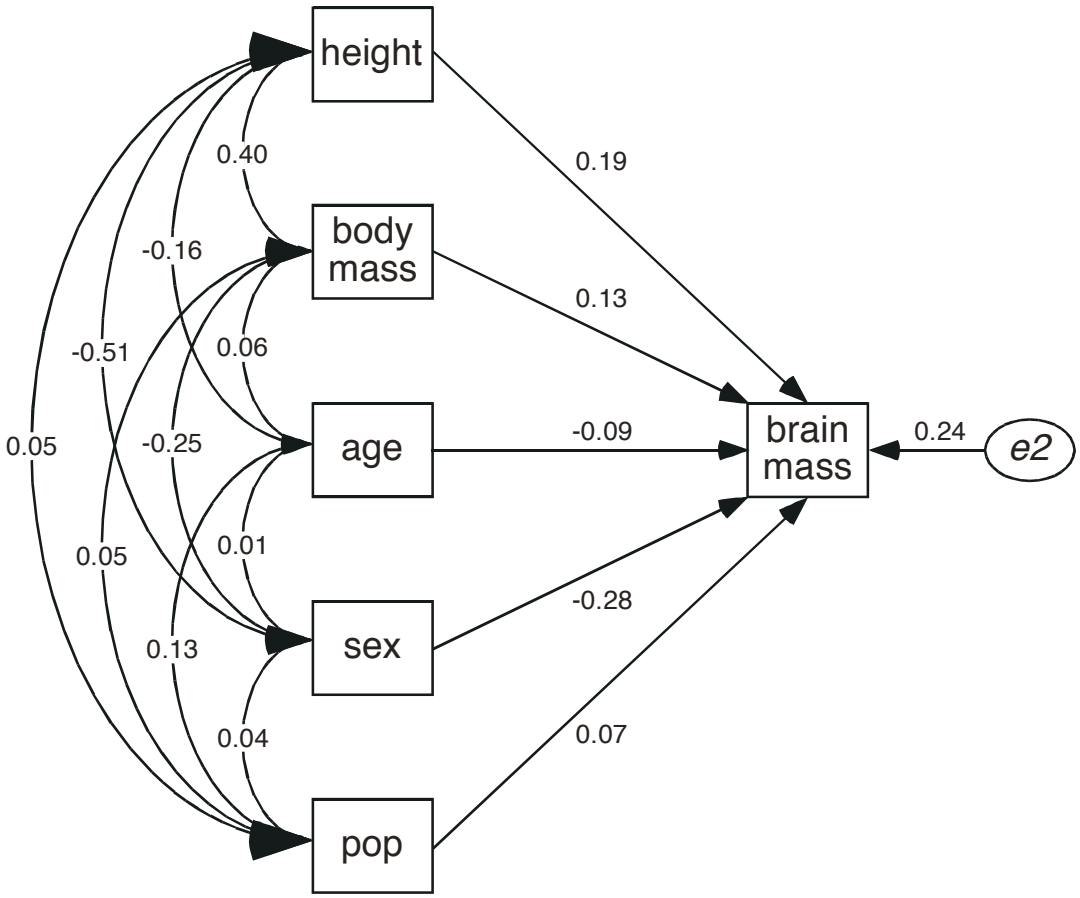
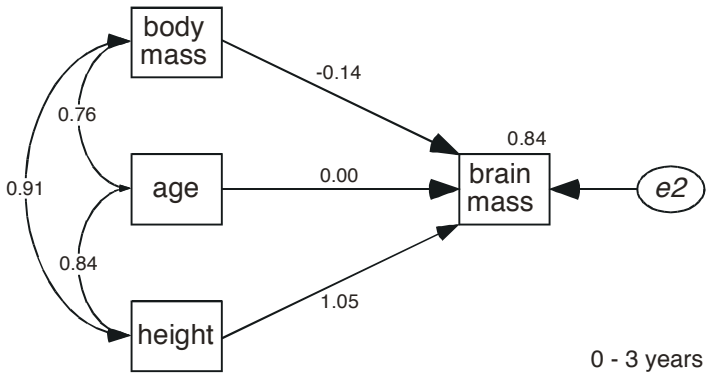
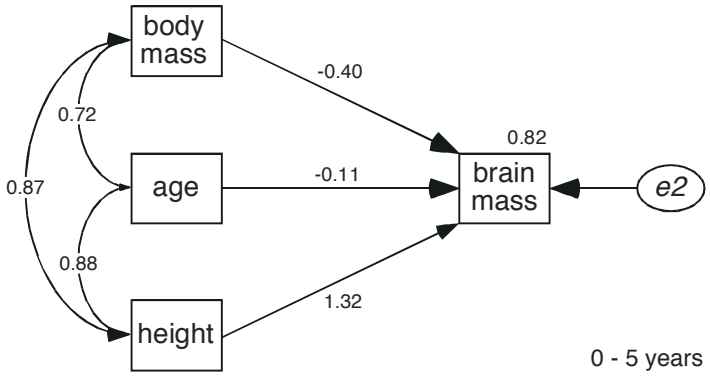

height
body mass
age
sex
pop
brain mass
e2
0.19
0.13
-0.09
-0.28
0.07
0.24
0.40
-0.16
-0.51
0.06
-0.25
0.05
0.01
0.13
0.05
0.04

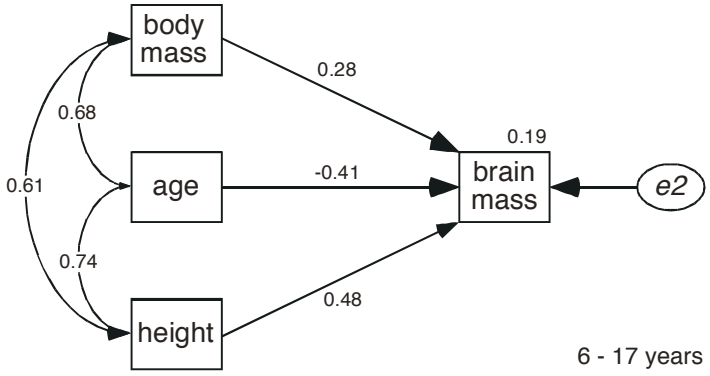
Figure 21 Series of multiple regression analyses using body mass, height and age to explain brain mass variation in different age cohorts. Note that the highest coefficient of multiple correlation (0.84) is observed during the 0-3 age cohort and this explanatory value rapidly decreases to reach the levels displayed in adulthood. e 2 = error term.



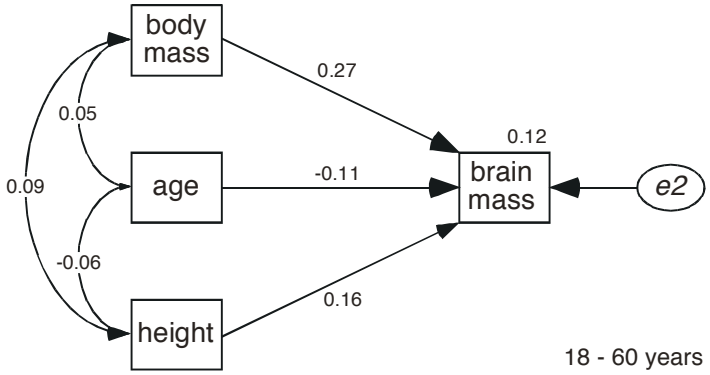
0 - 3 years



0 - 5 years



6 - 17 years



18 - 60 years

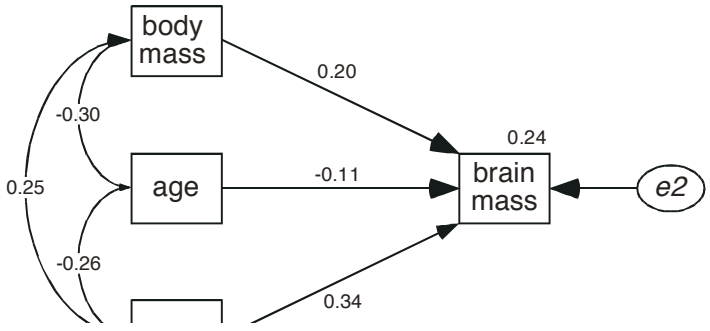
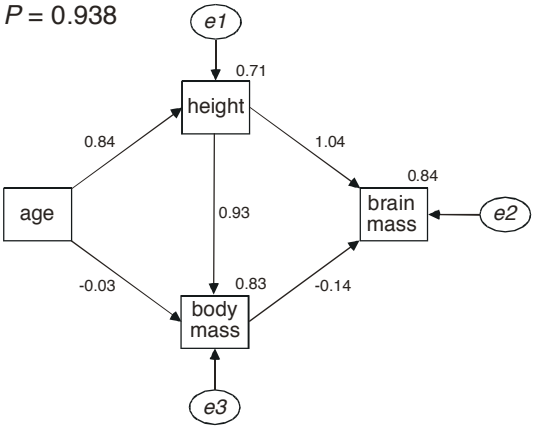
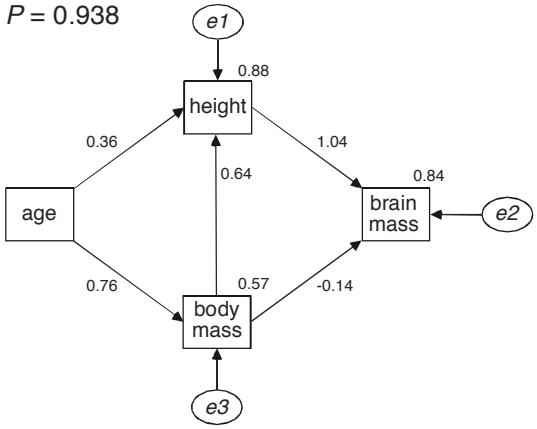
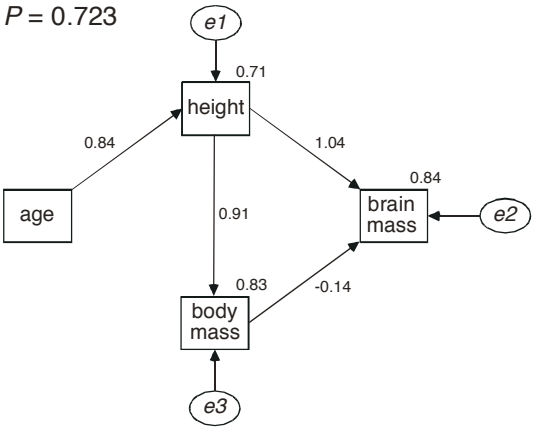
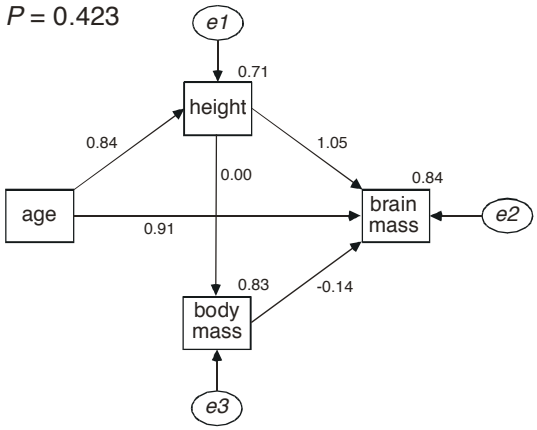


Figure 22 Path diagrams depicting four causal models for the relationship between brain mass, body mass, height and age. All four models were both successfully fit, i.e. $P > 0.05$ thus they significantly agree with the 0-3 year age cohort. When tested on other age cohorts such as the 18-60 year cohort these models were rejected and deemed to depart significantly from the data, reiterating the effect of a change in the brain and body size scaling relationships with age. The two best models as tested on the 0-3 year age cohort, were those with $P = 0.938$. In Model 1, height has both direct and indirect effects on brain mass. The direct effect of height on brain mass indicates that when height goes up by one standard deviation, brain mass goes up by 1.045 standard deviations. Body mass has a direct, inverse effect on brain mass of 0.14 standard deviations.


P = 0.938
e1
0.71
height
0.84
1.04
0.84
age
0.93
brain mass
e2
-0.03
0.83
-0.14
body mass
e3


P = 0.938
e1
0.88
height
0.36
1.04
0.64
0.84
age
brain mass
e2
0.76
0.57
-0.14
body mass
e3


P = 0.723
e1
0.71
height
0.84
1.04
0.84
age
0.91
brain mass
e2
0.83
-0.14
body mass
e3


P = 0.423
e1
0.71
height
0.84
1.05
0.00
0.84
age
0.91
brain mass
e2
0.83
-0.14
body mass
e3

4.4 Discussion

4.4.1 Average human brain mass

Using the entire sample of 11000 individuals for the age cohort of 18-60 years, the average human brain mass is approximated as being 1412 g (males 1447.95 g; females 1302.14 g). This is comparable to that reported in previous studies, where mean human brain mass ranges between 1169 g to 1498 g (See Table 26); however, brain size estimates are often derived from different sources and using a range of procedures and measures of brain size. For example, the average brain mass reported by both Strzalko (1974) and Holloway (1980) were both obtained using forensic material and the means closely approximate one another (i.e. mean =1367.1 g, males = 1432.2 g, females = 1302 g; and mean =1387.6 g, males = 1457.2 g females = 1317.9 g; for Strzalko (1974) and Holloway (1980) respectively). However, the two populations from which they were derived vary in that Strzalko used a Polish population whilst Holloway's estimate was recalculated using the Danish sample from Pakkenberg and Voigt (1964). Other studies such as that by Borowska and Golachowska (1935), based on a Slavic population and that by Milicer (1955) using a 19th century Australian Aborigine sample, vary not only in the samples used, but also in the measure of brain size, which for both population groups is that of cranial capacity as opposed to brain mass. Borowska and Golachowska's (1935) estimate of mean brain size is 1481.9 cubic centimeters (cc) (males 1545.5 cc; females 1396.1 cc) whilst Milicer's (1955) estimate is 1169.3 cc (males 1229.4 cc; females 1109.3 cc).

The use of cranial capacity has been drawn into question as a number of previous studies have shown that the endocranial vault may overestimate adult brain size by

approximately 20% (Brandes, 1936; Metteler, 1955; Tobias, 1994; Spocter *et al.,* 2007). Despite this relatively large overestimate, cranial capacity still remains a favoured measure of brain size for numerous researchers. An extension of this is the use of cephalometry on living individuals as performed by Henneberg *et al* (1985) in which a student population from Poland was used and yielded a mean brain size of 1498.3 cc (males 1589 cc; females 1407.6 cc). In order to extend the range of data and to produce brain mass estimates which are more representative of the human mean, meta-analyses have been used by certain researchers. The most notable has been the study performed by Beals *et al* (1984) in which a worldwide population average was derived from a database of cranial capacities from 122 human populations. The average human brain size as approximated by Beals *et al* (1984) is 1349.3 cc (males 1426.6 cc; females 1272 cc) and a similar analysis by Henneberg (1990) arrives at a comparable species average for *Homo sapiens* of 1350 cc. Both Beals *et al* (1984) and Henneberg (1990) make use of cranial capacity rather than brain mass to arrive at the species mean for *Homo sapiens*. The results obtained in the current study are well within range of that reported by both Henneberg (1990) and Beals et al (1984), but are larger and more closely associated with the values reported in more recent studies undertaken using the more validated Magnetic Resonance Imaging (MRI) procedures (e.g. Peters *et al.,* 1998; Bartley *et al.,* 1997; Bigler *et al.,* 1995).

Another area of discontinuity between the techniques used to derive estimates of average human brain size in various studies has been the use of relatively small sample sizes to arrive at population means. For example, the samples used by Strzalko (1974); Pakkenberg and Voigt (1964); Borowska and Golachowska (1935) and Henneberg et al

(1985) all use sample sizes of 700 individuals or less. In comparison the current study uses a dataset of 11000 individuals from three population groups and two 'ethnically' distinct groups. Magnetic resonance imaging of the human brain has facilitated the *in vivo* volumetric analysis of brain mass with great accuracy (Piven *et al.,* 1995; Raz *et al.,* 1997; Bartley *et al.,* 1997; Bigler *et al.,* 1995; Caviness *et al.,* 1996; Geidd *et al.,* 1996; Peters *et al.,* 1998); however, the cost of this procedure has made large scale analyses with large sample sizes (the like of the current study) unfeasible. Although the brain mass of healthy individuals can now be determined with great accuracy, the range of variability of the samples as being reflective of the variation in brain size for *Homo sapiens* is drawn into question. In this regard we believe that while imaging does provide accurate results, the use of a reliable, large dataset is appropriate for defining the mean brain mass for humans.

Whilst the analyses of Beals *et al* (1984) and Henneberg (1990) attempt to overcome this pitfall by incorporating population means into their final estimate of average brain size, they suffer from the mathematical error of taking 'means of means' instead of 'weighting' the means as is conventional procedure in meta analyses of this type. This results in a lower average brain size estimate than might actually be representative of the species. For example although the actual average brain mass in this study is 1411.89 g, if individual group means as represented in Table 16 are summed and a mean is calculated from this sum, the new mean is 1362.69 g. The mean of means for this study would thus match more closely with that reported by Beals *et al* (1984) and Henneberg (1990), although it is obvious that this isn't the actual mean for the sample. In fact when calculating the mean of means for brain mass of the African and European

(Hamburg) samples, an estimate of 1351.1 g is derived whilst when using the two geographically similar population groups (i.e. South African European and African samples) an estimate of 1355.34 g is arrived at which is virtually identical to that reported by both Beals *et al* (1984) and Henneberg (1990).

A much better way of performing this type of meta analysis would be to correct for sample size by 'weighting' the means relative to the number of individuals in the sample (Sokal & Rolf, 1969). For this current study, the mean brain mass calculated by 'weighting' the mean brain mass from each population relative to the sample size for that population is 1411.85 g, which is only 0.04 g away from the actual mean calculated. Using the data presented in Table 26 (to the exclusion of that presented by Beals *et al*., 1984; Henneberg, 1990 and the data in this current study) a 'weighted' mean for brain size as calculated from the literature is approximately 1413 g, which is virtually identical to that obtained in this current study. This result argues in favour of a revaluation of average brain mass towards values in the range of 1411 g to 1413 g. Another statistical descriptive often accompanying reported measures of mean brain size is the standard deviation for the sample understudy. In this current study the standard deviation of brain mass is 153 g. The range for standard deviation from previous studies is between 92.6 g - 167 g. Our standard deviation is very similar to that reported by Henneberg (1990) and Olivier and Tissier (1975) who report a standard deviation of 157 g whilst Tobias (1968) reports a slightly higher standard deviation of 167 g. Thus, once techniques are corrected for, our database is in strong agreement with previous studies, and revises the average brain mass and body mass for humans as a species. This revision is important in terms of

understanding human brain evolution and the use of data in comparing changes in brain mass over time in the hominid lineage.

