

**Prenatal androgen effects and social
evolution in haplorhine primates: Evidence
from the second-to-fourth digit ratio
(2D:4D)**

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by

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This thesis is dedicated to my father
Gary Christopher Brown

Abstract

Prenatal androgens play a key role in sexual differentiation. In humans and rhesus macaques prenatal androgens have been implicated in variation in the development of sexually selected behaviours and cognitive abilities in both males and females. The primacy of prenatal androgens in organising traits linked to sociality suggests great pertinence to the evolutionary substrate that underpins social behaviours in all higher primates.

In humans the second-to-fourth digit ratio (2D:4D) is a marker for prenatal androgen effects. Low 2D:4D has been indirectly linked to higher foetal androgens and high 2D:4D has been indirectly linked to lower foetal androgens. Individuals with low 2D:4D express higher competitiveness, promiscuity and more masculinised social cognitive abilities. Low 2D:4D is also more common in polygynous societies. This thesis uses 2D:4D as an anatomical biomarker with phylogenetic comparative methods to investigate co-variation between prenatal androgen effects and traits linked to sexual selection in haplorhine primates. In the process, measurements were taken of the 2nd and 4th digits of 1286 captive individuals from 74 species.

An intra-specific study of 2D:4D and dominance ranks in female rhesus macaques is presented and shows that low 2D:4D is more common in higher ranking females. The result suggests that prenatal androgens may be implicated in supporting dominance ranks across generations. A comparison of digit ratios in mother-infant dyads in the same cohort shows heritability of 2D:4D to be high; values are similar to humans and more distantly related taxa. Moving to inter-specific analyses: results enclosed provide the first robust evidence that 2D:4D generalise across a taxonomic group and striking parallels are shown between cross-species analyses and results from earlier human 2D:4D studies. In particular, evidence is presented that 2D:4D is lower in polygynous species with higher levels of intra-sexual competition and higher in pair-bonded species with lower levels of intra-sexual competition. These studies also show that 2D:4D co-varies with both core behavioural characteristics and androgen profiles in catarrhines: in comparison to the great apes, Old World monkeys exhibited low 2D:4D, higher intra-sexual competition and more reactive androgen profiles. These differences might reflect prenatal androgen effects on programming neuro-psychological pathways that potentiate social behaviours and social bonding patterns according to different levels of sexual selection.

Focussing on the great apes; 2D:4D is shown to increase from *Pongo sp.* to *Homo sapiens*. It is proposed that this reflects a decrease in prenatal androgens and androgen sensitivity across this clade. That being the case; fossilised digit bone ratios permitted prenatal androgen effects to be traced across extinct apes and hominins. The findings indicate that Miocene apes might have experienced a down-regulation of the androgen response. Reducing masculinisation is also detected across time in *Australopithecus afarensis*, but not *Ardipithecus ramidus*. Evidence from Middle and Late Pleistocene hominins indicates that prenatal androgen effects continued to decrease over evolutionary time. The most recent increase in digit ratios appears to have coincided with shifts in social organisation as modern humans entered a new 'adaptive space' with the advent of agriculture at the beginning of the Holocene. These changes are consistent with hypotheses proposing a 'feminisation' or 'domestication' event coinciding with reductions in dominance and competitive behaviours throughout hominin evolution, right up until relatively recent times.

Analyses of 2D:4D identify prenatal androgen effects as strong candidates in the evolution of sexually selected behaviours and social bonding in haplorhines. Application of 2D:4D could thereby improve our current understanding of primate social evolution if incorporated within research into the bio-behavioural processes that underpin sociality across taxonomic groups. Finally, because hominin evolution is so strongly associated with the emergence of specialised cognitive adaptations to cope with changes in social organization, 2D:4D could prove to be a window into our own past.

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Preface

Data were compiled and analysed Emma C. Nelson. The thesis and manuscripts were constructed and written by Emma C. Nelson. Sections of this thesis have been published elsewhere. Editorial assistance from co-authors was provided in the chapters below.

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Chapter 7

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Additional publications linked to this project

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Nelson, E., Manning, J.T. and Sinclair, A.G.M. 2006. Using the 2nd to 4th digit ratio (2D:4D) to sex cave art hand stencils: factors to consider. *Before Farming* (on-line), 2006/1. Article 6:1-7.

Burriss, R.P., Little, A.C. and Nelson, E. 2006. 2D:4D ratio and its relationship with sexually dimorphic facial characteristics and perceived dominance. *Archives of Sexual Behavior*, 36:377-384.

Chapter 1

Introduction

1.1: Introduction

From the moment we are born we are thrust into a social context and begin to learn about relationships; the way we learn and the way we respond is guided by our biological sex. To this end we are similar to most other primate species. Unlike other primates, however, we have a unique ability to effortlessly predict the behaviours of others and understand their emotions. This not only impacts the way we conduct our reproductive relationships and our friendships, but also how we structure our societies. These adaptations may be rooted in cellular processes that take place before we are born, the precursors of which evolved in order to prepare individuals for a journey through life within an extended social landscape. The interaction between prenatal developmental mechanisms and the socially complicated extra-uterine world may have played a pivotal role in primate sociality and human evolution. This thesis is about the evolution of one of those mechanisms: prenatal androgen effects.

Prenatal androgen effects are defined here as “*the combined effects of androgen hormones, such as testosterone, with cellular responsiveness to androgens*”. This cellular responsiveness is conferred by the androgen receptor gene that dictates the speed at which the cells respond to androgens.

1.1.1: Prenatal androgen effects (PAE) and sexual selection

Prenatal androgen effects (PAE) play a central role in sexual selection through their action on sexually dimorphic anatomical traits and behaviours. Darwin (1871) formulated his theory of sexual selection after observing the struggle of individuals (usually males) to compete for access to mates and the adaptations that emerge in order to facilitate mating. The principal of the theory follows; in animals that internally gestate their offspring it is the female that is responsible for carrying the pregnancy and nurturing the young, so, unlike males, females can always be sure that the offspring belong to them. It is this asymmetry in offspring investment and paternal uncertainty that leads to differences in male and female reproductive strategies; females focus their energy on gestation and infant care, but also by being discerning in their choice of mates (Trivers 1974; 1985; Møller & Swaddle 1997). For males it is more advantageous not to expend time and energy in paternal care but to focus on

mating with many females. For primates relatively long gestation periods and infant dependency mean that females become the limiting sex which intensifies competition between males. This is a somewhat idealised portrayal of sexual selection theory as male primates can be choosy about their mates and can invest in offspring, and females primates do compete over males (Geary 2002). The theory does, however, highlight the inherent conflict between male and female reproductive strategies.

The differing selective pressures on males and females lead to the evolution of sexually dimorphic characters that match the needs of male and female reproductive strategies (Geary 2002; Plavcan 2004). For example, male sexually selected characters that are linked to their higher PAE are mostly associated with intra-sexual competition (e.g., aggression, dominance, status, risk taking). Female traits associated with their lower PAE manifest in energy acquisition, increased nurturing skills and risk aversion (Geary 2002). These prenatally programmed characteristics bias developmental trajectories along male- and female-typical pathways and become accentuated through sex-linked play and social learning of gender roles. Sex-linked characteristics are expressed in similar ways in juveniles across primate species; higher PAE primes young males for more physical play and lower PAE facilitates more interest in infants in females (Wallen 2005; Collaer & Hines 1995; Hines 2010). These hormones are therefore centrally placed within the framework of sexual selection theory (e.g., Hines 2010; Knickmeyer & Baron-Cohen 2006).

1.1.1.1: PAE: Evidence from sexual differentiation

PAE play a key role in sexual differentiation in mammals. Sexual differentiation is achieved through masculinisation¹ of the foetus (see Wallen & Baum 2002; Wallen 2005). The expression of genes on the Y-chromosome in males triggers a sexually differentiating cascade of events that begins with the development of the primordial gonads into testes under the control of the *SRY* gene on the Y-chromosome. Androgens secreted by the testes (e.g., testosterone; dihydrotestosterone) masculinise the endocrine system by organising the hypothalamic–pituitary–gonadal axis (HPG) towards a male functional pattern (see Pfeiffer 1936; see Fowden & Forhead 2009). Without this process the gonads develop into ovaries and the low androgen environment programmes the endocrine system for feminisation of the

¹ Phoenix *et al.* (1959) used the term masculinisation for the organisational effects of prenatal androgens. The terminology has since been refined; PAE are defined as having *masculinising* effects, referring to an increase in male-like characters and behaviours, but also *defeminising* effects referring to a decrease in female-like characters and behaviours (Whalen 1974; Thornton *et al.* 2009). In this thesis Phoenix *et al.*'s original terminology is used.

foetus (Pfeiffer 1936). The ovaries in females are largely quiescent during this period, although they have the potential to produce both androgens and estrogens. Female foetuses are also potentially exposed to androgens produced by their own adrenal glands (Knickmeyer & Baron-Cohen 2006). The intra-uterine hormonal environment may also be altered by endocrine signals from the placenta in response to external stimuli; these signals can be sex-specific (i.e., differ in accordance with the sex of the foetus; Fowden *et al.* 2008; Fowden & Forhead 2009). It is unclear if androgens cross the healthy placenta in humans (Manning 2007a, p 18). Androgens do, however, cross the healthy placenta in other mammals including non-human primates (see Wallen & Baum 2002; Wallen 2005; Dloniak *et al.* 2006). In smaller mammals, such as rodents, the brain is masculinised by the conversion of androgens to estrogens via a process called aromatisation (see Fitch & Bimonte 2002). However, brain masculinisation in primates largely occurs as a direct response to prenatal androgens (Wallen & Baum 2002; Wallen 2005; also see Kragie 2002). Although there is evidence to suggest that early genetic effects may also be implicated in the masculinisation process (Arnold 1996) the evidence strongly identifies PAE as the primary factors that masculinises higher primates.

1.1.1.2: PAE: Evidence from macaque studies

Fifty years ago there was an assumption that although sexual differentiation in primary sexual characteristics (e.g., the reproductive system) happened before birth, masculine and feminine social behaviours were believed to be learnt (see Thornton *et al.* 2009 Wallen & Hassett 2009). This perception had to be radically revised when it was shown that doses of androgens given to pregnant female guinea pigs (a rodent that gives birth to precocial² young) permanently masculinised both the anatomy and the behaviours of their female offspring (Phoenix *et al.* 1959). Phoenix *et al.* (1959) describe how prenatal androgens ‘organise’³ the foetal tissues at the cellular-level during ‘critical phases’ when tissues are receptive to physiological alteration by PAE (Fowden & Forhead 2009). Organisation is the term used for the mechanisms that permanently alter tissues (Fowden & Forhead 2009). In response to developmental signals steroid hormones bind to their receptors on the surface of cells. These effects alter the form and function of tissues via processes of cell growth, cell proliferation and cell death (Fitch & Bimonte 2002). At maturity the tissues undergo

² Prococial animals are those that are born in a relatively mature state; their eyes are open and they are able to move locomote unaided. This contrasts the altricial state in which young are born helpless, underdeveloped, with eyes are closed (Gould 1977).

³ The term ‘programming’ is also used to refer to organisational processes (MacLusky & Naftolin 1981; Lucas 1991).

additional changes under the influence of circulating sex hormones; this process is termed 'activation'. Activation leads to the expression of sex-typical adult tissue function that is primed for reproduction. The 'organisational-activational' hypothesis remains a tenet of developmental biology.

Realising the groundbreaking importance of their study, Phoenix and co-workers immediately switched their focus from guinea pigs to an animal model more closely resembling humans (e.g., Goy 1968; Phoenix 1974; 1980; Phoenix *et al.* 1977). They chose the rhesus macaque (*Macaca mulatta*); a species that is developmentally similar to humans (although it develops four times faster; Wallen 2005), lives in complex social groups and expresses clearly identifiable sex-linked behaviours that are expressed in ways similar to humans. The focus of these studies was to understand the development of sex-linked social behaviours by experimentally manipulating PAE. The aim was to understand PAE on anatomical and social development by priming foetuses with prenatal androgens and tracking their social interactions over growth.

The studies on captive rhesus macaques that were set up after the progenitor paper in guinea pigs (Phoenix *et al.* 1959) focussed on giving pregnant rhesus females different doses of androgens at different points during their pregnancy. The androgenised female offspring were then monitored and their development was compared to females growing up in the same environment but whose mothers had not received androgens during pregnancy. The females who had received high doses of androgens early in gestation developed genitals that were indistinguishable from those of males. Through development the masculinised females displayed behaviours that were more typical of males than of females, such as higher incidences of rough-and-tumble play and foot-clasp mounting. Vocalisations were masculinised and interest in infants was diminished (masculinised). Play initiation, however, remained more female-like (reviewed in Wallen 2005). In adult females that had been exposed to high PAE a dose of androgens prompted male-like initiation of sexual behaviour and copulatory behaviour (activational effects). The endocrine function of these adult females was also shown to be abnormal. By reducing the dose of androgens given to the mother late in pregnancy the androgenised female offspring developed normal genitalia, but male-typical behaviours continued to be expressed throughout adult life.

More recently studies have focused on exposing female foetuses to smaller doses of androgens during gestation and observing their development within more naturalistic social environments (as opposed to individually caged or peer-reared within a laboratory setting). In these studies prenatal androgens were high enough to alter HPG function of the females,

but were not high enough to masculinise their genitals. These mildly androgenised females did not significantly differ from the control females and androgenised females all went through puberty and reproduced normally (Wallen 2005; Wallen & Hassett 2009). The studies suggested that a complex social environment acts to modify prenatal androgen-induced predispositions (Wallen 1996; 2005; Champagne & Curley 2005; Zehr *et al.* 2005). When females develop within normal social environments the potential for masculinised behaviours to emerge in mildly androgenised females is governed more by social learning. Social learning promotes the development of appropriate behaviours and discourages inappropriate behaviours (Datta 1988). The potential for organisational effects on behaviour to be adapted by environmental factors (e.g., learning from others) allows behaviour to be fine-tuned according to the individual's social landscape (Wallen 1996; Champagne & Curley 2005; Thornton *et al.* 2009).

Interestingly, blocking effects of prenatal androgens in the female foetuses (via administration of the androgen receptor blocker flutamide to the mothers during pregnancy) resulted in some abnormalities of the reproductive system with the effects being similar to studies in which very low doses of androgens were given to mothers. This suggests that, in female rhesus macaques, some androgens are essential for normal sexual development (see Herman *et al.* 2000).

There appears to be striking similarities in the pattern of PAE across gestation in rhesus macaques and humans. In human males there is a rise in prenatal androgens in response to differentiation of the testes and HPG function between 8-18 weeks of gestation. These levels drop to match female hormonal profiles until birth. Parturition (birth) stimulates a brief rise in androgens which fall to childhood levels (low) within 2 weeks of birth. The second postnatal peak in androgens occurs around 8 weeks in males, returning to childhood levels within 6 months (see Baron-Cohen *et al.* 2004; McIntyre 2006). Low levels are then maintained until puberty. The rhesus macaque also exhibits an early rise in androgens (week 5-6) which then falls but increases again before birth (Resko & Ellinwood 1981; Resko 1985). This is followed by a postnatal rise until 3-4 months when androgens levels fall to female levels and remain low until puberty at around 2.5-4 years (Mann *et al.* 1984; Terasawa & Fernandez 2001). Organisational effects mostly occur during prenatal and early postnatal critical phases when the body and brain tissues are undergoing maximal change. More recently it has been shown that hormonally induced changes in the brain during adolescence displays similar organisational changes to those in the foetus and neonate. This suggests that, although the most profound changes occur in early development, puberty is a time of both organisational and activational processes (Wallen 2005; Fitch & Bimonte 2002).

1.1.1.3: PAE: Evidence from human studies

Due to ethical constraints it is not possible to manipulate the human prenatal environment so studies of PAE in humans have utilised 'natural experiments'; medical disorders that expose the foetus to extremes of PAE for their genetic sex or situations in which PAE can be estimated for individuals and compared to expressions of sex-linked traits during development in the same individual. For example, amniotic fluid samples taken as part of the foetal screening process in normal pregnancies can be used as a proxy for foetal circulatory androgens (Baron-Cohen *et al.* 2004).

1.1.1.3.1: PAE: Evidence from medical disorders in humans

The most studied medical disorders with known extremes in PAE are; congenital adrenal hyperplasia (CAH) in which female foetuses are exposed to high (male-like) levels of androgens from the adrenal glands, polycystic ovary syndrome (PCOS) in which female foetuses are exposed to high (male-like) levels of androgens from the ovaries (Abbott *et al.* 2005) and complete androgen insensitivity syndrome (CAIS) in which genetic (XY) males have deficient androgen receptors preventing masculinisation of the tissues by PAE (for reviews see Collaer & Hines 1995; Hines 2010; Berenbaum & Beltz 2011). These studies strongly indicate that exposure to low (or absent) PAE feminises behaviour in males and exposure to high PAE masculinises behaviour in females (Hall *et al.* 2004; Abbott *et al.* 2005; Cohen-Bendahan *et al.* 2005; Pasterski *et al.* 2008; Hines 2010). These findings are therefore concordant with studies in rhesus macaques (see Wallen 2005). Additionally, it has been reported that female monkeys exposed to high PAE exhibit symptoms consistent with PCOS in humans (Abbott *et al.* 2005).

1.1.1.3.2: PAE: Evidence from normal variation in humans

Studies in rodents and pigs show that female foetuses that gestate between two male foetuses are masculinised via transfer of androgens from the males (vom Saal 1989; Rohde-Parfet *et al.* 1990; Even *et al.* 1992). To investigate if this occurs in humans, studies have compared females from opposite sex (OS) twins with females from same sex (SS) twins to see if OS female twins are more masculinised. OS females show evidence of masculinisation but these effects are either only weakly detectable or absent (reviewed in Cohen-Bendahan *et al.* 2005).

Studies have also used prenatal androgens assayed from amniotic fluid samples obtained while the child was gestating (at around 17 weeks; Finegan *et al.* 1989; Grimshaw *et al.* 1995a; 1995b). Amniotic androgens have been shown to be reflective of foetal levels of prenatal androgens during the first prenatal peak when major body systems are being organised (see Cohen-Bendahan *et al.* 2005). The findings are generally indicative of mild masculinisation of females exposed to higher foetal androgens (e.g., play behaviour is more similar to boys than girls), but results are somewhat contradictory as some studies did not detect any differences (see Cohen-Bendahan *et al.* 2005). These kinds of investigations are likely to be age-sensitive as behaviours adapt to follow social norms as young females develop into adults.

1.1.1.3.3: PAE: Evidence from social development in humans

Humans show distinctive sex differences in social learning with females having superiority in this domain. These effects (female superiority) are shown in neonates (Connellan *et al.* 2000) and throughout early infancy, with infant females showing more interest in faces, interactions between individuals and having higher rates of eye contact than infant males (reviewed in Knickmeyer & Baron-Cohen 2006). Females also develop theory of mind⁴ earlier than males and, as adults, are generally more attuned to the feelings (empathy) and thoughts of others which enables them to fine-tune their behaviour in accordance with these perceptions (reviewed in Knickmeyer & Baron-Cohen 2006; Geary 2002). From birth males appear more interested in abstract patterns (Connellan *et al.* 2000) and show tendencies towards developing restricted interests in subjects that involve rule-based systems (so called systemising; Baron-Cohen 2002; Knickmeyer *et al.* 2005). Empathising and systemising represent the two ends of a spectrum which incorporate normal human cognitive variation as well as extreme empathisers that are ‘system-blind’ and extreme systemising who are ‘mind-blind’ and autistic (Baron-Cohen 2002). Male-type social development is expressed in an extreme form in individuals with autistic spectrum disorder (ASD); a developmental condition that is manifested in varying levels of impairment and, in its severest form, is characterised by poor social cognition, often with learning difficulties (Baron-Cohen 1995). In its mildest form individuals with ASD integrate well into society and often excel in

⁴ Theory of mind is a suite of cognitive adaptations that may be unique to humans that emerge around the age of 1 year with the understanding and sharing of intentions with others. Theory of mind becomes fully-functional around the age of 4 when children begin to understand that others may have thoughts, beliefs, goals that differ from their own. Language may be an integral aspect of human theory of mind and theory of mind is essential for empathy (Leslie 1987; Tomasello & Rakoczy 2003; Tomasello *et al.* 2005).

examinations and occupations linked to mathematics and physical systems or rule-based phenomena (systemizing; Baron-Cohen *et al.* 1998).

The incidence of ASD is four times higher in males than females suggesting that this complex condition may be linked to masculinisation during sexual differentiation (Knickmeyer & Baron-Cohen 2006). Some individuals with ASD have endocrine systems that over-produce androgens and this can be associated with higher levels of aggression and early signs of puberty (Trojman *et al.* 1997). These individuals also tend to exhibit hyper-masculine scores in cognitive tests (Ingudomnukul *et al.* 2007) and perform poorly on standard measures of empathy (e.g., 'reading the mind in the eyes' test [RMET]; Empathy Quotient [EQ]; Baron-Cohen *et al.* 2001a; Baron-Cohen & Wheelwright 2004). Children with ASD have been shown to have more close relatives (unaffected by ASD) in professions linked to engineering, while unaffected students studying mathematics, engineering and physics have more relatives with ASD than students studying literature (Baron-Cohen *et al.* 1997; 1998). Unaffected healthy males score higher than unaffected healthy females using standardised tests of systemising (e.g., Autistic-Spectrum Quotient; Baron-Cohen *et al.* 2001a). A recent study in unaffected individuals, EQ and Systemising Quotient (SQ) were shown to be largely independent in females, but men expressed these traits in a more continuous form; men used systemising approach to deal with empathising problems (Valla *et al.* 2010). Based on this accumulating evidence, and theories proposing links between early androgens and brain lateralisation (see Baron-Cohen *et al.* 2004 for a review), Baron-Cohen hypothesised that high PAE may be implicated in the aetiology of ASD and that ASD in its more extreme form may be an exaggeration of early masculinisation processes (Baron-Cohen 2002).

In order to test this theory an understanding of how PAE influence normal social development was needed. The Cambridge Foetal Testosterone Project was set up to tackle this objective (Knickmeyer & Baron-Cohen 2006; Baron-Cohen *et al.* 2004). The series of studies used measures of prenatal androgens assayed from amniotic samples taken during normal pregnancies and correlated these measures with the social development of the infants from which the sample was taken. The ongoing research is showing that higher PAE are associated with lower incidence of eye contact (at aged 1 year), lower vocabulary size (at aged around 2 years), poorer empathising ability (Knickmeyer *et al.* 2006a) and poorer quality social relationships at aged 4 years in both boys and girls (Knickmeyer *et al.* 2005). Children exposed to higher PAE score higher on standard tests measuring autistic traits (e.g., SQ), and poorly on standard tests measuring empathy (e.g., EQ; reviewed in Ingudomnukul *et al.* 2007). Adult females with ASD and the mothers of children with ASD have a higher

incidence of testosterone-related disorders than control females (e.g., polycystic ovary syndrome (PCOS); Ingudomnukul *et al.* 2007). In addition, females with congenital adrenal hyperplasia (CAH) have been shown to score higher in standard tests measuring autistic traits than healthy female controls (Knickmeyer *et al.* 2006b). This evidence suggests that high PAE masculinises the brain and channels social cognition towards systemising and away from empathising.

One of the most limiting manifestations of ASD is the reduced ability of individuals to express empathy and to form close emotional social bonds with people other than primary caregivers (see Tomasello *et al.* 2005). This may be linked to the antagonistic effects of prenatal androgens on the neuro-circuitry of reward systems (Carter 2007; Lim *et al.* 2005; Broad *et al.* 2006). Oxytocin (OT) and arginine vasopressin (AVP) are peptide hormones produced in the posterior pituitary that are associated with reproduction and sexual behaviour in mammals, but are also implicated in social bonding (Young & Wang 2004; Broad *et al.* 2006; Donaldson & Young 2008). The peptides are molecularly very similar and while both OT and AVP have effects on males and females, in the social domain OT imparts more of an effect in females whilst AVP imparts more of an effect in males (Dunbar 2010b). In female mammals OT is implicated in the formation of the mother-infant bond and in male and female humans it enhances the ability to interpret the facial expressions and the emotions of others (Domes *et al.* 2007; Guastella *et al.* 2008). OT has also been shown to improve empathetic ability of males with ASD (Guastella *et al.* 2010). A recent study has linked variation in a host of polymorphic candidate genes related to sex-hormone production and uptake, social bonding (OT) and neural development to differences in EQ/SQ scores in participants with ASD and normal unaffected participants (Chakrabarti *et al.* 2009). Several of the candidate genes were shown to differ among the ASD participants.

OT appears to have a modulating action on the neural reward cascade via its effects on opioid and dopamine receptors; the interactions between neuro-hormonal processes in the reward pathway are complex, interdependent and highly conserved (Insel 1997; Donaldson & Young 2008). In humans and other higher primates, intimate social interactions between individuals causes the release of endorphins that promote feelings of well-being, OT is released and this promotes feelings of calmness and trust between the pair, at the same time dopamine acts to store the memory of the interaction which serves to reinforce the bond and make it more likely to be repeated (Lim *et al.* 2005; Donaldson & Young 2008; Dunbar 2010b). This reward cascade (described in simplistic terms here; see Broad *et al.* 2006 for a detailed description) is believed to underpin social attachment in humans; similar effects may occur in other primates during social grooming episodes (Keverne *et al.* 1989; Dunbar

2010b). These processes appear to be disrupted in individuals with ASD and healthy individuals exposed to high androgens under test conditions (Dawson *et al.* 2005; Carter 2007; van Wingen *et al.* 2010; also see Rilling *et al.* in press). These links have led to the proposal that one of the contributing factors in ASD could be an over-expression of androgens and an under-expression of OT leading to an impaired ability to read the mental states of others and a reduction in emotional reward elicited by close social interaction with others (Carter 2007; see van Honk *et al.* 2010; also see Insel *et al.* 1999; Dawson *et al.* 2005).

1.1.1.4: PAE: A potential role in social evolution?

Social cognitive abilities in humans have been presented within an evolutionary framework (Knickmeyer & Baron-Cohen 2006). The model proposes that sexual selection along with sexual division of labour imposed different neuro-cognitive pressures on males and females in early human societies (although it remains unclear when sexual division of labour evolved; see Kuhn & Stiner 2006; Gamble *et al.* 2010). For females enhanced social skills such as empathy and showing concern for others are proposed to have been advantageous because females migrate out of their kin-groups and acceptance by unfamiliar individuals requires females to build social bonds with strangers in order to form a secure environment for rearing offspring (Geary 2002; Taylor *et al.* 2000). In males higher PAE is proposed to have been advantageous for the development of weapon skills (e.g. via enhancement of spatial abilities and hand-eye coordination) and fighting ability (e.g., aggression, dominance, risk-taking, low empathy). Systemising would have been useful for devising hunting and warfare strategies (e.g., predicting seasonal movements of animals, judging devising attacks; Baron-Cohen 2004; McIntyre & Edwards 2009). It has been proposed that ASD may represent an exaggeration of the male phenotype (Baron-Cohen 2002; 2004). In most contemporary human societies individuals that are hyper-masculinised, socially distant and low in empathy stand out as being abnormal; these characteristics may have been less conspicuous in our ancient ancestors as men vied for control over resources and females (Foley & Gamble 2009).

As PAE plays a key role in organizing sexually selected traits and sociality in humans and non-human primates we might expect the prenatal phase of development to be under strong selective pressure. Variation in PAE across primate species may therefore be useful in informing research paradigms that investigate social evolution. The Social Brain Hypothesis (SBH; Byrne & Whiten 1988; Whiten & Byrne 1997; Dunbar 1998; also see Humphrey 1976) was originally conceived to explain why primates, particularly humans, had unusually

large brains for their body size. Over time the SBH has altered its focus according to the research findings and has now broadened to incorporate studies of brain size and social systems across Vertebrate Orders (Shultz & Dunbar 2007). In most vertebrates pair-bonded species are shown to have the largest brain size, while in primates it is promiscuous species living in large social groups that have the biggest brains (Shultz & Dunbar 2007). A subsequent study tested primates separately from other Orders and found that they do follow the vertebrate pattern (pair-bonded species have larger brains), but only when group size is controlled for (Dunbar 2010a). Unlike other vertebrates, however, the intense relationships formed between haplorhines living in large social groups are not confined to reproductive partners but appear to be extrapolated in a pair-bond-like way to other individuals in order to form enduring ‘friendships’ (Silk 2002b; also see Curley & Keverne 2005; Broad *et al.* 2006). Maintaining such close bonds becomes cognitively challenging when group size is large and friendship networks are extensive. This style of social bonding may lie at the root of primate brain evolution because brain size is accentuated in promiscuous primate species that live in large groups (Shultz & Dunbar 2007; see Dunbar & Shultz 2010).

The proximate mechanisms that underpin primate social bonds, and which ultimately form the evolutionary substrate for human sociality, are still poorly understood (Dunbar & Shultz 2007a; 2010). Three of the candidates implicated in the maintenance of these special bonds in primates are OT, AVP and dopamine (Curley & Keverne 2005; Donaldson & Young 2008; Dunbar 2010b); these hormones act as neuro-modulators and neuro-transmitters within the reward pathway and appear to be altered by PAE and circulating androgens (Carter 2007; van Honk *et al.* 2011). Investigating how PAE co-vary with social behaviours in extant primates might refine our understanding of hominin social evolution.

1.1.2: The second to fourth digit length ratio (2D:4D)

It is not possible to perform comprehensive cross-species analyses in primates using data based on standard methods of investigating PAE (e.g., experimental manipulation of prenatal androgens and assays of amniotic fluid). Over the last decade an anatomical marker of PAE⁵ has emerged that makes it possible to carry out this research; the 2nd (2D) to 4th (4D) digit length ratio (2D:4D). As a consequence of the pioneering work of John Manning, the 2D:4D biomarker has now been used extensively to investigate PAE on human development, adult

⁵ The specific prenatal androgens (e.g., testosterone, dihydrotestosterone; adrenal androgens) that influence 2D:4D are unknown.

sexually selected behaviours and sex-linked diseases (Manning 2002a; 2007a; Voracek & Loibl 2009).

1.1.2.1: 2D:4D: The history

Documentation of sexual differences in the relative lengths of the fingers has a long history. Associations between masculinity and a long ring finger (4th digit) were noted in the diaries of Casanova (1894; apparently his ring finger was lengthy). Sexual differences in the 2nd and 4th digits first appear in published accounts by Ecker (1875) whose comparative studies of humans and non-human primates led him to believe that a long 4th digit was the primitive pattern (see Peters *et al.* 2002). Ecker's opinions were echoed by the anthropologist and anatomist Adolph Schultz whose observations of primate and human foetuses indicated to him that a long 4th digit was characteristic of arboreal primates and that digit length proportions were fixed *in utero* (Schultz 1926; 1947). Phelps (1952) also noted the sex difference in the two digits and was aware of Schultz's finding that finger proportions were fixed prenatally. Her study of similarities in finger length proportions within families led her to the conclusion that the pattern was driven by genetic factors. Rösler (1957) published a series of papers on sex-linked finger patterns in 2D:4D that formed the basis of his doctoral dissertation (for an overview see Rösler 2007; Voracek *et al.* 2008c). On reading Phelps (1952) account of sex differences in the digits, psychologist Glenn Wilson suspected that the sexually dimorphic pattern in 2D:4D may be linked to prenatal sex hormone differences in males and females (see Wilson 2010). He used 2D:4D to assess personality in women from self-measured finger lengths collected in a public poll advertised in the Daily Express newspaper. The results showed that women with low 2D:4D (high PAE) described themselves as having more a masculinised personality type (e.g., assertive and competitive), while women with higher 2D:4D (signalling low PAE) described themselves as more feminised (e.g., gentle and feminine; Wilson 1983).

This body of literature was not known to evolutionary biologist John Manning in the 1980s to late 1990s when he was working on fluctuating asymmetry (FA)⁶ and sexual selection (see Wilson 2010). Over the years Manning had come to notice consistent sexual differences in the relative lengths of the index and ring fingers of his adult study subjects; males tended to

⁶ Fluctuating asymmetry in bilateral anatomical traits is a measure of developmental stability/instability which reflects the genetic and environmental stress experienced by individuals over growth. A more symmetrical individual signals a phenotype that is able to withstand developmental stress. Signals of symmetry are used by females in mate choice as a measure of 'good genes' and are predictive of male fitness (see Møller & Swaddle 1997; Trivers *et al.* 1999).

have long ring fingers and shorter index fingers, but in females these fingers were more equal in size. Working with Robert Trivers on the Jamaican Symmetry Project, a longitudinal study of FA in Jamaican children (Trivers *et al.* 1999), Manning saw the same sexual pattern in the fingers of the children that he had noticed in the adults (Manning *et al.* 2000b). He postulated that these differences might be associated with sexual differentiation. The fact that they were similarly expressed in both children and adults suggested that the relative lengths of the digits (2D:4D) were probably fixed early in development. The observation that 2D:4D did not change at puberty, when sex hormones increase markedly, supported the idea that the ratio was probably fixed early in life (Manning *et al.* 1998; Manning 2002a).

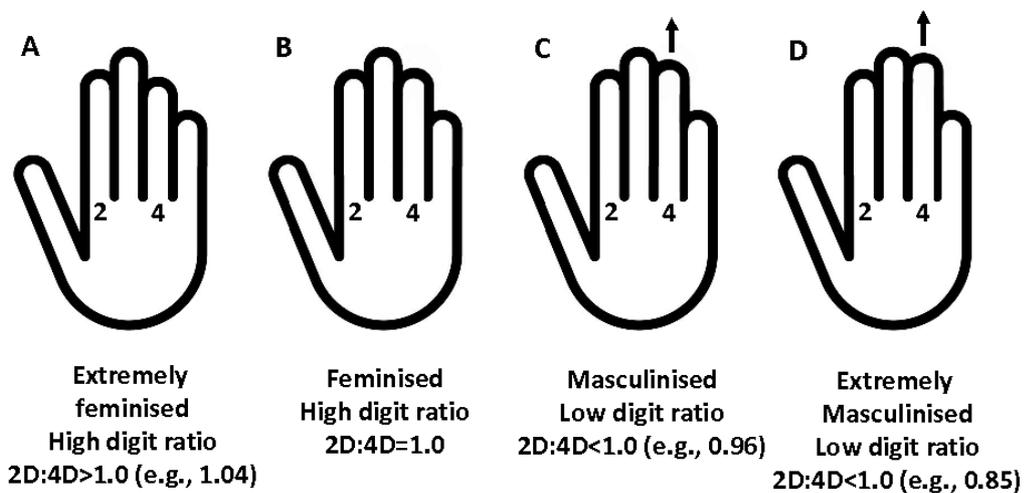


Figure 1.1: High 2D:4D and low 2D:4D: what do they look like? Human females have 4th digits (4D; ring finger) that are slightly shorter than their 2nd digits (2D; index finger), which produced a digit ratio that is greater than 1 (**A**), or their 2D and 4D are about the same length producing a digit ratio close to unity (i.e., equal to 1) (**B**). Prenatal androgens lengthen the 4D; therefore human males have a longer 4D relative to their 2D (**C & D**). This leads to a low ratio (<1.0). As such, a masculinised 2D:4D is one that is lower on average than that of females (i.e., <1.0). A feminised 2D:4D is one that is average for females (i.e., ~1.0) or higher (>1.0). The absolute values differ slightly between human populations (see Manning *et al.* 2000a).

1.1.2.2: 2D:4D: Developmental framework

In the progenitor publication Manning *et al.* (1998) briefly outlined the bio-genetic mechanisms they believed underpinned the developmental links between sexual differentiation and digit morphology (see below). The study showed that despite considerable variation a low 2D:4D (2D:4D < 1) was more common in males and was inferred to be associated with high PAE and masculinisation, and a high 2D:4D (2D:4D > 1) was more

common in females and was associated with feminisation⁷ and low PAE (see Fig. 1.1). In their large sample of adults they showed that low 2D:4D (masculine-type) was associated with higher testosterone levels in men, while high 2D:4D (feminine-type) was correlated with high female-linked hormones in females (e.g., estrogen, prolactin and luteinising hormone). Their findings also showed that men with feminine-type (high) 2D:4D had more feminine hormonal profiles and a higher incidence of low sperm counts. They postulated that the programming (organisational) effects of prenatal sex hormones during sexual differentiation are reflected in 2D:4D and adult hormonal profiles. Their results implied a link between 2D:4D and evolutionary fitness because adult male sex hormones are higher in males with a masculine (low) 2D:4D and adult female sex hormones are higher in females with a feminine (high) 2D:4D. Furthermore, their study indicated that a switching of this pattern between the sexes signalled compromised reproductive functioning (Manning *et al.* 1998; also see Manning *et al.* 2000a; Manning & Fink 2011).

The mechanism proposed to underpin these associations is androgen sensitivity in the homeobox (*HOX*) gene cluster (Manning *et al.* 1998). *HOX* gene proteins encode the blueprint for the body's structure. The posterior *HOXA* and *HOXD* genes organise the development of the terminal limb-bud (digits) and parts of the reproductive systems (including the gonads, baculum and penis (Zákány *et al.* 1997; Kondo *et al.* 1997; Montavon *et al.* 2008; also see Chiu & Hamrick 2002). For example, hand-foot-genital syndrome is caused by a mutation on *HOXA* 13 which leads to abnormal development in all the named structures (Mortlock & Innis 1997). By manipulating the *HOX* gene proteins in *HOXA* 13 and *HOXD* 11-13 a progressive reduction in digit size and digit number can be induced in mice (Zákány *et al.* 1997). In the same study, genotypes were manipulated to show that as the D4 reduced in size, there was a corresponding reduction in the size of the baculum. *HOX* gene transcription appears to be sensitive to sex hormones (Soto & Sonnenschein 1999; Daftery & Taylor 2006). Interactions between hormones and *HOX* genes facilitate the generation of functional and structural variation via the utilisation of evolutionary conserved developmental mechanisms (Daftery & Taylor 2006). As such, variation in PAE could potentially alter *HOX* proteins leading to subtle changes in digit lengths. D4 appears to be particularly sensitive to PAE/*HOX* genes interactions such that individuals exposed to high prenatal androgens have longer 4D relative to their 2D (low 2D:4D; Zákány *et al.* 1997). It is conjectured that relative digit lengths (e.g., 2D:4D) signal the organisation of the reproductive system by PAE as a consequence of *HOX* gene pleiotropy (Voracek & Manning

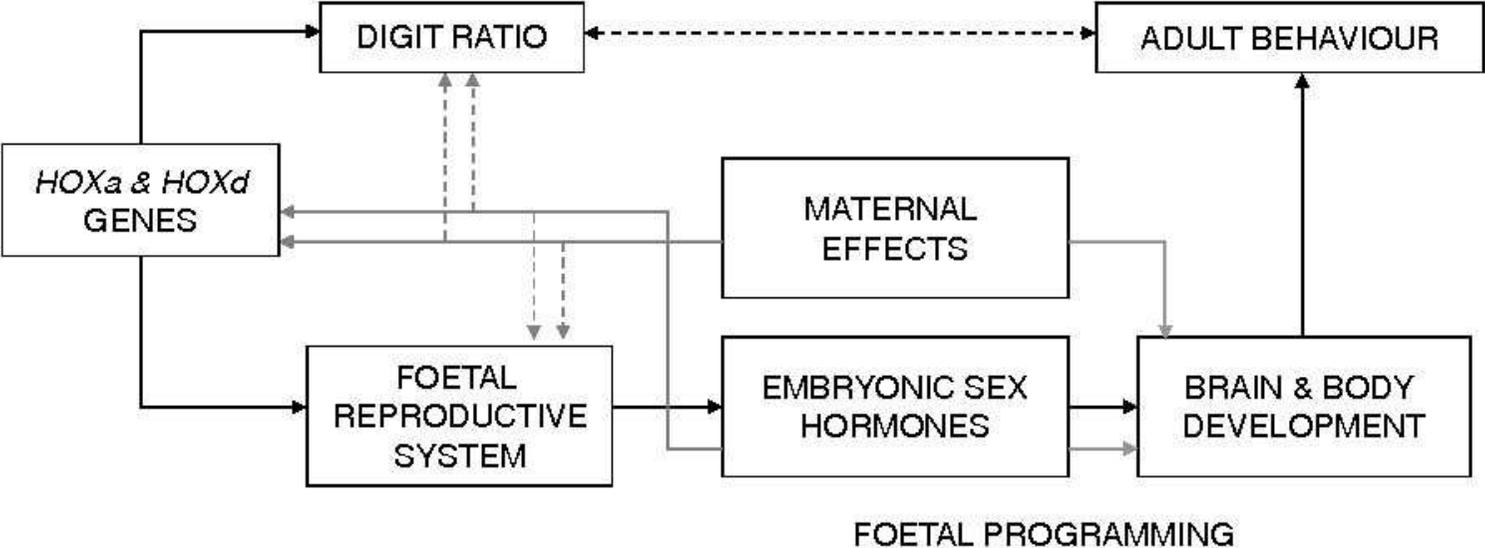
⁷ Manning proposes that high 2D:4D is linked to prenatal estrogens. There is no convincing evidence to link prenatal estrogens to 2D:4D in humans, which might be expected given that the primary mode of sexual differentiation is via PAE.

2003; Fig. 1.2). The overarching theory is that 2D:4D reflects programming effects of androgens on the foetus and subsequent postnatal development; specifically the efficiency of the reproductive system to masculinise males or to remain quiescent to allow the feminisation of females.

Although this mechanism is still not understood in humans, a recent study in mice provides strong support for links between PAE acting through the fourth digit (but not the second) and thereby influencing 2D:4D in the expected directions (Zheng & Cohn, 2011). When the mouse embryo was exposed to estrogens or the androgen receptor gene was blocked, the fourth digit was feminised (similar in size to the second digit). When the embryo was exposed to high androgens or the estrogen receptor was blocked, the fourth digit was masculinised (longer than the second) (Zheng & Cohn, in press).

Developmental linkage between the genital and limb buds may have evolved early in the history of land vertebrates (Manning 2002a). Studies in mice show that reducing *HOX* gene proteins induces loss of digits (Zákány *et al.* 1997). As these products are pared down the autopod (hand/foot) moves through a stage of polydactyly (many digits). Before the evolution of the pentadactyl digital pattern (five digits) the fossil record indicates that ancient aquatic tetrapods had up to eight digits (polydactyl) which may have allowed the autopod to function as a paddle (Zákány *et al.* 1997). Manning (2002a) posits that the vertebrate limb was remodelled towards a five-digit form when animals began moving out of an aquatic environment. At the same time adaptations to the reproductive system were taking place. Aquatic or amphibious vertebrates reproduce in water; fertilisation occurs by males ejaculating directly on to the eggs (Manning 2007a, p 11). Life on dry land required gamete fusion to occur internally. It is possible that these adaptations came about by recruiting the same *HOX* genes which led to shared regulatory mechanisms between the simultaneously evolving limb (polydactyly) and genital bud (Zákány *et al.* 1997).). As *HOX* genes are highly phylogenetically conserved across taxa (van der Hoeven *et al.* 1996) Manning hypothesised that sexual dimorphism in 2D:4D should generalise across taxonomic groups with pentadactyl limbs (Manning 2002a).

Figure 1.2: Proposed bio-genetic mechanisms underpinning the inferred relationships 2D:4D and PAE. Black arrows the known pathway of development. Grey arrows represent the hypothesised influences on structures of prenatal androgens from the foetus and the mother. Adapted from Forstmeier *et al.* 2008.



Direct evidence of links between 2D:4D and PAE come from a study in which testosterone was given to the mothers of gestating rats. Both behaviour and digit ratios were masculinised (inferred lower 2D:4D) in their adult female offspring (Talarovičová *et al.* 2008). Studies have now been performed on a wide range of vertebrate taxa with different developmental patterns and sexual determination (see Lombardo & Thorpe 2008; Adkins-Regan *et al.* 1995; Lance 1997). In mammals masculinisation is manifested in lower 2D:4D while in birds and scaly reptiles masculinisation *might* be indicated by high 2D:4D (Saino *et al.* 2006; Chang *et al.* 2006). The close evolutionary links between birds and scaly reptiles (Shedlock & Edwards 2009) have been proposed as an explanation for similarities in sexual dimorphism in 2D:4D in these two groups (Chang *et al.* 2006; Chang 2008). However, high variation in sex-linked patterns of 2D:4D within and between taxonomic groups has yielded highly inconsistent patterns (see Lombardo & Thorpe 2008; Lombardo *et al.* 2008). It is still not clear if sexual dimorphism in 2D:4D generalised across vertebrates in a consistent way.

1.1.2.3: 2D:4D: Studies in humans

Links between 2D:4D and PAE have largely been indirect because of the operational challenges and ethical restrictions that govern invasive research of the intra-uterine environment. Studies of digit ratios in deceased foetuses indicate that sexual dimorphism in 2D:4D is evident as early as 9 weeks of development (Malas *et al.* 2006; Galis *et al.* 2010; also see Garn *et al.* 1975). Females with CAH and PCOS that have been masculinised *in utero* have lower, more masculinised 2D:4D (inferred higher PAE) than healthy controls (Brown *et al.* 2002; Ökten *et al.* 2002a; Hönekopp & Watson 2010; but see Buck *et al.* 2003; Catrall *et al.* 2005; but see Lujan *et al.* 2010a; 2010b). Infants aged 2 years with a higher testosterone-to-estradiol ratio (assayed from their amniotic fluid) have lower 2D:4D (inferred higher PAE) than those with a lower testosterone hormone ratio (Lutchmaya *et al.* 2004). 2D:4D was found to be lower (inferred higher PAE) in female twins who had gestated next to a male twin compared to females with a same sex twin suggesting that prenatal androgens from the male twin masculinise the digit ratios of the female twin (van Anders *et al.* 2006; but see Medland *et al.* 2008). Females with low 2D:4D (inferred higher PAE) exhibit masculinisation in areas of their hippocampus (Kallai *et al.* 2005). Smoking in pregnancy increases prenatal androgen levels. Mother who smoked during pregnancy had sons with lower 2D:4D (inferred higher PAE) than controls; females ratios were not affected (Rizwan *et al.* 2007). Importantly, 2D:4D was shown to be lower (inferred higher PAE) in children with ASD and their unaffected close relatives compared with healthy controls (Manning *et al.* 2001). Individuals with low 2D:4D (inferred high PAE) score consistently higher on tests aimed at detecting systemising traits (e.g., SQ), but score lower on test detecting empathy

(e.g., EQ; von Horn *et al.* 2010; Manning *et al.* 2010; Wakabayashi & Nakazawa 2010; but see Voracek & Dressler 2006b). Males with low 2D:4D (inferred high PAE) have an androgen receptor gene that is more sensitive to androgens (Manning *et al.* 2003a; but see Hurd *et al.* 2011). Genetic males (XY) with complete androgen insensitivity syndrome (CAIS) who develop as females due to defective androgen receptors have 2D:4D ratios more similar to healthy females (inferring lower PAE) than healthy males (Berenbaum *et al.* 2009).

1.1.2.3.1: 2D:4D: Links to sexual selection

In humans low 2D:4D (inferred high PAE) is repeatedly associated with competitive behaviours particularly those linked to sporting achievement (see Manning 2002a; 2007a), but also behaviours that reflect drives for dominance, status, and mates (e.g., Manning & Fink 2008; Millett & Dewitte 2009; but see Koehler *et al.* 2004). These kinds of sexually selected behaviours may involve displays of aggression, strength, risk-taking and courtship and are associated with low 2D:4D (inferred high PAE; e.g., Fink *et al.* 2006c; Millet & Dewitte 2009; Coates & Page 2009; Longman *et al.* 2011; Stenstrom *et al.* 2011; Hönekopp 2011). Studies of these behaviours have focussed less on women but the evidence suggests that females with low 2D:4D (inferred high PAE) are also masculinised in these kinds of traits even though they may be expressed differently (e.g., reduced risk-aversion: Hönekopp *et al.* 2006a; dominance; Manning & Fink 2008). For example aggression appears to be more physically and overtly displayed in males, while in females it may be more indirect (see Voracek & Schicker 2010). Additionally, low 2D:4D (inferred high PAE) in both sexes has been associated with a propensity towards promiscuity (Clark 2004; Hönekopp *et al.* 2006b; but see Putz *et al.* 2004; Rahman *et al.* 2005). These behavioural relationships prompted Fink *et al.* (2006c) to propose that 2D:4D should be viewed within a framework of sexual selection.

Low 2D:4D (inferred high PAE) in males has been linked to higher sperm quality (Manning *et al.* 1998; Wood *et al.* 2003; but see Firman *et al.* 2003; Bang *et al.* 2005; Seo *et al.* 2010) and larger family size (Manning *et al.* 2000a; Manning *et al.* 2003b). High 2D:4D (inferred low PAE) in males is linked to poor sperm quality and a more female-like hormonal profile (Manning *et al.* 1998; also see Wood *et al.* 2003). High 2D:4D (inferred low PAE) in females is linked to larger family size (Manning *et al.* 2000a; Manning *et al.* 2003b) and more female hormonal profile. Low 2D:4D (inferred high PAE) in females is associated with delayed menarche and slower sexual development (Matchock 2008; Manning & Fink 2011;

but see Helle 2010). These relationships highlight the antagonistic effects of variation in PAE in males and females (see Manning *et al.* 2000a).

Across populations 2D:4D appears to co-vary with sexual selection (indexed by marriage systems) with polygynous societies expressing lower 2D:4D ratios than monogamous populations (Manning *et al.* 2004b; Manning 2007a; also see Manning *et al.* 2000a). Despite relatively poor sampling of polygynous groups, this finding is the first to link 2D:4D to sexual selection at the population level. These patterns are consistent with research on the human androgen receptor gene (ARG) showing that the expression of the gene is most sensitive in populations in which polygyny more prevalent (e.g., sub-Saharan Africa; Murdock 1967; Kittles *et al.* 2001; Manning 2007a; 2007b). Higher intra-sexual competition in polygynous societies (e.g., Møller & Welch 1990; Madhavan 2002; Bove & Valeggia 2009) may therefore be supported by higher PAE; integral to these effects may be the action of a more sensitive ARG.

In summary; what evidence we have in humans linking prenatal androgens to 2D:4D points to higher PAE causing low 2D:4D. What evidence we have in humans regarding masculinisation of social behaviour points to high PAE. What evidence we have in humans shows low 2D:4D to be associated with masculinisation of social behaviour.

1.1.2.4: 2D:4D: The Feminised Ape Hypothesis

Based on observations of correlations between humans 2D:4D and the ARG (Manning *et al.* 2003a) Manning (2007a) hypothesised about changes to PAE that might have a bearing on human evolution. The ARG has been shown to decrease in sensitivity over primate evolution, with humans expressing the least sensitivity to androgens (Choong *et al.* 1996; Hong *et al.* 2006; also see Manning *et al.* 2003a). Manning linked this evidence to the findings of Ecker (1875) and Schultz (1924) of low 2D:4D (inferred high PAE) in promiscuous primates. Manning theorised that low 2D:4D in primates was probably linked to higher sensitivity of the ARG. He reasoned that the pattern seen in non-human primate species may follow the same pattern observed across human populations (low 2D:4D, higher sensitive of the ARG, higher levels of sexual selection; Manning 2007a). The fact that 2D:4D is higher (PAE is inferred to be lower) in humans than in promiscuous non-human primates and higher still in monogamous human populations indicated to Manning that a reduction in male-male competition (sexual selection) might have occurred over hominin evolution. If this was the case, he reasoned further, then this effective 'feminisation' may have facilitated the emergence of social adaptations linked to low PAE such as empathy,

language skills and may be greater intelligence (Manning 2007a; 2007b). The trade-off for increased social intelligence and feminisation, however, was a reduction in male fertility brought about by a down-regulation of the ARG (von Eckardstein *et al.* 2001; Manning *et al.* 2003a) and higher incidence of heart disease due to the detrimental effects of low PAE on the cardiovascular system (Manning & Bundred 2001; Fink *et al.* 2006a; Ozdogmus *et al.* 2010). In Manning's hypotheses Man is described as a Feminised Ape⁸ (Manning 2007a) and Manning conveniently outlines the gaps in his theory; a lack of 2D:4D data in apes and fossil hominins. Investigating 2D:4D in non-human primates might allow us to bridge those gaps and to elucidate the proximate mechanisms (linked to PAE) implicated in the evolution of human sociality.

1.2: Thesis approach

This thesis takes a top-down approach to investigating variation in 2D:4D across closely related taxa; namely haplorhine primates. It uses the evidence from studies of PAE on behaviour in macaques and humans, in addition to the evidence of links between 2D:4D and androgenised behaviours, to make the assumption that 2D:4D is likely to reflect PAE in other primates. Haplorhines are the most obvious group to compare with the human evidence because humans are within the suborder *Haplorrhini* and, as such, their developmental and biological profiles will be more similar to those primates than to more distantly related taxa. Haplorhines are also well studied which allows for systematic study of social behaviour across taxonomic groups. In order to carry out comparative analyses I make the assumption that 2D:4D in non-human haplorhines is associated with PAE in the same way that we believe it to be in humans. Lower 2D:4D is inferred to be associated with higher PAE and a more masculinised profile and higher 2D:4D is inferred to be linked with lower PAE and a more feminised profile.

1.2.1: Thesis aims

Broad aims:

1. To look across haplorhine species to see if the patterns between 2D:4D and sexually selected traits in humans are reflected at higher taxonomic levels.

⁸ I use the term the 'Feminised Ape Hypothesis'.

2. To look for patterns in the distribution of 2D:4D across haplorhines that might implicate PAE in broad-scale changes in sociality through primate evolution.
3. To investigate if variation of 2D:4D in extant apes might be informative about the role of PAE in hominin social evolution.

1.2.2: Thesis outline

Each chapter is effectively a stand-alone study. Chapter 2 will review methodologies in published human and animal-based studies in anticipation of introducing the haplorhine sample used in this thesis. The chapter then describes the methods used to collect the haplorhine data and how phylogenetic non-independence of traits were controlled for; a requirement for cross species analyses. The results of an analysis of 2D:4D across vertebrates is described. Chapter 3 presents a cross-species analysis of 2D:4D and behavioural variables linked to sexual selection; namely social system and intra-sexual competition. Chapter 4 investigates if 2D:4D co-varies with anatomical markers of sexual selection; namely body, canine and brain size as well as dimorphism in these characters. Chapter 5 focuses on within-species variation by presenting a case study investigating associations between 2D:4D and social dominance rank in female rhesus macaques (*Macaca mulatta*) from Cayo Santiago Island, Puerto Rico. It also presents the first analysis of heritability of 2D:4D in non-human primates by examining the variation between mother and infant 2D:4D ratios. Chapter 6 returns to the issue of proximate mechanisms in order to understand how species-level 2D:4D might correlate with factors more closely associated with potentiating behaviours; namely variation in circulating testosterone levels and variation in androgen receptor gene sensitivity. Chapter 7 utilises the findings in Chapter 3 by using fossil digit ratios to predict the social systems of extinct hominoids and hominins. Finally, Chapter 8 presents a review of the preceding chapters. In this chapter I discuss how cross-species results compare with findings in humans and how they fit within broader research themes. The main discussion also addresses how the findings might help us to understand changes in sociality through hominin evolution. The strengths and limitations of each study are presented along with recommendations on how the research themes might be extended. In the conclusion I return to the aims of the thesis to address whether they have been achieved.

Chapter 2

2D:4D, the haplorhine dataset and variation across vertebrates

2.1: Introduction

The majority of 2D:4D published studies (over 450 research papers and two books; Manning 2002a; 2007a) (reviewed in Voracek & Loibl 2009; Voracek 2011) have been carried out on humans, with a much smaller proportion (approximately 8%) performed on non-human animals. 2D:4D has been largely localised within the psychology discipline, which has impacted the kinds of studies performed (see for overviews Manning 2002a; 2007a; Voracek & Loibl 2009). The human 2D:4D literature is bias towards publishing positive findings and these studies tend to cite the same supporting research papers (Voracek & Loibl 2009). In recent years more authors and journals appear willing to publish negative or neutral findings (e.g., Vehmas *et al.* 2006) and more recently attention has been turned to scrutinising measurement methods and understanding how these might impact the accuracy of 2D:4D within and between samples (Manning *et al.* 2005; Voracek *et al.* 2007a; Caswell & Manning 2009; Kemper & Schwerdtfeger 2009; Allaway *et al.* 2009).

Techniques used to measure human digits have generally been limited to taking direct measurements from the skin surface or obtaining measurements from images of the hand, but less standardised techniques have also been employed (e.g., Bang *et al.* 2005; Jürimäe *et al.* 2008). More recently the research sphere has broadened to include 2D:4D in non-humans animals.

2.1.1: Aims

The aim of this chapter is to:

- 1) Review the methodologies of 2D:4D in humans and non-human animal research.
- 2) Presents new data (the haplorhine dataset) that expands the sample within the Primate Order.

- 3) Present the first attempt to compare variation in 2D:4D across vertebrates using a phylogenetically controlled technique.

2.2: Measurement of human 2D:4D

The digits are numbered from 1 (for the thumb) through to 5 (little finger; Romer 1955). Thus the index finger is digit 2 (2D) and the ring finger is digit 4 (4D). 2D:4D is calculated by dividing the length of the 2nd digit (index finger) by the length of the 4th (ring finger; Fig. 2.1; for a more detailed outline of this calculation see Appendix 2.1).

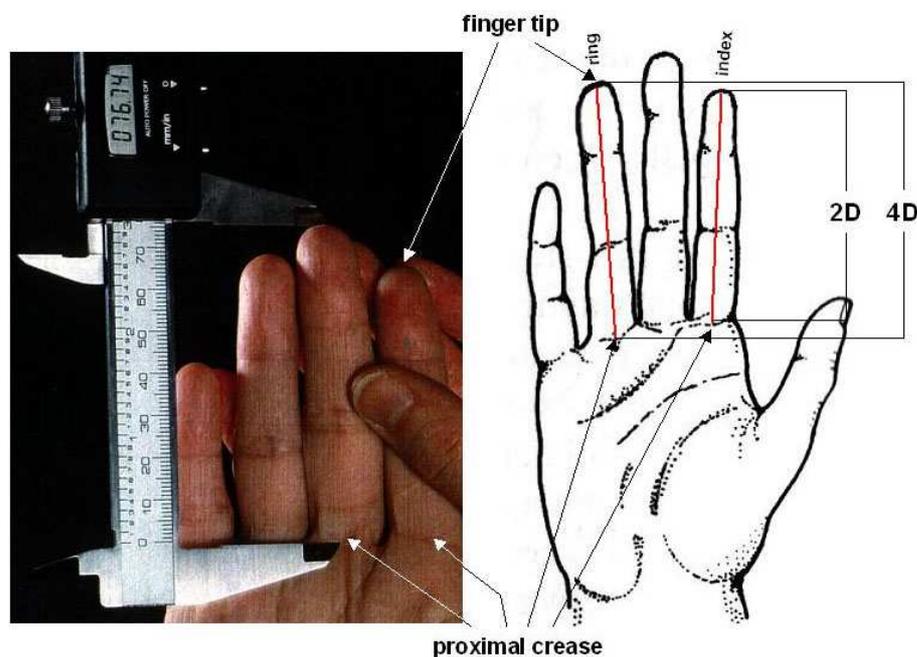


Figure 2.1: Direct measurement of digit lengths from the proximal crease to the fingertip. The length of the 2nd digit (2D; index finger) and the 4th digit (4D; ring finger) can be obtained using digital vernier callipers (resolution 0.01mm) or a standard ruler (0.1 mm).

In order to obtain an accurate 2D:4D ratio and compare results across studies the length of the 2D and 4D must be measured from the same landmarks using the same method (Fig. 2.1; Manning *et al.* 2005). Digit length is conventionally measured on the palmer surface of the hand, from the proximal crease at the base of the digit (basal crease; crease closest to the palm) to the tip of the finger in the mid-line (excluding the nail; Fig. 2.1; Manning *et al.* 1998; Manning 2002a). The standard position for measuring digit lengths one in which the participant is asked to hold their fingers together (adducted) in an extended position (Fig. 2.1). This allows the anatomical landmarks to be clearly identified. This is important because the absolute size differences between the two digits can be relatively small in humans and

slight deviations from the standard position can lead to inaccuracies in measurements. For example ulna deviation of the wrist elongates 4D and radial deviation lengthens the 2D (Weissenberg 1895; quoted in Peters *et al.* 2002; Robertson *et al.* 2008). The precision of a digit measurement is gauged via calculations of internal consistency performed on two sets of measurements from the same digit. These may be taken by the same observer with a delay of time between measuring bouts or by a different observer. Low values of internal consistency, and therefore low precision of measurements, are likely to impact on subsequent statistical analyses.

2.2.1: Measurement methods

2.2.1.1: Direct measurement methods

Soft tissue finger length can be measured directly from the skin surface with a set of digital vernier callipers (Fig. 2.1) or a ruler (see Manning *et al.* 2007a). This method does not distort the soft tissue elements of the finger; this is important as both the hard and soft tissues of the digits appear to be influenced by prenatal androgens (Buck *et al.* 2003). Direct measurement using digital vernier callipers is considered the most appropriate method (Manning & Hill 2009; Manning *et al.* 2010).

Compared to the calliper method (resolution 0.01 mm) using a ruler (0.1mm) leads to less precise values, but the ruler method still provides highly repeatable data and is comparable to the calliper method (Voracek *et al.* 2007a; Burriss *et al.* 2007). Ruler measurements become less accurate, however, for self-measurements (Manning *et al.* 2007a; Caswell & Manning 2009; Hönekopp & Watson 2010). Self-measurement is when individuals' measure their own fingers. The accuracy of self-measures improves significantly when an experienced measurer is present to guide the participant through the procedure (Burriss *et al.* 2007).

2.2.1.2: Indirect measurement methods (soft-tissue)

The indirect method takes digit length measurements from a photocopy or scan of the hand (Fig. 2.2). Printouts of the image are measured using vernier callipers or a ruler and digital images are measured using software applications (i.e., Adobe measuring tool; Fig. 2.2). Specialised software has also been developed specifically for measuring 2D:4D (i.e., AutoMetric 2.2; DeBruine on-line application quoted in Kemper & Schwerdtfeger 2009; also see Burriss *et al.* 2006).

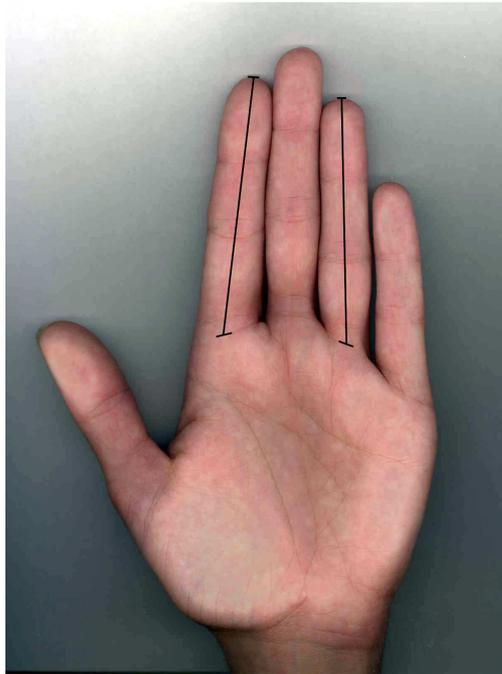


Figure 2.2: Scanned hand with digits measured using measuring software. Digits can be measured using software metric tools such as Adobe Photoshop©. Unlike photocopies, scanned images can be magnified in order to attain better resolution of the landmark points (see Allaway *et al.* 2009).

2D:4D has also been calculated from hand ink prints (Hall 2001; Ronalds *et al.* 2002; Hall & Love 2003). Using the ink print technique landmarks on the proximal creases of the digits are apparent but landmarks on the finger tips are not fully captured; this can reduce finger length by at least 2 mm (John Manning pers. comm.). Tracing around the hand has also been used (Bang *et al.* 2005; Jürimäe *et al.* 2008). The technique was first described by Ecker 1875. This method can elongate digit lengths due to deviations in the angle of the pencil and the splaying out of digits. It also fails to provide information on the proximal crease of the digit; the web-spaces between the splayed out digits have to be used proxy for the proximal crease. In addition, splaying of the digits can lead to small changes in finger length (John Manning pers. comm.). Despite the potential disadvantages, however, this method does appear comparable to direct measures taken from the skin surface (Voracek & Dressler 2006a).

The same principal has been applied to measure finger lengths from hand outlines in order to use 2D:4D as a method of identifying the sex of artists of hand stencils from prehistoric cave sites (Chazine & Noury 2006; Snow 2006). These stencils (hand outlines) were created by blowing paint from the mouth (directly or via a straw) onto the back of a hand placed flat upon the cave wall. A research group working in Indonesia have developed a digital imaging

technique named Kalimain© to sex hand stencils (Chazine & Noury 2006). The details of the method have not been fully published or validated but their brief description of the technique suggests that the program combines calculations of 2D:4D with other anthropomorphic traits such as hand size, palm width and finger length to estimate the sex of the individual (Chazine & Noury 2006; also see Nelson *et al.* 2006).

2.2.1.3: Other measurement methods

Early studies of human fingers investigated the concept of *digital formula*. This method compares the relative lengths of the fingers rather than actual ratios (reviewed in Peters *et al.* 2002). Digit formula has recently been used to identify the sex of an individual by visually judging differences between the relative lengths of the 2nd ray length compared the 4th ray (length of metacarpals plus phalanges) from radiographs. Radiographs were categorised as 2D<4D, 2D>4D or 2D=4D and these groupings have been shown to correspond to 2D:4D (e.g., low 2D:4D; high 2D:4D; 2D:4D ~1; Robertson *et al.* 2009). Mal-positioning of the hands during the X-ray procedure, however, made some hands difficult to classify (Robertson *et al.* 2008).

Recently Loehlin *et al.* (2009), using a large sample of photocopied hands of Austrian twins (n=800) devised a new method to compare digit lengths, termed '*rel*' (also see Stenstrom *et al.* 2011). The *rel* measure is calculated by dividing the length of a given finger by the sums of all the other fingers (but not the thumb) (i.e., $2D/(2D+3D+4D+5D)$; see Voracek 2009). The authors state that this method may be superior to 2D:4D for investigating sex differences (Loehlin *et al.* 2009). This concept was tested by Voracek (2009) using a similar sized but more ethnic diverse sample. Comparisons of the models did not suggest that *rel* was more advantageous for investigating sex differences than 2D:4D. However, *rel* appears to be informative in terms of highlighting sex-linked developmental gradients (growth fields) across the hand. Females had relatively longer 2D and 3D, while males had relatively longer 4D and 5D. These sex-linked effects lead to more marked ratios for nonadjacent digits than adjacent digits (Voracek *et al.* 2008a; Voracek 2009; also see Takai 1979) and may be associated with early androgenic effects on limb development (see Oxnard 2000, p 249-250; Voracek, 2006).

A small number of studies have used more unconventional methods of digit measurement; ratio of the second and fourth digit tip relative to the third digit tip (Ramesh & Murthy 1977), summing the length of the second and fourth digits from scans ('*digit extension*'; Millet *et al.* 2005), the length of the second digit minus the fourth digit (Arato *et al.* 2004)

and digit lengths measured from a fixed central point on the wrist to the digit tip from hand outlines (Jürimäe *et al.* 2008). While these studies are informative about hand morphology and possibly prenatal androgen effects (PAE), their methodological distinctness limits their use in comparative studies of human 2D:4D.

2.2.1.4: Summary of measurement methods (soft-tissue)

The advantages of direct measures are that the soft-tissues of the finger are not distorted which can occur with indirect methods and should therefore provide a more accurate assessment of PAE (see below). A recent study has shown, however, that intra-observer repeatability is lower for direct measurements compared to indirect measurements (Allaway *et al.* 2009). This is to be expected given that soft-tissues distort and images do not. The differences in internal consistency for direct and indirect methods quoted by Allaway *et al.* (2009) based on the intra-class correlation coefficient (ICC; McGraw & Wong 1996) may significantly differ, but both methods remain relatively precise (indirect: ICC=0.96; direct: ICC=0.93).

Other disadvantages of direct measurements is that data collection has to be arranged around the availability of participants (unless they are providing self-measures) and this can be inconvenient and time consuming (Manning *et al.* 2005). This is made more difficult when sample sizes are large or when participants are only available for short periods. Self-measures with a ruler appear to be particularly prone to extreme values (Manning *et al.* 2007a; Manning & Fink 2008; Caswell & Manning 2009) but overall results are similar to values obtained by more experienced researchers (Burriss *et al.* 2007; but see Manning *et al.* 2010).

Imaging the hands from which indirect measurements are taken has the advantage of providing a permanent visual record that can be conveniently measured many times by different observers (Manning *et al.* 2005). The method is highly repeatable (Voracek *et al.* 2007a; Kemper & Schwerdtfeger 2009; Allaway *et al.* 2009) and data collection from images is less time consuming than direct measures (Kemper & Schwerdtfeger 2009). The disadvantage of this approach, however, is that imaging process can distort soft-tissues and this may affect the estimation of PAE. In a study by Manning *et al.* (2005) comparing 2D:4D ratios calculated from direct measurements and photocopies it was found that 2D:4D ratios derived from photocopies were lower than those based on directly measurements. This led to greater sex differences in the photocopied derived ratios compared to 2D:4D based on direct measurements (Manning *et al.* 2005; also see Allaway *et al.* 2009; but see Voracek &

Dressler 2006a; Voracek & Offenmüller 2007). When the differences in finger lengths were examined it was found that there was a tendency for imaged second digits to be shorter or unchanged from directly measured values and for the fourth digits to be longer or unchanged from directly measured. The fact that sex differences were stronger from the photocopy-derived 2D:4D than from directly measured ratios suggests that male and female fingers distort differently or are captured differently during the imaging process; male fingers distorted more than females. Additionally when the direct and indirect measures were compared between males unselected for sexual orientation and those who identified themselves as homosexual, the second digits of the homosexual group were shorter, while the fourth digits of unselected males were longer in 2D:4D ratios taken from photocopies but not in 2D:4D measured directly from the skin surface (Manning *et al.* 2005).

The shape of the finger pads is known to differ across the fingers and differ in males and females (Serina *et al.* 1997; Murai *et al.* 1997; Voracek *et al.* 2008b). This variation in pad size and elasticity of the tissues leads to differential distortion when pressing the hand against a hard surface such as a scanner plate. This evidence is suggestive of possible prenatal sex-linked developmental effects on fingertip morphology, possibly via interaction with the androgen receptor gene (see Manning 2002a, 2007a; Manning *et al.* 2003a). The distortion effects will be compounded by deviations in the pressure exerted by individuals as they press their hand on the scanner or photocopy plate. Functional influences on the soft-tissues (either as a result of natural selection or adaptations through life) cannot be ruled out.

A study addressing the relationship between finger tip morphology and 2D:4D was performed by Voracek *et al.* (2008b). A method for calculating Fingertip Index (FTI) was devised which incorporated the size of the fingertip (fingertip length) and the area of the fingertip in the sagittal plane. FTI was larger in males and larger on the left hand. Across the digits fingertips, 4D was the largest then 5D>3D>2D. Measurements were correlated with 2D:4D based on digit measurements from scanned hands (calliper measured from printed out images; n=40 males; 40= females). No relationship was found between FTI and scan-based 2D:4D. Studies have yet to investigate the potential relationships between FTI and 2D:4D based on direct measurements.

Manning *et al.* (2005) has proposed that the imaging process itself (photocopying or scanning) may produce distortions in the shape of the fingertip when the hand is converted from a three-dimensional shape to a two-dimensional image. In a study of facial dimorphism direct measurements derived from both self-measured and experimenter-measured 2D:4D yielded higher correlations with target traits than indirectly derived 2D:4D (Burriss *et al.*

2007; Almasry *et al.* 2011). A recent large study (n=1413 individuals) found differences in genetic co-variance with digit ratio when 2D:4D methods were compared (i.e., 2D:4D ratios of twins based on digit measurements derived from photocopies and those from digit measurements derived from digital scans from the same sample; Medland & Loehlin 2008). These findings indicate that different imaging methods appear to impact differently on 2D:4D and that this may potentially influence the outcomes of analyses.

To circumvent some of these effects it has been proposed by Manning *et al.* (2005) that within a study all digit data should be collected using the same technique; digit lengths calculated from indirect digit measurements should not be mixed with those derived from direct measurement techniques within the same study.

2.2.2: Measurements derived from bones

2D:4D ratios have also been calculated from bones. Summing the lengths of the three ray phalanges provides a measurement of bony digit length (Buck *et al.* 2006; Vehmas *et al.* 2006; McIntyre *et al.* 2005; 2006; Robertson *et al.* 2008; Galis *et al.* 2009; Bloom *et al.* 2010). Bone-derived 2D:4D from radiographs have been shown to significantly correlate with soft-tissue based 2D:4D in children (Manning *et al.* 2002a; also see Peters *et al.* 2002), although ratios calculated from bone lengths are generally lower than those based on soft-tissue lengths (e.g., Galis *et al.* 2009). The exact reason for this is not understood but may be linked to the fact that placement of the proximal crease of the digit does not directly overlay to the metacarpal-phalangeal joint (Voracek *et al.* 2007b; Fig. 2.3). This mismatch is most marked in the 4D. As such, the bony 4D is markedly longer than that soft-tissue digit length. Additional parallax effects associated with X-ray imaging techniques may also confound radiographic derived 2D:4D (Camasta *et al.* 1991).

A study of radiographs taken over growth in a large sample of children (left hand only) found that the sex differences in the hand bones are higher in the intermediate and distal phalanges of the fourth digit; these bones were longer in males compared to females in the same age class (McIntyre *et al.* 2005; 2006). However, as PAE also appear to be apparent in the finger pads (Serina *et al.* 1997; Murai *et al.* 1997; Buck *et al.* 2006; Vehmas *et al.* 2006) calculating 2D:4D from bones alone will inevitably fail to capture some of the PAE even though bone derived measurements will be more accurate (i.e., because measurements taken from a hard bone tissue will be more stable than measurements taken from soft-tissue landmarks that are prone to vary; but see Camasta *et al.* 1991).

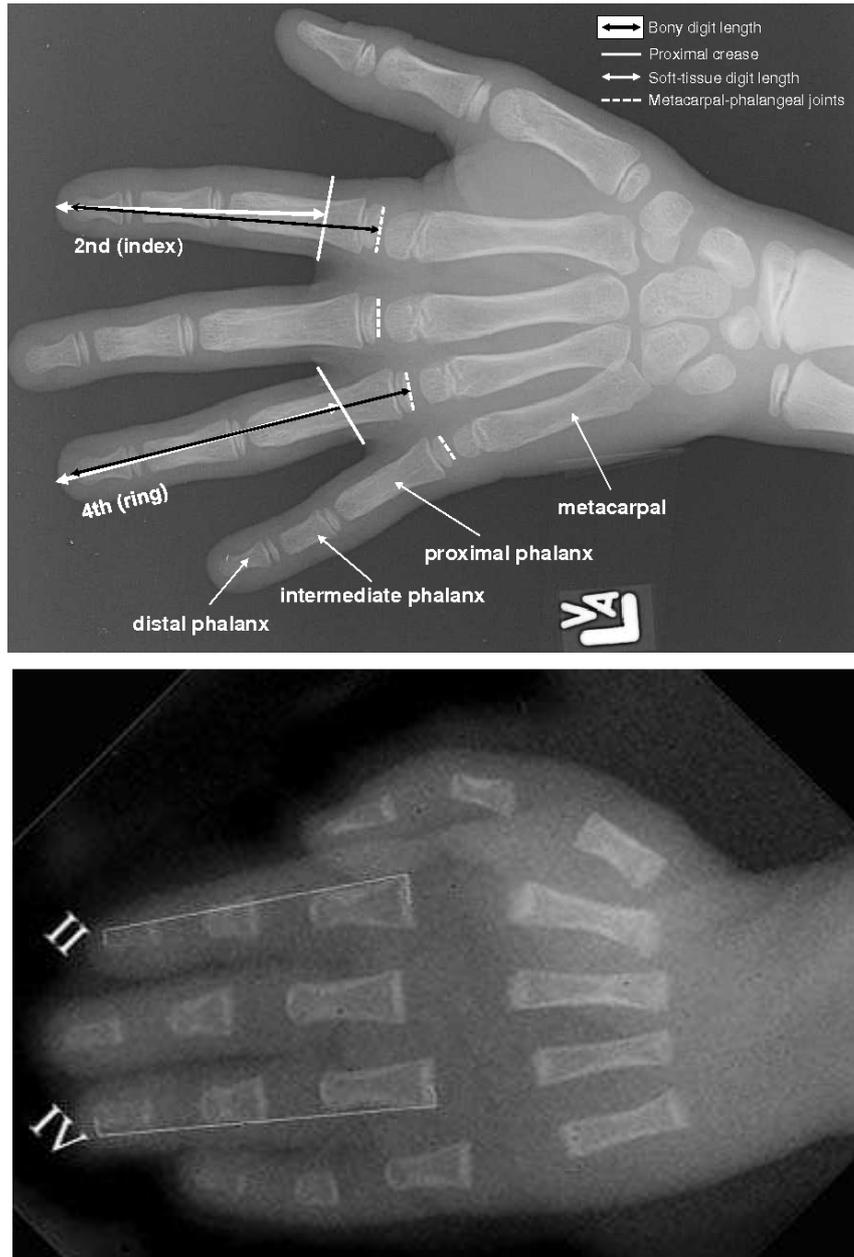


Figure 2.3: Comparison of radiograph derived digit lengths with soft tissue digit lengths. In adults the longer length of the bony 4D (ring finger) compared to the bony 2D is apparent (black arrows) (top image). In this image of a juvenile's hand the un-fused epiphyses of the 3 phalangeal bones of each digit are visible. Bottom image: X-ray of a foetus's hand. After Galis *et al.* 2009.

Ratios of the metacarpals and metatarsals (the metapodials) from the 2nd and 4th ray (in the hands and feet) have been compared with the summed lengths of phalanges from 2D and 4D (McFadden & Bracht 2009; also see McFadden & Shubel 2002). Metacarpal ratios were not found not to correlate with summed phalangeal bone length ratios in a study of human and

non-human primates bones from museum collections (McFadden & Bracht 2003; 2005; 2009). However these studies did not include distal phalanges in their measurements which may have contributed to the lack of relationship between the differently derived bone ratios (see McIntyre *et al.* 2005). Recently a study using radiograph derived bone measurements on an elderly human clinical cohort found that the ratios of the 2nd metacarpal and the 4th metacarpal (2M:4M) showed significant correlations with bone 2D:4D in the expected direction; males had lower 2M:4M ratios than females (Robertson *et al.* 2008; also see Zhang *et al.* 2008).

2.2.2.1: Summary of bones measurements

Although radiograph derived data yields lower 2D:4D ratios than soft-tissue derived data sexual dimorphism in the ratios is in the expected direction (males < females) and is evident from the first trimester of gestation (see Galis *et al.* 2009). During prenatal growth 2D:4D has been shown to increase in a stable manner as a consequence of positive growth in 2D. In children PAE effects appear to be largely concentrated in the 4D (McIntyre *et al.* 2005; 2006) although growth of immature digits appears to be highly variable (Bloom *et al.* 2010; Manning 2010). As with other imaging techniques, such as photocopying, there is the potential for loss of data in radiographic images due to poor positioning and failure to capture the full bone length (Robertson *et al.* 2008). Additional factors such as parallax effects can also distort length measurements but these factors may be minimised by adhering to standard procedures (Camasta *et al.* 1991).

2D:4D from bone derived measurements provide an additional means of capturing PAE and provides the potential for investigating ancient populations (see McFadden & Bracht 2003). For example McFadden and Bracht (2009) have recently used museum specimens to calculate bone-derived digit ratios of matapodials (metacarpals and metatarsals). However, museum-based collections must ensure that disarticulated hand bones are correctly assigned to an individual; incorrect assignment will lead to erroneous results. Osteological studies show that disarticulated proximal phalanges can be confidently assigned to the correct ray, although this is not possible for intermediate or distal phalanges (Case & Heilman 2006).

2.2.4: Factors to consider

Comparisons of direct and indirect methods suggest that the prenatal hormonal environment not only contributes to differences in soft-tissue and bony finger length but also to fingertip

morphology (Manning *et al.* 2005; also see Murai *et al.* 1997; Voracek *et al.* 2008b). Male and female fetuses are exposed to different intra-uterine sex hormones as part of the sexual differentiation process (Phoenix *et al.* 1959; Wallen & Baum 2002) and sex differences in 2D:4D are evident from 9 weeks of development (Malas *et al.* 2006; Galis *et al.* 2009). It has recently been shown that the finger proportions of children conceived by a method of artificial conception (intra-cytoplasmic sperm injection) differ from children conceived normally (Sutcliffe *et al.* 2010). It is possible that low fertility in parents is passed on to their children and this is reflected in subtle differences in their digit ratios (see Manning *et al.* 1998). Perturbations in prenatal sex hormones have been associated with variation in sexual orientation (see Rahman & Wilson 2003; McFadden *et al.* 2005) and these appear to impact indirect measurements of 2D:4D (see Manning *et al.* 2005). Differences in tissue responses may also be found between ethnic groups. For example variation in 2D:4D has been shown between populations from different geographical regions (e.g., Manning *et al.* 2000a; Loehlin *et al.* 2006; Trivers *et al.* 2006; Helle & Laaksonen 2009). These effects may interact with population-level differences in the androgen receptor gene (ARG), which is known to differ across populations (Kittles *et al.* 2008) and correlate with 2D:4D (Manning *et al.* 2003a).