4.4.2 Population differences in brain mass.

Brain size differences have been reported between various population/ethnic groups and have been accorded differing explanations and significance; however, explanations that invoke hierarchical levels of intelligence and the link to brain size have recently reappeared in the literature (e.g. Rushton, 1988, 1991, 1992, 1994, 1997, 1998; Rushton & Ankney, 1996). In this current study it has been shown that although there exists differences between the mean brain mass of the African and European population groups, these differences are small and may be largely attributed to differences in body size between the two populations. Although tests for significant difference in mean brain mass between the African and European population are positive (i.e. $P<0.05$), a test for heterogeneity in slope indicated significant homogeneity in the slopes for brain mass between the two population groups (See Table 19). Furthermore, a visual assessment of the means and range of data for the two groups (Fig. 18) strongly reinforces the similarity existing between the two populations. When the 95th percentile ellipses of the male and female data points from the population groups were drawn, tremendous overlap was observed between the plots (Fig. 17). This overlap is seen to be even more dramatic when one considers the standard deviation in brain mass (i.e. 153 g) and that adding or subtracting this amount from the mean brain mass for each sex and population group would result in an immediate 'crossing over' into the range of the remaining groups. This reiterates the point, that the 'differences' observed between the ethnic/population groups

lie well within the expected variation for brain mass and challenges the validity of proposed links between brain size and intelligence within *Homo sapiens*. An extension of this argument has been laid forth by more recent clinical findings showing that numerous individuals with clinically defined microcephaly, are functionally normal and often have normal IQs (Sells, 1977; Sassaman & Zartler, 1982; Rossi *et al.,* 1987). That variation exists in human brain size and that this has very dubious links with IQ is perhaps best highlighted by the recorded brain volume for the Nobel prize wining novelist Anatole France. According to Gould (1981) Anatole France had a post mortem brain volume of 933 cc / 1017 grams, a measure which lies within the range for *Homo erectus*. Clearly the case for Anatole France serves as one clear example to doubt the credibility of claims for links between brain size and intelligence being partitioned along the lines of population affinity.

4.4.3 Sex difference in brain mass

Sex differences in mean brain mass have been reported in the literature and this study is no exception. As indicated in Table 16, mean male brain mass is larger than that of females (male 1447.95 g; female 1302.14 g) and this pattern is maintained across the population groups. Numerous other studies have indicated the same result, the reasons for which and its significance have been the subject of debate for numerous researchers. Our results are most notably closest to that reported by Strzalko (1974) and Holloway (1980) who report a mean male and female brain mass of 1432.2 g and 1302 g and 1457.2 g and 1317.9 g respectively. A test for significant difference between male and female brain mass indicates low probabilities of the means and variances in brain mass for males and

females being equal (Table 17). However, mean body mass and height are also largely different between the sexes, indicating that an associated difference in body mass or height largely accompanies the difference in brain mass. Male and female regression slopes of brain mass versus body mass were also largely heterogenous and are believed to be reflective of the greater variation in body mass between the sexes. In comparison, tests for similarity in slope between height versus brain mass regression equations indicates the existence of significant homogeneity in slope between males and females of the South African European and African populations, however this is not the case for the European (Hamburg) sample.

Whether differences in brain mass between the sexes can be accounted for by body size has been a matter of interest to numerous researchers. Even as early as 1892 Marshall had asserted that the use of height as a covariate to brain mass could not "wholly explain" the sex difference in brain mass between males and females. This point was substantiated by Gould (1981) who indicated that even after correction for height the difference between male and female brains is approximately 113 g. Skullerud (1985) obtained a similar result and indicated that the sex difference between male and female brain mass is in the range of 110-115 g. The sex difference (without controlling for body parameters) as calculated from the samples used in this current study ranged between 102 –150 g as based on the 18-60 age cohort.

Our analysis indicated that the strength of relationship between brain mass and body mass, and brain mass and height, varies tremendously with age, and that the strongest linear correlations between brain mass and body parameters occurs during the first 2.5 years of life (Table 21). Following this, there is a dramatic drop in the strength of

relationship between the variables under study after which the value of both height and body mass in explaining brain mass is reduced to the levels quoted in most texts (i.e. r ~ 0.13-0.29). If the change in r-value is taken to be reflective of the change in the strength of relationship between brain mass and body parameters at different ages, it is no wonder that the sex difference in brain mass persists despite correction for either height or body mass. This suggests a 'decoupling' of the scaling relationship between brain mass and body parameters to allow for greater variation in both height and body mass with increases in age. A possible reason for why brain mass is tightly correlated with body parameters early in life may be the existence of a developmental constraint which exerts its effect most notably during the first few years of life. In this regard, subsequent diminishing of the strength of association in later life is thus indicative of the end of a crucial period in the developing organism. An example of this type of waning of a developmental constraint may be the disappearance of potential pleiotropic effects of genetic expression patterns during the developmental period, which in humans may last for sometime in the post-natal period.

The amount of sexual dimorphism in brain mass is smallest during the first 5 years of life and then subsequently increases to the levels displayed in adulthood (Table 18). Coincidentally the amount of sexual dimorphism in body mass and height are also at minimum levels during the first few years of life and increases with advancing age. This is indicative of the constraint exhibited in scaling relationships between brain and body parameters early in life followed by a subsequent relaxation and the onset of greater variation. That male and female brain mass do not differ dramatically from one another is most apparent in an analysis of the 95th percentile ellipses (Figs. 14, 15). Substantial

overlap is evident between male and female ellipses and this is also apparent from the plots in Figures 17 and 18. Once again when considering the standard deviation in brain mass (i.e. 153 g) and subtracting this from the mean male brain mass for each sub sample, the female mean comes to lie even closer within the range of the male data points, thus reiterating that the size dimorphism in brain mass between the sexes lies well within the expected variation for human brain mass. This result again questions the validity of claims for substantial differences in brain size between the sexes and especially any claims between brain size and intelligence within *Homo sapiens*. Although the size difference between male and female brain mass is hardly substantial enough to warrant an investigation of any cognitive significance in size, the origin of this size difference is still a valid area of interest. We attempted to investigate this phenomenon by using a test for heterochrony as implemented by Rice (2002). Our analysis indicates that transformation of the female brain growth curve and subsequent testing using the more appropriate Kolmogorv-Smirnov test reveals that males have relatively larger mean brain masses than females and that this may be attributed to a slightly prolonged period of growth of the male brain.

Despite these differences in mass of the brain and body between males and females, an analysis of encephalization quotients (EQ) revealed that there is no difference in the relative size of the brain between the sexes. Our calculated EQs are somewhat lower than those previously published. For example, Jerison's EQ of 8.07 for humans (Jerison, 1973).The major difference between these studies and the present one is the difference in the estimate of brain and body mass, as well as the regression equation used. Given that our estimates are based on an extremely large dataset for all three possible

variants in the calculation of EQ, we believe that the present results represent a more accurate assessment of the situation. Thus, in previous studies the relative brain size of modern humans appears to have been overestimated.

Our analyses, whilst showing that there are differences in mean brain mass between males and females, these differences are not substantial enough to warrant an investigation of links to intelligence and brain size and that the differences between the sexes fall well within the expected variation in brain mass. Moreover, we find no difference in the EQ of the sexes when calculated using the large dataset analyzed in the present study. Jersion (1973) postulated that EQ was a direct measure of biological intelligence. If EQ is taken in this sense, then there is no difference in intelligence between the sexes of *Homo sapiens*.

4.4.4 Multiple regression analysis and causal modelling of brain mass variation

The use of multiple regression analysis indicates that as with the use of univariate analyses, body parameters are most closely related to brain mass during the first 3 years of life. The reason for this is postulated to be the result of a constraint that limits the amount of early variation in body size. A potential causal model for the explanation of brain mass variation indicates that height is the largest contributor to brain mass variation during this period, followed by body mass and then age. Our analyses also indicate that both sex and population affinity, play a minor role in explaining brain mass variation for *Homo sapiens*. The role of climate as a potential explanation for the variation in human brain mass is drawn into question by our reanalysis of the Beals *et al* (1984) dataset.

Accordingly climatic zone appears to explain a lesser amount of the global variation in human brain mass than either body mass or height. This result reiterates the importance of accounting for body parameters, especially in analyses concerned with exploring brain mass variability. The correlative and potentially constraining influence of body parameters (such as height and weight) on brain mass may not be overlooked and must have played a substantial role in channelling changes in hominid brain size. The possibility that these constraints may have been 'lifted' in intensity, 'decoupled' or potentially 'reversed' during the course of our hominid ancestry remains a feasible argument. Evidence for this is reflected in the plasticity of the allometric equations defining the relationship between brain mass and body parameters early in life as opposed to the weaker correlations later and the incremental variation in both brain and body mass with advancing age.

4.5 Conclusion

This study has shown that differences in brain mass between and within population groups are small and may be the result of variation in body parameters such as height and body mass, whilst factors such as sex, population affinity and climate play a lesser role. That *Homo sapiens* represent a single homogenous group is undisputable and is once again reiterated by the current analysis of brain mass. These results seriously question the validity of intraspecific links between brain size and intelligence due to the tremendous overlap between the sexes and population groups. In addition paleoanthropologists should find the levels of variation in brain mass for *Homo sapiens* useful as a guide to the interpretation of brain size variation in fossil hominid taxa.

Chapter 5

Allometric Scaling of Brain Size and Tempo in Primate and Mammalian Neural Systems Evolution.

5.1 Introduction

One of the most striking features in the evolution of brain size across mammals is the strong correlation between body and brain mass. This negative allometric scaling is a clear constraint on the evolution of variation in brain mass in mammals, i.e. changes in brain mass appear to be a predictable outcome of changes in body mass. This correlation has been known for well over a century (DuBois, 1897), but was formalized into a coherent theory by the seminal publication of Jerison (1973). What is clear from comparisons of body and brain mass across mammals is that certain groups do deviate from a mammalian norm, this being most clearly expressed in the primates (Jersion, 1973) and cetaceans (e.g. Manger, 2006). For non-primate-non-cetacean mammals, there is a strongly statistically significant and predictable negative allometric scaling that shows for every doubling in body mass, the mass of the brain increases by around 1.65 times (e.g. Armstrong, 1990; Manger, 2006). Mammals in general show some degree of deviation or variation, around this norm, with some species having a larger brain mass than you might predict from body mass and others the opposite. The primates, excluding humans, show a significant and order specific deviation to that seen in other mammals, in that the average brain mass is around two times larger than you would predict from knowledge of the brain and body mass of other mammals (e.g. Jerison, 1973). The non-human primates are seen to exhibit a negative allometric scaling relationship that is similar to that seen across mammals generally, but with higher than predicted brain

weights. When body mass in the non-human primates is doubled, the brain mass increases by approximately 1.69 times (e.g. Manger, 2006).

This primate – non-primate dichotomy is of interest in both understanding the history of the evolution of brain size in humans (e.g. Manger, 2005a) and in understanding trends and tempos in the evolution of the mammalian brain in general. For the most part, studies of the evolution of the mammalian brain have come to the fundamental conclusion that larger brains are more complex and differentiated (e.g. Stephan *et al.,* 1981). This line of reasoning emanates from cross species comparisons that do not take into account the phylogenetic relationships of the animals compared. This basic axiom often also ignores the level of organization within the brain that is compared across phylogenetically unrelated species. Thus, our understanding of brain evolution in mammals is being stymied by theorists who do not take into account the potential effects of phylogenetic constraints or scaling laws of form when discussing evolution of this organ. Phylogenetic constraints and scaling laws of form play important, if not the most important, roles in the genesis of any new form (Gould, 2002). Gould (2002) has surmised that these two avenues of evolutionary change may occur at a far higher relative frequency than adaptive avenues of change. While these avenues of change may not have received a great deal of attention previously in terms of studies of brain evolution, recently Manger (2005b) gathered evidence indicating that at the systems level of organization in the mammalian brain, there appears to be strong phylogenetic constraints apparent in that members of the same mammalian order do not appear to show differences in the complement of homologous subdivisions of systems despite differences in brain size, phenotype or life history.

The present study addresses some of these issues by examining allometric scaling within mammals at the phylogenetic level of the order and compares this with documented changes in the organization of the brain at the systems level. To undertake this, the scaling of the primate brain and subgroups of primates is compared to the scaling of the general mammalian brain and orders within the mammals. The case is then made for a potential correlation between constraints acting on brain mass within and across phylogenetic groupings with constraints acting upon changes at the system level of neural organization. This approach may lead to some predictability in findings across mammals adding a level of potential understanding to the study of brain evolution at the systems level that is not yet fully appreciated.

5.2 Materials and methods

In total 821 brain mass and body mass records for the Class Mammalia were collated from numerous published sources (Bininda-Emonds et al., 2001; Crile and Quiring, 1940; Stephan et al., 1981). Of these were representatives from 11 different Orders namely: Didelphimorphia (10); Dasyuromorphia (7); Xenarthra (9); Insectivora (8); Rodentia (26); Chiroptera (338); Carnivora (216); Artiodactyla (22); Diprotodontia (19); Perissodactyla (10) and Primates (156). In addition the estimated brain and body masses of 13 hominid species were also added to the existing database for later analysis (Chapters, 1, 2 and 3).

The analysis was split into four major parts: a) an investigation of the brain: body mass scaling relationship exhibited in the primate brain and how it compares to that of other non primate mammals; b) a test for homogeneity in slope between non primate

mammalian orders; c) a test for homogeneity in slope between the major sub-ordinal categories within the Order Primates; and d) a comparison of slopes and tests for homogeneity between primate sub-ordinal levels and orders from the Class Mammalia.

Bivariate trait relationships were analysed by fitting regression analysis lines using reduced major axis to the log base 10 transformed dataset as implemented by the statistical package (S)MATR: Standardised Major Axis Tests & Routines (Version 2.0; © Falster, 2003-2005). Slopes were fitted across the species within each group with 95% confidence intervals calculated following (Pitman 1939). In order to visualise these bivariate relationships, analysis plots depicting these relationships were constructed using the linear fit and graphing function in PAST (Version.1.18; PAST © Hammer & Harper, 1999-2005). A regression slope common to both groups was then estimated following Warton and Weber (2002), and using a likelihood ratio method as implemented by the statistical package (S)MATR: Standardised Major Axis Tests & Routines (Version 2.0; © Falster, 2003-2005). The significance of this estimate was determined by testing for significant heterogeneity among the group slope estimates by means of a permutational procedure (Manly, 1997). After fixing the position of individual points along the estimated common slope, residuals were permuted among groups 1000 times wherewith the common slope and test statistic was recalculated after each iteration. This method is analogous to that proposed by (Freedman & Lane, 1983) for linear regression, and has been shown to maintain close to exact significance levels in small samples for linear models (Anderson and Robinson 2001).

5.3 Results

Brain mass and body mass were strongly correlated within each of the groups studied (Table 27). The coefficients of determination range typically between 0.8 – 0.97 indicating that 80 % to 97 % of the variation in brain mass can be accounted for by variation in body mass. These high correlation coefficients were also associated with low probabilities of the two variables being uncorrelated and ranged between 0 - 5.73 x 10^{-133}. Low probabilities of uncorrelation argue strongly in favour of the existence of a brain: body mass scaling constraint that regulates changes in brain size across various taxa and taxonomic groups. The slopes for each of the taxonomic groups are also of interest as they give an indication of the rate of change in brain mass with changes in body mass and how these may correlate with changes at various levels of organization within the brain.

5.3.1 Non-human primate and mammalian scaling

While the non-human primates scale in a manner reflecting their greater relative brain mass as compared to the mammals (as indicated by the difference in the *Y* intercept), our observations indicate that the non-human primate slope (0.727) appears remarkably similar in magnitude to that of the mammalian class (0.749) (Figure 23). It should be noted here that the non-human primates include all extant primates for which we could find measures of brain and body mass to the exclusion of *Homo sapiens*, and the generalized mammalian group includes all extant mammals for which we could find measures of brain and body mass to the exclusion of the cetaceans as they show a phenotypically unusual form of scaling (Manger, 2006). Visual inspection of these results by plotting the respective regression lines seemed to confirm at least graphically if not statistically, the similarity between slopes derived for these two taxonomic groups as

the regression lines appear to be almost parallel. While speculation as to the whether these curves display similar enough slopes may be approached from a visual basis, a more sound and objective analysis, would be to test for homogeneity in slope using statistical procedures. In this regard a set of tests for common slopes between groups was undertaken (Table 28), and these tests for homogeneity in slope prove positive and indicate that the non-human primates do indeed have a slope comparable to that of the mammalian class. This first analysis indicates that it is likely that changes in the size of the brain associated with changes in the size of the body occur under the same type of scaling constraint in the non-human primates and the generalized mammalian class, i.e. a class level form of scaling is found for the non-human primates.

5.3.2 Scaling within mammalian orders

Of the several Mammalian orders used in this study, four groups were investigated to assess whether they displayed similar intra-ordinal 'uniformity' in slope. The orders included for analysis as there was enough data available (more than 20 combined brain-body mass estimates) were the carnivores, rodents, diprotodonts and artiodactyls. Visual assessment of the derived regression slopes was suggestive of slopes that were less steep than that determined for the mammalian class in general or the non-human primates. But the derived regression slopes appeared to be relatively parallel between these groups (Figure 24). The four mammalian orders analyzed were all seen to have regression slopes that lie between 0.518 – 0.667 in magnitude (Table 27); however when using a more objective statistical approach to testing for homogeneity in slope, the nature of the visual similarities observed for the Orders became apparent (Table 29). Tests indicate that within the four orders studies, the diprotodonts share a common slope

with the artiodactyls, carnivores and rodents, whilst rodents and carnivores also have statistically similar slopes. Thus, as with the first analysis it seems likely that changes in the size of the brain and correlated changes in the size of the body follow an order specific scaling constraint in the non-primate mammals.

5.3.3 Scaling within primate groupings

While the subgroups in the mammalian analysis represented mammalian Orders, the subgroups of the primate comparison were comprised of taxonomic groups taken below the level of the Order. These groups are as follows: the Pongidae & Hylobatidae group (Apes); Hominidae group (Hominids); Haplorrhini (New and Old World Monkeys) and Strepsirrhini (Prosimians). Unlike the comparison of the mammalian components, these slopes appear more variable though the apes (0.572) and the Haplorrhini (0.654) groupings superficially displayed slopes of a similar magnitude as that observed for mammalian orders (Figure 25). A notable exception to this is the very steep slope calculated for the hominid regression line (1.496) which included humans as one of the data points. Of all the groups examined in the current study, only the hominid regression showed positive allometry (i.e. slope > 1), the remainder being negative allometry (slope < 1). On statistical inspection a test for homogeneity in slope revealed that within the Order Primates, the Apes share a common slope with the Haplorhini (0.654) and Strepsirrhini (0.705), whilst as expected from the visual assessment of the curves, the hominid slope is significantly different from all other slopes within the primate Order (Table 30). Thus from the analyses presented above, three major primate groupings, of taxonomic levels below that of the Order, display similar scaling relationships between brain mass and body mass, and is potentially indicative of a grouping specific scaling

constraint. The clear separation and juxtaposition of the hominid slope from that of the rest of the primate groups is reflective of a scaling relationship in brain and body size which is specific to hominids, and originated at the onset of this family.

5.3.4 Scaling in primate groupings compared with mammalian orders

These analyses allow us to ask the question – do sub-categories within the Primate Order scale in the same fashion as some of the Orders comprising Mammalia? In order to answer this a cross taxonomic analysis for homogeneity in slope needed to be undertaken. It is apparent that the regression lines for a number of the primate groups are visually parallel to that of the mammalian orders (Figure 26), whilst the magnitude for most of these slopes are relatively close except for that of the hominids. Statistical analysis for homogeneity however revealed surprising results (Table 31). The apes were shown to share a common slope with a number of the mammalian orders (i.e. Artiodactyls; Diprotodonts; and Carnivores). The Haplorrhini slope was significantly homogenous with that of the Carnivores; Diprotodonts and Rodents. Statistically homogenous slopes also existed between the primate Strepsirrhini sub-order and the mammalian Order Rodentia. Not surprising, the hominid slope was not homogenous to any of the groups understudy.

Table 27 Regression estimates based on least square regression analysis for the samples understudy. N = sample size; R^2 = Pearsons correlation coefficient; P (Uncorr) = Probability of brain mass and body mass being uncorrelated; LowCIs = Lower confidence interval for slope; UppCIs = Upper confidence interval for slope; Interc. = Intercept.