Human 2D:4D is known to change slightly in males and females over childhood although sexual dimorphism remains stable over growth (Trivers *et al.* 2006). There are no perceivable changes in 2D:4D at puberty (Manning 2002a) and the ratio stabilises in adulthood (Manning *et al.* 2004a; McIntyre *et al.* 2005; 2006; Trivers *et al.* 2006; Gillam *et al.* 2008). In older adults intra-digital joint spaces reduce as part of the ageing process (Harris *et al.* 1992; Vehmas *et al.* 2006; Zhang *et al.* 2008) and soft-tissue elasticity decreases, particularly in menopausal women (Kurabayashi 2004; Sumino *et al.* 2004; also see Zhang *et al.* 2008). This indicates that 2D:4D may alter in later life and that tissues of immature and aged hands may distort differently in different age classes. These soft-tissue effects may also differ between hands and even across digits (Voracek *et al.* 2008b; Voracek 2009). We currently do not know how direct and indirect digit measurements compare in immature and older individuals or how 2D:4D may be affected by the ageing process in adults in terms of intra-individual changes over a lifetime. Additionally, links between 2D:4D and disease (see Manning & Bundred 2001; Zhang *et al.* 2008) make it difficult to disentangle normal degenerative effects on 2D:4D from changes in hormonal conditions that might also affect the tissues of the hand.

Multiple confounding factors interacting with methodologies can lead to problems comparing results across studies of 2D:4D. For example early studies mostly used direct

measurements, while more recent studies have tended to use indirect methods (Voracek and Loibl 2009). It is possible that the use of differing methodologies and lack of controls for confounds such as ethnicity and age may be partly responsible for the failure to replicate some findings such as the inconsistencies in results of multiple studies investigating 2D:4D and homosexuality (see McFadden *et al.* 2005). These factors also highlight the importance of measurement rigor and the need for careful controls of within- and between-sample factors (Manning & Hill 2009).

Evidence from radiographic studies suggests that bone-derived data offer an additional method of studying digit ratios, although PAE may be weaker than soft-tissue based measures. However, unlike soft-tissue measures radiographic and skeletal collections provide the potential for investigating PAE between contemporary human populations as well as historical and prehistoric periods, including extinct hominins (McFadden and Bracht, 2003; 2009).

2.2.5: Measures of internal consistency

Relationships between 2D:4D and target traits are often weak and measurement error can affect relationships, particularly if sample sizes are small (Peters *et al.* 2002; Voracek *et al.* 2007a; Manning & Fink 2008). Testing the reliability (internal consistency) of a method is done by estimating observer error of repeat measures taken by the same data collector or more than observer (Voracek *et al.* 2007a). The main methods used to estimate measurement error are Pearson's correlation coefficient (r), Cronbach's alpha, and intra-class correlation coefficient (ICC) (via a two-way mixed effect model with absolute-agreement definition; McGraw & Wong 1996). ICC is the most widely used measure and the most appropriate, as, unlike Pearson's r and Cronbach's alpha, ICC takes into consideration possible scale and location shifts in data; measurement sets with unequal variances and different means, respectively (McGraw & Wong 1996; see Voracek *et al.* 2007a).

Comparisons of ICCs for indirect methods show that precision is highest in methods that use software to measure the digital images (e.g., Adobe PhotoShopTM and Autometric; DeBruine on-line application) and less precise using manual methods (e.g., calliper or ruler to measure photocopied hands; see Kemper & Schwerdtfeger 2009; Allaway *et al.* 2009). However Voracek *et al.* (2007a) did not detect differences in ICCs between ruler and calliper methods in a study measuring intra-observer error in a set of 50 hand scans measured by 16 experienced investigators. ICC values were found to be higher for individual finger length measurements but lower when length measures are transformed into 2D:4D (for left and

right hands separately). ICC's improve, however, when 2D:4D is transformed into mean value for an individual (2D:4D based on mean digit lengths of both hands; see Appendix 2.1). Results also showed that similarities in the precision of measurements using different instruments (calliper and ruler).

2.2.6: Summary of methods in humans

In humans adopting a standard hand position allows for comparisons of results between studies using the same methodology. Direct measures are considered to be the ideal method because fingertips are not distorted and landmarks are clearly visible (Manning & Hill 2009; Manning & Fink 2008; Manning *et al.* 2007a). However the direct method can be less precise because soft-tissue landmarks are not static and can alter slightly when the calliper is placed on them. The widespread use of indirect methods has been instrumental in expansion of the discipline. In summary, both direct and indirect techniques have advantages and disadvantages; neither is ideal.

2.3: Studies of digit lengths in non-human animals

This section will review methodologies used in studies of 2D:4D in non-human animals; rodents, lizards, birds and non-human primates.

2.3.1: Measurements of rodent digits

Studies of 2D:4D have been performed on laboratory rats, laboratory mice, the wood mouse and field voles (Table 2.1). In general the rear paws have been favoured over the forepaws because the digits of the forepaws are smaller and more strongly curve making them difficult to measure (Manning *et al.* 2003c; Brown *et al.* 2002a; Leoni *et al.* 2005). The age range between samples (from foetal to adult) increases the size variation of digits across studies (Table 2.1). Of the studies that have published data on digit length, sizes were shown to range between 2.0-2.8 mm for 2D and 2.8-3.75 mm for 4D of adult laboratory mice (data taken from a graph; Manno 2008) and in juvenile laboratory rats averages are shown to be around 5.8 mm for 2D and 6.5 mm 4D (McMachan *et al.* 2004). Most studies used a microscope for direct measures and for taking digital images (but see Leoni *et al.* 2005).

Table 2.1: Rodent Studies. NI=Not indicated; #formaldehyde; F= forepaws, B= rear-paws; J=juvenile, A=adult; ^=ICC; *= Correlations for soft tissue; **= Correlations for X-rays; r=Cronbach's Alpha.

Genus	Species	Common name	n male	n female	Age	Live Dead	Limb	Method	Technique	Repeatability	Ref
<i>Mus</i>	NI	Lab mouse	15	17	J	D	B	D	Ruler	NI	Brown <i>et al.</i> 2002a
<i>Mus</i>	NI	Lab mouse	20	19	A	D	B	D	Ruler with pin	NI	Brown <i>et al.</i> 2002a
<i>Mus</i>	NI	Lab mouse	70	41	NI	?	B	NI	NI	NI	Manning <i>et al.</i> 2003c
<i>Mus</i>	strains ID	Lab mouse	93	92	A	D	B	I	Digital photo	r=0.92	Bailey <i>et al.</i> 2005
<i>Mus</i>	NI	Lab mouse [#]	494	519	NI	D	B	I	Digital photo	L=0.66, R=0.54 [^]	Yan <i>et al.</i> 2008
<i>Mus</i>	NI	Lab mouse	39	39	A	L	F&B	I	Digital photo	Stated to be high	Manno 2008
<i>Mus</i>	strain ID	Lab mouse	44	51	Foetal	D	B	I	Digital photo	r>0.61	Hurd <i>et al.</i> 2008
<i>Apodemus</i>	<i>sylvacticus</i>	Wood mouse	16	26	NI	D	B	D	Callipers (digit bones)	R ² =0.95	Leoni <i>et al.</i> 2005
<i>Microtus</i>	<i>agrestis</i>	Field vole		66	J	D	B	I	Digital photo & X-ray	r=0.50* r=0.88**	Lilley <i>et al.</i> 2010
<i>Microtus</i>	<i>agrestis</i>	Field vole		66	A	D	B	I	Digital photo & X-ray	r=0.52* r=0.88**	Lilley <i>et al.</i> 2010
<i>Rattus</i>	NI	Lab rat	5	5	J	D	F	I	Scanned	NI	McMechan <i>et al.</i> 2004
<i>Rattus</i>	<i>norvegicus</i>	Wister rats	8	8	A	L	F	D	Callipers	digits r>0.98	Talarovičová <i>et al.</i> 2009

2D:4D studies on rodents have been performed on live animals as well as sacrificed specimens and formaldehyde preserved specimens (Table 2.1). The technique of gluing hands to a board in order to maintain and fixed position has been favoured by some studies. Live animals are hand restrained for measurement (Talarovičová *et al.* 2008; Manno 2008). Indirect methods are more popular than direct measurements (Table 2.1).

2.3.1.1: Direct measurements

During direct measurement of the digits the hands can be manipulated to enable landmarks to be clearly visualised. For example in a study on live rats digits were measured with callipers while the animals were hand restrained (Talarovičová *et al.* 2008). It might be expected that small movements of the animal during measurement would have a negative impact on the accuracy of digit measurements. However in the study by Talarovičová *et al.* (2008) repeatability of measurements was very high ($r=0.98$). It is not stated if a time gap was left between repeated measurements; re-measuring digits without a time gap will increase correlations between repeated measures although a decrease in the handling time will minimise stress in the animal. To increase accuracy of direct measures in sacrificed mice Brown *et al.* (2002a) placed a pin at the zero point of a ruler (Fig. 2.4) and measured digit length by positioned the pin in the web spaces between digits. However, Bailey *et al.* (2005) found high variation in the depth of the webbing in between the digits in their sample of 8 strains of inbred mice while the basal crease of the digit did not vary. This suggests that, in mice, using the web between the digits as a proxy for the basal crease may be inappropriate.

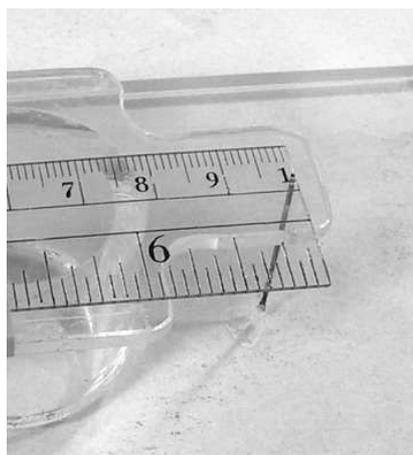


Figure 2.4: Attachment of a pin on a ruler to increase measurement accuracy of rodent digits. After Brown *et al.* 2002a.

2D:4D ratios have been calculated from direct measures taken from digit bones dissected from the hands of the wood mouse (Leoni *et al.* 2005). Soft-tissue digit length was not measured in this sample. In addition, the distal phalanges were not measured due their small and fragile state. Measurements taken using callipers directly from the bones were found to yield higher repeatability after a time gap of at least a day ($R^2=0.95$; Table 2.1).

2.3.1.2: Indirect measurements

The advantages of digitally imaging rodent hands are that the tiny landmarks can be highly magnified and measured several times. Digital techniques also allow hands to be inverted (rendering them all to left or right hands) to minimise measurement bias. However, measures of internal consistency range widely across studies and species (Table 2.1). Manno (2008) used digital images to measure digit lengths of both sets of paws of live mice by restraining them on a scanner. An example of one of the scans (Fig. 2.5) clearly shows, however, that this method is inappropriate for visualising digit lengths in this species; raised areas on the palm of the paw do now allow the digits to be laid flat and this prevents accurate linear measurements of digit length and visualisation of the proximal creases of some of the digits.

Lilley *et al.* (2010), in a study of field voles, also used an imaging method but in contrast to Manno (2008) rear paws were digitally photographed through a microscope. However the authors' comment that multiple creases around the proximal crease caused difficulties in identifying the correct landmark. They actually state that "one [crease] had to be chosen" (Lilley *et al.* 2010). This indicates that an element of guesswork was involved in some of the measurements. In the same study the bony digit lengths of the field voles were also measured from digitised radiographs of field vole's forepaws (glued onto card). Measurement of bones from radiographs provided more reliably measurements and yielded significantly higher correlations between repeats than soft-tissue derived ratios (Lilley *et al.* 2010; Table 2.1).

Visualisation of landmarks has also been shown to be problematic in foetal samples. Hurd *et al.* (2008) correlated 2D:4D with intra-uterine position in foetal mice (i.e., if a pup gestated next to two males, two females or a male and a female at 18 days of gestation). The authors state that the proximal crease was not visible on the foetal 4D and used an extrapolation method based upon the webbing on 3D by extending a line between the basal mounds of the palm and proximal digits on images of the paw (Fig. 2.6). Despite visualisation problems correlations between repeated measures were satisfactory ($r=0.72-0.61$ for the two raters).



Figure 2.5: Digital scan of male mouse right hind-paw.
After Manno, 2008.

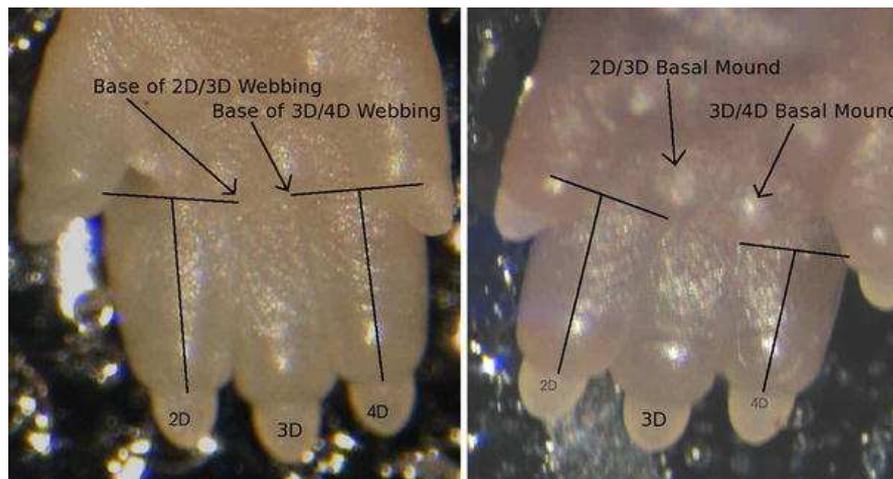


Figure 2.6: Measuring digits in mice. Left image, left hind- paw of a mature male mouse.
Right image, hind-paw of a foetal male mouse.
After Hurd *et al.* 2008.

2.3.1.3: Summary of rodent studies

Leoni *et al.* (2005) argue that the small sizes of rodent digits will increase measurement error. Measurements derived from different tissues (bone and soft-tissues) and individuals from different age classes are also expected to increase variability in digit length measurements. Attempts to improve precision have led to indirect methods being favoured, with a number of studies attempting to standardised paw position (e.g., gluing the paw to

cardboard before imaging). However, indirect methods may not capture vital information such as proximal creases or/and may distort digits lengths through soft tissue changes or mal-positioning (i.e., the inability to lay digits flat on the scan plate; Fig. 2.5). These factors are highlighted by evidence of morphological differences in the paws within strains, between strains, between ages-classes and between limbs within and between species (e.g., Manning *et al.* 2003c; Bailey *et al.* 2005). Thus different indirect methods may be required for different animals and possibly different limbs. These issues potentially confound comparisons of 2D:4D across studies and across species within the Rodent Order. Direct measurements using standard landmarks may be the most appropriate methodology. Ideally these should be taken on euthanased or restrained animals, as preservation techniques (formaldehyde; Yan *et al.* 2008) can alter the structure of soft-tissues (see Rubolini *et al.* 2006).

All except one (Leoni *et al.* 2005; wood mice) of the rodents studies to date have been performed on laboratory reared animals. The fact that 2D:4D varies between strains of laboratory mice (Bailey *et al.* 2005) suggest that the ratios will also vary between wild-born populations and should be considered when comparing across studies. Species names and strain-type of animals should also be provided in all studies along with the results of repeatability tests; this standard practise is currently not adhered to. It is of note that some studies make conclusions based upon unreliable measurement methods (e.g., Hurd *et al.* 2008; Lilley *et al.* 2010). In human 2D:4D research images of hands that do not yeild clear visualisation of anatomical landmarks are rejected from the sample because even small errors in digit lengths can lead to big changes 2D:4D (Voracek *et al.* 2007a). In animals as small as rodents, these effects are likely to be magnified (Leoni *et al.* 2005).

2.3.2: Measurement of reptile digits

Studies of 2D:4D have been performed on green anoles, wall lizards and tree skinks (Table 2.2). One study obtained direct digit measurements from a live animal and two studies used indirect methods from preserved animals (Table 2.2). In lizards the forefeet were favoured because rear feet have an unusual shape and are often damaged in preserved specimens (Rubolini *et al.* 2006). Both sets of feet were measured in the green anoles (*Anolis carolinensis*; Lombardo & Thorpe 2008; Table 2.2). All measurements were on wild-born, mature individuals. Absolute length sizes are only published for green anoles with mean lengths ranging between 3.35-4.38 mm for 2D and 5.51-10.40 mm for 4D (Lombardo & Thorpe 2008).

2.3.2.1: Direct measurements

Digit length measurements from green anoles were obtained using callipers from live animals restrained by hand. Landmarks used in this species are the same as for humans; proximal crease on the ventral aspect of the hand to a point on the digit tip where the nail emerges. Correlations between repeated digit measurements were high ($r > 0.96$).

2.3.2.2: Indirect measurements

Digit length measurements from hand images of dead green anoles (*Anolis carolinensis*), lizard (*Podarcis muralis*) and the tree skink (*Mabuya planifrons*) were obtained using measuring software from digital images taken through a dissecting microscope and from scanned images (see Lombardo & Thorpe 2008). All individuals had been preserved for an extensive period in 75% alcohol. The forefeet of the lizard and skink species were fixed in an extended position and inserted into small clear plastic tubes fixed with tape (Fig. 2.7).

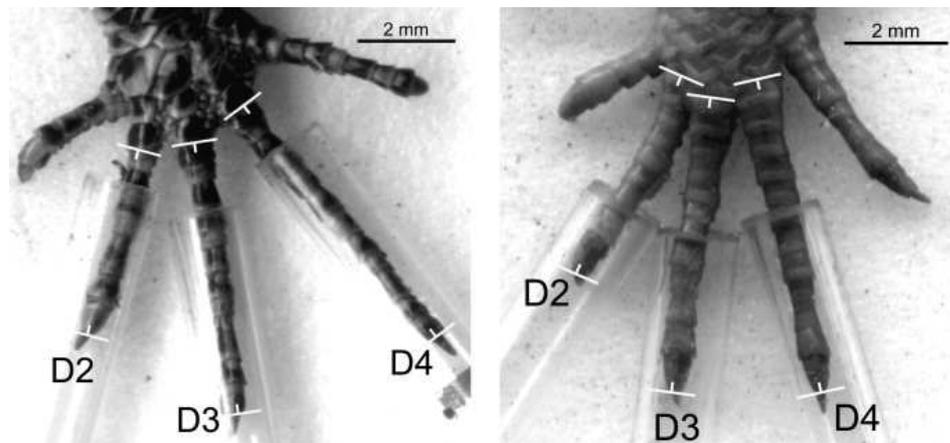


Figure 2.7: Landmarks in the forefoot in lizards (dorsal aspect). Right wall lizard (*P. muralis*) and left tree skink (*M. planifrons*). Digits fixed in plastic tubes.
After Rubolini *et al.* 2006.

Table 2.2: Reptile studies. # preserved in chemicals; F= forefoot, B= rear-foot; A=adult;*= Correlations on digit repeats; ^ ICC's of front left forefoot 2D:4D, ^^ ICC's of front right forefoot 2D:4D; r=Cronbach's Alpha.

Genus	Species	Common name	n male	n female	Age	Live Dead	Limb	Method	Technique	Repeatability	Ref
<i>Anolis</i>	<i>carolinensis</i>	Green anoles	61	87	A	L	F&B	D	Callipers	r>0.96*	Chang <i>et al.</i> 2006
<i>Podarcis</i>	<i>muralis</i>	Wall lizard [#]	18	18	A	D	F	I	Scanned	r=0.94*	Rubolini <i>et al.</i> 2006
<i>Mabuya</i>	<i>planifrons</i>	Tree skink [#]	17	11	A	D	F	I	Scanned	r=0.87*	Rubolini <i>et al.</i> 2006
<i>Anolis</i>	<i>carolinensis</i>	Green anoles [#]	25	25	A	D	F&B	I	Digital photo	^=0.78, ^^=0.44	Lombardo & Thorpe 2008

Contrary to human measuring techniques (and those of most other animals), digit measurements for lizards were taken from the dorsal (back) aspect of the foot (Fig. 2.8). In the wall lizard the proximal landmark was taken from the second dorsal scale, in the skink the joint is obscured by a scale and was estimated as being located at the “middle point of the line perpendicular to the digit axis and tangential to the imprecated scale covering the metacarpals” Rubolini *et al.* 2006 (Fig. 2.7). In the green anoles conventional landmarks were used (i.e., taken from the proximal crease of the digit on the palmer surface). Correlations between repeated digit measurements across reptile species were high ($r > 0.87$), but in the only study to quote ICC values, results for 2D:4D were much lower for the right forefoot compared to the left (ICC=0.78 v 0.44) indicating that the method had low measurement precision (Table 2.2).

2.3.2.3: Summary of reptile studies

Only green anoles have been used in more than one study. This species shows evidence of sexual dimorphism in 2D:4D ratios in the right rear feet in direct measurements on live animals (Chang *et al.* 2006). However, sexual dimorphism in 2D:4D was not detected in this species in a study using indirect measurements (Lombardo & Thorpe 2008). Two species of lizard had their digits measured from the dorsal (back) aspect of the foot. It was not made clear if this aspect was chosen because the palm did not show standard landmarks or for some other reason. In lizards the digits do hyperextend against the substrate so it may be appropriate to use landmarks on the dorsal aspect of the foot. There have been no studies showing that taking digits lengths from the ventral aspect of the hand/foot correspond to measurements taken on the dorsal aspect. More research is needed to ascertain the impact of different measurement methods on digit lengths and 2D:4D. The effects of chemicals on soft-tissue preservation and how these effects may vary across species also need to be understood and controlled for.

2.3.3: Measurement of bird digits

Studies of digit ratios in birds have been performed in 10 species (Table 2.3). Selection for wings in the forelimb forces studies to focus on the pedal digits. Some studies have measured digits from the dorsal aspect of the foot (Romano *et al.* 2005; Saino *et al.* 2007; Leoni *et al.* 2008; Fig. 2.8) while the others have used the palmer aspect (Table 2.3). All but one study (Lombardo *et al.* 2008) have used live bird. Six studies have used wild-caught birds; the remainder were on captive samples (Table 2.3).

Table 2.3: Bird studies. NI=No information; R= right foot; L=left foot; J=juvenile, A=adult; Cal=callipers; *=wild caught; ~=dorsal aspect of foot; #= 1D:3D only ; r=Cronbach's Alpha.

Genus	Species	Common name	n M	n F	Age	Live Dead	Limb	Method	Technique	Repeatability	Ref
<i>Taenipygin</i>	<i>guttata</i>	zebra finch	47	56	A	L	R	I	Cal (ink prints)	r>0.9	Burley & Foster 2003
<i>Phasianus</i>	<i>colchicus</i>	ring-neck pheasant	153	133	A	L	R&L	D	Cal~	r2>0.66	Romano <i>et al.</i> 2005
<i>Taenipygin</i>	<i>guttata</i>	zebra finch	258	242	A	L	R	D	Cal	r=0.88	Forstmeier 2005
<i>Taenipygin</i>	<i>guttata</i>	zebra finch	15	15	A	L	R	I	Cal (ink prints)	r0.79	Forstmeier 2005
* <i>Passer</i>	<i>domesticus</i>	house sparrow	46	20	A	L	R&L	I	Cal (pinholes)	R=r=0.54; L=r=0.77	Navarro <i>et al.</i> 2007
<i>Phasianus</i>	<i>colchicus</i>	ring-neck pheasant	50	62	A	L	R&L	D	Cal~	r>0.65	Saino <i>et al.</i> 2007
* <i>Ficedula</i>	<i>albicollis</i>	flycatcher	70	0	A	L	R	I	Cal (pinholes)	2Dr=0.77; 4Dr=0.70	Garamszegi <i>et al.</i> 2007
* <i>Hirundo</i>	<i>rustica</i>	barn swallow	44	45	A	L	R&L	I	Cal (pinholes)	R=r2=0.73; L=r2=0.73	Dreiss <i>et al.</i> 2008
<i>Taenipygin</i>	<i>guttata</i>	zebra finch		23	A	L	R	D	Cal	ref Forstmeier 2005	Forstmeier 2008
*# <i>Puffinus</i>	<i>maurentanicus</i>	puffin		100	A	L	?	D	Cal	NI	Genovart <i>et al.</i> 2008
<i>Corvus</i>	<i>corone</i>	hooded crow		70	A	D	R&L	I	X-ray (pixals)	r=>0.94	Leoni <i>et al.</i> 2008
<i>Corvus</i>	<i>corone</i>	hooded crow		70	A	D	R&L	D	Cals~	r=>0.94	Leoni <i>et al.</i> 2008
* <i>Passer</i>	<i>domesticus</i>	house sparrow	8	12	A	L	R&L	I	Digital photo	ICC=0.34	Lombardo <i>et al.</i> 2008
* <i>Trachycinata</i>	<i>bicolor</i>	tree swallow	39	38	A	L	R&L	I	Digital photo	ICC=0.34	Lombardo <i>et al.</i> 2008
<i>Melospittacus</i>	<i>undulates</i>	budgerigar	16	25	A	L	R&L	I	Digital photo	ICC=0.20	Lombardo <i>et al.</i> 2008
<i>Gallus</i>	<i>domesticus</i>	chicken	12	12	A	L	R&L	D	Cal	ICC=0.27	Lombardo <i>et al.</i> 2008

2.3.3.1: Direct measurements

Callipers were used to take direct measurements. The only study to use ICC to estimate measurement error (Lombardo *et al.* 2008) showed that direct digit measurements taken from chickens had very low repeatability (ICC=0.27; Table 2.3).

2.3.3.2: Indirect measurements

A variety of methods have been used to obtain indirect digit measurements from birds. One study used ink prints (Burley & Foster 2004). Forstmeier (2005) tested the ink print method but found it to have low repeatability and rejected it from his methodology. Another study fixed weights to the nails of digits to ensure extension was fully maintained during the measuring procedure (Leoni *et al.* 2008). Two studies placed the bird's foot on a piece of white cardboard and used small pins to pierce holes in the cardboard at points between the digits (web-space) and at the ends of the digits (Navarro *et al.* 2007; Dreiss *et al.* 2008). Length measurements were then taken from the web space pin hole to the digit tip pin hole. However, it can be seen that in the pheasant (Fig 2.8) that the web space does not correspond to the proximal part of the digit. In addition the thickness of the inter-digital web may vary between individuals; a similar observation has been made between strains of mice (Bailey *et al.* 2005; see above). Digit measurements taken from digital photos using computer graphics programmes yields low ICC values compared to other indirect techniques (Lombardo *et al.* 2008; Table 2.3). Finally, digit lengths have also been estimated by calculating the summed lengths of phalanges from radiographs and these have been shown to correspond to soft-tissue measurements taken directly from the same bird (Leoni *et al.* 2008).

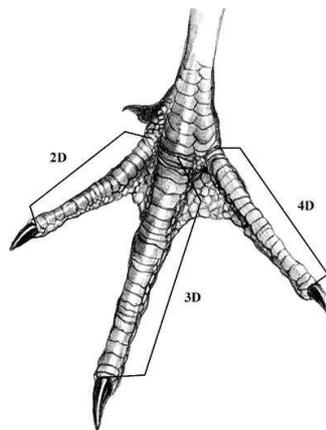


Figure 2.8: Landmarks in the forefoot in *Phasianus colchicus* (dorsal aspect).
After Saino *et al.* 2007.

2.3.3.3: Summary of bird studies

As with other taxonomic groups of non-human animals with varying foot morphology, bird studies have employed a variety of methods to measure digit length. It has been proposed that stronger selection processes to constrain foot morphologies in some bird species may obscure PAE on relative digit lengths (see Burley & Foster 2006; Lombardo *et al.* 2008). In addition, it is not known if foot digit ratios approximate to ratios of the forelimbs even though these limbs are adapted for flight (wings); forelimb bone ratios may also signal PAE. Finally, for the one study that has employed ICC, values were poor indicating low measurement precision (ICC=0.20-0.34; Lombardo *et al.* 2008).

2.3.4: Measurement of non-human primate digits

Studies have been performed on three species of primate; guinea baboons (*Papio papio*), chimpanzees (*Pan troglodytes*) and bonobos (*Pan paniscus*; Table 2.4). All were on sedated animals from captive facilities although the *Pan sp.* data were taken from individuals in Congolese sanctuaries, which suggest that they might have been wild-born. The same anatomical landmarks used in human studies are used in non-human primates.

2.3.4.1: Direct measurements

Digit length measurements of Guinea baboons were taken using a ruler (right hands only; Roney *et al.* 2004). The zero point of the ruler was placed on the proximal crease and the value was read-off at the tip of the digit. Due to time constraints measurements were only taken once.

2.3.4.2: Indirect measurements

A digital scanner (Canon CanoScan™) was used to image the hands of sedated bonobos and chimpanzees. Digits were measured using the Adobe PhotoShop measuring tool. Only those individuals who that had a scans with clearly identifiable landmarks for both hands were used in the study; scans of 18 chimpanzees and 5 bonobos had to be rejected due to unclear images and, in a few cases, digit injuries (McIntyre *et al.* 2009). Twenty percent of scans were measured by a second observer and ICC was used to assess repeatability. ICC values were higher for the right than the left (Table 2.4).

Table 2.4: Primate studies. A=adult; M=multiple age ranges; R=right hand, L=left hand; *= ICC bonobos and chimpanzee sample were pooled.

Genus	Species	Common name	n male	n female	Age	Live Dead	Limb	Method	Technique	Repeatability	Ref
<i>Papio</i>	<i>papio</i>	Guinea baboon	11	21	A	L	R	D	Ruler	Not repeated	Roney <i>et al.</i> 2004
<i>Pan</i>	<i>paniscus</i>	bonobo	27	12	M	L	R&L	I	Scanned	*R=0.886; L=0.709	McIntyre <i>et al.</i> 2009
<i>Pan</i>	<i>troglodytes</i>	chimpanzee	39	40	M	L	R&L	I	Scanned	*R=0.886; L=0.709	McIntyre <i>et al.</i> 2009

2.3.4.3: Summary of non-human primate studies

In the two studies published to date (three species), both direct and indirect methods have been used; however authors using the scanned approach did experience some difficulties in imaging the hand due to the naturally flexed position of the fingers. Scanning hands is known to distort soft tissues of the finger tips in humans (Manning *et al.* 2005; Allaway *et al.* 2009), it is therefore not unreasonable to assume that similar distortional effects will also occur in non-human primates. Direct measurements would seem more appropriate for primates because the hand rests in a flexed position and fingers cannot be extended and adducted on request.

2.4: Summary of published methodologies

2D:4D has now been now been investigated in four taxonomic groups; scaly lizards, birds, rodents and primates. These studies are constrained by many factors (outlined above), most are associated with variation in limb anatomy. Research in humans indicates that indirect measurements may be more convenient and precise, but indirect measurements may distort finger lengths and reduce the clarity of landmarks. This may be particularly important for non-human taxa in which variation in hand/paw morphology is high (e.g., reptiles). As a result, direct measurements of digits represent the most appropriate method of measuring digit length for studies of 2D:4D.

2.5: Main study: An extension of non-human primate 2D:4D research

Higher primates - *Haplorrhini* - are the suborder within the Order Primates to which humans belong. This means that developmental processes of haplorhines will be more similar to those of humans than to reptiles or altricial mammals. Although haplorhines inhabit a wide variety of ecological settings which impact on hand structure (Jouffroy *et al.* 1993; Richmond 2007) hand morphology across the Order is generally similar (Napier 1980; Ankel-Simons 2000). Haplorhines represent an appropriate model to compare against human 2D:4D research.

The main aim of this thesis is to investigate patterns of the 2D:4D across species to investigate relationships between PAE and social behaviour. In order to achieve this aim, two main problems areas were identified:

1) Access to data: Conscripting institutions that house primates to provide digit measurements. Obtaining data from a broad range of species and obtain sample sizes that were large enough to provide meaningful statistical results.

2) Accuracy of the data: Ensuring standardisation of the measuring procedure so that cross-species comparisons are not distorted by imprecision of data derived from many institutions.

These issues will be addressed in the following sections.

2.5.1: Access to data

Haplorhine primates can be found in the wild, in captivity but free-ranging, or captive within a facility (e.g., a zoo or a primate research facility). Primates are less commonly found in domestic settings (Duarte-Quiroga & Estrada 2003). All of these groups, apart from domestically kept primates, were considered for data provision. Establishing collaborations with researchers studying wild species that were also planning to sedate individuals, however, proved difficult to arrange in the timeframe of this study. A dataset was collected from a free-ranging population as part of an intra-specific study (see Chapter 5), but all other data was obtained from captive animals. Access to sedated captive primates is highly restricted for health and safety reasons, data was therefore collected by the staff from the zoo or primate facility.

Soft-tissue digit length ratios from captive primates can be obtained under two circumstances; a) While the animal is sedated; b) After death.

a) Research facilities regularly sedate their animals to perform health checks or for clinical purposes. Zoos do not tend to sedate their animals on a regular basis. As such measurements from zoo primates had to be collected on an opportunist basis while the animal was sedated for another procedure (e.g., wound repair or contraceptive implant).

b) The National Museums of Scotland (NMS) acquire rarer species of primates for their collections from zoos throughout Europe and agreed to collect digit length data from cadavers. However, due to potential distorting affects of freezing and thawing on the tissue, it was decided that measurements from dead animals and live animals

should not be mixed within the same dataset. Data from cadavers was utilised separately in Chapter 7.

2.5.1.1: Petitioning institutions

Standard letters were sent to 160 zoos and research facilities that were known to house haplorhine primates. If the institution did not reply within two months a brief e-mail was sent to the research co-ordinator or facility manager to remind them of the study. If there was no response to the second appeal no further contact was made. If the institution expressed an interest in the study, an information pack was posted to them which contained a copy of the protocol (Appendix 2.2a), a laminated measurement instruction sheet (Appendix 2.3), a data collection spread sheet and a ruler.

2.5.2: Study protocol

The study protocol included background information, hypotheses, rationale for the use of particular primate species, time-frame of study, step-by-step guide, data analyses and information on the dissemination of results (Appendix 2.2a). The protocol was approved by the School of Archaeology, Classics and Egyptology Research Committee, University of Liverpool; see Appendix 2.2b).

Due to the opportunistic nature of data collection it was impossible to predict how much data would be provided, so a decision was made to collect data from all digits not just 2D and 4D. If there was a poor response to data collection in general, measurements from other digits would allow a comparative study to be performed on all digit ratios. Data collectors were provided with detailed instructions in the form of a step-by-step procedure and images of the anatomical landmarks (Appendix 2.3). Potential errors arising from misidentification of digits and incorrectly locating the proximal crease was also addressed on this sheet. Data collectors were encouraged to contact the primary investigator (myself) if they were unclear about any aspect of the technique. For health and safety reasons I requested that measurements be obtained only from primates that were sedated in a state registered zoos or primate facility. The most convenient and cost effective method of measuring the digits was to use a ruler. However, as digital callipers provide more precise measurements than rulers (see Burriss *et al.* 2006), institutions were encouraged to use digital callipers if they were available. Measuring instructions were provided for both ruler and calliper use (Appendix 2.3).

As 2D:4D has been shown to vary over growth in humans (Trivers *et al.* 2006), it was requested that digit lengths be taken only from mature individuals; selecting mature individuals reduced the potential development effects on 2D:4D. Maturity was broadly defined as the ability of an individual to reproduce. Mature individuals with injured or malformed digits were excluded from the study.

2.5.2.1: Methodology

Measurement procedure was based upon Manning *et al.* (1998) for calliper measurements and Manning *et al.* (2007a) for ruler measurements.

Instructions:

1. Ensure the finger being measured remain as straight as possible throughout the procedure (Fig. 2.1; Fig. 2.9).
2. Looking at the palm of the hand, locate the mid-point of the crease at the base of the finger. If using callipers, open the jaws wider than the digit to be measured (Fig. 2.10).
3. Place the zero of the ruler exactly on the ruler exactly on this point. If using callipers place the jaw tip of the calliper exactly on this point.
4. While maintaining the finger in extension (straight), gently place the ruler along the length of the middle of the finger; the mid-line. Or use the callipers to measure the same trajectory.
5. Looking directly at the ruler (or the digital display), record the length at the tip of the soft-tissue of the finger. Please do not include the finger nail or compress the soft tissue of the finger tip as this will distort the digit ratio. Record the length to the nearest millimetre.
6. Please repeat the measurements, if possible.

Due to time, staffing levels and safety issues it was not always possible for staff to adhere fully to these instructions and in many cases only data on 2D and 4D digits were provided and in many cases measurements were not repeated.

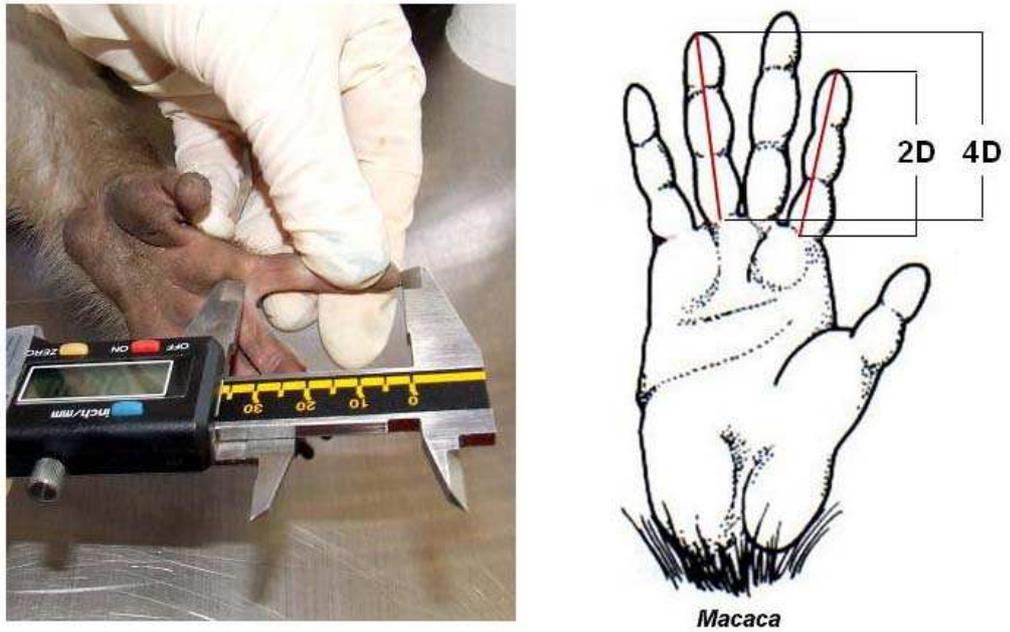


Figure 2.9: Measuring primate digit lengths (also see Appendix 2.3).

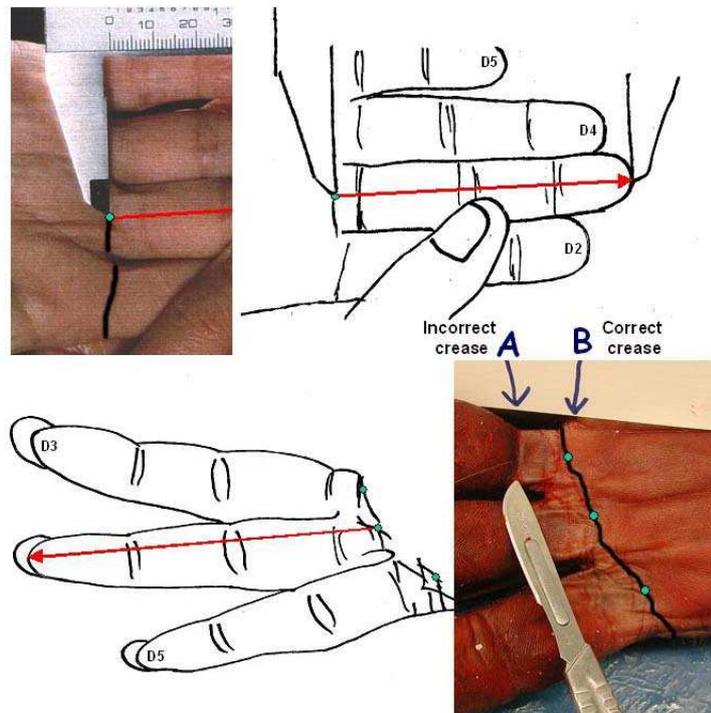


Figure 2.10: Identifying landmarks on the proximal crease at the base of the digits.

2.5.2.2: Landmarks

Identifying the correct landmarks is a perennial problem in 2D:4D studies human studies (Voracek *et al.* 2007a) and misidentification of landmarks leads to error (Manning *et al.* 2007a). As primate data was collected by different people from multiple institutions it was important to ensure data collectors had a correct understanding of the measurement procedure. A detailed instruction sheet with images was produced to provide instructions on to how to identify the landmarks (Fig. 2.10, see Appendix 2.3).

In human studies subjects are compliant and are able to hold the hand still and position their fingers in an extended (straight) and adducted (together) position (Fig. 2.10, top left). Despite the short time available for measuring digits in sedated primates it was possible to achieve a standard human-like hand position in all aspects other than digit adduction. As all individuals were measured with their fingers in a splayed position this deviation from the human standard is unlikely to have introduced gross error into the subsequent analyses of primate 2D:4D.

2.5.3: Protocol review

For the study protocol to be approved it first had to be reviewed by the institution's research co-ordinator or their research and ethics committee. In the U.S.A. this body is the Institutional Animal Care and Use Committee (IACUC) and is linked to the National Institutes of Health. The IACUC is a board that ensures all animal research adheres to the guidelines set by state governing bodies and the zoos own safety and ethical protocols. Most zoos permit a data collection period of one year, after which time the study protocol had to be resubmitted for review by the research committee. However, for this study many institutions were willing to collect data over the requested period of two years without the protocol having to be reviewed.

2.5.3.1: Communication and data transfer

A web site (www.digitratio.com) was set up to provide contact and study information. On the web site the aims of the research were clearly stated, background information was provided supported by referenced literature and a copy of the protocol and measurement procedure was available to download. Information was also provided on the study's progress (e.g., poster presentations, publications). There was a page stating my links with the

University of Liverpool and academic qualifications as well as a page addressing ethics and safety issues. In the initial letter appealing for data, institutions' were encouraged to view the web site before they committed to data collection. Informal feedback indicated that the web site had a positive effect on the willingness of institutions to take part in the study.

The web site also provide a portal for uploading data using a drop-down box system in which digit measurements and information such as the date, sex and age, could be inputted quickly. Once data was downloaded by staff it was automatically formatted into a excel spread sheet. Data from institutions could also be e-mailed as an attached excel file, or in a text format or sent in the mail. The web site was checked every couple of days. On receipt of new data a short acknowledgement e-mail was sent to the institution and the contribution each institution made to the database was logged.

The opportunistic nature of the data collection meant that many months could pass without contact with some institutions. Due to the long data collection period (2 years) there was the potential for loss of contact with some institutions. A concerted effort was made to maintain links. For example, if there had been no data forthcoming from an institution for three months, a short 'up-date' e-mail was sent to the link person. This often prompted them to send data they had been storing up ready to down load.

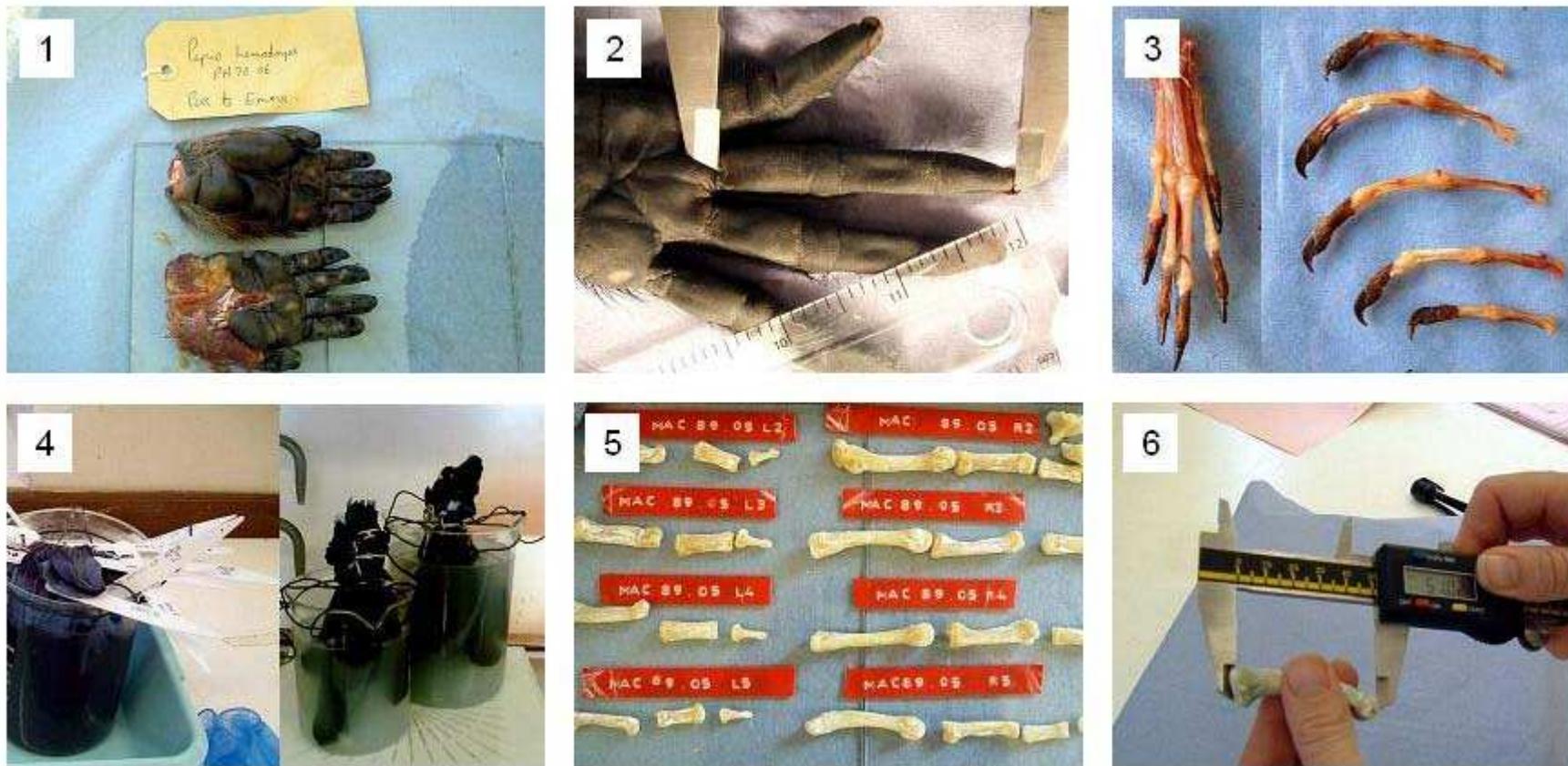
Each year, for the full period of the study, an annual report was sent out each institution. This included an update on data collection (size of database), conference presentations and copies of publications.

Data was also collected on a group of rhesus macaques from the Caribbean Primate Research Centre, Puerto Rico. Methodologies pertaining to the collection of those data are outlined in Chapter 5.

2.5.4: Data from primate cadavers

Data was also collected on a small sample of deceased captive primates (n=17) provided by the National Museums of Scotland; data from deceased individuals were not included in the main dataset but was used in comparative methods outlined in Chapter 6.

Figure 2.11: Dissection of primate hands. 1) Hands of *Papio hamadryas* specimen with label from National Museums of Scotland; 2) 4th digit of *Ateles hybridis* being measured by digital callipers; 3) Dissection of hand of *Leontopithecus chrysomelas*; 4) Dissected digits were confined in mesh bags and clearly labelled (left). Bags were then immersed in a biological solution and boiled until the bones were clear of flesh (right); 5) Cleaned bones were dried and labelled; 6) Disarticulated phalanges were measured with digital callipers.



Hands were dissected (Fig. 2.11) to enabled comparisons to be made between soft-tissue digit length and hard-tissue digit length (summed from the lengths of the three phalanges).

2.5.4.1: Dissection method

1. All specimens were clearly labelled with a reference number and this ensured that each specimen could be matched with its specific data (e.g., sex, species, age group; Fig. 2.11).
2. Soft tissue finger length of the dismembered hand was measured with digital callipers.
3. The majority of the soft-tissue was removed from the hands and the bones disarticulated.
4. Dissected digits were confined in mesh bags and clearly labelled. Bags were then immersed in a biological solution and boiled until the bones were clear of flesh (Fig. 2.11, image 4 & 5). Bagging the samples ensured that all boned could be correctly assigned to the same digit.
5. Cleaned bones were dried and labelled.
6. Disarticulated phalanges were measured with digital callipers using the measuring protocol outlined in Chapter 6.

2.6: Haplorhine dataset: Results of data collection

2.6.1: The sample

A total of 160 institutions were petitioned. These included zoos throughout the world and National Primate Research Centres (NPRC) in the USA. Data collection ran over two years but data continued to be submitted after that time.

2.6.1.1: Response rates

Sixty-three out of 160 institutions agreed to review the protocol (57 zoos and 6 NPRC); a response rate of 39.4% (see Fig. 2.12). All 63 institutions agreed to provide data on an opportunistic basis. Of those, 66.6% (37 zoos and 5 NPRC) provided measurements, while 33.3% did not.

2.6.1.2: Sample size and species diversity

Data was collected from total of 1286 individuals; 463 males and 823 females (Table 2.5). NPRC submitted 44% (566 individuals) of the total data for the study. Regional differences in zoos were observed with more data provided by U.S.A. than European zoos (Fig. 2.12).

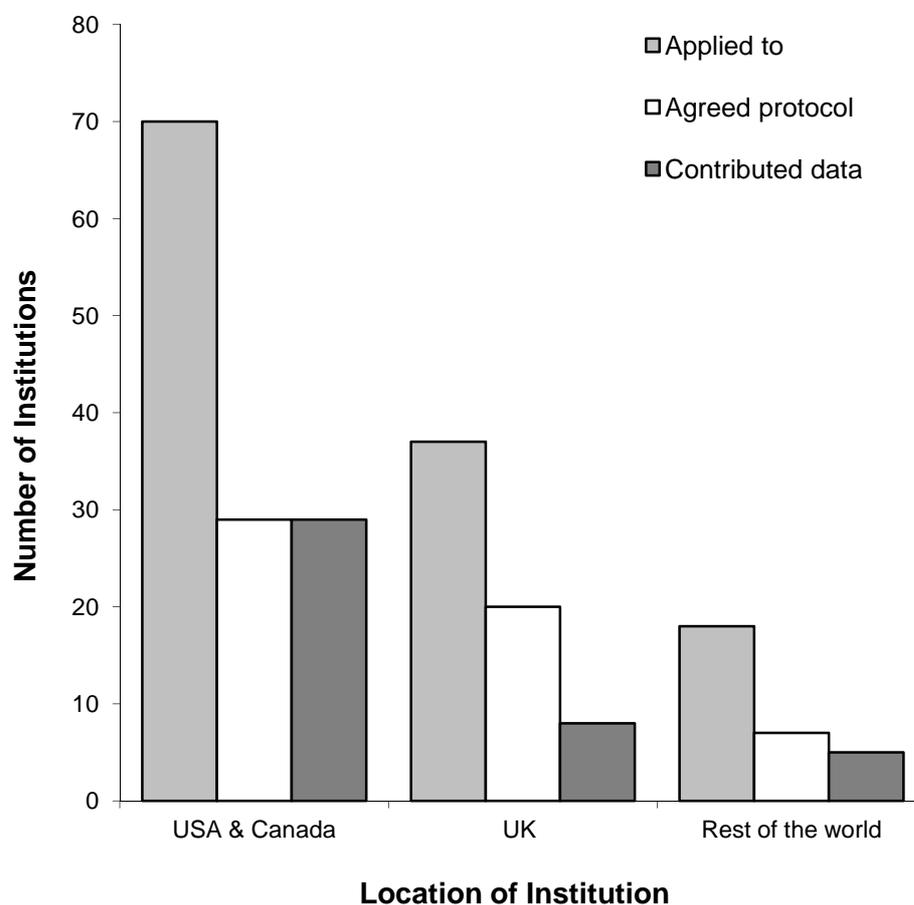


Figure 2.12: Institutions that supplied data (by region). Institutions approached to take part in the study compared to those that agreed and those that supplied data.

OWM (Old World monkeys; Cercopithecoidea) represented the largest super family (50.1%) and NWM (New World monkeys; Ceboidea) the smallest (18.1%; Fig. 2.13). Females

represented 64.3% of the total dataset. Data on captive primate reflected the species preferred by zoos (e.g., apes/hominoids; Melfi 2005). OWM were the predominant species in data provided by NPRC (Fig. 2.13).

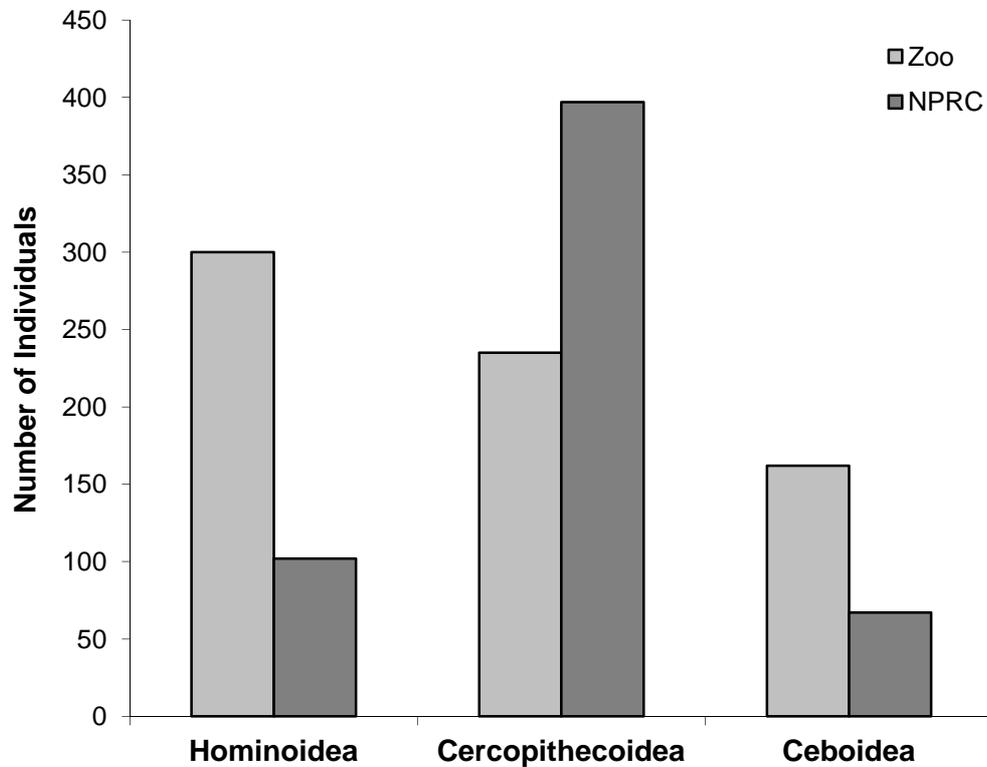


Figure 2.13: Individuals within haplorhine super families supplied by institutions (zoo or National Primate Research Centres; NPRC).

2.6.2: Precision of digit measurements

Data was collected on 74 species; sample sizes were highly variable (Table 2.5). In 16.2% of the species sampled only one individual was measured. Looking within each super family at the species with the largest samples size, *Pan troglodytes* represented 62.7% the of total data for apes, *Macaca mullata* represents 57.6% of the total data for OWM and *Callithrix jacchus* represents 30.1% of the total data for NWM. Out of the total of 1286 individuals, 97.9% had the 2D and 4D of their right hands measured, while 94.6% had the digits measured from the left hand. For the whole sample 59.6% of digits were measured with a ruler and 40.4% with callipers. Repeated measurements were only available for 667 right hands and 660 left hands (Appendix 2.5).

Table 2.5: Haplorhine dataset.

Genus	Species	Male	Females	Genus	Species	Male	Females	Genus	Species	Male	Females
Hominoidea				Cercopithecoidea				Cercopithecoidea continued			
<i>Hylobates</i>	<i>agilis</i>	0	1	<i>Allenopithecus</i>	<i>nigroviridis</i>	4	3	<i>Macaca</i>	<i>arctoides</i>	1	0
<i>Hylobates</i>	<i>hoolock</i>	0	1	<i>Cercocebus</i>	<i>albegina</i>	3	2	<i>Macaca</i>	<i>fascicularis</i>	6	9
<i>Hylobates</i>	<i>klossii</i>	2	0	<i>Cercocebus</i>	<i>galeritus</i>	1	1	<i>Macaca</i>	<i>fuscata</i>	8	9
<i>Hylobates</i>	<i>lar</i>	4	2	<i>Cercopithecus</i>	<i>ascanius</i>	0	1	<i>Macaca</i>	<i>mulatta</i>	56	310
<i>Hylobates</i>	<i>moloch</i>	1	1	<i>Cercopithecus</i>	<i>campbelli</i>	0	1	<i>Macaca</i>	<i>nigra</i>	1	2
<i>Hylobates</i>	<i>muelleri</i>	0	2	<i>Cercopithecus</i>	<i>diana</i>	6	2	<i>Macaca</i>	<i>silenus</i>	0	1
<i>Hylobates</i>	<i>pileatus</i>	1	1	<i>Cercopithecus</i>	<i>erythrotis</i>	3	0	<i>Macaca</i>	<i>sylvanus</i>	1	1
<i>Nomascus</i>	<i>concolor</i>	0	3	<i>Cercopithecus</i>	<i>hamlyni</i>	2	1	<i>Mandrillus</i>	<i>leucophaeus</i>	4	4
<i>Nomascus</i>	<i>leucogenys</i>	3	3	<i>Cercopithecus</i>	<i>lhoesti</i>	1	3	<i>Mandrillus</i>	<i>sphinx</i>	11	17
<i>Symphalangus</i>	<i>syndactylus</i>	7	7	<i>Cercopithecus</i>	<i>mona</i>	2	2	<i>Papio</i>	<i>anubis</i>	0	1
<i>Gorilla</i>	<i>gorilla</i>	21	39	<i>Cercopithecus</i>	<i>neglectus</i>	10	4	<i>Papio</i>	<i>hamadryas</i>	11	15
<i>Pan</i>	<i>paniscus</i>	13	12	<i>Cercopithecus</i>	<i>petaurista</i>	0	4	<i>Papio</i>	<i>papio</i>	11	21
<i>Pan</i>	<i>troglodytes</i>	104	148	<i>Cercopithecus</i>	<i>wolffi</i>	2	0	<i>Colobus</i>	<i>guereza</i>	6	18
<i>Pongo</i>	<i>pygmaeus</i>	8	18	<i>Chlorocebus</i>	<i>aethiops</i>	12	10	<i>Presbytis</i>	<i>comata</i>	0	1

Table 2.5: Haplorhine dataset continued.

Genus	Species	Male	Females	Genus	Species	Male	Females	Genus	Species	Male	Females
Cercopithecoidea continued				Ceboidea				Ceboidea continued			
<i>Presbytis</i>	<i>melalophos</i>	1	5	<i>Callimico</i>	<i>goeldii</i>	4	0	<i>Cebus</i>	<i>apella</i>	9	11
<i>Pygathrix</i>	<i>nemaeus</i>	1	0	<i>Callithrix</i>	<i>argentata</i>	1	0	<i>Saimiri</i>	<i>sciureus</i>	3	9
<i>Trachypithecus</i>	<i>auratus</i>	0	2	<i>Callithrix</i>	<i>geoffroyi</i>	4	8	<i>Callicebus</i>	<i>donacophilus</i>	2	3
<i>Trachypithecus</i>	<i>cristatus</i>	1	0	<i>Callithrix</i>	<i>jacchus</i>	36	33	<i>Callicebus</i>	<i>moloch</i>	21	12
<i>Trachypithecus</i>	<i>francoisi</i>	6	8	<i>Callithrix</i>	<i>pygmaea</i>	2	0	<i>Callicebus</i>	<i>torquatus</i>	2	0
<i>Trachypithecus</i>	<i>obscurus</i>	2	5	<i>Leontopithecus</i>	<i>chrysomelas</i>	3	4	<i>Pithecia</i>	<i>pithecia</i>	5	1
				<i>Leontopithecus</i>	<i>rosalia</i>	5	5	<i>Alouatta</i>	<i>caraya</i>	10	12
				<i>Saguinus</i>	<i>bicolor</i>	1	0	<i>Ateles</i>	<i>belzebuth</i>	0	1
				<i>Saguinus</i>	<i>geoffroyi</i>	4	1	<i>Ateles</i>	<i>fusciceps</i>	2	0
				<i>Saguinus</i>	<i>imperator</i>	3	5	<i>Ateles</i>	<i>geoffroyi</i>	1	4
				<i>Saguinus</i>	<i>midas</i>	6	4	<i>Ateles</i>	<i>hybridus</i>	1	2
				<i>Saguinus</i>	<i>oedipus</i>	1	1	<i>Ateles</i>	<i>paniscus</i>	0	3
				<i>Cebus</i>	<i>albifrons</i>	0	1	<i>Lagothrix</i>	<i>lagotricha</i>	0	2
										463	823
								Total		1286	

2.6.2.1: Case study: Precision of measurements in humans studies

Internal consistency of the haplorhine dataset was estimated using intra-class correlation coefficients (ICC; McGraw & Wong 1996). It has been proposed that indirect measurements taken from images are more precise than direct measurements taken from the skin surface (Voracek & Offenmüller 2007; Allaway *et al.* 2009). To observe how ICC in human 2D:4D studies vary according to different measurement approaches (direct versus indirect), ICC values were taken from the published studies (Fig. 2.14; Appendix 2.4 for references).

Data was only sufficient to compare ICCs for mean values for 2D and 4D of each hand and mean 2D:4D collected from the literature (i.e., ICCs could not be established for repeats on individual finger lengths). Results show that in humans direct methods had statistically higher measurement precision than indirect methods for each hand ratio (R2D:4D, $t=-5.12$, $p<0.01$; L2D:4D, $t=-4.22$, $p<0.01$), but methods did not differ in ICCs for mean 2D:4D (M2D:4D, $t=-1.21$, $p=0.26$; Fig. 2.14). A similar pattern was shown by Voracek *et al.* (2007a). Thus direct measures (e.g., taken by ruler or callipers) present a more reliable method of measuring digit lengths than indirect methods; although both show relatively high repeatability over all (see Allaway *et al.* 2009).

2.6.2.2: Precision of measurements in the haplorhine dataset

ICC values were calculated for the haplorhine dataset for measurements taken with a ruler and with callipers. Values for haplorhines accord with those shown in humans (Voracek *et al.* 2007a); ICC values were higher for repeated measurements of fingers but lower for the repeats of ratios for each hand and ameliorated when 2D:4D from the left and right hands was converted to a mean (see Table 2.6 haplorhines; Fig. 2.14 for humans). ICC values for the haplorhine dataset were within range of ICC values for direct methods quoted in human studies (Table 2.6)

There were no significant differences within super families between ICC values for 2D:4D ratios calculated using a ruler and those calculated using callipers (right hand, $t=0.78$, $p=0.54$, $df=2$; left hand, $t=1.06$, $p=0.40$, $df=2$) or between hands using the same method (callipers, $t=-0.26$, $p=0.82$, $df=2$; ruler, $t=-0.447$, $p=0.70$, $df=2$; Table 2.7). This supports the conclusion of Voracek *et al.* (2007a) that rulers and callipers yield similar results.

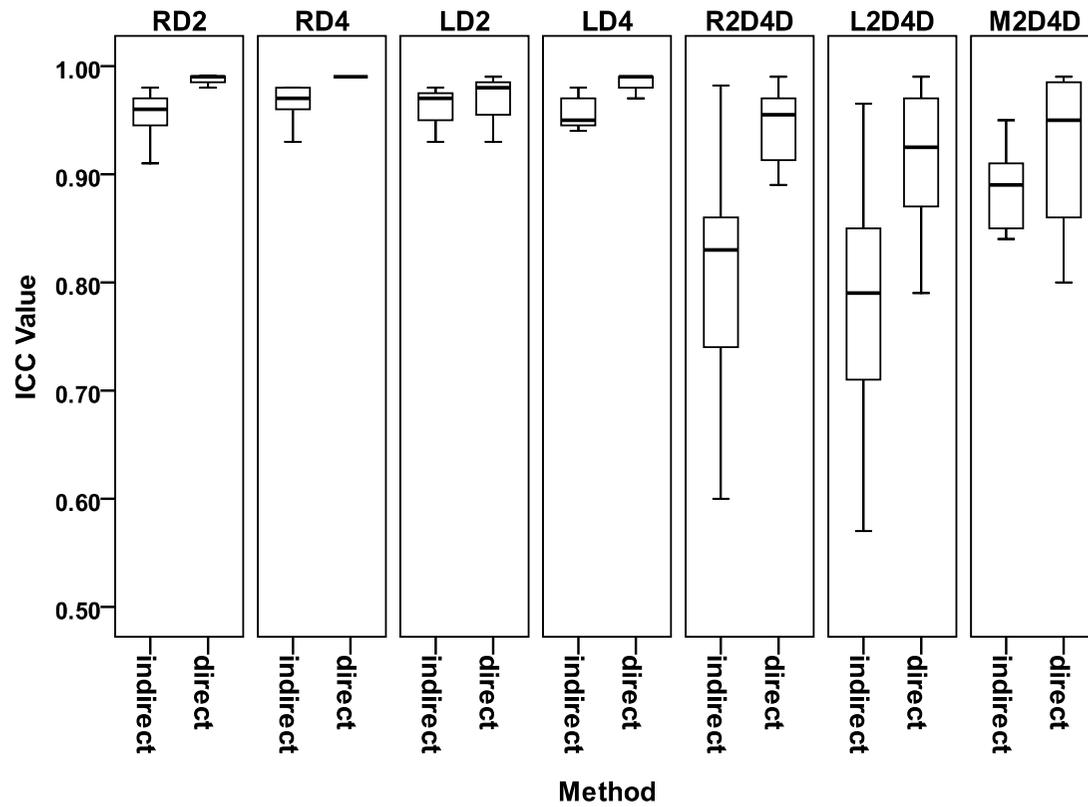


Figure 2.14: Intra-class correlation coefficients between indirect and direct methods in human 2D:4D studies (see Appendix 2.4 for reference sources). RD2= right second digit; RD4 = right fourth digit; LD2= left second digit; LD4= left fourth digit; R2D:4D= right 2D:4D; L= left 2D:4D; M2D:4D= mean 2D:4D.

Repeated Measures	Method	ICC	F	p	df
Left D2	Callipers	0.999	1870.47	<0.0001	192
	Ruler	0.999	1155.04	<0.0001	466
Left D4	Callipers	0.999	1560.51	<0.0001	193
	Ruler	0.999	1391.46	<0.0001	466
Right D2	Callipers	0.999	1912.06	<0.0001	197
	Ruler	0.999	1280.00	<0.0001	469
Right D4	Callipers	0.999	2763.99	<0.0001	196
	Ruler	0.999	135.44	<0.0001	468
Left 2D:4D	Callipers	0.953	21.79	<0.0001	192
	Ruler	0.935	15.48	<0.0001	465
Right 2D:4D	Callipers	0.965	28.67	<0.0001	196
	Ruler	0.926	13.56	<0.0001	467
Mean 2D:4D	Callipers	0.974	38.82	<0.0001	202
	Ruler	0.970	32.96	<0.0001	473

Table 2.6: Intra-class correlation coefficients for repeated measures in haplorhines. F=variance result; p=probability; df=degrees of freedom).

As primates are highly variable in their body size (apes are larger than OWM which in-turn are larger than NWM; Smith & Jungers, 1998; Lindenfors & Tullberg, 1998). Methods were also compared between super families to detect if there was any differences in repeatability in according with hand size. Significant differences were shown in ICC values between super families (controlling for method; $F_{2,8}=16.34$, $p<0.01$) with ape values being higher than NWM values and both apes and NWM values being higher than OWM ICC values (see Table 2.7). However, ICC values for all super families still remain within acceptable limits for human studies (Voracek *et al.* 2007a) which indicate that the results of subsequent analyses are not the product of measurement error but reflect real differences in 2D:4D between species.

Repeated Measures	Method	Group	ICC	F	p	df
Left 2D:4D	Callipers	Apes	0.975	40.62	<0.0001	74
		OWM	0.868	7.79	<0.0001	107
		NWM	0.901	9.96	0.001	9
	Ruler	Apes	0.970	33.24	<0.0001	113
		OWM	0.744	3.94	<0.0001	248
		NWM	0.901	10.00	<0.0001	102
Right 2D:4D	Callipers	Apes	0.991	113.00	<0.0001	75
		OWM	0.877	8.08	<0.0001	110
		NWM	0.884	8.12	0.002	9
	Ruler	Apes	0.964	28.04	<0.0001	112
		OWM	0.736	3.78	<0.0001	249
		NWM	0.932	14.66	<0.0001	104
Mean 2D:4D	Callipers	Apes	0.987	77.48	<0.0001	80
		OWM	0.909	11.11	<0.0001	111
		NWM	0.934	14.04	<0.0001	9
	Ruler	Apes	0.994	170.28	<0.0001	114
		OWM	0.793	4.84	<0.0001	251
		NWM	0.950	19.99	<0.0001	106

Table 2.7: Intra-class correlation coefficients for repeated measures between super-families.

2.6.2.3: Precision of measurements in the dissected data

Data derived from a small and varied set of dissected primate hands allowed comparisons between soft- and hard- tissues (addressed in context in Chapter 6). Soft-tissue measurements were taken from 17 individuals from 15 species and hard-tissue data was taken from 11 of those individuals (dissected and prepared at the University of Liverpool (Fig. 2.11; Table 2.8). Pairs of hands from 6 individuals could not be dissected. ICC values for soft-tissue and hard-tissue length measurements are presented in Table 2.9. ICC values were higher for repeated measurements on hard-tissues than on soft-tissue measurements.

Table 2.8: Deceased sample (n=17). Dissected hands (n=11 pairs). * Both soft- and hard-tissue data; M = mature; I - immature

Genus	Species	Sex	Age	Genus	Species	Sex	Age
* <i>Ateles</i>	<i>hybridis</i>	F	M	* <i>Macaca</i>	<i>mulatta</i>	M	M
* <i>Ateles</i>	<i>fusciceps</i>	M	M	* <i>Mandrillus</i>	<i>sphinx</i>	M	I
<i>Callithrix</i>	<i>argonata</i>	M	M	* <i>Normanscus</i>	<i>leucogenys</i>	M	M
* <i>Cebus</i>	<i>apella</i>	M	M	* <i>Pan</i>	<i>trogodytes</i>	M	M
* <i>Hylobates</i>	<i>lar</i>	F	M	* <i>Papio</i>	<i>hamadryas</i>	F	M
<i>Hylobates</i>	<i>lar</i>	M	I	<i>Papio</i>	<i>hamadryas</i>	F	M
<i>Lemur</i>	<i>catta</i>	M	M	* <i>Presbytis</i>	<i>melalophos</i>	F	M
<i>Leontopithecus</i>	<i>chrysopygus</i>	F	M	<i>Siamiri</i>	<i>sciureus</i>	F	M
* <i>Leontopithecus</i>	<i>chrysomelas</i>	F	M				

Table 2.9: Intra-observer correlation coefficients (ICC) valued for repeated digit measurements in the decreased sample. ICCs for means ratios are calculated by comparing the means of left and right hands for the first and second sets of measurements. *=soft tissue measurements.

Dissected hands Length measurement	ICC Values						
	Tissue	Left hand	df	Right hand	df	Mean ratio	df
Digit 2*	Soft	1.00	16	0.95	16		
Digit 4*	Soft	1.00	16	1.00	16		
Mean 2D:4D*	Soft	0.92	16	0.63	16	0.84	14
Proximal phalange (PP) 2	Bone	1.00	10	1.00	10		
Proximal phalange (PP) 4	Bone	1.00	10	1.00	9		
Mean 2PP:4PP	Bone	1.00	10	1.00	9	1	10
Intermediate phalange (IP) 2	Bone	1.00	10	1.00	9		
Intermediate phalange (IP) 4	Bone	0.97	10	1.00	9		
Mean 2IP:4IP	Bone	0.88	10	0.91	9	0.934	9
Distal phalange (DP) 2	Bone	1.00	9	0.99	9		
Distal phalange (DP) 4	Bone	1.00	8	1.00	8		
Mean 2DP:4DP	Bone	0.99	8	0.98	8	0.994	8

2.6.2.4: Comparisons of measurements across institutions

To check for systematic bias in measurements across institutions 2D:4D ratios of species measured at different zoos and facilities were compared (intra-specific comparisons). Only one species, the saki monkey (*Pithecia pithecia*), out of 74 exhibited significant differences in measurements between institutions (n=2 institutions; $F_{1,2}=51.21$, $p<0.01$). When measurements for each species were compared across institutions only 3.37% of pair wise comparison showed significantly different ratio values across institutions. This indicates that the occurrence of systematic bias in the measurement of 2D:4D across institutions is unlikely to have impacted the measurements.

2.7: Controlling for phylogenetic effects in cross-species analyses

The bushy pattern of evolution (speciation; see Wood 2010) means that related taxa are not evolutionary free-agents but form part of a group with connected histories and sets of traits (Felsenstein 1985). To consider species as independent is to assume that adaptations shared between closely related species evolved separately, which is not the case (Cheverud *et al.* 1985; Fig. 2.15A). When analyzing cross-species data controls have to be incorporated to account for evolutionary non-independence of traits across taxa (also see Grafen 1989; Harvey & Pagel 1991).

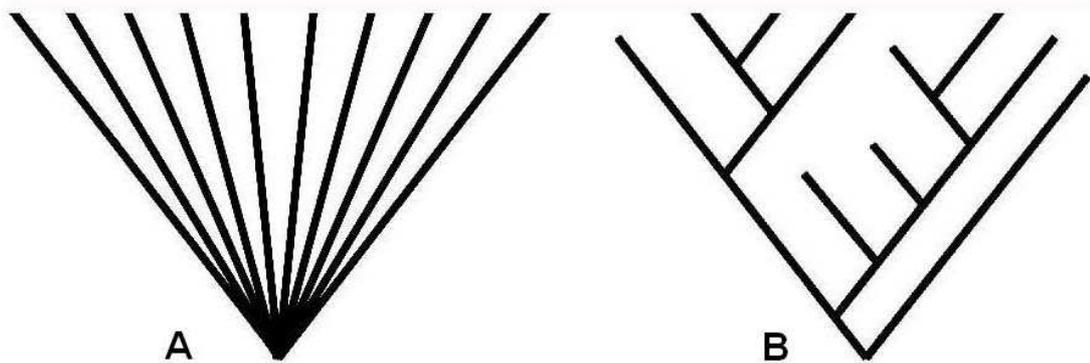


Figure 2.15: Phylogenetic trees. A) The free-phylogeny (or star-phylogeny) assumes that species evolved independently of each other, while the branching phylogeny (B) depicts a connected pattern known to characterise evolutionary change.

After Garland and Carter 1994.

In brief, modern phylogenetic statistical programs create matrices based upon the branching patterns⁹ of phylogenetic information and use the data (branch pattern, branch distance and a Brownian-motion model; Felsenstein 1985; Grafen 1989; Harvey & Pagel 1991) to control for phylogenetic autocorrelation; inertia in traits between species formed as a consequence of evolutionary relatedness (Cheverud *et al.* 1985; Pagel 1997). Phylogenetic autocorrelation is calculated by estimating the ‘specific component’ which is a measure of change after cladogenesis (adaptation) and the ‘phylogenetic component’ which is a measure of the effects of common ancestry which provides a constraint on evolutionary change (Cheverud *et al.* 1985; Pagel 1997). These variables can be summed up in one value known as Pagel’s lambda (λ). The closer λ is to 1 the stronger the phylogenetic effect on the relationship between the variables being modelled. The Moran’s I calculation (Moran 1950) can be used to test how strong the phylogenetic signal is within individual sets of data (e.g., 2D:4D, species weight). For example applying Moran’s I to values for body weight across species the results produced will provide an expected value based on the assumption that species are unrelated (independent) and predicted value based upon the branching pattern of species relatedness in the matrix constructed from the phylogenetic tree (see footnote). If the predicted value differs significantly from the expected value then the dataset is assumed to be influenced by phylogenetic relatedness (Felsenstein 1985; Grafen 1989; Harvey & Pagel 1991).

There are now numerous methods based upon these principles (reviewed in Nunn & Barton 2001) but many are unable to incorporate discrete values (integers) with continuous variables. This is a disadvantage in comparative analyses as studies often involve combining both types of data. Here I use a phylogenetic generalised least-squares (PGLS) method (Grafen 1989) as it is able to incorporate both discrete and continuous variables and thus facilitates investigations between 2D:4D (continuous) and classifications of traits linked to ecological niche (often reduced to discrete variables).

2.7.1: Materials and methods

To demonstrate the phylogenetic method 2D:4D ratios of non-primate taxa were taken from the literature (Table 2.10; see Appendix 2.4 for reference sources) and compared with the haplorhine dataset (see Appendix 2.5). Human data was taken from the literature (Appendix 2.4) and used as a comparison within haplorhines. In this analysis I use data from all 74 species in the haplorhine dataset to obtain broad-scale patterns across vertebrates. 2D:4D

⁹ Phylogenetic trees used to build the matrices throughout this thesis are based on Purvis 1995 and Opazo *et al.* 2006.

data for some species did not include measurements from both sexes and data from both hands and, for some species, data was only available from one individual (see Appendix 2.5). In cases in which only one hand was measured I assumed that the non-present hand would have approximately the 2D:4D ratio in the hand for which data was available. This can be justified as correlations of left and right hands (at the species level) were high in those species that had a full complement of measurements (left on right 2D:4D, females: $R^2=0.805$; males: $R^2=0.863$; mean: $R^2=0.91$).

It is acknowledged when a species is represented by only one individual is unlikely to be representative a species (Schillaci & Schillaci 2009). In subsequent chapters analyses are based upon species data taken from samples that have 2D:4D ratios of both males and females and have data for both hands (i.e., those species in which data are lacking for one hand or from one sex have been excluded from the dataset). Consequently the analyses throughout the rest of this thesis are based upon a dataset of 44 species (Appendix 2.5).

Order	Species	Common Name	Male 2D:4D	Female 2D:4D	Mean 2D:4D
Rodent	<i>Mus musculus</i>	Lab mouse	0.985	1.021	1.003
Rodent	<i>Apodemus sylvaticus</i>	Wood mouse	0.957	0.963	0.960
Rodent	<i>Rattus rattus</i>	Black Rat	0.891	0.913	0.902
Rodent	<i>Microtus agrestis</i>	Field Vole	1.017	1.015	1.016
Bird	<i>Passer domesticus</i>	House sparrow	1.010	0.993	1.002
Bird	<i>Hirundo rustica</i>	Barn swallow	1.075	1.077	1.076
Bird	<i>Trachycineta bicolor</i>	Tree swallow	0.955	0.932	0.943
Bird	<i>Melopsittacus undulates</i>	Budgerigar	0.733	0.698	0.715
Bird	<i>Gallus domesticus</i>	Chicken	0.875	0.864	0.869
Bird	<i>Taeniopygia guttata</i>	Zebra finch	0.890	0.840	0.865
Lizard	<i>Anolis carolinensis</i>	Green lizard	0.484	0.437	0.461
Lizard	<i>Podarcis muralis</i>	Wall lizard	0.637	0.602	0.619
Lizard	<i>Mabuya planifrons</i>	Skink	0.731	0.723	0.727

Table 2.10: Non-primate 2D:4D data. For measures of variance in the studies see references Appendix 2.4.

2.7.2: Statistical methods

Analysing data using both non-phylogenetic (non-phy) and phylogenetic (PGLS) methods enables the magnitude of phylogenetic effects to be judged. A general linear model (GLM) was used to investigate non-phylogenetic relationships between species mean 2D:4D. Paired t-tests were used to examine relationships between male and female 2D:4D ratios within species. The Kolmogorov-Smirnov Test was used to look for heteroskedasticity (skew) in the data. All tests were non-significant ($p > 0.05$).

Phylogenetic trees were constructed within McClade software (Maddison & Maddison 2001) and were based on published trees for primates (Purvis 1995; Opazo *et al.* 2006; Appendix 2.6) and vertebrates (Steiper & Young 2006; Vidal & Hedges 2005; Huchon *et al.* 2007; Hackett *et al.* 2008; see Fig. 2.16). In McClade the trees were converted into a matrix using Phylogeny Inference Package (PHYLIP; Maddison & Maddison 2001). PGLS analysis was performed using the 'R' statistical software (Ihaka & Gentleman 1996) and the Analysis of Phylogenetics and Evolution (APE) (Paradis *et al.* 2004) with code provided by R.P. Duncan. 'R' statistical software uses the information from the PHYLIP matrix to control for species relatedness (phylogenetic autocorrelation) when performing correlations on species data (see above).