Group	N	R^2	P (Uncorr)	Slope	$LowCI_s$	$UppCI_s$	Interc.
Primates	156	0.890	8.27 * 10-76	0.7269	0.6862	0.7675	-0.828
Other mammals	636	0.982	0	0.749	0.741	0.757	-1.286
Artiodactyla	22	0.898	2.23*10-11	0.5183	0.4367	0.5998	-0.234
Carnivora	218	0.939	5.73*10-133	0.6552	0.6327	0.6776	-0.889
Diprotodontia	19	0.887	2.45*10-18	0.5643	0.4612	0.6674	-0.819
Rodentia	27	0.955	1.81*10-9	0.6671	0.6074	0.7269	-1.107
Pongidae & Hylobatidae	8	0.970	8.19*10-6	0.5717	0.472	0.6715	-0.177
Hominidae	13	0.800	3.72*10-5	1.4958	0.9991	1.9924	-4.168
Haplorrihini	83	0.862	1.52*10-36	0.6586	0.6002	0.7169	-0.552
Strepsirrhini	25	0.930	8.34*10-15	0.7046	0.6214	0.7877	-0.92

Table 28 Common slope and associated P value Matrix used for comparing the Order Primates and the Class Mammalia (excluding the Order Primates). Numbers in **BOLD** indicate the derivation of an acceptable common slope where P>0.05 and thus the slopes can be regarded as homogenous. (a, b) = slope, Probability of the slopes being heterogeneous.

Group	Primates	Other mammals
Primates	-1	(0.748 ,**0.293**)
Other mammals	(0.748 ,**0.293**)	-1

Table 29 Common slope and associated P value Matrix used for comparing the slopes of some of the Orders within the Class Mammalia (excluding the Order Primates). Numbers in **BOLD** indicate the derivation of an acceptable common slope where P>0.05 and thus the slopes can be regarded as homogenous. (a, b) = slope, Probability of the slopes being heterogeneous.

Groups	Artiodactyla	Carnivora	Diprotodontia	Rodentia
Artiodactyla	-1	(0.647 ,0.004)	(0.536 ,**0.496**)	(0.619 ,0.010)
Carnivora	(0.647 ,0.004)	-1	(0.651 ,**0.086**)	(0.657 ,**0.707**)
Diprotodontia	(0.536 ,**0.496**)	(0.651 ,**0.086**)	-1	(0.641 ,**0.095**)
Rodentia	(0.619 ,0.010)	(0.657 ,**0.707**)	(0.641 ,**0.095**)	-1

Table 30 Common slope and associated P value Matrix used for comparing four major definitive groups within the Order Primates. Numbers in **BOLD** indicate the derivation of an acceptable common slope where P>0.05 and thus the slopes can be regarded as homogenous. (a, b) = slope, Probability of the slopes being heterogeneous.

Groups	Pongidae & Hylobatidae	Hominidae	Haplorrihini	Strepsirrhini
Pongidae & Hylobatidae	-1	(0.598 ,0.004)	(0.630 ,**0.102**)	(0.644 ,0.029)
Hominidae	(0.598 ,0.004)	-1	(0.674 ,0.003)	(0.732 ,0.010)
Haplorrihini	(0.630 ,**0.102**)	(0.674 ,0.003)	-1	(0.675 ,**0.366**)
Strepsirrhini	(0.644 ,0.029)	(0.732 ,0.010)	(0.675 ,**0.366**)	-1

Table 31 Common slope and associated P value Matrix derived from a comparison of the subcomponents of the Order Primates and the Class Mammalia. Numbers in **BOLD** indicate the derivation of an acceptable common slope where P>0.05 and thus the slopes can be regarded as homogenous. (a, b) = slope, Probability of the slopes being heterogeneous.

Groups	Pongidae & Hylobatidae	Artiodactyla	Carnivora	Diprotodontia	Hominidae	Haplorrihini	Rodentia	Strepsirrhini
Pongidae & Hylobatidae	-1	(0.546 ,**0.374**)	(0.650 ,**0.092**)	(0.569 ,**0.914**)	(0.598 ,0.003)	(0.630 ,**0.114**)	(0.637 ,**0.097**)	(0.644 ,0.030
Artiodactyla	(0.546 ,**0.374**)	-1	(0.647 ,0.005)	(0.536 ,**0.479**)	(0.547 ,0.004)	(0.614 ,0.007)	(0.619 ,0.004)	(0.610 ,0.003
Carnivora	(0.650 ,**0.092**)	(0.647 ,0.005)	-1	(0.651 ,**0.069**)	(0.658 ,0.002)	(0.656 ,**0.910**)	(0.657 ,**0.716**)	(0.659 ,**0.236**
Diprotodontia	(0.569 ,**0.914**)	(0.536 ,**0.479**)	(0.651 ,**0.069**)	-1	(0.607 ,0.005)	(0.634 ,**0.112**)	(0.641 ,**0.078**)	(0.650 ,0.039
Hominidae	(0.598 ,0.003)	(0.547 ,0.004)	(0.658 ,0.002)	(0.607 ,0.005)	-1	(0.674 ,0.003)	(0.682 ,0.008)	(0.732 ,0.006
Haplorrihini	(0.630 ,**0.114**)	(0.614 ,0.007)	(0.656 ,**0.910**)	(0.634 ,**0.112**)	(0.674 ,0.003)	-1	(0.663 ,**0.853**)	(0.675 ,**0.358**
Rodentia	(0.637 ,**0.097**)	(0.619 ,0.004)	(0.657 ,**0.716**)	(0.641 ,**0.078**)	(0.682 ,0.008)	(0.663 ,**0.853**)	-1	(0.680 ,**0.439**
Strepsirrhini	(0.644 ,0.030)	(0.610 ,0.003)	(0.659 ,**0.236**)	(0.650 ,0.039)	(0.732 ,0.006)	(0.675 ,**0.358**)	(0.680 ,**0.439**)	-1

Figure 23 Reduced major axis regression analysis plots of brain mass against body mass for the Order Primates (excluding human) and the Class Mammalia.

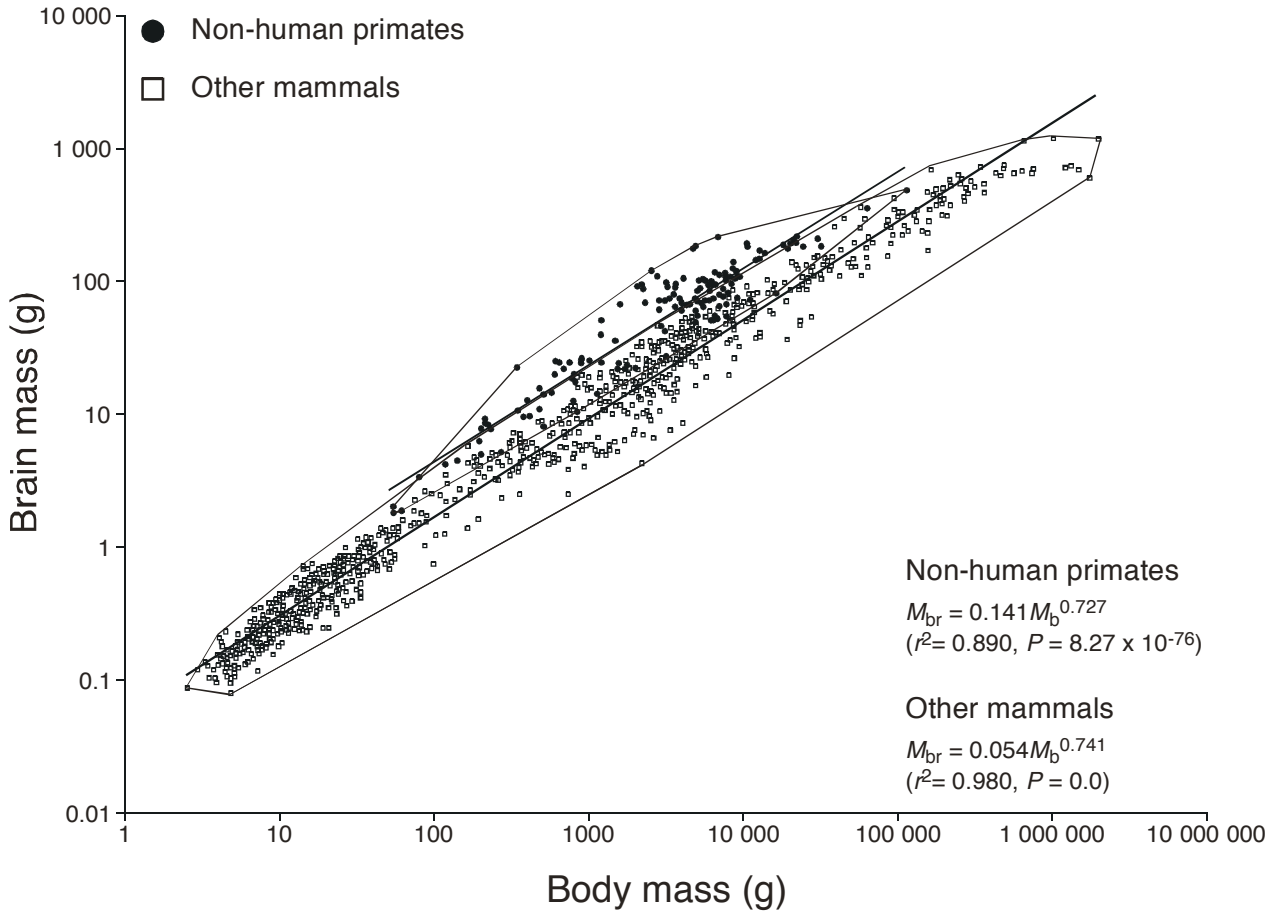

Non-human primates
Other mammals
Brain mass (g)
10 000
1 000
100
10
1
0.1
0.01
1
10
100
1000
10 000
100 000
1 000 000
10 000 000
Body mass (g)
Non-human primates
$M_{br} = 0.141 M_b^{0.727}$
($r^2 = 0.890$, $P = 8.27 \times 10^{-76}$)
Other mammals
$M_{br} = 0.054 M_b^{0.741}$
($r^2 = 0.980$, $P = 0.0$)

Figure 24 Reduced major axis regression analysis plots of brain mass against body mass for four major Orders of the Class Mammalia.

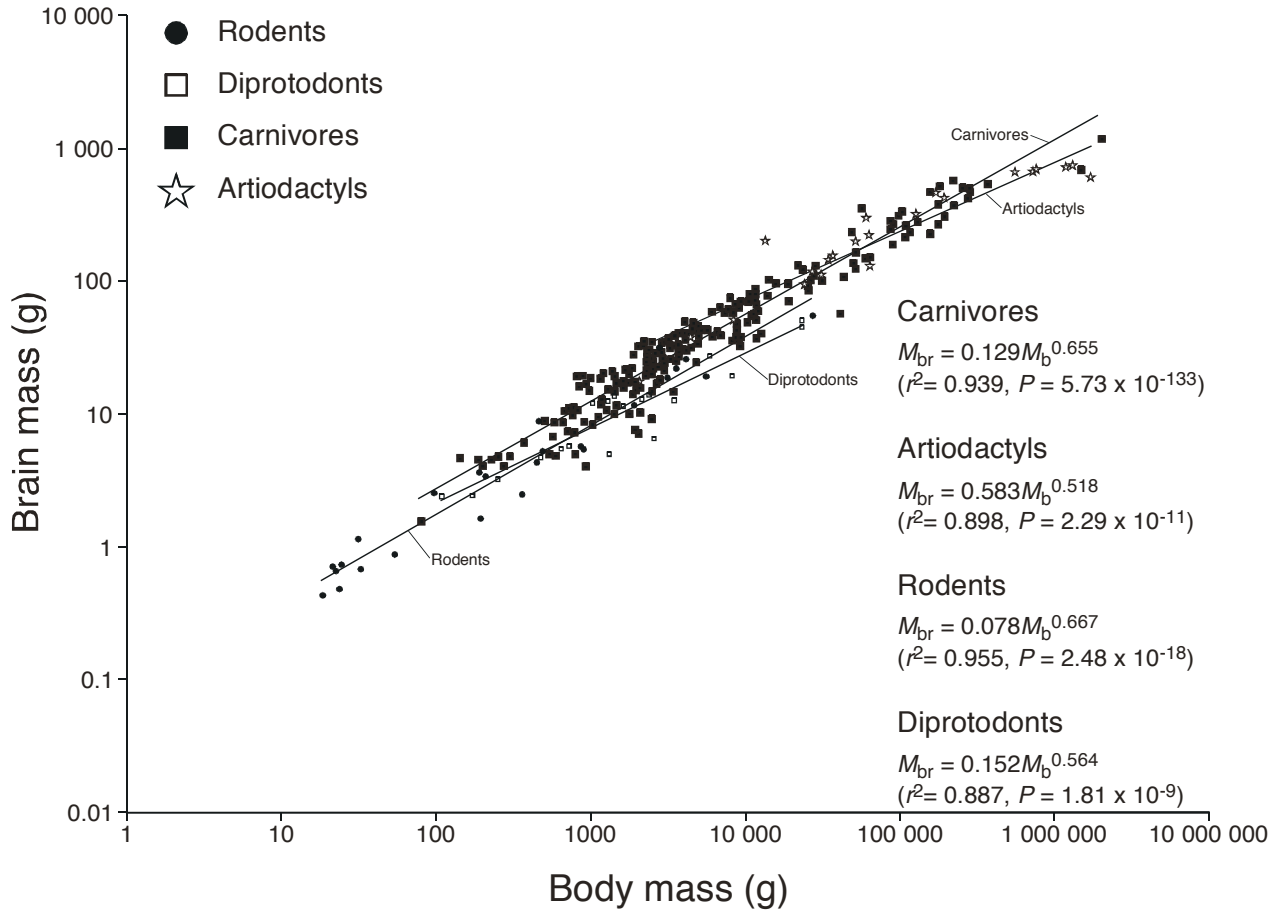

Rodents
Diprotodonts
Carnivores
Artiodactyls
Brain mass (g)
Body mass (g)
10 000
1 000
100
10
1
0.1
0.01
1
10
100
1000
10 000
100 000
1 000 000
10 000 000
Carnivores
Artiodactyls
Diprotodonts
Rodents
Carnivores
$M_{br} = 0.129M_b^{0.655}$
(r^2= 0.939, $P = 5.73 \times 10^{-133}$)
Artiodactyls
$M_{br} = 0.583M_b^{0.518}$
(r^2= 0.898, $P = 2.29 \times 10^{-11}$)
Rodents
$M_{br} = 0.078M_b^{0.667}$
(r^2= 0.955, $P = 2.48 \times 10^{-18}$)
Diprotodonts
$M_{br} = 0.152M_b^{0.564}$
(r^2= 0.887, $P = 1.81 \times 10^{-9}$)

Figure 25 Reduced major axis regression analysis plots of brain mass against body mass for four major groups of the Order Primates.

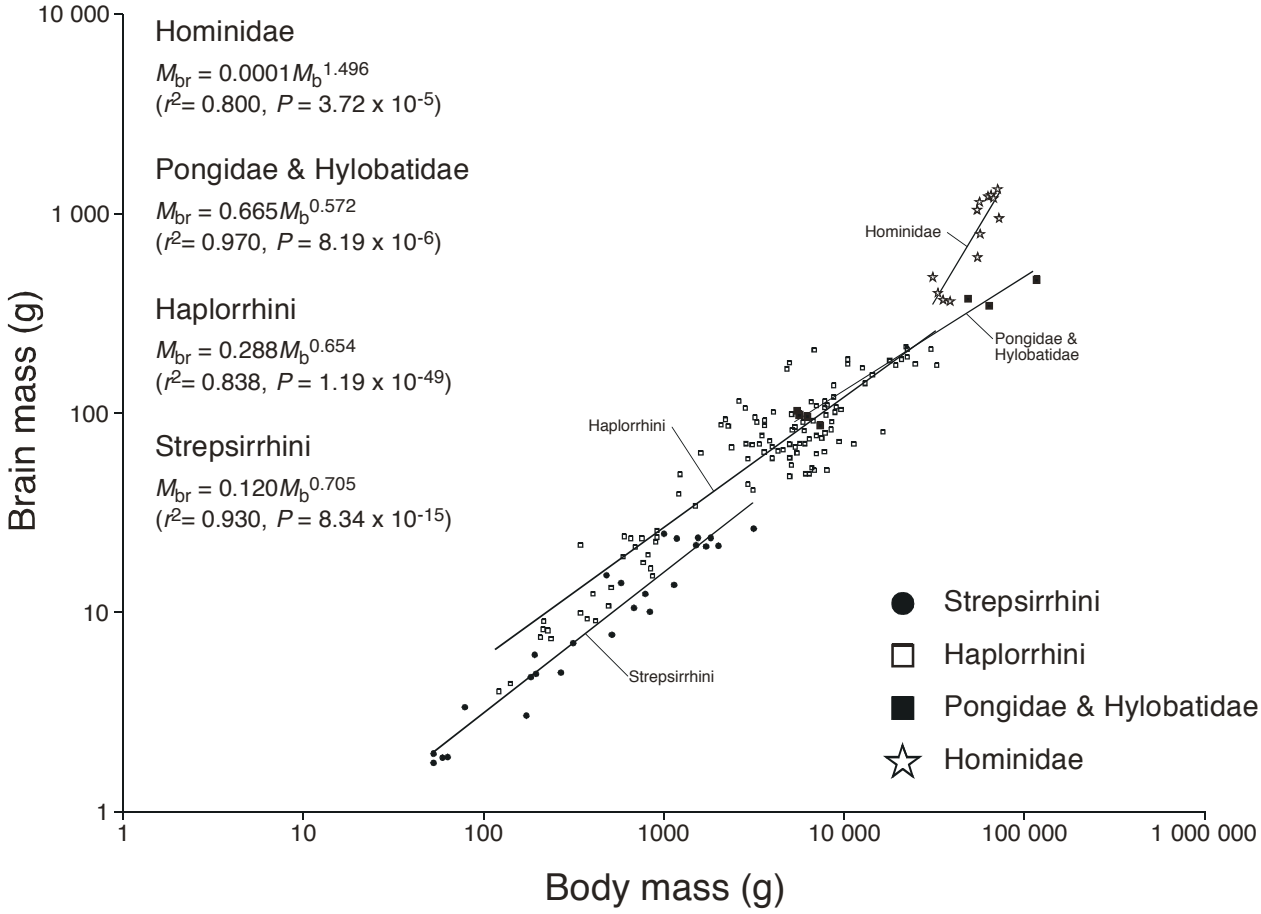
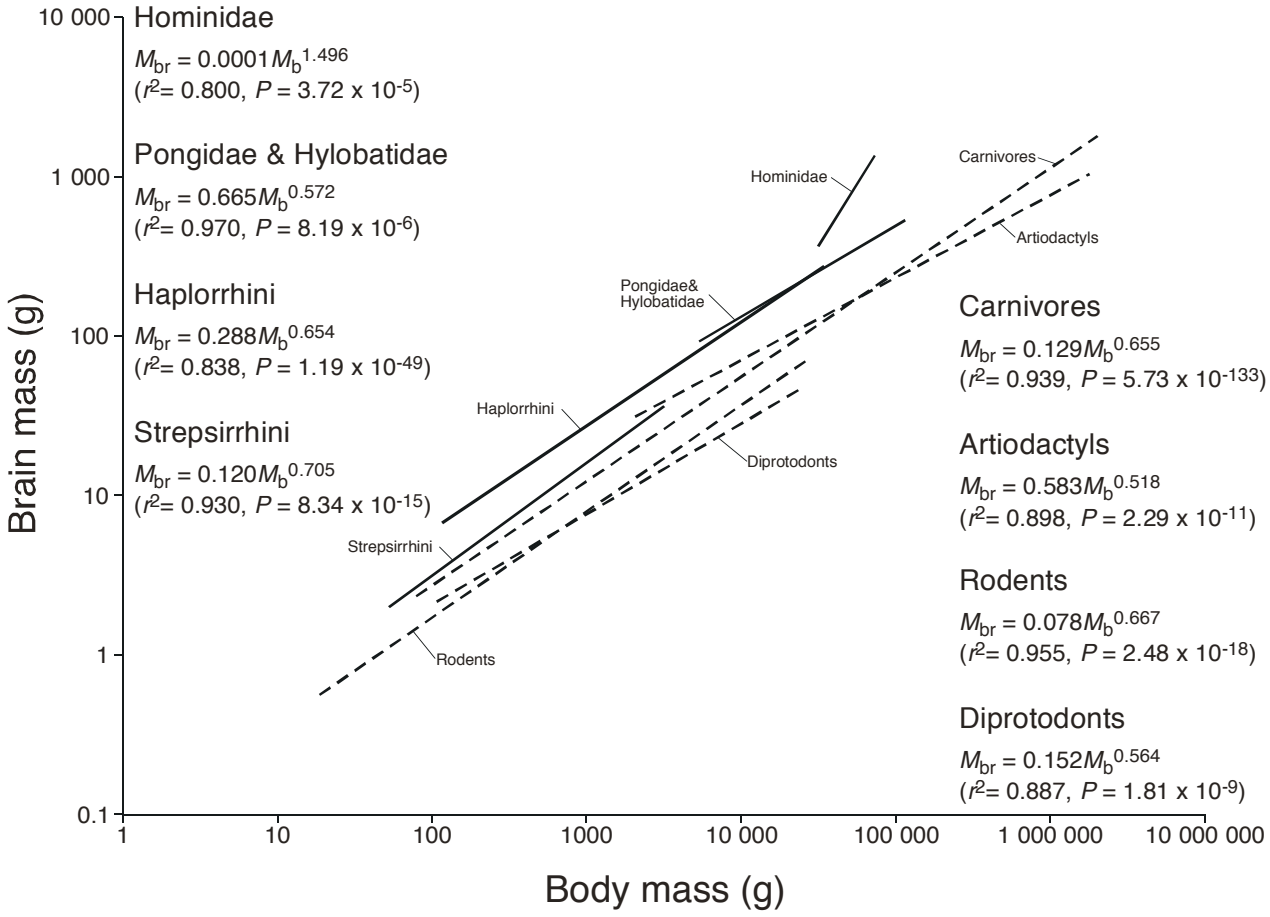

Hominidae
$M_{br} = 0.0001 M_b^{1.496}$
(r^2= 0.800, $P = 3.72 \times 10^{-5}$)
Pongidae & Hylobatidae
$M_{br} = 0.665 M_b^{0.572}$
(r^2= 0.970, $P = 8.19 \times 10^{-6}$)
Haplorrhini
$M_{br} = 0.288 M_b^{0.654}$
(r^2= 0.838, $P = 1.19 \times 10^{-49}$)
Strepsirrhini
$M_{br} = 0.120 M_b^{0.705}$
(r^2= 0.930, $P = 8.34 \times 10^{-15}$)
Brain mass (g)
10 000
1 000
100
10
1
Body mass (g)
1
10
100
1000
10 000
100 000
1 000 000
Hominidae
Pongidae & Hylobatidae
Haplorrhini
Strepsirrhini
Strepsirrhini
Haplorrhini
Pongidae & Hylobatidae
Hominidae

Figure 26 Reduced major axis regression analysis plots of brain mass against body mass for four major groups of the Order Primates and four major Orders of the Class Mammalia.


Hominidae
$M_{br} = 0.0001 M_b^{1.496}$
(r^2= 0.800, $P = 3.72 \times 10^{-5}$)
Pongidae & Hylobatidae
$M_{br} = 0.665 M_b^{0.572}$
(r^2= 0.970, $P = 8.19 \times 10^{-6}$)
Haplorrhini
$M_{br} = 0.288 M_b^{0.654}$
(r^2= 0.838, $P = 1.19 \times 10^{-49}$)
Strepsirrhini
$M_{br} = 0.120 M_b^{0.705}$
(r^2= 0.930, $P = 8.34 \times 10^{-15}$)
Carnivores
$M_{br} = 0.129 M_b^{0.655}$
(r^2= 0.939, $P = 5.73 \times 10^{-133}$)
Artiodactyls
$M_{br} = 0.583 M_b^{0.518}$
(r^2= 0.898, $P = 2.29 \times 10^{-11}$)
Rodents
$M_{br} = 0.078 M_b^{0.667}$
(r^2= 0.955, $P = 2.48 \times 10^{-18}$)
Diprotodonts
$M_{br} = 0.152 M_b^{0.564}$
(r^2= 0.887, $P = 1.81 \times 10^{-9}$)
Hominidae
Pongidae& Hylobatidae
Haplorrhini
Strepsirrhini
Rodents
Carnivores
Artiodactyls
Diprotodonts
Brain mass (g)
10 000
1 000
100
10
1
0.1
1
10
100
1000
10 000
100 000
1 000 000
10 000 000
Body mass (g)

5.4 Discussion

The present study compared the allometric relationship between the parameters of brain mass and body mass in a range of extant mammalian species, and how the relationship between these parameters change with specific phylogenetic groupings. The first key observation made, confirming that seen in many other studies (e.g. Jerison, 1973), is that as an order, the non-human primates show a brain:body scaling that is parallel to that seen in mammals in general, but with greater relative brain size. Thus, we conclude that the non-human primates exhibit a brain:body scaling that can be considered, when compared to mammals, as a class level type of scaling. Second, we found that when non-primate mammalian orders are examined on their own, a "typical" ordinal level type of scaling is found, one with a slope less steep than the class level. Again, relative brain mass may change between the orders, but the slopes of the regressions are for the most part parallel. When dividing the primates into four subordinal groups, prosimians, old and new world monkeys, apes, and hominids, we found that for the first three of these groups the relationship between brain and body mass is very similar to the type of scaling found within mammalian orders – an ordinal type of scaling. Hominids were a major exception to these scaling trends, and were the only group to show a positive allometry in the relationship between brain mass and body mass. Here we explore the possibility that the observed differences in scaling within these phylogenetic groupings may be instructive, or representative, of distinct changes in the structure of the brain at the organization level of the system in terms of the complement of homologous identifiable subdivisions (Manger, 2005).

5.4.1 Examples of stasis and change in systems level organization in non-primate mammals

In an earlier study Manger (2005) proposed that changes in the number of identifiable components of the brain at the organization level of the system (these components being discrete nuclei or cortical areas), would remain stable within a specific mammalian order irrespective of brain mass, life history or phenotype, but will either increase or decrease in number when species belonging to different orders were compared. The cholinergic, catecholaminergic and serotonergic systems, being readily identifiable with immunohistochemical techniques and having been compared in a number of mammalian species are examples of systems that are relatively easy to compare.

In two species of monotremes, the platypus and echidna, 52 components of these systems were found, all being identical in each species, despite a 3 fold difference in brain size, and dramatic differences in life history and phenotype (Manger *et al.,* 2002a,b,c). Within rodents, these systems have found to be similar across species (Da Silva *et al.,* 2006), again despite major differences in phenotypes and life history. Within the rodents, there is a nucleus, the C3 catecholaminergic nucleus of the medulla, that is specific to rodents alone (Smeets and Gonzalez, 2000). In the carnivores that have been studied, the cat and the dog, it was found that there was no difference in the number and homology of identifiable subdivisions of the catecholaminergic system (Kitahama *et al.,* 1994), despite differences in the brain size, phenotype, life history and the 55 million year period since these species shared a common ancestor. However, when species from different orders are compared, the number and complement of these subdivisions can vary quite significantly, even though they may share many features in common (e.g. Smeets and Gonzalez, 2000). For example, the A4 and A6 dorsal subdivisions of the locus coeruleus complex are only present in the rabbit (Blessing *et al.,* 1978), tree shrew (Murray *et al.,* 1982) and

primates (Felten *et al.,* 1974; Garver and Sladek, 1975; Jacobowitz and McLean, 1978; Schofield and Everitt, 1981; Smeets and Gonzalez, 2000) and absent in all other mammals studied. There are three subdivisions of the catecholaminergic system that are specific to the bottlenose dolphin (Manger *et al.,* 2004). The monotremes and the insectivores lack the cholinergic parabigeminal and Edinger-Wetphal nuclei (Manger *et al.,* 2002a; Karasawa *et al.,* 2003) that are found in other mammals, while the monotreme possess the serotonergic paraventricular organ of the hypothalamus (Manger *et al.,* 2002c) which is not found in other mammals.

Within the cerebral cortex, in the two species of monotremes studied, the number and homology of cortical areas were clear with both species having the identical complement (Krubitzer *et al.,* 1995a). In a series of studies on the cortical visual system of the ferret, it has been seen that the number of cortical areas in the ferret is likely to be the same as that seen in the cat, despite a 5-fold difference in brain size, extensive binocular vision in the cat compared to lateralized eyes in the ferret, and a 55 million year period of divergence (Manger *et al.,* 2005). In a study of five species of shrews belonging to the order Soricomorpha, Catania et al. (1999) found evidence for an invariant pattern of cortical area organization across these species despite an almost five fold difference in body mass (no brain masses reported); however, there were less somatic sensory cortical areas in the shrews than in the closely related hedgehog (Catania *et al.,* 2000) which belongs to the order Erinaceomorpha, which in turn appears to have less cortical visual areas than seen in the tenrec, belonging to the order Tenrecoidea (Krubitzer *et al.,* 1997). When larger cross order comparisons are made across mammals, it is clear that the representatives of the different orders have different complements of cortical areas. For example, Rosa (1999) provides a summary diagram comparing representatives of different

orders, demonstrating clearly that each species from the different orders has its own complement of cortical visual areas. Others have also providx_ similar and more broadly based diagrams representing these differences between individual species of different orders (Krubitzer and Kahn, 2003).

From this foregoing discussion it becomes apparent that there is the possibility that the number of homologous subdivisions within the neural systems of species of the same order are likely to be similar, and that these number of divisions will differ between orders. This view is in contrast to that provided by many, where gradualistic changes and eventual separation of nuclei and cortical areas is thought to occur as a result of increases in overall brain, or cortical, size (e.g. Changizi and Shimojo, 2005; Kaas, 2006). However, if we accept that within orders, subdivisions of systems are stable (Manger, 2005), we can propose a correlation between the ordinal brain: body mass scaling observed and the timing of changes in the evolution of additional subdivisions of systems such that both these changes will occur at the genesis of a new mammalian order and will be stable thereafter.

5.4.2 Examples of stasis and change in systems level organization in primates

There is a tendency in the comparative literature to describe changes in the differentiation of neural systems as being associated with increases in the size of the brain, such that larger brains have more differentiated or complexly organized neural systems (i.e. more nuclei and cortical areas) than smaller brains (e.g. Stephan *et al.,* 1981; Rosa, 1999; Krubitzer and Kahn, 2003; Changizi and Shimojo, 2005; Kaas, 2006). The discussion above challenges this pervading view for non-primate mammals, but what about primates? It has been assumed that the larger brain size of

humans should be associated with significant changes in the organization of neural systems, especially with language (e.g. Tobias, 1995), but is this really the case, and if so, how and when might this occur?

The first point to elucidate here is to determine whether there are examples of consistency in the subdivisions of systems across various primate species, i.e. are there systems that have the same complement of homologous nuclear subdivisions in all primate species? The strongest examples of consistency stem from the literature on the neuromodulatory systems. Of three species studied, the common marmoset, baboon, and human (Everitt *et al.,* 1988; Satoh and Fibiger, 1985; Mesulam *et al.,* 1989), 26 homologous subdivisions of the cholinergic system were found, including the absence of a subdivision (the medullary tegmental field) seen in other mammals. For the catecholaminergic system, 27 homologous subdivisions were found in the pygmy marmoset, squirrel monkey, macaque monkey, and human (Felten *et al.,* 1974; Garver and Sladek, 1975; Jacobowitz and McLean, 1978; Schofield and Everitt, 1981). Thus for the neuromodulatory systems, 53 homologous subdivisions, and only these subdivisions, have been found in a range of primates of very different brain sizes, phenotypes and life histories. In examining a range of primaZü thalami, Jones (1998) could find no major differences in the parcellation of the nuclei across species, despite major differences in size, concluding that the only real difference in the thalami of various primate species was one of size and not nuclear differentiation. However, even given these examples of order specific complements of neural systems that appear to be constained in their evolution, many still believe that cortical areas must be added with increasing brain size in primates (e.g. Kaas, 1995, 2006; Karlen and Krubitzer, 2006).

One of the major problems in comparing the areal organization of primate cerebral cortex is that there appears to be as many schema of cortical organization as there are laboratories working on this problem. This difficulty is highlighted by Rosa (1997, his figure 1), where four different models of the organization of visual cortex in the owl monkey are presented. In these models the only consistent visual cortical area is V1, as even the borders of V2 are different (and this summarizes 7 studies). Interestingly, Rosa (1997) proposes a model whereby all simian primates share the same basic layout of cortical visual areas, and later (Rosa, 1999) indicates that prosimians have fewer visual cortical areas than simians. In a similar vein, Kaas (2004) has proposed that there are 11 visual cortical areas in common to all primates. Culham and Kanwisher (2001) make the case for the homology of 5 posterior parietal visual areas in macaque monkeys and humans.

The somatomotor cortex has also been extensively studied across primate species, and while the primary motor and somatosensory areas are a common feature, the number of other areas differs between reports. For example, Wu et al. (2000) found evidence of 10 cortical motor areas in the prosimian galago that were direct homologues of the motor cortical areas found in macaques. In a subsequent study of the somatosensory cortex, Wu and Kaas (2003) found evidence for three somatosensory cortical areas lateral to SII and PV, whereas only a single cortical area has been defined in this region for the macaque monkey (Krubitzer *et al.,* 1995b). The cited studies are but some of the examples of the differing schema proposed for the organization of cerebral cortex in primates and there are many more available in the literature, but the central point to be made here is that there is a great deal of variance in these reports.

In the current study we divided primates into four phylogenetically related groups to examine differences in brain and body scaling. By doing this, we observed that three of the groupings evinced scaling that is similar to that seen by mammalian orders. Above we made the tentative link between scaling differences in orders and the appearance of novel subdivisions in systems for mammals in general. Here we attempt to make this link in primates, given the above scenario of a great deal of stasis in the non-cortical organization of neural systems, the variance in reports of cortical area organization, and the complete lack of readily comparable data of the organization of cerebral cortex in apes.

Rosa (1997, and Rosa and Tweedale, 2001, 2005) makes the case that the organization of visual cortex in the common marmoset and macaque monkeys consist of an equal number of homologous subdivisions, including (in their terminology) V1, V2, VP/DLp, VA/DLi, DLa, MT, DM, DA, and M. Rosa (1997, 1999, and Rosa et al, 1997) however, indicates that there are less cortical visual areas in the prosimian galago than are found in the simian marmoset and macaque. Moreover, in Rosa and Tweedale (2005, their figure 3) the manner in which the diagram is drawn indicates a great deal of potential space for additional cortical areas in the visual system of humans as compared with the macaque monkey. Thus, if we follow the studies of Rosa and colleagues, we have the possibility of additions of cortical areas between the groups we have demonstrated as having changes in the brain:body scaling relationship, and stasis within those where the scaling is consistent. In the somatosensory system of primates, a change in numbers of cortical areas is seen between the galago and macaque, but in this case the macaque has less cortical areas lateral to SII and more cortical areas posterior to primary somatosensory cortex than

the galago. Thus again, there are differences in the subdivisional complement of the systems between the groupings studied allometrically here.

5.4.3 Possible evolutionary scenarios

The link made herein between the appearance of additional subdivisions of neural systems related to the evolution of a new mammalian order and a subsequent altered and order specific allometric scaling appears to hold relatively true for mammals in general. Obviously a great deal more comparative work would be required to consolidate such a proposal, but on the basis for current information, it is as likely an evolutionary scenario as any forwarded to date. Moreover, this hypothesis does not ignore phylogenetic relationships, and the confounding observations of some species with larger brains having less subdivisions of particular systems than those with small brains (e.g. the mouse *vs* dolphin locus coeruleus comparison made in Manger, 2005) is also overcome.

We are left with two possibilities in the current situation considering primates. First of all, there appears to be a lot of order specific stasis in the non-cortical regions of the primate brain. This would not be parsimonious with the groupings made in the current study, and would negate the link made between allometric scaling trends of the brain and body and changes in the number of homologous subdivisions of the neural systems. On the other hand, the organization of the areas of the cerebral cortex do, at least to some extent, appear to be coincident with these allometric differences in the groupings made. If this were the case, then the groupings and scaling revealed may make it possible to reveal some predictability in the evolution and organization of the primate cerebral cortex at the systems level. If, for arguments sake, further studies across a range of prosimians revealed that they shared a common plan of

cortical organization and that there were fewer cortical areas found than in a common plan of cortical organization for new and old world monkeys, the scenario proposed here would apply. Again, apes may reveal a common organizational plan with more cortical areas than those found in new and old world monkeys. The positive allometric scaling found in hominds may reveal yet another point in primate evolution when additional cortical areas may be added. The increasing number of cortical areas may allow, at the systems level, for increasing degrees of freedom in terms of information processing, and thus correlate with the observable increases in behavioural sophistication (Kaas, 1995).

5.5 Conclusion

While it is obvious that the proposal forwarded in this study is tentative and for the most part speculative, if true, it would provide some sense of order in our understanding for primate cortical evolution, and indeed evolution of neural systems across mammals as a whole. A great deal more work is required to validate or negate the current hypothesis, but the observations made to date provide a basis of support. The major problems in arriving at conclusions regarding systems level evolution in mammals are dual, firstly there is the inherent gradualistic thinking ingrained in the literature indicating that larger brains are more complex, and second, especially for primate cortical organization, is the variance in schemes of organization proposed for the different species studies. Once these problems are dealt with, the current proposal, or newer more novel proposals may lead to a greater understanding of primate and mammalian brain evolution.

Chapter 6

Concluding remarks

6.1 Introduction

Much of the variation in organ size is attributable to the size of the body and the brain is no exception to this rule (Mayr, 2002). Regression analysis has revealed that across species, correlations between body mass and brain mass are usually in the order of 0.9 to 0.98, suggesting that at least 90% of the variation in brain mass may be explained by body mass. As a consequence of this observation numerous researchers have and continue to tailor their comparative research questions towards controlling for the scaling effect of body mass on brain mass (Jerison, 1973).

However, even after taking the effects of body size into account, brain mass still differs between various taxonomic groups (e.g. Barton, 2007; Jerison, 1979; Chapter 5). For humans in particular both the absolute and relative size of the brain, poses serious questions concerning the mechanism and agent responsible for hominid brain expansion (Chapter 4). In particular the residual variation in human brain mass (after accounting for body size) has been the focus of attention to various adaptationists suggesting that it is indicative of selection for greater cognitive abilities for foraging, sociality or other aspects of behavioural ecology (Dunbar, 1998; Aiello & Wheeler, 1995). A major difficulty in discerning between these selective forces is that the data representing rival hypotheses are largely inter-related. For example, Dunbar (1998) has argued for sociality as being the main selective agent for primate brain size and intelligence, an idea encapsulated by his 'Social brain hypothesis'. Unfortunately, group size and diet (or foraging behaviour) as invoked by Eisenberg and Wilson (1978) and Clutton-Brook and Harvey (1980) are often correlated with

each other and are compounded by the considerable error associated with the measurement of this data.