The PGLS output indicates strength of relationships between variables with phylogenetic controls in the form of conventional F- and p-values, and also provides a measure for strength of phylogenetic auto correlation (Moran's I) within sets of variables and a measure of strength of phylogenetic auto correlation within the model (Pagel's λ).

2.7.3: Results of comparisons across Vertebrate Orders

Results show that digit ratios significantly differed across orders (non-phy; $F_{3,55}=30.32$, $p < 0.01$; PGLS; $F_{3,52}=5.76$, $p < 0.01$, $\lambda=0.86$). Moran's I for 2D:4D; observed = 0.25, predicted = -0.02 , ± 0.04 , $p=0.009$, shows that phylogenetic effects on 2D:4D are strong across primates. The fact that Pagel's λ was low (zero) in non-primate comparison could be an effect of poor sample sizes in non-primate species compared with the primate sample. Paired analyses (between orders) indicated that only the 2D:4D ratios of scaly lizards remained significantly different (lower) after phylogeny was controlled for (PGLS) in the analysis (Table 2.11).

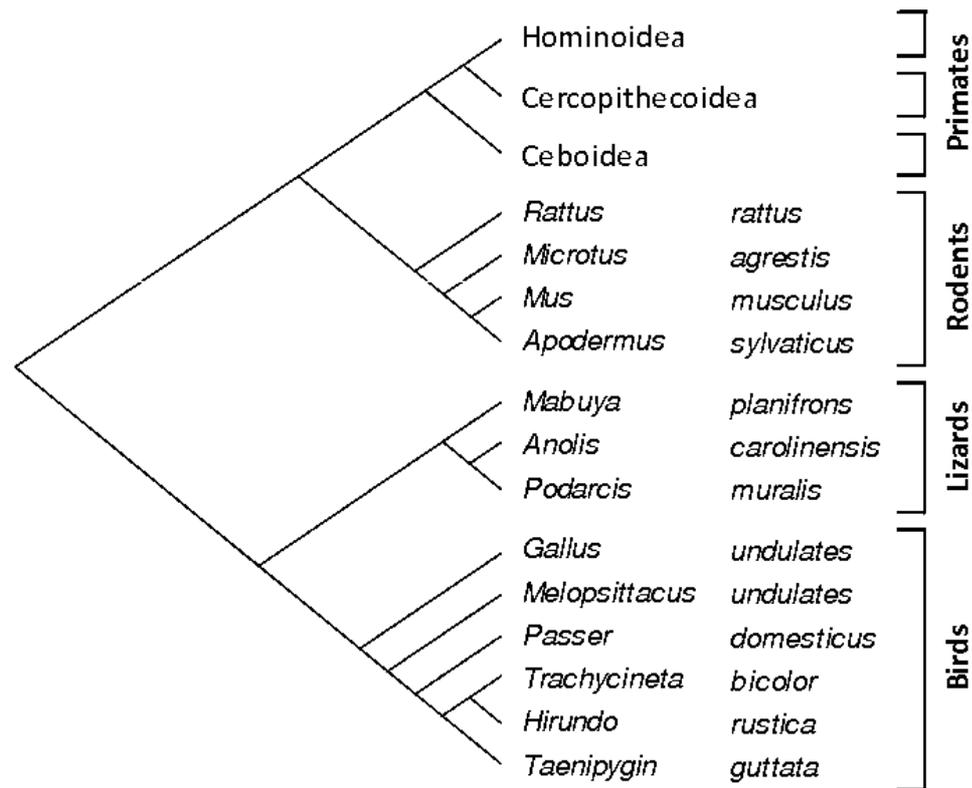


Figure 2.16: Phylogenetic tree for vertebrate taxa. Primates: Steiper & Young 2006; lizards (Order Squamata): Vidal & Hedges 2005; rodents: Huchon *et al.* 2007; birds (Class Aves): Hackett *et al.* 2008¹⁰. Primate data taken from this study (see Appendix 2.5). Non-primate data was sample taken from the literature (see Table 2.10 and Appendix 2.4).

Comparisons	Non-phylogenetic Analyses			Phylogenetic (PGLS) Analysis			
	F	p	df	F	p	df	λ
Primates - Rodents	3.79	0.06	1,44	0.20	0.07	1,45	0.997
Primates - Lizards	31.15	<0.0001	1,44	4.01	0.05	1,44	0.916
Primates - Birds	0.39	0.54	1,47	0.01	0.96	1,47	0.980
Rodents - Lizards	24.32	0.01	1,4	18.88	0.01	1,5	0
Rodents - Birds	1.15	0.32	1,7	0.61	0.46	1,8	0
Lizards - Birds	11.71	0.01	1,7	9.20	0.02	1,7	0

Table 2.11: Non-phylogenetic and phylogenetic analyses between Vertebrate Orders. Significant values ($p \leq 0.05$) in bold type.

¹⁰ Shedlock & Edwards (2009) show that birds are closely related to turtles and crocodiles. Many taxonomists consider it valid to place birds within the Class Reptilia; many ornithologists do not agree.

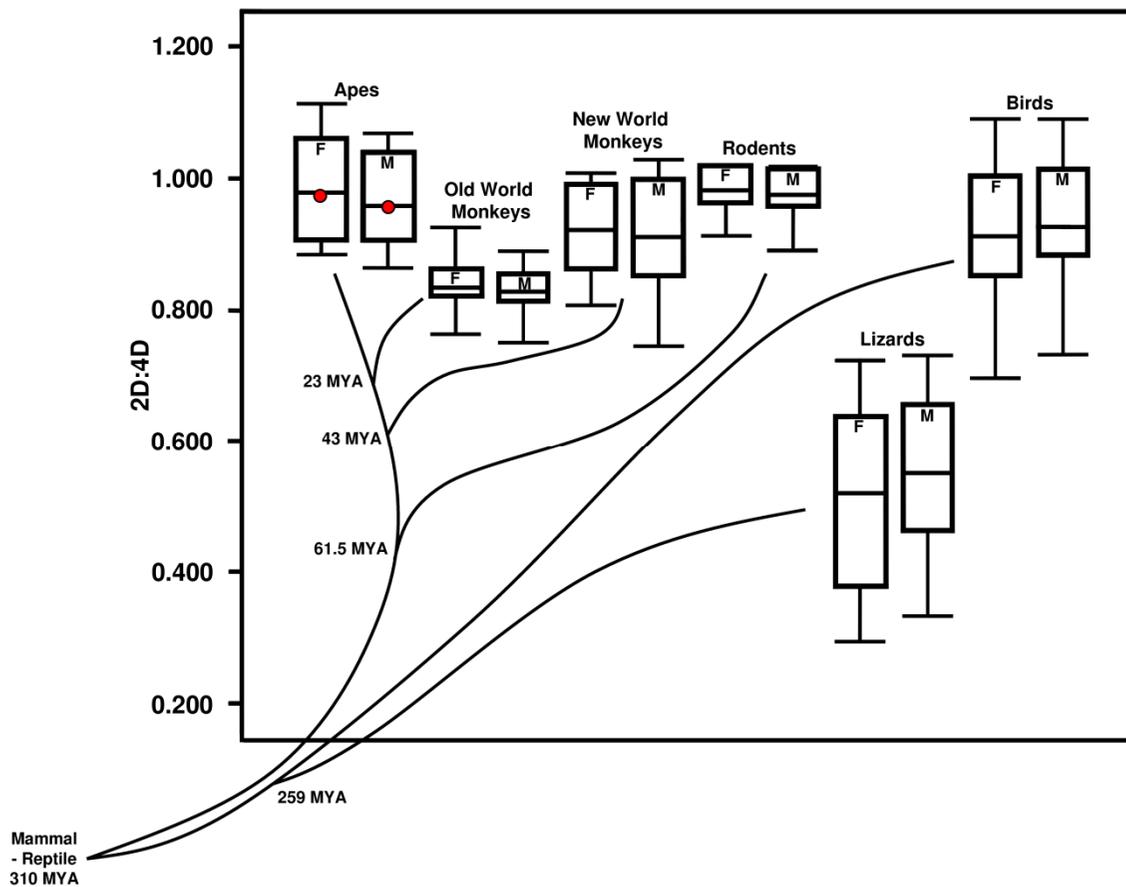


Figure 2.17: Box-plot phylogram of 2D:4D across vertebrate orders (primates analysed by order). F=females; M=males; dots represent mean 2D:4D for humans for comparison. Bars within boxes are redrawn to show mean values. Branch divergence dates were taken from Steiper & Young 2006; Benton & Donoghue 2007. For data references sources for non-primates and humans see Appendix 2.4.

These relationships are more clearly shown in a box-plot phylogram that incorporates time-depth and branching sequences (Fig. 2.17). Human mean 2D:4D ratios (represented by dots) are used as a comparison and sit snugly within the Hominoidea (apes; Fig. 2.17). Patterns of sexual dimorphism in 2D:4D are shown to differ at higher taxonomic levels; males have lower mean ratios than females in mammals (primates and rodents), while the opposite appears to be the case for birds and lizards, grouped here within the Class Reptilia (Shedlock & Edwards 2009; Fig. 2.17).

Paired t-tests were used to examine differences between male and female means (within species; Table 2.12). At the species level significant differences between male and female 2D:4D ratios were shown for lizards and birds; there were no significant sex differences within rodents or primates. The 2D:4D ratios for humans were analysed separately from non-human primates and show significant sex differences (Table 2.12). Care was taken to only compare the mean ratios of males and females within studies.

	Male		Female		t	p	df
	Mean	sd	Mean	sd			
Primates	0.886	0.084	0.892	0.082	0.905	0.370	1,45
Rodents	0.973	0.052	0.985	0.047	1.670	0.170	1,4
Lizards	0.564	0.162	0.527	0.182	-2.769	0.050	1,4
Birds	0.934	0.113	0.915	0.127	-2.610	0.035	1,7
Humans	0.964	0.020	0.977	0.020	8.662	<0.001	92

Table 2.12: Differences between male and female 2D:4D ratios within Vertebrate Orders. Humans added for comparison. All comparisons based on mean taken from published studies. See Appendix 2.4 for reference sources. Significant values ($p \leq 0.05$) in bold type.

2.8: Discussion

2.8.1: Methodologies

The main body of 2D:4D research has been performed on humans (see Voracek & Loibl, 2009). The majority of these studies use the same anatomical landmarks with the hand held in a standardised position (Fig. 2.1; Fig. 2.2). Methodologies can be divided into two main categories; direct and indirect. In humans indirect methods have been shown to reduce 2D:4D (Manning *et al.* 2005; Allaway *et al.* 2009). It has been recommended that data

derived using indirect and direct approaches should not be mixed within the same analyses (unless the differences between methods are being compared). Due to the distortion effects imposed on digit length by indirect methods it is recommended that and direct measurements be used when possible (Manning & Hill 2009).

It has been shown that indirect measurements are more repeatable than direct methods (Voracek & Offenmüller 2007; Allaway *et al.* 2009). However, using published ICC values from studies I have shown that both direct and indirect methods yield comparatively high repeatability values (Fig. 2.14). It is noteworthy that a large proportion of the studies using direct data had John Manning as primary author or co-author (Appendix 2.4). Assuming that he collected the data in studies in which he was primary author, we might expect a highly skilled measurer to obtain digit measurements with high levels of precision and this effect contributed in the high ICC values for soft-tissue data over all. Nevertheless repeatability estimates have been shown to be high for both direct and indirect methodologies in human studies (Voracek *et al.* 2007a; Allaway *et al.* 2009) and direct measurements of haplorhine primates yield comparably ICC values (Table 2.6; Table 2.7).

In comparison to human studies, studies of 2D:4D in non-human animals are poorly represented (~8% of the total number of publications). The research themes in non-human studies have largely focused on investigating sexual dimorphism in 2D:4D rather than associations with behaviour or anatomical measures (but see Bailey *et al.* 2005; Talarovičová *et al.* 2009). The results of these studies, however, have been inconsistent. To date it has not been possible to make meaningful comparisons between non-human studies because species within taxonomic groups are very limited and those that are published often employ different methodologies. Non-human studies also adopt both direct and indirect approaches but methods have had to be adapted to accommodate differences in hand/foot morphologies. Measures of internal consistency, when published, suggest that many of these approaches are experimental and need refining (see Table 2.1-2.4). Variation in the results of the cross-species analysis is likely to be associated with factors other than PAE, which may not necessarily impact on within-species studies but may yield much greater effects in cross-species analyses (e.g., differences in hand/foot morphology). These issues will be addressed in the following sections.

2.8.2: Morphology

Within species some sets of limbs have been favoured for study over others. For example in birds digit lengths are calculated from pedal (foot) digits because of the development of

wings in the fore-limbs. In scaly lizards and rodents measurements have been taken from both the fore- and hind-limbs, but in some rodent studies the hind-paw has been favoured for study because the digits of the fore-paw are highly curved and very small and thus difficult to measure (Brown *et al.* 2002a; Manning *et al.* 2003c). Hand and foot morphology can also greatly differ within taxonomic groups (Rubolini *et al.* 2006; Chang *et al.* 2006; Lombardo & Thorpe 2008; Chang 2008). Within rodents the soft-tissue components of the hands also appear to vary between species (e.g., Manno 2008; Lilley *et al.* 2009) and within individuals during development (Hurd *et al.* 2008). Mostly researchers have tried adapt the same anatomical landmarks used in human studies, but others have opted to use more novel landmarks such as measurements taken from the dorsal (back) surface of the hand (or foot; Rubolini *et al.* 2006). Huge variation in limb-morphology across vertebrates makes it unlikely that one standardised approach can be devised for used in all animals. As a result comparisons of digit ratios at higher taxonomic levels can only provide general patterns (Fig. 2.17).

2.8.3: Patterns of development and sexual differentiation

Voracek (2006) has drawn attention to the fact that vertebrates develop cranio-caudally (head to tail) and this may alter the influence of sex hormones on digit ratios from the two sets of limbs. Correlations between hand and foot 2D:4D ratios in humans have been found to be low although the overall pattern is similar (McFadden & Shubel 2002). Comparison between mouse hind-paw ratios (across all digits) and human hand digit ratios show similar patterns (Manning *et al.* 2003c), while differences in 2D:4D ratios between the fore-and hind-paw in scaly lizards are more extreme (Lombardo & Thorpe 2008). It is possible that the digits of different limbs may be differently affected by PAE (Voracek 2006) and could contribute to increased variation within and between species in comparative studies. However, these differences may relate to functional effects on limb morphology.

In egg-laying vertebrates sex hormones derived from the mother are deposited in the yolk sac (e.g., Schwabl 1993; Gil *et al.* 2004) while in placental mammals the maternal-foetal-placental-unit is active throughout gestation and plays an important role in hormone regulation throughout gestation (e.g., Kragie, 2002; Fowden & Forhead 2004; Fowden *et al.* 2008). Mammals therefore have the potential to be exposed to higher variations in sex hormones than oviparous taxa in which yolk sex-hormones levels are finite. Within mammals developmental differences between taxonomic groups may also impact on 2D:4D. For example, the genitals of altricial mammals (e.g., most rodents) are highly underdeveloped at birth and require appropriate maternal responses to stimulate sex

hormones that to the development of the reproductive system (i.e., licking the infant genitals; Baum *et al.* 1996). The maternal behaviour of mothers may affect digit ratios in altricial (underdeveloped) mammals. More studies of animals with differing developmental and life-history patterns are required to investigate potential interactions on digital development and 2D:4D.

Within vertebrates, mammals have different mechanisms of sexual determination compared with reptiles (birds and most lizards; Lombardo *et al.* 2008). In mammals, males are the heterogametic sex while in birds and most lizards females are the heterogametic sex (Adkins-Regan *et al.* 1995; Lance 1997). As the development of the gonads (ovary and testes) is influenced by genes on the sex chromosomes, differences in sexual determination could differentially influence foetal sex hormone production which could impact on *HOX* gene expression (Zákány *et al.* 1997; Daftary & Taylor 2006). Different mechanisms of sexual determination across Vertebrate Classes may underpin Chang's Phylogenetic Constraints Model (Chang *et al.* 2006; Chang 2008) which proposes that 2D:4D ratios in more closely related taxonomic groups should be more similar (but see Lombardo & Thorpe 2008). However, this was discounted by Lombardo and Thorpe (2008) because they did not detect any consistent sex differences across non-human species in their descriptive study of 2D:4D (also see Lombardo *et al.* 2008).

2.8.4: Sample size

It is important to appreciate that the sample sizes available for non-primates animals are small in comparison with human studies and the new non-human primate sample (haplorhine dataset). While it is acknowledged that sample sizes for some individual species within the haplorhine dataset are also small, and could therefore yield erroneous results, in analyses between closely related species these effects should be minimised in tests across vertebrates.

2.8.5: The haplorhine dataset

The response from zoos and primate research centres to an appeal for digit length data was very good. Measurements were collected from over 1200 individuals from 74 species. This can be considered an exceptionally large dataset for a captive primate study. The haplorhine dataset provides the potential for analyses across species within the same suborder in a set of animals that are closely related and developmentally similar to humans. This allows for more realistic comparisons with the main body of 2D:4D research.

Despite differences in modes of locomotion (e.g., Jouffroy *et al.* 1993; Richmond 2007), hand morphology remains broadly similar across the haplorhines (Napier 1980; Ankel-Simons 2000). This means that the anatomical landmarks for digit length measurement used in human studies can also be used for non-human primates. All digits in the haplorhine dataset were measured directly with a ruler or callipers according to a standard set of instruction that were simple to follow. Comparisons of intra-observer error showed no significant difference in ICC valued between measurement methods (ruler versus callipers) (Table 2.6). These results indicate that it was appropriate use pool data from the two methods.

There was no significant difference in species data between institutions. ICC vales were significantly lower in OWM when compared to apes and NWM (Table 2.6; Table 2.7). However, these effects are not likely to affect the results because repeated measurements were still highly statistically significantly related in OWM (i.e., ICC valued still indicated high precision of measurements). These results strongly suggest that variation between species is unlikely to be attributable to measurement error or measurement bias across institutions and more likely to reflect real intra- and inter-specific differences in 2D:4D.

2.8.6: Large-scale phylogenetic effects on 2D:4D

The haplorhine dataset allows large-scale differences in ontogenetic processes across Orders to be analysed for the first time using phylogenetic controls. The results show a consistent pattern of variation in 2D:4D ratios within and between vertebrate groups. The findings support the Phylogenetic Constraint Model (Chang *et al.* 2006; Chang 2008) which proposes that sexual dimorphism in 2D:4D will be more similar between more closely related species, such as scaly lizards and birds, than between birds and mammals or scaly lizards and mammals (Shedlock & Edwards 2009). The results show that in mammals males have lower 2D:4D than females, while in birds and scaly lizards, males have higher 2D:4D ratios than females (Fig. 2.17; Table 2.12). Scaly lizards appear to be the most distinctive group with 2D:4D ratios that are significantly lower than other taxa (Fig. 2.17). The sample upon which these results are based is bigger than that of Lombardo & Thorpe's (2008) on which they based a descriptive analysis of 2D:4D across taxa. These results are the first to robustly support the proposal by Manning (2002a) that 2D:4D should generalises across species with similar limb morphology because of common genetic links (via the same *HOX* genes).

2.8.7: Summary

This is the first study to clearly show that sexual dimorphism in 2D:4D generalises across taxa (Manning 2002a). Broad patterns indicate that 2D:4D is lower in males than females in Class Mammalia, while in the Class Reptilia (birds and scaly lizards; Shedlock & Edwards 2009) males have higher 2D:4D than females. These patterns correspond to differences in sexual determination (Fig 2.17). The results indicate the 2D:4D values differ markedly across vertebrates.

This chapter has reviewed methodologies used in human and non-human 2D:4D research. In comparison to human studies, non-human animal 2D:4D studies tend to use small samples and employ distinct methodologies that have been developed to cope with a wide array of forelimb and hind limb morphologies. These factors are likely to confound comparative analyses across lower-order taxonomic analyses.

Aside from publications associated with this project (see Preface) there have only been two published studies on 2D:4D in haplorhine primates providing data on three species (Table 2.4). The haplorhine dataset has expanded the sample for rhesus macaques (*Macaca mullata*) by 366 individuals, for chimpanzees (*Pan troglodytes*) by 250 individuals and for bonobos (*Pan paniscus*) by 25 individuals (Table 2.4; Appendix 2.5). It has also increased the dataset from 3 to 74 species (n=1286 individuals). Unlike the majority of 2D:4D studies in non-human animals, the methods used in this study parallel those used in humans. The most appropriate methodology was implemented; measurements were taken directly from the skin surface of the digits using procedures that have been shown to be highly repeatable and do not cause soft-tissue distortion. Although the nature of the study necessitated that data be collected by many people from many institutions (n=63), there was no overall measurement bias detected in the sample. Accuracy of data was good; in general ICC values were very high and similar to valued in human studies (Table 2.6; Table 2.7). This indicates that variation of 2D:4D data used in this thesis is unlikely to be a product of poor measurement but reflects real differences between species.

Chapter 3

2D:4D and behavioural indicators of sexual selection in haplorhines¹¹

3.1: Introduction

A substantial body of indirect evidence supports the hypothesis that the second-to-fourth digit length ratio (2D:4D) negatively correlates with prenatal androgen effects (PAE) in humans (see Manning 2002a; 2007a; Chapter 1). The fourth digit (ring finger) appears to be particularly sensitive to PAE such that individuals exposed to high PAE have longer ring fingers relative to their index fingers (McIntyre *et al.* 2005; 2006). Although there is substantial overlap, 2D:4D tends to be lower in males than females (McFadden & Shubel 2002) with differences evident from nine weeks of development (Malas *et al.* 2006; Galis *et al.* 2010). The mechanism proposed to underpin these associations is androgen sensitivity in the homeobox (*HOX*) gene cluster (Manning *et al.* 1998; also see Daftary & Taylor 2006). The posterior *HOXa* and *HOXd* genes organise the development of the terminal limb-bud (digits) and parts of the reproductive system (including the gonads, penile bone and penis) (Zákány *et al.* 1997; Kondo *et al.* 1997; Montavon *et al.* 2008). Additionally 2D:4D has been shown to be moderately to highly heritable between generations (see Voracek & Dressler 2009). Although the physiological interplay between genetic and gestational effects (see below) is not completely understood, it has been proposed that 2D:4D can be used as a proxy for early androgen exposure and can be informative about the development of androgenic-programmed traits (Manning 2002a).

A wide ranging literature has built up that demonstrates links between 2D:4D and sexually selected behavioural traits in humans (Fink *et al.* 2006c; Voracek & Loibl 2009). Low 2D:4D, which potentially indicates high levels of androgen exposure during development and masculinisation of the foetus, has been linked in both sexes to increased intra-sexual competition (Hönekopp *et al.* 2006a), mate seeking (Clark 2004; Hönekopp *et al.* 2006b) and higher levels of aggression and risk taking (e.g. Benderlioglu & Nelson 2004; Bailey & Hurd 2004; Hönekopp 2011). In males, lower 2D:4D has been associated with higher dominance (Neave *et al.* 2003; Manning & Fink 2008; but see Koehler *et al.* 2004), status-seeking (Millet & Dewitte 2008), risk-taking (Stenstrom *et al.* 2011), physical strength (Fink *et al.*

¹¹ Citation for this chapter: Nelson, E. & Shultz, S. 2010. Finger length ratios (2D:4D) in anthropoids implicate reduced prenatal androgens in social bonding. *American Journal of Physical Anthropology*, 141:395-405.

2006c), facial attractiveness (Ferdenzi *et al.* 2011) and behaviours that attract mates (Roney & Maestripieri 2004). 2D:4D is negatively associated with reproductive success in males, but positively associated with fitness in females (Manning & Fink 2008). Low male 2D:4D has been linked to a higher number of sexual partners in males (Hönekopp *et al.* 2006b), although this was unsupported by an earlier study (Rahman *et al.* 2005). Similarly, higher sperm quality and plasma testosterone has been linked with lower 2D:4D in some studies (Manning *et al.* 1998), but is not supported by others (e.g., Neave *et al.* 2003; Benderlioglu & Nelson 2004; Bang *et al.* 2005; Hönekopp *et al.* 2007; Muller *et al.* 2011; McIntyre *et al.* 2011). In contrast to traits linked with low 2D:4D (inferred high PAE), high ratios (inferred low PAE) have been implicated in the development of prosocial behaviours and sensitivity in children (Williams *et al.* 2003; Fink *et al.* 2007). Although intra-population trends tend to be in the predicted direction (low 2D:4D = more masculine traits), correlations are generally weak and some studies have been difficult to replicate (Putz *et al.* 2004). However, these issues may, in-part, be associated with methodological inconsistencies (see Manning & Fink 2008) and sampling differences (i.e. variation in genetic and maternal effects within and between samples).

Relationships between 2D:4D and sexual selection appear to be stronger at the population-level (Manning *et al.* 2000a; Manning *et al.* 2003b; Manning *et al.* 2004a). Within ethnic groups male and female ratios are highly correlated, but between populations the ratios of males and females in some groups appear more masculinised than the ratios of other groups. Manning (2007a) proposed that population differences may be linked to marriage systems and levels of polygyny. High competition between males for females (Møller & Welch 1990; Madhavan 2002; Bove & Vallengia 2009), as is found in polygynous systems, is associated with higher circulating testosterone levels (Gray 2003; Alvergne *et al.* 2009) and could lead to selection for high PAE (Manning *et al.* 2004b; Manning 2007a, p141). High circulating androgens promote male competitive behaviour (Klein 2000; McIntyre *et al.* 2011) and mechanisms facilitating dominance and aggression may be most adaptive in populations where males experience strong competition for access to females (Gray 2003). However, it is not known how long it takes for changes in 2D:4D within a population to occur if levels of sexual selection increase or decrease (i.e., does it take a generation or several generations for digit ratios to alter?) Additionally, in species that experience high competition for resources (mates and food), exposure to high prenatal androgens may also be important in supporting female social hierarchies (Ostner *et al.* 2003; Dloniak *et al.* 2006).

3.1.1: Prenatal androgen effects (PAE) and social development

Prenatal androgens organise the brain and body tissues at a cellular level according to sex-specific patterns (Collaer & Hines 1995; Cooke *et al.* 1998; Fitch & Bimonte 2002). They are crucial to sexing the male phenotype, but also play a role in female development (Pfeiffer 1936; Phoenix *et al.* 1959; Herman *et al.* 2000). Studies observing individual differences in social behaviour after manipulation of prenatal androgens in female rhesus macaques suggest that high levels of PAE may be implicated in programming some masculinised behaviours such as foot-clasp mounting and rough-and-tumble play, although these were dependent on dosage and timing of the treatment (Goy *et al.* 1988; Wallen 1996, 2005). Expression of sex-linked social behaviours (whether manipulated by hormone treatments or not), however, were also influenced by social learning and context (Wallen 1996; 2005; Champagne & Curley 2005). In humans, studies correlating the development of social behaviours with prenatal testosterone (PT), assayed from amniotic fluid, also suggest that PAE may influence neural pathways implicated in social development (Knickmeyer & Baron-Cohen 2006). Lower levels of amniotic fluid PT were associated with higher sociality scores in infants sampled (i.e. higher frequency of eye contact, higher scores in assessments of parent-child relationship quality), while higher levels of PT were linked to lower sociality scores. Although females had higher sociality scores than males, children of both sexes exposed to high PT had lower scores for social relationship quality and higher scores for restricted interests (Knickmeyer *et al.* 2005). It is proposed that sex-differences in bonding-style are a product of adaptation to differences in reproductive investment between males and females (Knickmeyer & Baron-Cohen 2006).

Variation in sensitivity of the androgen receptor gene (ARG) has been linked to differences in social behaviour in the normal population (Comings *et al.* 2002; Rajender *et al.* 2008 Aluja *et al.* 2011) as well as autistic individuals (Henningsson *et al.* 2009). Low 2D:4D has been associated with a more sensitive ARG (Manning *et al.* 2003a; Manning (2007b; 2007a, p136-141).

3.1.2: 2D:4D in non-human taxa

On the basis of the associations between 2D:4D and sex-linked traits, Fink *et al.* (2006c) proposed that variation in human 2D:4D should be viewed within a framework of sexual selection theory. Demonstrating relationships between 2D:4D and levels of sexual competition in other species would extend this conclusion. *HOX* genes are strongly conserved within and between taxonomic groups (Zákány *et al.* 1997; Kondo *et al.* 1997;

Montavon *et al.* 2008), associations between 2D:4D and traits organised by PAE should therefore be common in pentadactyl organisms (Manning 2002a, p17). Although 2D:4D has been studied in a number of diverse vertebrate species (see Chapter 2) there have been no comprehensive cross-species studies of the relationship between 2D:4D and sexually selected behaviours. Most non-human studies have concentrated on demonstrating intra-specific sex differences in 2D:4D or bone derived ratio (McFadden & Bracht 2005) but these have not always been in the expected directions, that is, males do not always have lower 2D:4D ratios than females (see Roney *et al.* 2004; Rubolini *et al.* 2006).

A few studies have looked at sexually selected physical traits within species, but these have yielded conflicting results. For example, hind-limb 2D:4D is negatively related to tail length in barn swallows (Dreiss *et al.* 2007), but positively related to visible badge size in male house sparrows (Navarro *et al.* 2007), although both are sexually selected characters. In mice, aggression is significantly related to 2D:4D but in the opposite direction to humans (2D:4D was positively related to aggression in the mice) (Bailey *et al.* 2005). However, a recent study in which rats were administered androgens during pregnancy, showed that the 2D:4D ratios of their adult offspring (males and females) were more masculinised (lower) than controls (Talarovičová *et al.* 2008). In addition sex-linked activity patterns became more masculinised in the females compared to controls. A cross-taxa study in two distantly related lizard species showed that sexual dimorphism in 2D:4D differed between the taxa (Rubolini *et al.* 2006). In primates lower 2D:4D ratios in chimpanzees (*Pan troglodytes*) compared to bonobos (*Pan paniscus*) are hypothesised to be associated with the more competitive social behaviour of chimpanzees compared to the more tolerant social-style observed in bonobos (McIntyre *et al.* 2009).

Comparisons between closely related species, such as haplorhine primates, should provide for more meaningful comparisons with the main body of 2D:4D research (humans) than comparisons with more distantly related taxa (e.g., lizards). As haplorhines will have more similar biological profiles and social behaviours in this group has been well studied and it relatively easy to categorise (Dixson 1998). In addition 2D:4D may be informative about changes to the ARG. Manning *et al.* (2003a; also see Manning 2007a; 2007b) has hypothesised that reduced sensitivity to androgens (signalled by changes in the ARG) in hominoids (apes) may be linked to changes in social cognition and reproduction. If these differences are reflected in primate digit ratios they may provide valuable insights into the physiological mechanisms that underpin evolution changes in primate sociality (Shultz & Dunbar 2007).

3.1.3: Aims of the study

This is the first study to investigate variation in 2D:4D across a taxonomic group (haplorhine primates). Here I test the concept that digit ratio is linked to sexual selection by investigating how 2D:4D varies according to social systems within and between anthropoid super families. Assuming that PAE are associated with differences in strength of sexual selection in anthropoids, I predict that:

- 1) 2D:4D will be lower (inferred higher PAE) in males than females.
- 2) Species mean ratios will be associated with levels of sexual competition. Lower 2D:4D (inferred higher PAE) will be associated with more promiscuous (non-pair-bonded) social systems and higher levels of inter-male competition, while higher 2D:4D (inferred lower PAE) will be linked to lower levels of sexual selection (pair-bonded systems) and lower levels of inter-male competition.
- 3) 2D:4D will be lower in both females and males that experience high levels of intra-sexual competition.
- 4) Based upon broad classifications of human mating behaviour (Harcourt *et al.* 1981) I expect human 2D:4D to be positioned between the ratios of pair-bonded (PB) and non pair-bonded (NPB) taxa within the Hominoidea (apes).

3.2: Sample and methods

3.2.1: Subjects

Length measurements from the index (2D) and ringer (4D) fingers were obtained for both hands from 1085 anaesthetised, mature, captive primates from 37 anthropoid species sampled from 63 zoos and primate research centres (Table 3.1; see Chapter 2). The staff in the separate institutions collected measurements (see Appendix 3.1). While this method is not ideal, it was the only way to ensure large sample sizes and a broad range of species. 34 species out of a total of 37 species were sampled from multiple institutions (Table 3.1; see Appendix 3.1). There were no systematic biases in species 2D:4D across institutions (Section 2.6.2.4; Chapter 2).

Only species for which data was available for both males and females and, of these species, only those in which data was available for both hands were included in the analysis (n=37 species). Please see section 4.3.6 (additional note) at the end of the Results section.

Table 3.1: Sample variables. Categories: substrate, Plavcan & van Schaik 1992; social system and mating system, Plavcan 2004; inter-female competition levels, Sterck *et al.* 1997; inter-male competition levels, Plavcan & van Schaik 1997 (see Appendix 3.1 for 2D:4D for both sexes and species body weights).

Species	F	M	F	M	F	M	Sub ^a	SS ^b	MS ^c	F	M
	n	n	2D:4D	SD	2D:4D	SD				Comp ^d	Comp ^e
<i>Hylobates lar</i>	2	4	1.067	0.01	1.064	0.01	A	PB	PB	1	1
<i>Nomascus leucogenys</i>	2	2	1.020	0.05	1.008	0.04	A	PB	PB	1	1
<i>Hylobates pileatus</i>	1	1	1.113	0.00	1.068	0.00	A	PB	PB	1	1
<i>Symphalangus syndactylus</i>	7	7	1.051	0.04	1.021	0.09	A	PB	PB	1	1
<i>Gorilla gorilla</i>	39	22	0.907	0.04	0.928	0.06	A/T	NPB	UM	2	3
<i>Pan paniscus</i>	12	13	0.930	0.03	0.924	0.03	A/T	NPB	MM	2	2
<i>Pan troglodytes</i>	149	104	0.917	0.06	0.898	0.05	A/T	NPB	MM	2	2
<i>Pongo pygmaeus</i>	18	8	0.889	0.05	0.869	0.04	A	NPB	UM	2	3
<i>Cercopithecus aethiops</i>	10	12	0.829	0.04	0.824	0.05	A/T	NPB	MM	4	4
<i>Cercopithecus diana</i>	2	2	0.899	0.06	0.884	0.07	A	NPB	UM	3	3
<i>Cercopithecus lhoesti</i>	3	1	0.886	0.03	0.853	0.00	A/T	NPB	UM	3	3
<i>Cercopithecus mona</i>	2	2	0.893	0.08	0.841	0.07	A/T	NPB	UM	3	3
<i>Cercopithecus neglectus</i>	4	10	0.854	0.04	0.821	0.05	A/T	NPB	UM	3	3
<i>Colobus guereza</i>	18	6	0.790	0.06	0.781	0.05	A	NPB	UM	3	3
<i>Macaca fascicularis</i>	9	6	0.832	0.03	0.839	0.03	A/T	NPB	MM	4	4
<i>Macaca fuscata</i>	9	8	0.822	0.03	0.850	0.04	A/T	NPB	MM	4	4
<i>Macaca mulatta</i>	242	53	0.822	0.04	0.813	0.04	A/T	NPB	MM	4	4
<i>Macaca nigra</i>	2	1	0.820	0.02	0.848	0.00	A/T	NPB	MM	4	4
<i>Mandrillus leucophaeus</i>	4	4	0.820	0.08	0.875	0.05	A/T	NPB	MM	4	4
<i>Mandrillus sphinx</i>	17	11	0.856	0.03	0.823	0.03	A/T	NPB	MM	4	4
<i>Papio hamadryas</i>	15	11	0.862	0.04	0.854	0.04	T	NPB	MM	2	4
<i>Presbytis melalophos</i>	3	1	0.758	0.03	0.799	0.00	A	NPB	UM	3	3

Table 3.1: Sample variables continued. Categories: substrate, Plavcan & van Schaik 1992; social system and mating system, Plavcan 2004; inter-female competition levels, Sterck *et al.* 1997; inter-male competition levels, Plavcan & van Schaik 1997. (see Appendix 3.1 for 2D:4D for both sexes and species body weights).

Species	F n	M n	F 2D:4D	SD	M 2D:4D	SD	Sub ^a	SS ^b	MS ^c	F Comp ^d	M Comp ^e
<i>Trachypithecus francoisi</i>	8	4	0.791	0.03	0.776	0.04	A	NPB	UM	3	3
<i>Trachypithecus obscura</i>	5	2	0.792	0.02	0.798	0.03	A	NPB	UM	3	3
<i>Alouatta caraya</i>	12	10	0.897	0.03	0.914	0.03	A	NPB	MM	2	4
<i>Ateles geoffroyi</i>	4	1	0.913	0.04	0.902	0.00	A	NPB	MM	2	2
<i>Callicebus donacophilus</i>	3	2	0.852	0.02	0.845	0.01	A	PB	PB	1	1
<i>Callicebus moloch</i>	12	20	0.858	0.02	0.852	0.04	A	PB	PB	1	1
<i>Callithrix geoffroyi</i>	8	4	0.903	0.05	0.950	0.06	A	PB	PB	1	1
<i>Callithrix jacchus</i>	33	36	0.928	0.06	0.912	0.08	A	PB	PB	1	1
<i>Leontopithecus chrysomelas</i>	4	3	0.994	0.04	0.984	0.03	A	PB	PB	1	2
<i>Leontopithecus rosalia</i>	5	5	0.981	0.03	0.989	0.02	A	PB	PB	1	2
<i>Pithecia pithecia</i>	1	5	0.756	0.00	0.740	0.04	A	NPB	MM	2	2
<i>Saimiri sciureus</i>	9	3	0.902	0.03	0.893	0.03	A	NPB	MM	4	3
<i>Saguinus imperator</i>	5	3	0.993	0.03	1.012	0.01	A	PB	PB	1	2
<i>Saguinus midas</i>	4	6	1.007	0.04	1.013	0.02	A	PB	PB	1	2
<i>Saguinus oedipus</i>	1	1	1.007	0.00	1.029	0.00	A	PB	PB	1	2
Total Individuals = 1085	684	394									

^aSubstrate; A=arboreal; A/T=arboreal/terrestrial; T=terrestrial; ^bSocial System; PB=pair-bonded; NPB=non-pair-bonded; ^cMating System; PB=pair-bonded; UM= uni-male; MM=multi-male-multi-female; ^dFemale competition: 1=pair-bonded, 2=dispersed-egalitarian, 3=resident-egalitarian, 4=resident-nepotistic; ^eMale competition: 1=low frequency-low intensity, 2=high frequency-low intensity, 3=low frequency-high intensity, 4=high frequency-high intensity.

3.2.2: 2D:4D measurements

Institutions were provided with an identical set of detailed instructions and images highlighting anatomical landmarks (Appendix 2.3). Measurement procedure followed that used for humans (Manning 2002a; Manning *et al.* 2007a); lengths were measured to the nearest millimetre along the midline of the digit from the proximal crease (at the base) to the top the fingertip, with callipers or a ruler (see Chapter 2). Digits were held extended throughout, but not maintained in adduction. Repeated measurements were requested and both sets of measurements were taken by the same observer. However obtaining two sets of measurements was not always possible due to time and safety constraints placed on handling a sedated primate. In the absence of repeated measurements single values were used.

An individual's 2D:4D was calculated by dividing the length of 2D by length of 4D. Individuals' data within were then pooled to form a species mean values. All analyses are based upon mean 2D:4D ratios for species or sexes within species. Intra-class correlation coefficient (ICC) was used to assess repeatability of mean 2D:4D (average-score ICCs with absolute-agreement definition; McGraw & Wong 1996).

3.2.3: Social variables

3.2.3.1: Social System

The term 'social system' was used in a general sense and each system incorporates variability in mating and social behaviour (for a more refined definition see (Kappeler & van Schaik 2002). Categories were based upon recognised classifications of social system taken from Plavcan (2004). Species were defined as either pair-bonded (PB) or non pair-bonded (NPB; Table 3.1). Species were classified as PB if males usually bond with one female. Polyandrous species were also placed within this category as the primary bond is between reproductive partners and has evolved as an extension of a monogamous social system (van Schaik & Kappeler 2003). As such, a PB social system in this study defines a more general social system termed 'social monogamy' by van Schaik & Kappeler (2003); the terms 'PB' and 'monogamy' are used interchangeably in this study. Pair-bonding is virtually absent in Old World monkeys; there were no pair bonded Cercopithecoidea in the data set. Males in species that generally mate with multiple females were placed in the NPB (promiscuous) category.

Categories of mating system were also compared. These were taken from Plavcan (2004) and were trichotomous, pair-bonded, uni-male and multi-male-multi-female (Table 3.1; also see Appendix 3.1).

Missing data or revised social systems were added for *Hylobates sp.* (Fuentes 1999), *Cercopithecus diana* (Byrne *et al.* 1983), *Trachypithecus francoisi* (Anderson *et al.* 2004) *Callicebus donacophilus* (Fuentes 1999); *Pithecia pithecia* (Norconk, 2006); *Callithrix geoffroyi*, (Anderson *et al.* 2004); *Leontopithecus chrysomelas* (De Vleeschouwer *et al.* 2000) and *Saguinus imperator* (Baker & Woods 1991).

3.2.3.2: Competition levels

Two published methods of assessing levels of intra-sexual competition were used: 1) Intra-male competition (Plavcan & van Schaik 1997; see Appendix 3.1) and intra-female competition (Sterck *et al.* 1997). Missing data for intra-male competition were estimated from references above. For intra-female competition missing data (i.e. data not included in Sterck *et al.* 1997) were added for *Pongo pygmaeus* (Goossens *et al.* 2006), *Mandrillus sphinx* (Charpentier *et al.* 2005), *Mandrillus leucophaeus* (Hadidan & Bernstein 1979), *Presbytis melalophos*, *Trachypithecus francoisi*, *Trachypithecus obscura* (Sterck *et al.* 1999), *Alouatta caraya* (Jones 1982) and *Pithecia pithecia* (Norconk 2006).

3.2.3.2.1: Intra-male competition

Categories of intra-male competition (Table 3.1; see Appendix 3.1) are based on competition intensity and competition frequency (levels 1 to 4) (Plavcan & van Schaik 1997). Low frequency-low intensity competition is classified as level 1, level 2 is high frequency-low intensity competition, level 3 is low frequency-high intensity competition, and level 4 is high frequency-high intensity competition. Level 1 is primarily associated with socially monogamous species. It is noteworthy that, based on new evidence, Plavcan and van Schaik (1997) altered the classification status of *Leontopithecus sp.* and *Saguinus sp.* from level 1 to level 2 as these species are group living so the frequency of male interaction is higher than true pair-bonded taxa (see Plavcan & van Schaik 1997, p 68). Level 2 is primarily associated with male-bonded groups (male philopatry) (e.g. *Pan sp.*; *Ateles sp.*) that have high levels of tolerance towards each other (e.g., higher ranking males are tolerant towards lower ranking males which is not generally the case in multi-male groups of Old World monkeys). Level 3 is mostly associated with harem social systems and seasonally mating multi-male species,

while multi-male and multi-female species, such as *Macaca sp.* and *Mandrillus sp.*, are placed into the level 4 category that incorporates species in which males are intolerant of other males most of the time.

3.2.3.2.2: Intra-female competition

Levels of intra-female competition are based on female dispersal and social relationships (Sterck *et al.* 1997) (Table 3.1; see Appendix 3.1). A category for pair-bonded species was also included (but was not presented in the original paper by Sterck *et al.* 1997). The pair-bonded category (level 1) includes species in which both sexes dispersed and primarily lived in heterosexual pairs (e.g. Hylobatids) or socially monogamous groups (i.e. the Callitrichids). Level 2 includes species in which females disperse and social relationships are classed as egalitarian (e.g. Hominae, *Ateles sp.*). Level 3 includes species in which females are philopatric and social relationships are classed as egalitarian (e.g., Colobines and most *Cercopithecus sp.* species). In level 4, females are generally philopatric, but social relationships are more competitive and often nepotistic (e.g. *Macaca sp.*).

3.2.3.2.3: Other variables

To check for possible influences of body size on 2D:4D, mean species 2D:4D was regressed on mean species body weight (Smith & Jungers 1997; Lindenfors & Tullberg 1998) while controlling for phylogenetic effects using PGLS methods (see Statistical methods). As hand morphology is also associated to substrate use (Jouffroy *et al.* 1993; Richmond 2007), substrate use was also incorporated (terrestrial, terrestrial/arboreal, and arboreal) as a factor in all analyses. Substrate was taken from Plavcan & van Schaik (1992) (Table 3.1; see Appendix 3.1).

For human 2D:4D ratios (used for comparative species within hominoids), published means were used taken from self-measured finger lengths from a large sample from varying ethnic backgrounds (Manning *et al.* 2007a).

3.2.4: Statistical methods

The data were normally distributed (Kolmogorov-Smirnov Test). Sample sizes for species were highly variable, but there was no evidence of heteroskedasticity in the data, so the analyses were not weighted. Sex differences were evaluated by comparing individual

measurements within species. Associations between pair-bonding, competition measures and 2D:4D were evaluated using species means.

Phylogenetic trees were constructed using evolutionary relationships from published sources (Purvis 1995; Opazo *et al.* 2006, see Appendix 2.6). Relationships were analysed using both non-phylogenetic tests (i.e., ANOVA or General Linear Model; GLM) and Phylogenetic Generalised Least Squares (PGLS) analysis (Grafen 1989). PGLS analysis was executed in 'R' (Ihaka & Gentleman 1996) using APE package (Analysis of Phylogenetics and Evolution) (Paradis *et al.* 2004) with code provided by R.P. Duncan. Pagel's λ was used to estimate the degree of phylogenetic autocorrelation within models (Pagel 1997) (see Chapter 2). Results of Moran's I values indicate high phylogenetic autocorrelation in continuous variables used in this study ($p < 0.001$; Appendix 3.2). As such, the use of PGLS analysis is warranted.

Values stated in the text are PGLS unless otherwise stated. Significance was set to $p < 0.05$ and two-tailed tests were used (unless otherwise stated). F=variance result; p=probability; df=degrees of freedom.

3.2.5: Repeatability estimates

562 individuals in the sample had repeat measurements. 441 of these were obtained using a ruler and 121 with callipers. Intra-class correlation coefficients (ICC; McGraw & Wong 1996) for left-hand 2D:4D ruler measurements ICC's = 0.884, $F_{440,440} = 16.218$, $p < 0.001$, calliper measurement ICC's = 0.910, $F_{120,120} = 21.190$, $p < 0.001$. For right-hand 2D:4D ruler measurements ICC's = 0.859, $F_{440,440} = 13.168$, $p < 0.001$, calliper measurement ICC's = 0.918, $F_{120,120} = 23.320$, $p < 0.001$. Ruler measurements had lower ICC values than calliper derived values, however ICCs for both techniques are high (Voracek *et al.* 2007a; see Chapter 2) and therefore indicate that it is valid to use data derived from both of these methods in the same analysis. High measurement concordance overall may be attributable to the minimal lag times between repeated measurements. However, due to safety and staffing constraints when dealing with anaesthetised animals, this could not be avoided. Large sample size should serve to ameliorate these effects. It was concluded that 2D:4D measurements reflect real differences between individuals and this, in turn, will reflect between-species differences (see Chapter 2).

3.3: Results

3.3.1: 2D:4D and large scale phylogenetic differences

2D:4D varied across super families ($F_{2,36}=11.33$, $p<0.001$). The 2D:4D ratios of Cercopithecoidea (OWM) were significantly lower than the Hominoidea (apes) ($F_{1,23}=41.45$, $p<0.001$) and Ceboidea (NWM; $F_{1,28}=21.51$, $p<0.001$). Although, 2D:4D ratios of Ceboidea were lower than Hominoidea, the difference is not significant ($F_{1,20}=2.45$, $p=0.13$). Families within the Hominoidea and the Cercopithecoidea, but not Ceboidea, significantly differed in their 2D:4D ratios (Table 3.2).

3.3.2: 2D:4D and social system

Across the whole sample, with and without controlling for phylogeny, PB taxa had higher 2D:4D ratios (inferred lower PAE) than NPB taxa (Table 3.3; Fig. 3.1). Within super families (Hominoidea and Ceboidea), PB species had significantly higher 2D:4D ratios than NPB species (Table 3.3).

The 2D:4D ratios of PB Hominoidea were higher than Ceboidea species ($F_{1,11}=7.34$, $p=0.02$) (Fig. 3.1). NPB Ceboidea did not differ from those of NPB Hominoidea or Cercopithecoidea (Ceboidea-Hominoidea, $F_{1,5}=0.045$, $p=0.84$; Ceboidea-Cercopithecoidea, $F_{1,16}=0.34$, $p=0.57$). 2D:4D ratios of the Cercopithecoidea (NPB) were less variable and were significantly lower than NPB Hominoidea ($F_{1,16}=44.31$, $p<0.001$) (Fig. 3.1). 2D:4D was higher in PB than NPB anthropoids. The 2D:4D ratios of PB and NPB NWM and apes did not differ. Cercopithecines had lower 2D:4D than NPB apes, but not significantly lower than NPB NWM.

Correlations between 2D:4D and mating system (PB, single male and multi-male) were highly significant ($F_{2,33}=9.06$, $p<0.0001$, $\lambda=1$). However, this effect was due to strong differences between PB and NPB species as there were no significant differences in 2D:4D within NPB species when mating systems were divided into uni-male and multi-male-multi-female species ($F_{1,21}=0.16$, $p=0.69$, $\lambda=0.99$; Fig. 3.2). These results are consistent with those for sexual dimorphism in body and canine size (Plavcan 2000; 2004).

Table 3.2: 2D:4D between taxonomic families. PGLS=phylogenetic least-squares; F=variance result; p=probability; df=degrees of freedom.

Super Family	Family	n	Non-phylogenetic Analysis			PGLS Analysis			
			F-value	p	df	F-value	p	df	λ
Hominoidea	Hylobatidae-Hominidae	8	52.36	<0.001	7	20.85	0.006	5	0
Cercopithecoidea	Cercopithicidae-Colobinae	16	29.99	<0.001	15	28.29	<0.001	12	0
Ceboidea	Atelidae-Cebidae	10	3.18	0.112	9	0.57	0.466	8	1
	Atelidae-Pitheciidae	5	5.77	0.096	4	1.26	0.344	3	1
	Cebidae-Pitheciidae	11	20.48	0.001	10	2.62	0.140	9	1

Table 3.3: 2D:4D and social system. PGLS=phylogenetic least-squares; F=variance result; p=probability; df=degrees of freedom.

Species mean 2D:4D	Group	n	Non-phylogenetic Analysis			PGLS Analysis			
			F	p	df	F	p	df	λ
	All species	37	32.47	<0.001	33	19.40	<0.001	33	1
Pair-bonded v Non pair-bonded species	Hominoidea	8	33.36	0.002	5	20.85	0.006	5	0
	Ceboidea	13	4.05	0.069	11	14.81	0.003	11	0.984

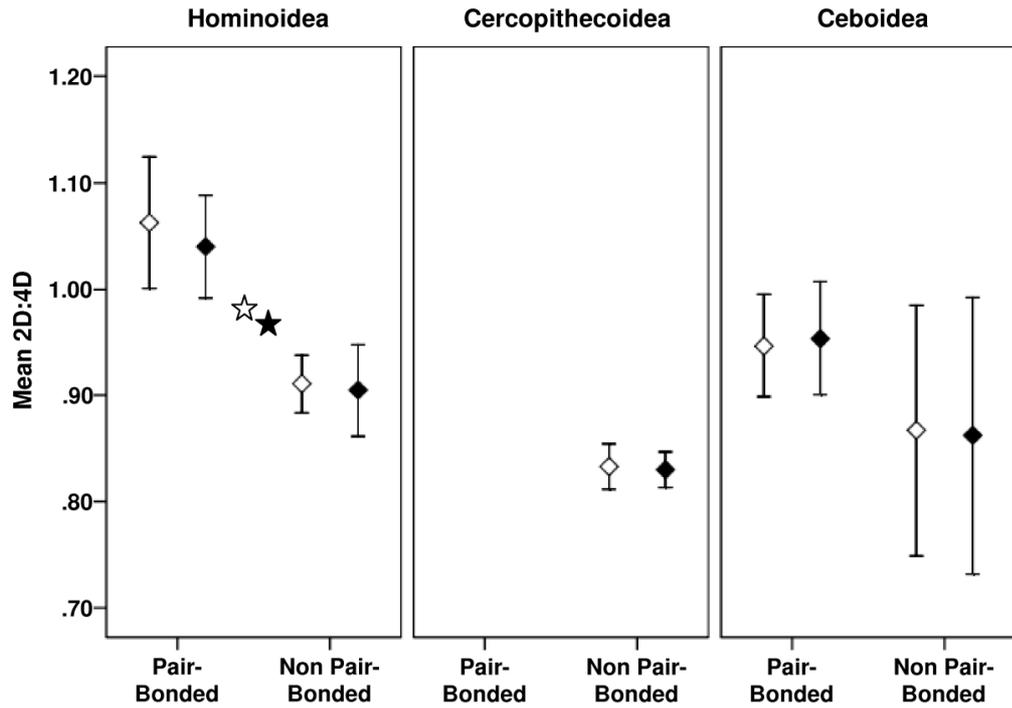


Figure 3.1: 2D:4D between pair-bonded and non pair-bonded taxa within super families. Open symbols represent females and black symbols males. Stars represent human values for comparison. Bars represent 95% confidence intervals. Hominoidea = apes; Cercopithecoidea = Old World monkeys (OWM); Ceboidea = New World monkeys (NWM).

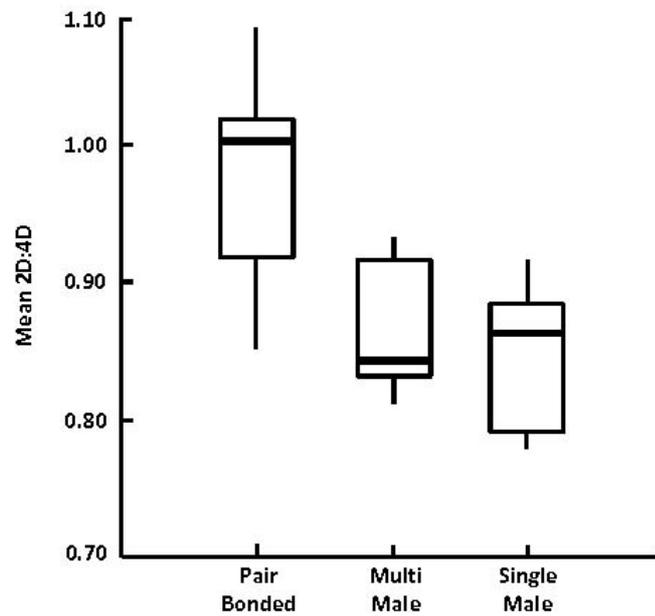


Figure 3.2: 2D:4D across mating systems. There were no significant differences between promiscuous species (multi-male and single male species). Bars represent 95% confidence intervals.

Ape species (Hominoidea) had significantly different mean 2D:4D ratios ($F_{1,7}=53.19$, $p<0.001$). In pair-wise comparisons, PB gibbon species (Hylobatidae) had significantly higher 2D:4D ratios than the NPB great apes (Hominidae) ($p<0.001$). Within the Hominidae, the ratios of *Pongo pygmaeus* were significantly lower ($p<0.05$) than ratios of African ape species (Homininae; Fig. 3.3). Ratios of the Homininae did not significantly differ from each other. Human 2D:4D ratios (Manning *et al.* 2007a) were transposed onto 2D:4D patterns within the Hominoidea. As predicted, human 2D:4D ratios fell between the ratios of PB and NPB taxa (Fig. 3.1; Fig. 3.3) and were significantly different from both (humans *versus* NPB apes, $t=6.75$, $p<0.001$; humans *versus* PB apes, $t=6.69$, $p<0.001$).

3.3.3: 2D:4D and intra-sexual competition

Male and female competitive regimes were highly associated (Likelihood ratio = 61.83, $p<0.001$, $n=37$). Thus, PB species tended to have low levels of both male and female competition, whereas species with high level of female competition also have high levels of male competition. 2D:4D was significantly associated with intra-male competition estimates using non-phylogenetic tests; lower 2D:4D ratios (inferred higher PAE) were associated with higher levels of competition, while higher 2D:4D ratios (inferred lower PAE) were associated with lower levels of competition (Table 3.4; Fig. 3.4).

Due to high levels of phylogenetic autocorrelation (lambda values presented in Table 3.4) this association was not robust to phylogenetic analysis (PGLS analysis). However, if socially monogamous *Saguinus sp.* and *Leontopithecus sp.* (Goldizen 2003) (level 2; high frequency-low intensity) are reclassified as level 1 (low frequency-low intensity), the category to which they were originally assigned (see Plavcan & van Schaik 1997), the association is robust to phylogenetic control (species 2D:4D; $F_{3,31}=7.66$, $p<0.001$).

2D:4D and intra-female competition levels were strongly associated, even after controlling for a strong phylogenetic signal (Table 3.3). Lower 2D:4D ratios (inferred higher PAE) were associated with higher levels of intra-female competition and female philopatry (e.g., staying within the natal group), and higher 2D:4D ratios (inferred lower PAE) were significantly associated with lower levels of competition and heterosexual or female dispersal (Table 3.4; Fig. 3.4).

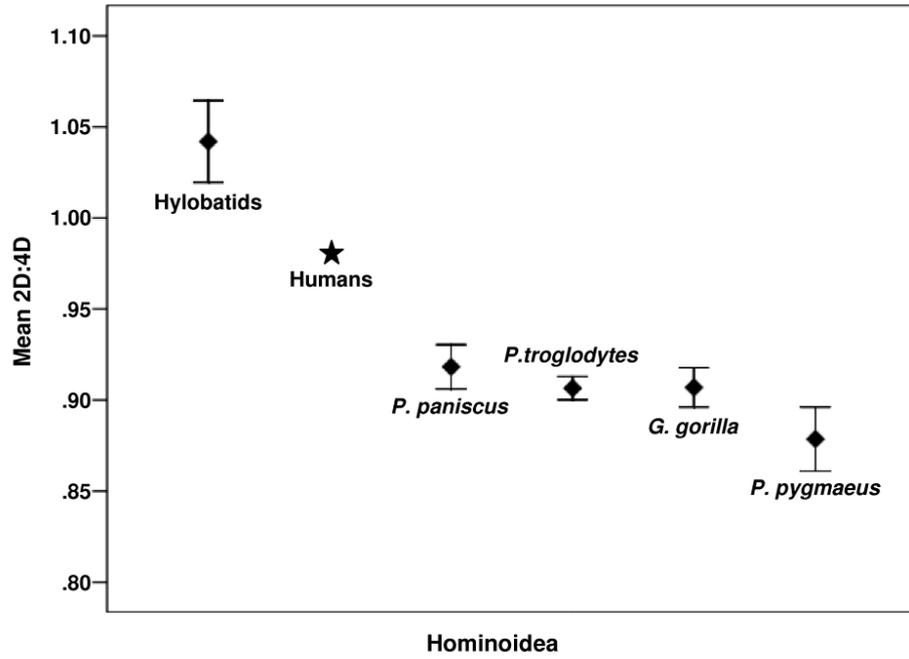


Figure 3.3: 2D:4D across hominoids. Star represents human values for comparison. Bars represent 95% confidence intervals.

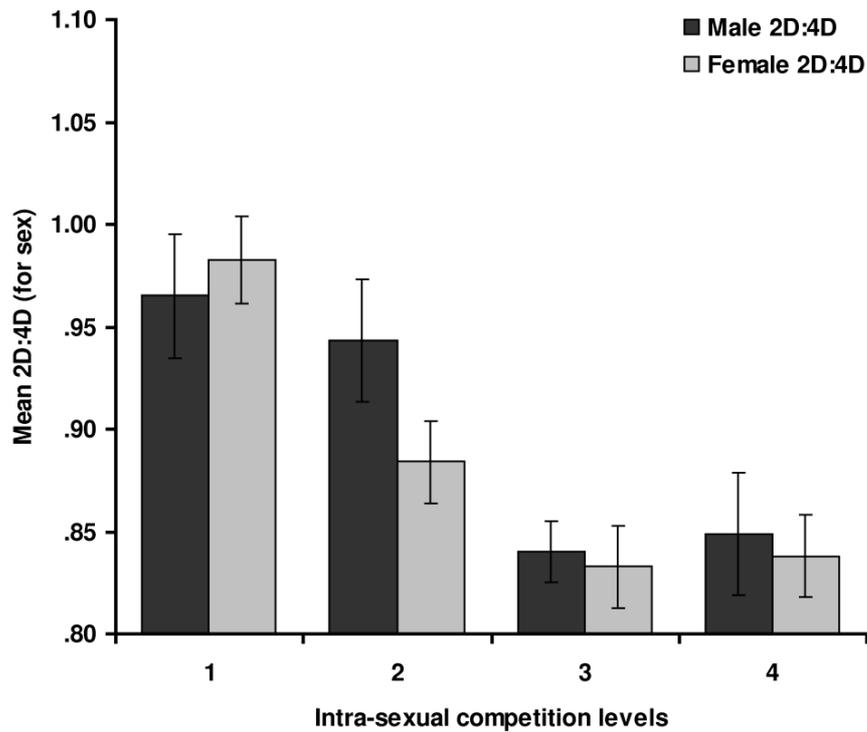


Figure 3.4: 2D:4D and intra-sexual competition levels. Male mean 2D:4D and intra-male competition levels and female mean 2D:4D and intra-female competition levels. Level 1=low frequency-low intensity; Level 2=high frequency-low intensity; Level 3=low frequency-high intensity; Level 4=high frequency-high intensity. Bars represent standard errors.

3.3.4: 2D:4D and sex differences

Male and female 2D:4D ratios were significantly correlated across the whole sample ($R^2=0.90$, $p<0.001$, $df=36$). Controlling for species differences a near significant difference in sex across the dataset was found (species: $F_{36,1047}=53.76$, $p<0.001$; sex: $F_{1,1047}=3.48$, $p=0.06$).

Males in NPB species had significantly lower 2D:4D ratios than females (males 0.845 ± 0.003 s.e., females 0.852 ± 0.003 s.e., species: $F_{23,817}=23.82$, $p<0.001$; sex: $F_{1,817}=5.13$, $p=0.02$). There was no sex difference, however, in PB species (males 0.971 ± 0.008 s.e., females 0.973 ± 0.008 s.e., species: $F_{13,174}=21.84$, $p<0.001$; sex: $F_{1,174}=0.04$, $p=0.85$). Within the hominoids there were significant differences in mean 2D:4D ratios between males and females (species: $F_{7,375}=32.95$, $p<0.001$; sex: $F_{1,375}=4.41$, $p=0.04$). However, there were no significant sex differences in other super families (Cercopithecoidea and Ceboidea).

Significant sex differences in 2D:4D ratios at the species level were detectable only in competition level 2 for both males and females (high frequency-low intensity (species: $F_{3,316}=24.49$, $p<0.001$; sex: $F_{3,316}=6.62$, $p=0.01$; dispersed-egalitarian; species: $F_{3,415}=13.39$, $p<0.001$; sex: $F_{3,415}=3.74$, $p=0.05$). Within this level females had significantly lower 2D:4D ratios than males. Sex differences in the other categories (in both male and female competition classifications systems) were non-significant.

3.3.5: 2D:4D, substrate and body size

There was no significant relationship between mean 2D:4D and species body weight across the whole sample ($r^2=0.06$ $df=36$, $F_{1,36}=2.216$, $p=0.15$) (Table 3.5). Significant relationships were found within the arboreal Hominoidea, which was due to large differences in 2D:4D and body weight between the Hylobatidae and the Pongidae. Relationships were also absent between in all other super families.

Table 3.4: 2D:4D and intra-sexual competition. F=variance result; p=probability; df=degrees of freedom.

Model		Non-phylogenetic Analysis			PGLS Analysis				
		F	p	df	F	p	df	λ	AIC
Inter-male competition	Species 2D:4D	3.37	0.032	36	1.30	0.292	31	1	-123.31
	Male 2D:4D	3.37	0.032	36	0.99	0.409	31	1	-113.24
Inter-female competition	Species 2D:4D	10.94	<0.001	36	7.88	0.001	31	1	-142.20
	Female 2D:4D	9.36	<0.001	36	6.12	0.002	31	0.98	-129.37

Table 3.5: 2D:4D and species weight for substrate. F=variance result; p=probability; df=degrees of freedom.

Substrate	Super family	n	R ²	F	p	df
Arboreal	Hominoidea	5	0.951	28.26	0.01	4
	Cercopithecoidea	5	0.774	4.37	0.125	4
	Ceboidea	13	0.21	0.52	0.49	12
Arboreal-Terrestrial	Hominoidea	3	0.026	0.00	0.98	2
	Cercopithecoidea	10	0.147	0.20	0.67	9

3.3.6: Additional analysis

Subsequent to the publication of this chapter (see Preface) more data were collected on a further six species: *Allenopithecus nigroviridis*, *Cercopithecus hamlyni*, *Macaca sylvanus*, *Cercocebus albegena*, *Cercocebus galeritus*, *Ateles hybridus*. Their digit ratios were added to the dataset increasing the total number of species to 43 (see Appendix 2.5). Here I reanalyse the expanded dataset.

Comparisons in 2D:4D between super families were rendered non significant using PGLS analysis ($F_{2,40} = 0.80$, $p=0.46$, $\lambda=1$). However, correlations across families remained strong ($F_{6,35}=35.11$, $p<0.0001$, $\lambda=0$) and there was no change to the strength of the correlation with social system ($F_{1,41}=14.51$, $p<0.001$, $\lambda=0.99$) or mating system ($F_{2,40}=9.05$, $p<0.001$, $\lambda=0.99$). As in the main analysis no significant differences in 2D:4D were found between uni-male and multi-male-multi-female species ($F_{1,28}=0.43$, $p=0.52$, $\lambda=0.71$). Correlations across female intra-sexual competition levels remained strong ($F_{3,38}=4.58$, $p=0.01$, $\lambda=0.99$) and male intra-sexual competition levels were still non-significant ($F_{3,39}=0.89$, $p=0.46$, $\lambda=1$) unless *Saguinus sp.* and *Leontopithecus sp.* were reassigned to level 1 ($F_{3,39}=4.55$, $p=0.01$, $\lambda=0.99$).

Controlling for species differences by sex across the dataset remained close to significance with the addition of the new data (species: $F_{42,1204}=43.75$, $p<0.001$; sex: $F_{1,1204}=3.22$, $p=0.06$). Within the NPB category males still had significantly lower 2D:4D ratios than females (species: $F_{30,1022}=42.24$, $p<0.001$; sex: $F_{1,1022}=6.10$, $p=0.02$). However, there remained no sex difference in PB species (species: $F_{12,182}=22.06$, $p<0.001$; sex: $F_{1,182}=0.03$, $p=0.89$).

In sum, the additional data reduced the differences in 2D:4D between super families, although differences remained strong between families. Over all, the results remained the same.

3.4: Discussion

This is the first study of variation in 2D:4D across a taxonomic group and shows that the relationship between 2D:4D and sexual selection in anthropoid primates accords with that proposed for humans (Manning, 2007a); 2D:4D is lower (inferred higher PAE) in polygynous species NPB and species with high intra-sexual competition and 2D:4D is higher

(inferred lower PAE) in PB species and species with low levels of intra-sexual competition. In humans low 2D:4D (inferred high PAE) has been linked with intra-sexual competition (Hönekopp *et al.* 2006a), promiscuity (Clark 2004; Hönekopp *et al.* 2005b), aggression (Benderlioglu & Nelson 2004; Bailey & Hurd 2004), and perceived dominance (Neave *et al.* 2003; Manning & Fink 2008). High 2D:4D has been associated with sensitivity and prosocial behaviour (Williams *et al.* 2003; Fink *et al.* 2007). These results are consistent with prenatal androgens in non-human primates potentially promoting the development of competitive and aggressive behaviours, which can ultimately be manifested in increased intra-sexual competition, polygynous social systems and dominance hierarchies (see Dloniak *et al.* 2006). Conversely, low PAE may be important in potentiating behaviours linked to pair-bonding and co-operative breeding (i.e. co-ordination and tolerance) (Shultz & Dunbar 2007; Dunbar 2010a). The results are similar to studies of variation in circulating testosterone levels associated with male reproductive effort (see Alvergne *et al.* 2009).

Analysis of 2D:4D at higher taxonomic levels (super families) also point towards qualitative differences between behavioural and competitive regimes across primate lineages. The consistent relationship between 2D:4D and social systems (PB-NPB) both across and within taxonomic groups, suggests that PAE may provide a mechanistic explanation for the development of species-specific and lineage social behaviour. Social systems in hominoid (ape) and platyrrhine (NWM) species are highly variable, with PB, co-operative breeding, male and female philopatry characteristic of different species in these lineages. This variation necessitates differences in physiological pathways that support different social systems within taxonomic groups. In contrast, OWM (Cercopithecoidea) have low, and relatively invariant 2D:4D ratios (inferred high PAE), which are coupled with relatively invariant social grouping patterns and high levels of intra-sexual competition. Differences in OWM (compared to the other super families) suggest that female philopatry is a specialised response to a competitive ecological niche rather than a highly conserved ancestral adaptation and, as such, OWM are unlikely to present a good model for early hominin social evolution (Strier 1990; Di Fiore & Rendall 1994). Conversely, variability in social systems and 2D:4D in extant NWM and apes leads us to believe that social systems of early primates probably showed similar flexibility. Great apes show higher levels of behavioural flexibility than monkeys (Barrett *et al.* 2003) and this is likely to have been crucial to the evolution of human sociality (Hare 2004; Tomasello *et al.* 2005; Herrmann *et al.* 2007). New evidence is showing that down-regulation of the androgen response in panins (bonobos and chimpanzees) which is reflected in their 2D:4D ratios (McIntyre *et al.* 2009), may be implicated in the evolution of complex forms of communication such as empathy (Wobber *et al.* 2010a; Rilling *et al.* in press; also see Hare & Tomasello 2005a). Interestingly, humans

2D:4D ratios are where we might expect them to be, couched within apes between PB and NPB species (Fig. 3.1). Higher 2D:4D in humans compared to great apes suggests lower PAE and is consistent with the idea of reducing masculinisation and increasing feminisation in the hominin lineage (Manning 2007a); these changes may have been critical to the evolution of human sociality.

In primates, the mechanisms by which steroid hormones affect development patterns, and especially that of the brain, are not well understood. Androgens have been shown to affect brain differentiation across a wide number of non-primate vertebrate species (Cooke *et al.* 1998; Fitch & Bimonte 2002). However, although it appears that estrogens or the aromatization of androgens into estrogens are not implicated in sexual differentiation in higher primates (Wallen & Baum 2002; Wallen 2005) gestational androgens can originate from the gonads and from the adrenal glands, and can either be maternally or foetally derived (Rabinovici & Jaffe 1990; Mesanio & Jaffe 1997). If maternally generated hormones are important, there is the opportunity for maternal effects to play an additional role; in species with female kin-bonded dominance rank hierarchies (i.e. *Macaca sp.*) maternal dominance may impact on foetal exposure to androgens (see Dloniak *et al.* 2006).

In addition to variability in circulating levels of androgens, PAE may also be the result of intrinsic differences in androgen sensitivity (Manning *et al.* 2003a). Increased tri-nucleotide (CAG) repeats in the androgen receptor gene (ARG) lead to reduced androgen sensitivity; extreme numbers of repeats are associated with the androgen insensitivity syndrome (Chamberlain *et al.* 1994). In humans, low 2D:4D and aggressive behaviour is associated with low CAG repeat numbers (Rajender *et al.* 2008; Aluja *et al.* 2011). Longer CAG repeats in the ARG gene in the Hominoidea have led to reduced ARG sensitivity in comparison with OWM (Choong *et al.* 1998; Hong *et al.* 2006). Thus the polymorphic expansion of the ARG in the Hominid clade may provide a genetic vehicle that facilitates male-bonded co-operative sociality found in apes and humans. Of all primates the ARG is the most polymorphic and the least sensitive in humans (Andrés *et al.* 2004). Unfortunately, knowledge of the structure and variation in the ARG is limited in OWM and no data is available for NWM.

Over all sampled species, there was a trend for males to have lower ratios than females. However, these sex effects were not consistent over all species. NPB species (i.e., those with higher levels of male-male competition) had significantly lower male than female 2D:4D ratios, whereas there was no difference between sexes in PB species. This is consistent with the predictions and the assumption that male-male competition may be a key factor driving

PAGE. More challenging was that the pattern was not consistent over all taxonomic groups; male apes had lower ratios than females but in other groups the difference was non-significant. One potential explanation is that the genes responsible for androgen sensitivity are sexually antagonistic, such that strong selection on male reproductive characteristics can have knock on effects on females. Consequently, the androgenic mechanisms associated with the competitive regimes of both males and females are strongly associated, such that both males and females experience either high or low levels of intra-sexual competition. These effects have been shown to occur in promiscuous species where reproductive skew between males and females is high (Rice 2000; Rice & Chippendale 2001; also see Manning *et al.* 2000a). The only competition category where there was some discrepancy between male and female competition strength was in the Level 2 of the intra-sexual competition categories, which is also where there were sex differences (Fig 3.4; but see below).

Although it is clear that the mechanisms by which PAGE is manifested (i.e. whether the androgens are maternally or foetally derived, how sensitive they are to environmental effects, and how important underlying genetic variation is in determining sensitivity), the key issue is that there is substantial evidence that links androgen and estrogens to sexual differentiation of the brain during gestation (Cooke *et al.* 1998; see Chapter 1). Therefore, assuming that 2D:4D reflects PAGE, these results suggest that early androgen exposure may be implicated in priming behaviours associated with sexual competition in adult haplorhine primates. Additionally, circulating androgens (and other hormones) clearly play a role throughout an individual's life, not just prenatally. Thus although not conclusive, the consistency and strength of the associations that have documented here are certainly provocative and consistent with the existing 2D:4D framework.

A caveat must also be made about variation in hand morphology within and between taxonomic groups. It could be argued that some differences in inter-specific 2D:4D may be attributable to phylogenetic inertia or locomotor adaptations to substrate use (Jouffroy *et al.* 1993; Richmond 2007). Additionally some elements of primate hand anatomy may be associated with *HOX*-gene pleiotropy between anatomical structures (Zákány *et al.* 1997; Kondo *et al.* 1997). Phylogeny and substrate use (arboreality and terrestriality) were controlled for in all analyses to try to remove functional and evolutionary influences on hand morphology, yet relationships between 2D:4D and social behaviours were maintained. Thus, if variation in digit ratios were primarily a consequence of functional variation in hand morphology, large-scale relationships would be obscured.

Finally, the methodology draws attention to shortcomings in classifications of inter-male competition (Plavcan & van Schaik 1997). A re-evaluation of the classification by Plavcan & van Schaik (1997) moved Callitrichids from Level 1 (PB) into Level 2. Level 2 incorporates species in which males are primarily philopatric and are in frequent contact, but are tolerant (e.g. *Pan* sp.; *Ateles* sp.). The problem with this change is that Level 2 no longer constitutes a coherent cohort of species in terms of male-male competition. Arguably Callitrichids males do exhibit frequent tolerant interactions and some species show male philopatry (McGrew & McLuckie 1986), but despite high flexibility in social behaviour (Dunbar 1995) the social system is primarily pair-bonded (Dunbar 2010a). The social structure of Callitrichids differs markedly from the other larger bodied non-human primate species in Level 2 which weakens the power of the classification. This factor probably explain the marked sexual differences in 2D:4D in this Level 2 because Callitrichids (high 2D:4D) are assigned to Level 1 in female intra-sexual competition levels (Sterck *et al.* 1997). Evidence from 2D:4D places Callitrichids within the range of other PB NWM and indicates that competition levels should be reappraised.

3.4.1: Summary

Analysis of 2D:4D at large scale taxonomic groupings across anthropoid species has allowed speculation on how broad-scale trends in PAE may underpin sexual selection and aspects of anthropoid sociality. Within catarrhines apes and OWM show distinctly different androgen profiles which appear to be consistent with broad differences in their social behaviour and brain size (Di Fiore & Rendall 1994; Barrett *et al.* 2003). It must be emphasised that PAE does not program social behaviour *per se*, as social behaviour is, by definition, manifested through interactions between individuals and thus cannot occur *in utero*. Rather it is suggested that 2D:4D may inform us about how variation in PAE, including variation in ARG, differentially programs the brain and body tissues and how physiological foundations for social development vary between related taxa. Exploring the relationship between sex steroid receptor sensitivity and neuro-endocrine pathways (e.g., OT and AVP) that impact sociality across vertebrates may uncover an ontogenetic basis for affiliative and competitive behaviour and augment our understanding of mechanisms underpinning social bonding (Walum *et al.* 2008; Israel *et al.* 2008;2009; Rilling *et al.* in press; also see van Honk *et al.* 2011).

I have shown that in general sex differences in 2D:4D in promiscuous species follow the same patterns; within species males have lower 2D:4D (inferred higher PAE) than females. Furthermore, digit ratios of both sexes were found to be low in species that exhibit high

levels of male and female intra-sexual competition. These findings mirror results from human studies (e.g., Fink *et al.* 2006a; Manning & Fink 2008) and adds to the evidence suggesting that PAE are also important for female, as well as male, social development (Voracek & Schicker 2010).

Finally, these results are the first to show human 2D:4D in evolutionary context. Human 2D:4D ratios are couched within the Hominoidea positioned, as predicted, between pair-bonded Hylobatidae (gibbons) and the promiscuous Hominidae (great apes) (Fig. 3.1 and 3.3). This result fits with evidence from comparative analysis of variation in relative testis size and sperm competition within primate social systems (Harcourt *et al.* 1981; Anderson & Dixson 2002; Dixson 2009). Recently a loss-of-function deletion within the ARG has been linked to the loss of penile spines in humans (McLean *et al.* 2011). This characteristic is only seen in PB primates (Dixson 1998; 2009) and provides further support of a reduction in sexual selection in the human lineage (Manning 2007a). It is interesting to speculate if evolutionary shifts in PAE within the African apes, and associated changes in the AR gene, have contributed to the socio-behavioural flexibility of apes, and ultimately of humans (see Manning *et al.* 2003a; Manning 2007b; Hare & Tomasello 2005a; Rilling *et al.* in press).

Chapter 4

2D:4D and anatomical indicators of sexual selection in haplorhines

4.1: Introduction

Human studies show consistent relationships between 2D:4D and behaviours linked to sexual selection (see Manning 2002a; 2007a; Fink *et al.* 2006c; see Chapter 3). These relationships are now supported by the cross-species analyses in the higher primates (see Chapter 3; McIntyre *et al.* 2009). Taken together, these findings strongly suggest that associations between 2D:4D and sexually selected social behaviours generalise across haplorhines.

PAE play a primary role in sexual differentiation in male and female mammals (Phoenix *et al.* 1959; Wallen & Baum 2002; Wallen 2005; Herman *et al.* 2000) and are also implicated in the development of secondary sexual characteristics and changes in body composition (Phillips 2002; Klein 2000). Although puberty remains the phase in which sexual dimorphism in body proportions develop under the activational effects of sex hormones (Bardin & Catterall 1981; Frisch 1984; Zehr *et al.* 2005 Callewaert *et al.* 2010), the template for these changes is programmed before birth (Pfeiffer 1936; Phillips 2002). As such, we might expect 2D:4D to correlate with sexual selected anatomical characteristics. The inconsistent results from the few studies in human studies (e.g., Fink *et al.* 2003; Danborn *et al.* 2010) may therefore simply reflect the fact that humans exhibit relatively low levels of anatomical dimorphism (Ruff 1994) signalling their lower levels sexual selection.

Alternatively, it may indicate that other developmental factors (e.g., growth hormones, pubertal sex hormones) and environmental effects on morphology over growth (e.g., high or low nutrient intake) obscure the programming by PAE. These concepts can be tested by analysing co-variance between traits at higher taxonomic levels where differences in relationships between 2D:4D and sexually dimorphic anatomical characters should be more marked.

In the next section I introduce the target variables against which 2D:4D will be correlated; namely body size (weight), and body size dimorphism, maxillary canine height and maxillary canine height dimorphism (shortened to canine dimorphism) and brain size and brain size dimorphism. Relationships between brain size and sexual selection are reviewed within the context of the Social Brain Hypothesis (Byrne & Whiten 1988; Dunbar 1998). I

will begin by showing why these traits are widely accepted as proxies for variation in sexual selection (e.g., strength of male-male competition).