A more traditional approach has been to use the often rare and fragmentary evidence provided by the fossil record (e.g. Tobias, 1971; Wood & Collard, 1999; Falk *et al.,* 2000). As a source of evidence fossil hominid remains have contributed substantially to our pool of ideas concerning the mode, tempo and mechanism of hominid brain expansion. Whilst favoured as an approach closely aligned to elucidating the biology of intermediate hominid forms, the fossil record presents its own unique set of challenges and problems. Central to this is the much needed estimation of fossil hominid brain and body mass estimates and what the expected level of variation in brain mass is in a single hominid species.

Each chapter within this thesis has been an attempt to address a specific problem related to the use of fossil remains to explain hominid brain evolution. Chapter two addressed the problem of fossil hominid body mass prediction and revealed that cranial variables show similar predictive capability as those of postcranial elements. This chapter sought to emphasis the utility of cranial variables for body mass prediction, not only because of their availability and species diagnostic value, but also because of the existence of constraints which guide the scaling relationship between craniofacial elements and body mass. From this chapter much needed accurate predictions of fossil hominid body masses were derived and methods which take phylogeny into account as well as a method of variance partitioning was implemented for the first time in a question concerning the prediction of fossil hominid body mass.

Chapter three was concerned with addressing the other major problem regarding the use of fossil material, and that is the accurate prediction of hominid

brain mass. In this study magnetic resonance imaging of comparative primate brain scans were used and yielded a mean adult percentage difference between endocranial volume and brain volume of about 20%. Using a regression analysis of endocranial volume versus brain mass, an equation was derived from which brain mass for fossil hominid species could be calculated. This technique allows paleoneurologists to bring their comparative cranial capacity and body mass analyses in line with the traditional variables of body mass and brain mass as used in analyses of extant species. This chapter also provided the first preliminary set of brain mass estimates for selected hominid species, whilst also indicating by means of variance partitioning that the maintenance of the stable isometric relationship between brain mass and endocranial volume is likely to be the result of a strong developmental constraint governing the scaling attributes of neural and non-neural endocranial components.

Chapter four was primarily concerned with addressing the question of variation in brain mass within a single species, in this case *Homo sapiens*. This chapter attempted to overcome a central problem of comparative studies and that is obtaining measures for the limits of human brain size. In this analysis 18 000 data points of brain mass and associated body mass, height and age were analysed for sex and population differences.

Results indicated that hierarchical views of intelligence and brain size between sexes and population groups were unfounded as the majority of the variation in brain mass could be accounted for by differences in body size. This study saw the novel approach of structural equations modeling and residual transformation applied to the question of brain mass variability. The effects of body parameters in describing brain mass was reiterated by an analysis of the relatively reduced effect of climate in explaining both body size and brain size. An adjusted estimate of average human

brain mass in the range of 1411g to 1413 g was proposed which brings the estimate of average human brain mass more in line with values obtained from MRI studies. The 'diminishing' of constraining forces on the brain: body size axis later in life and the facilitation of greater variation, is argued to be indicative of the lifting of a developmental constraint which controls body size scaling during an integral period of development in the human fetus and infant.

Chapter five was concerned with addressing a key theoretical problem in studies of comparative brain: body mass allometry in mammals and p÷{mates and this is the problem of possible brain reorganization. In this study three groups of primates were shown to scale like mammalian orders, a result that is predicted to be indicative of changes in complexity at the systems level of organization. This argument is taken as suggesting that within primates there exists a potential group specific scaling law, with Prosimians having fewer cortical areas than Old World and New World Monkeys, which in turn may have fewer cortical areas than Apes, whilst Apes may have fewer cortical areas than hominids. For example, Kaas (2006) indicates that modern humans have more than 150 cortical areas, while the macaque monkey has around 80. This extent of reorganization, or addition of cortical areas may add a great deal to behavioural flexibility of the organism possessing more cortical areas by increasing the degrees of freedom in which information can be processed or associated. The consideration of phylogeny in analyses of complexity and the consideration of various levels of brain organization is emphasized by this chapter.

What remains is an application of the techniques outlined in all previous chapters to a case study and to observe what potential explanatory value this approach may add to the scientific literature.

In this regard, perhaps no better case study for a test of the allometric approach exists than that presented by the recently announced remains of a fossil hominid on the island of Flores, Indonesia. This discovery announced in 2003 by Morwood and colleagues, was given the scientific designation of *Homo floresiensis* and has subsequently captured the imaginations and attention of both the scientific and non-scientific community (Brown et al 2004; Falk et al 2005; Morwood et al 2005; Brumm et al 2006; Conroy & Smith, 2007). Central to the controversy which surrounds the taxonomic categorization of this species is its notable diminutive stature of around one meter, a measured cranial capacity of between 380-417 cc, and the recovery of a relatively advanced stone tool culture normally associated with the cerebrally superior (at least in size) species of *Homo erectus*. A point of contention exists concerning the assertion that a species with a cranial capacity similar to that of a chimpanzee could display the stone tool capability of an advanced hominid like *Homo erectus*. The debate concerning this species has seen the academic community split into two major camps: 1) those who assert that *H.floresiensis* is a microcephalic/pathological human group and 2) those who believe it represents a new hominid species derived from insular dwarfing of a *H. erectus* population to give rise to this unusual occurrence of body proportions. What could the approach outlined in the preceding chapters reveal about the taxonomic affiliation of this species and its apparent anomalous brain: body size scaling?

6.2 Predicting body mass and brain mass for *Homo floresiensis*

While the body dimensions for *Homo floresiensis* has been estimated as being similar to that of AL 288-1 suggesting a body mass of about 27 kg based on the femoral cross sectional area (Schoenemann, 2006). An estimate of body mass has as

yet not been derived from the cranium. Morwood et al (2005) provide a measurement of the foramen magnum length (28 mm) and breadth (21 mm) for this species while measurements for orbital length and orbital breadth were derived from a digital version of the published photographs available in the initial publication (Morwood et al 2005). Photographs were scanned using an Epson 3170 Photo-scanner and the images were saved as image files for subsequent measurement. The 'edge detection tool' accompanying the program IrfanView 3.09 was then used to delineate between the orbital boundaries whereafter the program ImageJ Version.1.3 (Abramoff *et al*., 2004) was then used to open and calibrate the image files and to measure the straight line distance between the upper and lower margins of the orbital cavity and the straight distance between maxillofrontale and ectoconchion (see definition in Chapter 2 and Figure 31 A & B). Using the Whole primate regression equations derived in Chapter 2 the species body mass for *Homo floresiensis* was estimated from the cranial measurements.

This yielded a mean orbital and mean foramen magnum based species body mass of 29.6 kg and 31.3 kg respectively. This estimate for body mass is consistent with that derived from the postcranial skeleton, once again reinforcing the utility of cranial elements for body mass prediction (Chapter 2).

However, could the cranium be used to predict brain mass for this species? As shown in Chapter 3, although the cranial capacity/endocranial volume overestimates brain volume by approximately 20 %, a strong predictive relationship exists between endocranial volume and brain mass. The derived regression equation for this relationship (See Chapter 3, Brain weight (g) = 0.8177 * Endocranial volume (cc) – 14.138) was used to estimate brain mass for *Homo floresiensis*. Using the more recently computed cranial capacity for this species of around 400 cc (Falk *et al.,*

2005) it is predicted that the species brain mass for *Homo floresiensis* is approximately 341.22 grams.

6.3 How does the brain: body mass scaling of *Homo Floresiensis* compare with that of Human pygmies, other hominids, and micrencephalics?

The estimated brain mass and body mass for *Homo floresiensis* was compared with that of other groups to see whether the scaling relationship may be indicative of any affinity shared with a particular group. Data for body mass, brain mass and or cranial capacity was compiled from a number of sources. Non human primate brain and body mass data were obtained from various the prepublished sources (Bininda-Emonds et al., 2001; Crile and Quiring, 1940; Stephan et al., 1981).

A modified version of the Beals *et al* (1984) dataset formed the core of the world human population sample with populations omitted in cases where cranial capacity and associated body mass had not been recorded. Measures of cranial capacity were converted to brain mass by using the equation derived in Chapter 3. Comparative fossil hominid brain and body mass data were obtained from various sources (Abbate *et al.,* 1998; Aiello and Dean, 1990; Asfaw *et al.,* 1999, 2002; Falk *et al.,* 2005; McHenry, 1992; Rightmire *et al.,* 2006; Ruff *et al.,* 1987; Senut *et al.,* 2001; Zollikofer *et al.,* 2005)The species represented here were : *Australopithecus afarensis*; *Australopithecus africanus*; *Paranthropus boisei*; *Homo habilis*; *Homo rudolfensis*; *Homo erectus*; *Homo heidelbergensis*; *Homo neanderthalensis* and *Homo sapiens*.

Brain and body mass data for extant modern human micrencephalics were obtained on request from a published dataset by Michael Hofman (Hofmann, 1984). This dataset consisted of brain and body mass records for 84 micrencephalic humans

aged between 0 to 74 years of age however only adult individuals (i.e. 18 years and older) were included in this current comparison. Hofman (1984) defined micrencephaly as individuals who had brain masses greater than three standard deviations lower than his normal comparative sample. Although he recognized two categories of micrencephaly (i.e. primary and secondary) he had not partitioned this dataset accordingly. Ward's clustering procedure was performed using PAST (Version.1.18; PAST © Hammer & Harper, 1999-2005) to see whether the dataset could be split into two major groups using the measures of body height, brain mass and body mass. The two micrencephalic groups are from here on referred to as Micrencephalic group 1 and Micrencephalic group 2, with the Micrencephalic group 1 appearing to be categorized by larger body and brain dimensions. Note that this distinction is pragmatic and is aimed at correcting for the possible admixture of data from the two clinically defined micrencephalic groups.

Human pygmy male and female body mass estimates were obtained from mean values published by Gusinde (1961) in a study describing the somotalogy of the Ayom pygmies from New Guinea. Gusinde (1961) also published mean head measurements for the Ayom population but for obvious reasons had not included measurements of brain mass or cranial capacity. The extensive list of linear craniometric dimensions for the Ayom were then used to estimate required endocranial volumes using the formula given by Williams et al (1995): Males 0.000337 (L-11) x (B-11) x (Ht-11) + 406.01; Females: 0.000400 (L-11) x (B-11) x (Ht-11) + 206.60 (Manjunath, 2002; Williams et al , 1995).This formula simply requires a measure of head length, head breadth and auricular height to derive an estimate of endocranial volume. Subsequently endocranial volumes were derived for the pygmy population, whereafter these volumes were converted to brain mass using

the equation relating endocranial volume to brain mass (see Chapter 3). Graphs representing the relationships between the *Homo floresiensis* and the other groups were then subsequently drawn and revealed the following results (Figs 27-30).

Firstly, the dichotomy between the hominid and ape line is most apparent (Figure 27) with the steep slope displayed in hominids being indicative of a distinct scaling relationship established for this group. A shown in Chapter 5 a comparison of the hominid and ape (Pongidae and Hylobatidae) regression slopes proves to be significantly different from one another (i.e. P(Heterogenous) < 0.05).

Figure 28 shows the regression lines computed for the world human population and hominid samples. Note that the human mean values calculated in Chapter 4, have been included in the hominid sample. When *Homo floresiensis* is plotted alongside this graph its scaling relationship in brain mass and body mass appears to be consistent with that displayed for the entire hominid group. This suggests that whatever selective forces had been responsible for the dramatic change in body size displayed in this species, had not effected the overall scaling constraint governing the proportionality of hominid brain and body mass dimensions. Thus what is clear from the above analysis is that the brain of *Homo floresiensis* scales just as one would predict for a hominid of its body size.

But how does the brain mass of *Homo floresiensis* compare with that of the human pygmy and the two micrencephalic groups obtained from Hofman (1984)? In comparison to the Human Ayom Pygmies it is apparent that *Homo floresiensis* exhibits a different scaling relationship in brain and body mass. The pygmy data points are strongly grouped with the human data points from different geographical regions and are visually distinct from *Homo floresiensis* (See Figure 29). Homo floresiensis also appears to scale differently from that of the two micrencephalic

groups. The distinction between *H.floresiensis* and the micrencephalic dataset is made most apparent by an observation of the mean brain and body masses for the two micrencephalic groups (See Figure 30). As observed in Figure 30 *H.floresiensis* appears visually distinct from the microcephalic groups in its brain: body mass scaling relationship and appears to share a scaling affinity with the Australopithecines. This observation is supported by recently published studies of brain shape in *H. floresiensis* and human microcephalics (Falk et al., 2007).

6.4 The paradox which is *Homo floresiensis*

This result presents a problem to our interpretation of the taxonomic designation of *Homo floresiensis* a point highlighted by Conroy and Smith (2007) who attempted to calculate the size of brain components for fossil species including *H.floresiensis.* Conroy and Smith (2007) conclude that if we are to take the original assertion of LB1 as being correct (i.e. that it represents a non-pathological specimen of Homo which despite its small brain size displayed advanced cultural attributes), then we are faced with a paradox in that 1cm^3 of *H.floresiensis* brain could not possibly be functionally equivalent to 1cm^3 of either a modern chimpanzee or human brain.

Whilst this conclusion may seem to be a serious blow to those arguing that *H. floresiensis* is a credible species, it also inadvertently raises concern regarding the well-accepted axiom that 'bigger brains are more complex'. This line of argument is epitomised by Stephan et al (1981) who categorically state: "The two variables of a structure, (i.e.) size and differentiation, in general do not vary independently. Increased size is almost always accompanied by progressive differentiation and *vice versa*." This form of reasoning has widely permeated comparative studies and has

fostered enquiry towards explaining the behavioural repertoire of large brained species on the premise that they represent complex, uniquely adapted neural systems.

However, the brain consists of hierachical levels of organisation (such as molecular, synaptic, single neuron, cortical areas and whole systems) and evolutionarily significant changes may occur at any of these levels and may or may not result in cascading effects upon the information processing capacity of the entire system, without necessarily having to increase or decrease brain size, and thus lead to changes in behavioural sophistication. Manger (2005) proposed that systems level organisation remains consistent across an Order and this concept formed a major part of the contribution in Chapter 5, in which Order specific scaling relationships were further investigated.

As argued for in both Manger (2005) and the Discussion in Chapter 5, there exists several examples to the contrary that bring the validity of the 'bigger brains are more complex axiom' into question. These examples are widespread and essentially argue for stasis in the number and complement of brain components at the organisational level of the system. A favoured example cited by Manger (2005) for stasis is that exhibited by the two Monotremes, the platypus and echidna who despite a three fold difference in brain size, contrasting life history, diet and phenotypes, still display consistency in all 52 components of their neuromodulatory systems. Rodents too appear to display similar consistency across species (Da Silva *et al.,* 2006) as do cats and dogs (Kitahama *et al.,* 1994). Homology of cortical areas amongst various species are also displayed within the cerebral cortex. Krubitzer *et al* (1995) have shown that the two species of monotremes studied display an identical complement of cortical areas. The cortical visual system of the ferret and the cat is also likely to be the same despite differences in brain size (Manger *et al.,* 2005), while Catania *et al*

(1999) found invariant patterns of cortical area organisation in shrews of various sizes. In primates, stasis in the cholinergic system of the marmoset, baboon and human (Everitt *et al.,* 1988; Satoh & Fibiger, 1985; Mesulam *et al.,* 1989) have also been shown with 26 homologous subdivisions of the cholinergic system being identified. In a study of the pygmy marmoset, squirrel monkey, macaque and human, 27 homologous subdivisions of the catecholaminergic system have been identified (Felter *et al.,* 1974; Garver & Sladek, 1975; Jacobowitz & Mclean, 1978; SChofred & Everitt, 1981). Sadly however, thus far only these subdivisions have been found and identified and more work needs to be done to approach a complete assessment of homology between primate species. What is emphasised however is that increases or decreases in brain size are not necessarily associated with increases or decreases in complexity, and that there exists several cases where organisational levels such as the number of cortical areas, have remained the same despite changes in brain size.

To return to the paradox presented by *H. floresiensis*, changes in the size of the brain of *H. floresiensis* need thus not be associated with changes in complexity, at least at the systems level, as evidence lent from contemporary comparative neurology would seem to suggest, and thus the possibility exists that $1cm^3$ of H. *floresiensis* brain is not functionally equivalent to $1cm^3$ of either a chimp or human brain. This perspective suggests a serious reconsideration of the manner in which trends in hominid brain size are currently viewed. Progressionist notions, strictly reliant on advancing brain size as a proxy for complexity both in structure and behaviour may need to be abandoned for a synthesis of observations based on comparative studies of extant species and the various levels of organisation at which these have been observed. This restructuring of the paradigmatically held view that ‘bigger brains are more complex’, may usher in a new era in paleoanthropology in which observations

of fossil hominid cranial capacities may be brought more in line with the contemporary work presented by modern neurology.

So what does this mean for *H. floresiensis* in terms of brain organization and behavioural sophistication? The parameter examined above, brain mass *vs* body mass, indicates that *H. floresiensis* may be considered to be a new species of hominid. However, with its small brain, can it demonstrate the purported behavioural sophistication? The analysis undertaken in Chapter 5, indicates that *H. floresiensis* may in fact have a brain organization, in terms of the number of identifiable areas of the cerebral cortex, that resembles the situation seen in modern humans. If the hominids indeed have far more cortical areas than the apes, and if *H. floresiensis* is a hominid, this would separate *H. floresiensis* from the apes in terms of cognitive and behavioural flexibility. This increase in the number of cortical areas, in the order of 30 to 40 areas (Kaas, 2006), even with a smaller brain size, would indicate the possibility of greater flexibility in the processing and association of neural information. This change at the systems level, even if not accompanied by changes at other levels of organization within the brain (Conroy and Smith, 2007), has the potential to lead to changes in the behaviour of the organism. This view of brain evolution and primate brain evolution specifically, may help, at least theoretically, to unravel the paradox of *H. floresiensis*.

Using *H.floresiensis* as a case study it has been demonstrated that the approach outlined in all previous chapters adds a novel perspective both in terms of practical body and brain mass estimation and theoretical implications for assessing the functional capabilities of fossil hominid species. What is still unaccounted for is an

assessment of the relative contribution of constraints and adaptation to the evolution of hominid brain and body size.

6.5 The role of constraints in hominid brain and body size evolution

It is has been the aim of the current study to provide data that may contribute to the assessment of the relative influence of structural laws of form, phylogenetic constraints, and adaptive factors during the course of primate and hominid brain evolution.

Using results obtained from subsequent investigations, constraints have been shown to play a critical role in the scaling attributes of the primate and hominid brain. In Chapter 2 variance partitioning revealed that developmental constraints governing the scaling attributes of the skull and body may explain up to 50 % of the variation in body mass whereas phylogenetic constraints are purported to have played a lesser role (i.e. 0.8 -3.6 %). In Chapter 3, scaling attributes between the neural and non neural components of the cranial vault suggested that the relative occupancy of the skull by the brain is highly constrained. 96 % of the variation in both brain mass and the residual endocranial space was explained by the developmental constraint controlling the scaling of these two variables with the cranial vault. That constraints are far more pliable than traditionally thought has been shown in Chapter 4 and intraspecific analyses of scaling attributes in humans is a prime example of this. The poor regression coefficients typical reported in intraspecific curves have been shown to arise during development as greater variation in body parameters are allowed with age. That grade shifts in the scaling of brain: body size exists is highlighted in Chapter 5 with the key emphasis being that primate groups scale like Mammalian Orders and that this may be reflective of Order specific scaling attributes. Constraints are integral

to correlated evolution between structures but the brain has several levels of organisation and changes effected at these various levels may have cascading affects on the information processing ability and behavioural repertoire of a species. Thus, this represents a unique problem in that even correlated changes between the brain and body size may not necessarily impact upon the 'complexity' of the neural system as functional integrity may be maintained via higher output states initiated at levels of organisation such as that of the molecule, neuron or cortical area. However, it is only through implementation of research protocols that take a pluralistic approach to an understanding of the role of both constraints and adaptation in the formation of the brain, that Order specific changes and the levels of organisation at which they may be act could fully be understood.