4.1.2: Anatomical correlates of sexual selection in non-human haplorhines

4.1.2.1: Body size and sexual selection

Early observations of primate behaviour noted that size dimorphism was higher in non-pair-bonded (NPB) promiscuous species than pair-bonded (PB) monogamous taxa and deduced that male-male competition was a prime force in the evolution of size dimorphism (Darwin 1871). In species in which males do not generally provide paternal care, males increase their reproductive potential by being promiscuous and competing for females (Trivers 1972; 1985). In promiscuous primates there is a high tendency for males who are bigger, stronger and more dominant to have priority of access to females (Altmann *et al.* 1996). Males social organisation is dictated by female distribution and grouping patterns which are, in-turn governed by the distribution of food resources in the landscape (Wrangham 1980; van Schaik 1989; Sterck *et al.* 1997; Lindenfors *et al.* 2004). For example, when females clump together one male may be able to monopolise them and a uni-male (UM) social system may evolve, when females are more dispersed multi-male-multi-female (MMMf) grouping may arise. Male reproductive skew is considered high when only one, or a few, males are reproducing within the social group (e.g.,UM). Selection for bigger size enables males to fight for harems and defend them once acquired. In MMMf species these effects are less marked because female group size is larger and one, or a few males, cannot prevent other males from mating. In PB monogamous species, male and female reproductive potential is approximately equal (Holland & Rice 1999; Rice 2000) and monomorphism is common (Plavcan 2000).

Variation in male reproductive skew should therefore be reflected in levels of body size dimorphism which should reflect strength of sexual selection across species. However, these relationships are not straightforward as male competition and social strategies vary within social systems which can alter levels of reproductive skew (Pawlowski *et al.* 1998; Altmann 2000; Plavcan 2004). For example, if males with intermediate body sizes regularly obtain mating opportunities (e.g., by forming coalitions against larger dominant males) selection for larger male size will be reduced (Plavcan 2001). Males mating inside and outside their social group can influence overall variance in male reproductive ability in both groups (Lawler 2009; also see Emlen & Oring 1977; Mitani *et al.* 1996). These kinds of factors mean that sexual dimorphism in body size cannot be differentiated within promiscuous mating systems

(e.g., UM; MMMF; Plavcan 2001; Fig. 4.1). This suggests that simple classifications based on male-male competition are not sensitive enough to be informative about variation in sexual selection in promiscuous species when body size dimorphism is the target variable (see Clutton-Brock *et al.* 1977; Leutenegger & Chilverud 1982; 1985).

Plavcan and van Schaik (1992; Plavcan *et al.* 1995) elaborated upon an existing classification based on variation in inter-male competition (Kay *et al.* 1988) by incorporating information in levels of tolerance between males (*'intensity'*) and the levels of the potential *frequency* males compete with each other (see Chapter 3). Unlike other categorical approaches this classification is able to accommodate variation within social systems. Level 1 (low-frequency-low intensity) is primarily associated with socially monogamous PB species in which male competition intensity and frequency are both low. Level 2 (high-frequency-low intensity) incorporates species that may be primarily PB with one breeding pair, but live in large mix-sexed groups such as callitrichids. Level 2 also includes other species that live in MMMF groups in which the potential for male competition is present but males are largely tolerant of each other, such as *Pan sp.* and *Ateles, sp.* Level 3 and 4 (low frequency-high intensity, high frequency-high intensity, respectively) incorporates variability in male-male competition within the remaining promiscuous species (UN and MMMF). Species in these higher categories (3 and 4) are all NPB and are grouped according to variables such as levels of male reproductive skew, seasonal breeding, dominance hierarchies and the potential for short tenure in uni-male group (see Plavcan 2004, p 232-233). Grouping species according to more varied behavioural patterns reflects the superiority of this classificatory system over more simplistic categories; however it is still somewhat subjective. For example, the extent of inter-male competition exhibited by the array of species in Level 2 appears to be much broader than in other levels.

Using this approach, dimorphism was shown to increase within increasing intensity and frequency of male-male competition, but levels of dimorphism overlapped between categories (Fig. 4.2). Level 3 and 4 species showed similar levels of dimorphism (Plavcan & van Schaik 1997). Level 2 incorporates both PB and NPB promiscuous species and NPB taxa (see above) and species in this category exhibited lower male-male competition and lower body size dimorphism compared to males in the other NPB categories (levels 3 and 4). Strong correlations between body size dimorphism and male competition categories indicated that sexual selection plays a pivotal in body size dimorphism (Fig. 4.2; Plavcan and van Schaik (1997). The fact that body size dimorphism in primates is evident in neonates (showing similar levels of dimorphism to adult levels) suggests that developmental patterns might be programmed by PAE (Smith & Leigh 1998).

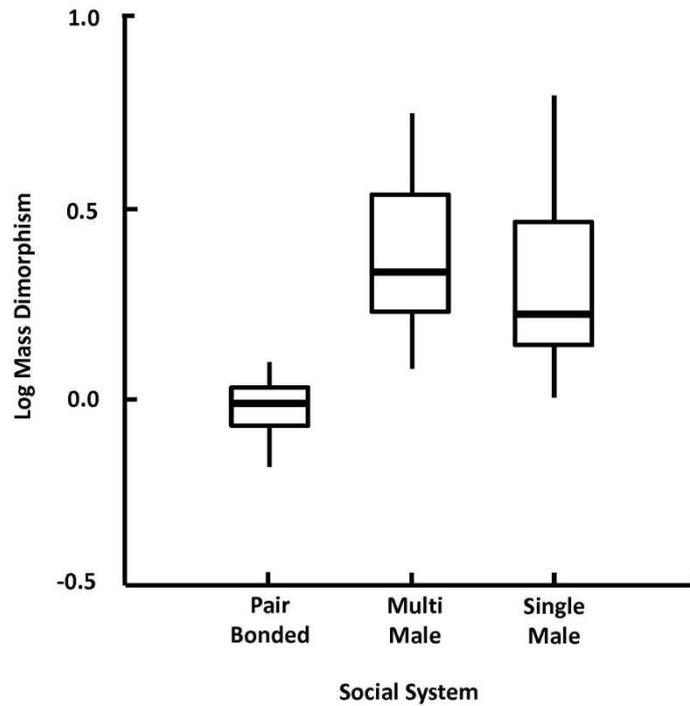


Figure 4.1: Body mass (size) dimorphism and social system across haplorhine primates After Plavcan 2000. Differences between single male (UM) and multi male (MMMMF) species were not significant.

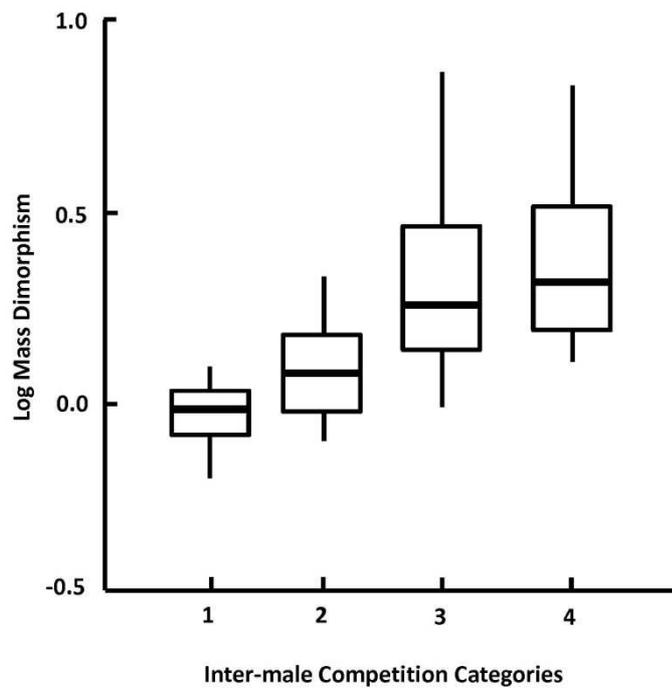


Figure 4.2: Body mass (size) dimorphism and male competition categories in haplorhine primates. After Plavcan 2000. 1=low frequency-low intensity; 2=high frequency-low intensity; 3=low frequency-high intensity; 4=high frequency-high intensity.

Selection for larger body size in males in terrestrial species (but less so in arboreal taxa), appears to have led to an increase in overall body size through haplorhine evolution (Cope's Rule; see Lindenfors & Tullberg 1998; Lindenfors 2002; Clutton-Brock *et al.* 1977; Leutenegger & Cheverud 1982; 1985). Levels of body size dimorphism increase with increasing male size (Rensch's Rule; Fairbairn 1997; but see Lindenfors & Tullberg 1998). This may be associated with many factors such as predator defence by males in terrestrial species (Leutenegger & Kelly 1977; see Plavcan 2001 for a review). As male size increases female size also increases as a correlated response associated with shared genes for body size (Fairbairn 1997; Lindenfors 2002; also see Lande 1980). However, increased size in females, while being beneficial in terms of predator defence, slows reproductive rate because larger animals develop more slowly and produce larger young (Foley 1987; Lindenfors 2002). Here stabilising selection may act to optimise female body size for reproduction, while male body size, which is less constrained is able to increase beyond the female limits leading to body size dimorphism. Selection for large body size therefore has antagonistic effects on males and females. Over time modifier genes act to minimise these antagonistic effects and may lead to a de-coupling of characters between the sexes (Holland & Rice 1999; Rice & Chippendale 2000; Manning *et al.* 2000a; Lindenfors, 2002).

Superimposed upon these effects is the influence of social factors on male and female growth trajectories within species (Leigh 1995; Leigh & Shea 1995; Janson & van Schaik 1993). Detailed studies show that body size dimorphism can occur within species when males mature later by extending their growth pathway compared to females, termed bi-maturation (sex differences in age at attainment of adult size; Wiley 1974) or by males growing faster over a given time period than females or by females maturing more slowly or more rapidly than males (Shea 1986). Any combinations of these processes can lead to variations in the extent of body size dimorphism (or to monomorphism; Plavcan 2001). These processes can occur independently of each other and can also vary between closely related species (Leigh 1992). Hormonal changes across ontogeny also impact growth trajectories and add to the complexity of body size differences in males and females (Berinstein *et al.* 2007; 2008). Hormones linked to growth do not necessarily positively correlate with adult size (e.g., insulin-like growth factor), although estradiol and testosterone do appear to be important predictors of growth and size in some species (Berinstein *et al.* 2007; 2008). This may be due to direct effects on the tissues or by stimulating growth hormones via up-regulation of the hormone receptors (see Zuloaga *et al.* 2008; Roney *et al.* 2010).

In general, the influences of sex hormones and growth hormones on ontogenetic patterns are highly variable and tend to be poor predictors scaling differences between species (Berinstein *et al.* 2007; 2008). Environmental factors can have a marked effect on body growth and these factors can influence males and females differently (Gray & Wolfe 1980 Turner *et al.* 1997; Diverse Populations Collaborative Group 2005).

In sum, body size and body size dimorphism are associated with levels of sexual selection but these relationships are weakened by many intrinsic and extrinsic factors.

4.1.2.2: Canine size and sexual selection

Although sexual dimorphism occurs across the dentition in non-human primates (Lucas *et al.* 1986), dimorphism is accentuated in the canines. As a consequence, research on dental dimorphism has mostly concentrated on variation in canine (maxillary crown height) dimorphism. I therefore focus on that character here.

Similar to body size dimorphism correlations with simple categories of social system identified significant differences in canine size dimorphism between species in PB and NPB groups, but no significant differences within NPB categories (Fig. 4.3; Harvey *et al.* 1978; see Plavcan & van Schaik 1992). Applying their classification of inter-male competition Plavcan & van Schaik (1992) found that canine dimorphism increased with increasing sexual selection; higher canine dimorphism was most strongly associated with higher levels of male-male competition (Fig. 4.4). Relationships were stronger for competition ‘intensity’ (linked to how intolerant males are of each other); canine dimorphism increased in accordance with male reproductive skew. However, male-male competition did not explain the all variation in canine dimorphism; female canine size also showed high variability.

Plavcan and van Schaik (1997) demonstrated that female-female competition also impacted canine dimorphism and this varied according to female social relationships. ‘Frequency’ had the opposite effect to that in males; females in low frequency species had bigger canines and this served to lower sexual dimorphism. It is proposed that more isolated females compete more intensely over resources compared to group living females and this increases canine size in these females. In contrast, females in high frequency species often obtain food or defend food patches via coalitionary support from allies or close female kin; female canines do not need to be big because the weapon is the threat of the group. In these species canine dimorphism is increased because female canines are small compared to those of males. These coalition-effects also reduce male canine size when males group together as a force. In

level 2 promiscuous species (Fig. 4.4) males form coalitions with male peers and kin (e.g., *Pan troglodytes*). In these species canine dimorphism is reduced because male canine size is reduced and brought closer to that of females.

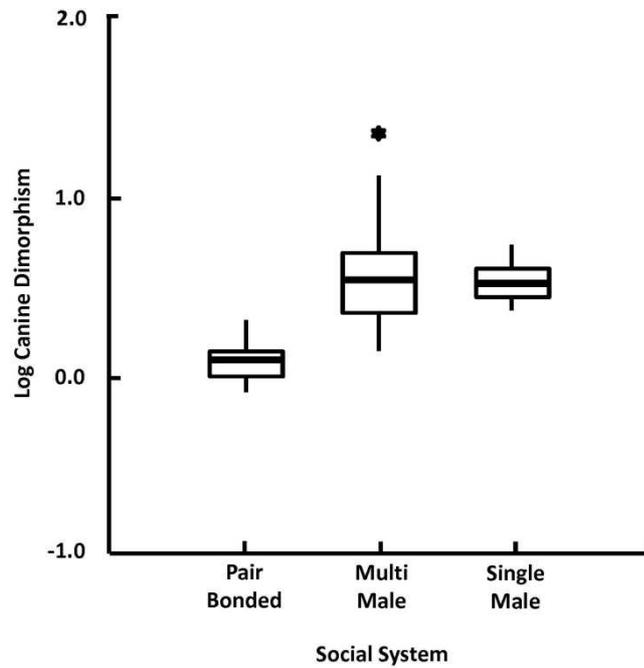


Figure 4.3: Canine dimorphism and social system across haplorhine primates. After Plavcan 2004. Differences between single male (UM) and multi male (MMMF) species were not significant.

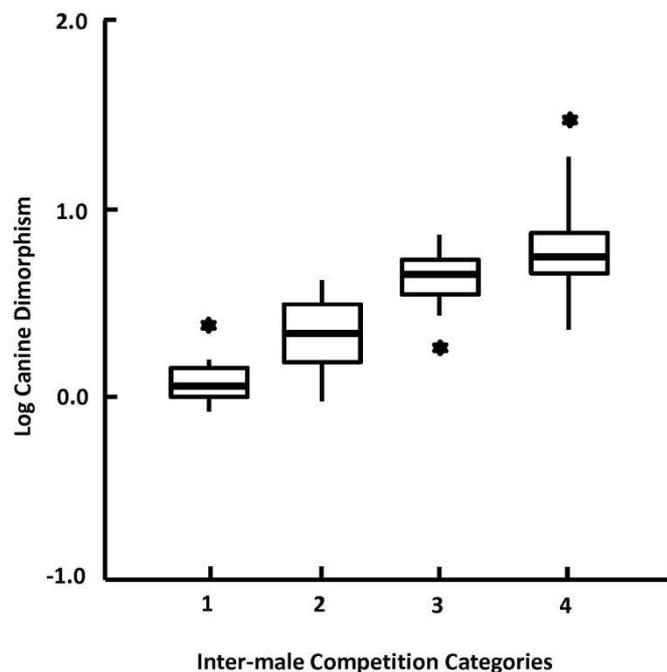


Figure 4.4: Canine dimorphism and male competition categories in haplorhine primates. After Plavcan 2000. 1=low frequency-low intensity; 2=high frequency-low intensity; 3=low frequency-high intensity; 4=high frequency-high intensity.

Differential selection on male and female canine size across species may correspond to variation in the ontogenetic pathways of dental development within species. The limited studies that have examined the ontogeny of canine formation show that the bi-maturation is the primary developmental factor driving canine dimorphism (Swindler 1985; Schwartz & Dean 2001; Leigh *et al.* 2005). The timing of development between males and females within species is also linked to social competition (Leigh *et al.* 2005). For example in UM gelada baboons (*Papio hamadryas*) males have to migrate from the natal group and compete for females when they are still juveniles; in this species male canine development is accelerated early in their juvenile phase compared to that of females. This contrasts with male canine development in mandrills (*Mandrillus sp.*) that live in MMMF group. In this species development is delayed to avoid conflict with mature males until males grow larger and stronger (Leigh *et al.* 2005). In hominoids canine development follows a bi-maturational pattern with males taking longer to form their canines than females (Schwartz & Dean 2001). In PB monogamous species there is no appreciable differences in canine (or body) dimorphism (Thoren *et al.* 2006; Leigh *et al.* 2005).

Canine dimorphism is higher in terrestrial species than arboreal taxa (Plavcan 2001) and is most marked in males of savannah-living species. In species occupying open environments males play a bigger role in predator defence than females (Plavcan & van Schaik 1992). This is believed to increase selection for increased canine size in males which leads to increased canine dimorphism. It is notable that these effects appear to be unrelated to body size and body dimorphism, which is also known to be higher in promiscuous, terrestrial taxa (Plavcan & van Schaik 1992; but see Thoren *et al.* 2006). This apparent mismatch between body size dimorphism and canine size dimorphism is also expressed in colobines. Colobines are promiscuous, arboreal catarrhines that exhibit relatively low body size dimorphism yet possess moderate canine dimorphism (Plavcan 2001).

Comparative analyses indicate that body and canine ontogeny are de-coupled (Schwartz & Dean 2002; Leigh *et al.* 2005). Canine development is known to be highly genetically constrained (Harila-Kaera *et al.* 2001) although subtle sex differences have been linked to PAE in humans (see Dempsey *et al.* 1999). Body size, in contrast, is less constrained and is freer to vary with environmental factors (Herculano-Houzel 2009).

In sum, evidence indicates that canine size and canine size dimorphism are associated with levels of sexual selection but these relationships are highly variable across species and do not necessarily correlate with levels of body size dimorphism.

4.1.2.3: Brain size and sexual selection

Across primates brain development begins early and is highly conserved (Smith 1989; Martin *et al.* 1994). Sex differences in the brain are programmed by PAE (Phoenix *et al.* 1959; McClusky & Naftolin 1981; Arnold & Gorski 1984; Wallen 2005). Programming continues into postnatal life in primates (Lindenfors *et al.* 2007; Schulz *et al.* 2009), although the extent of these postnatal processes (e.g., neurogenesis and cell pruning) are likely to be constrained by phylogenetic differences in brain development (Gould 1977, p 68; Bandeira *et al.* 2009). Brain size and body size are tightly correlated and scale in relation to each other according to biological principals (see Jerison 1973, Martin 1981; Shultz & Dunbar 2010). Within species females (usually the smaller sex) therefore have absolutely smaller brains, but sex differences in brain size are largely lost when body size is controlled for. Across taxonomic groups disassociations in brain size and body size can occur because body size can vary according to environmental factors, but brain size is more constrained (Herculano-Housel 2009; also see Shultz & Dunbar 2010).

As sexual dimorphism in brain size is not marked it provides little information about selection processes on males and females within species. Studies have therefore focussed on how species brain size or relative brain size (e.g., neocortex ratio) correlate with sexual selection or how internal brain architecture linked to either male or female reproductive-drives vary with sexual selection across species.

Studies examining brain size and social behaviour across primates are often couched within the broad research theme of the Social Brain Hypothesis (SBH; Byrne & Whiten 1988; Dunbar 1998). The theory was originally formulated to explain why primates have large brains (for body size) compared to other taxa (Byrne & Whiten 1988; Whiten & Byrne 1997) but gradually evolved into a framework investigating the role of sociality in ecological problem-solving (e.g., acquiring food, mates, avoiding predators). Solving ecological problems enables individuals to survive and reproduce and doing this within a group confers benefits such as predator avoidance (Lee 1994; Shultz *et al.* 2004). However, group life also imposes problems. In order to maintain group cohesion decisions made by individuals to acquire resources have to factor in group-level needs; decisions become a compromise between selfish actions, maintaining bonds with close allies and co-operating to maintain group cohesion (Hamilton 1964; Shultz & Dunbar 2007). As group size increases, decision-making processes get more complex and monitoring social relationships become more cognitively intensive and “wrong decisions in the context of sexual and parental behaviour

could be very costly for sustaining reproductive success” (Keverne *et al.* 1996). As the social environment becomes more complex, brain size increases. Neocortex size increases with total brain size. The frontal lobe is situated within the neocortex and is the hub of cognitive processing often termed ‘executive function’ (Kolb & Wishaw 1996). The frontal lobe has increased disproportionately through primate evolution (Finlay & Darlington 1995), although this size change still remains within the scaling relationships for primates (Semendeferi *et al.* 1997; Herculano-Houzel 2009).

Correlations between neocortex size and measures of sexual selection in primates have been found. NPB species have bigger neocortices than PB species, suggesting that that male competitive behaviour may have been a driving force in brain evolution (Sawaguchi & Kudo 1990). This is supported by a positive relationship across primates between relative neocortex size and higher body size dimorphism and socioeconomic sex ratio (SSR; the number of adult females per males in a breeding social group; Sawaguchi 1997; but see Schillaci 2006). Neocortex ratio was found to negatively predict mating success and dominance rank in males when male groups size was controlled for (Pawlowski *et al.* 1998). This suggests that lower ranking males are able to employ strategies against higher ranking males that increase their mating potential and reduce levels of male reproductive skew (see Byrne & Corp 2004; Higham & Maestriperi 2010). In encephalised primates individuals circumvent restrictions imposed by dominance hierarchies by employing social strategies to improve fitness (Pawlowski *et al.* 1998; Byrne & Corp 2004)

As males and females are under differing selective pressures in terms of reproductive fitness and these pressures are somewhat antagonistic (see Introduction). It would be of interest to see how selection impacts male and female brain architecture across primates. Unfortunately, this kind of information is relatively limited (e.g., Stephan *et al.* 1981; Semendeferi *et al.* 1997; Rilling & Insel 1999). Of those data that are available the sexes are most often pooled, probably as a consequence of low sample sizes, and this prevents the study of sex-specific effects on brain morphology. As such, researchers have developed novel comparative methods to investigate how differing selective pressures on sexual selection and sociality may have differently impacted brain morphology in the two sexes. For example Lindenfors *et al.* (2007) extended a line of research initiated by Lindenfors (2005) by looking at how levels of sexual selection co-vary with brain structures known to be associated with behaviours linked to male-male competition (e.g., mesencephalon, diencephalon) and female sociality (e.g., neocortex). At the species-level higher levels of male-male competition correlated positively with brain architecture linked to aggressive behaviour and with motor centres that deal with body control (e.g., the septum), but correlated negatively with areas

linked to aggressive control (e.g., inhibition of behaviour). No significant relationship was found between male-competition and neocortex size. In contrast female group size was positively correlated with neocortex size (Lindenfors *et al.* 2007). This latter finding supports previous findings (Lindenfors 2005) showing that neocortex size was larger in species that have bigger female social networks and was interpreted as a sign that primate sociality is driven by females group size which, in turn, dictates male group size (Lindenfors 2005). Differing selection pressures on male and females impose changes on different parts of the brain. However, it is currently not possible to know if these neurological structures exhibit sexual dimorphism or are similar in size as a consequence of genetic correlation between the sexes.

Evidence of the influence of sexual selection on brain architecture is supported by investigations into genetic imprinting through primate brain evolution (Keverne *et al.* 1996). Genetic imprinting is the expression of genes in a parent-specific manner (maternal or paternal) and these genes are considered in conflict, in terms of evolutionary fitness, due to the differing interests of the parental genome (Moore & Haig 1991; also see Rice 2000). Male imprinted genes are selected to promote the survival of each offspring, while selection of female imprinted genes is concerned with maternal needs and nourishment of her current offspring and subsequent offspring. For example paternal imprinting is often linked to increased size while maternally expressed genes are linked to conserving energy and smaller size. These effects reflect male- and female-biased reproductive strategies. Based on patterns of parental genomic imprinting in rodent brains, research across primate species shows that maternally imprinted genes are focussed in the 'executive brain' (neocortex and striatum) and this increases through primate evolution (see Dunbar 1998). This increase occurs alongside a decrease in size of the paternally imprinted 'emotional brain' (hypothalamus, septum; Keverne *et al.* 1996). These adaptations have led to shift towards behaviours based on decision making with a concomitant reduction in behaviours controlled by hormonal-drives. In catarrhines these changes have occurred alongside reductions in olfactory sensory input and increases in visual processing (Barton 1998; Curley & Keverne, 2005). The fact that maternally imprinted genes have been favoured over male imprinted genes across primate evolution does not disadvantage male reproduction as males also benefit from large brains and strategy-based reproductive behaviours (Keverne *et al.* 1996; Byrne & Whiten 1988; Whiten & Byrne 1997; Pawlowski 1998).

Investigating relationships between brain size and sexual selection across taxonomic groups shows that primate sociality is characterised by a strong group size effect (Shultz & Dunbar 2007). However, it has been shown within the SBH paradigm that it is both the quantity of

relationships (group size) the quality of the social bonds that, together, impose strong selective forces in primate brain size evolution (Dunbar & Shultz 2007a; 2007b). Looking across non-primate taxonomic groups (e.g., ungulates, birds, carnivores), PB social systems appears to be more cognitively expensive to maintain than other social systems (Shultz & Dunbar 2007). PB relationships require high levels of monitoring of reproductive partners so that feeding and infant care is synchronised (Dunbar 2010a; Dunbar & Shultz 2010). In many vertebrate species large groups cluster as aggregations of individuals to avoid predation (e.g., herds; Lee 1994); the bonds that form are more transient and demand less monitoring than PB relationships. NPB primates deviate from other mammalian and avian taxa because the qualities of the social relationships primates form appear to be pair-bond-like (i.e., primate species that live in large groups do not live in transient aggregations but form highly organised societies which are often underpinned by closely monitored 'friendships'; Silk 2002a; Shultz & Dunbar 2007; Dunbar 2010a). Maintaining complex societies like these is more cognitively demanding and may explain why group size has such an impact on primate sociality and brain evolution (Shultz & Dunbar 2007).

Phylogenetic effects on brain size across haplorhines are also apparent and suggest large-scale developmental differences between super families (but see Herculano-Houzel 2009). Apes have larger brains than Old World monkeys, who, in turn have larger brains than New World monkeys (Dunbar 1998; Barratt *et al.* 2003; Roth & Dickie 2005). Apes appear to have a better understanding of mind-states than monkeys (reviewed in Tomasello *et al.* 2005; also see de Waal & Aureli 1999) and consistently do better in cognitive testes than monkeys (see Barrett *et al.* 2003; but see Amici *et al.* 2009). Ape intelligence may be associated with neural adaptations that have evolved to circumvent the cognitive challenges incurred by having to keeping track of social relationships during periods of separation (fission-fusion social systems; Barrett *et al.* 2003; Aureli *et al.* 2008). In contrast, in most monkey species individuals remain in close proximity within the social group which means that monitoring and maintaining social bonds between allies and potential reproductive is less cognitively demanding. These phylogenetic differences in brain size may correspond to qualitative differences in sociality.

Evidence from relationships of 2D:4D across social systems (Chapter 3) show that PB species tend to have high 2D:4D (inferred low PAE) and NPB primates tend to have lower 2D:4D (inferred higher PAE) (Fig. 3.1; Chapter 3). In the light of SBH we might expect to see some general patterns; high 2D:4D ratios to be more common in smaller brained species and lower 2D:4D ratios to be more prevalent in non-pair-bonded taxa. Differences may also occur within super families.

4.1.3: Aims and predictions

Aims: The aim of this study is to correlate species mean 2D:4D against anatomical characteristics linked to sexual selection in haplorhines to test if 2D:4D reflects PAE on gross morphology at the species-level.

Predictions:

- 1) Based upon the results of Chapter 3 it is predicted that lower 2D:4D (inferred high PAE) will be associated with higher sexual dimorphism in body and canine size, larger brain size and bigger total group size across species.
- 2) Due to genetic constrains 2D:4D will be more strongly associated with canine dimorphism and brain size than with body size dimorphism.

4.2: Materials and methods

4.2.1: Sample

The sample consisted of 44 haplorhine species from captive populations (Table 4.1). A sample of *Cebus apella* (n=20) were added to the updated sample in Chapter 3 (see Section 3.3.6: Additional analysis and Appendix 2.5 for sample sizes on individual species).

4.2.2: Variables

4.2.2.1: Digit measurements and 2D:4D

See previous chapter for details of how the 2D:4D sample was collected and measured. In this analysis mean values for male and female 2D:4D and a species mean are used (see Table 4.1; Appendix 2.5 provides data on male and female 2D:4D and sample sizes). A dimorphism value based upon male and female 2D:4D value is also calculated and analysed. To obtain this value for 2D:4D conventional methods for calculating dimorphism were followed; male mean values were divided by female mean values. Cohen's *d* was calculated to show the magnitude of the difference between male and female 2D:4D. Cohen's *d* represents the size effect between male and female mean 2D:4D; a negative value indicates males have a lower ratio (inferred higher PAE) than females. For Cohen's *d* an effect size of 0.2 to 0.3 is considered be small, around 0.5 a medium size effect and 0.8 or above to be

large (see Dunst *et al.* 2007). As the calculation of Cohen's *d* requires a standard deviation value for each group, values are missing in those species represented by only a single male or a single female (even though both sexes are represented).

4.2.2.2: Body size measurements

4.2.2.2.1: Body size and body size dimorphism

Body size was based on body weight data was taken from published sources (Smith & Jungers 1997; Lindenfors & Tullberg 1998). In the analysis I used mean values for males and females and an overall species mean (see Table 4.1; see Appendix 4.1 for sexed values). Body size dimorphism was calculated by dividing male mean body weight by female mean body weight (Table 4.1). These values were log-transformed.

Neonatal body size was taken from Smith and Leigh (1998) and data was available for 31 species (see Appendix 4.2 for the data). Again, male mean values were divided by female mean values. These values were log-transformed.

4.2.2.2.2: Canine size and canine size dimorphism

Canine size, specifically maxillary crown height, was taken from published articles (Plavcan 2004; Thorén *et al.* 2006). In the analysis mean values for males and females are used as well as an overall species mean (see Table 4.1; see Appendix 4.1 for sexed values). Canine size dimorphism was calculated by dividing male mean maxillary crown height by female mean maxillary crown height. These values were log-transformed.

4.2.2.2.3: Brain size and brain size dimorphism

Endocranial volume (ECV) was used as a proxy for total brain size (Isler *et al.* 2008). In the analysis a mean values for males and females as well as an overall species mean (see Table 4.1 see Appendix 4.1 for sexed values). I estimated brain size dimorphism by dividing male ECV by female ECV. These values were log-transformed.

Table 4.1: Main sample variables (species-level). See Appendix 4.1 and 2.5 for data on males and females. A=arboreal; A/T=arboreal/terrestrial; T=terrestrial.

Genus	Species	Substrate	Body Dimorphism	Canine Dimorphism	Species ECV	Species 2D:4D	sd	Cohen's <i>d</i>	2D:4D Dimorphism	Group Size
<i>Pan</i>	<i>paniscus</i>	A/T	1.30	1.39	341.29	0.918	0.029	-0.34	0.988	63
<i>Pan</i>	<i>troglydytes</i>	A/T	1.19	1.42	368.35	0.907	0.051	-0.34	0.981	53
<i>Gorilla</i>	<i>gorilla</i>	A/T	1.63	1.74	490.41	0.907	0.041	0.63	1.028	7
<i>Pongo</i>	<i>pygmaeus</i>	A	2.23	1.69	377.38	0.879	0.043	-0.53	0.976	5
<i>Nomascus</i>	<i>leucogenys</i>	A	1.01			1.023	0.03	0.62	1.013	2
<i>Symphalangus</i>	<i>syndactylus</i>	A	1.11	1.22	123.50	1.045	0.069	-0.32	0.979	2
<i>Hylobates</i>	<i>lar</i>	A	1.10	1.16	101.87	1.065	0.01	-3.03	0.982	2
<i>Hylobates</i>	<i>pileatus</i>	A	1.08		101.87	1.091	0.032		0.960	2
<i>Allenopithecus</i>	<i>nigroviridis</i>	A/T	1.98		58.20	0.869	0.049	0.19	1.010	15
<i>Chlorocebus</i>	<i>aethiops</i>	A/T	1.43	1.81	65.00	0.831	0.043	0.15	1.007	9.6
<i>Cercopithecus</i>	<i>l'hoesti</i>	A/T	1.81	1.84	74.20	0.873	0.031		0.954	29
<i>Cercopithecus</i>	<i>diana</i>	A	1.33	1.59	62.61	0.869	0.066	0.07	1.001	8
<i>Cercopithecus</i>	<i>hamlyni</i>	A	1.63			0.844	0.071		0.867	3
<i>Cercopithecus</i>	<i>neglectus</i>	A	1.77	1.72	65.97	0.834	0.041	-1.22	0.950	3
<i>Cercopithecus</i>	<i>mona</i>	A/T	1.76	1.91	61.84	0.874	0.074	-0.46	0.956	32
<i>Macaca</i>	<i>sylvanus</i>	A/T	1.12		93.93	0.782	0.045		0.921	10
<i>Macaca</i>	<i>nigra</i>	A/T	1.58	2.61	94.90	0.832	0.022		1.045	24
<i>Macaca</i>	<i>fascicularis</i>	A/T	1.67	2.26	63.98	0.835	0.029	0.28	1.008	15.6
<i>Macaca</i>	<i>mulatta</i>	A/T	2.07	2.09	88.98	0.819	0.036	-0.04	0.991	32.9
<i>Macaca</i>	<i>fuscata</i>	A/T	1.29	2.05	102.92	0.838	0.038	0.85	1.041	31

Table 4.1 continued: Main sample variables (species-level). See Appendix 4.1 and 2.5 for data on males and females. A=arboreal; A/T=arboreal/terrestrial; T=terrestrial.

Genus	Species	Substrate	Body Dimorphism	Canine Dimorphism	Species ECV	Species 2D:4D	sd	Cohen's <i>d</i>	2D:4D Dimorphism	Group Size
<i>Papio</i>	<i>hamadryas</i>	T	2.29	2.62	146.17	0.856	0.036	-0.20	0.991	38.1
<i>Cercocebus</i>	<i>albegina</i>	A	1.41	1.89		0.865	0.031	0.28	1.013	12.6
<i>Cercocebus</i>	<i>galeritus</i>	A/T	1.85	1.38		0.848	0.011		1.020	26
<i>Mandrillus</i>	<i>leucophaeus</i>	A/T	1.70	4.27		0.855	0.072	1.05	1.085	44
<i>Mandrillus</i>	<i>sphinx</i>	A/T	2.17		153.88	0.840	0.032	-0.82	0.975	368
<i>Colobus</i>	<i>guereza</i>	A	1.22	1.47	74.39	0.785	0.055	-0.24	0.984	4
<i>Trachypithecus</i>	<i>obscurus</i>	A	1.28	1.77	62.12	0.800	0.027	0.233	1.009	6
<i>Trachypithecus</i>	<i>francoisi</i>	A	1.05			0.780	0.038	-0.35	0.982	30
<i>Presbytis</i>	<i>melalophos</i>	A	1.02	1.71	64.85	0.781	0.035		1.050	38.1
<i>Callicebus</i>	<i>donacophilus</i>	A	1.00			0.825	0.025	-2.15	0.959	2
<i>Callicebus</i>	<i>moloch</i>	A	1.16	1.08		0.855	0.029	-0.15	0.993	2
<i>Pithecia</i>	<i>pithecia</i>	A	1.14	1.32	32.56	0.755	0.041		0.922	4.5
<i>Alouatta</i>	<i>caraya</i>	A	1.48	1.49	52.63	0.905	0.032	0.57	1.019	4.6
<i>Ateles</i>	<i>hybridus</i>	A	1.36		103.05	0.851	0.019		0.962	25
<i>Ateles</i>	<i>geoffroyi</i>	A	1.10	1.52	105.09	0.908	0.029		0.981	21
<i>Saimiri</i>	<i>sciureus</i>	A	1.25	1.41	24.14	0.898	0.036	-0.53	0.982	10.6
<i>Cebus</i>	<i>apella</i>	A	1.41	1.41	66.63	0.950	0.04	-0.22	0.991	12.6
<i>Saguinus</i>	<i>imperator</i>	A	1.00			1.003	0.022	1.71	1.027	6
<i>Saguinus</i>	<i>midas</i>	A	1.37	0.99		1.011	0.026	0.17	1.006	6
<i>Saguinus</i>	<i>oedipus</i>	A	0.95	1.00	9.76	1.018	0.016		1.022	4.6

Table 4.1 continued: Main sample variables (species-level). See Appendix 4.1 and 2.5 for data on males and females. A=arboreal; A/T=arboreal/terrestrial; T=terrestrial.

Genus	Species	Substrate	Body Dimorphism	Canine Dimorphism	Species ECV	Species 2D:4D	sd	Cohen's <i>d</i>	2D:4D Dimorphism	Group Size
<i>Leontopithecus</i>	<i>chrysomelas</i>	A	1.15			0.995	0.033	0	1.000	6.7
<i>Leontopithecus</i>	<i>rosalia</i>	A	1.04	1.18	12.83	0.985	0.021	0.56	1.017	5.8
<i>Callithrix</i>	<i>jacchus</i>	A	1.08	1.30	7.24	0.928	0.064	-0.02	0.999	5.6
<i>Callithrix</i>	<i>geoffroyi</i>	A	1.00			0.922	0.073	0.75	1.063	9

4.2.2.2.4: Other variables

Substrate measures for species were taken from Plavcan and van Schaik (1992). Total group size data were taken primarily from Smuts *et al.* (1987). These data were supplemented by estimates taken from Meijaard *et al.* 2010 for *Pongo pygmaeus*; Hall *et al.* 2006 for *Cercopithecus hamlyni*; Li *et al.* 2007 for *Trachypithecus francoisi*; van Roosmalen & Klein 1988 for *Ateles hybridus*; Fleagle 1998 for *Saguinus midas*; Cain 1998 for *Callithrix geoffroyi*.

4.2.3: Statistical methods

Data were first analysed using a non-phylogenetically (non-phy) controlled approach using a general linear model (GLM). Male and female species mean 2D:4D (dependent variable) were correlated with male and female target (predictor) variables (i.e., male 2D:4D and male canine size) while species mean 2D:4D were correlated against mean values for the target variables. Male, female and mean 2D:4D values were correlated in-turn with species dimorphism measures. Analyses of canine size and brain size (ECV) were also performed with body size as an additional predictor variable (see Shultz and Dunbar, 2010b). Relationships were analysed using both non-phylogenetic tests (i.e., General Linear Model; GLM) and PGLS analysis (Graften 1989). PGLS analysis was executed in 'R' (Ihaka & Gentleman 1996) using APE package (Analysis of Phylogenetics and Evolution) (Paradis *et al.* 2004) with code provided by R.P. Duncan. Pagel's λ was used to estimate the degree of phylogenetic autocorrelation within models (Pagel 1997; see Chapter 2). Phylogenetic trees were constructed using evolutionary relationships from published sources (Purvis 1995; Opazo *et al.* 2006; see Appendix 2.6 for phylogenetic trees).

Analyses were performed firstly using GLM for the whole sample (all species; males, female and species-level) and then for each super family; apes (Hominoidea), OWM (Cercopithecoidea) and NWM (Ceboidea) (males, female and species-level). GLM analysis indicates correlations between variables without controlling for phylogenetic relatedness between species. Analyses were then repeated using a phylogenetically controlled approach (PGLS). As evolutionary relationships must be controlled for in cross species analyses (Felsenstein 1985), the results of this study will focus on outputs from PGLS analysis (for full statistical output using GLM and PGLS; see Appendix 4.3). Main results, based on species mean values (PGLS), are tabulated in the results section below. The results of the remaining analyses are tabulated in Appendix 4.3.

Kolmogorov-Smirnov Tests were used to test skew in the variables. All of the variables were skewed (i.e., not normally distributed) except female 2D:4D ($p>0.01$) and canine size for males ($p>0.01$), females ($p>0.02$) and species ($p>0.02$). All variables were therefore log-transformed. Intra-class coefficient (ICC; McGraw & Wong 1996) was within range ($\sim p<0.01$) of those presented in Chapter 2 (section 2.6.2.2) and Chapter 3. High levels of phylogenetic autocorrelation expressed in significant Moran's I values were evident in all variables except sexual dimorphism in 2D:4D (expected=-0.02; observed=0.040; sd=0.03, $p=0.06$; see Appendix 4.4 for Moran's I for each variable). These results indicate that PGLS analysis was justified. Values in the text are PGLS unless otherwise stated. 2D:4D Significance was set to $p\leq 0.05$ unless stated otherwise. Substrate was not associated with 2D:4D in any of the analysis ($P>0.05$). F=variance result; p=probability; df=degrees of freedom.

4.3: Results

4.3.1: 2D:4D and body measurements

4.3.1.1: 2D:4D and body size

There were no significant associations between 2D:4D and body weight across the whole sample (Table 4.2). In apes, lower 2D:4D was significantly associated with higher body weight in female ($F_{1,6}=29.48$, $p<0.01$, $\lambda=0$). Within the ape clade males and females in the Hylobatidae have significantly higher 2D:4D ratios than those in the great apes (Hominidae) (males: $F_{1,6}=60.80$, $p<0.001$, $\lambda=0$; females: $F_{1,6}=43.43$, $p<0.001$, $\lambda=0$), but body weight did not significantly differ between the two groups (males: $F_{1,6}=3.61$, $p=0.11$, $\lambda=0$; females: $F_{1,6}=3.18$, $p=0.12$, $\lambda=0$; Fig. 4.5). In Gorilla males had a higher 2D:4D than females with is contrary to expected (Fig. 4.5). This pattern is not uncommon in the haplorhine data as can be seen from the positive Cohen's d values in Table 4.1.

There were no significant relationships between 2D:4D and body size in the OWM or the NWM (Table 4.2; Appendix 4.3).

4.3.1.2: Body size dimorphism

Across the whole sample there was no relationship between 2D:4D and body size dimorphism (Table 4.2) although a weak trend was apparent in the expected direction; 2D:4D decreased with increasing body dimorphism. Within super families relationships were only significant for male OWM (Cercopithecoidea) in which a *positive* relationship was found between male 2D:4D and body dimorphism; males had higher 2D:4D (inferred lower PAE), and higher body dimorphism than males in the Colobinae family (2D:4D: $F_{1,19}=8.74$, $p<0.01$, $\lambda=0$; Body dimorphism: $F_{1,19}=12.91$, $p<0.01$, $\lambda=0$).

Sample	Model		PGLS Analysis			
			F	p	df	λ
All	Species 2D:4D	Species body weight	2.49	0.12	42	0.99
All	Species 2D:4D	Body dimorphism	0.32	0.57	42	0.99
All	2D:4D dimorphism	Body dimorphism	0.32	0.58	42	0.00
Apes	Species 2D:4D	Species weight	4.04	0.09	6	1.00
Apes	Species 2D:4D	Body dimorphism	1.95	0.21	6	1.00
Apes	2D:4D dimorphism	Body dimorphism	0.06	0.82	6	0.00
OWM	Species 2D:4D	Species body weight	0.62	0.44	19	0.87
OWM	Species 2D:4D	Body dimorphism	3.04	0.10	19	0.63
OWM	2D:4D dimorphism	Body dimorphism	0.12	0.73	19	0.87
NWM	Species 2D:4D	Species body weight	0.04	0.84	13	0.91
NWM	Species 2D:4D	Body dimorphism	0.01	0.91	13	0.96
NWM	2D:4D dimorphism	Body dimorphism	0.18	0.68	13	1.00

Table 4.2: 2D:4D and body weight and body weight dimorphism in adults (PGLS). For a more detailed breakdown see Appendix 4.3.

4.3.1.3: Neonatal body size and dimorphism

Across the whole sample relationships between 2D:4D and neonatal body weight were in the expected direction; low 2D:4D was associated with higher neonatal weight but the correlation was not significant (Table 4.3). Low 2D:4D was also associated with higher neonatal dimorphism across the whole sample, but again, this was lost when phylogeny was controlled for (Table 4.3; see Appendix 4.3 for GLM results). There were no relationships between these variables within the OWM or NWM (Table 4.3).

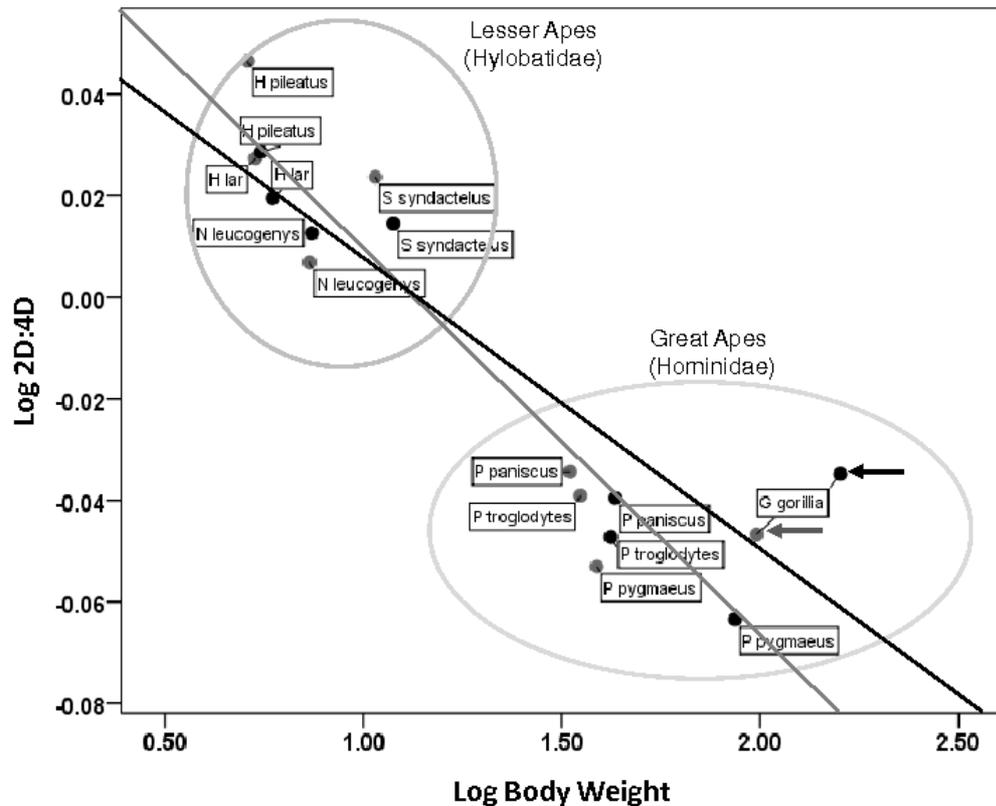


Figure 4.5: 2D:4D and body weight within apes. Gray dots are females ($R^2=0.87$); black dots are males ($R^2=0.82$). Gorilla 2D:4D ratios are higher in males than females, contrary to expectations (marked by an arrow).

Sample	Model	PGLS Analysis			
		F	p	df	λ
All	Species 2D:4D Neonatal dimorphism	0.40	0.53	29	0.99
All	2D:4D dimorphism Neonatal dimorphism	0.01	0.97	29	0.00
Apes	Species 2D:4D Neonatal dimorphism	0.41	0.54	10	0.72
Apes	2D:4D dimorphism Neonatal dimorphism	0.26	0.62	8	0.00
OWM	Species 2D:4D Neonatal dimorphism	3.21	0.10	10	0.91
OWM	2D:4D dimorphism Neonatal dimorphism	1.15	0.30	10	0.00
NWM	Species 2D:4D Neonatal dimorphism	0.17	0.68	7	1.00
NWM	2D:4D dimorphism Neonatal dimorphism	0.13	0.72	7	1.00

Table 4.3: 2D:4D and body weight and body weight dimorphism in neonates (PGLS). For a more detailed breakdown see Appendix 4.3.

4.3.2: 2D:4D and canine measurements

4.3.2.1: Canine size

Across the whole sample there was no relationship between 2D:4D and canine size (Table 4.4). Within super families correlations with canine size were only significant for male Old World monkeys; male 2D:4D increased (PAE decrease) with increasing male canine size ($F_{1,12}=4.90$, $p = 0.05$, $\lambda=0.42$). These patterns are contrary to predictions. Relationships remained positive after controlling for body weight (canine size: $F_{1,11}=5.31$, $p=0.04$; body weight: $F_{1,11}=0.69$, $p=0.46$, $\lambda=0.22$) and may be associated with high 2D:4D (and large canine size) in male *Mandillus leucophaeus* compared to other OWM. Male 2D:4D was also higher in males *M. leucophaeus* than in females signalled by a large size effect (Cohen's $d=1.05$).

There were no relationships between these variables within the Hominoidea or Ceboidea (Table 4.4; see Appendix 4.3 for full statistical output).

4.3.2.2: Canine size dimorphism

There were no significant relationships between 2D:4D and canine size dimorphism across the whole sample, although the trend was in the expected direction (Table 4.4; Fig. 4.6). Removing *M. leucophaeus* from the analysis improved the relationships ($F_{1,25}=3.40$, $p=0.07$, $\lambda=0.98$).

There was a significant relationship between dimorphism in 2D:4D and canine size dimorphism but this relationship was contrary to expectations ($F_{1,26}=5.55$, $p=0.03$, $\lambda=0$). When these relationships are analysed in the other super families, significance is only maintained in the OWM (Table 4.4). This relationship was opposite to predictions due to the effects of the *M. leucophaeus*. There were no relationships between 2D:4D and canine size dimorphism within the apes or NWM (Table 4.4).

Table 4.4: 2D:4D and canine size and canine size dimorphism (PGLS). For a more detailed breakdown see Appendix 4.3.

Sample	Model		PGLS Analysis					
			F	p	df	λ	F	p
All	Species 2D:4D	Species canine size	0.20	0.66	26	0.96		
All	Species 2D:4D	Species canine size + Body weight	0.15	0.70	25	0.98	2.38	0.135
All	Species 2D:4D	Canine dimorphism	1.66	0.21	26	0.97		
All	2D:4D dimorphism	Canine dimorphism	5.55	0.03	26	0.00		
Apes	Species 2D:4D	Species canine size	0.13	0.73	4	1.00		
Apes	Species 2D:4D	Species canine size + Body weight	0.05	0.83	3	1.00	0.20	0.25
Apes	Species 2D:4D	Canine dimorphism	3.50	0.13	4	1.00		
Apes	2D:4D dimorphism	Canine dimorphism	1.07	0.36	4	1.00		
OWM	Species 2D:4D	Species canine size	0.62	0.45	12	0.78		
OWM	Species 2D:4D	Species canine size + Body weight	0.51	0.41	11	0.77	0.02	0.87
OWM	Species 2D:4D	Canine dimorphism	0.89	0.36	12	0.81		
OWM	2D:4D dimorphism	Canine dimorphism	5.05	0.04	12	0.17		
NWM	Species 2D:4D	Species canine size	0.06	0.81	6	0.90		
NWM	Species 2D:4D	Species canine size + Body weight	0.00	0.98	5	0.90	0.13	0.724
NWM	Species 2D:4D	Canine dimorphism	1.50	0.27	6	1.00		
NWM	2D:4D dimorphism	Canine dimorphism	0.47	0.52	6	1.00		

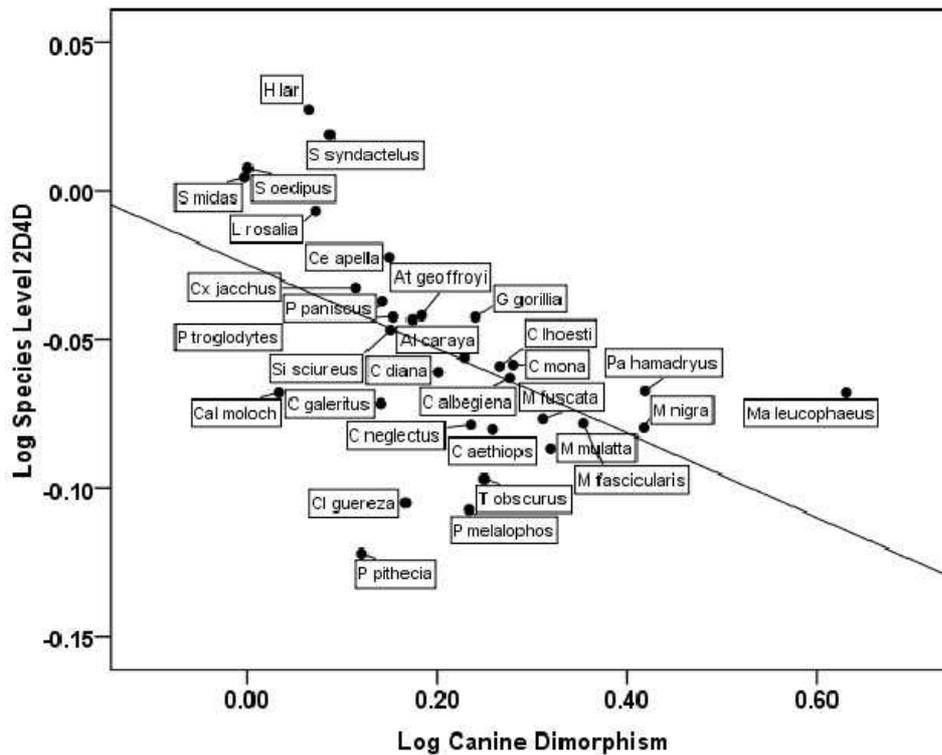


Figure 4.6: 2D:4D and canine dimorphism across the whole sample ($p=0.21$; $R^2=0.27$). Relationships improved after removing *Mandrillus leucophaeus* ($p=0.07$; $R^2=0.35$)

4.3.3: 2D:4D and brain measurements

4.3.3.1: Brain size (endocranial volume; ECV)

Relationships between 2D:4D and ECV were not significant across the whole sample, as were most analyses within super families (Table 4.5). Within the apes, the Hylobatidae had significantly higher 2D:4D ratios and significantly lower ECV than great apes (2D:4D: $F_{1,5}=100.13$, $p<0.001$, $\lambda=0$; ECV: $F_{1,5}=86.91$, $p<0.001$, $\lambda=0$) and 2D:4D was significantly negatively related ECV, but significance was lost once body size was controlled for (Table 4.5). No relationships were found in these variables when OWM and NWM were analysed separately from apes (ECV: $F_{1,22}=0.34$, $p=0.66$, $\lambda=0.94$) or when apes and OWM were analysed together (ECV: $F_{1,20}=0.07$, $p=0.38$, $\lambda=1$).

Table 4.5: 2D:4D and endocranial volume (ECV) and brain size dimorphism (PGLS). For a more detailed breakdown see Appendix 4.3.

Sample	Model		PGLS Analysis					
			F	p	df	λ	F	p
All	Species 2D:4D	ECV	0.21	0.64	30	0.99		
All	Species 2D:4D	ECV + body weight	1.39	0.24	29	1.00	1.27	0.269
All	Species 2D:4D	ECV dimorphism	0.13	0.72	30	0.99		
All	2D:4D dimorphism	ECV dimorphism	3.23	0.08	30	0.00		
Apes	Species 2D:4D	ECV	68.98	<0.001	5	0.00		
Apes	Species 2D:4D	ECV + body weight	3.55	0.13	4	0.00	0.17	0.702
Apes	Species 2D:4D	ECV dimorphism	0.56	0.49	5	1.00		
Apes	2D:4D dimorphism	ECV dimorphism	0.16	0.70	5	0.00		
OWM	Species 2D:4D	ECV	0.00	0.99	14	0.83		
OWM	Species 2D:4D	ECV + body weight	0.46	0.51	13	0.74	0.57	0.4622
OWM	Species 2D:4D	ECV dimorphism	0.28	0.11	14	0.84		
OWM	2D:4D dimorphism	ECV dimorphism	4.64	0.07	14	0.00		
NWM	Species 2D:4D	ECV	0.57	0.47	7	1.00		
NWM	Species 2D:4D	ECV + body weight	0.30	0.60	6	1.00	0.02	0.882
NWM	Species 2D:4D	ECV dimorphism	0.10	0.76	7	0.79		
NWM	2D:4D dimorphism	ECV dimorphism	1.45	0.27	7	0.32		

4.3.3.2: Brain size dimorphism

There were no relationships between 2D:4D and brain size dimorphism (Table 4.5; also see Appendix 4.3). Analysing NWM and OWM together with apes removed, relationships remained non-significant: $F_{1,23}=0.21$, $p=0.64$, $\lambda=0.86$) and analysing ape and OWM together with NWM removed (ECV: $F_{1,21}=0.01$, $p=0.99$, $\lambda=1$).

4.3.4: 2D:4D and group size.

There were no associations between 2D:4D and group size across the sample although the trend was in the predicted direction; 2D:4D decreased with increasing group size (Table 4.6). There were no significant relationships within super families (Table 4.6) or when monkeys were analysed with apes excluded ($F_{1,23}=0.35$, $p=0.56$, $\lambda=0.87$).

Sample	Model		PGLS Analysis			
			F	p	df	λ
All	Species 2D:4D	Group size	0.10	0.75	42	0.99
All	2D:4D dimorphism	Group size	1.81	0.19	42	0.00
Apes	Species 2D:4D	Group size	0.09	0.78	6	1.00
Apes	2D:4D dimorphism	Group size	0.01	0.89	6	0.00
OWM	Species 2D:4D	Group size	0.52	0.48	19	0.84
OWM	2D:4D dimorphism	Group size	2.81	0.11	19	0.00
NWM	Species 2D:4D	Group size	0.24	0.63	7	1.00
NWM	2D:4D dimorphism	Group size	0.11	0.73	13	0.40

Table 4.6: 2D:4D and group size (PGLS). Also see Appendix 4.3.

4.4. Discussion

Relationships between 2D:4D, body and canine size and body and canine dimorphism were not significant after controlling for phylogenetic relatedness but exhibited trends in the predicted direction; lower ratios were associated with higher levels of dimorphism.

Relationships were stronger for canine size dimorphism than body size dimorphism. Lower 2D:4D ratios were associated with bigger groups but not significantly so. There were no

associations with brain size measures. The results suggest that 2D:4D is a poor predictor of the effects on PAE on programming sexually selected anatomical characteristics. These findings are in stark contrast to strong relationships between 2D:4D and sexual selection indexed by social systems and competition levels in Chapter 3.

Weak relationships between digit ratio and body measures might be expected as 2D:4D is largely fixed *in utero* and target variables are commonly obtained on adult individuals. Body size changes radically through growth and growth trajectories are influenced by many postnatal factors (e.g., sociality, diet, latitude, climate). In *Cercopithecus aethiops* variation in body mass dimorphism across wild Kenyan populations was found to be associated with changes in female body mass responding to increased food availability, latitude and rainfall (Turner *et al.* 1997). These effects were only evident in females and no differences in males could be detected. In humans height varies between populations in accordance with sexual selection (see Kanazawa & Novak 2005 for a review; but see Gray & Wolfe 1980) but is also impacted by environmental factors (Gray & Wolfe 1980; Ruff 1994). For example, low protein availability within some societies has been shown to have a bigger impact on male height (stunts growth) than female height (Gray & Wolfe 1980). These environmental effects on sex-linked growth trajectories can alter sexually dimorphic height patterns across populations. Relationships between 2D:4D and stature in humans are weak (Lippa 2003; Barut *et al.* 2008) or non-significant (Rahman *et al.* 2005; Hönekopp & Watson 2010) and body weight shows a similar relationship (Fink *et al.* 2003; Rahman *et al.* 2005; but see Danborn *et al.* 2008). Poor correlations between 2D:4D and body size in humans and other primates may reflect the impact of postnatal environmental variability growth trajectories (e.g., Turner *et al.* 1997; Diverse Populations Collaborative Group 2005).

If PAE does impact growth trajectories we might expect to see stronger relationships between 2D:4D and neonatal size and dimorphism, as extraneous postnatal influences (e.g., pubertal growth, postnatal environmental factors) can be discounted a potential confound. Sexual dimorphism in neonatal body size is evident in most primates and these measures correlate with adult levels of body dimorphism across species (Smith & Leigh 1998). Variation in birth weight is largely governed by the intra-uterine environment (Penrose 1952) and prenatal androgens have been proposed as possible candidates for greater male size at birth in primates (Smith & Leigh 1998). In humans sex differences in body size and 2D:4D have been detected as early as 8-12 weeks (Bukowski *et al.* 2007; Malas *et al.* 2006; Galis *et al.* 2010). This corresponds to a sharp rise in foetal testosterone as the testes begin to function (McIntyre 2006). In a sample of Nigerian (human) newborns, lower 2D:4D (inferred higher PAE) was associated with higher birth weight in males, while high 2D:4D

was associated with higher birth weight in females (Danborno *et al.* 2010). No associations with birth weight were found in a smaller sample from the UK (Ronalds *et al.* 2002), although decreasing size (crown-to-heel length) was associated with increased 2D:4D (PAE decreased) in males. Birth weight is an important indicator of subsequent health status in adult humans; lower birth weight and size dimensions are strongly associated with chronic disease in later life (Loos *et al.* 2001; 2002; Fowden & Forhead 2004). The health status of an individual has consequences for evolutionary fitness as healthy males are more able to compete for females and are more likely to be chosen as mates (Rhodes *et al.* 2003; Roberts & Little 2007). This is also likely to be the case for other primates. Across haplorhines 2D:4D and neonatal body size and size dimorphism did exhibit weak trend in the expected direction; low 2D:4D was associated with higher neonatal weight and size dimorphism. However these relationships were not significant which suggests that prenatal growth factors, other than PAE, have already influenced body size before birth (e.g., insulin-like growth factor; Bernstein *et al.* 1997; Loos *et al.* 2001; 2002). A recent study in humans reported no differences in birth weight in neonates subsequently diagnosed with genetic and medical abnormalities (e.g., congenital adrenal hyperplasia) linked to extreme levels (high and low) of prenatal androgens (Miles *et al.* 2010).

Although PAE may not significantly influence birth weight in humans (Miles *et al.* 2010; clinical sample) this may not be the case for species with higher levels of sexual selection. Evidence from 2D:4D alludes to the possibility that PAE may be implicated in programming growth trajectories in species with higher levels sexual selection because as 2D:4D decreases (PAE increases) body dimorphism increases. However, non significant results suggest that these relationships are weak and may not be straightforward (Bernstein *et al.* 2007; 2008). Adult size and dimorphism in primates can be the product of different growth processes, even between closely related species (Leigh 1992). For example, levels of body size dimorphism in bonobos and chimpanzees are similar (Lindenfors & Tullberg 1998), but developmental trajectories differ. In bonobos (*Pan paniscus*) males extend growth in relation to females. In chimpanzees, development is delayed in females, but is accelerated in males (Leigh 1992; Leigh & Shea 1995). These differences may arise in chimpanzees as a consequence of feeding competition in females (delaying maturity) and male involvement in coalitions associated with territorial patrols (speeding maturity; Reno *et al.* 2003). 2D:4D also differs between these two species with bonobos having higher ratios (closer to human values), than chimpanzees (McIntyre *et al.* 2009; Fig. 4.1). Chimpanzees also exhibit an increase in sexual dimorphism in 2D:4D from around 7-8 years of age; female ratios increased with age while male ratios decreased. These changes coincide with the sharp increase in testosterone in male chimpanzees and the onset of puberty in both males and

females at around 8 years of age (Martin *et al.* 1977; Goodall 1986). This suggests that circulating sex hormones may influence digit morphology and in adult chimpanzees. Bonobos, in contrast, showed stability in sex differences in 2D:4D across the same age range and in this respect they follow the human pattern (Manning 2002a; Manning *et al.* 2004a). Sex-linked age-related changes in adult 2D:4D in some species (e.g., chimpanzees), but not others (e.g., bonobos and humans) will weaken cross-species correlations with target variables (see below). This suggests that 2D:4D may be a better reflector of PAE in some species but not in others.

Results indicate stronger relationships between 2D:4D and canine dimorphism (but not canine size) than between 2D:4D and body size measures. They also suggest that PAE may be more influential in programming male and female differences in canine development in species with higher levels of sexual selection because lower 2D:4D (inferred higher PAE) is associated with higher male-male competition and promiscuous social systems (see Chapter 3). As the formation of the permanent dentition begins *in utero* (Harila-Kaera 2001) we might expect to see correlations between 2D:4D and canine measures. Relationships were in the predicted direction and approached significance after the removal of an outlier (*M. leucophaeus*); low 2D:4D (inferred higher PAE) was associated with higher measures of canine dimorphism (Fig. 4.6). However, genetic influences cannot be discounted. In a study of same-sex (SS) and opposite-sex (OS) twins, females with a male co-twin had significantly larger teeth than females with a SS twin (Dempsey *et al.* 1999). This effect was attributed hormonal transfer of androgens from the male to the female twin, and implies that PAE are implicated in early sexual dimorphism of the human dentition. However, canine size exhibited the least size change in the OS twin (Dempsey *et al.* 1999), which may be indicative of the high genetic constraints on canine size (Harila-Kaera 2001; but see Kaushal 2007). Evidence from human studies may not be a good comparative model for other haplorhines, especially those species with high levels of sexual selection. Schwartz and Dean (2001) demonstrated that although canine crown formation times between males and females did not differ in humans, they did significantly differed in promiscuous great apes.

Brain size in primates is positively related to higher levels of sexual selection and group size (Sawaguchi 1996; Dunbar 1998; Shultz & Dunbar 2007). As 2D:4D also co-varies with social system and competitive behaviour (Chapter 3) we might expect to find that species with lower 2D:4D (inferred higher PAE) that live in larger groups to have bigger brains. This was not found. Across the whole sample relationships between 2D:4D and ECV and brain size dimorphism (based on ECV measures) were not significant after body size was controlled for.

Prenatal androgens influence the internal architecture of the brain (McClusky & Naftolin 1981; Arnold & Gorski 1984) and, in humans PAE have been linked to social development (e.g., Baron-Cohen 2002; Knickmeyer *et al.* 2005) and are reflected in 2D:4D (e.g., Manning *et al.* 2001; 2010). These sex-effects may be linked to androgenic effects on neural pathways linked to social bonding and social reward (van Honk *et al.* 2011; van Wingen *et al.* 2010; Rilling *et al.* in press). In non-human primates, operational and ethical difficulties preclude investigations of size differences in brain structures between males and females (see Lindenfors 2005; Lindenfors *et al.* 2007), however there is good evidence to show that sexual selection exerts a strong selective force on neural tissue producing sex-linked differences in brain architecture (e.g., Jacobs 1996; Gur *et al.* 1999; Lindenfors *et al.* 2007; Yan *et al.* 2010). Furthermore, variation in 2D:4D in females has been shown to be associated with masculinisation in sub-regions of hippocampus; low 2D:4D (inferred high PAE) was linked to male-typical smaller, left-side volumes in the posterior hippocampus (Kallai *et al.* 2005). However no differences were found in other asymmetric structures such as the amygdalae and total hippocampal formation, which are known to be replete with estrogen and androgen receptors (Pomerantz *et al.* 1985; O'Keefe *et al.* 1993; Cooke *et al.* 2003). This evidence suggests that, while 2D:4D may not be informative about the evolutionary development of total brain size or brain size dimorphism; it may be insightful about the impact of sex hormones on brain architecture, although these patterns are likely to be complex and difficult to investigate at the species-level.

Sampling bias appears to impact correlations with 2D:4D. In *G. gorilla* and *M. leucophaeus* males had higher mean 2D:4D ratios than females. This is contrary to the expected pattern. This pattern (higher ratios in males than females) was also evident in several other species in the dataset (signalled by positive Cohen's *d* values; Table 4.1) and has also been reported in a small group of captive guinea baboons (*Papio papio*; Roney *et al.* 2004). In humans there is considerable overlap in 2D:4D values between the sexes, but on average male digit ratio is lower than female digit ratio within populations (Manning 2002a; Mills 2002). How can 2D:4D ratios be higher in males than females when males are exposed to higher PAE? Circulating androgens (e.g., testosterone) vary within and between primate species (Coe *et al.* 1992) and are highly responsive to environmental factors (social and biological; e.g., Whitten & Turner 2004; Schulz *et al.* 2009). Although PAE are likely to vary less than circulating testosterone in adults due to maternal buffering of the intra-uterine environment, it is likely that taxonomic differences in prenatal sex hormones will still be evident. Intra-specific levels of PAE are additionally influenced at a local level by maternal effects which contribute to foetal programming (Mousseau & Fox 1998; Kaiser & Sachser 2009).

Foetal programming is a physiological process that prepares the foetus for extra-uterine life. Cellular-level changes occur as a response to subtle variations in placental function which alters in accordance to external stimuli such as dietary insufficiency, disease and stress (Phillips 2002; Fowden & Forhead 2009; Matthews and Philips 2010). Females are highly responsive to changes in nutrient availability and ecological variables such as climate, latitude and rainfall (Turner *et al.* 1997) and these factors are also likely to impact pregnant females. Changes in nutrition and health are known to influence placental function and this gives rise to foetal programming (see Phillips 2002; Fowden & Forhead 2004; 2009; Fowden *et al.* 2008). If local environmental effects induce changes in PAE via maternal effects on foetal programming then 2D:4D may differ between populations. Thus sampling of males and females from different populations could skew sex-differences in 2D:4D.

The social status of an individual may also impact PAE via maternal effects. For example in humans, males and females with lower 2D:4D (inferred higher PAE) consider themselves as more dominant and exhibit more aggression than males and females with higher 2D:4D (Benderlioglu & Nelson 2004; Bailey & Hurd 2004; Manning & Fink 2008). PAE has been inferred to be higher in foetuses of more socially dominant female hyenas based on measurements of maternal faecal testosterone (Dloniak *et al.* 2006; but see East *et al.* 2009). Faecal testosterone levels positively correlate with maternal rank and with levels of aggression in the hyena pups (Dloniak *et al.* 2006). If dominance is linked to variation in PAE within populations then a skewed pattern of sexual dimorphism in 2D:4D may arise if a dataset samples 2D:4D from many submissive males (high 2D:4D) and many dominant females (low 2D:4D). Ecological and dominance effects may not be mutually exclusive as more dominant individuals have priority access to food and are therefore likely to grow faster and be heavier than lower status individuals (Zehr *et al.* 2005).

Weaknesses in correlations with 2D:4D may also arise as a consequence of correlations between data derived from captive and from wild animals. 2D:4D data were predominantly taken from captive primates, while measurements of anatomical characteristics were mostly taken from wild-caught individuals (published sources; Lindenfors & Tullberg 1998; Smith & Jungers 1997; Thoren *et al.* 2006; Isler *et al.* 2008). Comparatively high nutritional intake and low energy expenditure of captive animals compared to wild animals may lead to differences in growth and development (see Smith & Jungers 1997, p 526), although Isler *et al.* (2008) found no significant differences in ECV between individuals that were captive bred and wild caught in their sample and I could not detect significant differences in 2D:4D ratios of captive female rhesus macaques (*Macaca mulatta*) and free-living population. It is

possible, however, that rearing conditions impact 2D:4D in some captive-born species and this serves to weaken relationships with measures of anatomical characters measured in wild-born individual.

4.4.1: Summary

Sexual selection has, without doubt, had a significant influence on the evolution of sexually selected body size in haplorhines (Plavcan 2001; Shultz & Dunbar 2007). Sexual dimorphism in body size is impacted by a myriad of intrinsic and extrinsic factors that serve to increase variation in this characters (e.g., Turner *et al.* 1997; Harila-Kaera 2001; Leigh *et al.* 2008). A key finding from reviewing ontogenetic studies is that although body, brain and canine size may correlate (to a greater or lesser degree) across primate species, the development of these structures can be de-coupled from each other (Clegg & Aiello 1999; Schwartz & Dean 2001; Herculano-Houzel 2009). It appears that body size is much freer to vary with postnatal factors than brain or canine size (Smith 1989; Martin *et al.* 1994; Schwartz & Dean 2001; Herculano-Houzel 2009) and, as such, body size and body dimorphism is a much weaker proxy of sexual selection (Plavcan 2001).

In conclusion, the predictions of this study were not met. That fact that relationships between 2D:4D and target variables were mostly in the expected direction, even though statistic parameters were achieved, suggests that some of these characters are probably programmed by PAE within the same critical prenatal phases. However, these effects are obscured over growth due to radical changes in allometry of anatomical characters. These findings are in line with weak or non significant relationships between 2D:4D and anatomical variables in humans. In the light of these results, and those of the Chapter 3, it can be concluded that 2D:4D is not a good predictor of PAE on sexually selected anatomical traits but may be more informative about PAE on the brain, specifically in programming core species-level differences in neural structures linked to potentiating social behaviour.

Chapter 5

2D:4D, female dominance rank and heritability in rhesus macaques¹²

5.1: Introduction

Cross-species studies in haplorhines have shown that 2D:4D co-varies with social system and intra-sexual competition (Chapter 3), but not anatomical traits linked to sexual selection (Chapter 4). This chapter investigates relationships between 2D:4D and social behaviour by looking to see if relationships exist at the intra-specific level in a haplorhine primate species. Specifically, a case study is presented investigating relationships between 2D:4D and social dominance rank in female rhesus macaques (*Macaca mulatta*). It also calculates heritability levels of 2D:4D by analysing variation in mother macaques and their infant offspring.

5.1.1: 2D:4D: An anatomical marker for prenatal androgens effects

In humans 2D:4D is sexually dimorphic from nine weeks of prenatal life (Malas *et al.* 2006; also see Galis *et al.* 2010). Lower 2D:4D ratios are inferred to be associated with higher PAE and 2D:4D tends to be lower in males than females within a population (Manning 2002a; McIntyre *et al.* 2005). 2D:4D has been shown to correlate negatively with direct and indirect measures of prenatal androgens (Manning *et al.* 2007a). The mechanisms underlying these relationships are not clear, but are believed to be linked to common developmental pathways between the fingers and reproductive system. The distal limb buds (digits) and the genital bud are controlled by the same groups of phylogenetically conserved *HOX* genes (Zákány *et al.* 1997; Kondo *et al.* 1997; Montavon *et al.* 2008) and *HOX* gene transcription appears to be sensitive to sex hormones (Soto & Sonnenschein 1999; Daftery & Taylor 2006). As *HOX* genes are strongly phylogenetically conserved within and between taxonomic groups (Zákány *et al.* 1997), it has been proposed that genetic association between 2D:4D and PAE should be common across four-limbed vertebrates (Manning 2002a, p 17).

¹² Citations for this chapter: Nelson, E., Hoffman, C.L., Gerald, M.S. & Shultz, S. 2010. Finger length ratios (2D:4D) and dominance rank in female rhesus macaques from Cayo Santiago. *Behavioral Ecology and Sociobiology*, 64:1001-1009.

Nelson, E. & Voracek, M. 2010. Heritability of digit ratio (2D:4D) in rhesus macaques (*Macaca mulatta*). *Primates*. 51:1-5.

Studies of heritability of digit ratios support this contention. Twin studies of 2D:4D have found high narrow-sense (genetic effects on phenotypic variance) heritability for 2D:4D ($h^2 = 50-80\%$) and shared environmental influences (non-genetic effects) on 2D:4D to be small (Paul *et al.* 2006b; Voracek & Dressler 2007c; Gobrogge *et al.* 2008; Medland & Loehlin 2008). Similar results have been found for familial relationships in humans, such as mother-offspring and sibling-sibling comparisons, which yield heritability values of between 41% and 69% (Ramesh & Murty 1977; Marshall 2000; Manning 2002a; Voracek & Dressler 2009). High heritability levels have also been calculated for zebra finches ($h^2 = 70-80\%$; Forstmeier 2005; Forstmeier *et al.* 2008). The moderate to high heritability levels quoted in these studies suggest that non-shared environmental influences on 2D:4D (such as maternal effects and epigenetic factors) are low to moderate (see Gobrogge *et al.* 2008). Additionally, there is some evidence to suggest that birth order and sex of older siblings may influence PAE/2D:4D relationships (Williams *et al.* 2000; Saino *et al.* 2006), possibly via interactions between maternal physiology and parity, and this might impact on familial resemblance (Williams *et al.* 2000; James 2001; Saino *et al.* 2006; also see Fowden & Forhead 2009, p 617). Comparisons of 2D:4D between siblings and parent-offspring dyads in both humans and non-human animals (zebra finches) also show similarities and suggest that heritability of 2D:4D may also generalise across taxa (see Voracek & Dressler 2009). Genetic estimates combined with indirect links between digit ratios and PAE (outlined above), have led to 2D:4D being widely employed to study androgenic-programming effects on shaping human sex-linked traits and behaviours (Voracek & Loibl 2009).

In humans, low 2D:4D ratios (inferred high PAE) are associated with dominance-related behaviours in both sexes. Low 2D:4D individuals tend to be more competitive (Manning & Taylor 2001; Manning *et al.* 2007b), risk-taking (Schwerdtfeger *et al.* 2010; Stenstrom *et al.* 2011), show higher physical strength (Fink *et al.* 2006c; but see van Anders 2007) and more aggression compared to individuals with higher 2D:4D (Bailey & Hurd 2004; Benderlioglu & Nelson 2004; Millet & Dewitte 2007; 2009; Voracek & Schicker 2010.). In addition, low 2D:4D individuals exhibit higher drives for social status (Millet & Dewitte 2008; also see Coates *et al.* 2009), rate themselves as more dominant (Manning & Fink 2008) and are perceived as more dominant than individuals with higher 2D:4D ratios (Neave *et al.* 2003). In contrast to traits linked with low 2D:4D (inferred high PAE), high ratios (inferred low PAE) have been associated with emotional sensitivity in children (Fink *et al.* 2007; Williams *et al.* 2003). Thus 2D:4D appears to reflect the influence of PAE on social development, and particularly on behaviours linked to intra-sexual competition and sexual selection in humans (see Fink *et al.* 2006c).

One approach to understanding the physiological interplay between genetic and gestational effects is to compare 2D:4D within an evolutionary framework. As the main body of 2D:4D research is in humans, a framework considering species closely related to humans would be the most informative. Haplorhine primates offer the best comparative model as they reside within the same suborder as humans and will therefore have more similar biological profiles to humans than more distantly related species such as lizards or birds. Rhesus macaques (*Macaca mulatta*; family Cercopithecidae) are a particularly well studied primate species that show similarities in development to humans (Wallen 1996; 2005; Thornton *et al.* 2009). The Caribbean Primate Research Centre (CPRC) manages a free-ranging colony of macaques on a small island (15.2 ha) located 1 km off Puerto Rico's south-eastern coast. The CPRC has maintained detailed socio-demographic records on the macaque colony since the 1940s. This information has facilitated decades of macaque research (see Rawlings & Kessler 1983; Maestripietri 2007). Annual trapping of the animals make it possible to investigate variables such as 2D:4D, female dominance rank and familial resemblance of traits (a measure of genetic heritability).

5.1.2: Female dominance rank in rhesus macaques: A case for prenatal androgens?

Rhesus macaques exhibit competitive behaviours at all levels of the social hierarchy (Maestripietri 2007). Females macaques exhibit strong matrilineal bonds that determine dominance relationships within the group (Thierry *et al.* 2000). A female's position within the hierarchy is established during the juvenile period. Dominance ranks are passed from mother to daughter ('inherited') with younger sisters usually outranking older sisters (Datta 1988; Walters & Seyfarth 1987). Rank acquisition occurs through a gradual process, during which support is provided by the mother and other close kin (Chapais 1992; 2004; Datta 1988; Holekamp & Smale 1991). A female's rank therefore becomes established as a result of her own experiences, by the reactions of others that interact with her and by recognition of her status by other members of the group. These socio-behavioural mechanisms of dominance rank inheritance are widely acknowledged and well understood (Chapais 2004).

Although social processes are the primary mechanism of dominance rank inheritance, there is some evidence from a number of species to suggest that prenatal androgens may play a role in the ontogeny and maintenance of dominance behaviours in many taxa. For example, exposure to high prenatal androgens has been shown to masculinise social behaviour in female rhesus macaques and humans (e.g., Wallen 1996; Thornton *et al.* 2009; Hall *et al.* 2004; Kaiser & Sachser 2005). In humans, sex steroids vary with birth order (Bernstein *et al.*

1986) and may be implicated in competition between siblings (see Saino *et al.* 2006). In some bird species, yolk androgens (which are allocated by the mother) change with egg laying order (Gil *et al.* 2004) and appear to modify competitive behaviour in offspring (Groothuis *et al.* 2005). In canaries (*Serinus canaria*), yolk androgens have been shown to correlate positively with the social rank of siblings (Schwabl 1993).

The ability to confer signals about the extra-uterine world to a developing foetus, via maternal effects, is adaptive in that it physiologically prepares the individual for life in that environment (Mousseau & Fox 1998). Maternal effects, therefore, vary according to environmental conditions, but the effects on the foetus can be sex-specific and transmitted across generations (e.g., Kaiser & Sachser 2005; Champagne & Curley 2005; Matthews & Phillips 2010). Studies have consistently shown that exposing pregnant mammals to specific stressors (i.e., overcrowding or unfamiliar conspecifics) masculinises daughters and feminizes sons (Wallen & Baum 2002; Kaiser & Sachser 2005). More recently the research focus has broadened to understand how maternal effects in mammals adapt offspring under normative or non-adverse conditions (Kaiser & Sachser 2005; 2009).

In healthy human individuals, differences in prenatal androgen (assayed from amniotic fluid samples) appear to alter behavioural developmental pathways (Knickmeyer & Baron-Cohen 2006). However, it is not known the degree to which these effects on the foetus are genetic (e.g., androgens secreted by the foetus itself), environmental (e.g., maternal effects), or due to epigenetic combinations of these influences (Champagne & Curley 2005). Nevertheless, these effects may be manifested in the form of different physiologically induced predispositions (Wallen 1996; 2005). The social landscape then acts to adapt these differences making some behavioural tendencies more prone to develop than others (Wallen 1996). The interaction between physiological and social learning processes is adaptive in that it provides a means of fine-tuning behaviour according to social and physical variations in the environment in which the individual is developing (Maestripieri 2003 Champagne & Curley 2005; Kaiser & Sachser 2005).

Research on wild spotted hyenas (*Crocuta crocuta*) suggests a link between prenatal androgens and the transfer of dominance behaviour (Dloniak *et al.* 2006). Spotted hyenas have social systems and mechanisms of rank inheritance that resemble those of cercopithecine primates, particularly the *Macaca* genus (Engh *et al.* 2000; Holekamp & Smale 1991). In female hyenas, levels of faecal androgens late in pregnancy are positively related to dominance rank (Dloniak *et al.* 2004). Offspring of mothers with higher levels of faecal androgens are more aggressive and display higher rates of mounting behaviour than

pups of mothers with lower faecal androgens (Dloniak *et al.* 2006). The inference is that the level of prenatal androgens a pup experiences is, to some extent, governed by maternal effects. Exposure to high PAE primes aggressive behaviour in young pups, such that pups of higher-ranking mothers are predisposed to be more confrontational, and therefore, be more adapted to maintaining their socially ‘inherited’ dominant status. More recently it has been shown that, amongst orphaned spotted hyena pups adopted by other females, the dominance rank of the fostered animal was determined by the rank of the surrogate mother (and her social support), not by the rank of the genetic mother (East *et al.* 2009). It is likely, however, that the early life experiences of an individual (i.e., the style of mothering an infant receives as well as the foster mother’s dominance rank within the group) will act to modify and adapt prenatally inherited predispositions (see Maestripieri 2003; 2004).

Taken together this evidence from social studies suggests that offspring of high-ranking females are permitted to be more dominant because they inherit their mother’s dominance rank, and the biological data indicate that early exposure to high androgens from higher ranking mothers predispose infants to be more confrontational. Investigating heritability 2D:4D and comparing these correlations between 2D:4D and social behaviour may allow us to understand nature/nurture effects. If female dominance rank is ‘inherited’ only through socio-physiological effects occurring *after* birth in this species, we might expect to see no relationship with the PAE biomarker (2D:4D). If female dominance rank is influenced by genetic effects of PAE on predispositions, then higher heritability of 2D:4D should be seen.

5.1.3: Aims of the study

The aims of the study are:

- 1) To investigate relationships between 2D:4D and dominance rank in female rhesus macaques (Part I).
- 2) To use a family-resemblance approach to estimate the heritability of 2D:4D in mother-offspring dyads in the same population (Part II).

5.2: Materials and methods

5.2.1: Subjects

Data were collected during the 2008 trapping season on Cayo Santiago. The island's free-ranging rhesus macaque colony was established in 1938, and the present-day population (approximately 900 individuals) is directly descended from the founder group, originating from the Indian subcontinent (Rawlings & Kessler 1986). Genetic and behavioural analyses indicate no significant inbreeding (McMillan & Duggleby 1981; Mason & Perry 1993; Charpentier *et al.* 2007), although it is acknowledged that female dispersal patterns might potentially reduce genetic diversity (Chepko-Sade & Sade 1979) which may impact on heritability factors.

Measurements from 60 adult females and 25 of their infants (25 mother-infant pairs; 8 female and 17 male) were collected over a four-week period between January and February. The females in this study were primarily being sampled for a larger, unrelated study. Consequently, this sample size was restricted to those females being sedated for that study so I could not sample all adult females and their infants within a matriline.

Within the sample for this study the age of adult females varied between four and 24 years of age, and infants between one and four months. Individuals came from Groups F, R, and S. Females were sampled from two matriline from social group F (065 and 004) and from social group R (116r and DM) and from one matriline from social group S (116s) (see Table 5.1). A matriline is defined as a female kin lineage that can be traced back to the original matriarch of the groups. The matriarchs of groups F and R were born in the 1950s, and the matriarch of group S can be traced to the 1980s, when matriline 116 (originally residing in social group R) split into two lineages, the smaller of which moved out of R to form a new social group; group S (116s).

CRPC staff captured the monkeys in nets or by hand between 0830 hours and 1200 hours inside feeding corrals. Individuals were transferred immediately to standard cages and were held in the cages overnight. Each monkey had access to water and monkey chow during overnight housing, and all infants were kept with their mothers. The following morning, females and their offspring were sedated with ketamine. Approximately 5-10 minutes after ketamine injection, morphometric measures were taken. Following measurement, trained veterinary staff monitored monkeys every 10 minutes until they had recovered completely¹³.

¹³ All data were collected in accordance with the National Institutes of Health Guide for the Care and Use of Laboratory Animals. The study was approved by the School of Archaeology, Classics and Egyptology's Ethics Committee (University of Liverpool, UK) and the Institutional Animal Care and Use Committee (IACUC) of the University of Puerto Rico, Medical Sciences Campus.

Table 5.1: Social group and matriline variables in Part I (adult females). Figures in parentheses represent total number of adult females in the matriline.

Social Group	Rank	n	Mean 2D:4D	sd	Age	sd	Weight (Kg)	sd	Matriline	Rank	n	Mean 2D:4D	sd	Age	sd	Weight (Kg)	sd
F	High	11	0.828	0.03	15.6	5.2	9.6	0.6	065	High	6 (42)	0.838	0.03	16.4	5.2	9.3	2.1
									004	Low	5 (38)	0.824	0.03	14.7	5.6	9.9	2.3
R	Middle	35	0.826	0.04	11.5	5.7	8.5	1.8	116r	Middle	6 (11)	0.833	0.04	10.1	5.3	8.6	1.3
									DM	Low	29 (61)	0.822	0.04	11.9	5.7	8.5	1.9
S	Low	14	0.832	0.04	14.6	4.8	7.8	1.8	116s	Low	14 (31)	0.832	0.04	14.6	4.8	7.8	1.8

5.2.2: Measurement procedures

Once fully sedated, individuals were placed on a weigh-sling (Detecto™) and weighed to the nearest 0.1 Kg. The 2D and 4D of each hand were then measured, with measurements taken from the proximal crease at the base of the digit (crease closest to the palm) to the tip of the extended digit without compressing the soft-tissues of the fingertip. Procedure follows (as near as possible) that of Manning (2002a). All digit measurements were taken twice (to the nearest 0.01mm), using a vernier digital calliper (Guo Gen™); the digits of the left hand were measured (2D, then 4D) first, and then the digits of the right hand (2D, then 4D) were measured. The second set of measurements was taken immediately after the first, using the same procedure. During the measurement procedure, the hands of most of the sedated adult monkeys remained relaxed, but in the few instances that female's hands became tonic. In these cases an assistant gently opened the hand and held the digits in extension to facilitate length measurements. Assistance was also provided to help measure the infants' hands, as the digits were small. Means of the two right-hand (R2D:4D) measurements and the two left-hand (L2D:4D) measurements were averaged to create individual mean digit ratios (M2D:4D).

5.2.3: Determination of dominance rank

Each female was followed twice a week for a minimum of 16 weeks in the period immediately preceding the trapping period. Female rank within the matriline was estimated via observation of antagonistic interactions (observational data provided by the Christy Hoffman, University of Chicago). Christy Hoffman recoded initiators and recipients of both aggressive (i.e., threats, chases) and submissive behaviours (i.e., withdrawals, screams, grimaces) during 30 minute time periods. Submissive individuals were scored as lower ranking than aggressive individuals, and dominance was only scored when submissive actions were clearly observed. As dominance rank data is available for all females within matriline (not just the study sample), it was possible to assign *absolute ranks*. Absolute rank is defined as the rank order of a female in relation to the rank of all other females within the matriline. For example, the absolute ranks of the study females in matriline 004 were 21, 22, 25, 26 and 37.

However, since I could only measure the digits of a sample of females within a matriline (as noted above), I also calculated the *proportional dominance rank in matriline* (see Zehr *et al.* 2005) as a method of controlling for the large group-size differences between kin lineages.

The proportional dominance rank in a matriline is calculated by dividing the absolute rank of the female within the matriline by the total number of mature females in the matriline. For example, the absolute ranks of females in matriline 004 are divided by the total number of females in that matriline ($n = 38$; see Table 5.1) yielding proportional ranks of 0.52, 0.58, 0.66, 0.68 and 0.97; the lower the value the higher the rank.

Social groups and matriline within the social groups could be ranked as high, middle or low (Table 5.1). Social group rank and matriline rank were assessed by observing the direction of displacements at favoured sites. For example, if one group displaced another at a feeding patch, the displaced group would be considered lower ranking than the displacing group. Based on observations of individual females and their birth histories, families could also be ranked linearly according to the ranks of the females within them. A family consisted of a matriarch, her daughters, and her granddaughters.

5.2.4: Statistical methods

5.2.4.1: Part I: Adult females

2D:4D and age were normally distributed (Kolmogorov-Smirnov Tests), but body weight was not; body weight was therefore log-transformed. A general linear model (GLM) was used to investigate relationships between 2D:4D and dominance rank within and between social groups, matriline, and families. I hypothesized that a female's 2D:4D was influenced by maternal androgens and largely fixed *in utero*. I therefore assigned 2D:4D as the independent variable and dominance rank as the dependent variable. Age and body weight were considered as covariates. For comparisons of dominance rank within social groups I coded females linearly; the highest-ranking female in the highest-ranking matriline as 1, while the lowest-ranking female in the lowest-ranking matriline (within the same group) was assigned the lowest number (of the total sampled for that social group).

It is important to note that in the regression of 2D:4D and family rank (Table 5.3; Fig. 5.1), I pooled data of families from two social groups (DM and 116s) to increase the power of the analysis. However, I controlled for social-group differences by assigning 'social group' as fixed factor. A paired *t* test (two tailed) was used to evaluate differences in 2D:4D between left and right hands and younger and older sisters within families. Sample sizes within groups were highly variable (see Table 5.1), but analyses were not weighted since there were no relationships between variances of 2D:4D and sample size.

5.2.4.2: Part II - Mother-infant dyads

All data were normally distributed. Data were analyzed using GLM was used to test across categorical variables and linear regression for continuous variables. Cohen's *d* (see Dunst *et al.* 2007) was used to estimate the size effect of 2D:4D between male and female infants. Pearson's correlation coefficient (*r*) was used to examine relationships between 2D:4D, age, and body weight. Paired *t* tests (two-tailed) were used to compare mother and infant 2D:4D ratios. Following standard practice of familial resemblance studies of 2D:4D, heritability (h^2) values for mother-infant dyads were calculated by doubling the Pearson correlation coefficients (Ramesh & Murty 1977; Voracek & Dressler 2009).

Significance is at the $p \leq 0.05$; F=variance result; p=probability; df=degrees of freedom; R^2 is the correlation coefficient explaining (in a linear regression) the extent of the effect of the independent variable on the dependent variable.

5.2.5: Repeatability of measurements

Intra-observer measurements of repeatability were quantified with intra-class correlation coefficients (ICC), according to a two-way mixed-effects model with absolute-agreement definition (McGraw & Wong 1996). Adult females ICC's = R2D:4D, 0.895; L2D:4D, 0.942; M2D:4D, 0.955 (all p 's < 0.0001); Infants: ICC = R2D:4D, 0.651; L2D:4D, 0.783; M2D:4D, 0.797 (all p 's < 0.02). These results indicated that 2D:4D ratios of infants were somewhat less repeatable than those of their mothers, which is understandable due to the smaller size of infant hands at this age.

5.3: Results

5.3.1: Part I: Adult females

5.3.1.1: 2D:4D, age, and weight in adult females

Mean 2D:4D (\pm standard deviation) across the whole sample was 0.827 ± 0.04 . L2D:4D and R2D:4D were not significantly different (left: 0.830 ± 0.05 ; right: 0.825 ± 0.04 ; $t_{59} = 0.776$, $p = 0.447$), as such the main findings are based on M2D:4D. Mean values for this group of free-living female rhesus macaques are within range of M2D:4D for captive populations (see Chapter 3). Age structure differed between social groups ($F_{2,59} = 3.37$, $p = 0.04$; Table 5.1).

There was also a trend for body weight to increase with the increasing dominance rank of social groups ($F_{2,59}=2.79$, $p=0.07$; Table 5.1). I controlled for both of these variables in all analyses to reduce the possibility of Type I errors (Grafen & Hails 2002), as age and body weight are known to covary with dominance rank in rhesus macaques (Datta 1988; Zehr *et al.* 2005).

5.3.1.2: 2D:4D and dominance rank in adult females

5.3.1.2.1: Relationships within and between social groups

I first addressed whether there was a relationship between M2D:4D and dominance rank between social groups. These relationships were not significant ($F_{2,59}=0.10$, $p=0.90$). Next I investigated relationships within the social group. Analyzing social groups separately there was a significant relationship between M2D:4D and dominance rank for social group S (Table 5.2), which was the only group composed of a single matriline (see Table 5.1). In social group S, significant interactions between dominance rank in social group, age and body weight were also found ($F_{1,10}=12.05$, $p=0.006$); older and heavier females in this social group tended to be higher ranking.

Group	Variable	F	p	R ²	df	Power
F	M2D:4D	0.001	0.99	0.09	1,7	0.05
	Age	0.004	0.95		1,7	
	Body weight	0.57	0.47		1,7	
R	M2D:4D	1.28	0.27	0.06	1,30	0.20
	Age	0.35	0.56		1,30	
	Body weight	0.28	0.60		1,30	
S	M2D:4D	6.25	0.03	0.59	1,10	0.64
	Age	8.38	0.02		1,10	
	Body weight	7.53	0.02		1,10	

Table 5.2: Mean 2D:4D (M2D:4D) and dominance rank within social groups. Rank in social group was assigned as the dependent variable, and M2D:4D was assigned as a covariate along with age and body weight. Note that Group S only contains one matriline (116s; Table 5.1).

5.3.1.2.2: Relationships within and between matriline

No differences were found between matriline in Groups F and R, respectively (between matriline in Group F, $F_{1,10} = 0.10$, $p = 0.76$; matriline in Group R, $F_{1,33} = 0.50$, $p = 0.50$). In contrast, within matriline (across all sampled individuals), M2D:4D ratio decreased (higher PAE) significantly, as the proportional dominance rank in matriline increased ($F_{1,55} = 6.86$, $p = 0.01$, $R^2 = 0.12$; Observed power = 0.73); age and body weight were not significantly related to proportional dominance rank (age: $F_{1,55} = 0.42$, $p = 0.52$; body weight: $F_{1,55} = 0.05$, $p = 0.82$). Relationships between M2D:4D and absolute rank within matriline across the whole group also approached significance ($F_{1,55} = 3.48$, $p = 0.07$, $R^2 = 0.06$; Observed power = 0.45); again, age and body weight were not significantly related to absolute rank (age: $F_{1,55} = 0.09$, $p = 0.77$; body weight: $F_{1,55} = 0.001$, $p = 0.99$). Thus lower M2D:4D ratios, indicative of higher PAE, were more common in higher ranking females.

5.3.1.3: Relationships between ranked families within matriline

Small sample sizes for three of the six matriline (no more than six individuals in matriline 065, 004, and 116r; see Table 5.1) meant that it was not possible to compare across families in these matriline in a meaningful way. However, matriline DM and 116r were larger and contained individuals from seven ranked families within both matriline; DM: families = 7; individuals = 27; 116s: families = 7; individual = 14). As matriline DM and 116s are from different social groups, R and S, respectively (Table 5.1), possible effects of 'social group' were controlled for (see Methods), and treated age and weight as covariates. 2D:4D ratios and matrilineal family rank were significantly negatively correlated; members of high-ranking families exhibited lower 2D:4D (Table 5.3; Fig. 5.1).

5.3.1.4: 2D:4D and birth-order effects in adult females

To test for possible birth-order effects, where possible (within families) 2D:4D ratios of oldest sisters were compared with their youngest sisters ($n = 12$ pairs); relationships were not significant (M2D:4D: $t_{11} = -0.39$, $p = 0.71$).

Ranked Families	F	p	R ²	df	Power
M2D:4D	6.99	0.01	0.19	6,37	0.73
Social group	0.004	0.95		1,37	
Age	0.92	0.34		1,37	
Body weight	0.56	0.46		1,37	

Table 5.3: 2D:4D of ranked families in matriline DM and 116s, with family rank assigned as the dependent variable and M2D:4D as a covariate alongside age and body weight. I pooled data of families from different social groups (R and S; see Methods) to increase the power of the analysis, but controlled for social group differences by assigning social group as the fixed factor.

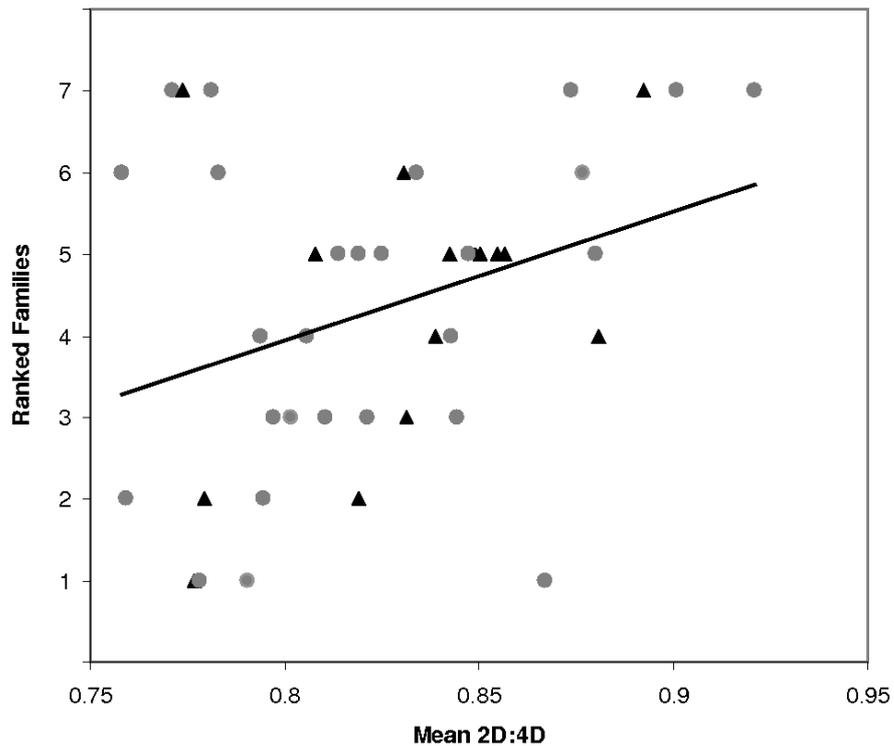


Figure 5.1: 2D:4D of individuals within ranked families in matriline DM (n=29; gray dots) and 116s (n=14; black triangles). Family rank was assigned as the dependent variable. Highest ranking family =1; lowest ranking family =7. Data from matriline DM were analyzed together, while controlling for social group as a fixed factor (R²=0.19).

5.3.2: Part II - Mother-infant dyads

5.3.2.1: Sex differences in infant 2D:4D

Male infants did not exhibit significant differences in L2D:4D with female infants (0.833 ± 0.037 for females vs. 0.820 ± 0.025 for males; $t_{23}=1.09$, $p=0.29$, Cohen's $d=-0.45$) or in R2D:4D (0.816 ± 0.034 for females vs. 0.808 ± 0.028 for males; $t_{23}=-0.58$, $p=0.57$, Cohen's $d=-0.27$). M2D:4D of male and female infants respectively were $0.814 (\pm 0.022)$ and $0.824 (\pm 0.027)$, with an effect size differences (Cohen's $d=-0.43$). Size effects for the infants are larger than that for a larger sample of adult rhesus macaques from various captive populations (Cohen's $d=-0.23$).

5.3.2.2: 2D:4D, age, and weight in mother-infant sample

Comparisons between infants' R2D:4D (mean and SD: 0.811 ± 0.03) and L2D:4D (0.824 ± 0.03) were not significant ($r=0.30$, $p=0.15$), and infants' R2D:4D was somewhat, but not significantly, lower than their L2D:4D (paired t test: $t_{23}=1.91$, $p=0.07$). Mother's R2D:4D and L2D:4D were significantly positively related ($r=0.50$, $p=0.01$). Digit ratios did not significantly differ between mothers and infants (mother-daughter; L2D:4D, $t_7=0.80$, $p=0.45$; R2D:4D, $t_7=2.11$, $p=0.07$) and mother-son (L2D:4D, $t_{16}=-0.91$, $p=0.38$; R2D:4D, $t_{16}=-1.83$, $p=0.09$).

Mean body weight for mothers was 8.22 kg (± 1.53). Mean body weight for female infants was lower than for males infants (female mean: 0.85 kg, ± 0.21 ; male mean: 1.03 kg, ± 0.21), and this difference was significant when age was controlled for ($F_{1,24}=6.08$, $p=0.01$; Table 5.4). There was no correlation between maternal body weight and infant body weight ($r=0.241$, $p=0.25$). Body weight of individuals did not significantly differ between social groups (mothers, $F_{1,24}=1.58$, $p=0.22$; infants, $F_{1,24}=3.02$, $p=0.10$) or ranked matriline (mothers, $F_{1,24}=1.79$, $p=0.19$; infants, $F_{1,24}=1.29$, $p=0.27$). Mothers' and infants' body weight did not correlate with mothers' absolute rank in matriline (mothers, $F_{1,24}=0.14$, $p=0.71$; infants, $F_{1,24}=1.70$, $p=0.20$) or proportional rank in matriline (mothers, $F_{1,24}=0.27$, $p=0.61$; infants, $F_{1,24}=0.81$, $p=0.38$).

Table 5.4: Social, physical and age variables in Part II (mother-infant dyads).

Social Groups	Rank	Relatives	Sex	n	Mean 2D:4D	sd	Age	sd	Weight	sd	Matriline	Rank	n	
F	High	Mothers	F	8	0.824	0.02	16.2	5.42	8.7	1.7	065	High	5	
											004	Low	3	
		Infants	F	3	0.821	0.03	0.2	0.04	0.8	0.2		065	High	2
												004	Low	1
			M	5	0.812	0.02	0.2	0.05	1.2	1.0		065	High	3
												004	Low	2
R	Middle	Mothers	F	9	0.822	0.03	15.0	6.49	8.1	1.7	DM	Low	9	
		Infants	F	4	0.830	0.03	0.2	0.09	1.2	0.8		DM	Low	4
M	5		0.802	0.02	0.2	0.09	1.2	1.0		DM	Low	5		
S	Low	Mothers	F	8	0.824	0.04	12.5	3.35	7.8	1.1	116s	Low	8	
		Infants	F	1	0.804	0.00	0.3	0.00	1.0	0.0		116s	Low	1
M	7		0.824	0.02	0.4	0.40	1.3	1.1		116s	Low	7		

Mean age of mothers was 14.6 years (± 5.32) and the mean age of infants was 3 months (± 0.07 ; Table 5.4). Female infants had a lower mean age than males (female mean: 73 days, ± 0.07 ; male mean: 83 days, ± 0.06), but the ages did not significantly differ ($F_{1,24}=3.92$, $p=0.14$). Unlike the larger adult female sample in Part I (see above), mothers' R2D:4D in this study (Part II) significantly increased with age ($r=0.47$, $p=0.02$), but not their L2D:4D ($r=0.20$, $p=0.34$). Mothers' and infants' age did not significantly differ between social groups (mothers, $F_{1,24}=2.08$, $p=0.16$; infants, $F_{1,24}=0.80$, $p=0.38$) or ranked matriline (mothers, $F_{1,24}=1.73$, $p=0.20$; infants, $F_{1,24}=0.27$, $p=0.61$) and did not correlate with mothers' absolute rank (mothers, $F_{1,24}=0.72$, $p=0.41$; infants, $F_{1,24}=1.44$, $p=0.24$) or proportional rank in matriline (mothers, $F_{1,24}=0.31$, $p=0.58$; infants, $F_{1,24}=0.76$, $p=0.39$).

5.3.2.3: 2D:4D and dominance rank in mother-infant sample

Unlike the larger, adult sample (Part I; $n=60$), in this study (Part II; $n=25$ dyads) there was no relationship between mothers' 2D:4D and absolute rank (M2D:4D, $F_{1,21}=0.15$, $p=0.70$, $R^2=0.04$, Observed power =0.07) or proportional rank in matriline (M2D:4D, $F_{1,21}=0.07$, $p=0.80$, $R^2=0.02$, Observed power =0.06;). This was also the case for infants, which were assumed to be the same rank as their mothers (absolute rank, M2D:4D, $F_{1,21}=0.02$, $p=0.89$, $R^2=0.07$, Observed power =0.05; proportional rank, M2D:4D, $F_{1,21}=0.04$, $p=0.84$, $R^2=0.04$, Observed power =0.05). Age and body weight were factored into each analysis, but were non-significant.

5.3.2.4: Heritability of 2D:4D

Mother-infant resemblance (based on 25 dyads, with both sexes of infants combined) was strong and significant for R2D:4D ($r=0.58$, $p=0.002$), but was not significant for L2D:4D ($r=0.31$, $p=0.13$). Hence the heritability estimate was high, in fact off the upper boundary, for R2D:4D (i.e., $h^2 > 100\%$), but lower for L2D:4D and $h^2=62\%$ (Fig. 5.2).

These findings (higher h^2 for the right hand than for the left) also generally held in separate analyses when accounting for offspring sex. Mother-daughter resemblance (based on 8 dyads, R2D:4D: $r=0.53$, $p=0.18$; L2D:4D: $r=-0.12$, $p=0.78$), was not as strong as mother-son resemblance (based on 17 dyads, R2D:4D: $r=0.62$, $p=0.008$; L2D:4D: $r=0.42$, $p=0.09$).

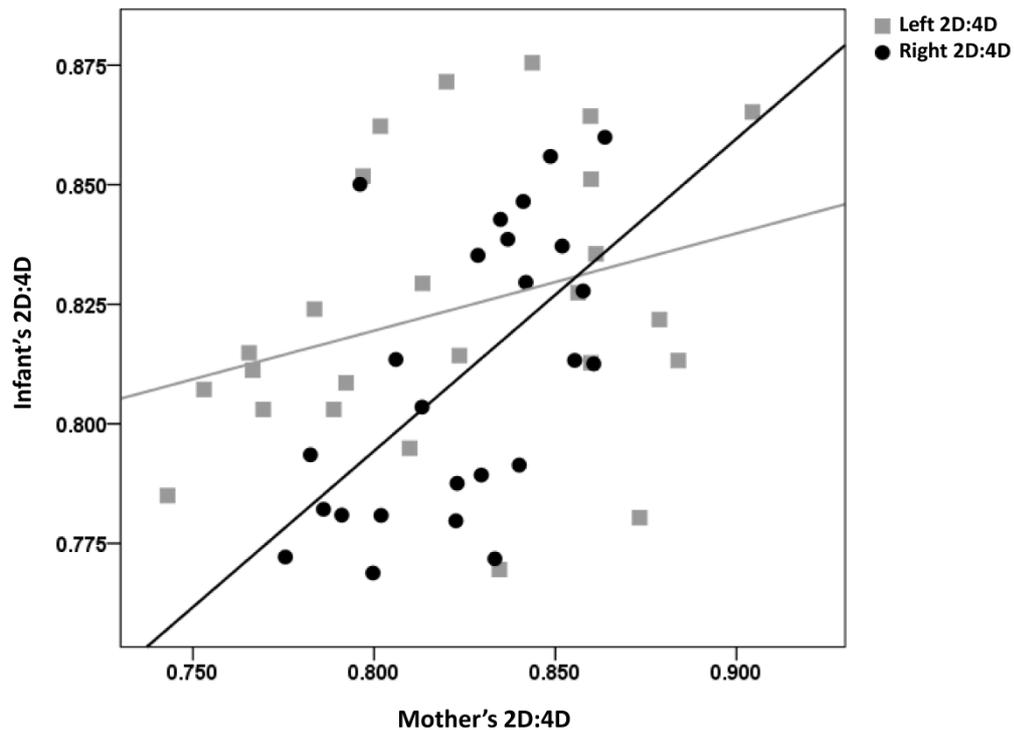


Figure 5.2: Relationships between mother and infant 2D:4D ratios (Right 2D:4D $R^2=0.34$; left 2D:4D $R^2=0.1$). Heritability (h^2) estimates were calculated by doubling the Pearson correlation coefficients r . Right 2D:4D ($r=0.58$, $p=0.002$) has higher heritability (off the upper boundary, i.e., $h^2 > 100\%$) than the left hand 2D:4D ($r=0.31$, $p=0.13$, $h^2=62\%$).

5.4: Discussion

This is the first study to show a relationship between 2D:4D and dominance rank in non-human primates. It is also the first to provide heritability values for 2D:4D in a non-human primate. Lower 2D:4D ratios were associated with high dominance rank, while higher 2D:4D ratios were linked to lower dominance rank in a group of free-ranging adult female rhesus macaques residing on Cayo Santiago. Relationships were most marked between ranked families within matriline with 2D:4D explaining 19% of variance in family dominance rank (Table 5.3; Fig. 5.1). In contrast, no differences in 2D:4D were found between matriline or between social groups. Relationships were therefore only evident between related individuals within matriline. These results are similar to relationships between 2D:4D and dominance-linked behaviours in humans (e.g., Hönekopp *et al.* 2006a; Millet & Dewitte 2007; see also Manning & Fink 2008) and between dominance-related behaviours and PAE (inferred from faecal samples) in wild spotted hyenas (Dloniak *et al.* 2006). Similarities are also shown in the heritability levels of 2D:4D based on mother-infant dyads; values were found to be within the range of human twin and family studies of 2D:4D.

5.4.1: 2D:4D and dominance rank in adult female rhesus macaques

The life experiences of individuals can differ markedly as a consequence of social rank (Bastian *et al.* 2003; Sapolsky 2005), and the physiological and psychological factors experienced by a gestating female are known to impact the development of the foetus (e.g., Herman *et al.* 2000; Thornton *et al.* 2009). It might be expected the stable dominance ranks in the Cayo Santiago colony (Stucki *et al.* 1991) and the pattern by which groups' fission along female kin lines (Chepko-Sade & Sade 1979) provide the potential for differences in 2D:4D to arise between unrelated individuals and groups. However, this was not the case; differences were only detected amongst related individuals within matriline. It is possible that the transfer of males leads to gene flow and prevents significant differences in 2D:4D from emerging between longstanding, differently ranked social groups. Female philopatry and the nepotistic social structure (Thierry *et al.* 2000), on the other hand, provides the substrate for inter-individual differences to emerge within matriline via maternal effects. Although a females' dominance rank within the social group is likely to influence her overall quality-of-life, it is her support network of close female kin that she spends most time with (Chauvin & Berman 2004; Datta 1988). Consequently, a female's close kin tend to be her direct competitors so the levels of competition she experiences will be most strongly linked to her dominance rank within the matriline than her rank within the whole social group.

In rhesus macaques competition for resources is evident at all levels of the social structure (Maestriperi 2007). A mother's ability to alter the physiology of her foetus via maternal effects provides a mechanism for her to potentially improve the fitness of her offspring before birth. Abnormal conditions, such as extreme maternal stress, have been shown to masculinise and defeminise daughters and adversely affect fertility (Wallen & Baum 2002; Kaiser & Sachser 2005). Kaiser and Sachser (2005; 2009) have proposed, however, that prenatal androgenisation of females may be adaptive. It is conjectured that when the normal social environment is highly competitive (e.g., at high population densities), it might be advantageous for female offspring to be androgenised in order to promote dominance-traits that equip them to obtain and defend valuable resources. Although high PAE may compromise fertility, androgenised females may still fare better than other females because they can monopolize resources (Kaiser & Sachser 2005; 2009). Similar mechanisms have been proposed for relationships between female dominance rank and PAE in wild spotted hyenas; a species in which resource competition is intense and higher ranking females have preferential access to food (see Dloniak *et al.* 2006). It is proposed that in species such as rhesus macaques and hyenas, in which dominance rank is 'inherited' and clearly stratified, PAE may have a less marked influences in species with more egalitarian and less clearly

defined social structures. Effects may be magnified in the Cayo Santiago monkeys because the island setting itself may promote social competition by restricting the distances over which animals can range in search of new resources as well as restricting their ability to avoid rivals.

In rhesus monkeys higher-ranking females have preferential access to resources (Silk, 2002b). When dominance hierarchies are stable, high-ranking females do not need to defend their dominance rank by expressing high levels of aggression (Higley *et al.* 1996; Westergaard *et al.* 2003). However, even when social relationships are stable, dominant females tend to show higher rates of low-level aggression towards other females (Higley *et al.* 1996; Westergaard *et al.* 2003). Higher ranking mothers are also quicker than lower ranking mothers to intervene aggressively on behalf of their offspring (Maestriperi 1994). The social mechanisms that underpin dominance-rank inheritance in rhesus macaque societies permit higher-ranking females to impose their authority down the hierarchy often without threat of retaliation to themselves. However, it is also possible that the physiological profile of higher ranking adult females (inferred higher PAE) predisposes them to be more aggressive when dealing with potential threats to status (see Millet & Dewitte 2008; 2009) or to be quicker at detecting threats to their status, enabling them to respond faster to those cues (see Coates *et al.* 2009; Coates 2009). For lower-ranking monkeys, inappropriate expression of impulsive or aggressive behaviour is more likely to lead to punishment. Exposure to lower PAE (as indexed by higher 2D:4D) in lower ranking females, in conjunction with the social learning they experience as low ranking individuals, may predispose them towards more inhibited responses which would be socially adaptive over the longer term (e.g., winner-and-loser effects: Drummond & Canales 1998; also see Champagne & Curley 2005; Mehta & Josephs 2006).

I speculate that covariation in 2D:4D might therefore indicate different PAE on programming developmental trajectories linked to the formation of predispositions or personality types. The social landscape then provides the potential for some PAE-linked behaviours to be expressed, or reduces the potential for others to emerge during the course of an individual's development (Knickmeyer & Baron-Cohen 2006; Wallen 1996; 2005). If this is the case, these effects may be associated with promoting the development of rank-appropriate behaviours. Certainly, in macaque societies, as in many primate societies, appropriate behaviour is adaptive (Bastian *et al.* 2003; Maestriperi 2003), while rank-inappropriate behaviour can be detrimental and may even lead to severe injury and death (Bastian *et al.* 2003; Westergaard *et al.* 2003). An extension of this behaviour is that individuals recognize social hierarchies and maintain their position within the pecking-order.

Thus, while behavioural processes undoubtedly provide the primary mechanism for dominance rank inheritance in Old World monkeys (e.g., Chapais 1992; 2004; Datta 1988; Holekamp & Smale 1992), physiological processes have some influence (see Maestriperi 2003). Even small inter-individual rank-related differences in PAE may support female dominance rank inheritance within and between generations of cercopithecine primates.

In humans, exposure to higher PAE (indexed by low 2D:4D) appears to predispose individuals towards expressions of dominance and these effects have been demonstrated in both sexes (e.g., Bailey & Hurd 2005; Benderlioglu & Nelson 2004; Manning & Fink 2008). Differences in 2D:4D may correlate with different adaptive approaches to situations that increase evolutionary fitness, such as avoiding harm or acquiring resources. For example Millet & Dewitte (2009) have shown that after exposure to aggressive cues (e.g., violent video and aggressive words within written sentences) low 2D:4D (inferred high PAE) individuals tend to respond aggressively under test conditions, while individuals with higher 2D:4D (inferred low PAE) become more friendly. It is postulated those individuals with low 2D:4D (inferred high PAE) may be predisposed to react strongly in potentially aggressive situations in order to maintain status. Conversely, for individuals with higher 2D:4D (inferred low PAE) it may be more adaptive to tackle a potentially stronger and more aggressive opponent by adopting a non-threatening manner (Millet & Dewitte 2009). If these findings generalise across taxa they might suggest that, in macaques, rank-related differences in 2D:4D could be associated with PAE on the development of rank-appropriate behaviours.

In humans, 2D:4D and digit lengths have been shown to vary with birth order and the sex ratio of older siblings (Williams *et al.* 2000; Saino *et al.* 2006). Offspring digit ratios have also been associated with maternal fecundity (Saino *et al.* 2006). It has been proposed that variation in offspring 2D:4D could occur via maternal-androgenising effects that change according to the mother's physical and social environment (Kaiser & Sachser 2005). However, maternal effects may also be associated with the *condition* of the mother, such as maternal age and the physiological traces of previous offspring on maternal tissues (i.e., the notion of tissue memory; see Williams *et al.* 2000; Fowden & Forhead 2009, p 617). These factors could serve to confer cellular-level signals to the foetus about the extra-uterine environment; such as maternal health or potential sibling rivalry (see Saino *et al.* 2006). Evidence from a study of changes in digit ratio according to birth order might suggest that 2D:4D ratios will be lower in younger daughters of rhesus mothers (PAE increase; see Saino *et al.* 2006), but no birth-order effects on 2D:4D within families were found: younger, higher ranking sisters did not have more masculinised (lower) digit ratios than older, lower ranking sisters. These findings therefore suggest that the pattern of strict youngest daughter

ascendancy within families in rhesus macaques is not supported by PAE. However, the fact that associations are shown between dominance ranks within matrilineal families (which are rank-ordered by the youngest daughter ascendancy rule) suggests that we should see a relationship. This indicates that the sample size used in this test (n=12 sister-sister dyads) is too low to test this hypothesis.

Do individuals with low 2D:4D have higher circulating androgen levels? Relationships between 2D:4D and serum androgens have been shown in male, but not in female guinea baboons (*Papio papio*; Roney *et al.* 2004). Associations in humans generally indicate no relationship (Hönekopp *et al.*, 2007; Muller *et al.* 2011; McIntyre *et al.* 2011), although parallels are evident between the behavioural responses of individuals with low 2D:4D (inferred high PAE) and individuals with high testosterone levels (Mazur & Booth 1998; Grant & France 2001). It is possible that 2D:4D could be reflecting PAE on programming neuro-endocrine pathways (Pfeiffer 1936; Fowden & Forhead 2009) and/or genetic sensitivity to circulating androgens (e.g., Manning *et al.* 2003a). For example, in mammals (guinea pigs), masculinisation of female offspring is associated with male-typical patterning and up-regulation of androgen receptors in certain brain regions (Kaiser & Sachser 2005). In humans, low 2D:4D ratios in males (females were not tested) have been associated with more sensitive androgen receptor gene expression (Manning *et al.* 2003a; but see Hurd *et al.* 2011). If higher ranking female rhesus macaques with lower 2D:4D are more sensitive to androgens compared to lower ranking females, these effects could lead to different responses to androgen-inducing situations in adulthood, even though absolute levels of the circulating androgens may not vary significantly between individuals.

5.4.2: 2D:4D in infant rhesus macaques

This study has provided the first opportunity to investigate sex differences in 2D:4D in a cohort of non-human primate individuals that have only been minimally influenced by postnatal influences (mean age 3 month). Among infant monkeys males had lower 2D:4D than females. Although these sex differences were not significant, they were in the expected direction; males are normally exposed to higher PAE and will therefore generally have lower 2D:4D than females. Mean 2D:4D in this infant sample were similar to values for captive adult rhesus macaques in the haplorhine database (see Appendix 2.5), and size effects between male and female infant ratios were also in range of those presented for humans (see Voracek *et al.* 2007c). If, as suggested by the results, interactions do exist between maternal-androgenising effects and female dominance rank in rhesus monkeys, then it is possible that

these rank-related mechanisms may act to blur sex differences in 2D:4D ratios in this species (e.g., higher ranking females having more male-like 2D:4D ratios).

5.4.3: Heritability of 2D:4D in rhesus macaques

The heritability values are based upon mothers and very young infants. This study design might be advantageous in that the potential impact on 2D:4D from possible postnatal growth and environmental influences can be considered minimal. However, the disadvantage of this approach is that heritability can be inflated due to the exclusion of possible non-shared environmental effects on the digit ratio occurring through growth. For example, right 2D:4D was shown to increase with age among adult females in Part II, but not in Part I; Part II females (those mothers with infants) formed a subset of the Part I sample. Age effects were not observed in infant monkeys which might be attributable to this group's narrow age range. In adult chimpanzees (*Pan troglodytes*) sex differences in 2D:4D have been shown to increase with age (McIntyre *et al.* 2009). Humans (and bonobos; *Pan paniscus*) differ from this pattern in that 2D:4D increases slightly during childhood while sexual dimorphism remains consistent (McIntyre *et al.* 2005; 2009; Trivers *et al.* 2006). Changes in 2D:4D through life (within and between species) are likely to confound heritability estimates based upon familial resemblance, and this should be considered in future analyses.

Heritability values were higher in the right hand compared to the left (Fig. 5.2). These estimates, particularly the lower correlation for left 2D:4D and the high heritability estimate for right 2D:4D may stabilize in larger samples. The differences in heritability between hands in this study (higher for the right-side than for the left) are concordant with results from another familial study (Voracek & Dressler 2009), but differ from twin studies that show higher heritability values for left hands than for right hands (Gobrogge *et al.* 2008; Medland & Loehlin 2008). Some of these contradictory findings may be associated with environmental factors that are reported to have a stronger influence on one side of the body than the other (see Flegr *et al.* 2008). Familial resemblance of 2D:4D between mothers and their infants yielded high heritability values over all (Fig. 5.2). This suggests that genetic and gestational contributions to the expression of 2D:4D are substantial and strengthens the conjecture that relationships between 2D:4D and PAE may generalize across taxa (Manning 2002a).

Heritability of 2D:4D was higher between mothers and sons than between mothers and daughters. A familial resemblance study in humans, however, showed stronger heritability through the male line (i.e., father-son, brother-brother; Voracek & Dressler 2009), which

suggests that 2D:4D may be inherited through Y-linked genes (i.e., *Sry*, sex determining gene). Inheritance of 2D:4D via the Y-chromosome, conflicts with the idea of X-linked associations (i.e., via the androgen receptor gene; see Manning *et al.* 2003a) and the findings of no evidence for sex-linked inheritance in a recent twins study (see Medland & Loehlin 2008). However, given the general conformity of high heritability estimates for 2D:4D from this study, several human studies (twin and family), and two parent-offspring studies of zebra finches, it seems unlikely that mechanisms of genetic inheritance differ between macaques and other taxa. It is possible that the results shown here (higher heritability between mothers and sons) may be a consequence of low sample size ($n=25$ mother offspring dyads), particularly for females ($n=8$ mother-daughter dyads), making the results prone to error. Additionally, the application of different measurement methodologies may also impact heritability estimates. The digits ratios in this study are based on direct measurements of finger lengths, whereas heritability studies use measurements taken from hand images such as photocopies (e.g., Gobrogge *et al.* 2008; Medland & Loehlin 2008; Voracek & Dressler 2009). It is now recognized that this imaging process can distort finger lengths in sex-specific ways (Manning *et al.* 2005; Caswell & Manning 2009; see Chapter 2), so when possible, direct measurements of hands should be used (Manning & Hill 2009). In addition, it is known that soft tissue changes occur in response to cyclical and short-term changes in hormonal concentrations in males and females (Scutt & Manning 1996; Manning *et al.* 2002). These factors, along with age-related changes (see above), are likely to impact heritability estimates of 2D:4D and confound cross-study comparisons.

5.4.4: Summary

Social behaviour undoubtedly provides the primary mechanism by which dominance rank is attained in cercopithecine primates, however the preliminary results presented in this study support the idea that PAE, indexed by 2D:4D, might also contribute to maintaining dominance hierarchies. Evidence from 2D:4D also suggests that higher PAE is involved in higher dominance related behaviours in humans. The 2D:4D ratios of adult female rhesus monkeys suggest that PAE is positively related to dominance rank with effects being most evident between related individuals. Familial resemblance of 2D:4D indicates that heritability is high in this population and mirrors levels in humans and other taxa. The rank-associated patterns shown in the larger study suggest that a proportion of this familial resemblance may be linked to prenatal maternal effects which may occur via physiological responses to competition between relatives within matriline. Finally it is proposed that variation in PAE between ranked individuals might influence whether individuals react to social situations in rank-appropriate manner. If correct, these effects are likely to be highly

adaptive in strongly bonded hierarchical social systems, as even small differences in an individual's abilities to dominate and confront others appropriately are likely to impact fitness.

Chapter 6

2D:4D, testosterone and the androgen receptor gene in haplorhines

6.1: Introduction

In this chapter I shift from looking at broad trends in behaviour within and between haplorhine species to looking at how species-level 2D:4D correlates with factors more closely associated with *potentiating* behaviours; specifically, variation in adult circulating androgens levels (testosterone) and variation in androgen receptor gene sensitivity (ARG).

The introduction will focus on the genetic and cellular mechanisms that influence adult circulating testosterone levels and considers the circumstances in which they may be related to 2D:4D and sexually selected behaviours. The discussion will speculate on how the results of the analyses may inform us about big questions surrounding primate social behaviour and hominin evolution.

6.1.1: Circulating testosterone (CircT)

In adults circulating testosterone (circT) levels are higher in males than females (Tortora & Anagnostakos 1990; Gouchie & Kimura 1991) and show moderate heritability (Aitken-Harris *et al.* 1998). In males circT is important for sperm formation, maintenance of muscle mass and bone metabolism (Tortora & Anagnostakos 1990). In humans and non-human primates circT is often associated with the male phenotype, but it is also important for female development (Herman *et al.* 2000) and is instrumental in potentiating behaviours linked to sexual selection in both sexes (e.g., aggression, dominance; Mazur & Booth 1998; Grant & France 2001; Alvergne *et al.* 2009; McIntyre *et al.* 2011).

CircT levels differ across haplorhine populations (including humans) (Gray 2003; Whitten & Turner 2004; 2009; Alvergne *et al.* 2009), co-vary with seasonality (e.g., Gordon *et al.* 1976; Cerda-Molina *et al.* 2009) and time-of-day (e.g., Goodman *et al.* 1974; also see Dixson 1980; Archer 1991). CircT also changes over life-stages. For example, in humans and other haplorhines (Mann *et al.* 1984; Resko 1985) birth triggers a surge in luteinizing hormone and in male neonates this leads to a sharp increase of circT that approach adult male levels. Within one to two weeks these levels fall, but increase again at around four to six months, at which time they remain at childhood (low) levels until puberty (Forest 1990). In the

parturient peak, sex hormone binding globulin (SHBG) is low (causing unbound T to be high), but SHBG is higher at the second peak causing unbound T to be low (Forest 1990; McIntyre 2006). The significance of these early postnatal peaks and the variation in bound and unbound T has on subsequent development is still not clearly understood, although there is evidence to show that they may be associated with cell proliferation in the testes (Fouquet *et al.* 1984) and penile growth (Brown & Dixson 1999).

Male circT increases in anticipation of mating opportunities (e.g., Herndon *et al.* 1981; Gray *et al.* 2004; Alvergne *et al.* 2009), the magnitude of the increase may vary according to social status such as dominance rank; higher ranking individual exhibit higher levels of circT (e.g., Czoty *et al.* 2009; also see Mazur & Booth 1998.). In some haplorhine species male circT has been shown to change around the time of their offspring's birth and remains low while their offspring are young (Story *et al.* 2000; Zeigler & Snowdon 2000; Gray 2003). In pair-bonded species this may be a mechanism for mate guarding (Zeigler & Snowdon 2000) and increasing tolerance in males that have to partake in infant care. Male circT also declines with age (Martin *et al.* 1977; Storey *et al.* 2000) in association with cell degeneration, pituitary sensitivity and, possibly loss of status and mating opportunities (Masur & Booth 1998). In females circT varies across the menstrual cycle (e.g., Machatachke *et al.* 2006) and rises over pregnancy (Castracane *et al.* 1998; Beehner *et al.* 2005). The magnitude of the increase may vary positively according to dominance rank (see Beehner *et al.* 2005; also see Grant & France 2001). As with males, female circT can also decline with age (Labrie *et al.* 1997).

CircT in both sexes alter with social system and vary widely throughout life within and between individuals (e.g., see Bernhardt *et al.* 1998; Mazur & Booth 1998; Dabbs *et al.* 2001; Grant & France 2001). These fluctuations contrast with genetic stability of androgen sensitivity throughout life that is more conserved (e.g., timing of the prenatal peak; Resko & Ellinwood 1981; Resko 1985; McIntyre 2006).

6.1.2: Cellular and genetic mechanisms

Foetal programming is a process in which variation in the prenatal environment during 'critical phases' have lasting and lifelong effects (MacLusky & Naftolin 1981; Phillips 2002). Critical phases occur when tissues become 'switched-on' and receptor genes distributed throughout the foetal tissues become sensitive to local environmental influences (e.g., hormones; Rhind *et al.* 2001; Fowden & Forhead 2004; 2009; Fowden *et al.* 2008). Prenatal androgen effects (PAE) are a critical factor in programming sex-linked neuro-

endocrine axes that control adult hormone production (Phoenix *et al.* 1959; Goy & McEwen 1980; Phillips 2002; Fowden & Forhead 2009). PAE differ between the sexes (Auyeung *et al.* 2009) with low prenatal androgens imprint the hypothalamus and pituitary towards a female cyclical pattern and high prenatal androgens imprinting a male tonic pattern (Pfeiffer 1936; Fowden & Forhead 2009, p 617). Abnormalities in prenatal androgen levels during critical phases (i.e., high levels in females and low levels in males) can lead to disrupted functioning of the neuro-endocrine axes and are have been implicated reduced infertility and some chronic diseases (Phillips 2002; Davies & Norman 2002; Fowden & Forhead 2009); 2D:4D has been shown to correlate with some of these conditions (see Manning 2002a).

High androgens *in utero* appear to have a negative impact on the development of the reproductive system. Females exposed to excessively high PAE, such as in congenital adrenal hyperplasia and polycystic ovary syndrome, suffer menstrual irregularities and infertility (i.e., Abbott *et al.* 2005; Merke & Bornstein 2005; Hickey *et al.* 2002). Women with these conditions have low 2D:4D ratios compared to healthy controls (Brown *et al.* 2002c; Ökten *et al.* 2002; Cattrall *et al.* 2005; but see Buck *et al.* 2003; Lujan *et al.* 2010a; 2010b). Delayed menarche and slow pubertal development has also been linked to low 2D:4D (inferred high PAE) in healthy females (Matchock 2008; Manning & Fink 2011; but see Helle 2010). In contrast, factors known to decrease prenatal androgen production in males can lead to reproductive tract abnormalities and infertility (Rhind *et al.* 2001; Davies & Norman 2002). High 2D:4D (inferred low PAE) in men is associated with low quality sperm and small family size (Manning *et al.* 2003b; Wood *et al.* 2003; but see Firman *et al.* 2003; Bang *et al.* 2005; Seo *et al.* 2010). Variation in PAE have antagonistic effects according to sex; high PAE are detrimental to females, but beneficial to males, while low PAE are detrimental to males but beneficial to females (Manning *et al.* 2000a; Rice 2000; Rice & Chippendale 2001). This antagonism appears to be stronger in promiscuous species because of selection for high androgens to support male-male competition (see Holland & Rice 1999). This highlights the key role of this group of hormones in sexual selection (Rice 2000; Trivers 1972; see Chapter 3).

Unlike the antagonistic effects of PAE on male and female reproductive potential, high and low PAE appear to impact male and female social development in similar ways (see Baron-Cohen *et al.* 2004; Knickmeyer *et al.* 2005; 2006) and excessively high PAE have been implicated in autistic spectrum disorder (ASD; Baron-Cohen 2002). Understanding the development of normal social skills and the processes involved in interpreting social information is important because impaired social ability impacts quality of life and fitness (see Tomasello *et al.* 2005; Domes *et al.* 2007). Results from studies looking at how PAE

(assayed from amniotic fluid samples) influences the social development of on healthy children are showing boys and girls exposed to higher amniotic androgens (prenatally) exhibit milder forms of the key characteristics of ASD (e.g., restricted interests, poorer social skills, poorer relationship quality; Knickmeyer *et al.* 2005; 2006). Exposure to higher PAE enhances rule-based thinking processes termed, ‘systemising’ (Baron-Cohen 2002). Systemisers excel in dealing with tasks that can be solved through logical, cause-and-effect processes (Baron-Cohen 2003). Systemisers often have difficulty in interpreting social cues as they are non-rule-based and often highly nuanced. In contrast, individuals exposed to lower prenatal androgens express enhanced social skills that appear to be underpinned by abilities to key into the mental states and emotions of others; termed ‘empathising’ (Baron-Cohen 2002). People with high empathising skills are able to predict the behaviours of others and alter their behaviour accordingly (Knickmeyer & Baron-Cohen 2006). Categorising individuals as ‘systemisers’ or ‘empathisers’ is useful in defining broad-types of social cognition and in identifying individuals at the extreme ends of the autistic spectrum.

In the normal population, low 2D:4D (inferred high PAE) in two-year-old children has been linked to high testosterone-estradiol ratios assayed from amniotic fluid samples (Lutchmaya *et al.* 2004). Studies of 2D:4D in individuals with ASD show that the 2D:4D decreases (PAE increases) with increasing severity of the disorder (Manning *et al.* 2001). Additionally, the unaffected parents and siblings of individuals with ASD also have lower 2D:4D ratios than close relatives of normal controls (Manning *et al.* 2001). This supports evidence of the high heritability and the complex aetiology of ASD (Henningsson *et al.* 2009). The concept that 2D:4D is a stronger index of PAE than of prenatal estrogens is supported by a significant negative correlation between 2D:4D and a conventional test of systemising ability (Systemising Quotient), but no relationship between 2D:4D and a standard test of empathising ability (Empathising Quotient; Manning *et al.* 2010; also see von Horn *et al.* 2010; Wakabayashi & Nakazawa 2010; but see Voracek & Dressler 2006b). Correlations with 2D:4D were stronger for SQ, than EQ and 2D:4D in females showed much weaker or no relationship (but see Valla *et al.* 2010). Recently a study has shown that the ability to judge empathy in healthy females with low 2D:4D was significantly reduced using standard measures (‘reading the mind in the eyes’ test of empathy) after being given a sublingual dose of testosterone (van Honk *et al.* 2011). It has been recently proposed that high androgen profiles may down-regulate neural pathways linked to empathising and social bonding (Carter 2007; van Honk *et al.* 2011; Guastella *et al.* 2010; also see Rilling *et al.* in press). This indicates that correlations between 2D:4D and androgens levels in adults may not just be about absolute levels of androgen (e.g., testosterone) but is also associated with the

overall responsiveness of the hypothalamic-pituitary gonadal axis (HPG) that is programmed by PAE early in foetal life (Pfeiffer 1936; Fowden and Forhead 2009).

Prenatal androgens, however, only form part of the PAE (Breedlove 2010). The androgen receptor gene (ARG) provides the mechanisms for converting hormones into tissue responses (programming) via a cascade of interactions (Hermanson *et al.* 2002; Zuloaga *et al.* 2008; Fowden & Forhead 2009). Hormonal receptors are distributed in different patterns within and upon the surface of organs (Adesanya-Famuyiwa *et al.* 1999; Roselli *et al.* 2001). The sensitivity of the X-linked ARG is dictated by repeat sequencing of a polymorphic polyglutamine stretch encoded by the cysteine, adenine, and guanine (CAG) nucleotides. These polyglutamine regions are involved in interactions between ligands that change the affinity of the receptor to coactivator proteins (Hermanson *et al.* 2002). Up-regulation of hormone receptors during critical phases of tissue programming sets off a cascade of transcription events that correspond to interactions between the level of hormones present and sensitivity of the receptors to those hormones. Evidence from molecular genetics indicates that mean CAG repeat length (CAGn) is associated with speed at which the hormone receptor complex bind to DNA; short CAGn sequences are associated with high affinity of the ARG to coactivators and thus are more sensitive to androgens. In contrast, longer CAGn sequences appear to have inhibitory effects and are associated with low ARG responsiveness to androgens (Westberg *et al.* 2001; Manning *et al.* 2003a; also see Hermanson *et al.* 2002).

In humans the normal range of CAGn is 11–30 with a mean of 21 (Manning 2007b). CAGn differs between populations (Kittles *et al.* 2001) with an apparent positive selection towards CAGn >21 (but <30) across human groups. Variation in CAGn within the normal range appears to be differentially linked to sexual selection. Short CAGn are linked to higher aggression and impulsivity (i.e., Rajender *et al.* 2008; Aluja *et al.* 2011), poor social attachment and poor relationship quality (Comings *et al.* 2002), lower intelligence (Kooy *et al.* 1999) and higher sperm quality (von Eckardstein *et al.* 2001, but see Komori *et al.* 1999.). In contrast, longer CAGn repeats have been linked to lower fertility in males (von Eckardstein *et al.* 2001), under masculinised genitalia (Lim *et al.* 2000), increased neuronal speed (Manning 2007b), but susceptibility to neurological disease (Stanworth *et al.* 2008; Hickey *et al.* 2002). In humans strong associations between CAGn and diseases suggest that variation in CAGn within our species can only occur within a limited range (Manning 2007b), as extremes of CAGn (very short and very long repeat sequences) are associated with rapid loss of function (Manning 2007b, Buchanan *et al.* 2004). It is not known if

polymorphisms of the ARG are associated with differences in reproductive potential or disease in other primate species.

The sensitivity of the ARG also plays a critical role in the homeostatic mechanisms of circT in healthy adults. CircT levels are controlled by a feedback mechanism and reactions to circT are tuned by CAGn (von Eckardstein *et al.* 2001). Longer CAGn are associated with higher baseline circT. This relationship adheres to the hypothesis that a less reactive ARG (longer CAGn) leads to increased *insensitivity* to circT leading to reduced suppression of hormonal feedback mechanisms in the HPG, which ultimately results in higher baseline circT (Krithivas *et al.* 1999; Stanworth *et al.* 2008). In contrast individuals with short CAGn have lower baseline circT but when surges in circT are required, such as in the anticipation of sexual or aggressive encounters, peaks in circT occur much faster and are higher in these individuals; shorter CAGn induce larger physiological effects from the same levels of androgens compared to individuals with high CAGn (Roney *et al.* 2010). Shorter CAGn have been linked to ASD (Henningsson *et al.* 2009; Schmidtova *et al.* 2010) and interactions between excessively high circT and shorter CAGn may induce predispositions towards higher aggression and precocious puberty (Tordjman *et al.* 1997).

There is now large body of evidence linking low 2D:4D (inferred high PAE) with sexually selected traits in humans and other primates (i.e., aggression, competitiveness, dominance). Low 2D:4D appears to be linked to shorter CAGn in human males (Manning *et al.* 2003a; but see Hurd *et al.* 2011). Based on these findings we might expect low 2D:4D to correlate with low baseline circT due to increased tone of the HPGA induced by short CAGn, but correlations have been contradictory between 2D:4D and circT and a recent meta-analysis found no relationship between human 2D:4D and testosterone levels in humans (Hönekopp *et al.* 2007; also see Muller *et al.* 2011; McIntyre *et al.* 2011). This may not be surprising given the liability of circT and the analytical difficulties this variability presents (see Gray *et al.* 1991; Hönekopp *et al.* 2007). In addition, if 2D:4D reflects PAE on *sensitising* the HPG to adult hormones, absolute levels of testosterone would not necessarily correlate with 2D:4D within individuals. It may be expected, however, that detecting associations between 2D:4D, circT and CAGn will be increased by looking across species because averaging circT (for a species) should dampen the effect of any confounds linked to intra-specific variation and localised methodological error.

Through primate evolution there has been a decrease in sensitivity to circT signalled by longer CAGn (Choong *et al.* 1998; Hong *et al.* 2006; Fig. 6.1a). CircT levels also show phylogenetic differences (Coe *et al.* 1992; Fig. 6.1b) and more competitive species have

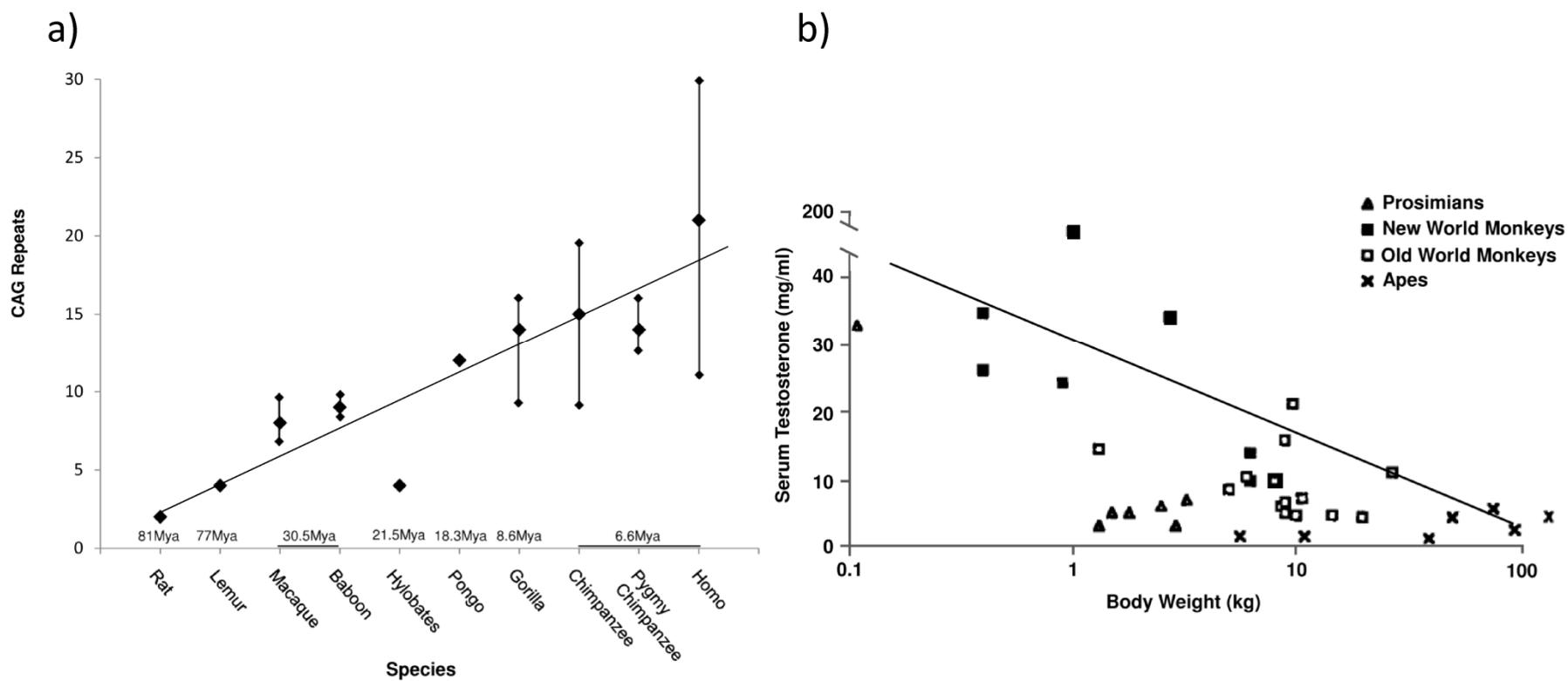
higher circT to support more intense levels of sexual selection (Klein 2000) these species also have larger brain size (Shultz & Dunbar 2007). Despite the understanding that androgens support sexually selected behaviours associations between 2D:4D, circT and CAGn have not been investigated. This study aims to draw together this evidence to see how it informs our knowledge of primate sociality.

6.1.3: Aims and predictions

Aim: To look at the patterns of relationships between species-level 2D:4D, circT and ARG sensitivity across primates. I also include data on salivary testosterone (salT) and fecal testosterone (faecT). General predictions:

- 1) It is predicted that measures of testosterone will be higher in males than females (Gouchie & Kimura 1991) and testosterone measures will be higher in NWM than Old World primates (Coe *et al.* 1992). Following the findings of Manning *et al.* (1998) and Roney *et al.* (2004) we might expect low 2D:4D (inferred high PAE) to be associated with high testosterone. Although evidence from Hönekopp *et al.* (2007; Muller *et al.* 2001; McIntyre *et al.* 2011) suggests that no relationships will be found.
- 2) Species with high 2D:4D may have longer CAGn and high testosterone (due to reduced neuro-endocrine feedback; Krithivas *et al.* 1999; Stanworth *et al.* 2008). Species with low 2D:4D may have short CAGn, according to the findings of Manning *et al.* (2003a) and lower background levels of testosterone. However, there is evidence to suggest that low 2D:4D and short CAGn may also be related to higher testosterone (see Roney *et al.* 2010).
- 3) We might expect stronger relationships between 2D:4D and CAGn than measures of testosterone because both digit ratio and the androgen receptor gene are stable within individuals and vary less within species compared to testosterone levels.

Figure 6.1: a) Variation in mean CAG repeats and divergence times in primate evolution (rat used as outgroup comparison). Increased mean CAG repeats (CAGn) indicate increasing insensitivity to androgens. Increased range of CAG repeats within species indicates polymorphic expansion in the androgen receptor gene (ARG). Divergence times taken from Steiper & Young 2006), Benton & Donoghue 2007 and Chatterjee *et al.* 2009. (CAGn references see Appendix 6.4). b) Circulating testosterone and body weight across the Primate Order. After Coe *et al.* 1992.



6.2: Materials and methods

6.2.1: 2D:4D data

Primate 2D:4D data (Appendix 2.5) was supplemented with human 2D:4D (means of population means) published in Manning *et al.* (2007a). For details on the haplorhine dataset see Chapter 2.

6.2.2: Literature search

6.2.2.1: Data collection: Testosterone

The search engine *Primate Lit* was used along with *Pub Med* and *Google Scholar*. The search terms inputted were: ‘serum testosterone’; ‘plasma testosterone’; ‘circulating testosterone’; ‘faecal testosterone’; ‘salivary testosterone’; ‘serum androgens’; ‘plasma androgens’; ‘circulating androgens’; ‘testosterone AND androgens’, ‘urinary testosterone’, ‘urinary androgens’. After a preliminary analysis of the data, urinary testosterone was omitted from the study as species levels were published in different values that could not be converted without data on urine volumes which are commonly published (ng/ml Creatinine; pmol/mg Creatinine).

I searched for studies that provided actual adult testosterone values of species for which I also had 2D:4D values (Appendix 2.5). Testosterone values were available for 9221 from 58 published papers circT (n=9095 individuals, 18 species), faecal T (n=104 individuals, 7 species) and salivary T (n=144 individuals; 4 species) (Table 6.1; Appendix 6.1). SsalT and feacT correlate closely with circT, although unlike circT the other sampling methods provide an average of testosterone for the preceding 24 hours (e.g., see Dloniak *et al.* 2004). Methods of assaying testosterone do vary across studies, but care was taken to ensure, as far as possible that the same elements were being measured. Testosterone values from graphs and values published in conference abstracts were not considered. Published testosterone values were in a variety of measures. For the analysis all testosterone values (circulating, faecal and salivary) were converted to the same measures (serum = ng/ml; faecal = ng/g, salivary = pg/ml).

Table 6.1: Species-level testosterone values. NS = not stated in the publication. ng/ml= nanograms per millilitre.

Species	Male			Female			Mean		Cohen's
	n	T	SD	n	T	SD	T	SD	d
<i>Homo sapiens</i>	4039	4.68	0.78	4257	0.49	0.13	3.28	2.20	7.49
<i>Pan paniscus</i>	2	1.2	NS						
<i>Pan troglodytes</i>	44	3.73	0.72	5	0.82	0.96	3.03	1.60	3.43
<i>Gorilla gorilla</i>	5	4.1	NS						
<i>Pongo pygmaeus</i>	2	2.4	NS						
<i>Symphalagus syndactylus</i>	2	1.6	NS						
<i>Hylobates lar</i>	1	1.6							
<i>Cercopithecus aethiops</i>	82	8.72	1.41						
<i>Macaca fascicularis</i>	210	10.28	4.31						
<i>Macaca mulatta</i>	179	6.17	2.93	42	0.40	0.25	5.02	3.52	2.78
<i>Macaca fuscata</i>	36	14.23	10.73						
<i>Macaca arcoides</i>	28	14.84	3.60						
<i>Papio hamadryas</i>	23	5.93	6.83	5	0.25	0.12	4.04	5.84	1.78
<i>Mandrillus sphinx</i>	12	14.22	10.28						
<i>Ateles geoffroyi</i>	7	8.53	6.32						
<i>Cebus apella</i>	12	34.30	NS						
<i>Saimiri sceurius</i>	90	78.53	49.69						
<i>Callithrix jacchus</i>	12	24.12	15.39						
	4786			4309					

Table 6.1: Species-level testosterone values continued. ng/ml= nanograms per millilitre.

Species	Faecal testosterone (faecT; ng/g)								
	Male			Female			Mean		Cohen's
	n	T	SD	n	T	SD	T	SD	d
<i>Pan troglodytes</i>	35	3.41	4.19						
<i>Symphalagus syndactylus</i>	1	0.36							
<i>Hylobates lar</i>	1	0.48							
<i>Macaca fuscata</i>	18	143.07	21.86						
<i>Papio hamadryas</i>	17	4.94	2.23	26	30.89	13.70	17.92	18.35	-2.77
<i>Alouatta caraya</i>	2	4.21	2.04	1	1.10	4.32	2.66	2.20	0.94
<i>Saguinus oedipus</i>	2	164.27	12.24	2	252.07	37.04	208.17	62.09	-3.18
	76			28					

Species	Salivary testosterone (salT; pg/ml)								
	Male			Female			Mean		Cohen's
	n	T	SD	n	T	SD	T	SD	d
<i>Homo sapiens</i>	77	95.16	36.93	44	78.24	44.38	88.39	35.50	0.41
<i>Pan troglodytes</i>	10	24.10	5.32						
<i>Macaca fascicularis</i>	4	54.72	17.93						
<i>Macaca mulatta</i>	9	42.40	9.71						
	100			44					

Variables that may alter testosterone (e.g., time of day, age, season) were not controlled for as the main aim was to obtain a general species means (for sex and for species). Published measures from fertile adults were used, but values from pregnant females were not used as extremes of testosterone in pregnancy may influenced species mean testosterone levels (see Beehner *et al.* 2005). Additionally, data was not used from cohorts of infertile males as it has been suggested that these values may be abnormal (Manning *et al.* 1998; also see Hönekopp *et al.* 2007). If the published study had applied a drug that could have influenced testosterone (e.g., alcohol), baseline testosterone levels or control group measures were used.

When groups of individuals were measured the mean values were used. In studies of multiple measures of the same individuals the mean for that individual was calculated (and the standard deviation; SD). The value was then pooled with the mean values from the other individuals (calculated in the same way) before an overall mean (and SD) was calculated. This method aimed to control for multiple measures and minimise pseudo-replication of data. In studies in which one overall value was given, that value was used along with the published variation measure (e.g., SD). Some testosterone values were published without a measure of SD or standard error of the mean values (see Table 6.1).

6.2.2.2: Data collection: Androgen receptor gene (ARG)

The same search engines (see above) were used for the ARG. The search terms: ‘androgen receptor gene’ and ‘primates’ and ‘CAG’ were used to look for CAGn values for non-human primate species, as opposed to expression of receptors on specific body tissues. The androgen receptor gene has been intensively studied in humans and the normal range of CAGn for our species has been well established (11-30 CAGn; Manning 2007b) and is consistently affirmed (e.g., Krithivas *et al.* 1999; Stanworth *et al.* 2008). It was not possible to include all human-based studies; so an extensive sub-sample of publications from the human literature was used. Values were taken from publications investigating normal populations or from control groups in the papers studying disease (as opposed to cohorts with a disease). Values were pooled to represent species-level CAGn. The total number of individual from which the androgen receptor gene was sequenced was 2848 from a total of 10 species (Appendix 6.2).

6.2.2.3: Other variables taken from the literature

Data on species body weight (kg) were taken from Lindenfors & Tullberg (1998) (see Appendix 3.1; 4.1). Data on substrate (e.g., arboreal, terrestrial, arboreal/terrestrial) were

taken from Plavcan and van Schaik (1992). To investigate relationships with strength of sexual selection (inter-male competition) variables were correlated with published data on categories of social systems (Plavcan 2004). Humans were classified as pair-bonded (PB; Dunbar 2010a). Total group size was also used as a measure of competition. Groups size measures were primarily taken from Smuts *et al.* (1987; see Chapter 4). Measures of endocranial volume were also included (ECV; Isler *et al.* 2008; see Appendix 4.1) as another measure of intra-specific competition (see Chapter 4).

6.2.3: Statistical analysis

For within species analyses (e.g., sex differences) testosterone levels (circT, salT, faecT) between males and females were compared using a one-way ANOVA and paired t-tests. Cohen's *d* was also used to estimate the effect size between the sexes, with a small size-effect indicated by 0.2, a medium size-effect by 0.5, and a large size-effect by 0.8 (see Dunst *et al.* 2007). Negative values indicate higher testosterone values in females compared to males (i.e., contrary to expectations).

Male testosterone values were correlated with male 2D:4D and male weight for species and, when available, female testosterone levels were correlated with female 2D:4D and female weight for species. Results of correlations between males and females 2D:4D and testosterone are placed in Appendix 6.3. Sexual differences in each testosterone category are investigated, but the main analyses focus on mean values for species. Male and female testosterone values within their categories (i.e., circT, salT, faecT) were pooled¹⁴ and converted into mean values for species with SD to indicate levels of variance within species (Table 6.1). A non-phylogenetically (non-phy) controlled general linear model (GLM) and phylogenetic least squares (PGLS) analyses were used to test relationships in testosterone between species (for details on PGLS analysis see Chapter 2). In cross species analyses between primate grades (i.e., apes versus old world monkeys) the effect of primate grouping is based on pair-wise comparisons between estimated marginal means for species in the GLM. In PGLS analyses primate grade was used as the fixed factor. For analyses of testosterone and CAGn male circT was used as this was the most comprehensive dataset. In the analyses testosterone was assigned as the dependent variables assuming that prenatal programming by PAE (reflected in the 2D:4D) influences testosterone levels. Results are described for PSGL analyses only because phylogeny must be controlled for. GLM results

¹⁴ Male and female testosterone were pooled within their respective categories to form mean values for species (i.e., categories were not mixed).

are tabulated so comparisons can be made between non-phy GLM and phylogenetically controlled PGLS analyses.

As the power of PGLS is compromised when analysing small sample sizes, sample with less than four species were not analysed. As cross-species analyses also necessitate a number of species per super family to provide statistical rigor so I chose to concentrate on circT and faecT only as the samples constituted species from all groups of haplorhines (apes, OWM, NWM) whereas salT sample had species values for two closely related apes and two closely related OWM (Table 6.1). 2D:4D was normally distributed (Kolmogrov-Smirnov Test; $p > 0.05$), but testosterone and body weight were not ($p < 0.01$). Analyses were therefore performed on log-transformed variables. There was no relationship between sample size and standard deviation within species ($F_{1,19} = 0.54$, $p = 0.47$, $R^2 = 0.03$).

Moran's I (a test of phylogenetic autocorrelation; see Chapter 2) indicated that phylogenetic relatedness significantly impacted species-level testosterone levels, 2D:4D ratios and body weight (Moran's I $p < 0.001$), and emphasized the important of using PGLS analysis.

6.3: Results

6.3.1: Sex differences in testosterone levels within species

Male and female testosterone levels were highly variable between species and within species (Table 6.1). Cohen's d values indicate medium to large effect sizes (Table 6.1). In accordance with predictions circT levels were lower in females when compared to males (Table 6.1) but these differences only reach significance in humans ($F_{1,8} = 80.16$, $p < 0.001$) and *M. mulatta* ($F_{1,19} = 14.89$, $p = 0.001$). Negative Cohen's d values for *P. hamadryas* and *S. oedipus* indicate that females had higher faecT than males in this sample (opposite to expected predictions), but these sex differences were not significant ($t = -1.84$, $p = 0.32$, $df = 3$). For salT human males had higher mean levels but they did not significantly differ from those of females ($F_{1,4} = 0.22$, $p = 0.67$).

6.3.2: Testosterone levels and 2D:4D across species

6.3.2.1: Circulatory testosterone (circT)

Higher male circT levels were associated with lower 2D:4D (inferred higher PAE) and lower body weight (Table 6.2; Fig 6.2). The effect was stronger for body weight ($R^2=0.58$) than for 2D:4D ($R^2=0.44$). Male 2D:4D and body weight were not significantly correlated (PGLS: $F_{1,16}=1.16$, $p=0.30$, $\lambda=0.93$). There were no significant effects for female circT when variables were analysed separately or analysed within the same model ($p>0.05$; Table 6.3). No associations with substrate categories were found ($p>0.05$).

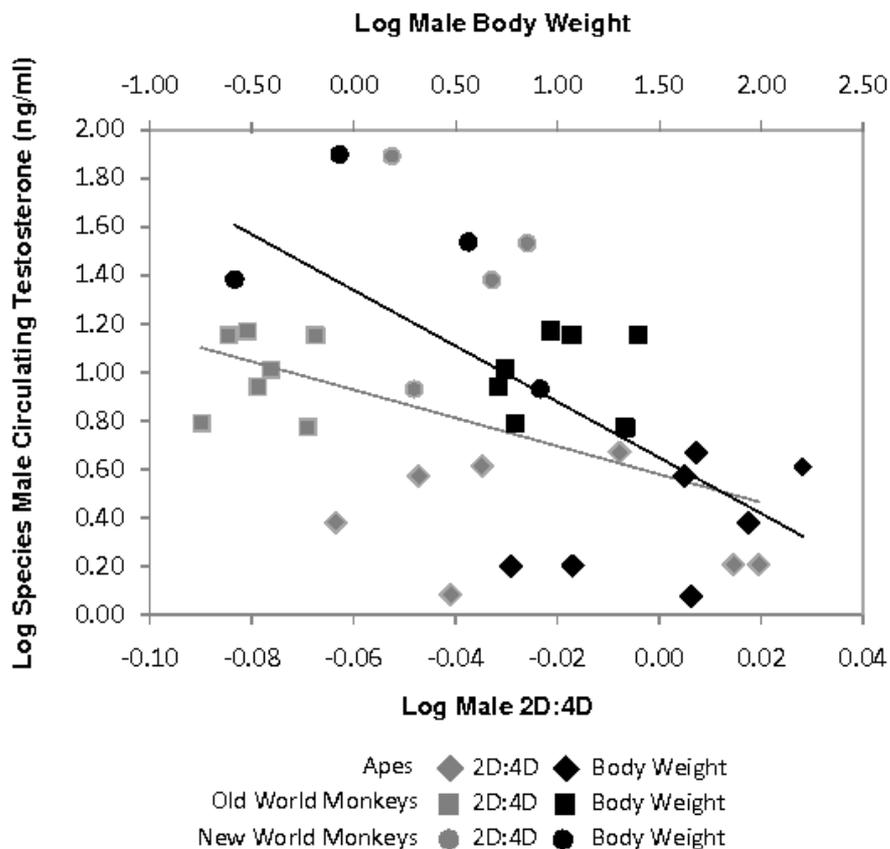


Fig. 6.2: CircT, 2D:4D ($R^2 = 0.44$) and body weight ($R^2=0.58$) across the primate order.

6.3.2.1.1: CircT and sexual election

Correlations between male circT levels and social system (PB and NPB) were not significant (PGLS: $F_{1,16}=0.46$, $p=0.51$, $\lambda=0.64$). Relationships with total group size¹⁵ were also non significant (PGLS: $F_{1,16}=0.88$, $p=0.69$, $\lambda=0.68$). 2D:4D did not correlate with total group size

¹⁵ Bigger group have higher levels of sexual selection (Shultz & Dunbar 2007).

(PGLS: $F_{1,16}=0.11$, $p=0.74$, $\lambda=0.96$). Looking across apes, there was no significant relationship between male circT and group size (PGLS: $F_{1,5}=0.80$, $p=0.41$, $\lambda=0$). Social system was not analysed within apes due to small sample size. Substrate use was not related to social system or total group size ($p>0.05$).

6.3.2.1.2: 2D:4D and circT in haplorhines

This analysis investigated how the main variables differed between evolutionary distinct primate groups (Table 6.4; Fig. 6.3). Substrate use was not related to any of the subsequent correlations ($p>0.05$).

OWM had significantly higher circT and significantly lower 2D:4D ratios than great apes (Table 6.4a,b; Fig. 6.3a,c). Body size did not significantly differ between these two super families in the PGLS analysis (Table 6.4c; Fig. 6.3b). NWM had significantly higher circT and significantly higher 2D:4D ratios than OWM (Table 6.4a,b; Fig. 6.3a,c). Body size was significantly smaller in NWM compared to OWM (Table 6.4c; Fig. 6.3b). NWM had significantly higher circT but significantly lower body sizes than apes (Table 6.4 a,c; Fig. 6.3a,b). 2D:4D ratios did not significantly differ between these two super families (Table 6.4b; Fig. 6.3c). Although NWM had significantly higher circT than both apes and OWM, circT did not significantly differ between groups when Old World primates (OW monkeys and apes) were pooled and analysed against NWM (Table 6.4a). This was also the case for 2D:4D and body size (Table 6.4b,c).