Figure 27 Least square regression analysis plots of brain mass against body mass for Apes and Hominids (including humans).

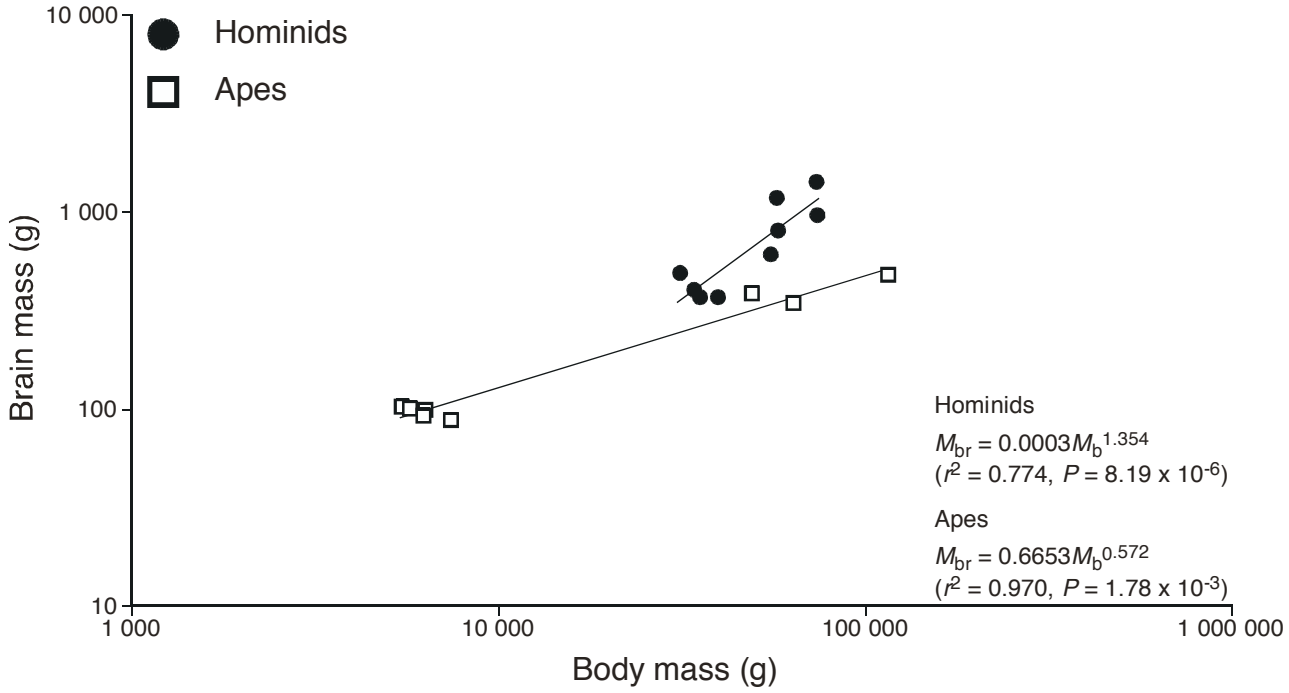

Hominids
Apes
10 000
1 000
100
10
Brain mass (g)
1 000
10 000
100 000
1 000 000
Body mass (g)
Hominids
$M_{br} = 0.0003 M_b^{1.354}$
($r^2 = 0.774$, $P = 8.19 \times 10^{-6}$)
Apes
$M_{br} = 0.6653 M_b^{0.572}$
($r^2 = 0.970$, $P = 1.78 \times 10^{-3}$)

Figure 28 Least square regression analysis plots of brain mass against body mass for the World Human population and Hominids (including human average). *Homo floresiensis* datapoint is also plotted though not included in the regression.

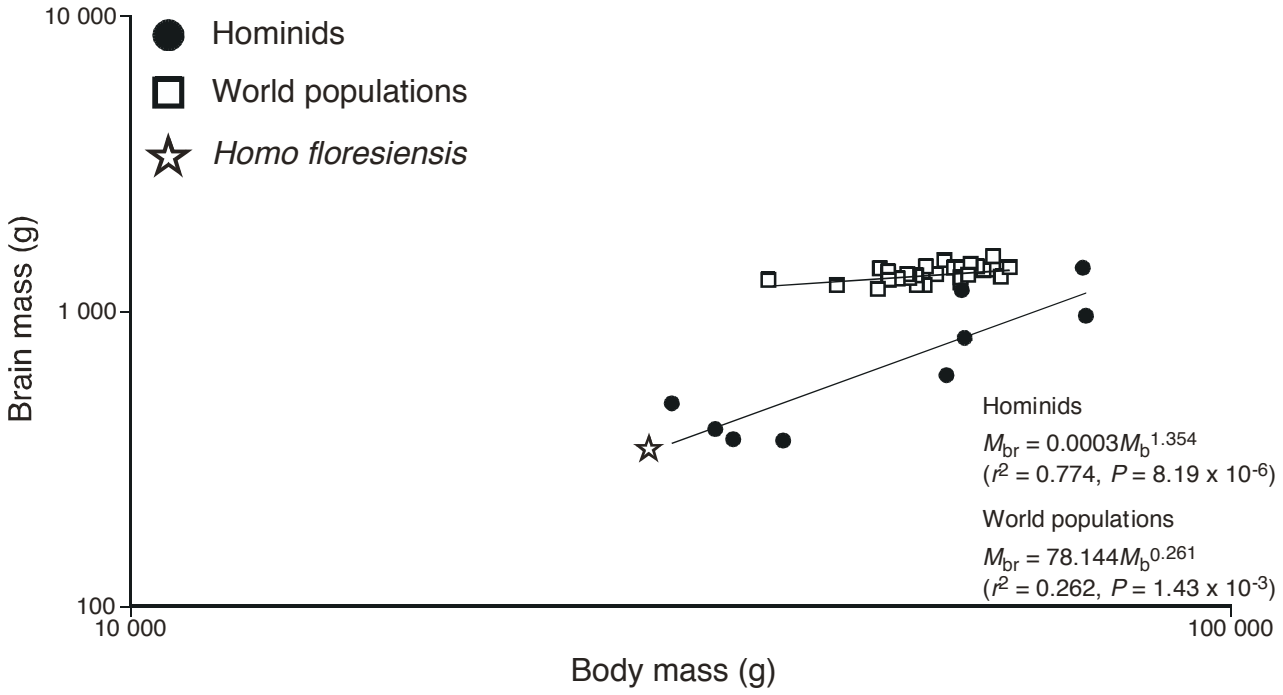

10 000
1 000
100
Brain mass (g)
Hominids
World populations
Homo floresiensis
Hominids
$M_{br} = 0.0003M_b^{1.354}$
($r^2 = 0.774$, $P = 8.19 \times 10^{-6}$)
World populations
$M_{br} = 78.144M_b^{0.261}$
($r^2 = 0.262$, $P = 1.43 \times 10^{-3}$)
10 000
100 000
Body mass (g)

Figure 29 Scatterplot of brain mass versus body mass for all the samples studied.

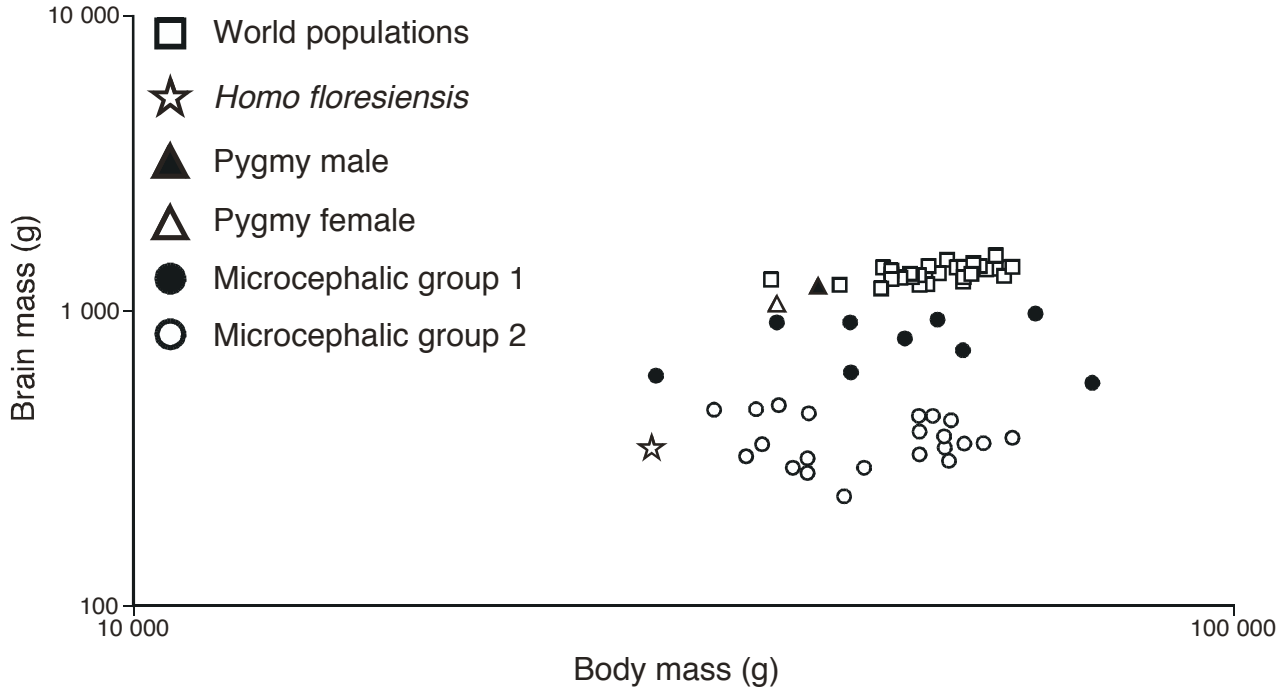

10 000
1 000
100
Brain mass (g)
World populations
Homo floresiensis
Pygmy male
Pygmy female
Microcephalic group 1
Microcephalic group 2
10 000
100 000
Body mass (g)

Figure 30 Least square regression analysis plots of brain mass against body mass for all the samples studied. The mean value for the microcephalic groups are plotted though not included in the regression analyses. The *Homo floresiensis* datapoint is also plotted though not included in the regression.

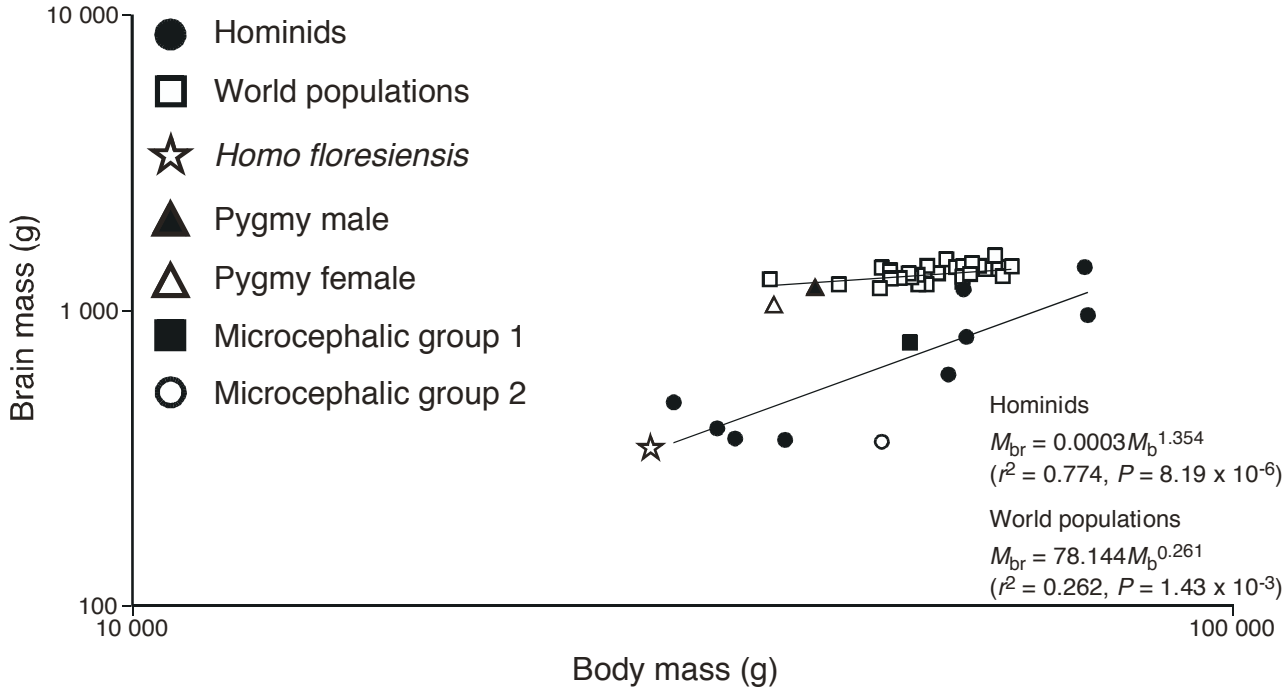
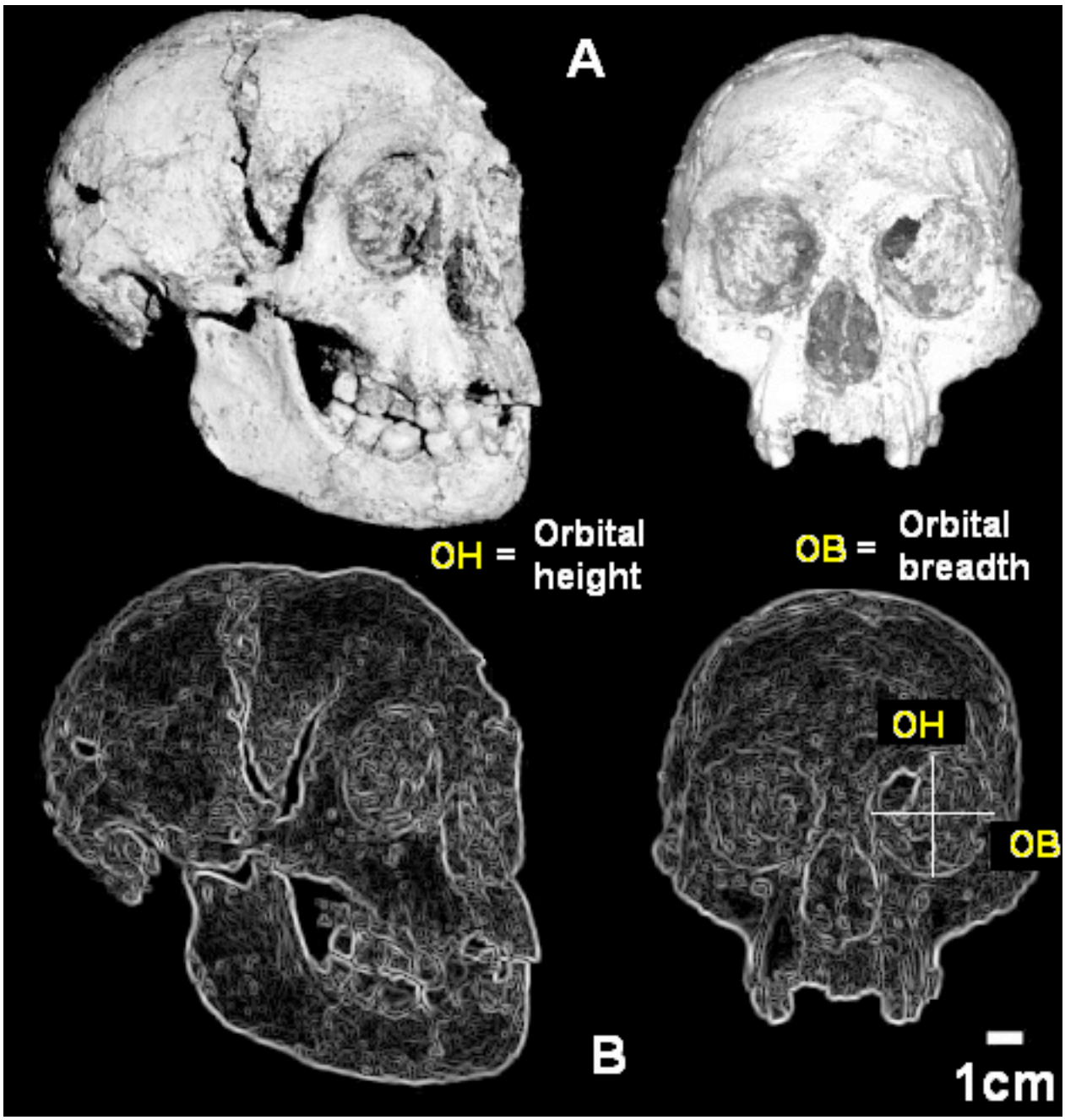

10 000
1 000
100
10 000
100 000
Brain mass (g)
Body mass (g)
Hominids
World populations
Homo floresiensis
Pygmy male
Pygmy female
Microcephalic group 1
Microcephalic group 2
Hominids
$M_{br} = 0.0003 M_b^{1.354}$
($r^2 = 0.774$, $P = 8.19 \times 10^{-6}$)
World populations
$M_{br} = 78.144 M_b^{0.261}$
($r^2 = 0.262$, $P = 1.43 \times 10^{-3}$)

Figure 31 (A) Original photographs of Homo floresiensis taken from Morwood et al (2005). (B) Edge detection reconstruction made from the original figures indicating the orbital margins from which measurements were taken to calculate body mass.


A
OH = Orbital height
OB = Orbital breadth
OH
OB
B
1cm

References:

Abbate, E., Albianelli, A., Azzaroli, A., Benvenuti, M., Tesfamariam, B., Bruni, P., Cipriani, N., Clarke, R.J., Ficarelli, G., Macchiarelli, R., Napoleone, G., Papini, M., Rook, L., Sagri, M., Tecle, T.M., Torre, D. & Villa, I. (1998) A one-million-year old *Homo* cranium from the Danakil (Afar) Depression of Eritrea. Nature 393:458-460.

Abouheif, E. & Fairbairn, D.J., (1997) A comparative analysis of allometry for sexual size dimorphism: Assessing Rensch's rule. American Naturalist 149:540–562.

Abouheif, E. (1999) A method for testing the assumption of phylogenetic independence in comparative data. Evolutionary and Ecological Research 1:895–909.

Abramoff, M.D., Magelhaes, P.J. & Ram, S. J. (2004) Image Processing with Image J. Biophotonics International 11:36-42.

Aiello, L.C. & Wood, B.A. (1994) Craniofacial variables as predictors of hominid body mass. American Journal of Physical Anthropology 95:409-426.

Aiello, L.C. & Dean, M.C. (1990) An Introduction to Human Evolutionary Anatomy. London: Academic Press.

Aiello, L.C. & Wheeler, P. (1995) The expensive tissue hypothesis. Current Anthropology 36:199-211.

Alexander, R.D., Hoogland, J.L., Howard, R.D., Noonan, K.M. & Sherman,P.W. (1979) Sexual dimorphism and breeding systems in pinnipeds, ungulates, primates and humans. In: Evolutionary Biology and Human Social Behavior: An Anthropological Perspective (N.A. Chagnon & W. Irons, eds.). Scituate, M.A.: Duxbury Press, pp. 402-435.

Allman, J.M. (1999) Evolving brains. New York: Scientific American Library.

Ankney, C.D. (1992) Sex differences in relative brain size: The mismeasure of woman, too? Intelligence 16:329-336.