Within the apes patterns become more complex. Correlations between 2D:4D and circT were not significant (Non-phy: $F_{1,4}=0.38$, $p=0.57$, $R^2=0.07$; PGLS: $F_{1,4}=0.96$, $p=0.34$, $\lambda=0$). Compared to the most great apes (Hominidea), the Hylobatidae (*S. syndactylus* and *H. lar*) had high 2D:4D but low circT. Within the great apes male circT was significantly lower in *P. paniscus* compared to the other great ape species ($t=-16.85$, $p=0.002$, $df=4$, mean difference $=-0.74$). Correlations between 2D:4D and circT remained non significant after removing *P. paniscus* from the analysis (PGLS: $F_{1,4}=2.07$, $p=0.23$, $\lambda=1$). With *P. paniscus* and Hylobates removed there was a trend towards a positive relationship between 2D:4D and circT in the remaining great ape species, but relationships were not significant (Non-phy: $F_{1,3}=10.70$, $p=0.08$, $R^2=0.84$; PGLS; $F_{1,2}=4.27$, $p=0.18$, $\lambda=0$).

Model		Non-Phylogenetic (GLM)				Phylogenetic (PGLS)			
Dependent	Independent	F	p	R ²	sd	F	p	sd	λ
Male circT	2D:4D	5.98	0.03	0.59	15	4.61	0.05	15	0
	Weight	15.33	<0.01			12.47	<0.01		
Female circT	2D:4D	0.08	0.82	0.39	4	0.03	0.9	1	0
	Weight	0.31	0.68			0.09	0.82		
Male faecT	2D:4D	13.27	0.02	4	0.82	7.73	0.05	4	0
	Weight	15.23	0.02			8.86	<0.01		
Male salT	2D:4D	20.93	0.14	1.00	0.95	3.22	0.32	1	0
	Weight	16.01	0.16			2.52	0.36		

Table 6.2: Testosterone, 2D:4D and body weight (variables analysed together). Variables entered into the model together. 2D:4D and body weight were not correlated ($p>0.05$). Substrate removed from the analysis ($p>0.05$). For data on species variables see Table 6.1; Appendix 2.5; 3.1; 4.1) CircT=circulating testosterone; faecT= faecal testosterone; salT= salivary testosterone.

Model		Non-Phylogenetic (GLM)				Phylogenetic (PGLS)			
Dependent	Independent	F	p	R ²	sd	F	p	sd	λ
Male circT	2D:4D	3.08	0.10	0.11	16	0.46	0.51	16	0.64
Male circT	Weight	11.58	<0.01	0.38	16	1.32	0.27	16	0.59
Male circT	Substrate	1.17	0.30	0.01	16	0.77	0.48	16	0.73
Female circT	2D:4D	0.51	0.55	-0.19	2	0.09	0.79	2	1
Female circT	Weight	1.05	0.41	0.02	2	0.53	0.54	2	0
Female circT	Substrate	1.00	0.42	0.00	2	2.49	0.23	2	1
Male faecT	2D:4D	0.74	0.43	0.05	5	0.5311	0.50	5	0
Male faecT	Weight	1.39	0.29	0.06	5	0.992	0.37	5	0
Male faecT	Substrate	0.19	0.68	-0.16	5	0.51	0.634	5	0.67
Male salT	2D:4D	0.58	0.53	-0.16	2	1.28	0.38	2	0
Male salT	Weight	0.00	0.97	-0.50	2	0.00	0.98	2	0
Male salT	Substrate	3.51	0.20	-0.46	2	7.10	0.12	2	1

Table 6.3: Testosterone and study variables (variables analysed separately). For samples sizes see Table 6.1. CircT=circulating testosterone; faecT= faecal testosterone; salt= salivary testosterone.

Evolutionary Group	Non-Phylogenetic (GLM)				Phylogenetic (PGLS)			
	F	p	R ²	sd	F	p	sd	λ
a) Circulating testosterone								
Apes v OW monkeys	31.23	>0.01	0.72	12	26.66	<0.01	12	0
OW monkeys v NW monkeys	6.68	0.03	0.43	9	5.47	0.04	9	0
NW monkeys v Apes	31.18	>0.01	0.77	9	25.50	>0.01	9	0
OW primates v NW primates	11.99	<0.01	0.43	16	3.33	0.09	16	0.58
b) 2D:4D								
Apes v OW monkeys	19.16	<0.01	0.62	12	16.86	>0.01	12	0
OW monkeys v NW monkeys	42.67	>0.01	0.83	11	31.90	>0.01	9	0
NW monkeys v Apes	0.82	0.39	0.08	9	0.82	0.39	9	0
OW primates v NW primates	0.42	0.52	0.03	16	0.04	0.85	16	0.91
c) Body weight								
Apes v OW monkeys	6.96	0.02	0.37	12	0.58	0.46	12	1
OW monkeys v NW monkeys	8.03	0.02	0.47	9	6.57	0.03	9	0
NW monkeys v Apes	15.24	<0.01	0.63	9	12.47	>0.01	9	0
OW primates v NW primates	13.26	<0.01	0.45	16	2.59	0.13	16	0.62

Table 6.4: Comparisons between evolutionary distinct groups. For data on species variables see Table 6.1; Appendix 2.5; 3.1; 4.1. OW = Old World; NW = New World.

6.3.2.2: Faecal testosterone (FaecT)

Significant relationships between 2D:4D and faecT when both 2D:4D and body weight were entered into the model together (Table 6.2). Higher faecT levels were associated with lower 2D:4D (inferred higher PAE) and lower body weight (Table 6.2). Variables were not significantly associated when analysed separately (Table 6.3). Patterns were similar to those shown for circT. Females were not analysed due to small sample size (n=3 species).

6.3.2.3: Salivary testosterone (SalT)

There were no significant correlations between male salT and the other variables (Table 6.2; Table 6.3). Females were not analysed due to small sample size (n=1 species).

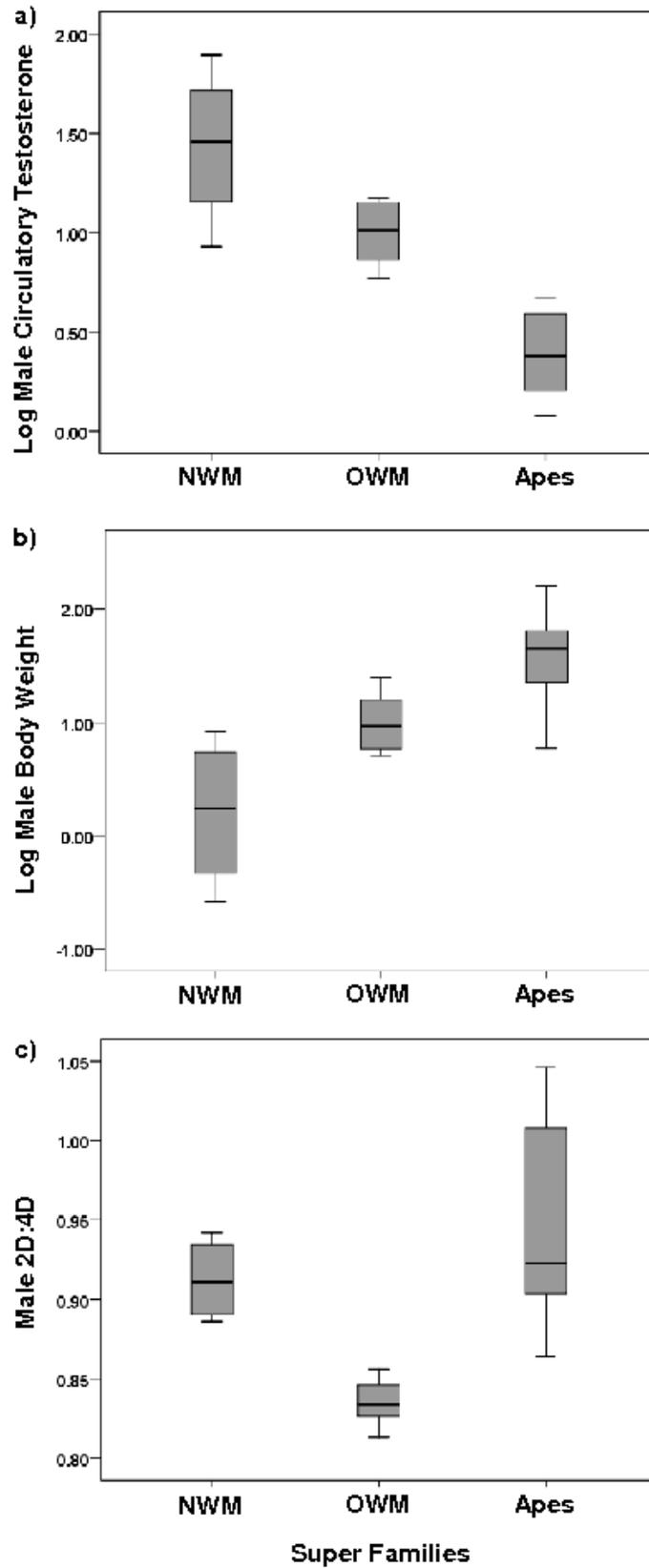


Figure 6.3: Differences in variables across super families. For sample details see Table 6.1. OWM = Old World monkeys; NWM = New World monkeys. 2D:4D values are not log-transformed in the figure, but were log-transformed in all analyses.

6.3.3: Associations with androgen gene receptor gene (ARG) sensitivity

ARG sensitivity is indicated by mean CAG repeat (CAGn) length; as CAGn repeats get longer sensitivity to testosterone decreases. Lemur species were also included in the sample to increase variation within primates (T levels for *Lemur catta* taken from Coe *et al.* 1992; Table 6.5). There are no published data on CAGn in NWM.

Species	2D:4D	sd	Mean CAGn	Range	Group Size
<i>Homo sapiens</i>	0.98	0.05	21	11-30	150
<i>Pan paniscus</i>	0.91	0.03	14	13-16	63
<i>Pan troglodytes</i>	0.91	0.05	15	8-19	53
<i>Gorilla gorilla</i>	0.91	0.04	14	8-17	7
<i>Pongo pygmaeus</i>	0.88	0.04	12	0	5
<i>Symphagus syndactylus</i>	1.05	0.07	4	0	3.4
<i>Hylobates agilis</i>	0.97	0	4	0	3.4
<i>Papio hamadryas</i>	0.86	0.04	9	8-10	38.1
<i>Macaca fascicularis</i>	0.84	0.03	8	7-9	15.6

Table 6.5: CAGn, ARG expansion range and mean group size across species. See Appendix 6.3 for references. Group sizes taken from Smutts *et al.* 1987 and Chapter 4.

2D:4D and androgen gene receptor gene sensitivity: Across species 2D:4D was highly statistically significantly related to CAGn but only when outliers – the gibbons (*S. syndactylus* and *H. lar*) - were removed from the analyses (Table 6.6b; Fig. 6.4a); higher 2D:4D ratios were associated with longer CAGn (a less sensitive ARG). Removing the *Lemur sp.* did not alter the result. The positive relationship was maintained within apes with gibbons excluded (Table 6.6b).

Testosterone and androgen gene receptor gene sensitivity: Across the whole sample no significant relationships were found between male circT and CAGn (Table 6.6a). Incorporating circT and weight into the same model did not alter significance (PGLS: $F_{1,4}=0.59$, $p=0.47$, $\lambda=0.76$). Body weight was significantly positively associated with CAGn with smaller species having shorter CAGn (Table 6.6c).

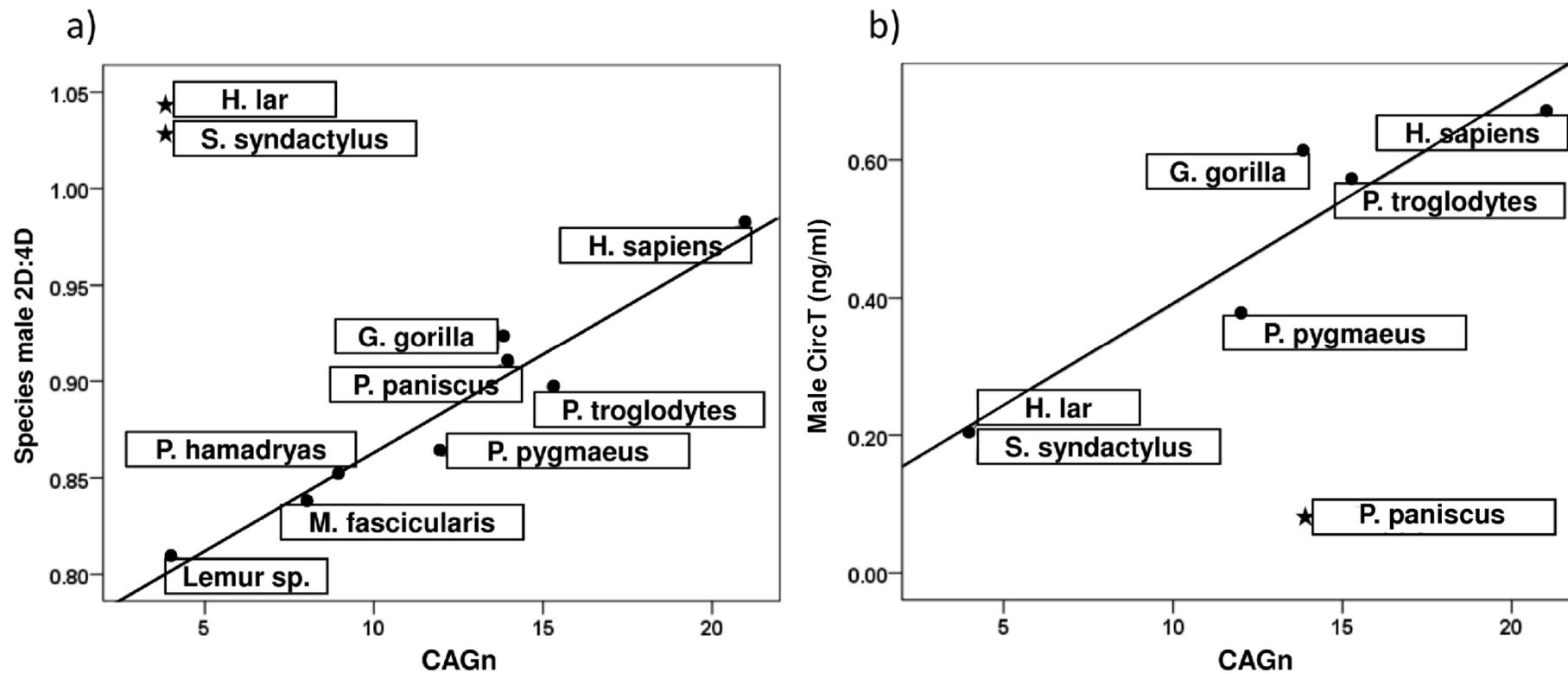
Table 6.6: CAGn and main study variables. For data on species variables see Table 6.1; Table 6.5; Appendix 2.5; 3.1; 4.1.

CAGn	Non-Phylogenetic (GLM)				Phylogenetic (PGLS)			
	F	p	R ²	sd	F	p	sd	λ
a) Circulating testosterone								
Whole sample (Table 7.5)	0.32	0.59	0.04	8	1.23	0.30	8	0.7
Apes only	4.35	0.09	0.47	5	3.11	0.14	5	0
b) 2D:4D								
Whole sample (Table 7.5)	0.00	0.99	0.00	8	1.26	0.29	8	1
Whole sample with gibbons removed	98.41	<0.01	0.94	6	52.33	<0.01	6	1
Apes only	2.08	0.21	0.29	5	0.04	0.53	5	1
Apes with gibbons removed	16.61	0.03	0.85	3	16.69	0.03	3	1
c) Body weight								
Whole sample (Table 7.5)	7.26	0.03	0.51	7	8.34	0.02	8	0
Whole sample with gibbons removed	1.88	0.23	0.27	5	5.93	0.05	6	0
Apes only	5.13	0.07	0.51	5	0.37	0.57	5	1
Apes with gibbons removed	0.732	0.46	0.2	3	0.36	0.86	3	1

*Substrate p>0.05

Gibbons = *S. syndactylus* and *H. Lar* (Table 6.5)

Figure 6.4: a) 2D:4D and CAGn across primate species. Outliers (stars) *H. lar* and *S. syndactylus* removed from the analysis ($R^2=0.94$); b) CircT and CAGn in apes. Increasing CAGn signals decreasing sensitivity of the ARG. *P. paniscus* is an outlier (star) and is removed from the analysis ($R^2=0.91$). All values were log-transformed.



Investigating great apes only, CAGn was significantly positively related to male circT after removing the outlier *P. paniscus* from the analysis (PGLS: $F_{1,4}=8.02$, $p=0.05$, $\lambda=1$; Fig. 6.4b).

Androgen gene receptor gene sensitivity and sexual selection: As data were only available for two pair-bonded species (from the same family) an analysis between CAGn and social system was not performed (see Methods). Lemurs were not analysed within the model. There was a positive association with CAGn and group size across species (PGLS: $F_{1,7}=5.12$, $p=0.04$, $\lambda=1$), significance was maintained when body weight was added into the model (PGLS: group size; $F_{1,6}=13.23$, $p=0.01$; body weight; $F_{1,6}=13.99$, $p=0.01$, $\lambda=0$); as group size and body weight increase, the ARG becomes less sensitive to androgens (signalled by longer CAGn; Fig. 6.5).

A near significant positive relationship was also shown between polymorphism in the ARG (indexed by the range of CAG repeats within the gene; see Table 6.5) and group size; increasing expansion of the ARG (increasing polymorphism) was associated with increasing group size (PGLS: $F_{1,7}=4.43$, $p=0.066$, $\lambda=1$). However, this trend was lost when body weight was added into the model (PGLS: group size; $F_{1,6}=3.14$, $p=0.16$; body weight; $F_{1,6}=0.07$, $p=0.79$, $\lambda=1$).

6.3.4: Associations with endocranial volume (ECV)

Across the sample there were no relationships between ECV (Isler *et al.* 2008) and 2D:4D and ECV and male circT (PGLS: $p>0.2$). Positive relationships were shown between ECV and body size (PGLS: $F_{1,8}=5.70$, $P=0.04$, $\lambda=1$). ECV and CAGn were positively related (PGLS: $F_{1,8}=14.14$, $p<0.01$, $\lambda=0.83$) and relationships remained significant when body weight was introduced into the model (PGLS: $F_{1,7}=5.04$, $p=0.056$, $\lambda=1$; Fig. 6.6). ECV was not related to group size (PGLS: $F_{1,7}=2.14$, $p=0.19$, $\lambda=1$). This result was not altered with the addition of body weight into the model ($p<0.1$).

In apes significant relationships between ECV and circT (PGLS: $F_{1,5}=7.12$, $p<0.04$, $\lambda=1$) and ECV and CAGn (PGLS: $F_{1,5}=27.7$, $P<0.01$, $\lambda=0$) were rendered non significant when body size was introduced into the models ($p>0.1$).

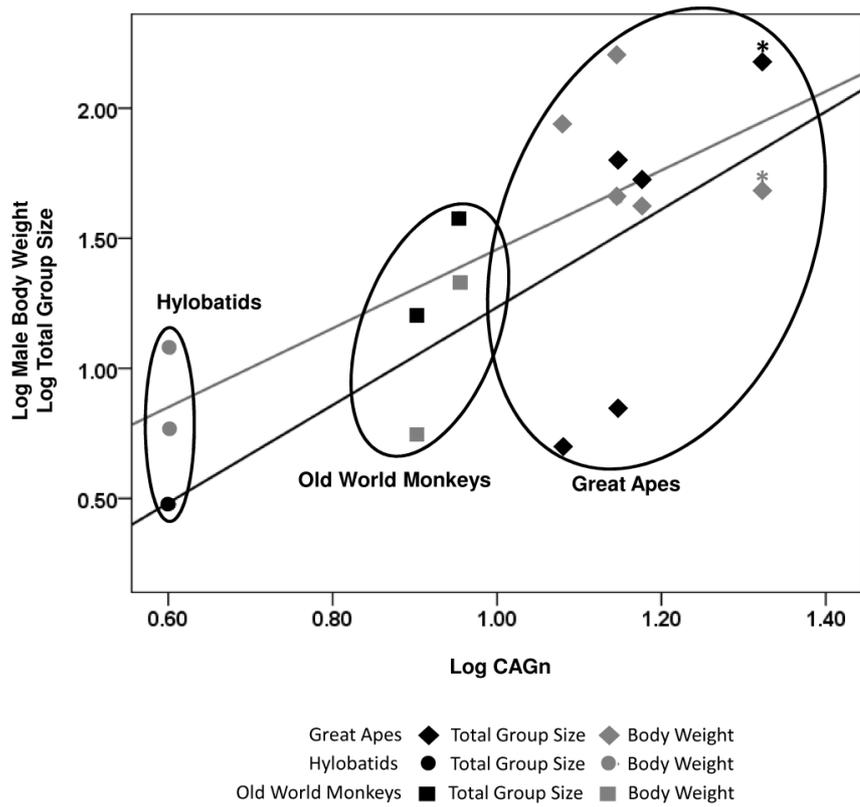


Figure 6.5: CAGn, total group size ($R^2=0.57$) and body weight ($R^2=0.57$). Humans indicated by a star above symbols.

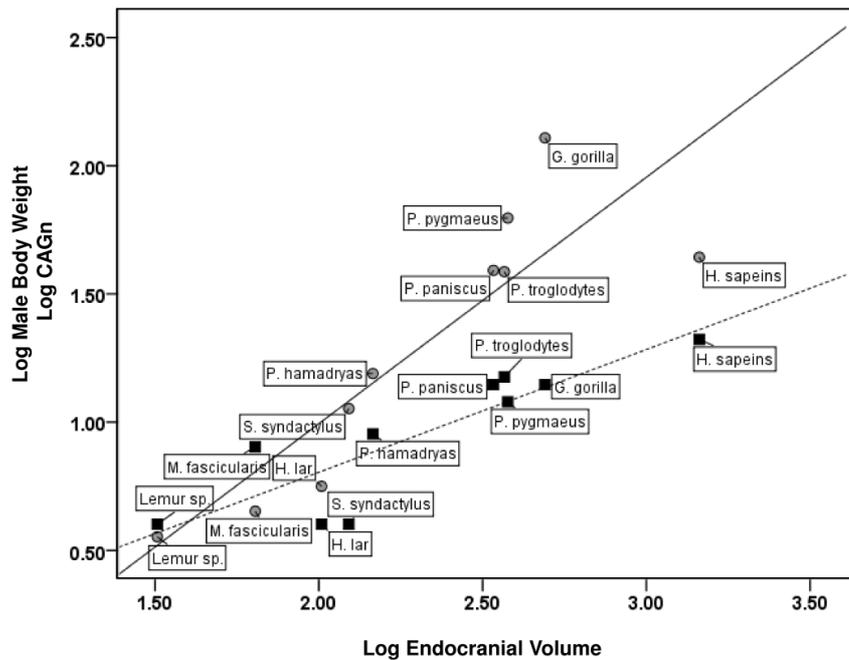


Figure 6.6: Endocranial volume, CAGn ($R^2=0.75$) and body weight ($R^2=0.77$). ECV and male species body weight = gray circles; ECV and CAGn = black squares (dashed line).

6.4: Discussion

The main findings of this study are; 1) Males had higher circT than females; 2) Across the sample lower 2D:4D (inferred higher PAE) and lower body weight was associated with higher circT, with the highest circT in NWM; 3) Within catarrhines, OWM and apes showed marked differences in androgen profiles with OWM species exhibiting lower 2D:4D (inferred higher PAE), higher circT and a more sensitive ARG than apes; 4) Decreasing sensitivity of the ARG (longer CAGn) was associated with larger group size and there was a positive trend ($p=0.07$) between expanding phenotypic variation of the gene (polymorphism) and group sizes; 5) Brain size was positively related to longer CAGn; 6) In general relationships with circT were poor.

Across the sample circT correlated with 2D:4D but only in conjunction with body size; with smaller Neotropical species having lower 2D:4D and higher circT (Fig. 6.2). The results for inter-specific circT levels support the findings of Coe *et al.* (1992) who demonstrated a strong link between circT and body size (also see Bernstein *et al.* 2008). Small NWM show highly elevated hormonal profiles compared to larger catarrhines (Coe *et al.* 1992; Fig. 6.3a). The reason for this pattern remains unclear but the pattern may be attributed to phylogenetic differences in reproductive physiology, steroid resistance and lower hormonal clearance in smaller monkeys (see Coe *et al.* 1992 for an overview; Gromoll *et al.* 2003). NWM also display a diversity of reproductive strategies that are underpinned by different physiological mechanisms to catarrhines (Zeigler & Snowdon 2000; Gromoll *et al.* 2003; Heyman 2003). For example NWM rely more on olfactory cues to up-regulate endocrine responses and potentiate sex-linked behaviours (Curley & Keverne 2005; Heyman 2003). These mechanisms imply that 2D:4D might be a less effective biomarker in Neotropical primates than in catarrhine primates. ARG data is lacking for platyrrhines; as such it was not possible to investigate how CAGn may play into relationships with the other study variables in NWM.

OWM species had lower 2D:4D (inferred higher PAE), shorter CAGn and higher circT than great apes. These traits may reflect the effects of high PAE on programming dominance-related behaviours that underpin the hierarchical, competitive social-styles that characterise OWM sociality (Di Fiore & Rendall 1994). OWM social organization is highly conserved and underpinned by female philopatry (Di Fiore & Rendall 1994; also see Strier 190). This has led to the development of strong female kin-bonded coalitions that compete with other female groups. Males migrate from their natal groups and compete for dominance with

unrelated males. These core characteristics appear highly conserved in OWM (Thierry *et al.* 2000; Di Fiore & Rendall 1994). However, levels of sexual selection and social behaviour do vary (e.g., asymmetry in hierarchies, affiliation, tolerance, retaliation; de Waal & Luttrell 1989; Thierry *et al.* 2000; Thierry 2004).

Within the genus *Macaca*, for example, studies mapping species-typical temperaments onto a phylogeny suggest that, while core-characteristics have remained largely unchanged, there has been an increase towards higher levels of aggression and increased nepotism in some lineages (e.g., *M. fascicularis* lineage (Thierry 1990; 2004; Thierry *et al.* 2000). Differences in levels of affiliation and prosocial behaviour between closely related species of *Macaca* may be linked to levels of the peptide oxytocin (OT; Rosenblume *et al.* 2002). In mammals OT has several functions related to reproduction but it is also linked to social bonding via promoting feelings of trust and calmness between individuals (Dunbar 2010b). In humans OT enhances abilities to read the mental states and emotions of others which govern empathetic responses (see Donaldson & Young 2008). New research on neural pathways linked to social bonding suggests that high PAE (indexed by 2D:4D) and high adult testosterone may reduce function in some regions of the emotion-reward circuitry in humans and non-human primates (see van Honk *et al.* 2011; Rilling *et al.* in press). In macaques OT (assayed from cerebral spinal fluid) was found to be higher in the more affiliative and gregarious bonnet macaque (*Macaca radiata*) than the more socially distant and aggressive pigtailed macaque (*Macaca nemestrina*; Rosenblume *et al.* 2002). It is noteworthy therefore that the bonnet macaque has lower circT than the pigtailed macaque¹⁶ and that OWM have higher circT and lower 2D:4D than apes (Coe *et al.* 1992; Fig. 6.3)

Apes have high 2D:4D, low circT and higher CAGn compared to OWM (Fig 6.4). The social systems of these two super families exhibit marked differences. Male apes are philopatric and form tolerant bonds with other males (see Ghiglieri 1987; Mitani 2009). Females migrate into the group and form bonds with other females (Newton-Fisher 2006). Great apes display dominance hierarchies but they are more egalitarian and less tightly enforced than those of OWM (Watts 2002; Foster *et al.* 2008). Sociality varies across apes (van Schaik & van Hooff 1996; Robbins *et al.* 2004; Mitani 2009) and this variation is evident between closely related species and may be linked to differences in species androgen profiles. The more tolerant and affiliative social style of female-bonded bonobos may be attributed to their lower PAE (inferred higher 2D:4D and down-regulated androgen response (lower circT; higher 2D:4D) compared to chimpanzees whose behaviour is governed somewhat by a competitive response (McIntyre *et al.* 2009; Wobber *et al.* 2010a; also see Parish & de Waal

¹⁶ There is no 2D:4D data available for *M. radiata* and *M. nemestrina*.

2000). Lower androgens in bonobos have been implicated in the more developed neural pathways linked to empathy (Rilling *et al.* in press). In humans testosterone has been shown to reduce the function of the same pathways and this may be linked to lower empathising skills in men compared with women (van Wingen *et al.* 2010; Knickmeyer & Baron-Cohen 2006).

If we consider the evidence that more masculine androgen profiles in humans are associated with higher levels of 'systemising' and rule-based thought processes we might hypothesise that the more competitive, rule-based dominance hierarchies of OWM might select for a high androgen profiles. In contrast, the generally tolerant social systems of great apes (especially bonobos) may necessitate a less reactive androgenic system that is (pre-) adapted towards reading emotions and thoughts (proto-empathizing) (de Waal & Aureli 1999; Fraser *et al.* 2008). The evolution of these cognitive processes in apes may have been important for maintaining social bonds and group cohesion when feeding strategies require the group to disperse (Aureli *et al.* 2008). The fission-fusion foraging strategies of great apes may have evolved in Miocene to allow larger group size to be maintained when food sources became patchy (Barrett *et al.* 2003; Aureli *et al.* 2008). Animals that exhibit a fission-fusion strategy have larger brain sizes and specialised adaptations for social communication (see Barrett *et al.* 2003; Aureli *et al.* 2008; Butti *et al.* 2009; Hakeem *et al.* 2009).

Larger group size and brain size were associated with decreasing sensitivity of the ARG (Fig. 6.4; Fig 6.6). A relationship trend ($p=0.07$) was also shown between increasing polymorphism of the ARG and increasing group size (see Fig 6.1a). Given the links between social behaviour and polymorphisms of other receptor genes (e.g., AVP and OT receptors; Hammock & Young 2005; Pritchard *et al.* 2007; Walum *et al.* 2008; Israel *et al.* 2008; 2009; dopamine receptor: Reuter *et al.* 2011) expansion of ARG provide the potential for increases in behavioural variation. Behavioural flexibility is likely to be adaptive in species that live in large populations because social complexity increases with increasing group size which placed an additional cognitive strain on maintaining social cohesion (Dunbar 1998; Dunbar & Shultz 2007b). Being able to adapt according to the actions of others may improve opportunities to increase reproductive fitness. Polymorphism of the ARG coupled with a reduction in androgen sensitivity (longer CAGn) may be associated with the diversity of social behaviours and increased intelligence in great apes compared to OWM.

Manning (2007b) has postulated that the expansion of CAGn through primate evolution may have occurred alongside changes in sexual selection and intelligence (Manning *et al.* 2003a; Manning, 2007a). In humans longer CAGn (within the normal range) may be associated with

increased neuronal transmission speed, faster thought processing and higher general intelligence (Manning 2007b). The findings of this study lend support to Manning's Feminised Ape Hypothesis of human evolution (Manning 2007a). It seems plausible that pre-adaptations towards 'feminisation' may have been a characteristic of the species that gave rise to the hominin lineage in the late Miocene (~6.6 Mya; Steiper & Hedges 2006).

As predicted higher 2D:4D ratios were associated with a reduction sensitivity of the ARG (see Manning *et al.* 2003a; Manning 2007a; but see Hurd *et al.* 2011). However, these relationships only became significant after the removal of the hylobatids (gibbons and siamangs). Hylobatids exhibit a short CAGn which may be a primitive trait retained to facilitate surges in circT required for mate competition (acquisition) and territorial defence in which aggression towards intruders can be lethal (Palombit 1993). Within great apes circT increased with decreasing ARG sensitivity (Fig 6.4b). This pattern fits with a lowered androgen response in apes; down-regulated ARG reduced hormonal feedback to HPG axis which results in higher baseline (Krithivas *et al.* 1999; Stanworth *et al.* 2008; Roney *et al.* 2010; see Fig. 6.4b). This within family (Hominidae) response differs from patterns across super families showing that circT decreases with decreased sensitivity of the ARG. Humans have the least sensitive ARG of all the apes. The recent discovery of a loss-of-function deletion within the ARG linked to the loss of penile spines in humans (McLean *et al.* 2011) is consistent with a down-regulation of androgen response (Dixson 1998; 2009) and provides additional support for a reduction in sexual selection in the human lineage (Manning 2007a).

Over all relationships between 2D:4D and CAGn were stronger than relationships with circT. This might be expected as testosterone is highly variable within individuals, between sexes, within species and between species (e.g., Gouchie & Kimura 1991; Coe *et al.* 1992; Alvergne *et al.* 2009). In contrast 2D:4D and ARG are much more stable throughout life. The lack of a relationship with circT does not, however, negate the fact that PAE programme neuro-endocrine axes (Pfeiffer 1936 Fowden & Forhead 2009; Hönekopp *et al.* 2007). The findings from this study are consistent with those in humans showing links between 2D:4D and ARG sensitivity (Manning *et al.* 2003a; but see Hurd *et al.* 2011) but no relationship between 2D:4D and testosterone (Hönekopp *et al.* 2007; Muller *et al.* 2011; McIntyre *et al.* 2011).

6.4.1: Summary

The advantage of species-level investigations is their ability to circumvent many of the localised extraneous effects that can obscure patterns within species. This approach has

allowed me to theorise on how phylogenetic differences in androgenic mechanisms might impact primate social systems and hominin social evolution. The paucity of data, however, makes interpretation speculative. Furthermore it is not known if 2D:4D is reflective of combined ARG effects and prenatal androgen levels (i.e., PAE), which is inferred in this thesis¹⁷ or only prenatal androgens or only ARG sensitivity (see Breedlove 2010). Nevertheless associations between higher and lower androgen profiles between catarrhine lineages are consistent with the sexually selected behavioural profiles we might expect to arise from them.

The differing circT and PAE between OWM and apes may underpin differences in social organization and social bonding adaptations (Barrett *et al.* 2003). Adaptations to species-level circulating androgens and PAE (including ARG sensitivity) may provide a mechanism by which sociality can be altered although the extent of these changes are likely to be limited by phylogenetic conservation of core characteristics (selecting environment canalization model; Eshel & Matessi 1998; Waddington 1942). It is possible that the Miocene lineage that gave rise to hominins may have already expressed a down-regulation in androgen response which predisposed hominins to (eventually) evolve more cooperative and egalitarian social systems (Aiello & Dunbar 1993; Dunbar 2010a; also see Hare 2004; Hare & Tomasello 2005a).

¹⁷ Breedlove (2010) has recently coined the phrase ‘prenatal androgen stimulation’.

Chapter 7

2D:4D and social systems in extinct hominids and hominins¹⁸

7.1: Introduction

Predicting social systems of extinct primates and hominins is crucial for understanding human palaeobiology, but is fraught with difficulty. Analyses of the haplorhine dataset show that 2D:4D co-varies with sexual selection and is a strong predictor of social systems and intra-sexual competitive behaviours, but is unrelated to sexually selected anatomical characters. This information is used as the basis of a methodology that enables the systems of extinct hominids to be predicted from fossilised digit bone ratios.

7.1.1: Predicting social systems of extinct primates and hominins

Sexual dimorphism in canine and skeletal features is the main method used to predict social behaviour in extinct primates (Plavcan & van Schaik 1992; Lockwood *et al.* 2007). In species in which males compete strongly for females, high male reproductive skew (e.g., when only one or a few males sire offspring within the group) manifests as sexual dimorphism in canine and body size; where intra-sexual competition is reduced, such as with social monogamy, dimorphism in these characters is reduced. Although this model provides a rule-of-thumb for most extant primates (Plavcan 2004; see Chapter 4), it is more problematic to apply to extinct primates, including hominins. Firstly, canine dimorphism is reduced in hominids compared to other primates (Reno *et al.* 2003; Plavcan *et al.* 2009). Secondly, estimates of skeletal size dimorphism from small, and often spatially and temporally dispersed fragmentary fossils, are prone to error (Plavcan 2004; Gordon *et al.* 2008). Furthermore, evidence suggests that sexual dimorphism in extinct primates may exceed levels in extant species (Scott *et al.* 2009); this indicates either a broader range of social systems in extinct species or potential systematic biases in estimating dimorphism.

The controversy resulting from inferences based on dimorphism in postcranial remains is exemplified by predictions of social systems for *Australopithecus afarensis*, which range from monogamous (Lovejoy 1981; Reno *et al.* 2003) to highly promiscuous (Lockwood *et*

¹⁸ Citation for this chapter: Nelson, E., Rolian, C., Cashmore, L. & Shultz, S. 2011. Digit ratios predict polygyny in early apes, *Ardipithecus*, Neanderthals and early Modern Humans but not in *Australopithecus*. *Proceedings of the Royal Society B.*, 278:1556-163.

al. 2007; Gordon *et al.* 2008). The debate has been reviewed in detail in a series of publications (Reno *et al.* 2003; Plavcan *et al.* 2005; Reno *et al.* 2005; Scott & Stroik 2006; Gordon *et al.* 2008) and hinges on a number of methodological issues that could significantly impact the estimated levels of dimorphism (reviewed in Gordon *et al.* 2008).

The story is not much clearer for other early hominins. Most recently, evidence has been put forward for low canine and body dimorphism in *Ardipithecus ramidus* leading to proposal that human-like, pair-bonded characteristics evolved early and therefore could be a cardinal trait of the hominin lineage (Lovejoy 2009). However, based on facial dimorphism and maturation rates, a gorilla-like harem social system has been proposed for the later *Paranthropus robustus*, (Lockwood *et al.* 2007), which appears to have differed from that of *Australopithecus africanus* (see Lockwood *et al.* 2007). Recently a *Pan*-like social system has been put forward for *Au. africanus* based upon strontium isotope analyses of dental fossil remains (Copeland *et al.* 2011). Marked levels of sexual dimorphism may also have characterised some populations of *Homo erectus* (Spoor *et al.* 2007; Ruff 2010) and *Homo heidelbergensis* (Arsuaga *et al.* 1997); dimorphism only approaches human-like levels in *Homo floresiensis* (Brown & Maeda 2009) and *Homo neanderthalensis* (Trinkaus 1980). Inferring social systems in stem hominins and hominoids is complicated by the fragmentary nature of Middle Miocene fossils (Begun 2004a) and is confounded further by the uncertainty of taxonomic assignment of fossils during this period (Wood & Harrison 2011). Given the difficulties associated with conventional methods of estimating social systems in fossils, employing alternative markers of sexual selection should be a key focus of research.

7.1.2: 2D:4D: an appropriate marker for social system?

The second-to-fourth digit ratio (2D:4D) is a proposed marker for prenatal sex hormones (Manning 2002a; 2007a), with lower ratios associated with higher prenatal androgen effects (PAE). In humans 2D:4D is sexually dimorphic such that within a population digit ratios are generally lower in males than females (Manning 2002a; 2007a). These relationships are supported by evidence of lower 2D:4D ratios in human with disorders linked to high PAE, such as autistic spectrum conditions (Manning *et al.* 2001), congenital adrenal hyperplasia (Hönekopp & Watson 2010), polycystic ovary syndrome (Cattrall *et al.* 2005; but see Lujan *et al.* 2010a; 2010b) and higher 2D:4D ratios in genetic males with androgen insensitivity syndrome (Berenbaum *et al.* 2009).

In humans, low 2D:4D is associated with dominance related behaviours in both sexes (see Chapter 5). Population differences in 2D:4D have been linked to marriage systems, with

polygynous populations expressing lower 2D:4D ratios compared to more monogamous groups (Manning 2007a). In non-human primates 2D:4D is strongly related to social system with; pair-bonded monogamous species have significantly higher digit ratios than in non promiscuous and more competitive species. These relationships appear to be particularly strong in hominoids (McIntyre et al. 2009; see Chapter 3).

The fact that 2D:4D is unrelated to anatomical measures of sexual selection (see Chapter 4) might suggest the 2D:4D keys into different developmental pathways and may therefore circumvent the pitfalls associated with the more conventional methods of predicting social systems (e.g., skeletal and canine size dimorphism). As the lengths of the underlying skeletal components of the digits (phalanges) are related to overlying soft-tissue finger length (Manning 2002a) the potential exists to use digit bone ratios, alongside the results of haplorhine 2D:4D studies, to investigate social behaviour in extinct primate species. The proximal phalanges are the most likely of the proximal phalangeal (PP) bones to be fossilised (because they are the largest) and can be assigned to the correct digit with confidence (Landsmeer 1955; Susman 1979). Here I investigate relationships between species-level manual PP ratios and social systems in extant hominoids and then use fossil bone ratios to predict the social systems of extinct hominoids.

7.1.3: Aim of the study

The aim of this study is to use knowledge of variation in 2D:4D ratios from extant hominoids alongside an extensive sample of hand bone ratios and discriminant function analysis with the objective of predicting the social systems from the fossilised hand bone ratios of five extinct hominids (hominins and hominoids) and one early anatomically modern human.

7.2: Materials and methods

7.2.1: Extant sample

Samples of extant ape species (contemporary humans and non-human hominoids) were taken from museum collections (scanned bones) and data on contemporary humans was supplemented with archaeological material (Table 7.1). All specimens were of known sex, in good condition with no visible skeletal pathologies. Metacarpals, proximal and intermediate phalanges were present in most specimens.

Table 7.1: Comparative sample of 2PP:4PP ratios. PB = Pair-bonded; NPB = Non Pair-bonded; INT = between PB and NPB. *Homo sapiens*: CNH, NNH, EC, GR, GC; *Pan troglodytes*: PCM, ANH, NNH, MCZ; *Gorilla gorilla*: PCM, ANH, NNH, MC; *Pongo pygmaeus*: NNH, BSM, MCZ; *Hylobates lar*: MCZ. CNH: Cleveland Museum of Natural History (Hamann-Todd); NNH: National Museum of Natural History (Terry Collection); EJ:Ecija; GR: Greenwich; GR: Great Chesterton; NW; PCM: Powell-Cotton Museum; ANH: American Museum of Natural History; MCZ: Harvard Museum of Comparative Zoology; BSM: Bayerische Staatssammlung, Munich; UT: Dept. of Anthropology, University of Texas.

Extant Species	Social System	n	Male		n	Female		Cohen's <i>d</i>	n	Species	
			2PP:4PP	sd		2PP:4PP	sd			2PP:4PP	sd
<i>Homo sapiens</i>	INT/PB	177	0.956	0.02	143	0.957	0.02	-0.06	320	0.957	0.03
<i>Pan troglodytes</i>	NPB	38	0.901	0.03	62	0.903	0.03	-0.06	100	0.902	0.03
<i>Gorilla gorilla</i>	NPB	62	0.918	0.03	50	0.920	0.02	-0.08	112	0.919	0.03
<i>Pongo pygmaeus</i>	NPB	15	0.901	0.02	31	0.911	0.02	-0.49	46	0.908	0.02
<i>Hylobates lar</i>	PB	26	1.009	0.01	20	1.009	0.01	-0.02	46	1.009	0.01

Table 7.2: Sources of contemporary human samples. ds = digital scan; dm = direct measures. The museum sample derived from the Hamann-Todd (Cleveland Museum of Natural History, USA) and Terry Collections (National Museum of Natural History, USA) (Rolian 2009). The archaeological samples derive from a medieval Islamic cemetery site at Écija, Spain; an Anglo-Saxon cemetery site Great Chesterford, UK; Greenwich Naval Hospital Cemetery, UK, dated to 1703-1869 (Cashmore 2009). The samples did not significantly differ in 2PP:4PP ($p>0.01$). Male and female 2PP:4PP ratios did not significantly differ within populations ($p>0.05$).

Sample	Method	Males			Females			Cohen's <i>d</i>
		n	2PP:4PP	sd	n	2PP:4PP	sd	
Museum sample	ds	106	0.956	0.02	107	0.956	0.03	-0.10
Great Chesterford, UK	dm	11	0.957	0.02	11	0.962	0.03	-0.12
Greenwich, UK	dm	28	0.960	0.02				
Écija, Spain	dm	32	0.955	0.02	25	0.955	0.02	0.04
Total	dm	177	0.957	0.02	143	0.958	0.02	-0.04

7.2.1.1: Museum (scanned) samples: *Homo sapiens*

Hand bones of 107 adult females and 106 adult males were also scanned from museum samples from the Terry Collection (National Museum of Natural History, U.S.A.) and the Hamann-Todd Collection (Cleveland Museum of Natural History, U.S.A.) (see Table 7.2). All specimens were of known sex, in good condition with no visible skeletal pathologies. Bones from both hands were available for 168 individuals and single hands were available for a further 45 individuals. Assignment of digit bone class (i.e., metacarpals, proximal phalanges, intermediate phalanges) is most accurate when all hand bones of that class are available (Case & Heilman 2006). Out of 213 only two individuals had a metacarpal (MC) missing (a MC 5 and a MC3) and three individuals had one of their PPs missing (one PP1 and two PP5s). An intermediate, and or distal phalanx was missing in 27 individuals

7.2.1.2: Museum (scanned) samples: Non-human hominoid species

Hand bones of 163 adult females and 141 adult males from four species from a number of museum collections were also scanned (Table 7.1). All specimens were of known sex, in good condition with no visible skeletal pathologies. Measurements were taken from one hand and all the metacarpals, proximal and intermediate phalanges were available and their positions assigned; distal phalanges were not identified (Rolian 2009).

Bones from museum specimens were assigned their anatomical positions by Dr. Campbell Rolian (Harvard University) as follows: If available, the articulated side of the skeleton was used to assign identity on the disarticulated side. Alternatively, the first, third and fifth proximal phalanges were identified based on size differences (Susman 1979). The 2PP and 4PP were then assigned in relation to the other three using the following criteria: (i) increased robusticity and presence of a bony flange on the proximal radial aspect of 2PP in humans and some African apes (Susman 1979; Christensen 2009), (ii) asymmetric head of the second compared to the fourth metacarpal, causing a bony lip on the ventral-radial aspect of the proximal end of 2PP for *Pongo*, African apes and humans (Landsmeer 1955; Susman 1979), (iii) larger surface area of the 2nd compared to 4th metacarpophalangeal joints (Susman 1979; pp. 218).

For scanning, bones were placed ventrally in anatomical position on a flatbed scanner (Microtek i320 ScanMaker) and imaged in TIFF format at 300dpi (Rolian 2009). Maximum length of the 2PP and 4PP were obtained by magnifying the image and placing the digital

ruler on the most distal point on the PP and then measuring to the most proximal point on the PP in pixels using TPSDig2 (Rohlf 2005). Each bone was measured twice and the mean values of bones provided the length value. I checked the assignment of the scanned phalanges (scanned by Dr. Campbell Rolian; see above), as far as was possible on a 2-dimensional image using the same morphological criteria as above.

Length measurements for adult human phalanges were also obtained from archaeological specimens (Table 7.2; data supplied by Dr. Lisa Cashmore). Bones exhibiting pathologies were excluded from the analysis, as were those that could not be confidently assigned using the morphological criteria (cited above). Phalanges were also rejected if the total number of hand bones (for the individual) was low, as this is known to lead to uncertainty in phalangeal assignment (Case & Heilman 2006). Sexing and ageing the burials was undertaken using standard osteological techniques (i.e., pelvic morphology and epiphyseal fusion; see Cashmore 2009). Additionally, if material could not be sexed, it was omitted from the dataset. Maximum length of hand bones was obtained using callipers (PowerfixTM; resolution 0.01 mm) by taking the straight distance from the middle point of the surface of the base to the topmost point of the head (Cashmore 2009). Intra-observer error for these data was shown to be within acceptable limits (Cashmore 2009).

Based on the anatomical features used to assign the proximal phalanges (outlined above), in the human sample (museum and archaeological collections combined) 2PP was shorter than 4PP in 97.7% of males and in 97.9% of females. This pattern (2PP<4PP) is consistent with phalangeal formulae in other great apes (Susman 1979).

7.2.2: Fossil sample

Flatbed digital scans were taken of high quality casts of one anatomically modern human (AMH) and four *Homo neanderthalensis* fossils archived at Washington University in St. Louis (permission to use the data was granted by Prof. Erik Trinkaus). AMH; Qafzeh 9 (right hand); Neanderthals; Kebara 2 (left hand); Shanidar 4 (right hand); La Ferrassie I (left hand); Le Regourdou (both hands). For the Le Regourdou fossils, the mean lengths of 2PP and 4PP from both hands were used to obtain a single value for the individual. Fossil phalanges were assigned, scanned by Dr Campbell Rolian and I measured them using the same methods as for the extant human sample. All scanned fossils were measured ten times and re-measured three months later to assess intra-observer error (see electronic supplementary material). Correct assignment of the scanned phalanges was also checked, as far as was possible on a 2-dimensional image, using the same morphological criteria above.

Data were also taken from the literature: *Australopithecus afarensis* A.L. 333 (n=1) (Bush *et al.* 1982; Alba *et al.* 2003); *Ardipithecus ramidus* ARA-VP6/500 (n=1) (Lovejoy *et al.* 2009); *Hispanopithecus laietanus* IPS18800 (n=1) (Almécija *et al.* 2007); *Pierolapithecus catalaunicus* IPS1350 (n=1) (Almécija *et al.* 2009); *Homo neanderthalensis* Spy II (Musgrave 1971). Note that among these specimens only the *Ardipithecus* hand fossils were found associated *in situ*. For the remaining fossils (except Spy II), phalangeal identification proposed in their respective descriptions was used. For the Spy Neanderthal measurements were taken from a published photographic image (Musgrave 1971).

Length measurements of the Spy II fossils 24c (2PP) and 24a (4PP) (Musgrave 1971; Semal *et al.* 2005) were taken ten times (for each bone) using set of digital callipers (Powerfix™; resolution 0.01 mm) from a photographic image (Musgrave 1971). The same publication also showed a photographic image of La Ferrassie I hand fossils which was one of the fossils that had been flatbed scanned image. The difference between 2PP:4PP ratios calculated from the photographed image of Le Ferrassie I and those calculated from the scanned image of the same hand fossils were compared by taking ten sets of phalangeal measurements (2PP, 4PP) from both images. Digital callipers were used to measure phalanges from the photographic image (mm) and tpsDig version 2 (Rohlf 2005) to measure the scanned phalanges (pixels). 2PP:4PP was calculated from these values (see below) and compared using a t-test. There were no significant differences in the 2PP:4PP ratio values calculated from measurements taken from the two different images ($t=0.76$, $p=0.49$, $df=9$) and the error was small (± 0.003 SE). A reasonable level of accuracy was therefore for Spy II fossils and thus included the Spy data measured from the photographic image in our sample.

I rejected fossils for which length looked like it had been altered significantly due to breakage or other post-depositional effects (e.g., the left 4PP in Qafzeh 9), and those in which the morphology of the bone looked to be abnormal (e.g., proximal end of the right 4PP in Kebara 2). However, fossils which may have been broken but whose length appeared minimally affected were retained (e.g., 2PP; ARA-VP-6/500-043) of *Ardipithecus* stated as having a length measurement of 43 mm ± 0.3 ; Lovejoy *et al.* 2009).

7.2.3: Ratio calculations

2PP:4PP was calculated for each individual (extant and extinct) by dividing the length of 2PP by the length of 4PP. In cases in which bones were available for the two hands, mean values of left and right 2PP:4PP were used to prevent pseudo-replication. A mean value

2PP:4PP and standard deviation for each extant species was calculated (Table 7.1). I tested for skew in the 2PP:4PP data using the Kolmogorov-Smirnov Tests; 2PP:4PP was normally distributed over the whole sample (species means: 0.28, $p=0.2$, $df=5$; individuals: 0.03, $p=0.19$, $df=535$). However, when individuals within species were analysed separately *Gorilla* data were skewed (0.11, $p<0.01$, $df=112$), so 2PP:4PP data for all the individuals in all the samples were log-converted.

7.2.4: Social system

The social system of extant species was defined by classifying each taxon as either pair-bonded monogamy (PB), which included species in which males usually mate with only one female, or polygynous, non pair-bonded (NPB), which included species in which males usually mate with more than one female (see Plavcan 2004). As contemporary humans do not fit strictly into a PB category (i.e., pair-bonds within a multi-male, multi-female social system) (Dunbar 2010a) separate analyses were performed with contemporary humans classified as PB and as intermediate (i.e., something other than PB or NPB).

7.2.5: Other variables

Data on extant ape 2D:4D ratios and associations with social systems were used as a comparative sample (see Chapter 3; Manning *et al.* 2007a). As substrate-use (e.g., arboreal; terrestrial; arboreal/terrestrial) is associated with variation in hand morphology (Jouffroy *et al.* 1993; Richmond 2007) categories of substrate were included in the analysis (Plavcan & van Schaik 1992) as a means of controlling for possible functional effects on digit ratios. Measures of species body weights were also included (Smith & Jungers 1997; Lindenfors & Tullberg 1998) to ensure 2D:4D was not being unduly influenced by size. Body dimorphism estimates were calculated by dividing male body weight by female body weight. Kolmogorov-Smirnov Tests indicated skew in body weight and body dimorphism data ($p<0.001$); skewed data were log-transformed.

7.2.6: Statistical analysis

I used t-tests to estimate differences between male and female species mean 2PP:4PP. Cohen's d (see Dunst *et al.* 2007) was used to assess the size effect between male and female species mean ratios within species (Table 7.1). A negative d value indicates lower male 2PP:4PP compared to females. Linear regression was used to compare species mean

2PP:4PP with species mean 2D:4D. This regression equation also allowed us to estimate 2PP:4PP from 2D:4D for a Zulu population (Manning *et al.* 2000a).

To control for phylogenetic effects, associations between extant species digit ratios (2PP:4PP and 2D:4D) and social systems were analyzed using Phylogenetic Generalized Least Squares (PGLS) analysis with an optimised lambda (using the Ape package in 'R') (see Chapter 2). In all PGLS analyses with social systems I checked to see if digit ratios correlated with body weight or substrate; these variables were removed from the model if not significant.

7.2.6.1: Monte Carlo method

For comparisons between extant and extinct taxa, a Monte Carlo resampling method was employed using custom-written routines in Matlab R2009b (Natick, MA) (Manly 1991). For fossil taxa with a sample size greater than $n=1$ (i.e., Neanderthals), the routine derives a distribution of population 2PP:4PP means in extant species based on the means of 10000 subsamples of n individuals (where n =number of individuals in the fossil sample) drawn randomly, with replacement, from each extant taxon. The distribution is then used to determine the probability of drawing a sample of each extant taxon of the same size as the fossil sample with a 2PP:4PP mean equal to or lower than the observed fossil population mean. The same approach was used to compare the extinct species with $n=1$: single individuals were drawn randomly from each extant taxon (10000 iterations), in order to derive 5% and 95% confidence levels for the distribution of 2PP:4PP ratios in each. For the Neanderthal sample the results were based on the percentage probability of sampling five individuals within the extant samples with the same 2PP:4PP ratios as the five Neanderthal fossils.

7.2.6.2: Discriminant function analysis (DFA)

DFA was used to predict the social systems of extinct taxa (individuals) based upon extant hominoid 2PP:4PP (individuals) and their known social systems. 2PP:4PP (logged values) was designated as the independent variable and social system (PB-NPB) as the grouping variable. As sample sizes for fossil species were very small (all but one represented by a single individual) a DFA was performed on the 2PP:4PP ratios of individuals (rather than mean for species values). As contemporary humans show some flexibility in social systems i.e., broadly pair-bonded (Møller & Welch 1990; Dunbar 2010a) DFA were run three times; once with humans assigned as PB, again with them assigned as intermediate (i.e., something

different from a PB and NPB social system) and lastly, they were omitted from the analysis. Based on 2PP:4PP data for extant species (Table 7.1); DFA predicted the social systems for the fossil species.

A step-wise DFA was also performed with both species mean 2PP:4PP and species mean body size dimorphism (logged values) inputted as dependent variables and social system as the grouping variable.

7.2.7: Testing for measurement error

Subsets of scanned human (n=25 males and n=25 females) and scanned non-human primate material (n=5 males and 5 females per taxon; total n=50) and all of the scanned fossils were re-measured using the same method after three months to check intra-observer reliability. To check that the scanning process itself did not influence the measurements 50 human phalanges from a collection at the University of Liverpool were scanned at the same resolution (Hewlett Packard 4470c Scanjet; 300 dip) as the bones scanned by Campbell Rolian. A week later the same sets of 50 phalanges were measured using digital callipers. The measurements were transformed into ratios (n=25 sets), and the ratios of the directly measured bones and the scanned bones were compared. Intra-class correlation coefficient (ICC) was used to test the repeatability of measurements (McGraw & Wong 1996).

7.2.7.1: Error estimation for the fossil sample

Phalanx length data of 50 humans (25 males and 25 females) and 60 non-human primates (30 males and 30 females) was pooled. Intra-class correlation coefficients (ICC) were performed on repeated measures from the scanned images (from the first and second measurement sets taken three months apart). All p-values <0.001, ICC=0.99 for 2PP ($F_{1,108}=721.96$); 1.00 for 4PP ($F_{1,108}=8575.96$); 0.979 for 2PP:4PP ($F_{1,109}=47.44$). Campbell Rolian also measured 30 scanned hands and the two datasets were compared; ICCs =1.00 for 2PP ($F_{1,27}=4060.57$); 1.00 for 2PP ($F_{1,27}=7015.56$); 0.993 for 2PP:4PP ($F_{1,28}=215.37$).

7.2.7.2: Error estimation for the fossil sample

ICCs were calculated for repeated measures of phalangeal length from scanned images of the fossils (three months apart). All p-values <0.001; ICC=0.999 for D2 ($F_{1,4}=721.79$); 1.00 for D4 ($F_{1,4}=857.96$); 0.993 for 2PP:4PP ($F_{1,5}=145.00$). Comparisons of ratios based on

scanned bone-sets (proximal phalanges in pixels) and direct measurements (in millimetres) did not significantly differ ($t=-1.91$, $p=0.07$, $df=24$).

7.3: Results

7.3.1: Sex differences in 2P:4PP in extant species

Consistent with the proposed relationships between digit ratios and PAE, within species males had lower 2PP:4PP ratios than females reflected in negative Cohen's d based on mean ratios (Table 7.1), although sex differences in 2PP:4PP were not significant ($p>0.1$) when individuals within species were compared (Table 7.3).

Extant species	t	p	df	Mean difference
<i>Homo sapiens</i>	0.49	0.62	318	0.001
<i>Pan troglodytes</i>	0.27	0.79	98	0.002
<i>Gorilla gorilla</i>	0.39	0.69	110	0.002
<i>Pongo pygmaeus</i>	0.16	0.13	44	0.009
<i>Hylobates lar</i>	0.09	0.93	44	0.004

Table 7.3: Comparisons between male and female 2PP:4PP (individuals) within species ($p>0.1$).

7.3.2: 2PP:4PP and 2D:4D and correlations with social systems

Ten primate hands were measured then dissected (see Chapter 2). The soft-tissue digit length for 2nd and 4th digits of each hand was each correlated against their respective proximal phalanx (right 2nd $R^2=0.85$; right 4th $R^2=0.83$; left 2nd $R^2=0.89$; left 4th $R^2=0.92$). These values indicate that soft-tissue digit length correlates highly with the proximal phalanx length of the same digit. Hominoid species mean 2PP:4PP was then regressed on to species mean 2D:4D ratios (see Chapter 3; Manning *et al.* 200a) and found the ratios to be highly correlated, despite 2PP:4PP being lower substantially than 2D:4D (with humans: $R^2=0.97$, $F_{1,4}=86.79$, $p<0.01$); without humans $R^2=0.96$, $F_{1,3}=49.23$, $p=0.02$).

Extant hominoid species were classified as pair-bonded (*Hylobates*) or non-pair-bonded (*Pan*, *Pongo*, *Gorilla*). 2PP:4PP was significantly lower in non pair-bonded (NPB) than pair-bonded (PB) species both when humans were classified as pair-bonded (PGLS, $F_{1,3}=11.55$, $p=0.04$, $\lambda=1$) and when they were removed from the analysis (PGLS, $F_{1,2}=49.78$, $p=0.02$, $\lambda=0$). These results are consistent with 2D:4D for the same species (with humans, PGLS $F_{1,3}=9.48$, $p=0.05$, $\lambda=1$, without humans, PGLS $F_{1,5}=20.85$, $p<0.01$, $\lambda=0$) (see Chapter 3). Correlations between digit ratios and body weight and substrate were not significant in PGLS analyses ($p>0.1$).

7.3.3: 2PP:4PP between extinct and extant hominids

Contemporary human mean 2PP:4PP was 0.957, $sd=0.02$ ($n=320$; $CL=0.92-0.95$), while Neanderthal 2PP:4PP mean was 0.928, $sd=0.03$ ($CL=0.89-0.96$). The Monte Carlo resampling analysis shows that only 19 out of 10000 resampled means in *H. Sapiens* are as low or lower than the Neanderthal mean (Fig. 7.1b). In other words, there is a ~0.2% probability of drawing a sample of five modern humans with a mean 2PP:4PP ratio as low as the observed Neanderthal mean (Table 7.4). The early AMH Qafzeh 9 is within range of contemporary human 2PP:4PP ratios (Table 7.4), but falls at the lower end of the range (Fig. 7.1a). The Qafzeh 9 2PP:4PP (0.935) is similar to (polygynous) Zulu 2PP:4PP (0.939); estimated by inputting 2D:4D values (0.95 ± 0.040) (Manning *et al.* 2000a) into hominoid regression equation.

Table 7.4 shows the probabilities (based on 10000 iterations) of sampling observed fossil 2PP:4PP values from each extant species. *Australopithecus* is within range of human 2PP:4PP, but falls outside the confidence interval of all other extant hominoids. In contrast, the Miocene apes and *Ardipithecus* have 2D:4D ratios that fall below the 5% confidence level for modern humans, but within the range of 2D:4D ratios for African apes and *Pongo*.

7.3.4: Predictions of social system for extinct hominids

Predicted social systems for Miocene apes and *Ardipithecus* were non pair-bonded and were stable across all discriminant function analyses (DFA) (Table 7.5; Fig. 7.2). Predicted social system for *Australopithecus*, early modern humans and Neanderthals with the highest 2PP:4PP ratios (Shanidar 4, Kebara 2, Le Ferrassie I), mirror the designated social system of contemporary humans (PB or intermediate), while the Neanderthals with the lowest 2PP:4PP ratios (Le Regourdou and Spy II) were classed as non pair-bonded in all analyses (Table 7.5; Fig. 7.1).

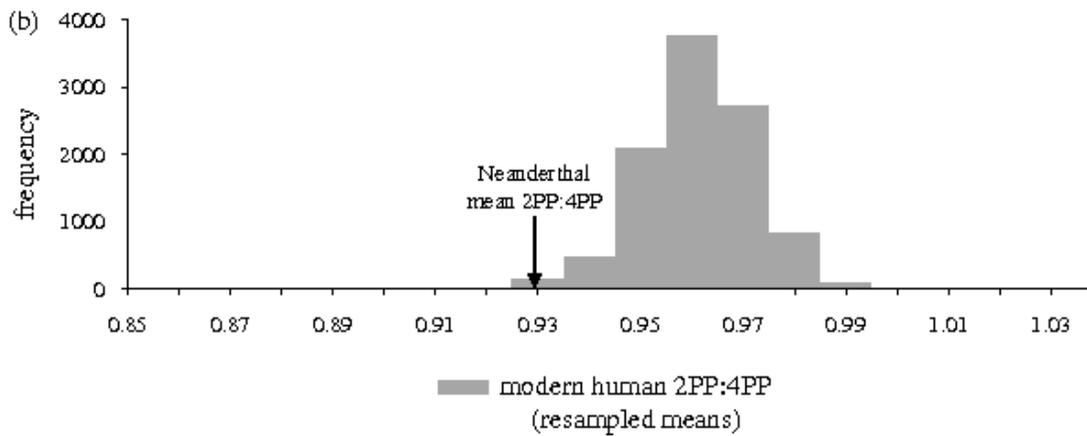
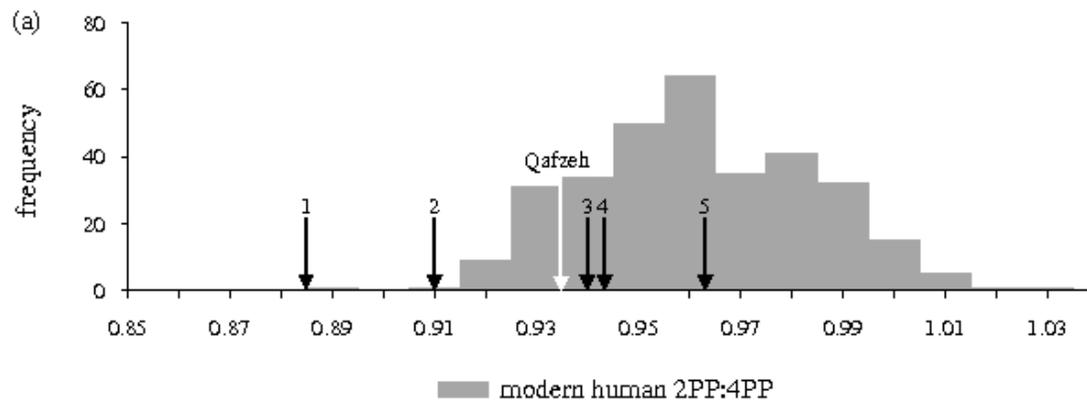


Figure 7.1: a) 2PP:4PP in the modern human sample (white arrow; Qafzeh 9) with Neanderthal fossils (Late Pleistocene) superimposed as black arrows: 1, Spy; 2, Le Regourdou; 3, Kebara; 4, La Ferrassie; 5, Shanidar. b) 2PP:4PP in the modern human sample with Neanderthal mean 2PP:4PP (black arrow). The Monte Carlo analysis shows there is a ~0.2% probability of drawing a sample of five modern humans with a mean 2PP:4PP ratio as low as the observed Neanderthal mean.

Table 7.4: Probability estimates. Percentage probabilities of sampling a 2PP:4PP ratio from the extant species (10000 iterations) that is as extreme as the observed fossil 2PP:4PP ratios. Neanderthal probabilities are based upon resampling five individual (see materials and methods for resampling procedures). WR = within range (i.e., between the 5% and 95% confidence levels (CL) for the extant population distribution).

Extant Species		<i>Homo sapiens</i>	<i>Hylobates lar</i>	<i>Pan troglodytes</i>	<i>Gorilla gorilla</i>	<i>Pongo pygmaeus</i>
2PP:4PP (n)		0.957 (320)	1.009 (46)	0.909 (100)	0.919 (112)	0.908 (46)
5-95% CL		0.923-0.995	0.992-1.028	0.858-0.947	0.873-0.949	0.878-0.940
Fossils (n)	2PP:4PP	Probability Estimates				
Qafzeh (1)	0.935	WR	0%	WR	WR	WR
Neanderthals (5)	0.928	0.19%	0%	1.59	WR	0.69%
<i>Australopithecus</i> (1)	0.979	WR	0%	0%	0.95%	0%
<i>Ardipithecus</i> (1)	0.899	0.14%	0%	WR	WR	WR
<i>Hispanopithecus</i> (1)	0.848	0	0%	4.31%	0%	0%
<i>Pierolapithecus</i> (1)	0.908	0.39%	0%	WR	WR	WR

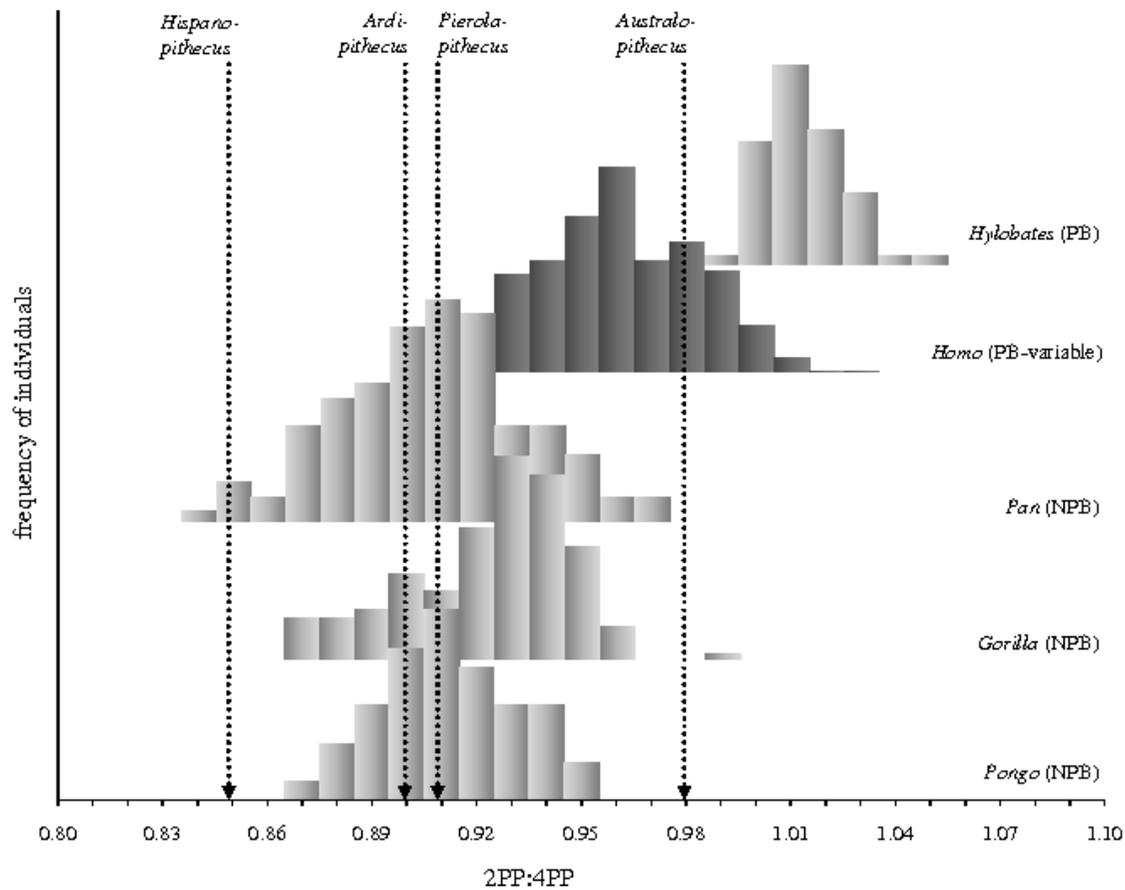


Figure 7.2: Extant ape 2PP:4PP with Miocene and Pliocene fossils transposed. Miocene apes and *Ardipithecus ramidus* are classified as non pair-bonded (NPB), while *Australopithecus* is classified as having a social system that is pair-bonded monogamous (PB) or intermediate i.e., between pair-bonded and non pair-bonded. Frequencies of individuals within species are scaled to the same dimensions; for sample sizes see Table 1).

Table 7.5: Discriminant function analysis: Predicting the social system of fossil hominids. PB=Pair-bonded; NPB=Non Pair-bonded; INT=something other than PB and NPB. Extant 2PP:4PP sample = *Homo sapiens*, *Pan troglodytes*, *Gorilla gorilla*, *Pongo pygmaeus*, *Hylobates lar*. Substrate and body weight $p>0.05$.

Species	Fossil	Predicted Social System		
		Humans as PB	Humans as intermediate	Humans removed
Anatomically Modern Human	Qafzeh	PB	INT	NPB
Neanderthal	Shanidar 4	PB	INT	PB
Neanderthal	Kebara 2	PB	INT	NPB
Neanderthal	La Ferrassie	PB	INT	NPB
Neanderthal	Le Regourdou	NPB	NPB	NPB
Neanderthal	Spy II	NPB	NPB	NPB
<i>Australopithecus</i>	A.L. 333	PB	INT	PB
<i>Ardipithecus</i>	ARA-VP	NPB	NPB	NPB
<i>Hispanopithecus</i>	IPS18800	NPB	NPB	NPB
<i>Pierolapithecus</i>	IPS1350	NPB	NPB	NPB
Wilks λ (df) P		0.52 _{1,622} $p<0.001$	0.40 _{,621} $p<0.001$	0.32 _{,302} $p<0.001$
Eigenvalue (Canoconical Correlation)		0.93 (0.70)	1.54 (0.78)	2.15 (0.82)
% of original cases correctly classified		82%	75%	99%

With humans removed from the analysis, 99% of original cases are classified correctly and all fossil specimens, except the Shanidar 4 Neanderthal and *Australopithecus*, are classified as non pair-bonded (Table 7.5). Comparisons between extant apes and Miocene and Pliocene hominids show *Australopithecus* to be positioned between pair-bonded monogamous gibbons and non pair-bonded apes, while *Ardipithecus*, *Pierolapithecus* and *Hispanopithecus* are all within the range of non pair-bonded great apes (Fig. 7.2).

For contemporary humans the percentage of original cases classified as correct in DFA was 56% when classed as PB and 42% when classed as intermediate. Thus contemporary human 2PP:4PP appears to sit in between NPB apes and the PB gibbons (Fig. 7.2). In NPB extant apes it was 98% for chimpanzees, 98% for gorillas, 100% for orang-utans and 100% for PB gibbons.

In addition, I also ran a step-wise DFA on extant species with species mean 2PP:4PP and species mean (logged) body size dimorphism as predictors of social system. 2PP:4PP was retained in the final model while body size dimorphism was dropped when humans were assigned as pair-bonded (2PP:4PP, Wilk's $\lambda=0.17$, $p=0.03$, $df=1$; body dimorphism, Wilk's $\lambda=0.54$, $p=0.21$, $df=1$) and when humans were assigned as intermediate (2PP:4PP, Wilk's $\lambda=0.018$, $p=0.02$, $df=2$; body dimorphism Wilk's $\lambda=0.54$, $p=0.54$, $df=2$). These results suggest that 2PP:4PP is a better predictor of social system in extant hominoids than body size dimorphism.

7.4: Discussion

Conventional methods of estimating social systems for fossil hominids have largely relied on estimations of sexual dimorphism in body size (e.g., Reno *et al.* 2003). This analysis suggests that digit ratio represents a more accurate predictor of social systems than body size dimorphism. Furthermore, the relationship between social system and digit ratio across extant haplorhines suggests that 2D:4D reflects links between prenatal androgen effects (PAE) and sexually selected social behaviours (see Chapter 3; McIntyre *et al.* 2009). These findings have been used to predict the social systems of extinct hominids from digit ratios calculated from fossil remains. Discriminant function analyses using 2PP:4PP of Middle and Late Miocene apes (*Pierolapithecus* and *Hispanopithecus*) predict they lived within promiscuous social systems, but that this pattern began to vary in Pliocene hominins. The predicted social system of *Ardipithecus* was non pair-bonded and differed from that of *Australopithecus*, predicted to be pair-bonded monogamous (Table 7.5; Fig. 7.2). During the

Late Pleistocene, social systems of *Homo sp.* may have been more promiscuous than those evident in most contemporary human populations (Fig. 7.1).

The inferred social system for *Australopithecus afarensis* tentatively supports the claim by Lovejoy (1981) and Reno *et al.* (2003) that this species may have been monogamous (contra Plavcan *et al.* 2005; Scott & Stroik 2006; Gordon *et al.* 2008). However, the inference for *Au. afarensis* is potentially biased by taphonomic factors. There remains a possibility that phalangeal bones from more than one individual are included in the A.L. 333 assemblage (Bush *et al.* 1982), and hence in the hand reconstructed by Alba *et al.* (2003). Similarly, *Hispanopithecus* and *Pierolapithecus* hand remains were found disassociated, although their identity may be more secure based on additional taphonomic and morphological consideration (Almécija *et al.* 2007; 2009). *Ardipithecus ramidus* fossil hand bones were found associated *in situ*, and this analysis of the phalanges conflicts with Lovejoy's recent claim, based on canine and body dimorphism, that *Ar. ramidus* was pair-bonded (Lovejoy 2009). It is noteworthy that the hominin status *Ar. ramidus* also remains in some doubt due to its fragmentary nature (Sarmiento 2010; Wood & Harrison 2011)

2PP:4PP ratio of the early anatomically modern humans (AMH), Qafzeh 9 (~90 Kya) was lower than mean values for most contemporary human populations and in this respect was similar to the mean value for the Neanderthal sample (~73-36 Kya; Fig. 7.1b). The 2PP:4PP of Qafzeh 9 is close to published digit ratios from a polygynous Zulu sample (Møller & Welch 1990; Manning *et al.* 2000a). These similarities suggest that both *Homo neanderthalensis* and early AMH may have lived within a (facultative?) polygynous social structure and may have expressed higher levels of male-male competition than most contemporary human populations. However, variance in Neanderthal digit ratios (Fig. 7.1a) might indicate that these Middle and Late Pleistocene hominins, like contemporary humans, exhibited some flexibility in their social systems and mating behaviour across populations.

Pair-bonding, in a broad-sense, is universal amongst humans (Dunbar 2010a), but it is not known when the transition from a promiscuous mating system to a stable bonded one occurred. The persistence of marked levels of skeletal dimorphism in *Homo* until the Middle Pleistocene (e.g., Arsuaga *et al.* 1997), combined with genetic evidence indicating that male population size (ancestral to people today) was low compared to females' until the spread of agriculture (Dupanloup *et al.* 2003), implies that human-like PB was not common until late in human evolution. The fact that human 2PP:4PP ratios fall between those of PB and non pair-bonded apes (Fig. 7.2) also suggests that human pair-bonding differs from that of other socially monogamous apes (gibbons). Unlike PB monogamous gibbons, humans live within

a multi-male-multi-female social system (Dunbar 2010a); the potential therefore remains for variation in levels of male-male competition. Maintaining this potential may be adaptive during range expansion across ecologically diverse environments. For example, prenatally androgenised individuals with their potential for aggression, risk-taking and status-seeking are likely to have been at the forefront of range expansion, whether colonization took place on virgin territory (i.e., land that were not occupied by other hominin species or out-groups) or took place on land that was already being defended by out-groups.

The fact that correlations between individuals' whole finger lengths relative to the lengths of the proximal phalanges were high and 2PP:4PP and 2D:4D are also very closely related and significantly correlate with hominoid social systems, suggests that 2PP:4PP also reflects a species-level androgenic response. This is supported by the findings that 2PP:4PP ratios show sex differences in the expected direction (Table 7.1) and is in line with a recent meta-analysis in humans showing similarities in sex differences between radiograph derived 2D:4D and 2D:4D based on finger lengths taken from the skin surface (Hönekopp & Watson 2010). Measurement methodologies do impact digit ratios and there is accumulating evidence to show that imaging the hand using scanners or photocopiers distorts the soft-tissue of the digits which lowers digit ratios and increases sex differences (see Hönekopp & Watson 2010; Manning *et al.* 2010). The data were not influenced by distortional factors because bones do not distort when imaged and the 2D:4D ratios used in the regression analysis are based on measurements taken directly from the skin surface (see Chapter 2; Manning *et al.* 2007a).

7.4.1: Summary

Based on associations between digit bone ratios and social systems, the evidence suggests that the social systems of Neanderthals and early AMH may have been similar and characterised by a more competitive social system than evident in most contemporary human populations. This is in line with increasing evidence showing developmental similarities between Neanderthals and early AMH (Ponce de León *et al.* 2008; Guatelli-Steinberg & Reid 2010). A promiscuous social system is indicated for the Miocene apes *Hispanopithecus laietanus* and *Pierolapithecus catalaunicus*. Due to problems with sampling of the data, the results are unable to resolve questions surrounding the social system of *Australopithecus afarensis*, although evidence from *Ardipithecus ramidus*, which is more securely assigned to an individual, suggests that social system of this early putative hominin was promiscuous. These findings suggest a shift to lower PAE across hominin evolution and lend support to Manning's Feminised Ape Hypothesis (Manning 2007a). However, evidence of high body

size dimorphism indicates that 'feminisation' did not approach modern human-like levels until late in hominin evolution (Arsuaga *et al.* 1997; also see Spoor *et al.* 2007; Ruff 2010).

The method used here is distinct from conventional approaches because 2D:4D is not associated with sexual dimorphism of anatomical character (e.g., body size; canine size; see Chapter 4). Indeed, analyses indicate that 2D:4D is actually a better predictor of social systems than body size dimorphism, at least in extant hominoids. Variation in effectiveness of conventional proxies used to predict social behaviour in fossils (e.g., canine and body dimorphism) may, in part, be linked disassociations in ontogeny (and probably in evolution) between development of the body, brain and dentition (see Chapter 4). Although sample sizes for fossils digit are small, this evidence suggests that, as more postcranial fossils become available, digit ratios could augment current methods of estimating social systems for fossil primates and our understanding of the diversity and complexity of human social evolution (Foley & Lee 1989; Foley & Gamble 2009; Dixson 2009).

Chapter 8

General discussion

8.1: Main findings and discussion

8.1.1: 2D:4D, the haplorhine dataset and variation across vertebrates

The haplorhine dataset: Data on mature captive haplorhine primates were provided by over 60 zoos and primate research centres. Measurements were taken by veterinary staff and each institution followed the same standardised procedure (Appendix 2.3). Data were collected on 1286 individuals (463 males and 823 females) from 74 species (Table 2.5; Appendix 2.5). There was no measurement bias across samples from different institutions. The precision of the data was good and within acceptable limits for human studies (ICC=0.74-0.99). This indicates that the 2D:4D data used in the analyses in this thesis reflected real differences and were not influenced by imprecise measurements.