Antonovics, J., & Tienderen, P.H. (1991) Ontoecogenophyloconstraints? The chaos of constraints terminology. TREE 6:141-172.

Asfaw, B., Gilbert, W.H., Beyene, J., Hart, W.K., Renne, P.R., WoldeGabriel, G., Vrba, E.S. & White, T.D. (2002) Remains of *Homo erectus* from Bouri, Middle Awash, Ethiopia. Nature 416:317-320.

Asfaw, B., White, T., Lovejoy, C.O., Latimer, B., Simpson, S. & Suwa, G. (1999) *Australopithecus garhi*: A new species of early hominid from Ethiopia. Science 284:629-635.

Bartley, A. J., Jones, D. W. & Weinberger, D. R. (1997) Genetic variability of human brain size and cortical gyral patterns. Brain 120:257–269.

Bauchot, R. & Stephan, H. (1961) Etude quantitative de quelques structures commisurales du cerveau des insectivores. Mammalia 25:314-341.

Beals, K.L., Smith, C.L. & Smith, M.D. (1984) Brain size, cranial morphology, climate and time machines. Current Anthropology 25:301-330.

Bigler, E. D., Johnson, S. C., Jackson, C. & Blatter, D. D. (1995) Aging, brain size and IQ. Intelligence 21:109–119.

Blessing, W.W., Chalmers, J.P. & Howe, P.R.C. (1978) Distribution of catecholamine-containing cell bodies in the rabbit central nervous system. Journal of Comparative Neurology 179:407-424.

Blinkov, S.M., & Glezer, I.I. (1968) The human brain in figures and tables: A quantitative handbook. New York: Plenum Press.

Blumenberg, B. (1985) Population characteristics of extinct hominid endocranial volume. American Journal of Physical Anthropology 68:269-280.

Borgognini Tarli, S.M. & Repetto, E. (1986) Methodological considerations on the study of sexual dimorphism in past human populations. Human Evolution 1:51-56.

Brandes, K. (1927) Liquorverhaltnisse an der Leiche und Hirnschwellung. Frankfurt Zeitschrift Pathologie 35:274-301.

Brown, P., Sutikna, T., Morwood, M., Soejono, R., Jatmiko, Z., Saptomo, E. & Due, R. (2004) A new small bodied hominin from the Late Pleistocene of Flores, Indonesia. Nature 431:1055–1061.

Brumm, A., Aziz, F., van den Bergh, G.D., Morwood, M.J., Moore, M.W., Kurniawan, I., Hobbs, D.R. & Fullagar, R. (2006) Early stone technology on Flores and its implications for *Homo floresiensis*. Nature 441:624–628.

Burt, D.B. (2001) Evolutionary stasis, constraint and other terminology describing evolutionary patterns. Biological Journal of Linnean Society 72:509-517.

Calder, WA. (1984) Size, Function, and Life History. Cambridge: Harvard University Press.

Carrier, D.R. (1987) The evolution of locomotor stamina in tetrapods; circumventing a mechanical constraint. Paleobiology 13:326-341.

Catania, K.C., Lyon, D.C., Mock, O.B. & Kaas, J.H. (1999) Cortical organization in shrews: evidence from five species. Journal of Comparative Neurology 410:55-72.

Catania, K.C., Collins, C.E. & Kaas, J.H. (2000) Organization of sensory cortex in the east African hedgehog (*Atelerix albiventris*). Journal of Comparative Neurology 421:256-274.

Caviness, V.S., Lange, N.T., Makris, N., Herbert, M.R. & Kennedy, D.N. (1999) MRI-based brain volumetrics: emergence of a developmental brain science. Brain and Development 21:289-295.

Changizi, M.A. & Shimojo, S. (2005) Parcellation and area-area connectivity as a function of neocortex size. Brain Behavior and Evolution 66:88-98.

Cheverud, J.M., Dow, M., & Leutenegger, W. (1985) The quantitative assessment of phylogenetic constraints in comparative analyses: sexual dimorphisms in body weight among primates. Evolution 39:1335-1351.

Chrzanowska, G., & Beben, A. (1973) Weight of the brain and body height in man between the ages of 20 and 89 years. Folia Morphologica (Warsaw) 32:391–406.

Clancy, B., Darlington, R.B. & Finlay, B.L. (2001) Translating developmental time across mammalian species. Neuroscience. 105:7-17.

Clutton-Brock, T.H. (1985) Size, sexual dimorphism and polygamy in primates. In: Size and Scaling in Primate Biology (W.L. Jungers, ed.) New York: Plenum Press, pp. 211-237.

Culham, J.C. & Kanwisher, N.G. (2001) Neuroimaging of cognitive functions in human parietal cortex. Current Opinions in Neurobiology 11:157–163.

Da Silva, J.N., Fuxe, K. & Manger, P.R. (2006) Nuclear parcellation of certain immunohistochemically identifiable neuronal systems in the midbrain and pons of the Highveld molerat (*Cryptomys hottentotus*). Journal of Chemical Neuroanatomy 31:37-50.

Damuth, J. & MacFadden, B.J. (1990) Body size in mammalian Paleobiology: Estimation and Biological Implications. Cambridge: Cambridge University Press.

Dechow, P.C. (1983) Estimation of body weights from craniometric variables in baboons. American Journal of Physical Anthropology 60:113-123.

Dekaban, A. S. & Sadowsky, D. (1978) Changes in brain weights during the span of human life: relation of brain weights to body heights and body weights. Annals of Neurology 4:345–356.

Delson, E., Terranova, C.J., Jungers, W.L., Sargis, E.J., Jablonski, N.G. & Dechow, P.C. (2000) Body mass in Cercopithecidae (Primates, Mammalian): estimation and scaling in extinct and extant taxa. Anthropological papers of the American Museum of Natural History number 83. New York: American Museum of Natural History.

Desdevises, Y., Legendre, P., Azouzi, L. & Morand, S. (2003) Quantifying phylogenetically structured environmental variation. Evolution 57:2647-2652.

Diniz-Filho, J.A.F., de Sant'Ana, C.E.R. & Bini, L.M. (1998) An eigenvector method for estimating phylogenetic inertia. Evolution 52:1247-1262.

Dubois, E. (1897) Sur le rapport de l' encéphale avec la grandeur du corps chez les Mammifères. Bulletin de la Societe anthropologie Paris, 4e série 8:337–374.

Dunbar, R.I.M. (1992) Neocortex size as a constraint on group size in primates. Journal of Human Evolution 22:469-493.

Dunbar, R.I.M. (1998) The social brain hypothesis. Evolutionary Anthropology. 6:178-190.

Elton, S., Bishop, L.C. & Wood, B. (2001) Comparative context of Plio-Pleistocene hominid brain evolution. Journal of Human Evolution 41:1-27.

Everitt, B.J., Sirkiä, T.E., Roberts, A.C., Jones, G.H. & Robbins, T.W. (1988) Distribution and some projections of cholinergic neurons in the brain of the common marmoset, *Callithrix jacchus*. Journal of Comparative Neurology 271:533-558.

Falk, D., Hildebolt, C., Smith, K., Morwood, M., Sutikna, T., Brown, P., Jatmiko Saptomo, E., Brunsden, B. & Prior, F. (2005) The brain of LB1, *Homo floresiensis*. Science 308:242–245.

Falk, D., Redmond, J. C., Guyer, J., Conroy, G.C., Recheis, W., Weber, G.W. & Seidler, H. (2000).Early hominid brain evolution: a new look at old endocasts. Journal of Human Evolution 38:695–717.

Falk, D., Hildebolt, C., Smith, K., Morwood, M.J., Jatmiko, Saptomo, E.W., Imhof, H., Seidler, H., Prior, F. (2007) Brain shape in human microcephalics and *Homo floresiensis*. Proc. Natl. Acad. Sci. USA. 104:2513-2518.

Felsenstein, J. (1985) Phylogenies and the comparative method. American Naturalist 125:1-15.

Felten, D.L., Laties, A.L. & Carpenter, M.B. (1974) Monoamine-containing cell bodies in the squirrel monkey brain. American Journal of Anatomy 139:153-166.

Fennema-Notestine, C., Ozyurt, I.B., Clark, C.P., Morris, S., Bischoff-Grethe, A., Bondi, M.W., Jernigan, T.L., Fischl, B., Segonne, F., Shattuck, D.W., Leahy, R.M., Rex, D.E., Toga, A.W., Zou, K.H. & Brown, G.G. (2005) Quantitative Evaluation of Automated Skull-stripping Method Applied to Contemporary and Legacy Images, Effects of Diagnosis, Bias Correction, and Slice Location. Human Brain Mapping 27:99-113.

Finlay, B.L. & Darlington, R.B. (1995) Linked regularities in the development and evolution of mammalian brains. Science 268:1578-84.

Finlay, B.L., Darlington, R.B. & Nicasto, N. (2001) Developmental structure in brain evolution. Behavioural and Brain Sciences 24:263-308.

Fleagle, J.G. (1999) Primate Adaptation and Evolution, 2nd edition. New York: Academic Press.

Gans, C. (1989) On phylogenetic constraints. Acta Morphologica Scandinavia 27:133-138.

Garland, T. & Ives, A. (2000) Using the past to predict the present: confidence intervals for regression equations in phylogenetic comparative methods. American Naturalist 155:346-364.

Garland, T., Harvey, P.H. & Ives, A.R. (1992) Procedures for the analysis of comparative data using phylogenetically independent contrasts. Systematic Biology 41:18-32.

Garver, D.L. & Sladek, J.R. (1975) Monoamine distribution in primate brain. I. Catecholamine-containing perikarya in the brain stem of *Macaca speciosa.* Journal of Comparative Neurology 159:289-304.

Giedd, J.N., Snell, J. W., Lange, N., Rajapakse, J. C., Casey, B. J., Kozuch, P. L., Vaituzis, A. C., Vauss, Y. C., Hamburger, S. D., Kaysen, D. & Rapoport, J. L. (1996) Quantitative magnetic resonance imaging of human brain development: Ages 4-18. Cerebral Cortex 6:551-560.

Gingerich, P.D. (1977) Correlation of tooth size and body size in living hominoid primates, with a note on relative brain size in *Aegyptopithecus* and *Proconsul*. American Journal of Physical Anthropology 47:395-398.

Gingerich, P.D. & Schoeninger, M.J. (1979) Patterns of tooth size variability in the dentition of primates. American Journal of Physical Anthropology 51:457-465.

Gittleman, J.L., Anderson, C.G., Kot, M. & Luh, H.K. (1996) Phylogenetic lability and rates of evolution: A comparison of behavioral, morphological and life history traits. In: Phylogenies and the Comparative Method in Animal Behaviour (E.P. Martins, ed.). Oxford: Oxford University Press, pp. 166-205.

Gould, S.J. (1975) On the scaling of tooth size in mammals. American Zoologist 15:351-362.

Gould, S.J. (1978) Morton's ranking of races by cranial capacity. Science 200:503-509.

Gould, S.J. (1981) Mismeasure of man. New York: Norton.

Gould, S.J. (2000) The structure of evolutionary theory. Cambridge MA: Belknap Press.

Gould, S.J. & Lewontin, R.C. (1979) The spandrels of San Marco and the Panglossian paradigm: a critique of the adaptionist program. Proceedings of the Royal Society of London. 205B:581-598.

Grafen, A. (1989) The phylogenetic regression. Philosophical Transactions of the Royal Society of London B 326:119-157.

Grausz, H.M., Leakey, R.E., Walker, A.C. & Ward, C.V. (1988) Associated cranial and postcranial bones of Australopithecus boisei. In: Evolutionary history of the "Robust" Australopithecines (F.E.Grine, ed.). New York: Aldine de Gruyter, pp. 127-132.

Gross, B.R. (1990) The case of Phillipe Rushton. Academic Questions 3:35-46.

Gurche, J.A. (1982) Early primate brain evolution. In: Primate brain evolution (E. Armstrong & D. Falk, eds.). New York: Plenum Press.

Stephan, H., Frahm, H. & Baron, G. (1981) New and revised data on volumes of brain structures in insectivores and primates. Folia Primatologia. 35:1–29.

Hammer, O., Harper, D.A.T. & Ryan, P.D. (2001) PAST: Paleontological Statistics software package for education and data analysis. Paleontologia Electronica 4:9.

Hartmann, P., Ramseier, A., Gudat, F., Mihatsch, M.J., Polasek, W. & Geisenhoff, C. (1994) Das Normgewicht des Gehirns beim Erwachsenen in Abhängigkeit von Alter, Geschlecht, Körpergrösse und Gewicht. Pathologie 15:165–170.

Harvey, P.H. & Pagel, M.D. (1991) The comparative method in evolutionary biology. Oxford UK: Oxford University Press.

Harvey, P.H. & Rambaut, A. (2000) Comparative analyses for adaptive radiations. Philosophical Transactions of the Royal Society of London B 355:1599-1605.

Haug, H. (1984) Macroscopic and microscopic morphometry of the human brain and cortex. A survey in the light of new results. Brain Pathology 1:123–148.

Henneberg, M. (1987) Hominid cranial capacity change through time: a Darwinian process. Human Evolution. 2:213-220.

Hennenberg, M. (1990) Brain size/body weight variability in *Homo sapiens*: Consequences for interpreting hominid evolution. Homo 39:121-130.

Henneberg, M. & De Miguel, C. (2001) Variation in hominid brain size: how much is due to method? Homo. 52:3-58.

Henneberg, M., Budnik, A., Pezacka, M. & Puch, A.E. (1985) Head size, body size and intelligence: intraspecific correlations in *Homo sapiens*. Homo 36:207-218.

Herndon, J.G., Tigges, J., Klumpp, S.A. & Anderson, D.C. (1998) Brain weight does not decrease with age in adult rhesus monkeys. Neurobiology of Aging 19:267- 272.

Ho, K-C., Roessmann, U., Straumfjord, J. V., & Monroe, G. (1980) Analysis of brain weight. II. Adult brain weight in relation to body height, weight, and surface area. Archives of Pathology and Laboratory Medicine 104: 640–645.

Holloway, R.L. (1980) Within species brain-body weight variability: A re-examination of the Danish data and other primate species. American Journal of Physical Anthropology 53:109-121.

Holloway, R.L. (1983). Human Brain evolution: A search for units, models and synthesis. Canadian Journal of Anthropology 3:215-232.

Hopkins, W.D., Marino, L., Rilling, J.K. & MacGregor, L.A. (1998) Planum temporale asymmetries in great apes as revealed by magnetic resonance imaging (MRI). Neuroreport 9:2913-2918.

Jacobowitz, D.M. & McLean, P.D. (1978) A brainstem atlas of catecholamine neurons and serotonergic perikarya in a pygmy primate (*Cebuella pygmaea*). Journal of Comparative Neurology 177:397-416.

Jerison, H.J. (1973) Evolution of the brain and intelligence. New York: Academic Press.

Jerison, H.J. (1979) Brain, body and encephalisation in early primates. Journal of Human Evolution 8:615-635.

Johnson, M.S. (1987) Adaptation and rules of form: chirality and shape in *Partula suturalis*. Evolution 41:672-675.

Jones, E.G. (1998) The thalamus of primates. In: The Primate Nervous System, Part II. Handbook of Chemical Neuroanatomy, vol. 14 (F.E. Bloom, A. Björklund, & T. Hökfelt, eds.). Amsterdam: Elsevier, pp. 1–298.

Jungers, W.L. (1985) Body size and scaling of limb proportions in primates. In: Size and Scaling in Primate Biology (W.L. Jungers, ed.). New York: Plenum Press, pp. 345-381.

Kaas, J.H. (1995) The evolution of isocortex. Brain Behavior and Evolution 46:187-196.

Kaas, J.H. (2004) Early visual areas: V1, V2, V3, DM, DL, and MT. In: The Primate Visual System (J.H. Kaas, & C.E. Collins, eds.). Boca Raton: CRC Press, pp. 139–159.

Kaas, J.H. (2006) Evolution of the neocortex. Current Biology 16:R910-R914.

Kappelman, J. (1996) The evolution of body mass and relative brain size in fossil hominids. Journal of Human Evolution 30:243-276.

Karasawa, N., Takeuchi, T., Yamada, K., Iwasa, M. & Isomura, G. (2003) Choline acetyltransferase positive neurons in the laboratory shrew (*Suncus murinus*) brain: coexistence of ChAT/5-HT (Raphe dorsalis) and ChAT/TH (Locus coeruleus). Acta Histochemica Cytochemica 36:399-407.

Karlen, S.J. & Krubitzer, L. (2006) The evolution of the neocortex in mammals: intrinsic and extrinsic contributions to the cortical phenotype. Novartis Foundation Symposium 270:146-159.

Kay, R.F. (1975) The functional adaptations of primate molar teeth. American Journal of Physical Anthropoloy 43:195-215.

Kay, R.F. & Simons, E.L. (1980) The ecology of Oligocene African Anthropoidea. International Journal of Primatology 1:21-37.

Kelley, J. (1993) Taxonomic implications of sexual dimorphism in Lufengpithecus. In: Species, Species Concepts, and Primate Evolution (W.H. Kimbel & L.B. Martin, eds.). New York: Plenum Press, pp. 429-458.

Kitahama, K., Nagatsu, I. & Pearson, J. (1994) Catecholamine systems in mammalian midbrain and hindbrain themes and variations. In: Phylogeny and Development of Catecholamine Systems in the CNS of Vertebrates (W.J.A.J. Smeets & A. Reiner, eds.). Cambridge: Cambridge University Press, pp. 183–205.

Krubitzer, L.A. & Kahn, D. (2003) Nature versus nurture revisted: An old idea with a new twist. Progress in Neurobiology 70:33-52.

Krubitzer, L., Manger, P., Pettigrew, J. & Calford, M. (1995a) The organization of somatosensory cortex in monotremes: in search of the prototypical plan. Journal of Comparative Neurology 351:261-306.

Krubitzer, L., Clarey, J., Tweedale, R., Elston, G. & Calford, M. (1995b) A redefinition of somatosensory areas in the lateral sulcus of macaque monkeys. Journal of Neuroscience 15:3821-3939.

Krubitzer, L., Kunzle, H. & Kaas, J.H. (1997) Organization of sensory cortex in a Madagascan insectivore, the tenrec (*Echinops telfairi*). Journal of Comparative Neurology 379:399-414.

Laird, A.K. (1967) Evolution of the human growth curve. Growth 31:345-355.

Lauder, G.V. & Liem, K.F. (1989) The role of historical factors in the evolution of complex organismal functions. In: Complex Organismal Functions: Integration and Evolution in Vertebrates (D.B. Wake & G. Roth, eds.). New York: John Wiley & Sons Ltd, pp. 63 -78.

Lee, S. & Wolpoff, M.H. (2003) The pattern of evolution in Pleistocene human brain size. Paleobiology 29:186-196.

Leigh, S.R. (1992) Cranial capacity evolution in *Homo erectus* and early *Homo sapiens*. American Journal of Physical Anthropology 87:1-13.

Leigh, S.R. & Cheverud, J.M. (1991) Sexual dimorphism in the baboon facial skeleton. American Journal of Physical Anthropology 84:193-208.

Leroi, A.M., Rose, M.R. & Lauder, G.V. (1994) What does the comparative method reveal about adaptation? American Naturalist 143:381-402.

Lestrel, P.E. & Read, D.W. (1973) Hominid cranial capacity versus time: a regression approach. Journal of Human Evolution 2:405-411.

Leutnegger, W. & Masterson, T.J. (1989) The ontogeny of sexual dimorphism in the cranium of Bornean orang-utans (*Pongo pygmaeus pygmaeus*): II. Allometry and heterochrony. Zeitschrift Morphologie Anthropologie 78:15-24.