Main findings: To illustrate the phylogenetic method a comparison of male and female 2D:4D ratios across vertebrates was performed by combining data from the haplorhine dataset with that of other non-human animals (published values; Chapter 2; Fig 2.17). In mammals 2D:4D was lower in males than females. In closely related birds and scaly lizards (see Shedlock & Edwards 2009) sexual dimorphism was in the opposite direction; males had higher 2D:4D than females.

Informing current understandings: This is the first study to assess the direction of sexual dimorphism in 2D:4D within an evolutionary framework. Previous analyses of inter-specific differences in 2D:4D have only been descriptive and failed to show consistent sexually dimorphic patterns across taxa (see Lombardo & Thorpe 2008; Lombardo *et al.* 2008). The inclusion of the haplorhine dataset and the use of the comparative method has enabled a more comprehensive and rigorous study to be carried out. The results support Manning's proposal that sex differences in 2D:4D should be generalised across taxa with similar limb development as a consequence of shared *HOX* genes (Manning 2002a; Zákány *et al.* 1997). However, this generalisation is limited. Mammals exhibited differences in the direction of sexual dimorphism in 2D:4D to scaly lizards and birds; Fig. 2.17). These two groups also differ in their mechanisms of sexual determination. In mammals males are the heterogametic sex, while in most reptiles (birds and lizards; see Shedlock & Edwards 2009) females are the heterogametic sex (Adkins-Regan *et al.* 1995; Lance 1997; Lombardo *et al.* 2008). As the

development of the gonads (ovary and testes) is influenced by genes on the sex chromosomes, differences in sexual determination could differently influence foetal sex hormone production, which could impact on *HOX* gene expression (Zákány *et al.* 1997; Daftary & Taylor 2006). Different mechanisms of sexual determination may underpin sexual differences in digit ratios (Lombardo *et al.* 2008). These findings support Chang's Phylogenetic Constraints Model (Chang *et al.* 2006; Chang 2008) which hypothesises that 2D:4D ratios in more closely related taxonomic groups should be more similar (but see Lombardo & Thorpe 2008).

Strengths and limitations: Comparative analysis between vertebrates has provided the first robust evidence of broad patterns of sexual dimorphism in 2D:4D. The evidence also indicates that comparative analyses should be confined to species within the Class Mammalia or within the Classes Reptilia and Aves (see Shedlock & Edwards 2009) in order to avoid potential confounds imposed by different mechanisms of sex determination.

The haplorhine dataset represents the first comprehensive collection of 2D:4D data across a taxonomic group. The large sample size was achieved by adhering to a carefully constructed data collection method. The dataset has several limitations. Firstly, having to obtain data from captive species means that the sample is biased towards species preferred by zoos and primate research centres (Melfi 2005; see Fig. 2.14). In non-pair-bonded (NPB) promiscuous species sample size is skewed towards females (Table 2.5). Sample sizes were lower in pair-bonded (PB) taxa and species rare in captivity. Secondly, captivity is known to impact primate development (e.g., Leigh 1994; Smith & Jungers 1997; Hare 2004) and these factors may influence PAE (and therefore 2D:4D). Thirdly, results indicate that while the precision of digit measurements were within the acceptable limits they may have been influenced by hand size; ICC values were lower in Old World monkeys (OWM) than in apes and New World monkeys (NWM; Table 2.7).

Extending the research: Increasing the diversity of species and sample sizes within the dataset would be advantageous to future studies. It would be of interest to examine how body size may influence 2D:4D measurements. For example, were very big hands measured more precisely than very small hands? Although no significant differences were detected in 2D:4D between a captive and a free-ranging sample of female rhesus macaques (see Chapter 5) it would be advantageous to carry out similar comparisons in other species which may be more sensitive to differences in environmental conditions.

8.1.2: 2D:4D and behavioural indicators of sexual selection in haplorhines

Summary and main findings: Chapter 3 investigated 2D:4D within and between super families to search for large-scale patterns between 2D:4D and sexually selected behaviours. 2D:4D was lower (inferred higher PAE) in NPB species and higher (inferred lower PAE) in PB species. Both male and female 2D:4D decreased (PAE increased) with increasing levels of intra-sexual competition. Relationships were weaker for NWM and stronger for catarrhines. OWM had low and relatively invariant 2D:4D (inferred high PAE) coupled with high levels of intra-sexual competition. This contrasts with higher and more variable ratios in apes and NWM. Human ratios were intermediate between PB and NPB great apes. Within great apes there was a clear increase in 2D:4D across evolution with humans having the highest 2D:4D (lowest inferred PAE).

Informing current understandings: Close correlation between males and female 2D:4D in haplorhine primates mirror sexually dimorphic patterns in 2D:4D across human populations (Manning *et al.* 2000a; Manning *et al.* 2004a; Manning *et al.* 2007a). This suggests that the genes that encode for PAE are inherited by both sexes via a shared gene-pool (Fig. 3.1; Fig. 3.4; see Manning *et al.* 2000a). This causes male and female 2D:4D to shift together according to the strength of sexual selection (see Lande 1980; see Manning *et al.* 2000a). For example a social system biased towards a male genetic profile selects for high PAE in males and this imposes high PAE on females via a correlated response (Lande 1980; Rice 2000; Rice & Chippendale 2001). This can be clearly seen in correlations between 2D:4D and intra-sexual competition (Fig. 3.4). However, selection for high PAE can be disadvantageous to females in terms of reproductive physiology (Wallen 2005; Manning & Fink 2011). Similarly, selection for low PAE in PB species can be disadvantageous to male fitness via negative effects on reproductive hormonal profiles and cardiovascular health (Manning *et al.* 1998; Manning 2002a). The mechanism that selects for traits that may be advantageous in one sex but disadvantageous to the other is termed ‘inter-genomic conflict of sexually antagonistic genes’ (Rice & Chippendale 2001). Manning *et al.* (2000a) used the explanatory framework to describe the sex-linked expression of 2D:4D across human populations. The framework is adopted here to interpret patterns of co-variation between 2D:4D and sexual selection across haplorhines.

8.1.2.1: 2D:4D and sexually antagonistic effects

When males are promiscuous their reproductive success over a lifetime is much higher than that of their mates. Selection for novel traits that increase fitness in promiscuous males

introduces sexual conflict because development of these traits often necessitates high androgens that impose costs on female reproductive fitness. These evolutionary effects are powerful and genes linked to male reproduction evolve quickly (Wykoff *et al.* 2000; Geary 2002; Dorus *et al.* 2004). These effects are much weaker in PB species because selection for a trait that reduces fitness in one sex also reduces it in the other (Holland & Rice 1999). Selection for a masculinised (high PAE) or a feminised (inferred low PAE) social systems move species' phenotypes in different directions along the masculinisation-feminisation continuum (Fig. 8.1). Maintenance of optimal and sub-optimal phenotypes (genetic variation) retains the potential for species to adapt to changes in environmental circumstances (Fig. 8.1; Eshel & Matessi 1998; Waddington 1942; also see Rice & Chippendale 2001). However, these evolutionary changes will be constrained by phylogenetic effects that may act to conserve core phenotypic traits (see Foley & Lee 1989; Di Fiore & Rendall 1994).

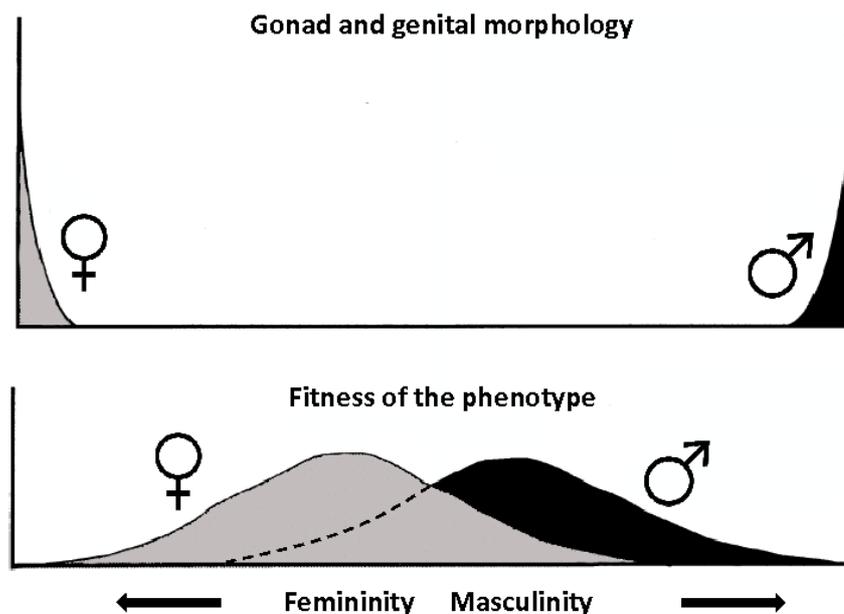


Figure 8.1: Inter-genomic conflict of sexually antagonistic genes. Although gonadal and genital morphology are discrete characters, variation in levels of fitness across the entire phenotype of a species indicate a continuous transition between males and females as a consequence of inter-sexual ontogenetic conflict. Many traits within each sex deviate from the optimum and may therefore not be the most adaptive for each sex.
After Rice & Chippendale 2001.

The fact that 2D:4D was found to be significantly lower in males (inferred higher PAE) than females in promiscuous NPB species, but not in PB species, might suggest that 2D:4D ratios are signalling the conflict between male advantage (increased male reproductive variance)

and female disadvantage due to high PAE (see Manning *et al.* 2000a). More divergent 2D:4D between males and females might signal the action of modifier genes that serve to stabilise selection and act to constrain the negative effects on PAE on female fitness. Female 2D:4D will therefore be constrained in order to maintain fitness (higher 2D:4D, inferred lower PAE), while in males 2D:4D is freer to decrease. In PB species reproductive variance is about the same in males and females and significant sex differences in 2D:4D were not detected in these taxa; as might be predicted by the explanatory framework (Holland & Rice 1999; Manning *et al.* 2000a).

8.1.2.2: 2D:4D and differences across lineages

Patterns between 2D:4D and social system across haplorhine species mirror relationships found in humans (indexed by marriage systems; Manning 2007a; also see Gray 2003; Alvergne *et al.* 2009). Differences in PAE within social systems may underpin key behavioural traits linked to variation in the strength of sexual selection. Low PAE in PB species channels behaviour towards feminisation which may promote low aggression, tolerance and the maintenance of enduring bonds between reproductive partners (Dunbar & Shultz 2010). These effects may also potentiate paternal care in some species (e.g., callitrichids and humans; Zeigler & Snowdon 2000; Gray *et al.* 2002; Gray 2003). In contrast, high PAE channels behaviour towards masculinisation which is important in supporting sexual and competitive behaviours in NPB promiscuous primates (Phoenix *et al.* 1959; Wallen 1996; 2005; Klein 2000).

Low and invariant 2D:4D ratios (inferred higher PAE) in OWM are consistent with their competitive, agonistic and impulsive social behaviours (Fig. 3.1; Plavcan and van Schaik 1997; Sterck *et al.* 1997). Social organisation is highly conserved across this super family (Di Fiore & Rendall 1994). In OWM males migrate into new groups and compete with unrelated males. Females are philopatric (remain within their natal group) and often form into kin-based dominance hierarchies in order to compete with other females kin groups. Female philopatry is a derived feature of OWM and may have evolved alongside adaptations linked to increases in competitive behaviour and higher PAE (Strier 1990; Di Fiore & Rendall 1994). Behaviours linked to high androgens are accentuated in the Cercopitheciinae and may have intensified after their split with the Colobinae after ~14 Mya (Raaum *et al.* 2005; also see Thierry *et al.* 2000). It is of note that in their phylogenetic analysis of primate social organization Di Fiore and Rendall (1994) found that the two prosimian genera that grouped most closely to OWM also show evidence of PAE on behaviour (*Lemur*: Ostner *et*

al. 2003; Drea 2007; *Propithecus*: Littlefield 2010). These species also have very low species mean 2D:4D; 0.802 and 0.674 respectively¹⁹ (see Oxnard 2000).

Great ape 2D:4D was significantly higher (inferred lower PAE) than OWM digit ratios. Great ape males are generally philopatric and form tolerant bonds (see Ghiglieri 1987; Mitani 2009), but social organization does vary across the clade (*Gorilla* sp.: Robbins *et al.* 2004; *Pongo* sp.: van Schaik & van Hooff 1996). Male chimpanzees (*Pan troglodytes*) form coalitions to hunt colobus monkeys and aggressively defend territorial boundaries (Mitani 2009) and exhibit dominance responses when faced with competition over food (Wobber *et al.* 2010a). Females migrate into the group and form bonds with other females (Newton-Fisher 2006) and males dominate females (Parish & de Waal 2000). In bonobo (*Pan paniscus*) societies females dominate males but also form close bonds with them. Females also form close bonds with other females (White 1992). Bonobos are highly gregarious, show higher levels tolerance and share food more readily than chimpanzees (Parish & de Waal 2000; Hare *et al.* 2007; Wobber *et al.* 2010a). McIntyre *et al.* (2009) found that 2D:4D in wild-born bonobos exhibited higher ratios than wild-born chimpanzees²⁰ and hypothesised that this might reflect their different social styles (Parish & de Waal 2000).

An unusual feature of great ape societies is their expression of fission-fusion (FF) dynamics (see Barrett *et al.* 2003; Aureli *et al.* 2008). FF involves the group reforming after periods of foraging in smaller parties as a mechanism to overcome feeding pressures induced by patchy food resources. In species that adopt this uncommon strategy tend to be more encephalised (e.g., elephants, dolphins; see Aureli *et al.* 2008). High FF poses a threat to social cohesion because friendships can potentially alter when individuals are separated from each other. This is not a problem for most OWM who tend to spend their day in full view of each other and can update relationships constantly²¹. Chimpanzees and bonobos have adapted to the 'out of sight, out of mind' cognitive hurdle of by evolving skills that allow them to hold relationships with others in their minds (proto-imagination; see Barrett *et al.* 2003; Dunbar & Shultz 2010). Tests in chimpanzees suggest a capacity to understand the feelings and intentions of others (proto-empathy; proto-theory of mind; Call & Tomasello 1998; de Waal & Aureli 1999; Fraser *et al.* 2008). Although these findings are contentious and difficult to test objectively (see Provinelli & Vonk 2006). Chimpanzees also appear to be able to inhibit

¹⁹ Prosimian data is a supplementary sample to the haplorhine dataset.

²⁰ 2D:4D ratios for these two species are similar to those in the captive sample in the haplorhine dataset (Table 3.1).

²¹ *Papio hamadryas* exhibit FF but re-congregate each night (Aureli *et al.* 2008).

responses in social bonding situations. This allows them time to reappraise social relationships when individuals reunite after a period of fission (Amici *et al.* 2008). Being able to inhibit responses appears much more difficult for OWM (Amici *et al.* 2008). In humans, impulsivity and reduced risk aversion is associated with a high androgen response (Aluja *et al.* 2011), high testosterone (see Roney *et al.* 2009) and low 2D:4D (Martel *et al.* 2008; Stenstrom *et al.* 2011).

In cross-species analyses correlations between 2D:4D and social systems were weaker for NWM (Table 3.2). This may be associated with differences in mechanisms that potentiate reproductive behaviour in NWM that may potentially obscure PAE on adult behaviour (Curley & Keverne 2005; also see Gromoll *et al.* 2003). There is, however, evidence from 2D:4D that suggests a convergence in social organisation of New World spider monkeys *Ateles sp.* and Old World chimpanzees. The two species share many similarities in their social structure including high FF dynamics, male philopatry, coalition formation (see Aureli *et al.* 2008, p 630) and cognitive abilities (Amici *et al.* 2008; 2009). The 2D:4D ratios of spider monkeys and chimpanzees are virtually indistinguishable (see Table 3.1) even though their global hand morphology is quite different (e.g., *A. geoffroyi* has a rudimentary thumb; Tague 2002). This convergence in 2D:4D, social structure and cognitive skill tentatively suggests that in some NWM PAE may induce similar effects on behaviour and cognition.

2D:4D and social systems were more variable in apes and NWM than in OWM (Fig. 3.1). This suggests that both apes and NWM retain the more primitive pattern of social flexibility even their social behaviours may be largely underpinned by different mechanisms (Curley & Keverne 2005). In catarrhines evolution, the divergence of the OWM and the apes from a common ancestor around 30 Mya (Steiper & Young 2006) led these super families to evolve along separate pathways characterised by differences in sexual selection and social organisation; OWM evolved a suite adaptations that facilitated highly competitive strategies that necessitated high PAE, while apes retained diversity in social systems which required more variable PAE (Fig. 3.1; see Di Fiore & Rendall 1994; Begun 2004b; Alba 2010). 2D:4D appears to index this variability. Within great apes (NPB) a gradual increase in 2D:4D is apparent from the *Pongo sp.* through to humans implying a decrease in PAE across the clade over evolution (assuming that digit ratios have not changed over time within great apes species; Fig. 3.3). The 2D:4D evidence is consistent with evidence showing a down-regulation of the androgen receptor gene (ARG) in great apes with humans having the least sensitive ARG (Choong *et al.* 1998; Andrés *et al.* 2004; Hong *et al.* 2006; Manning 2007b). This suggests that (proto-) 'feminisation' may stem back to the Middle Miocene (see Hare 2004).

8.1.2.3: 2D:4D and hominin social evolution

The Feminised Ape Hypothesis (Manning 2007a) bears some similarities to the Emotional Reactivity Hypothesis (Hare & Tomasello 2005a). Manning (2007a) hypothesised that over hominin evolution there had been a decrease in male-male competition co-incident with a reduction in PAE and an increase in intelligence. He based his evidence upon observations of differences in 2D:4D and the ARG sensitivity within humans and between humans and apes which signalled a feminisation rough hominin evolution (see Introduction; Section 1.1.2.4). Hare & Tomasello's (2005a) Emotional Reactivity Hypothesis proposed that selection for reduced competition, lower aggressive and increased cooperation led to a reduction in endocrine response. As hominins speciated into novel 'adaptive spaces' the bio-behavioural changes that had already taken place facilitated to elaboration of communication skills (e.g., proto-empathy and proto-theory of mind). This model is based upon observations of selective breeding in canines for temperaments low in aggression and high in attentiveness. Over many generations selection for these traits in dogs has led to the emergence of other characters such as delayed social development, more gracile and paedomorphic anatomical features and changes in hormonal profiles (Hare & Tomasello 2005a; Hare 2007; Wobber *et al.* 2010b; Konno *et al.* 2011). This suite of characteristics is expressed in socially tolerant bonobos (*Pan paniscus*) when compared with more competitive chimpanzees (Hare 2007; Wobber *et al.* 2010a; 2010b). The observations are of interest from a palaeo-anthropological perspective because this suite of characteristics is also apparent in modern humans (Lieberman 1998; Bogin 2003; Ruff 2005).

Both the Feminised Ape Hypothesis and the Emotional Reactivity Hypothesis (Hare & Tomasello 2005a) propose that through hominin evolution individuals that were more cooperative or 'tame' were favoured over those that were more aggressive and highly competitive. Although it is not clear how this might have come about, but it may be associated with need to update and reaffirm social relationships as group sizes increase as seems to be the case with bonobos (Mulavwa *et al.* 2010; also see Aureli *et al.* 2008; Dunbar 2010a). Comparative work in chimpanzees and bonobos show that many social interactions in chimpanzees hinge on competitiveness (Melis *et al.* 2006) which is supported by a more responsive androgenic profile (Wobber *et al.* 2010a). Androgenic responses appear to be down regulated in bonobos (Wobber *et al.* 2010a) which might explain their more developed neural adaptations linked to empathy responses (Rilling *et al.* in press). In humans the same pathways are known to be impaired by testosterone (van Wingen *et al.* 2010). This evidence suggests that reduced testosterone responses may have evolved in bonobos alongside

decreases in intra-sexual competition, increased cooperation and proto-empathy. These changes are consistent with changes seen in the domestication of dogs (see Hare & Tomasello 2005a).

Strengths and limitations: This is the first study investigating relationships between 2D:4D and sexually selected social behaviour across a taxonomic group: the *Haplorhini*. Cross-species approaches to studying 2D:4D circumvents many of the problems associated with intra-specific variability in target traits that might serve to obscure evolutionary patterns. This method permits the testing of important theoretical concepts of 2D:4D and supports the proposal that 2D:4D should be viewed within the framework of sexual selection theory (Fink *et al.* 2006c) and is consistent with the hypothesis that 2D:4D generalises across taxa because cross-species patterns mirror findings from human studies (Manning 2002a; 2007a; Manning *et al.* 2000a). Large-scale patterns in 2D:4D within catarrhines draw attention to evidence that implicate PAE in programming core behavioural profiles within lineages (OWM and apes). Investigating PAE within larger research paradigms, such as the Social Brain Hypothesis (SBH; Byrne & Whiten 1988; Dunbar 1998), may improve our understanding of the proximal mechanisms that underpin primate social relationships.

One of the limitations of this study is that 2D:4D proved to be no better than other anatomical markers of sexual selection (e.g., body and canine size dimorphism) when it was correlated against 3 categories of social system (Fig. 3.2; Fig. 4.1; Fig. 4.3); no significant differences could be detected between uni-male and multi-male-multi-female groups.

Extending the research: Across analyses potential influences of hand function on 2D:4D were controlled for by entering substrate categories into the models (e.g., arboreal, terrestrial; Plavcan & van Schaik 1992). Although differences on hand morphology on 2D:4D did not appear to weaken the results of this study, hand structure may impact digit ratios in some haplorhine families (Ankel-Simons 2000; Tague 2002). More detailed investigations of possible interactions between 2D:4D and hand function would help refine the haplorhine database (Stern & Susman 1983; Richmond 2007). Functional studies would be enhanced if they were could be combined with developmental studies of the effects of *HOX* gene proteins on digit morphology and the reproductive system, which is poorly understood in primates (Tague 2002; Chiu & Hamrick 2002).

8.1.3: 2D:4D and anatomical indicators of sexual selection in haplorhines

Summary and main findings: The previous chapter demonstrated strong associations between 2D:4D and social systems and intra-sexual competition across haplorhines. In Chapter 4 this evidence was extended to investigating relationships between 2D:4D and anatomical signals of sexual selection in haplorhines. Relationships between 2D:4D, body and canine size and body and canine dimorphism were not significant after controlling for phylogenetic relatedness, but dimorphism measures did exhibit weak trends in the predicted direction; low 2D:4D was associated with higher dimorphism. There were no associations with brain size measures. In conclusion 2D:4D was not associated with anatomical markers of sexual selection in haplorhine primates. This is in stark contrast to associations between 2D:4D and behavioural markers of sexual selection.

Informing current understandings: These findings are consistent with our current understanding of 2D:4D and its relationship with anatomical indicators of sexual selection. 2D:4D is formed early in development and changes minimally through life (Manning 2002a). In contrast, anatomical markers of sexual selection increase markedly over growth (Leigh 1995; Leigh & Shea 1995; also see Trivers *et al.* 2006). Sexual dimorphism is manifested by changes in the rate and timing of growth between males and females within species (Leigh 1992). Differing evolutionary pressures on growth trajectories lead to variability in these patterns across taxa by altering the timing and rate of maturation differently in males and females (Leigh 1995; Schwartz & Dean 2001; Bernstein 2007; 2008). Growth trajectories are additionally altered by extrinsic environmental factors at a local level (Turner *et al.* 1997; Gray & Wolfe 1980). Consequently similarities in sexual dimorphism (end-points) between species can be reached via different growth patterns. Condensing this degree of variability into a single measurement fails to capture developmental differences between the sexes and weakens the power of the sexual dimorphism ratio as a proxy for sexual selection (see Plavcan 2001). The fact that neonatal body size dimorphism correlates with adult body size dimorphism (Smith & Leigh 1997) but not with 2D:4D suggests that intra-uterine growth factors, other than PAE, have already influenced body size before birth (e.g., insulin-like growth factor; Bernstein *et al.* 1997). A recent study in humans found no relationship between PAE and neonatal weight (Miles *et al.* 2010). However, a lack of significant correlations with 2D:4D does not negate the fact that PAE may have played a part in programming growth trajectories early in prenatal development. Indeed trends in the expected direction suggest they do (Fig. 4.5; Fig 4.6).

There have been no studies of 2D:4D and dental morphology and no studies specifically investigating 2D:4D and body size dimorphism in humans. However, the null findings in this study are in line with relationships between 2D:4D and stature in humans which are weak (see Lippa 2003; Rahman *et al.* 2005; Barut *et al.* 2008; Manning 2010) or non-significant (Manning 2002a; also see Rahman *et al.* 2005). There is evidence to show that sexual selection impacts stature in humans; taller men and shorter women have higher reproductive success (Pawlowski *et al.*, 2000; Nettle, 2002) and this appears to be expressed at the population level (see Rahman *et al.* 2005); sexual dimorphism in stature is higher in polygynous populations than monogamous societies (see Kanazawa & Novak 2005 for a review; but see Gray & Wolfe 1980). It remains unclear, however, if these patterns have arisen via selection on increased male size or decreased female size or if cultural and environmental affects act to reorganise growth mechanisms generation-by-generation (see Kanazawa & Novak 2005). So while associations between 2D:4D and marriage systems suggest that 2D:4D should co-vary with human sexual dimorphism in stature, until the studies are done we might speculate that potential variation in male and female growth trajectories between human populations may also significantly weaken associations with PAE.

Evidence from developmental studies provides additional information on why relationships with 2D:4D are weak. Some anatomical proxies of sexual selection are highly variable while others more constrained (Waddington 1942). For example body size is much freer to alter whereas canine and brain size are much more tightly controlled by genetic factors (Smith 1989; Martin 1994; Harlia-Kaera *et al.* 2001; Schwartz & Dean 2001; Herculano-Houzel 2009). Disassociation of these traits pose problems for analysing brain size changes relative to body size changes across species because these calculations end up incorporating factors that impact on body size but that do not necessarily impact on brain size (e.g., ecological, energetic and functional constraints; Herculano-Houzel 2009; Shultz & Dunbar 2010). 2D:4D indexes PAE on programming brain responses that potentiate adult sexually selected behaviours across species and brain size is strongly related to sexually selected behaviours (Sawaguchi 1996; Schillaci 2006; Shultz & Dunbar 2007). Why did 2D:4D fail to correlate with brain size or brain size dimorphism in this study? Sexual dimorphism in the brain is manifested in the brain architecture, not relative size (e.g., Lindenfors *et al.* 2007; Yan *et al.* 2011). To date there has only been one study of 2D:4D and brain architecture. Low 2D:4D (inferred high PAE) in women (men were not tested) was associated with masculinisation of parts of the hippocampus (smaller posterior sub-structure on the left side; Kallai *et al.* 2005). Differences in species-level androgen responses have been associated with differences in neural circuitry linked to empathy in bonobos and chimpanzees (Rilling *et al.* in press)

Bonobos have more gray matter than chimpanzees (*Pan troglodytes*) in connections between the ventromedial prefrontal cortex and the amygdala (Rilling *et al.* in press). These pathways are associated with empathy response in humans and are impaired by testosterone (van Wingen *et al.* 2010). It is proposed that lower testosterone in more socially tolerant bonobos in comparison to more competitive chimpanzees (McIntyre *et al.* 2009; Wobber *et al.* 2010a) may be associated with enhanced (proto-²²) empathy in bonobos (Rilling *et al.* in press; see Herrmann *et al.* 2010). 2D:4D also differs between these two closely related species with different social behaviours (McIntyre *et al.* 2009; Table 3.3). Within humans, 2D:4D appear to reflect PAE on brain pathways linked to social bonding (e.g., empathy skills, OT and AVP response; Knickmeyer *et al.* 2006a; Cater 2007; van Honk *et al.* 2011). Studies of 2D:4D and brain architecture may prove informative about the effects of PAE on the organisation of sexually selected behaviours and social bonding in non-human primates.

Strengths and limitations: This study indicates that 2D:4D may be a better indicator of PAE on sexually selected behaviours (i.e., effects on brain programming) than on anatomical proxies of sexual selection (i.e., effects on programming growth). However these results must be interpreted with caution because of the potential confounds of condensing growth variation information in one species-level value. Of course using mean values is a practical and valid method of analysing species-level data, but it may be less appropriate for characters that can be the same across species but may have been reached via different developmental patterns (Leigh 1992; 1995; Schwartz & Dean 2001). It may be possible to refine measures of dimorphism by factoring in information on growth trajectories of males and females within species, but this is unlikely to improve correlations with adult body size dimorphism given the failure to find correlations between 2D:4D and neonatal body dimorphism. Finally, this study has also highlighted gaps in human 2D:4D research. As relationships between 2D:4D and sexual dimorphism have not been studied in humans it is difficult to know if the patterns will be similar to the findings shown here.

8.1.4: 2D:4D, dominance rank and heritability in female rhesus macaques

Summary and main findings: PAE appears to be implicated in supporting dominance rank hierarchies in spotted hyenas (*Crocuta crocuta*) that have similar social systems to macaques (Dloniak *et al.* 2006). Chapter 5 sampled 2D:4D in a group of free-living rhesus macaques

²² The prefix is used because the extent great apes are able to express cognition abilities such as theory of mind and empathy is still unclear (Tomasello *et al.* 2003; Provinelli & Vonk 2003).

(*Macaca mulatta*) to see if PAE co-varied with socially ‘inherited’²³ dominance rank. Low 2D:4D (inferred high PAE) was associated with higher-ranking females, while higher 2D:4D (inferred lower PAE) was associated with lower ranking females. A heritability analysis based on comparisons of 2D:4D in mother-infant dyads was also carried out. Results suggest heritability of 2D:4D is high in rhesus macaques and is within the range of estimates from humans and more distantly related taxa.

Informing current understandings: This finding from this study make an important contribution to primate research in several ways. It significantly advances our understanding of how social organisation is maintained across generations in OWM. There is a wealth of evidence to show that young cercopithecine females learn their rank from the actions and reactions of others around them (Datta 1988; Chapais 1992; 2004). It is this process of social learning that underpins the transfer of ‘inheritance’ of dominance rank from mothers to daughters (Walters & Seyfarth 1987). Evidence from 2D:4D suggests that PAE might also be implicated in maintaining the transfer of dominance rank from one generation to the next because higher ranking females had lower 2D:4D (inferred higher PAE). Offspring exposed to higher PAE via maternal androgenising effects are more aggressive and thus stand more chance at retaining their higher ranking position (see Dloniak *et al.* 2006). These behavioural predispositions are then reinforced, or discouraged, by social learning depending on the ‘inherited’ rank of the young female and the social context she grows up within. Thus there is a synergistic relationship between PAE and social learning processes; both serve to perpetuate rank across generations (see Wallen 1996; 2005). These effects are believed to be highly adaptive in species in which competition for resources is high (Dloniak *et al.* 2006; Kaiser & Sachser 2009).

The power of this study is increased because findings mirror those found in spotted hyenas using a different methodological approach (Dloniak *et al.* 2006). The similarity in results between these two studies is indicative of convergent evolution between these two distantly related mammals (Dloniak *et al.* 2006). This study also adds to the accumulating evidence that 2D:4D indexes the masculinising effects of prenatal androgens on females in both non-human primates (shown in this thesis) and humans (see Voracek & Schicker 2010). Similar to female rhesus monkeys, human females with low 2D:4D tend to be more dominant and exhibit more aggression (Wilson 1983; Manning & Fink 2008; see Voracek & Schicker 2010 for a review).

²³‘Inheritance’ in this context means passing on rank from one generation to another by learning ones place in the hierarchy (Datta 1988; Chapais 1992).

This study is the first to calculate heritability in 2D:4D in a non-human primate. The findings suggest that a proportion of the heritability effects in 2D:4D is a product of maternal effects on offspring. In the mother-infant sample heritability was shown to be higher in the right hand than in the left. Over all heritability the estimates accord with a recent study of familial resemblance of 2D:4D in humans (e.g., Voracek & Dressler 2009) and is in line with evidence showing higher PAE in the right hand in humans (Hönekopp & Watson 2010). This study also makes a significant contribution to the accumulating evidence that 2D:4D shows high heritability across species in distantly related taxonomic groups (see Forstmeier 2005; Forstmeier *et al.* 2008).

Strengths and limitations: This is the first study to investigate relationships between 2D:4D and dominance rank in nonhuman primates. This research was carried out on a cohort of free-living rhesus macaques descended from a wild colony established on Cayo Santiago Island in 1938 (Rawlings and Kessler 1986). As such the dominance ranks upon which this study is based have formed naturally over many generations. For a within-species study of behaviour it is important to collect data on non-captive individuals because both behaviour and development are impacted by the environment they develop within (Smith & Jungers 1997; Hare 2007; also see Wallen 2005). We currently do not know how the captive setting impacts influence 2D:4D.

Extending the research: The heritability study should be replicated using a much larger cohort. This should include multigenerational pedigree relationships (see Kruuk 2004), measures of assortative mating (see Voracek *et al.* 2007a), evidence of founder effects (see Chepko-Sade and Sade 1979) as well as other potentially relevant developmental factors. A better understanding of maternal and genetic effects on 2D:4D could be gained by including other familial dyads such as sibling-sibling, half-sibling or sire-offspring data. The ability to replicate this study, however, is highly constrained by the nature of the data; access to the digits of hundreds of sedated mother-infant pairs from free-living or wild populations will be challenging.

8.1.5: 2D:4D, testosterone and the androgen receptor gene in haplorhines

Summary and main findings: Chapter 6 focussed on the proximate androgenic mechanisms that potentiate sexually selected behaviours. Intra-specific levels of circulating testosterone were shown to be higher in males than females. Haplorhine species with lower 2D:4D tended to have lower body weights and small bodied NWM had the highest testosterone levels. OWM had lower 2D:4D (inferred higher PAE), higher circulating testosterone and a more

sensitive androgen ARG than great apes. Down-regulation of the ARG was associated with increasing group size and brain size.

Informing current understandings: Patterns of testosterone levels across haplorhines fit with existing evidence of high hormonal levels in smaller bodied NWM (Coe *et al.* 1992). Circulating testosterone levels significantly differed between OWM and apes. Higher levels in OWM support their highly competitive social systems underpinned by female philopatry and intra-sexual competition. Lower levels in apes are consistent with male philopatry and their comparatively more tolerant social behaviour. Within apes the lowest testosterone levels were in (NPB) bonobos and (PB) hylobatids; species that have high levels of tolerance between individuals (Brockelman *et al.* 1998; Parish & de Waal 2000).

The magnitude of circulating androgen responses on DNA within the tissues is mediated by the ARG. To recap: mean tri-nucleotide repeat sequences (CAG_n) in the ARG that are short, signal higher sensitivity to androgens (Roney *et al.* 2009). Short CAG_n in humans have been linked to traits such as aggression (Rajender *et al.* 2008), impulsivity (Aluja *et al.* 2011), low relationship quality (Comings *et al.* 2002) and high fertility (von Eckardstein *et al.* 2001). Longer CAG_n in humans are associated with lower responsiveness to androgens (Roney *et al.* 2009), lower fertility (von Eckardstein *et al.* 2001), smaller genitalia (Lim *et al.* 2000), but a hypothesised increase in general intelligence (Manning 2007b). In great apes there has been an increase in CAG_n over evolution with humans being the least sensitive to androgens (e.g., Choong *et al.* 1998; Hong *et al.* 2006; also see Manning *et al.* 2003a). The recent discovery of a loss-of-function deletion within the ARG has been linked to the loss of penile spines in humans (McLean *et al.* 2011) and is consistent with a reduction in male-male competition through hominin evolution (Dixson 1998; 2009).

A down-regulated androgen effect across great apes contrast with the low 2D:4D ratios and high androgen profiles of OWM (Fig. 8.2). It could be that selective changes to PAE may be the simplest mechanisms for taxa to ramp-up or down-regulate their social behaviours (John Manning pers. comm.). Small shifts in CAG_n along the polymorphic ARG might be sufficient to affect significant changes in a species social behaviour (Fig 6.1a). Variation in intra-specific PAE and circulating androgens may serve to fine-tune these changes at a local level (see Gray 2003; Alvergne *et al.* 2009).

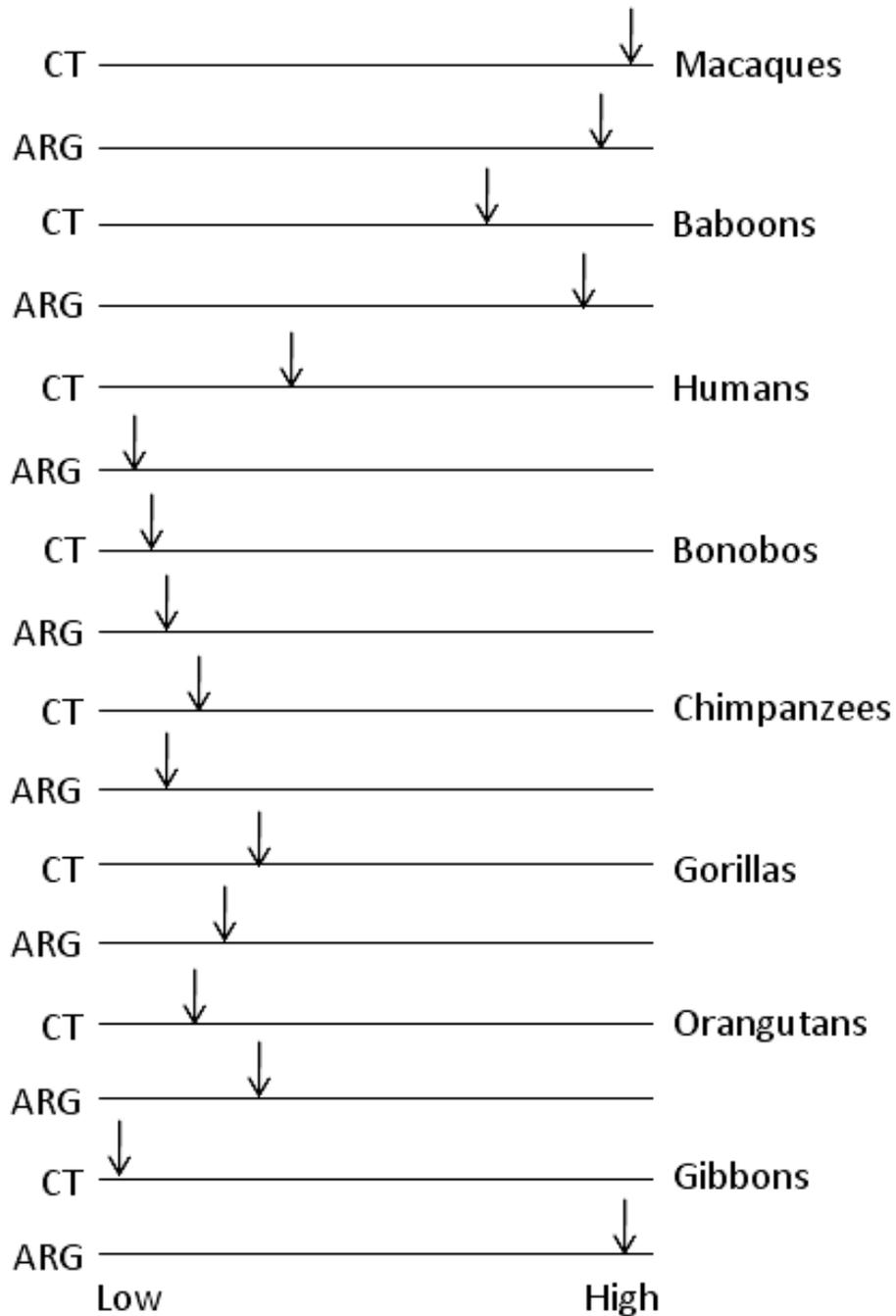


Figure 8.2: Androgen profiles in OWM and apes. Stylised image based upon the results from Chapter 7. Arrows represent species values of target traits relative to other species. CT; circulating male testosterone; androgen gene sensitivity signalled by CAGn. Lower CAGn = more sensitive to androgens. *Hylobates lar* and *Symphalangus syndactylus* are placed in a single profile (Gibbons) as their values did not significantly differ.

Polymorphisms allow species to exhibit more variability in phenotypic expression and potentially increase behavioural flexibility (Foley 2010). Diversity in social behaviour has also been linked to polymorphisms in other receptor genes linked to social bonding (e.g.,

AVP and OT receptors; Hammock & Young 2005; Pritchard *et al.* 2007; Walum *et al.* 2008; Israel *et al.* 2008; 2009; dopamine receptor: Reuter *et al.* 2011; also see Chakrabarti *et al.* 2009).

As social complexity increases in accordance with brain size and group size in primates (see Fig. 8.3; Dunbar 1998; Dunbar & Shultz 2007b) variation in behavioural flexibility and cognitive abilities may be critical for maintaining social relationships within large hierarchical groups. The pecking orders of dominance ranks mean that many individuals within the population will be prevented from optimising their energy and reproduction requirements (Rice & Chippindale 2001). The ability to employ different social strategies to acquire resources provides the potential for many more individuals (e.g., middle and lower ranking) to achieve the same endpoint - reproductive fitness - without jeopardising social cohesion. For instance, Pawlowski *et al.* (1998) showed that in large brained promiscuous species males are able to increase their reproductive potential by forming coalitions and employing social tactics to undermine the power of higher ranking males. Links to behavioural flexibility are supported by near positive relationship between CAGn and brain size ($p=0.056$ after controlling for body size; $p<0.01$ before body size controls); brain size increased in line with increasing CAGn (decreasing sensitivity to androgens) across the sample (Fig. 7.7).

Patterns between the structure of the ARG and group size within catarrhines show that expansion of the gene (polymorphism) within a species may be characteristic of bigger primate groups ($p=0.066$; Fig. 7.1a; Fig. 7.6). If polymorphism of the ARG and reductions in sensitivity to androgens facilitate group expansion, then these changes may partner the evolution of cognitive adaptations that allow humans to maintain close social relationships within larger, dispersed social groups (Aureli *et al.* 2008; Coward 2010). In addition, the close correlations between group size and brain size (Barrett *et al.* 2003; Dunbar 1998; 2009) and brain size and CAGn found in this study strongly suggest that changes in androgen profiles and sociality over hominin evolution are unlikely to be independent of brain size evolution (Fig. 8.3). Furthermore, it could be speculated that modulation of prenatal androgens via the polymorphic ARG and the known ramification on neural reward pathways (see van Honk *et al.* 2011) could potentially underpin key societal changes in *Homo* sp. (e.g., altruism; Israel *et al.* 2008; Reuter *et al.* 2011).

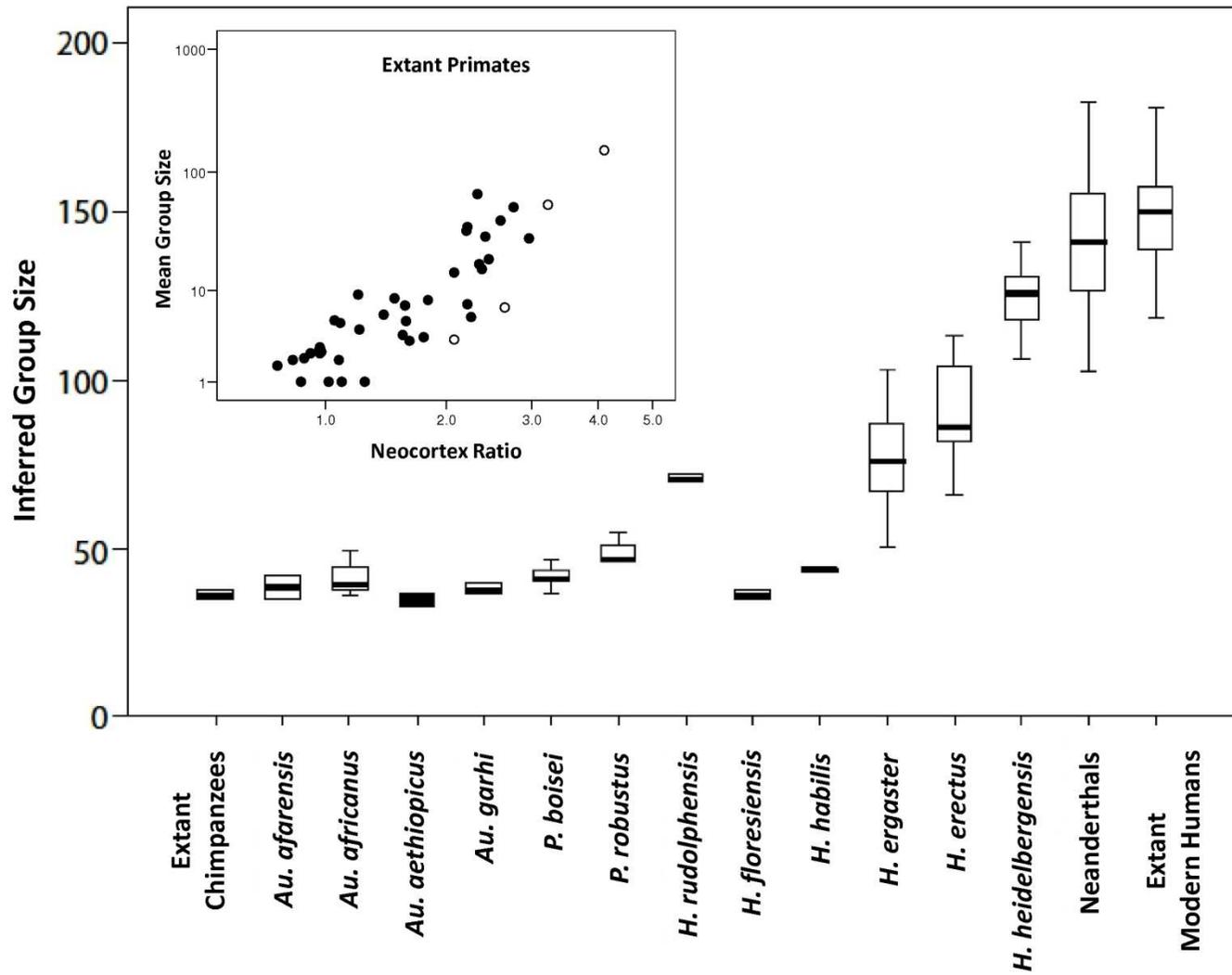


Figure 8.3: a) Group size in extant primates (small graph). The graph shows group size plotted against neocortex ratio in extant primates. Apes are the open symbols. Neocortex Ratio = neocortex ratio, the ratio of neocortex volume divided by the volume of the rest of the brain. Ape species are identified by open symbols; from lower left to top right: gibbons, gorillas, chimpanzees and modern humans. After Dunbar 2009.

b) Inferred group size for extinct hominins (large graph). This graph is based upon the regression of group size on neocortex ratio in extant primates (see above). ± 50 per cent and 95% ranges. After Gamble *et al.* 2011 based on equations from Dunbar 2009.

Relationships between 2D:4D and CAGn in humans are mirrored in the cross-species correlations; lower 2D:4D is associated with short CAGn (Manning *et al.* 2003a). 2D:4D and circulating testosterone were related across species (after body size was factored into the model), but correlations within human (intra-population) samples are generally not significant (Hönekopp *et al.* 2007; Muller *et al.* 2011; McIntyre *et al.* 2011; but see Manning *et al.* 1998; Manning *et al.* 2004c). A major problem with human studies is high variability in target traits within samples. Testosterone is a particularly difficult variable to capture because it is very labile (Gray *et al.* 1991). One method of circumventing these effects is to extend studies of human 2D:4D across populations with different social systems because circulating testosterone, ARG sensitivity and PAE have been shown to be higher in societies with higher levels of sexual selection (Kittles *et al.* 2001; Manning *et al.* 2007a; Alvergne *et al.* 2009).

Strengths and limitations: This study takes a more comprehensive approach to investigating evolutionary changes in androgen effects across haplorhines. The findings present additional evidence to show that OWM and apes have divergent androgen profiles that are consistent with differences in levels of sexual selection and sociality (Fig. 8.2). These profiles are consistent with PAE inferred by 2D:4D.

Recent studies within humans and across primates indicate that a down-regulation of androgens enhances empathy responses (Baron-Cohen *et al.* 2004; Guastella *et al.* 2008) while increased androgens may inhibit social bonding pathways (Carter 2007; van Honk *et al.* 2011; van Wingen *et al.* 2010). As androgens are known to impact human cognition (e.g., Baron-Cohen 2002; van Honk *et al.* 2011) and might be associated with variation in systemising and empathising abilities in human populations with different social systems, although this has not been tested; Simon Baron-Cohen pers. comm.; see Foley 2010). Large-scale differences in 2D:4D might also imply differences in programming neural reward pathways that maintain social bonding in primate lineages (e.g., OT and dopamine; Dunbar & Schultz 2010; Dunbar 2010b). Tantalising evidence exists in one study that shows differences in oxytocin in two macaque species with differing temperaments. The more socially distant pigtail macaque had lower oxytocin levels and higher circulating testosterone levels than the more socially gregarious and affiliative bonnet macaque; Rosenblume *et al.* 2002; Coe *et al.* 1992; see Chapter 6).

Higher OT and AVP are associated with promoting social bonding but they also play a role promoting feeling of social reward by stimulating the release of endorphins and by modulating the action of dopamine (Insel 1997; Dawson *et al.* 2005). For example, the

grooming of one primate by another causes a release of endorphins that promote feelings of well-being, OT is also released and this promotes feelings of calmness and trust between the dyad, while dopamine promotes the storing of social memories so that the behaviour is more likely to be repeated (Dunbar 2010b). This reward cascade is believed to underpin social attachment in humans and appears to be disrupted in individuals exposed to very high PAE (Insel 1997; Dawson *et al.* 2005; Carter 2007; also see Rilling *et al.* in press). Findings from 2D:4D support the evidence that PAE may be implicated in programming social bonding mechanisms (Manning *et al.* 2001; van Honk *et al.* 2011). PAE should therefore be incorporated into research paradigms investigating the evolution of sociality.

A major limitation of this study was the lack of control over the hormonal data taken from published sources. Although efforts were made to only use data analysed using similar techniques, variation in methods may have contributed to higher variation in testosterone values for some species in this study. In addition, there was very little published data on the NWM and OWM ARG. Studies of circulating testosterone are also poor in both NWM and apes. It is therefore possible that findings from this study may alter in the light of additional data.

Extending the research: It would be advantageous to replicate this study employing stricter methodological controls and incorporating a wider sample of species to test the hypothesis of two-androgenic pathways within catarrhines. It would be interesting to see how differences in inter-specific oxytocin levels or responses to OT and AVP differed between apes and OWM (see Rosenblume *et al.* 2002). OT is of particular interest because it is known to enhance the interpretation of social information (Donaldson & Young 2008; Guastella *et al.* 2008). It has also been implicated in facilitating and maintaining bonds over the longer term (Seltzer *et al.* 2010; also see Dunbar 2010b). Future studies should also address the co-variation between 2D:4D and ARG sensitivity within species.

Correlations in this study draw attention to the fact that it is still unclear how 2D:4D relates to PAE in terms of effects on the differing tissues within the digits (see Wallen 2009). For example is 2D:4D more indicative of ARG sensitivity than prenatal androgen levels (see Breedlove 2010)? Does subcutaneous fat impact 2D:4D (see Wallen 2009)? Do circulating hormonal effects impact digit lengths (Scutt *et al.* 1996)? Ethical restrictions prevent experimentation in order to disentangle genetic and cellular effects (Voracek 2011). However, this information may already be available; the elegant studies manipulating PAE in rhesus macaques provide the ideal research framework to directly test the impact of differing PAE on 2D:4D (Wallen 2005).

8.1.6: 2D:4D and social systems in extinct hominids and hominins

Summary and main findings: The findings of strong associations between 2D:4D and social systems were extended in Chapter 7 to predict the social systems of extinct hominids and hominins from fossil hand remains. Rather than the full digit length, the ratios of the 2nd to 4th proximal phalanges (2PP:4PP) was derived from a large sample of hominoid hand bones. As with soft tissue measurements, high 2PP:4PP in extant hominoids was associated with PB and low 2PP:4PP with promiscuous NPB mating systems. 2PP:4PP ratios in extant species were used to predict the social systems of extinct species from fossil 2P:4PP ratios. Main results indicate that the Miocene apes *Pierolapithecus catalaunicus* (~12.5 Mya) and *Hispanopithecus laietanus* (~9.5 Mya) and the Pliocene hominin *Ardipithecus ramidus* (~4.4 Mya) had social systems similar to extant NPB apes. *Australopithecus afarensis* (~3.2 Mya) differed from this pattern exhibiting a 2PP:4PP ratio that was consistent with PB. The early anatomically modern human (EAMH) Qafzeh 9 (~90kya) and Neanderthals (73-36 Kya) had higher ratios than NPB fossil apes and *Ar. ramidus* but had lower digit ratios than contemporary human populations. A polygynous social system was predicted for these Late Pleistocene hominins. 2PP:4PP was a more precise predictor of social system in extant hominoids than body size dimorphism.

Informing current understandings: The results augment current understandings of early ape and hominin evolution in several important ways. First, the results provide the only predictions of social systems for the Miocene apes *Pierolapithecus catalaunicus* and *Hispanopithecus laietanus*. This marks a significant finding because the fragmentary nature of Miocene fossil record makes predicting sexual dimorphism, and therefore social system, prone to high amounts of error (Begun 2004a). Predictions of a NPB social system for *Ar. ramidus* contradict the proposals of some researchers that this species was PB based on estimations of skeletal and canine size dimorphism (Lovejoy 2009). Close similarities in 2PP:4PP values between *Ar. ramidus* and the Miocene apes and extant great apes add to growing opinions that question the hominin status of *Ardipithecus* sp. (Sarmiento 2010; Wood & Harrison 2011).

The prediction of PB for *Au. afarensis prima facie* supports the assertions of Lovejoy (1981) and Reno *et al.* (2003) and adds to the on-going debate surrounding the social system of this species (see Gordon *et al.* 2008). However, it should be noted that the *Au. afarensis* material used in this study (A.L. 333) is less securely assigned to one individual than the other fossils in the sample (Bush *et al.* 1982; Alba *et al.* 2003). Intra-individual phalangeal data is critical

to the predictive power of the digit ratio method; these taphonomic concerns therefore reduce the reliability of the social system predictions for this hominin. The possibility of a PB social system in stem hominins, however, is not discounted. The ecological niches and geographic range of early apes appear to have been more diverse and less specialised than those of modern hominoids (Robson & Wood 2008; Begun 2009). Brain size and body size were variable in the Miocene (Begun 2004b; Alba 2010) and body size dimorphism is reported to be high suggesting high intra-sexual competition (Begun 2004b). This does not negate the possibility that a less competitive species also existed at this time. The fragmentary Middle Miocene fossil record is likely to be biased against the identification of a PB social system due to problems with sexing fossil material (see Begun 2004a, p 6). The fact that extant apes exhibit a range of social systems (including PB) in the face of reducing species diversity supports evidence that social systems may have been more variable in Miocene apes and the stem-hominin lineages that radiated out from them (see Foley & Lee 1989; Scott *et al.* 2009; Harrison 2010). The tendency to assume that there was a predominant ancestral social system for stem-hominins may actually narrow our thinking on the behavioural diversity of early hominins. For example, it is often assumed that a multi-male-multi-female social system was the ancestral state (see Foley & Gamble 2009), however extensive comparative studies of the primate reproductive system suggests the ancestral social system was either uni-male or monogamous (Dixson 2009).

Evidence from 2PP:4PP between the Miocene-Pliocene sample and the Late Pleistocene sample (3 Mya and 90 Kya) indicate a decrease in PAE which is consistent with reductions in intra-sexual competition. However, the lack of digit fossils (and therefore 2PP:4PP data) precludes predictions of social changes during this long and eventful phase in hominin evolution (see Fig. 8.4; Foley & Gamble 2009). The fact that body size dimorphism in some hominin species remained high until after 0.53 Mya (Arsuaga *et al.* 1997; Bischoff *et al.* 2007; also see Asuaga 2010) suggests that if a marked increase in brain size was associated with a shift towards a more human-like social structure around 0.5 Mya then it did not reduce male-male competition in all hominin species (Fig 8.4; see Aiello & Dunbar 1993; Dunbar 2009; 2010a). As such, high body size dimorphism in some Middle Pleistocene hominins conflicts with hypotheses proposing increasing feminisation-domestication through human evolution (Manning 2007a; Hare & Tomasello 2005a) and suggest that human-like social organisation may not have been in place until late in hominin evolution (Foley & Gamble 2009).

Neanderthals and EAMH are both predicted to have lived in polygynous social systems and predicted to be more competitive than most contemporary humans (Trinkaus 1980). High

variability of 2PP:4PP in the Neanderthal sample, however, implies that they may have had mating strategies that were as flexible as those of *Homo sapiens*; which is what we might expect from a hominin species with a similar brain size to humans. However, genetic evidence suggests that population size of *Homo neanderthalensis* was lower than that of *Homo sapiens*, and therefore probably falls below the estimates of inferred group size based upon group size/neocortex ratio correlations in primates (Fig 8.4; Aiello & Dunbar 1993; Dunbar 2009; Gamble *et al.* 2011). One of the on-going challenges in palaeo-anthropology is to understand how societies of Neanderthals, EAMH and more recent humans (>50 Kya) differed in their social structure and cognitive abilities (e.g., Kuhn & Stiner 2006; Foley & Gamble 2009; Gamble *et al.* 2010). The evidence presented here suggests PAE may be implicated in societal and cognitive differences in Late Pleistocene hominins (Fig. 8.3).

The shift to a recent modern human 2PP:4PP distribution and more generalised monogamy may not have occurred until the end of the Late Pleistocene with the evolution of agriculture around 11 Kya (Bramanti *et al.* 2009; Coward 2010)²⁴. Genetic evidence suggests that a monogamous PB social structure was adopted by expanding agrarian communities which replaced polygynous groups (Dupanloup *et al.* 2003; also see Frost 2006). Body size and skeletal robusticity significantly decreased in response to dietary and activity changes around this time (Spencer Larson 1995; Trinkaus 1997; Ruff 2005). Brain size also decreased and the shape of the cranium became more globular with a decrease in facial size (Lieberman 1998). The emergence of these physical traits, concomitant with decreases in PAE, suggests that Neolithic humans experienced a feminisation-domestication event as they moved into the novel 'adaptive space' created by an agricultural lifestyle. These changes may have supported the higher levels of within-group tolerance and cooperation necessary to maintain cohesion in groups with strong work obligations within a much larger societal structures. However, low PAE may also have enhanced cognitive abilities that enabled social cohesion to be maintained as populations expanded and fissioned and social networks became more extensive (e.g., circles of acquaintanceship; Dunbar 2009; Foley & Gamble 2009).

Strengths and limitations: This study is the first to extend the application of the digit ratio biomarker to the investigation of behaviour in extinct apes and hominins. This novel approach provides an additional method for inferring social behaviour from the fossil evidence. It provides support for the Feminisation Ape Hypothesis (Manning 2007a) by showing that digit ratios increase across hominin evolution and that the emergence of

²⁴ It is acknowledged that marriage systems vary between groups of contemporary people (e.g., foragers, pastoralists; see Muller *et al.* 2009).

modern human digit ratios was probably relatively recent. However, changes during the Neolithic are also consistent with the suite of traits that emerge with the domestication process (Hare & Tomasello 2005a; Hare 2007; Wobber *et al.* 2010b). It is unclear if feminisation and domestication are separate processes, whether one is transposed upon the other or if they act together in a synergistic way. Untangling the potential differences between these two processes, if indeed they are different, is beyond the scope of this thesis.

The 2PP:4PP method seems particularly appropriate for fossil hominoids because it is a more accurate predictor of social systems than body size dimorphism. Body size dimorphism is known to be a poor reflector of social system in extant primates (Plavcan 2001) and is notoriously difficult to predict from fragmentary fossils (Reno *et al.* 2003; Gordon *et al.* 2008). The on-going debates concerning the validity of methodologies of estimating body size dimorphism from fragmentary remains (see Gordon *et al.* 2008) highlight the importance of seeking alternative ways of estimating sexual selection for fossil hominins. The fact that 2PP:4PP does not correlate strongly with body size or body size dimorphism (Chapter 4) indicates that the ratio is targeting different PAE on behavioural development. This identifies the ratio as a marker of sexual selection that is independent of body size dimorphism and, as such, it can be used to augment current methods. The findings of this study are tentative as fossil sample sizes are small. The requirement for intra-individual sets of phalanges, and the fact that fossil phalanges are unlikely to ever constitute a large proportion of the hominin fossil record, also limit the use of this method.

Extending the research: As more fossil data become available it can be added into the existing model. The model could be extended to include other behavioural, physical and cultural markers of sociality; employing a phylogenetic comparative approach to analysing species' social markers as 'adaptive packages' presents a potentially powerful method for predicting changes in sociality across hominin evolution (see Foley & Gamble 2009). Potential functional effects on hand morphology in studies of extant primates are controlled for by incorporating substrate categories into the models (Plavcan & van Schaik 1992), however this method becomes problematic when analysing early hominin data because of their mixed locomotor repertoire. The model could be improved by introducing more refined categories of hand morphology based upon phalangeal curvature (Stern & Susman 1983; Richmond 2007). This 2PP:4PP method would also be applicable to investigating social changes in Neolithic and historic populations of modern humans. For example it should be possible to use proximal phalangeal data obtained from archaeological burials to trace transitions from polygyny to monogamy with the spread of agriculture into Europe (~7.5 Kya; Bramanti *et al.* 2009).

8.2: Conclusions

This final section returns to the aims of the thesis.

Aim 1: To look across haplorhine species to see if the patterns between 2D:4D and sexually selected traits in humans are reflected at higher taxonomic levels.

Relationships between 2D:4D and sexually selected behaviours in haplorhines were similar to those shown within and between human populations. No relationships were found between 2D:4D and sexually selected anatomical traits. Again, these findings are broadly consistent with human studies. In general the results imply that 2D:4D is a better indicator of PAE on brain pathways implicated in behavioural development than pathways that control sex differences in physical size. Sexual dimorphism in 2D:4D was evident at the species-level and was most apparent in species exhibiting higher levels of sexual selection; males had lower 2D:4D ratios than females. Male and female 2D:4D ratios were highly correlated within species. Similar patterns are shown in 2D:4D ratios within and between human populations. These findings are consistent with inter-genomic conflict of sexually antagonistic genes. Cross-species analyses between 2D:4D and sexually selected behaviours in haplorhines show extensive parallels to human 2D:4D studies and provide the first robust evidence that 2D:4D relationships generalise across a taxonomic group.

Aim 2: To look for patterns in the distribution of 2D:4D across haplorhines that might implicate PAE in broad-scale changes in sociality through primate evolution.

Digit ratio elicits a strong phylogenetic signal (Moran's I ; Pagel's λ) indicating that 2D:4D ratios are similar in closely related species. Similar phylogenetic constraints are shown for sexually selected core characteristics between related species. 2D:4D in NWM correlate with broad patterns of social behaviour but relationships were weaker than correlations across catarrhines. OWM and ape androgen profiles show divergent patterns and were consistent with differences in core social behaviours within these lineages. Increasing 2D:4D across great apes is indicative of a down-regulation of the androgen response over evolution. Large-scale phylogenetic differences in PAE in apes and OWM might underpin differences in social bonding mechanisms via programming effects on neuro-physiological pathways. These effects may be primarily a consequence of a reduction in ARG sensitivity in apes (indexed by an increase in 2D:4D ratios).

Aim 3: To investigate if variation of 2D:4D in extant apes might be informative about the role of PAE in hominin social evolution.

Reductions in 2D:4D and ARG sensitivity in extant hominids suggest that the Miocene species from which hominins evolved may have already had a reduced androgen response. A study of hominid and hominin digit bone ratios (2PP:4PP) detected an increase in 2PP:4PP (decrease PAE) between Miocene and Pliocene species and Late Pleistocene hominins. The most recent increase in digit ratios (decrease in PAE) appears to have occurred around ~11Kya when humans entered a new 'adaptive space' with the advent of agriculture. The results over all are consistent with the Feminised Ape Hypothesis (Manning 2007a) and are also line with Emotional Reactivity Hypothesis ('domestication'; Hare & Tomasello 2005a). Both hypotheses propose that a decrease in dominance-linked behaviour was integral to a decrease in endocrine response.

The aims of this thesis have been achieved. The series of studies have shown that 2D:4D generalises across haplorhine species at higher and low taxonomic levels. 2D:4D identifies PAE as candidates implicated in the development of sexually selected behaviours and social bonding pathways in haplorhine primates. This is of importance as the proximate mechanisms that underpin primate sociality, and which ultimately forms the evolutionary substrate for human sociality, are still poorly understood. PAE could improve our current understanding of primate social evolution if they are incorporated within research paradigms that investigate the bio-behavioural processes that potentially underpin sociality across taxonomic groups.

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Appendix 2

Appendix: 2.1: Calculation of 2D:4D from digit length measurements.

The 2D:4D ratio for each hand is calculated by dividing the mean (\bar{x}) of repeated length measurement of the second digit (2D; index) for the left hand by the mean of repeats of the fourth digit (4D; ring) of the left hand, the same formula is used for the right hand (see Manning, 2002). Thus:

$$\text{Left 2D:4D} = (\bar{x} \text{ 2D repeat measurements}) / (\bar{x} \text{ 4D repeat measurements})$$

For example data for a left hand might be 2D is 69.90 mm (first measurement) and 70.01 mm (second measurement) and for 4D is 72.28 mm (first measurement) and 72.52 mm (second measurement). The calculation is:

$$\text{Left 2D:4D} = ((69.90+70.01)/2) / ((72.28+72.52)/2) = (69.955) / (72.4) = 0.966$$

Data for a right hand might be 2D is 71.58 mm (first measurement) and 71.33 mm (second measurement) and for 4D is 75.27 mm (first measurement) and 75.96 mm (second measurement).

$$\text{Right 2D:4D} = ((71.58+71.33)/2) / ((75.27+75.96)/2) = (71.455) / (75.615) = 0.945$$

To calculate a mean 2D:4D values for the individual the value for 2D is obtained by calculating the mean of all repeats for 2D from both the left and right hands, similarly for 4D.

$$\text{Mean 2D:4D} = (\bar{x} \text{ 2D measurements for both hands}) / (\bar{x} \text{ 4D measurements for both hands})$$

The calculation is:

$$\text{Mean 2D:4D} = (\bar{x} \text{ of } 69.90;70.01;71.58;71.33) / (\bar{x} \text{ of } 72.28;72.52;75.27;75.96) = (70.71) / (73.26) = 0.955$$

²⁵ Each appendix's number corresponds to the chapter it is linked to

Appendix 2.2a: Study protocol (zoo).

PRIMATE DIGIT LENGTH DATA COLLECTION Zoo research protocol

Research project title

The length of the 2nd to 4th digit ratio (2D:4D) and its relationship to primate mating strategies and the evolution of human sociality.

Introduction

The length of the 2nd to 4th digit ratio (2D:4D) is sexually dimorphic in humans, primates and possibly other mammals (Phelps, 1952; Roney *et al.* 2004; McMechan *et al.* 2004) and is affected by levels of *in utero* sex hormones, specifically testosterone, estrogen and their derivatives (van Anders *et al.* 2006; Lutchmaya *et al.* 2004). Within human populations males have lower mean 2D:4D (shorter index finger compared to ring finger) than females. However, there is overlap between the sexes and variation between populations.

2D:4D correlates with many gender specific traits and behaviours, particularly reproductive and social abilities (see Manning, 2002a for an overview). For example, human males with low 2D:4D, compared to the population mean, tend to have higher sperm numbers per ejaculate and produce more offspring than males with high 2D:4D (Manning *et al.* 1998). Low 2D:4D has also been shown to correlate with more successful male-male competition behaviours such as greater levels of sporting and musical achievement (Manning and Taylor, 2001; Slumming and Manning 2000). A recent study has also linked low 2D:4D to increased levels of physical aggression in males, although no such relationship was shown in females (Bailey and Hurd, 2004).

Variation in 2D:4D has also been linked to behavioural differences between human populations; polygamous societies tending to have more masculinised digit ratios (low 2D:4D) than monogamous groups. Male-male competition seems to lead to selection for higher levels of foetal testosterone within a population resulting in lower 2D:4D (Manning *et al.* 2000a). Compared to humans, African ape 2D:4D (assessed from a few skeletal remains) is significantly more masculinised. It is possible that for African apes, exposure to higher levels of foetal testosterone affects adult male behaviour, allowing them to compete more successfully with other males for access to females, and possibly enhancing abilities to defend access to those females. A recent publication on 2D:4D in Guinea Baboons (*Papio papio*) suggests this may also be the case for cercopithecoid (Roney *et al.* 2004). It is worth noting that in this species the females had a more masculinised 2D:4D than the males. Lack of comparative primate data makes it impossible to investigate variation in inter- and intra-species 2D:4D, however.

Adolf Schultz (1947) commented on the differences between human and non-human primate digit ratios over 50 years ago. He observed that non-human primate digit ratios exhibited a more masculinised pattern than those of humans. Unfortunately he neglected to support this assertion with hard data. Based upon Schultz's observation and evidence of evolutionary change in the androgen receptor gene, Manning (2007a) hypothesised that a 'feminisation' had taken place through human evolution, but there is a lack of primate digit length data (particularly for African apes) to support this hypothesis.

If a 'feminisation' with increased foetal oestrogen exposure has taken place, it is hard to reconcile the mechanisms and timeframes of such an event, or events, with current evolutionary thinking. A late evolutionary change that might be linked to increased *in utero* estrogen is a reduction in skeletal robusticity (Churchill *et al.* 1996; Trinkaus, 1997). Conversely a move away from aggressive, male dominated mating strategies is thought to be a more ancient adaptation (Key and Aiello, 1999; Key, 2000). Language and higher levels

sociality, generally regarded as feminine characteristics, are estimated to have emerged half a million years ago (Dunbar, 2004), broadening mating strategies and facilitating the emergence of more complicated societies (Strum & Latour, 1997). These skeletal and social adaptations all point to selection for more feminised traits, which might also be reflected in fossil digit ratios.

Mating and social behaviours are central to primate and human group dynamics but prove difficult to trace in the archaeological record. The link between human 2D:4D and human inter-population mating behaviour seems strong, it could be that similar relationships will be found between inter-species mating behaviours in primates and digit ratio. Furthermore, it may be possible to use digit ratio as a means of tracing changes in mating strategies through human evolution.

2D:4D is normally obtained from soft-tissue measurement but correlation between whole finger length and bony digit length is good ($R^2=0.98$) (Manning, 2002a). If disarticulated archaeological and fossil material is to be interpreted, a relationship between total soft-tissue finger length and individual bony components of the digit must be established. It is hypothesised that the proximal phalanx will prove to be a good proxy for whole finger length, and there is some evidence to support this (Manning pers. comm.). Two studies have been designed to investigate bone and soft-tissue relationships: a human hand radiograph study (Royal Liverpool University Hospital Trust, UK) and a primate hand dissection study (National Museums of Scotland, UK).

The overall aims of this research project are to assess the validity of using 2D:4D as a tool to investigate the past and trace changes in mating behaviour through time. Tracking evolutionary change in hominin mating behaviours has to begin with an analysis of non-human primate 2D:4D. A non-human primate digit database will form the core of this research project.

Hypotheses

Species in which males are under intense pressure to compete for access to females will be exposed to high levels of foetal testosterone and will therefore be expected to have the lowest 2D:4D. Conversely, species with less competitive mating systems will be exposed to less prenatal testosterone and will be expected to have higher 2D:4D.

Objectives of primate digit data collection study

Build up a database of non-human primate digit lengths in order to;

1. Make a comparative study between humans and non-human primate digit ratios, with particular focus on correlating 2D:4D with measures of sexual selection and social complexity.
2. Preliminary analysis of relationships between non-human primate mating strategies and digit ratio use the index on inter-male competition formulated and utilised by Plavcan and van Schaik, 1992 and Kay *et al.* 1988. See below for preliminary results.

This database and analysis will provide the framework to enable metrics from fossil hominin digits to be incorporated into the study. It is hoped that the information derived from this aspect of the investigation will help inform us about temporal changes in human social evolution (Dunbar, 2004).

Rationale for conducting the project

While human 2D:4D continues to be extensively investigated and has shown some interesting results (see Manning, 2002a for an overview), non-human primate 2D:4D data is virtually nonexistent. We are only just beginning to understand how the prenatal

environment impacts on later life and 2D:4D can act as a window into this crucial time of growth and development. While the interests of this study lie in tracking evolutionary changes in mating and social behaviours, a database of non-human primate digit lengths may also inform us about factors such as disease susceptibility, fertility problems and may even provide insights into differences in timing of developmental phases between primate taxa (McFadden and Bracht, 2004; 2005).

Preliminary evidence to support the project

There is evidence in humans that the length ratio of the 2nd and 4th digits (2D:4D) is negatively correlated with prenatal testosterone (PT), and low male 2D:4D is associated with higher fertility and more successful male-male competitive behaviours. Human marriage systems may influence mean 2D:4D such that polygynous societies show high PT (indexed by low 2D:4D) while monogamous groups show low PT (indexed by high 2D:4D). Here we ask whether similar patterns of mean 2D:4D are evident in the mating systems of non-human primates.

The lengths of the 2nd and 4th digits from both hands were obtained from 276 anaesthetised captive primates (102 males). Mean 2D:4D was 0.839±0.072 for the right hand and 0.840±0.074 for the left, lower than typical human mean 2D:4D (0.98 to 1.00). Two-factor ANOVA with factors for sex (male, female) and taxon (Apes, Old World Monkeys, New World Monkeys, prosimians) with dependent variable right 2D:4D showed a significant main effect for taxon ($F=20.80$, $p=0.0001$) and non-significant effects for sex and the interaction. Apes had the highest mean 2D:4D (0.915). We excluded prosimians and performed a two-factor ANOVA (sex [male, female]) and mating system [1=low intensity, low frequency mating to 4=high intensity, high frequency mating]) with dependent variable right 2D:4D. There was a significant main effect for mating system ($F=9.19$, $p=0.0001$) with a reduction in mean 2D:4D with increasing competition for mates (1=0.906, 2=0.871, 3=0.853, 4=0.837). There were no significant sex or interaction effects.

We tentatively suggest that, on the evidence of 2D:4D, non-human primates have higher PT (indexed by lower 2D:4D) than humans, PT varies between taxa with the lowest PT (indexed by higher 2D:4D) found among Apes, and PT reduces with reducing sexual selection such that the lowest PT (indexed by highest 2D:4D) is found in mating systems with low intensity/low frequency mating. We consider the effects of lowered PT on human social evolution.

Justification for species and number of animals to be used

Variation in human 2D:4D is well documented but variation in non-human primate digit data is scarce. There is only one published study on non-human primate digit ratio, in a small sample of Guinea baboons (Roney *et al.* 2004; $n=31$). It is not possible to estimate inter- and inter-specific variation in non-human primate digit ratios without hard data because they seem to differ markedly from our own (based upon a small sample of skeletal remains and Roney *et al.* 2004). For valid comparative studies to be made, a large sample size is required. There is no restriction on sample size, although restrictions may be imposed if data from some species becomes too great.

As data can only be collected when a primate undergoes a general anaesthetic it is difficult to estimate the quantity of data that will be collected. For this reason data collection is set to run over a 2 year* period. It is hoped that this time frame will allow a significant amount of data to accumulate. *The data collection time frame can be negotiated.

Details of methods and experimental design of the study

Digit measurement: data collection involves the vet or nursing staff taking digit measurements from mature primates (any species, both sexes) while the animal is undergoing a general anaesthetic for a routine procedure such as a health check or

contraceptive implantation. Measurement is quick, simple and non-invasive; a ruler is placed on the mid-line of the digit and the reading is taken from the crease at the base of the digit to the fingertip, using a standardised technique. (Detailed instructions and diagrams are provided on a laminated sheet).

Accompanying information: some general information is also requested; the animal's zoo identification code, genus and species name, sex, age, weight (if possible) and the date of the procedure.

Data submission: an on-line facility is provided at www.digitratio.com. The Principal Investigator (Emma Nelson) will contact a link person every month and data can be passed on at this time. The link person would also be able to e-mail new data to the Principal Investigator at any time (enelson@liv.ac.uk). While it is acknowledged that data collection will add to the workload of your staff, it should not take longer than a couple of minutes to measure the digits.

Data analysis: numerical data will be analysed by the Kolmogorov-Smirnov test to determine normality. Where data is normally distributed it will be described in terms of mean and standard deviation, otherwise, non-parametric tests will be used. When comparing groups of data, the t-Test or ANOVA will be used for parametric data, and Mann-Whitney U test for non-parametric data. Where relationships are to be tested 'least squares' regression analysis will be performed on either raw data or transformed data, as appropriate.

Demands on the zoo staff

The vet or a member of veterinary staff will be required to take 2 sets of digit measurements from the primate while the animal is undergoing a general anaesthetic. The measurements should be entered onto the data sheet and, at a later time or date, submitted to the Principal Investigator (Emma Nelson) via e-mail (enelson@liv.ac.uk) or the on-line data sheet on the project's web site www.digitratio.com

Benefits to the zoo and primate breeding, conservation and health

This study will sample primates from the Europe, U.S.A., Canada and some regions of the African continent. So while this research may not directly benefit individual zoos and institutions, it should inform us about the impact of prenatal conditions on adult mating behaviours. Furthermore, the link between human 2D:4D and sex-dependant diseases may indicate that similar correlations could be found in non-human primates. It is possible that information derived from this study could be used alongside more conventional investigations to aid diagnosis of non-human primate health problems such as infertility. This project could, therefore, have relevance to primate breeding, conservation and health.

Dissemination of information

The PhD thesis is due to be submitted in 2010. It is anticipated that papers will be published prior to, and after the handing in date. Papers will be submitted to journals such as *Hormones and Behavior* and the *Journal of Human Evolution*.

Anticipated budget

A budget has not been set for primate digit data collection and analysis. I have so far been self-funded, and do not envisage problems with completing the project on this basis. Small grants are issued to this project on an annual basis from the University of Liverpool Faculty Postgraduate Research Fund.

Appendix 2.2a: Ethical approval.



THE UNIVERSITY
of LIVERPOOL

To: Emma Nelson
Doctoral Student
SACE

Re: Research approval

Date: 8th June 2006

School of Archaeology,
Classics and Egyptology

Hardley Building
Brownlow Street
Liverpool
L69 3GS

Telephone: 0151 794 5044
Facsimile: 0151 794 5057

Dear Emma Nelson

I write to inform you that SACE Research Committee has considered the ethical implications of your research and the documentation relating to the approval of your project by the external institutions with which you will be working. We are satisfied that your project has complied with the procedures and regulations relating to ethical matters established by the University. We are therefore happy to give approval to the continuance of your research.

Yours sincerely,

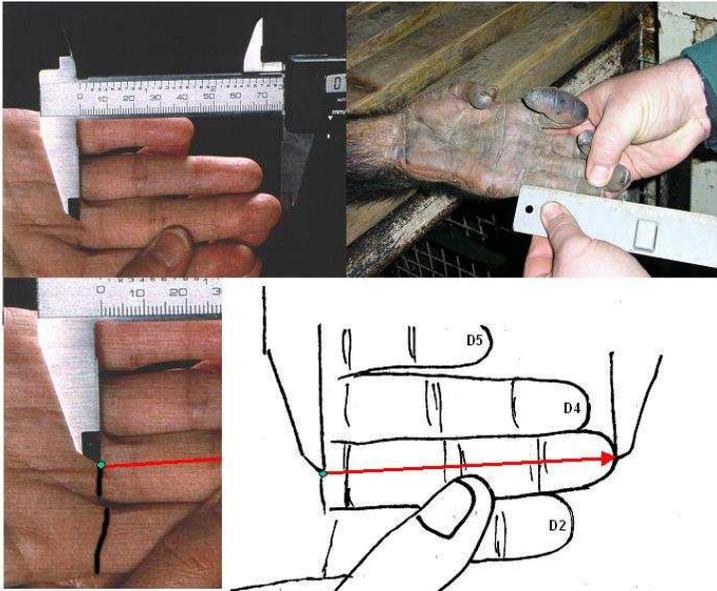
Dr. Doug Baird
Chair SACE Research committee



University switchboard
Telephone: 0151 794 2000
Facsimile: 0151 708 6502

Appendix 2.3: Digit measuring instructions.