Lewin, R. (1999) Human evolution: An illustrated introduction. 4th edition. New York: Blackwell Sciences, Inc.

Lewontin, R.C. (1992) Biology as Ideology: The Doctrine of DNA. New York: Harper Perennial.

Lin, L.I. (1989) Concordance correlation co-efficient to evaluate reproducibility. Biometrics 45:255-268.

Lovejoy,C.O. (1981) The origin of man. Science 211:341-350.

Manger, P.R. (2005a) Establishing order at the systems level in mammalian brain evolution. Brain Research Bulletin 66:282-289.

Manger, P.R. (2005b) Evolution of the modern human brain: P.V. Tobias's Hominization, Ockham's Razor and Asimov's Elephant. In: Voyages in Science: Essays by South African Anatomists in Honour of Phillip V. Tobias's 80th Birthday (G. Strkalj, N. Pather & B, Kramer, eds.). Pretoria, South Africa: Content Solutions, pp. 51-84.

Manger, P.R. (2006) An examination of cetacean brain structure with a novel hypothesis correlating thermogenesis to the evolution of a big brain. Biological Reviews of the Cambridge Philosophical Society 81:293-338.

Manger, P.R, Fahringer, H.M., Pettigrew, J.D. & Siegel, J.M. (2002a) Distribution and morphology of cholinergic neurons in the brain of the monotremes as revealed by ChAT immunohistochemistry. Brain, Behavior and Evolution 60:275-297.

Manger, P.R, Fahringer, H.M., Pettigrew, J.D. & Siegel, J.M. (2002b) Distribution and morphology of catecholaminergic neurons in the brain of monotremes as revealed by tyrosine hydroxylase immunohistochemistry. Brain, Behavior and Evolution 60:298-314.

Manger, P.R, Fahringer, H.M., Pettigrew, J.D. & Siegel, J.M. (2002c) Distribution and morphology of serotonergic neurons in the brain of the monotremes. Brain, Behavior and Evolution 60:315-332.

Manger, P.R., Fuxe, K., Ridgway, S. & Siegel, J.M. (2004) The distribution and morphological characteristics of catecholamine cells in the diencephalon and midbrain of the bottlenose dolphin (*Tursiops truncatus*). Brain, Behavior and Evolution 64:42-60.

Manger, P.R., Engler, G., Moll, C. & Engel, A. (2005) The anterior ectosylvian visual area of the ferret: a homologue for an enigmatic visual cortical area of the cat? European Journal of Neuroscience 22:706-714.

Manjunath, N. (2002) Estimation of cranial volume in dissecting room cadavers. Journal of the Anatomical Society of India 51:168-172.

Marshall, J. (1892) On the relation between the weight of the brain and its parts, and the stature and mass of the body in man. Journal of Anatomy and Physiology 26:445–500.

Martin, R.D. (1979) Phylogenetic aspects of prosimian behaviour. In: The study of prosimian behaviour (G.A. Doyle & R.D. Martin, eds.). New York: Academic Press, pp. 45-77.

Martin, R.D. (1982) Allometric approaches to the evolution of the primate nervous system. In: Primate brain evolution (E. Armstrong & D. Falk, eds.). New York: Plenum Press, pp. 39-57.

Mayr, E. (2002). What Evolution Is. London: Phoenix.

McHenry, H.M. (1974) How large were the australopithecines. American Journal of Physical Anthopology 40:329-340.

McHenry, H.M. (1992) How big were early Hominids. Evolution and Anthropology 13:15-20.

McHenry, H.M. (1994) Behavioural implications of early hominid body size. Journal of Human Evolution 27:77-87.

McHenry, H.M. & Berger, L.R. (1998) Body proportions in *Australopithecus afarensis* and *A.africanus* and the origin of the genus Homo. Journal of Human Evolution 35:1-22.

McHenry, H.M. & Coffing, K. (2000) Australopithecus to Homo: transformations in body and mind. Annual Review of Anthropology 29:125-46.

McKee, J. (2000) The riddled chain. Chance, coincidence, and chaos in human evolution. London: Rutgers University Press.

Mesulam, M.M., Geula, C., Bothwell, M.A. & Hersch, L.B. (1989) Human reticular formation: cholinergic neurons of the peduncuopontine tegmental nuclei and some cytochemical comparisons of forebrain cholinergic neurons. Journal of Comparative Neurology 281:611-633.

Mettler, F.A. (1995) Culture and the structural evolution of the neural system. James Arthur Lecture on the Evolution of the Human Brain. New York: American Museum Natural History.

Morwood, M., Brown, P., Jatmiko Sutikna, T., Saptomo, E., Westaway, K., Due, R., Roberts, R., Maeda, T., Wasisto, S. & Djubiantono, T. (2005) Further evidence for small-bodied hominins from the Late Pleistocene of Flores, Indonesia. Nature 437:1012–1017.

Murray, H.M., Dominguez, W.F. & Martinez, J.E. (1982) Catecholaminergic neurons in the brain stem of tree shrew (*Tupaia*). Brain Research Bulletin 9:205-215.

Nanney, D.L., Nyberg, D., Chen, S.S. & Meyer, E.B. (1980) Cytogeometric Constraints in Tetrahymena Evolution: Contractile Vacuole Pore Positions in Nineteen Species of the Tetrahymena pyriformis Complex. American Naturalist 115:705-716.

Nunn, C. & Barton, R. (2001) Comparative methods for studying primate adaptation and allometry. Evolutionary Anthropology 10:81-89.

Olivier, G. & Tissier, H. (1975) Correlations between major cranial diameters of man and pongidae. In: Primate functional morphology and evolution (R.H. Tuttle, ed.). The Hague: Elsevier, pp. 465-479.

Oxnard,C.E. (1987) Fossils, Teeth and Sex: New Perspective in Human Evolution. Seattle: University of Washington Press.

Pakkenberg, H. & Voigt, J. (1964). Brain weight of the Danes: A forensic material. Acta Anatomica.56:297–307.

Perrin, N. & Rubin, J.F. (1990) On dome-shaped norms of reaction for size-to-age at maturity in fishes. Functional Ecology 4:53-57.

Peters, M. (1993) Still not convincing evidence of a relation between brain size and intelligence in humans. Canadian Journal of Experimental Psychology 47:751-756.

Peters, M., Jäncke, L., Staiger, J.F., Schlaug, G., Huang, Y. & Steinmetz, H. (1998) Unsolved problems in comparing brain sizes in *Homo sapiens*. Brain and Cognition. 37:254-285.

Pigliucci., M. & Kaplan, J. (2000) The fall and rise of Dr Pangloss: adaptationism and the Spandrels paper 20 years later. TREE 15:66-70.

Pirie, R.L. (1978) Allometric scaling in the post-canine dentition with reference to primate diets. Primates 19:583-591.

Plavcan, J.M. (2003) Scaling relationships between craniofacial sexual dimorphism and body mass dimorphism in primates: Implications for the fossil record. American Journal of Physical Anthropology 120:38-60.

Plavcan, J.M. & van Schaick, C.P. (1992) Intrasexual competition and canine dimorphism in anthropoid primates. American Journal of Physical Anthropology 87: 461-477.

Purvis, A. (1995) A composite estimate of primate phylogeny. Philosophical Transactions of the Royal Society of London B 348:405-421.

Radinsky, L.B. (1977) Early primate brains: facts and fiction. Journal of Human Evolution 6:79-86.

Radinsky, L.B. (1982) Some cautionary notes on making inferences about relative Brain size. In: Primate brain evolution (E. Armstrong & D. Falk, eds.). New York: Plenum Press, pp. 29-39.

Ramachandran, V.S. (1997) Why do gentlemen prefer blondes? Medical Hypotheses 48:19-20.

Ravosa, M.J. & Ross, C.F. (1994) Craniodental allometry and heterochrony in two howler monkeys: *Alouatta seniculus* and *A. palliate*. American Journal of Primatology 33:277-299.

Raz, N., Gunning, F. M., Head, D., Dupuis, J. H., McQuain, J., Briggs, S. D., Loken, W. J., Thornton, A. E., & Acker, J. D. (1997) Selective aging of the human cerebral cortex observed in vivo: differential vulnerability of the prefrontal gray matter. Cerebral Cortex 7:268–282.

Rice, S.H. (1997) The analysis of ontogenetic trajectories: when a change in size or shape is not heterochrony. Proceedings of the National Academy of Sciences, USA 94:907-912.

Rice, S.H. (2002) The role of heterochrony in primate brain evolution. In: Human Evolution through developmental Change (N. Minugh-Purvis & K.J. McNamara, eds.). Baltimore: John Hopkins University Press.

Rightmire, G.P., Lordkipanidze, D. & Vekua, A. (2006) Anatomical descriptions, comparative studies and evolutionary significance of the hominin skulls from Dmanisi, Republic of Georgia. Journal of Human Evolution 50:115-141.

Rilling, J.K. & Insel, T.R. (1998) Evolution of the cerebellum in primates: differences in relative volume among monkeys, apes and humans. Brain, Behavior and Evolution 52:308-314.

Rilling, J.K. & Seligman, R.A. (2002) A quantitative morphometric comparative analysis of the primate temporal lobe. Journal of Human Evolution 42:505-533.

Roland, P., Svensson, G., Lindeberg, T., Risch, T., Baumann, P., Dehmel, A., Fredricksson, J., Halldorson, H., Forsberg, L., Young, J. & Zilles, K. (2001) A database generator for human brain imaging. Trends in Neuroscience 24:562-564.

Romanes, G.J. (1996) Cunningham's Manual of Practical Anatomy, Fifteenth edition. Vol. 3. Head and Neck. Oxford: Oxford University Press.

Rorden, C. & Brett, M. (2000) Stereotaxic display of brain lesions. Behavioral Neurology 12:191-200.

Rosa, M.G. (1997) Visuotopic organization of primate extrastriate cortex. In: Cerebral Cortex, vol. 12: Extrastriate Cortex in Primates (K.S. Rockland, J.H. Kaas & A. Peters, eds.). New York: Plenum Press, pp. 127–203.

Rosa, M.G. (1999) Topographic organization of extrastriate areas in the flying fox: implications for the evolution of mammalian visual cortex. Journal of Comparative Neurology 411:503–523.

Rosa, M.G. & Tweedale, R. (2001) The dorsomedial visual areas in New World and Old World monkeys: homology and function. European Journal of Neuroscience 13:421–427.

Rosa, M.G. & Tweedale, R. (2005) Brain maps, great and small: lessons from comparative studies of primate visual cortical organization. Philosophical Transactions of the Royal Society of London B 360:665-691.

Rosa, M.G., Casagrande, V.A., Preuss, T. & Kaas, J.H. (1997) Visual field representation in striate and extrastriate cortices of a prosimian primate (*Galago garnetti*). Journal of Neurophysiology 77:3193-3217.

Rossi, L.N., Candini, G., Scarlatti, G., Rossi, G., Prina, E. & Alberti, S. (1987) Autosomal dominant microcephaly without mental retardation. American Journal of Diseases of Children 141: 655-659.

Ruff, C.B., Trinkaus, E. & Holliday, T.W. (1997) Body mass and encephalisation in Pleistocene Homo. Nature 387:173-176.

Ruff, C.B., Trinkaus, E., Walker, A. & Larsen, C.S. (1993) Postcranial robusticity in Homo, I: temporal trends and mechanical interpretation. American Journal of Physical Anthropology 91:21-53.

Rushton, J.P. (1988) Race differences in behavior: A review and evolutionary analysis. Personality and Individual Differences 9:1009-1024.

Rushton, J.P. (1991) Mongoloid-Caucosoid differences in brain size from military samples. Intelligence. 15: 351-359.

Rushton, J.P. (1992) Cranial capacity related to sex, rank, and race in a stratified random sample of 6, 325 U.S. military personnel. Intelligence 16:401-413.

Rushton, J.P. (1994) Sex and race differences in cranial capacity from International Labour Office data. Intelligence 19:281-294.

Rushton, J.P. (1997) Race, evolution and behavior. A life-history perspective. New Brunswick, NJ: Transaction.

Rushton, J.P. (1998) Cranial size and IQ in Asian Americans from birth to age seven. Intelligence 25:7-20.

Rushton, J.P. & Osborne, R.T. (1995) Genetic and environmental contributions to capacity in black and white adolescents. Intelligence 20:1–13.

Rushton, J.P. & Ankney, C.D. (1996) Brain size and cognitive ability: Correlations with age, sex, social class and race. Psychonomic Bulletin and Review 3:21-36.

Sassaman, E.A. & Zartler, A.S. (1982) Mental retardation and head growth abnormalities. Journal of Pediatric Psychology 7:149-156.

Satoh, K. & Fibiger, H.C. (1985) Distribution of central cholinergic neurons in the baboon (*Papio papio*). I. General morphology. Journal of Comparative Neurology 236:197-214.

Schoenemann, P. T. & Allen, J.S. (2006) Scaling of brain and body weight within modern and fossil hominids: implications for the Flores specimen. American Journal of Physical Anthropology 129(supp. 42):159-160.

Schofield, S.P.M. & Everitt, B.J. (1981) The organization of catecholamine-containing neurons in the brains of the rhesus monkey (*Macaca mulatta*). Journal of Anatomy 132:391-418.

Schultz, A.H. (1940) The size of the orbit and of the eye in primates. American Journal of Physical Anthropology 26:389–408.

Sells, C.J. (1977) Microcephaly in a normal school population. Pediatrics 59:262-265.

Semendeferi, K. & Damasio, H. (2000) The brain and its main anatomical subdivisions in living hominoids using magnetic resonance imaging. Journal of Human Evolution 38:317-332.

Senut, B., Pickford, M., Gommery, D., Mein, P., Chebot, K. & Coppens, Y. (2001) First hominid from the Miocene (Lukeino Formation, Kenya). Compte Rendus de Acadamie Science Paris 337:137-144.

Seyfarth, R.M. & Cheney, D.L. (1992) Meaning and mind in monkeys. Scientific America December: 122-128.

Shattuck, D.W. & Leahy, S.R. (2001) Brainsuite: An automated cortical surface identification tool. Medical Image Analysis 8:129-142.

Shine, R. (1989) Ecological causes for the evolution of sexual dimorphism: a review of the evidence. Quarterly Review of Biology 64:19-46.

Singh, I.P. & Bhasin, M.K. (1968) Anthropometry. Delhi, India: Central Electric Press.

Sjogren, P., Elmberg, J. & Berglind, S.A. (1988) Thermal preference in the pool frog Rana lessonae: impact on the reproductive behaviour of a northern fringe population. Holarctic Ecology 11:178-184.

Smeets, W.J.A.J. & González, A. (2000) Catecholamine systems in the brain of vertebrates: new perspectives through a comparative approach. Brain Research Reviews 33:308–379.

Smith, R.J. (1993) Logarithmic transformation bias in allometry. American Journal of Physical Anthropology 90:215-221.

Smith, S.M. (2000) Robust automated brain extraction. Hum Brain Mapping 17:143-155.

Sokal, R.R. & Rohlf, F.J. (1995) Biometry: the principals and practice of statistics in biological research. San Francisco, CA: W. H. Freeman and Co.

Spocter, M.A. & Manger, P.R. (2007). The use of cranial variables for the estimation of body weight in fossil hominids. In Press: American Journal of Physical Anthropology.

Spocter, M.A., Gallagher, A. & Manger, P.R. (2007) Quantitative magnetic resonance imaging of the endocranial volume in humans and other primates: Predicting fossil hominid brain weights. In Press. American Journal of Physical Anthropology

Stebbins, G.L. (1966) Processes of organic evolution. Prentice-Hall, INC., Englewood Cliffs, New Jersey.

Stephan, H., Frahm, H. & Baron, G. (1981) New and revised data on volumes of brain structures in insectivores and primates. Folia Primatologica. 35:1-29.

Steudel, K. (1980) New estimates of early hominid body size. American Journal of Physical Anthropology 52:63-70.

Tobias, P.V. (1968) Cranial capacity in anthropoid apes, *Australopithecus* and *Homo habilis*, with comments on skewed samples. South African Journal of Science 64:81-91.

Tobias, P.V. (1971) The brain in hominid evolution. Columbia University Press, New York.

Tobias, P.V. (1985) Single characters and the total morphological pattern redined: the sorcting effected by a selection of morphological features of the early hominids, pp. 94-101, in Delson, E. (Ed.): Ancestors: The hard evidence. New York.

Tobias, P.V (1994). The craniocerebral interface in Early Hominids. In INTEGRATIVE PATHS TO THE PAST. Paleoanthropological Advances in Honor of F. Clark Howell. (Eds.) Corruccini, R.S and Ciochon, R.L. Prentics Hall, Englewood Cliffs, New Jersey.

Tobias, P.V. (1995) The Communication of the Dead. Earliest Vestiges of the Origin of Articulate Language. 17th Kroon Lecture. Amsterdam: Stichthing Nederland Museum voor Anthropologie en Praehstorie.

von Neumann, J., Kent, R.H., Bellinson, H.R. & Hart, B.I. (1941) The mean square successive difference. Annals Mathematics ad Statistics 12:153–162.

Wagner, G.P. (1988) The influence of variation and of developmental constraints on the rate of multivariate phenotypic evolution. Journal of Evolutionary Biology 1:45-66.

Warburton, K. (1989) Ecological and phylogenetic constraints on body size in Indo-Pacific fishes. Environmental Biological Fisheries 24:13-22.

Werdelin, L. (1987) Jaw geometry and molar morphology in marsupial carnivores: analysis of a constraint and its macroevolutionary consequences. Paleobiology 13:342-350.

Westoby, M., Leishman, M.R. & Lord, J.M. (1995) On mis-interpreting the 'phylogenetic correction'. Journal of Ecology 83:531-534.

Williams, P.L., Bannister, L.H., Berry, M.M., Collins, P., Dyson, M., Dussek, J.E. & Ferguson, M.W.J. (1995) Gray's Anatomy. In: Skeletal system. 38th Edition (R.W. Soames, ed.). London: Churchill Livingstone, pp. 612.

Wilson, E.O. (1975) Sociobiology: The New Synthesis. Harvard MA: Harvard University Press.

Wood, B.A. (1992) Origin and evolution of the genus *Homo*. Nature 355:783-790.

Wood, B. & Collard, M. (1999) The human genus. Science 284:65-71.

Wu, C.W. & Kaas, J.H. (2003) Somatosensory cortex of prosimian galagos: physiological recording, cytoarchitecture, and corticocortical connections of anterior parietal cortex and cortex of the lateral suclus. Journal of Comparative Neurology 457: 263–292.

Wu, C.W., Bichot, N.P. & Kaas, J.H. (2000) Converging evidence from microstimulation, architecture, and connections for multiple motor areas in the frontal

and cingulate cortex of prosimian primates, Journal of Comparative Neurology 423:140–177.

Wynn, T. (1991) Archeological evidence for modern intelligence. In: The Origin of human behavior (R.A. Foley, ed.). London: Unwin Hyman, pp 52-66.

Yushkevich, P.A., Piven, J., Hazlett, H.C., Smith, G.R., Ho, S., Gee, J.C. & Gerig, G. (2006) User-guided 3D active contour segmentation of anatomical structures: Significantly improved efficiency and reliability. In Press: Neuroimage.

Ziehe, M. & Gregorius, H.R. (1988) Selection caused by self-fertilization. II. Ecological constraints on selfing advantage. Journal of Evolutionary Biology 1:233-253.

Zollikofer, C.P.E., Ponce de León, M.S., Lieberman, D.E., Guy, F., Pilbeam, D., Likius, A., Mackaye, H.T., Vignaud, P. & Brunet, M. (2005) Virtual reconstruction of *Sahelanthropus tchadensis*. Nature 434:755-759.