Digit length measurement procedure



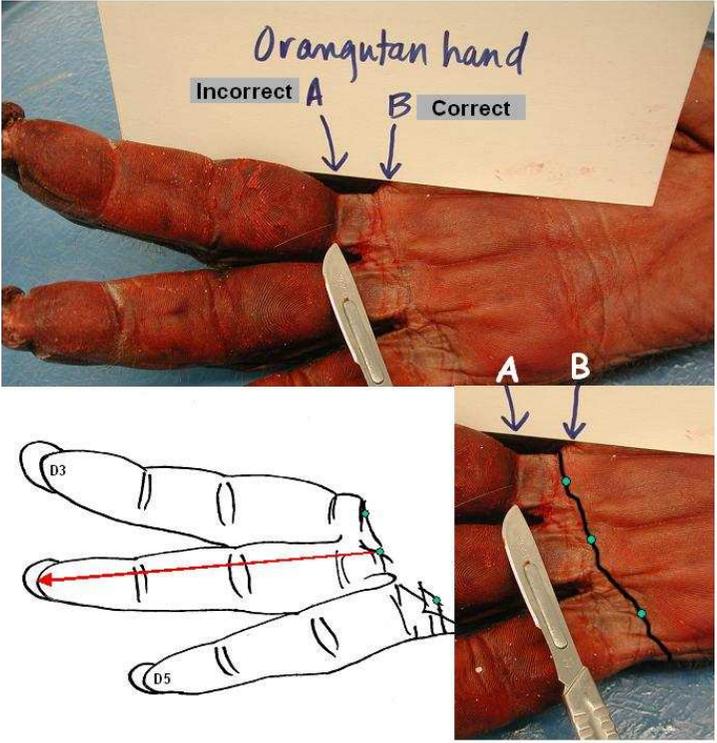
Procedure (please measure all digits except the thumb)

1. Ensure the finger being measured remains as straight as possible throughout the procedure.
2. Looking at the palm of the hand, locate the mid-point of the crease at the base of the finger. If using callipers, open them wider than the digit to be measured.
3. Place the 0 of the ruler exactly on this point. If using callipers place the jaw tip of the calliper exactly on this point.
4. While maintaining the finger in extension, gently place the ruler along the length of the middle of the finger, the mid-line. Or use the callipers to measure the same trajectory.
5. Looking directly at the ruler (or the digital display), record the length at the tip of the soft-tissue of the finger. Please do not include the finger nail or compress the soft tissue of the finger tip as this will distort the digit ratio. Record the length to the nearest mm.

Please measure the digit twice - if possible.

ECN_Protocol_0607

Identifying the proximal crease and landmarks



On some primates - this is a good example - the proximal crease can be quite 'broken' or there appears to be a number of creases, which makes identifying the crease difficult.

When the hand is in a relaxed position, the correct crease is the crease that forms a furrow at the base of the digits, closest to the palm (see other sheet for another example).

ECN_Protocol_0607

Appendix 2.4: References sources for human and non-primate 2D:4D data

Human 2D:4D data taken from:

Austin *et al.* 2002
Bailey & Hurd 2004
Bailey & Hurd 2005
Bang *et al.* 2005
Brosnan, 2006
Brown *et al.* 2002b
Coolican & Peters 2003
Csatho *et al.* 2003
Fink *et al.* 2003
Fink *et al.* 2004
Fink *et al.* 2006a
Fink *et al.* 2006b
Flegr *et al.* 2005
Flegr *et al.* 2008
Hall & Love 2003
Gobrogge *et al.* 2008
Hönekopp *et al.* 2006a
Hönekopp *et al.* 2006b
Kanchan *et al.* 2008
Kempel *et al.* 2005
Keogh *et al.* 2007
Koehler *et al.* 2004
Kuepper & Hennig 2007
Kyriakidis & Papaioannidou 2008
Loehlin *et al.* 2006
Lutchmaya *et al.* 2004

Human 2D:4D data taken from (cont.):

Manning & Leinster 2001
Manning & Peters 2009
Manning & Quinton 2007
Manning & Taylor 2001
Manning *et al.* 1998
Manning *et al.* 2000a
Manning *et al.* 2001
Manning 2002a
Manning 2002b
Manning *et al.* 2003a
Manning *et al.* 2003b
Manning *et al.* 2004a
Manning *et al.* 2004b
Manning *et al.* 2005
Manning *et al.* 2007a
Millet & Dewitte 2006
Ötken *et al.* 2002
Paul *et al.* 2006a
Pokrywka *et al.* 2005
Rahman & Wilson 2003
Rizwan *et al.* 2007
Robinson & Manning 2000
Ronalds *et al.* 2002
Trivers *et al.* 2006

Human 2D:4D data taken from (cont.):

Voracek & Dressler 2006a
Voracek & Dressler 2007b
Voracek & Offenmüller 2007
Voracek *et al.* 2007a
Voracek *et al.* 2007b
Wallian *et al.* 2008
Williams *et al.* 2003
Yang *et al.* 2009

Non-human 2D:4D references:

Lizards

Chang *et al.* 2006
Lombardo & Thorpe 2008
Rubolini *et al.* 2006

Bird

Burley & Foster 2004
Dreiss *et al.* 2007
Forstmeier 2005
Lombardo *et al.* 2008
Navarro *et al.* 2006

Rodents

Leoni *et al.* 2005
Lilley *et al.* 2009
Manning *et al.* 2003
McMachan *et al.* 2004

Appendix 2.5: Haplorhine 2D:4D dataset.

Species	Females							Males							Species						
	N ♀	Left	sd	Right	sd	Mean	sd	N ♂	Left	sd	Right	sd	Mean	sd	N	Left	sd	Right	sd	Mean	sd
<i>H. agilis</i>	1			0.97		0.97		0							1			0.97		0.97	
<i>H. hoolock</i>	1			1		1		0							1			1		1	
<i>H. klossii</i>	0							2	1.05	0.01	1.04	0.01	1.04	0.01	2	1.05	0.01	1.04	0.01	1.04	0
<i>H. lar</i>	2	1	0.01	1.11	0.07	1.07	0.01	4	1.05	0.04	1.08	0.03	1.05	0.01	6	1.04	0.04	1.09	0.04	1.07	0.01
<i>H. moloch</i>	1			0.98		0.98		1	0.99		1.02		1.01		2	0.99		1	0.03	1	0.02
<i>H. muelleri</i>	2	1		1.11	0.03	1.11	0.02	0							2	1		1.11	0.03	1.11	0.02
<i>H. pileatus</i>	1	1		1.13		1.11		1	1.03		1.11		1.07		2	1.01	0.02	1.12	0.02	1.09	0.03
<i>N. concolor</i>	3	1.01	0.03	1.04	0.02	1.02	0.01	0							3	1.01	0.03	1.04	0.02	1.02	0.01
<i>N. leucogenys</i>	3	1	0.03	0.99	0.03	1	0.03	3	1.02	0.08	0.98	0.01	1	0.04	6	1.01	0.05	0.98	0.02	1	0.03
<i>S. syndactylus</i>	7	1.06	0.06	1.05	0.05	1.06	0.04	7	1.06	0.11	1.02	0.09	1.03	0.09	14	1.06	0.08	1.03	0.07	1.05	0.07
<i>G. gorilla</i>	39	0.89	0.05	0.91	0.05	0.9	0.04	21	0.92	0.07	0.93	0.03	0.92	0.04	60	0.9	0.05	0.91	0.05	0.91	0.04
<i>P. paniscus</i>	12	0.93	0.03	0.92	0.05	0.92	0.03	13	0.91	0.03	0.92	0.04	0.91	0.03	25	0.92	0.03	0.92	0.04	0.92	0.03
<i>P. troglodytes</i>	148	0.91	0.06	0.91	0.05	0.91	0.05	104	0.9	0.07	0.89	0.06	0.9	0.05	252	0.91	0.06	0.91	0.06	0.91	0.05
<i>P. pygmaeus</i>	18	0.88	0.04	0.89	0.06	0.89	0.04	8	0.85	0.07	0.88	0.03	0.86	0.04	26	0.87	0.05	0.88	0.05	0.88	0.04
<i>A. nigroviridis</i>	3	0.88	0.05	0.85	0.02	0.86	0.03	4	0.88	0.07	0.87	0.07	0.87	0.07	7	0.88	0.06	0.86	0.05	0.87	0.05
<i>C. albegina</i>	2	0.88	0.04	0.83	0.03	0.86	0.04	3	0.87	0.04	0.87	0.03	0.87	0.04	5	0.88	0.04	0.85	0.03	0.87	0.03
<i>C. galeritus</i>	1	0.86		0.82		0.84		1	0.85		0.86		0.86		2	0.86	0.01	0.84	28	0.85	0.01
<i>C. ascanius</i>	1	0.76		0.79		0.77		0							1	0.76		0.79		0.77	
<i>C. campbelli</i>	1	0.79		0.81		0.80		0							1	0.79		0.81		0.8	
<i>C. diana</i>	2	0.88	0.05	0.86	0.07	0.87	0.06	6	0.86	0.08	0.88	0.07	0.87	0.07	8	0.87	0.07	0.87	0.06	0.87	0.07
<i>C. erythrotis</i>	0							3	0.82	0	0.82	0.01	0.82	0	3	0.82	0	0.82	0.01	0.82	0
<i>C. hamlyni</i>	1	0.91		0.94		0.93		2	0.78		0.83		0.8		3	0.82	0.08	0.87	0.07	0.84	0.07
<i>C. lhoesti</i>	3	0.9	0.05	0.86	0.05	0.88	0.03	1	0.84		0.85		0.84		4	0.88	0.05	0.86	0.04	0.87	0.03

Appendix 2.5: Haplorhine 2D:4D dataset continued.

Species	N	Females						N	Males						Species						
	♀	Left	sd	Right	sd	Mean	sd	♂	Left	sd	Right	sd	Mean	sd	N	Left	sd	Right	sd	Mean	sd
<i>C mona</i>	2	0.9	0.08	0.89	0.09	0.89	0.08	2	0.84	0.08	0.87	0.1	0.85	0.09	4	0.87	0.07	0.88	0.08	0.87	0.07
<i>C neglectus</i>	4	0.85	0.01	0.9	0.06	0.87	0.04	10	0.81	0.05	0.83	0.05	0.82	0.04	14	0.82	0.05	0.85	0.06	0.83	0.04
<i>C petaurista</i>	4	0.9	0.05	0.85	0.02	0.86	0.04	0							4	0.9	0.05	0.85	0.02	0.86	0.04
<i>C wolffi</i>	0							2	0.87	0.05	0.88	0.05	0.88	0.05	2	0.87	0.04	0.88	0.05	0.88	0.05
<i>C aethiops</i>	10	0.82	0.05	0.83	0.04	0.83	46	12	0.85	0.03	0.85	0.13	0.83	0.04	22	0.84	0.04	0.84	0.11	0.83	0.04
<i>M arctoides</i>	0							1	0.86		0.83		0.84		1	0.86		0.83		0.84	
<i>M fascicularis</i>	9	0.82	0.05	0.85	0.04	0.83	0.03	6	0.84	0.08	0.85	0.07	0.84	0.03	15	0.83		0.85		0.84	0.03
<i>M fuscata</i>	9	0.8	0.05	0.84	0.05	0.82	0.03	8	0.87	0.07	0.85	0.03	0.86	0.04	17	0.83	0.07	0.85	0.04	0.84	0.04
<i>M mulatta</i>	310	0.81	0.05	0.82	0.04	0.81	0.04	56	0.81	0.04	0.82	0.05	0.81	0.04	366	0.81	0.05	0.83	0.04	0.82	0.04
<i>M nigra</i>	2	0.79	0.02	0.85	0.01	0.82	0.01	1	0.88		0.84		0.86		3	0.82	0.05	0.85	0.01	0.83	0.02
<i>M silenus</i>	1	0.82		0.79		0.8		0							1	0.82		0.79		0.8	
<i>M sylvanus</i>	1	0.78		0.85		0.81		1	0.74		0.76		0.75		2	0.76	0.03	0.8	0.06	0.78	0.05
<i>M leucophaeus</i>	4	0.85	0.02	0.83	0.09	0.82	0.08	4	0.89	0.03	0.9	0.1	0.89	0.05	8	0.87	0.03	0.86	0.09	0.86	0.07
<i>M sphinx</i>	17	0.85	0.05	0.85	0.04	0.85	0.03	11	0.81	0.04	0.84	0.05	0.82	0.03	28	0.84	0.05	0.84	0.04	0.84	0.03
<i>P anubis</i>	1	0.93		0.84		0.88		0							1	0.93		0.84		0.88	
<i>P hamadryas</i>	15	0.86	0.04	0.86	0.04	0.86	0.04	11	0.88	0.05	0.83	0.05	0.85	0.04	26	0.87	0.04	0.85	0.05	0.86	0.04
<i>P papio</i>	21			0.83	0.05	0.83	0.05	11			0.88	0.05	0.88	0.05	32			0.85	0.05	0.85	0.05
<i>C guereza</i>	18	0.8	0.07	0.78	0.06	0.79	0.06	6	0.77	0.05	0.78	0.05	0.78	0.05	24	0.79	0.07	0.78	0.06	0.79	0.06
<i>P comata</i>	2	0.79	0.29	0.80	0.28	0.80	0.27	0							1	0.77		0.73		0.75	0
<i>P melalophos</i>	5	0.76	0.02	0.76	0.02	0.76	0	1	0.82		0.89		0.85		6	0.77	0.03	0.78	0.05	0.78	0.04
<i>P nemeaus</i>	0							1	0.79		0.86		0.82		1	0.79		0.86		0.82	

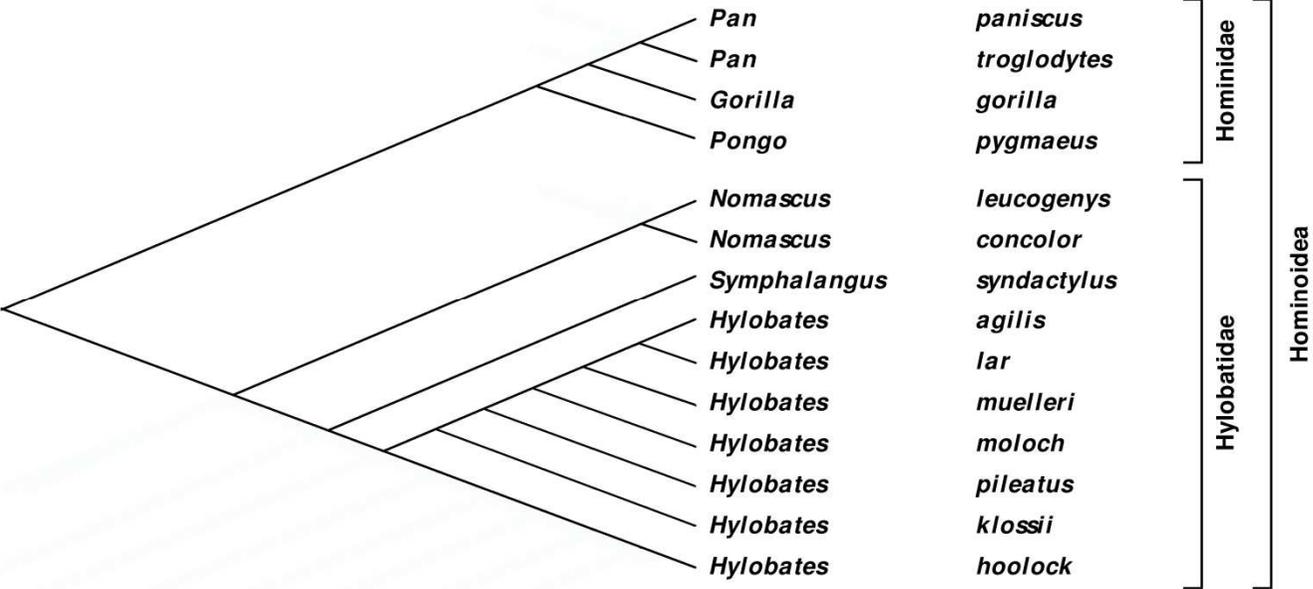
Appendix 2.5: Haplorhine 2D:4D dataset continued.

Species	N	Females						N	Males						Species						
	♀	Left	sd	Right	sd	Mean	sd	♂	Left	sd	Right	sd	Mean	sd	N	Left	sd	Right	sd	Mean	sd
<i>T auratus</i>	2	0.81	0.05	0.81	0.05	0.81	0	0							2	0.81	0.05	0.81	0.05	0.81	0
<i>T cristatus</i>	0							1	0.81		0.80		0.81		1	0.80		0.86		0.83	
<i>T francoisi</i>	8	0.79	0.04	0.79	0.03	0.79	0.03	6	0.78	0.05	0.77	0.05	0.77	0.04	14	0.78	0.04	0.78	0.04	0.78	0.04
<i>T obscurus</i>	5	0.79	0.03	0.8	0.03	0.8	0.03	2	0.77	0.07	0.84	0.03	0.81	0.03	7	0.79	0.04	0.82	0.03	0.8	0.03
<i>C goeldii</i>	0							4	0.96	0.05	0.96	0.03	0.96	0.04	4	0.96	0.05	0.96	0.03	0.96	0.04
<i>C argentata</i>	0							1	0.94		0.99		0.97		1	0.94		0.99		0.97	
<i>C geoffroyi</i>	8	0.92	0.06	0.89	0.09	0.9	0.06	4	0.94	0.06	0.98	0.17	0.96	0.09	12	0.93	0.06	0.92	0.13	0.92	0.07
<i>C jacchus</i>	33	0.93	0.07	0.93	0.07	0.93	0.06	36	0.91	0.07	0.94	0.08	0.93	0.07	69	0.92	0.07	0.93	0.08	0.93	0.06
<i>C pygmaea</i>	0							2	0.94	0.04	0.94	0.02	0.94	0.01	2	0.93	0.04	0.94	0.02	0.94	0.01
<i>L chrysomelas</i>	4	1	0.04	0.99	0.05	0.99	0.04	3	0.98	0.03	1.01	0.03	0.99	0.03	7	0.99	0.03	1	0.04	1	0.03
<i>L rosalia</i>	5	0.98	0.01	0.99	0.03	0.98	0.03	5	0.99	0.02	0.99	0.02	1	0.01	10	0.99	0.02	0.99	0.03	0.99	0.02
<i>S bicolor</i>	0							1	0.97		0.97		0.97		1	0.97		0.97		0.97	
<i>S geoffroyi</i>	1			1.12		1.12		4	0.99	0.03	0.97	0.01	0.98	0.01	5	0.99	0.03	1	0.06	1.01	0.06
<i>S imperator</i>	5	1	0.03	0.98	0.02	0.99	0.02	3	0.98	0.02	1.06	0	1.02	0.01	8	0.99	0.03	1.01	0.04	1	0.02
<i>S midas</i>	4	1.02	0.04	1	0.04	1.01	0.04	6	1.01	0.02	1.01	0.04	1.01	0.02	10	1.01	0.03	1.01	0.04	1.01	0.03
<i>S oedipus</i>	1	1		1.01		1.01		1	1		1.06		1.03		2	1		1.04	0.04	1.02	0.02
<i>C albifrons</i>	1	0.89		0.91		0.9		0							1	0.89		0.91		0.9	
<i>C apella</i>	11	0.93	0.06	0.95	0.06	0.94	0.06	9	0.96	0.04	0.92	0.03	0.95	0.04	20	0.95	0.05	0.95	0.05	0.95	0.05
<i>S sciureus</i>	9	0.9	0.04	0.91	0.03	0.9	0.03	3	0.88	0.05	0.89	0.01	0.89	0.03	12	0.89	0.04	0.9	0.03	0.9	0.04
<i>C donacophilus</i>	3	0.83	0.02	0.84	0.03	0.84	0.02	2	0.8	0	0.81	0.01	0.81	0.01	5	0.82	0.02	0.83	0.03	0.83	0.03
<i>C moloch</i>	12	0.87	0.03	0.84	0.03	0.86	0.02	21	0.86	0.05	0.85	0.04	0.85	0.03	33	0.87	0.04	0.84	0.03	0.86	0.03
<i>C torquatus</i>	0							2	0.95	0.12	0.9	0.13	0.92	0.12	2	0.95	0.12	0.9	0.13	0.92	0.12
<i>P pithecia</i>	1	0.78		0.84		0.81		5	0.73	0.03	0.76	0.05	0.74	0.04	6	0.74	0.03	0.78	0.05	0.76	0.04

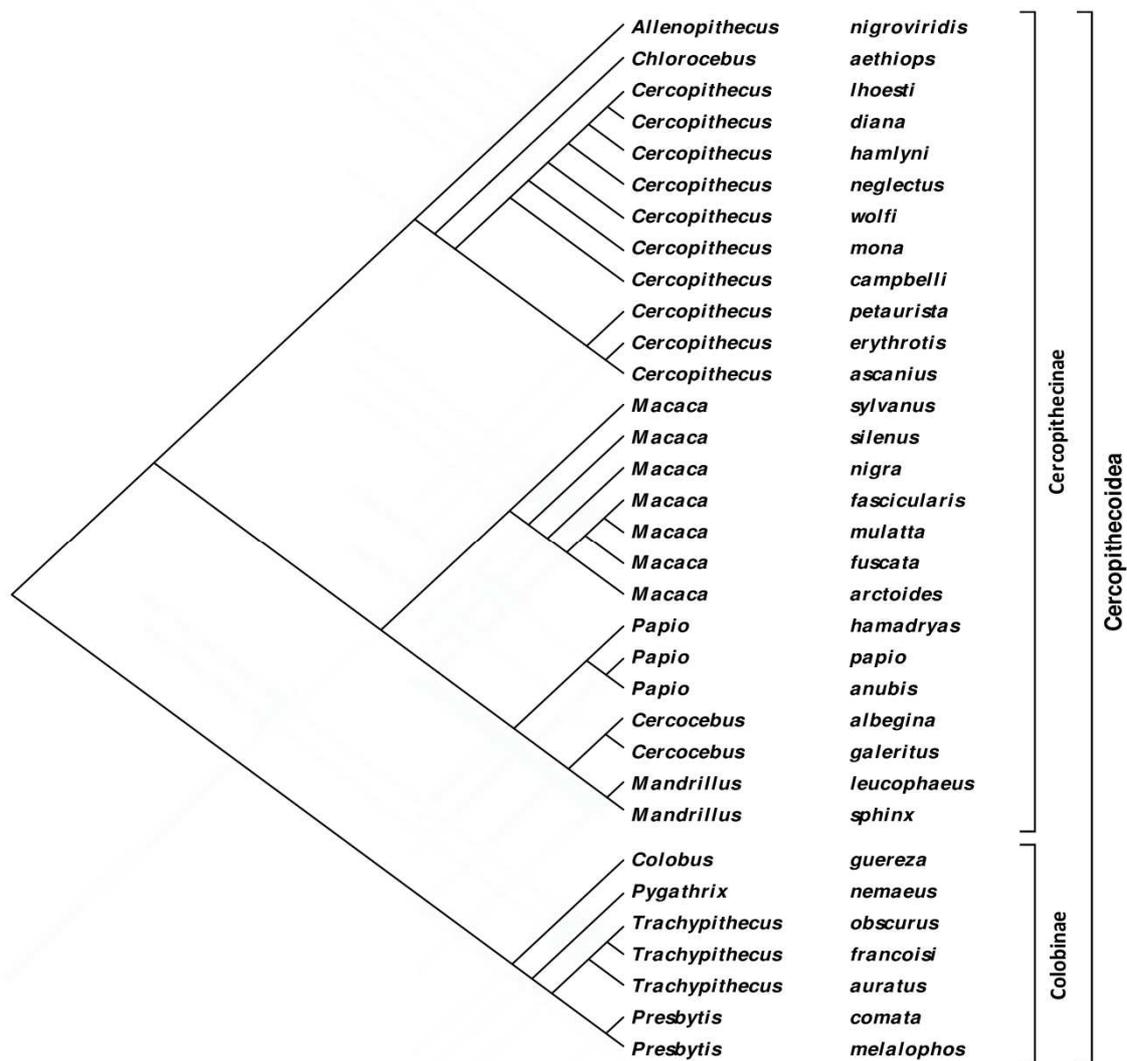
Appendix 2.5: Haplorhine 2D:4D dataset continued.

Species	Females							Males							Species						
	N ♀	Left	sd	Right	sd	Mean	sd	N ♂	Left	sd	Right	sd	Mean	sd	N	Left	sd	Right	sd	Mean	sd
<i>A caraya</i>	12	0.9	0.04	0.9	0.04	0.9	0.03	10	0.91	0.03	0.92	0.04	0.91	0.03	22	0.9	0.03	0.91	0.04	0.91	0.03
<i>A belzebuth</i>	1	0.95		0.93		0.94		0							1	0.95		0.93		0.94	
<i>A fusciceps</i>	0							2	0.91	0.1	0.95	0.05	0.93	0.07	2	0.91	0.1	0.95	0.05	0.93	0.07
<i>A geoffroyi</i>	4	0.9	0.04	0.92	0.04	0.91	0.03	1	0.92		0.87		0.9		5	0.9	0.03	0.91	0.04	0.91	0.03
<i>A hybridus</i>	2	0.86	0.05	0.86	0.04	0.86	0.01	1	0.86		0.8		0.83		3	0.86	0.04	0.84	0.04	0.85	0.02
<i>A paniscus</i>	3	0.96	0.06	0.93	0.06	0.94	0.06	0							3	0.96	0.06	0.93	0.06	0.94	0.06
<i>L lagotricha</i>	2	0.83	0.04	0.93	0.07	0.88	0.01	0							2	0.83	0.04	0.93	0.07	0.88	0.01
Total	823							463							1286						

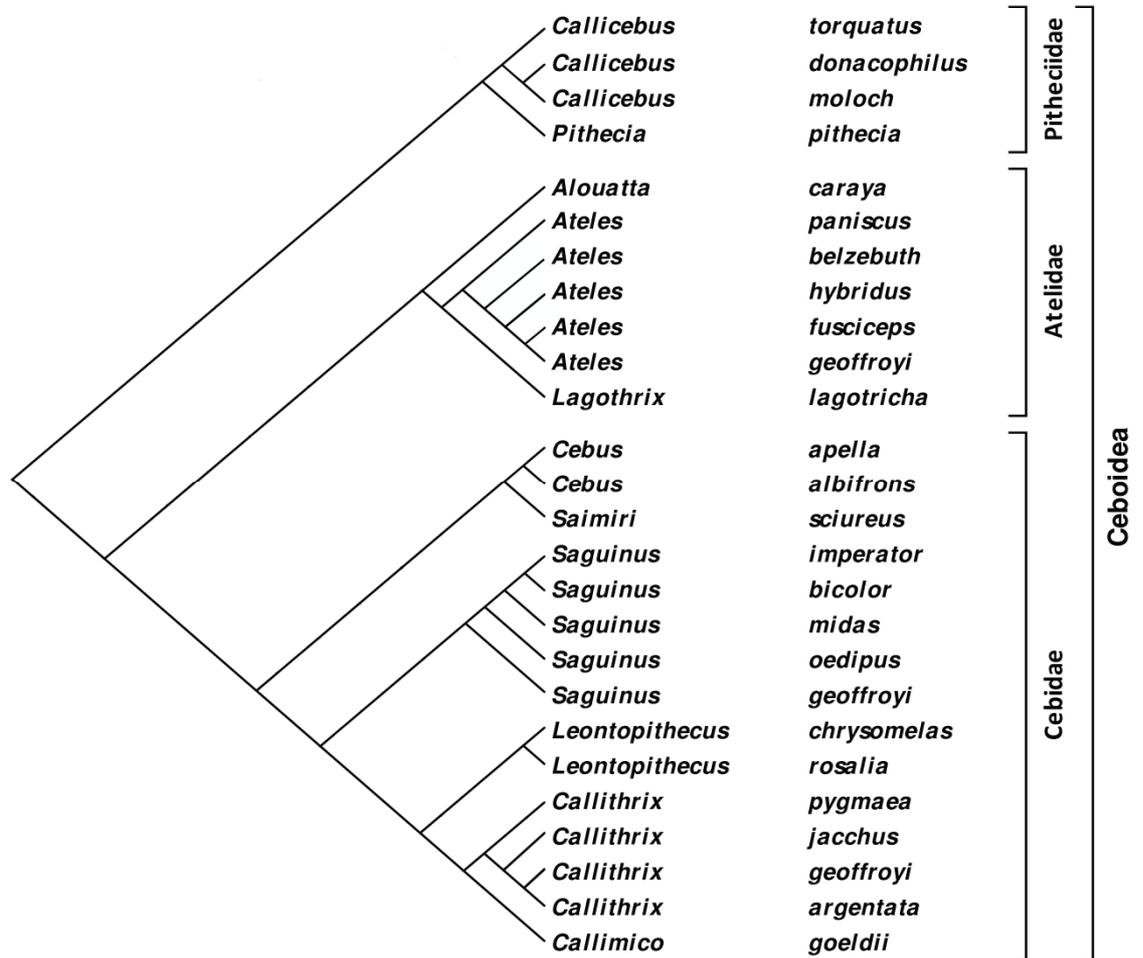
Appendix 2.6: Phylogenetic trees. Hominoidea (ape) phylogeny; Sub Order Haplorrhini; Pavorder: Catarrhini; Super Family: Hominoidea; Families: Hominidae, Hylobatidae. (Species order taken from Purvis, 1995).



Appendix 2.6: Phylogenetic trees continued. Cercopithecoidea (Old World Monkey) phylogeny; Sub Order Haplorrhini; Pavorder: Catarrhini; Super Family: Cercopithecoidea; Families: Cercpoithecinae, Colobinae (Species order taken from Purvis, 1995).



Appendix 2.6: Phylogenetic trees continued. Ceboidea (New World Monkey) phylogeny; Sub Order Haplorrhini; Pavorder: Platyrrhini; Super Family: Ceboidea; Families: Pitheciidae, Atelidae, Cebidae. (Species order taken from Opazo *et al.* 2006).



Appendix 3

Appendix 3.1: Study variables for 43 species. Categories: substrate, Plavcan & van Schaik 1992; social system and mating system, Plavcan 2004; inter-female competition levels, Sterck *et al.* 1997; inter-male competition levels, Plavcan & van Schaik 1997. Social System; PB=pair-bonded; NPB=non-pair-bonded; Mating System; PB=pair-bonded; UM= uni-male; MM=multi-male-multi-female; Female competition: 1=pair-bonded, 2=dispersed-egalitarian, 3=resident-egalitarian, 4=resident-nepotistic; °Male competition: 1=low frequency-low intensity, 2=high frequency-low intensity, 3=low frequency-high intensity, 4=high frequency-high intensity. Sub=Substrate; A=arboreal; A/T=arboreal/terrestrial; T=terrestrial.

Genus	Species	Social System	Mating System	Male Comp	Female Comp	Sub	Male Mass	Female Mass	N	Female Mean	sd	Male Mean	sd	Species Mean	sd	Zoo N
<i>Hylobates</i>	<i>lar</i>	PB	PB	LL	PB	A	5.90	5.34	6	1.07	0.01	1.05	0.01	1.07	0.01	4
<i>Hylobates</i>	<i>pileatus</i>	PB	PB	LL	PB	A	5.50	5.10	2	1.11		1.07		1.09	0.03	2
<i>Nomascus</i>	<i>leucogenys</i>	PB	PB	LL	PB	A	7.41	7.32	6	1.00	0.03	1.00	0.04	1.00	0.03	2
<i>Symphalangus</i>	<i>syndactylus</i>	PB	PB	LL	PB	A	11.88	10.71	14	1.06	0.04	1.03	0.09	1.05	0.07	7
<i>Gorilla</i>	<i>gorilla</i>	NPB	UM	LH	DE	AT	159.20	97.70	60	0.90	0.04	0.92	0.04	0.91	0.04	16
<i>Pan</i>	<i>paniscus</i>	NPB	MM	HL	DE	AT	43.00	33.20	25	0.92	0.03	0.91	0.03	0.92	0.03	4
<i>Pan</i>	<i>troglodytes</i>	NPB	MM	HL	DE	AT	42.00	35.20	248	0.91	0.05	0.90	0.05	0.91	0.05	17
<i>Pongo</i>	<i>pygmaeus</i>	NPB	UM	LH	DE	A	86.30	38.70	26	0.89	0.04	0.86	0.04	0.88	0.04	10
<i>Allenopithecus</i>	<i>nigroviridis</i>	NPB	UM	HH	RE	AT	6.30	3.18	7	0.86	0.03	0.87	0.07	0.87	0.05	3
<i>Cercocebus</i>	<i>albegina</i>	NPB	UM	HH	RE	A	9.00	6.40	5	0.86	0.04	0.87	0.04	0.87	0.03	1
<i>Cercocebus</i>	<i>galeritus</i>	NPB	UM	HH	RE	AT	10.20	5.50	2	0.84		0.86		0.85	0.01	1
<i>Cercopithecus</i>	<i>diana</i>	NPB	UM	LH	RE	A	5.20	3.90	8	0.87	0.06	0.87	0.07	0.87	0.07	3
<i>Cercopithecus</i>	<i>hamlyni</i>	NPB	UM	LH	RE	A	5.49	3.36	3	0.93		0.80		0.84	0.07	1
<i>Cercopithecus</i>	<i>lhoesti</i>	NPB	UM	LH	RE	AT	8.50	4.70	4	0.88	0.03	0.84		0.87	0.03	2
<i>Cercopithecus</i>	<i>mona</i>	NPB	UM	LH	RE	AT	4.40	2.50	4	0.89	0.08	0.85	0.09	0.87	0.07	2
<i>Cercopithecus</i>	<i>neglectus</i>	NPB	UM	LH	RE	A	7.00	3.96	14	0.87	0.04	0.82	0.04	0.83	0.04	4
<i>Chlorocebus</i>	<i>aethiops</i>	NPB	MM	HH	RN	AT	5.08	3.56	22	0.83	0.04	0.83	0.04	0.83	0.04	3

Appendix 3.1: Study variables for 43 species continued. Categories: substrate, Plavcan & van Schaik 1992; social system and mating system, Plavcan 2004; inter-female competition levels, Sterck *et al.* 1997; inter-male competition levels, Plavcan & van Schaik 1997. Social System; PB=pair-bonded; NPB=non-pair-bonded; Mating System; PB=pair-bonded; UM= uni-male; MM=multi-male-multi-female; Female competition: 1=pair-bonded, 2=dispersed-egalitarian, 3=resident-egalitarian, 4=resident-nepotistic; °Male competition: 1=low frequency-low intensity, 2=high frequency-low intensity, 3=low frequency-high intensity, 4=high frequency-high intensity. Sub=Substrate; A=arboreal; A/T=arboreal/terrestrial;T=terrestrial.

Genus	Species	Social System	Mating System	Male Comp	Female Comp	Sub	Male Mass	Female Mass	N	Female Mean	sd	Male Mean	sd	Species Mean	sd	Zoo N
<i>Macaca</i>	<i>fascicularis</i>	NPB	MM	HH	RN	AT	5.50	3.30	15	0.83	0.03	0.84	0.03	0.84	0.03	1
<i>Macaca</i>	<i>fuscata</i>	NPB	UM	HH	RN	AT	11.70	9.10	17	0.82	0.03	0.86	0.04	0.84	0.04	2
<i>Macaca</i>	<i>mulatta</i>	NPB	MM	HH	RN	AT	6.20	3.00	366	0.82	0.04	0.81	0.04	0.82	0.04	3
<i>Macaca</i>	<i>nigra</i>	NPB	MM	HH	RN	AT	10.40	6.60	3	0.82	0.01	0.86		0.83	0.02	2
<i>Macaca</i>	<i>sylvanus</i>	NPB	MM	HH	RN	AT	11.20	10.00	2	0.81		0.75		0.78	0.05	2
<i>Mandrillus</i>	<i>leucophaeus</i>	NPB	MM	HH	RN	AT	17.00	10.00	8	0.82	0.08	0.89	0.05	0.86	0.07	2
<i>Mandrillus</i>	<i>sphinx</i>	NPB	MM	HH	RN	AT	25.00	11.50	28	0.84	0.03	0.82	0.03	0.84	0.03	8
<i>Papio</i>	<i>hamadryas</i>	NPB	UM	HH	DE	T	21.50	9.40	26	0.86	0.04	0.85	0.04	0.86	0.04	5
<i>Colobus</i>	<i>guereza</i>	NPB	UM	HL	RE	A	10.60	8.70	24	0.79	0.06	0.78	0.05	0.79	0.06	13
<i>Presbytis</i>	<i>melalophos</i>	NPB	UM	HL	RE	A	6.70	6.60	6	0.76	0.00	0.85		0.78	0.04	2
<i>Trachypithecus</i>	<i>francoisi</i>	NPB	UM	HL	RE	A	7.70	7.35	12	0.79	0.03	0.77	0.04	0.78	0.04	5
<i>Trachypithecus</i>	<i>obscurus</i>	NPB	UM	HL	RE	A	8.30	6.50	8	0.80	0.03	0.81	0.03	0.80	0.03	3
<i>Callithrix</i>	<i>geoffroyi</i>	PB	PB	LL	PB	A	0.35	0.35	12	0.90	0.06	0.96	0.09	0.92	0.07	3
<i>Callithrix</i>	<i>jacchus</i>	PB	PB	LL	PB	A	0.26	0.24	69	0.93	0.06	0.93	0.07	0.93	0.06	4
<i>Leontopithecus</i>	<i>chrysomelas</i>	PB	PB	LH	PB	A	0.62	0.54	7	0.99	0.04	0.99	0.03	1.00	0.03	2
<i>Leontopithecus</i>	<i>rosalia</i>	PB	PB	LH	PB	A	0.62	0.60	10	0.98	0.03	1.00	0.01	0.99	0.02	4

Appendix 3.1: Study variables for 43 species continued. Categories: substrate, Plavcan & van Schaik 1992; social system and mating system, Plavcan 2004; inter-female competition levels, Sterck *et al.* 1997; inter-male competition levels, Plavcan & van Schaik 1997. Social System; PB=pair-bonded; NPB=non-pair-bonded; Mating System; PB=pair-bonded; UM= uni-male; MM=multi-male-multi-female; Female competition: 1=pair-bonded, 2=dispersed-egalitarian, 3=resident-egalitarian, 4=resident-nepotistic; °Male competition: 1=low frequency-low intensity, 2=high frequency-low intensity, 3=low frequency-high intensity, 4=high frequency-high intensity. Sub=Substrate; A=arboreal; A/T=arboreal/terrestrial;T=terrestrial.

Genus	Species	Social System	Mating System	Male Comp	Female Comp	Sub	Male Mass	Female Mass	N	Female Mean	sd	Male Mean	sd	Species Mean	sd	Zoo N
<i>Saguinus</i>	<i>imperator</i>	PB	PB	LH	PB	A	0.47	0.48	8	0.99	0.02	1.02	0.01	1.00	0.02	3
<i>Saguinus</i>	<i>midas</i>	PB	PB	LH	PB	A	0.59	0.43	10	1.01	0.04	1.01	0.02	1.01	0.03	4
<i>Saguinus</i>	<i>oedipus</i>	PB	PB	LH	PB	A	0.41	0.43	2	1.01		1.03		1.02	0.02	2
<i>Saimiri</i>	<i>sciureus</i>	NPB	UM	HL	RN	A	8.50	6.80	12	0.90	0.03	0.89	0.03	0.90	0.04	6
<i>Callicebus</i>	<i>donacophilus</i>	PB	PB	LL	PB	A	0.91	0.91	5	0.84	0.02	0.81	0.01	0.83	0.03	2
<i>Callicebus</i>	<i>moloch</i>	PB	PB	LL	PB	A	1.00	0.86	32	0.86	0.02	0.85	0.03	0.86	0.03	1
<i>Pithecia</i>	<i>pithecia</i>	NPB	MM	LH	DE	A	1.73	1.52	6	0.81		0.74	0.04	0.76	0.04	2
<i>Alouatta</i>	<i>caraya</i>	NPB	MM	HH	DE	A	6.80	4.61	22	0.90	0.03	0.91	0.03	0.91	0.03	7
<i>Ateles</i>	<i>geoffroyi</i>	NPB	MM	LH	DE	A	8.21	7.46	5	0.91	0.03	0.90		0.91	0.03	5
<i>Ateles</i>	<i>hybridus</i>	NPB	MM	LH	DE	A	9.50	7.00	3	0.86	0.01	0.83		0.85	0.02	3

Appendix 3.2: Moran's I values.

Species Variable	Moran's I			
	Expected	Observed	p	sd
Male 2D:4D	-0.03	0.41	<0.001	0.04
Female 2D:4D	-0.03	0.44	<0.001	0.04
Species mean 2D:4D	-0.03	0.44	<0.001	0.04
Male body weight	-0.03	0.35	<0.001	0.03
Female body weight	-0.03	0.40	<0.001	0.03
Species mean body weight	-0.03	0.35	<0.001	0.03

Appendix 4

Appendix 4.1: Sample variables. Body weight data taken from Smith and Jungers (1997) and Lindenfors and Tullberg (1998). Canine size taken from Plavcan (2004) and Thorén *et al.* (2006). Endocranial (ECV) volume taken from Isler *et al.* (2008). See Appendix 2.5 provides data on male and female 2D:4D and sample sizes.

Genus	Species	Male Weight	Female Weight	Species Weight	Male Canines	Female Canines	Species Canines	Male ECV	Female ECV	Species ECV
<i>Pan</i>	<i>paniscus</i>	43.0	33.2	38.1	15.6	11.2	13.4	356.3	326.3	341.29
<i>Pan</i>	<i>trogodytes</i>	42.0	35.2	38.6	21.7	15.3	18.5	386.2	350.5	368.35
<i>Gorilla</i>	<i>gorilla</i>	159.2	97.7	128.5	30.3	17.4	23.8	524.3	455.9	490.41
<i>Pongo</i>	<i>pygmaeus</i>	86.3	38.7	62.5	27.0	16.0	21.5	417.0	337.7	377.38
<i>Nomascus</i>	<i>leucogenys</i>	7.4	7.3	7.4						
<i>Symphalangus</i>	<i>syndactylus</i>	11.9	10.7	11.3	20.9	17.2	19.1	124.6	122.5	123.50
<i>Hylobates</i>	<i>lar</i>	5.9	5.3	5.6				103.3	100.5	101.87
<i>Hylobates</i>	<i>pileatus</i>	5.5	5.1	5.3				91.5	84.0	101.87
<i>Allenopithecus</i>	<i>nigroviridis</i>	6.3	3.2	4.7				65.4	53.7	58.20
<i>Chlorocebus</i>	<i>aethiops</i>	5.1	3.6	4.3	17.7	9.8	13.7	71.0	59.0	65.00
<i>Cercopithecus</i>	<i>lhoesti</i>	8.5	4.7	6.6	19.9	10.8	15.4	81.1	67.3	74.20
<i>Cercopithecus</i>	<i>diana</i>	5.2	3.9	4.6	19.6	12.3	16.0	70.1	55.2	62.61
<i>Cercopithecus</i>	<i>hamlyni</i>	5.5	3.4	4.4						
<i>Cercopithecus</i>	<i>neglectus</i>	7.0	4.0	5.5	20.0	11.6	15.8	71.1	60.9	65.97
<i>Cercopithecus</i>	<i>mona</i>	4.4	2.5	3.5	17.8	9.3	13.5	65.9	57.8	61.84
<i>Macaca</i>	<i>sylvanus</i>	11.2	10.0	10.6				97.5	87.9	77.93
<i>Macaca</i>	<i>nigra</i>	10.4	6.6	8.5	29.7	11.4	20.6	90.0	84.5	94.90
<i>Macaca</i>	<i>fascicularis</i>	5.5	3.3	4.4	24.1	10.7	17.4	65.8	62.1	63.98
<i>Macaca</i>	<i>mulatta</i>	6.2	3.0	4.6	17.0	8.1	12.6	93.7	84.3	88.98

Appendix 4.1: Sample variables continued. Body weight data taken from Smith and Jungers (1997) and Lindenfors and Tullberg (1998). Canine size taken from Plavcan (2004) and Thorén *et al.* (2006). Endocranial (ECV) volume taken from Isler *et al.* (2008). See Appendix 2.5 provides data on male and female 2D:4D and sample sizes.

Genus	Species	Male Weight	Female Weight	Species Weight	Male Canines	Female Canines	Species Canines	Male ECV	Female ECV	Species ECV
<i>Macaca</i>	<i>fuscata</i>	11.7	9.1	10.4	19.6	9.6	14.6	109.1	96.8	102.92
<i>Papio</i>	<i>hamadryas</i>	21.5	9.4	15.5	30.6	11.7	21.2	159.3	133.0	146.17
<i>Cercocebus</i>	<i>albegina</i>	9.0	6.4	7.7						
<i>Cercocebus</i>	<i>galeritus</i>	10.2	5.5	7.9						
<i>Mandrillus</i>	<i>leucophaeus</i>	17.0	10.0	13.5	50.0	11.7	30.8			
<i>Mandrillus</i>	<i>sphinx</i>	25.0	11.5	18.3				161.1	146.6	153.88
<i>Colobus</i>	<i>guereza</i>	10.6	8.7	9.7	20.3	13.9	17.1	79.0	69.8	74.39
<i>Trachypithecus</i>	<i>obscurus</i>	8.3	6.5	7.4	15.1	8.5	11.8	64.3	60.0	62.12
<i>Trachypithecus</i>	<i>francoisi</i>	7.7	7.4	7.5						
<i>Presbytis</i>	<i>melalophos</i>	6.7	6.6	6.7	14.0	8.2	11.1	70.5	59.2	64.85
<i>Callicebus</i>	<i>donacophilus</i>	0.9	0.9	0.9						
<i>Callicebus</i>	<i>moloch</i>	1.0	0.9	0.9	4.4	4.0	4.2			
<i>Pithecia</i>	<i>pithecia</i>	1.7	1.5	1.6	8.9	6.8	7.9	32.0	32.6	32.56
<i>Alouatta</i>	<i>caraya</i>	6.8	4.6	5.7	14.2	9.6	11.9	57.5	47.8	52.63
<i>Ateles</i>	<i>hybridus</i>	9.5	7.0	8.3				100.5	105.5	103.05
<i>Ateles</i>	<i>geoffroyi</i>	8.2	7.5	7.8	11.4	7.5	9.5	100.6	109.6	105.09
<i>Saimiri</i>	<i>sciureus</i>	8.5	6.8	7.7	7.1	5.0	6.1	24.3	24.0	24.14

Appendix 4.1: Sample variables continued. Body weight data taken from Smith and Jungers (1997) and Lindenfors and Tullberg (1998). Canine size taken from Plavcan (2004) and Thorén *et al.* (2006). Endocranial (ECV) volume taken from Isler *et al.* (2008). See Appendix 2.5 provides data on male and female 2D:4D and sample sizes.

Genus	Species	Male Weight	Female Weight	Species Weight	Male Canines	Female Canines	Species Canines	Male ECV	Female ECV	Species ECV
<i>Cebus</i>	<i>apella</i>	9.0	6.4	7.7	13.7	9.7	11.7	68.9	64.3	66.63
<i>Saguinus</i>	<i>imperator</i>	0.5	0.5	0.5						
<i>Saguinus</i>	<i>midas</i>	0.6	0.4	0.5	5.4	5.4	5.4			
<i>Saguinus</i>	<i>oedipus</i>	0.4	0.4	0.4	5.7	5.7	5.7	9.6	9.9	9.76
<i>Leontopithecus</i>	<i>chrysomelas</i>	0.6	0.5	0.6						
<i>Leontopithecus</i>	<i>rosalia</i>	0.6	0.6	0.6				13.0	12.7	12.83
<i>Callithrix</i>	<i>jacchus</i>	0.3	0.2	0.3				7.4	7.1	7.24
<i>Callithrix</i>	<i>geoffroyi</i>	0.4	0.4	0.4						

Appendix 4.2: Neonatal data (Smith and Leigh 1998). A= arboreal; A/T=arboreal/terrestrial (Plavcan and van Schaik 1992)

Genus	Species	Substrate	Neonatal Weight (g)			Neonatal Dimorphism	Species 2D:4D	Cohen's <i>d</i>	2D:4D Dimorphism
			Male	Female	Mean				
<i>Pan</i>	<i>paniscus</i>	A/T	1400.00	1494.00	1447.00	0.937	0.918	-0.34	0.988
<i>Pan</i>	<i>troglodytes</i>	A/T	1877.00	1814.00	1845.50	1.035	0.907	-0.34	0.981
<i>Gorilla</i>	<i>gorilla</i>	A/T	2251.00	1996.00	2123.50	1.128	0.907	0.63	1.028
<i>Pongo</i>	<i>pygmaeus</i>	A	1965.00	1653.00	1809.00	1.189	0.879	-0.53	0.976
<i>Nomascus</i>	<i>leucogenys</i>	A	481.00	500.00	490.50	0.962	1.023	0.62	1.013
<i>Symphalangus</i>	<i>syndactylus</i>	A	555.00	552.00	553.50	1.005	1.045	-0.32	0.979
<i>Hylobates</i>	<i>lar</i>	A	401.40	371.70	386.55	1.080	1.065	-3.03	0.982
<i>Hylobates</i>	<i>mulleri</i>	A	369.00	417.50	393.25	0.884	1.010		1.031
<i>Hylobates</i>	<i>moloch</i>	A	330.00	399.00	364.50	0.827	1.110		1.000
<i>Hylobates</i>	<i>pileatus</i>	A	385.00	405.70	395.35	0.949	1.091		0.960
<i>Chlorocebus</i>	<i>aethiops</i>	A/T	334.90	335.90	335.40	0.997	0.831	0.15	1.007
<i>Cercopithecus</i>	<i>neglectus</i>	A	455.00	450.00	452.50	1.011	0.834	-1.22	0.950
<i>Macaca</i>	<i>fascicularis</i>	A/T	353.10	326.10	339.60	1.083	0.835	0.28	1.008
<i>Macaca</i>	<i>mulatta</i>	A/T	490.30	466.30	478.30	1.051	0.819	-0.04	0.991
<i>Macaca</i>	<i>fuscata</i>	A/T	558.30	526.80	542.55	1.060	0.838	0.85	1.041
<i>Macaca</i>	<i>arcoides</i>	A	502.00	497.00	499.50	1.010	0.840		1.000
<i>Papio</i>	<i>hamadryas</i>	A/T	760.00	695.00	727.50	1.094	0.856	-0.20	0.991
<i>Papio</i>	<i>papio</i>	A/T	681.00	603.60	642.30	1.128	0.850		1.060
<i>Papio</i>	<i>anubis</i>	A/T	980.00	915.00	947.50	1.071	0.880		1.000
<i>Mandrillus</i>	<i>sphinx</i>	A/T	906.00	890.00	898.00	1.018	0.840	-0.82	0.975
<i>Colobus</i>	<i>guereza</i>	A	596.00	549.00	572.50	1.086	0.785	-0.24	0.984
<i>Pygathrix</i>	<i>nemaeus</i>	A	345.00	463.00	404.00	0.745	0.820		1.000

Appendix 4.2: Neonatal data continued (Smith and Leigh 1998).

Genus	Species	Substrate	Neonatal Weight (g)			Neonatal	Species	Cohen's	2D:4D
			Male	Female	Mean	Dimorphism	2D:4D	<i>d</i>	Dimorphism
<i>Lagothrix</i>	<i>lagotricia</i>	A	510.00	432.00	471.00	1.181	0.880		1.000
<i>Cebus</i>	<i>apella</i>	A	220.70	197.10	208.90	1.120	0.920		1.000
<i>Cebus</i>	<i>albifrons</i>	A	220.70	197.10	208.90	1.120	0.900		1.000
<i>Saimiri</i>	<i>sciureus</i>	A	112.50	106.40	109.45	1.057	0.898	-0.53	0.982
<i>Saguinus</i>	<i>oedipus</i>	A	44.00	42.10	43.05	1.045	1.018	0.44	1.022
<i>Leontopithecus</i>	<i>rosalia</i>	A	53.60	62.40	58.00	0.859	0.985	0.56	1.017
<i>Callithrix</i>	<i>pygmaea</i>	A	17.20	14.10	15.65	1.220	0.940		1.000
<i>Callithrix</i>	<i>jacchus</i>	A	30.15	30.23	30.19	0.997	0.928	-0.02	0.999
<i>Callimico</i>	<i>goeldii</i>	A	54.70	53.30	54.00	1.026	0.960	0.75	1.000

Appendix 4.3: Correlations between 2D:4D and study variables across the whole sample and for each super family. General Linear Model (GLM; non-phylogenetically controlled) results are shown first, followed by PLS results. Significant values in bold.

Sample	Model	GLM						
		F	p	df	R ²	Body Weight		
						F	p	
All	Male 2D:4D	Male weight	3.89	0.06	42	0.09		
All	Female 2D:4D	Female weight	2.49	0.12	42	0.05		
All	Species 2D:4D	Species weight	2.93	0.10	42	0.07		
All	Male 2D:4D	Body dimorphism	5.28	0.03	42	0.11		
All	Female 2D:4D	Body dimorphism	4.70	0.04	42	0.10		
All	Species 2D:4D	Body dimorphism	4.89	0.03	42	0.12		
All	Male 2D:4D	Male canine size	1.02	0.32	25	0.04		
All	Female 2D:4D	Female canine size	0.00	0.95	25	0.00		
All	Species 2D:4D	Species canine size	0.81	0.38	25	0.03		
All	Male 2D:4D	Male canine size + Body weight	1.09	0.31	24	0.03	0.263	0.612
All	Female 2D:4D	Female canine size + Body weight	0.22	0.64	24	0.02	0.449	0.509
All	Species 2D:4D	Species canine size + Body weight	0.83	0.37	24	0.04	0.189	0.668
All	Male 2D:4D	Canine dimorphism	7.40	0.01	30	0.20		
All	Female 2D:4D	Canine dimorphism	16.24	<0.001	30	0.35		
All	Species 2D:4D	Canine dimorphism	10.89	<0.01	30	0.27		
All	Male 2D:4D	ECV	0.44	0.51	30	0.01		
All	Female 2D:4D	ECV	0.19	0.67	30	0.01		
All	Species 2D:4D	ECV	0.19	0.67	30	0.01		
All	Male 2D:4D	ECV + body weight	0.98	0.33	30	0.01	1.34	0.26
All	Female 2D:4D	ECV + body weight	0.36	0.55	29	0.03	0.34	0.57
All	Species 2D:4D	ECV + body weight	0.96	0.34	29	0.05	0.66	0.42

Appendix 4.3: GLM (non-phylogenetically controlled) analysis across the whole sample continued.

Sample	Model	GLM				Body Weight	
		F	p	df	R ²	F	p
All	Male 2D:4D	ECV dimorphism	5.26	0.03	30	0.15	
All	Female 2D:4D	ECV dimorphism	4.49	0.04	30	0.13	
All	Species 2D:4D	ECV dimorphism	4.64	0.04	30	0.13	
All	Male 2D:4D	Male neonatal weigh	1.08	0.31	31	0.04	
All	Female 2D:4D	Female neonatal weight	0.44	0.51	31	0.02	
All	Species 2D:4D	Species neonatal weight	0.68	0.42	31	0.02	
All	Male 2D:4D	Neonatal dimorphism	10.42	<0.01	31	0.27	
All	Female 2D:4D	Neonatal dimorphism	10.47	<0.01	31	0.27	
All	Species 2D:4D	Neonatal dimorphism	10.66	<0.01	31	0.27	
All	Male 2D:4D	Group size	5.60	0.02	44	0.12	
All	Female 2D:4D	Group size	11.11	<0.01	44	0.21	
All	Species 2D:4D	Group size	7.21	0.01	44	0.14	
All	2D:4D dimorphism	Body dimorphism	0.33	0.57	42	0.01	
All	2D:4D dimorphism	Canine dimorphism	5.15	0.03	30	0.15	
All	2D:4D dimorphism	Neonatal dimorphism	0.10	0.92	31	0.00	
All	2D:4D dimorphism	Group size	1.90	0.18	44	0.04	
All	2D:4D dimorphism	ECV dimorphism	0.29	0.59	30	0.01	

Appendix 4.3: GLM (non-phylogenetically controlled) analysis of ape sample (Hominoidea) continued.

			GLM				Body Weight	
			F	p	df	R ²	F	p
Apes	Male 2D:4D	Male weight	27.05	<0.01	6	0.82		
Apes	Female 2D:4D	Female weight	39.31	<0.01	6	0.87		
Apes	Species 2D:4D	Species weight	43.03	<0.01	6	0.88		
Apes	Male 2D:4D	Body dimorphism	9.84	0.02	6	0.06		
Apes	Female 2D:4D	Body dimorphism	8.03	0.03	6	0.57		
Apes	Species 2D:4D	Body dimorphism	8.67	0.03	6	0.59		
Apes	Male 2D:4D	Male canine size	0.16	0.70	3	0.05		
Apes	Female 2D:4D	Female canine size	0.13	0.75	3	0.04		
Apes	Species 2D:4D	Species canine size	0.04	0.85	3	0.02		
Apes	Male 2D:4D	Male canine size + Body weight	0.43	0.58	2	0.62	3.05	0.22
Apes	Female 2D:4D	Female canine size + Body weight	0.11	0.77	2	0.75	4.95	0.16
Apes	Species 2D:4D	Species canine size + Body weight	0.64	0.51	2	0.77	6.63	0.12
Apes	Male 2D:4D	Canine dimorphism	0.01	0.93	3	0.00		
Apes	Female 2D:4D	Canine dimorphism	0.10	0.78	3	0.03		
Apes	Species 2D:4D	Canine dimorphism	0.04	0.85	3	0.02		
Apes	Male 2D:4D	ECV	44.87	<0.01	5	0.90		
Apes	Female 2D:4D	ECV	142.05	<0.001	5	0.97		
Apes	Species 2D:4D	ECV	96.57	<0.001	5	0.95		
Apes	Male 2D:4D	ECV + body weight	5.73	0.08	4	0.92	0.92	0.39
Apes	Female 2D:4D	ECV + body weight	14.57	0.02	4	0.98	1.59	0.28
Apes	Species 2D:4D	ECV + body weight	6.22	0.07	4	0.95	0.30	0.62

Appendix 4.3: GLM (non-phylogenetically controlled) analysis of ape sample (Hominoidea) continued.

			GLM				Body Weight	
			F	p	df	R ²	F	p
Apes	Male 2D:4D	ECV dimorphism	7.47	0.04	5	0.60		
Apes	Female 2D:4D	ECV dimorphism	6.01	0.05	5	0.55		
Apes	Species 2D:4D	ECV dimorphism	6.69	0.05	7	0.57		
Apes	Male 2D:4D	Male neonatal weigh	62.34	<0.001	10	0.89		
Apes	Female 2D:4D	Female neonatal weight	37.69	<0.001	10	0.82		
Apes	Species 2D:4D	Species neonatal weight	76.03	<0.001	10	0.91		
Apes	Male 2D:4D	Neonatal dimorphism	5.93	0.04	10	0.43		
Apes	Female 2D:4D	Neonatal dimorphism	3.78	0.08	10	0.33		
Apes	Species 2D:4D	Neonatal dimorphism	5.02	0.05	10	0.39		
Apes	Male 2D:4D	Group size	6.81	0.04	8	0.53		
Apes	Female 2D:4D	Group size	5.46	0.05	8	0.48		
Apes	Species 2D:4D	Group size	6.44	0.04	8	0.51		
Apes	2D:4D dimorphism	Body dimorphism	0.08	0.79	6	0.01		
Apes	2D:4D dimorphism	Canine dimorphism	1.57	0.28	4	0.28		
Apes	2D:4D dimorphism	Neonatal dimorphism	0.33	0.58	10	0.04		
Apes	2D:4D dimorphism	Group size	0.02	0.88	8	0.00		
Apes	2D:4D dimorphism	ECV dimorphism	0.23	0.65	5	0.04		

Appendix 4.3: GLM (non-phylogenetically controlled) analysis of Old World monkey (OWM) sample (Cercopithecoidea) continued.

							<u>Body Weight</u>	
			F	p	df	R ²	F	p
OWM	Male 2D:4D	Male weight	0.05	0.82	19	0.00		
OWM	Female 2D:4D	Female weight	6.17	0.02	19	0.25		
OWM	Species 2D:4D	Species weight	0.68	0.42	19	0.03		
OWM	Male 2D:4D	Body dimorphism	7.26	0.01	19	0.28		
OWM	Female 2D:4D	Body dimorphism	10.82	<0.01	19	0.36		
OWM	Species 2D:4D	Body dimorphism	16.33	<0.01	19	0.46		
OWM	Male 2D:4D	Male canine size	8.55	0.01	12	0.42		
OWM	Female 2D:4D	Female canine size	0.99	0.34	12	0.08		
OWM	Species 2D:4D	Species canine size	2.99	0.16	12	0.16		
OWM	Male 2D:4D	Male canine size + Body weight	10.25	<0.01	11	0.51	2.00	0.19
OWM	Female 2D:4D	Female canine size + Body weight	3.35	0.09	11	0.40	6.85	0.02
OWM	Species 2D:4D	Species canine size + Body weight	5.75	0.04	11	0.35	3.25	0.10
OWM	Male 2D:4D	Canine dimorphism	5.83	0.02	12	0.36		
OWM	Female 2D:4D	Canine dimorphism	0.42	0.53	12	0.03		
OWM	Species 2D:4D	Canine dimorphism	2.29	0.16	12	0.16		
OWM	Male 2D:4D	ECV	0.01	0.93	14	0.00		
OWM	Female 2D:4D	ECV	0.01	0.91	14	0.00		
OWM	Species 2D:4D	ECV	0.00	0.99	14	0.00		
OWM	Male 2D:4D	ECV + body weight	2.69	0.13	13	0.20	3.20	0.10
OWM	Female 2D:4D	ECV + body weight	3.64	0.08	13	0.37	7.59	0.02
OWM	Species 2D:4D	ECV + body weight	3.41	0.09	13	0.27	4.73	0.05

Appendix 4.3: GLM (non-phylogenetically controlled) analysis of Old World monkey (OWM) sample (Cercopithecoidea) continued.

			GLM				Body Weight	
			F	p	df	R ²	F	p
OWM	Male 2D:4D	ECV dimorphism	2.03	0.18	14	0.01		
OWM	Female 2D:4D	ECV dimorphism	0.13	0.72	14	0.01		
OWM	Species 2D:4D	ECV dimorphism	0.29	0.65	14	0.01		
OWM	Male 2D:4D	Male neonatal weigh	0.69	0.42	12	0.07		
OWM	Female 2D:4D	Female neonatal weight	1.05	0.33	12	0.10		
OWM	Species 2D:4D	Species neonatal weight	1.27	0.29	12	0.11		
OWM	Male 2D:4D	Neonatal dimorphism	0.47	0.83	12	0.01		
OWM	Female 2D:4D	Neonatal dimorphism	0.69	0.42	12	0.06		
OWM	Species 2D:4D	Neonatal dimorphism	0.95	0.77	12	0.01		
OWM	Male 2D:4D	Group size	1.25	0.28	21	0.06		
OWM	Female 2D:4D	Group size	0.44	0.53	21	0.02		
OWM	Species 2D:4D	Group size	0.44	0.51	21	0.02		
OWM	2D:4D dimorphism	Body dimorphism	0.16	0.69	19	0.01		
OWM	2D:4D dimorphism	Canine dimorphism	4.73	0.05	14	0.25		
OWM	2D:4D dimorphism	Neonatal dimorphism	1.38	0.27	12	12.00		
OWM	2D:4D dimorphism	Group size	3.11	0.09	21	0.14		
OWM	2D:4D dimorphism	ECV dimorphism	5.24	0.04	14	0.27		

Appendix 4.3: GLM (non-phylogenetically controlled) analysis of New World monkey (NWM) sample (Ceboidae) continued.

			GLM				Body Weight	
			F	p	df	R ²	F	p
NWM	Male 2D:4D	Male weight	2.86	0.12	13	0.18		
NWM	Female 2D:4D	Female weight	2.24	0.16	13	0.15		
NWM	Species 2D:4D	Species weight	2.04	0.18	13	0.14		
NWM	Male 2D:4D	Body dimorphism	0.32	0.26	13	0.14		
NWM	Female 2D:4D	Body dimorphism	0.15	0.71	13	0.01		
NWM	Species 2D:4D	Body dimorphism	0.15	0.70	13	0.01		
NWM	Male 2D:4D	Male canine size	0.14	0.72	6	0.02		
NWM	Female 2D:4D	Female canine size	0.01	0.94	6	0.00		
NWM	Species 2D:4D	Species canine size	0.04	0.85	6	0.01		
NWM	Male 2D:4D	Male canine size + Body weight	0.05	0.83	5	0.08	0.30	0.61
NWM	Female 2D:4D	Female canine size + Body weight	0.15	0.76	5	0.11	0.49	0.52
NWM	Species 2D:4D	Species canine size + Body weight	0.09	0.72	5	0.07	0.35	0.58
NWM	Male 2D:4D	Canine dimorphism	0.04	0.84	6	0.01		
NWM	Female 2D:4D	Canine dimorphism	0.04	0.85	6	0.01		
NWM	Species 2D:4D	Canine dimorphism	0.04	0.85	6	0.01		
NWM	Male 2D:4D	ECV	1.78	0.22	7	0.20		
NWM	Female 2D:4D	ECV	1.97	0.20	7	0.22		
NWM	Species 2D:4D	ECV	1.41	0.24	7	0.17		
NWM	Male 2D:4D	ECV + body weight	0.43	0.54	6	0.21	0.02	0.89
NWM	Female 2D:4D	ECV + body weight	0.47	0.52	6	0.22	0.02	0.89
NWM	Species 2D:4D	ECV + body weight	0.49	0.51	6	0.18	0.07	0.80

Appendix 4.3: GLM (non-phylogenetically controlled) analysis of New World monkey (NWM) sample (Ceboidae) continued.

			GLM				Body Weight	
			F	p	df	R ²	F	p
NWM	Male 2D:4D	ECV dimorphism	0.34	0.58	7	0.05		
NWM	Female 2D:4D	ECV dimorphism	0.60	0.81	7	0.01		
NWM	Species 2D:4D	ECV dimorphism	0.17	0.69	7	0.02		
NWM	Male 2D:4D	Male neonatal weigh	3.97	0.08	9	0.36		
NWM	Female 2D:4D	Female neonatal weight	4.47	0.72	9	0.39		
NWM	Species 2D:4D	Species neonatal weight	4.43	0.07	9	0.39		
NWM	Male 2D:4D	Neonatal dimorphism	3.03	0.12	9	0.30		
NWM	Female 2D:4D	Neonatal dimorphism	3.20	0.12	9	0.31		
NWM	Species 2D:4D	Neonatal dimorphism	2.19	0.13	9	0.29		
NWM	Male 2D:4D	Group size	3.97	0.08	9	0.01		
NWM	Female 2D:4D	Group size	4.47	0.07	9	0.02		
NWM	Species 2D:4D	Group size	4.43	0.07	9	0.02		
NWM	2D:4D dimorphism	Body dimorphism	0.66	0.43	13	0.05		
NWM	2D:4D dimorphism	Canine dimorphism	0.94	0.36	8	0.11		
NWM	2D:4D dimorphism	Neonatal dimorphism	1.14	0.27	7	0.17		
NWM	2D:4D dimorphism	Group size	0.00	0.97	15	0.00		
NWM	2D:4D dimorphism	ECV dimorphism	1.70	0.23	7	0.19		

Appendix 4.3: PGLS (phylogenetically controlled) analysis across the whole sample continued.

Sample	Model	PGLS						
		F	p	df	λ	Body Weight		
						F	p	
All	Male 2D:4D	Male weight	1.92	0.17	42	0.97		
All	Female 2D:4D	Female weight	2.32	0.16	42	0.99		
All	Species 2D:4D	Species weight	2.49	0.12	42	0.99		
All	Male 2D:4D	Body dimorphism	0.85	0.36	42	0.87		
All	Female 2D:4D	Body dimorphism	0.02	0.89	42	0.99		
All	Species 2D:4D	Body dimorphism	0.32	0.57	42	0.99		
All	Male 2D:4D	Male canine size	0.04	0.83	26	0.96		
All	Female 2D:4D	Female canine size	0.07	0.79	26	1.00		
All	Species 2D:4D	Species canine size	0.20	0.66	26	0.96		
All	Male 2D:4D	Male canine size + Body weight	0.60	0.45	25	0.98	2.66	0.115
All	Female 2D:4D	Female canine size + Body weight	1.08	0.31	25	0.98	4.19	0.05
All	Species 2D:4D	Species canine size + Body weight	0.15	0.70	25	0.98	2.38	0.135
All	Male 2D:4D	Canine dimorphism	0.38	0.54	26	0.96		
All	Female 2D:4D	Canine dimorphism	4.09	0.08	26	1.00		
All	Species 2D:4D	Canine dimorphism	1.66	0.21	26	0.97		
All	Male 2D:4D	ECV	0.00	0.98	30	0.97		
All	Female 2D:4D	ECV	0.02	0.90	30	0.98		
All	Species 2D:4D	ECV	0.21	0.64	30	0.99		
All	Male 2D:4D	ECV + body weight	0.63	0.44	29	0.98	0.85	363
All	Female 2D:4D	ECV + body weight	0.80	0.38	29	0.90	1.53	0.22
All	Species 2D:4D	ECV + body weight	1.39	0.24	29	1.00	1.27	0.269

Appendix 4.3: PGLS (phylogenetically controlled) analysis across the whole sample continued.

Sample	Model	PGLS				Body Weight	
		F	p	df	λ	F	p
All	Male 2D:4D	ECV dimorphism	1.16	0.29	30	0.98	
All	Female 2D:4D	ECV dimorphism	0.22	0.65	30	0.99	
All	Species 2D:4D	ECV dimorphism	0.13	0.72	30	0.99	
All	Male 2D:4D	Male neonatal weigh	3.22	0.83	29	1.00	
All	Female 2D:4D	Female neonatal weight	1.27	0.27	29	0.94	
All	Species 2D:4D	Species neonatal weight	1.83	0.19	29	0.99	
All	Male 2D:4D	Neonatal dimorphism	0.24	0.63	29	0.99	
All	Female 2D:4D	Neonatal dimorphism	1.28	0.27	29	0.93	
All	Species 2D:4D	Neonatal dimorphism	0.40	0.53	29	0.99	
All	Male 2D:4D	Group size	0.53	0.47	42	0.97	
All	Female 2D:4D	Group size	0.15	0.67	42	0.99	
All	Species 2D:4D	Group size	0.10	0.75	42	0.99	
All	2D:4D dimorphism	Body dimorphism	0.32	0.58	42	0.00	
All	2D:4D dimorphism	Canine dimorphism	5.55	0.03	26	0.00	
All	2D:4D dimorphism	Neonatal dimorphism	0.01	0.97	29	0.00	
All	2D:4D dimorphism	Group size	1.81	0.19	42	0.00	
All	2D:4D dimorphism	ECV dimorphism	3.23	0.08	30	0.00	

Appendix 4.3: PGLS (phylogenetically controlled) analysis of ape sample (Hominoidea) continued.

			PGLS				<u>Body Weight</u>	
			F	p	df	λ	F	p
Apes	Male 2D:4D	Male weight	1.64	0.27	6	1.00		
Apes	Female 2D:4D	Female weight	29.48	<0.01	6	0.00		
Apes	Species 2D:4D	Species weight	4.04	0.09	6	1.00		
Apes	Male 2D:4D	Body dimorphism	1.88	0.22	6	1.00		
Apes	Female 2D:4D	Body dimorphism	1.63	0.25	6	1.00		
Apes	Species 2D:4D	Body dimorphism	1.95	0.21	6	1.00		
Apes	Male 2D:4D	Male canine size	0.13	0.74	4	1.00		
Apes	Female 2D:4D	Female canine size	0.00	0.95	4	1.00		
Apes	Species 2D:4D	Species canine size	0.13	0.73	4	1.00		
Apes	Male 2D:4D	Male canine size + Body weight	0.02	0.89	3	1.00	0.64	0.479
Apes	Female 2D:4D	Female canine size + Body weight	0.01	0.91	3	1.00	1.99	0.252
Apes	Species 2D:4D	Species canine size + Body weight	0.05	0.83	3	1.00	0.20	0.25
Apes	Male 2D:4D	Canine dimorphism	1.43	0.28	4	1.00		
Apes	Female 2D:4D	Canine dimorphism	5.64	0.08	4	1.00		
Apes	Species 2D:4D	Canine dimorphism	3.50	0.13	4	1.00		
Apes	Male 2D:4D	ECV	5.09	0.07	5	1.00		
Apes	Female 2D:4D	ECV	101.46	<0.001	5	0.00		
Apes	Species 2D:4D	ECV	68.98	<0.001	5	0.00		
Apes	Male 2D:4D	ECV + body weight	3.36	0.14	4	1.00	1.13	0.34
Apes	Female 2D:4D	ECV + body weight	11.13	0.03	4	1.00	2.00	0.22
Apes	Species 2D:4D	ECV + body weight	3.55	0.13	4	0.00	0.17	0.702

Appendix 4.3: PGLS (phylogenetically controlled) analysis of ape sample (Hominoidea) continued.

			PGLS				Body Weight	
			F	p	df	λ	F	p
Apes	Male 2D:4D	ECV dimorphism	0.68	0.45	5	1.00		
Apes	Female 2D:4D	ECV dimorphism	0.25	0.64	5	1.00		
Apes	Species 2D:4D	ECV dimorphism	0.56	0.49	5	1.00		
Apes	Male 2D:4D	Male neonatal weigh	49.87	>0.001	8	0.00		
Apes	Female 2D:4D	Female neonatal weight	30.15	>0.001	8	0.00		
Apes	Species 2D:4D	Species neonatal weight	60.82	>0.001	8	0.00		
Apes	Male 2D:4D	Neonatal dimorphism	4.75	0.06	10	0.99		
Apes	Female 2D:4D	Neonatal dimorphism	0.33	0.57	10	0.21		
Apes	Species 2D:4D	Neonatal dimorphism	0.41	0.54	10	0.72		
Apes	Male 2D:4D	Group size	0.26	0.62	6	1.00		
Apes	Female 2D:4D	Group size	0.01	0.93	6	1.00		
Apes	Species 2D:4D	Group size	0.09	0.78	6	1.00		
Apes	2D:4D dimorphism	Body dimorphism	0.06	0.82	6	0.00		
Apes	2D:4D dimorphism	Canine dimorphism	1.07	0.36	4	1.00		
Apes	2D:4D dimorphism	Neonatal dimorphism	0.26	0.62	8	0.00		
Apes	2D:4D dimorphism	Group size	0.01	0.89	6	0.00		
Apes	2D:4D dimorphism	ECV dimorphism	0.16	0.70	5	0.00		

Appendix 4.3: PGLS (phylogenetically controlled) analysis of Old World monkey (OWM) sample (Cercopithecoidea) continued.

			PGLS				Body Weight	
			F	p	df	λ	F	p
OWM	Male 2D:4D	Male weight	0.06	0.80	19	0.47		
OWM	Female 2D:4D	Female weight	0.61	0.80	19	0.87		
OWM	Species 2D:4D	Species weight	0.62	0.44	19	0.87		
OWM	Male 2D:4D	Body dimorphism	6.68	0.02	19	0.00		
OWM	Female 2D:4D	Body dimorphism	2.40	0.14	19	0.80		
OWM	Species 2D:4D	Body dimorphism	3.04	0.10	19	0.63		
OWM	Male 2D:4D	Male canine size	4.90	0.05	12	0.42		
OWM	Female 2D:4D	Female canine size	0.22	0.65	15	0.79		
OWM	Species 2D:4D	Species canine size	0.62	0.45	12	0.78		
OWM	Male 2D:4D	Male canine size + Body weight	5.31	0.04	11	0.22	0.689	0.42
OWM	Female 2D:4D	Female canine size + Body weight	0.70	0.41	11	0.73	0.84	0.379
OWM	Species 2D:4D	Species canine size + Body weight	0.51	0.41	11	0.77	0.02	0.87
OWM	Male 2D:4D	Canine dimorphism	6.85	0.02	12	0.56		
OWM	Female 2D:4D	Canine dimorphism	0.24	0.63	12	0.83		
OWM	Species 2D:4D	Canine dimorphism	0.89	0.36	12	0.81		
OWM	Male 2D:4D	ECV	0.02	0.88	14	0.33		
OWM	Female 2D:4D	ECV	0.02	0.89	14	0.86		
OWM	Species 2D:4D	ECV	0.00	0.99	14	0.83		
OWM	Male 2D:4D	ECV + body weight	2.18	0.16	13	0.00	2.61	0.13
OWM	Female 2D:4D	ECV + body weight	0.87	0.37	13	0.00	127.00	0.279
OWM	Species 2D:4D	ECV + body weight	0.46	0.51	13	0.74	0.57	0.4622

Appendix 4.3: PGLS (phylogenetically controlled) analysis of Old World monkey (OWM) sample (Cercopithecoidea) continued.

			PGLS				Body Weight	
			F	p	df	λ	F	p
OWM	Male 2D:4D	ECV dimorphism	4.68	0.07	14	0.56		
OWM	Female 2D:4D	ECV dimorphism	0.84	0.38	14	0.90		
OWM	Species 2D:4D	ECV dimorphism	0.28	0.11	14	0.84		
OWM	Male 2D:4D	Male neonatal weigh	2.26	0.14	10	1.00		
OWM	Female 2D:4D	Female neonatal weight	1.43	0.25	10	0.23		
OWM	Species 2D:4D	Species neonatal weight	1.81	0.38	10	0.44		
OWM	Male 2D:4D	Neonatal dimorphism	2.88	0.12	10	0.98		
OWM	Female 2D:4D	Neonatal dimorphism	0.34	0.54	10	0.54		
OWM	Species 2D:4D	Neonatal dimorphism	3.21	0.10	10	0.91		
OWM	Male 2D:4D	Group size	0.69	0.42	19	0.36		
OWM	Female 2D:4D	Group size	0.16	0.69	19	0.87		
OWM	Species 2D:4D	Group size	0.52	0.48	19	0.84		
OWM	2D:4D dimorphism	Body dimorphism	0.12	0.73	19	0.87		
OWM	2D:4D dimorphism	Canine dimorphism	5.05	0.04	12	0.17		
OWM	2D:4D dimorphism	Neonatal dimorphism	1.15	0.30	10	0.00		
OWM	2D:4D dimorphism	Group size	2.81	0.11	19	0.00		
OWM	2D:4D dimorphism	ECV dimorphism	4.64	0.07	14	0.00		

Appendix 4.3: PGLS (phylogenetically controlled) analysis of New World monkey (NWM) sample (Ceboidae) continued.

			PGLS				Body Weight	
			F	p	df	λ	F	p
NWM	Male 2D:4D	Male weight	0.08	0.78	13	0.91		
NWM	Female 2D:4D	Female weight	0.01	0.90	13	0.95		
NWM	Species 2D:4D	Species weight	0.04	0.84	13	0.91		
NWM	Male 2D:4D	Body dimorphism	0.00	1.00	13	0.91		
NWM	Female 2D:4D	Body dimorphism	0.07	0.78	13	95.00		
NWM	Species 2D:4D	Body dimorphism	0.01	0.91	13	0.96		
NWM	Male 2D:4D	Male canine size	0.12	0.74	6	0.88		
NWM	Female 2D:4D	Female canine size	0.01	0.91	6	1.00		
NWM	Species 2D:4D	Species canine size	0.06	0.81	6	0.90		
NWM	Male 2D:4D	Male canine size + Body weight	0.00	0.97	5	0.88	0.157	0.71
NWM	Female 2D:4D	Female canine size + Body weight	0.10	0.77	5	1.00	0.39	0.561
NWM	Species 2D:4D	Species canine size + Body weight	0.00	0.98	5	0.90	0.13	0.724
NWM	Male 2D:4D	Canine dimorphism	1.72	0.24	6	1.00		
NWM	Female 2D:4D	Canine dimorphism	1.16	0.32	6	1.00		
NWM	Species 2D:4D	Canine dimorphism	1.50	0.27	6	1.00		
NWM	Male 2D:4D	ECV	0.42	0.54	7	1.00		
NWM	Female 2D:4D	ECV	0.35	0.57	7	0.93		
NWM	Species 2D:4D	ECV	0.57	0.47	7	1.00		
NWM	Male 2D:4D	ECV + body weight	0.30	0.60	6	1.00	0.06	0.8085
NWM	Female 2D:4D	ECV + body weight	0.27	0.62	6	0.92	0.08	0.78
NWM	Species 2D:4D	ECV + body weight	0.30	0.60	6	1.00	0.02	0.882

Appendix 4.3: PGLS (phylogenetically controlled) analysis of New World monkey (NWM) sample (Ceboidae) continued.

			PGLS				Body Weight	
			F	p	df	λ	F	p
NWM	Male 2D:4D	ECV dimorphism	0.25	0.63	7	0.70		
NWM	Female 2D:4D	ECV dimorphism	0.01	0.97	7	0.67		
NWM	Species 2D:4D	ECV dimorphism	0.10	0.76	7	0.79		
NWM	Male 2D:4D	Male neonatal weigh	0.16	0.69	7	1.00		
NWM	Female 2D:4D	Female neonatal weight	0.23	0.64	7	1.00		
NWM	Species 2D:4D	Species neonatal weight	0.25	0.63	7	1.00		
NWM	Male 2D:4D	Neonatal dimorphism	0.20	0.66	7	1.00		
NWM	Female 2D:4D	Neonatal dimorphism	0.19	0.67	7	1.00		
NWM	Species 2D:4D	Neonatal dimorphism	0.17	0.68	7	1.00		
NWM	Male 2D:4D	Group size	0.16	0.69	7	1.00		
NWM	Female 2D:4D	Group size	0.24	0.64	7	1.00		
NWM	Species 2D:4D	Group size	0.24	0.63	7	1.00		
NWM	2D:4D dimorphism	Body dimorphism	0.18	0.68	13	1.00		
NWM	2D:4D dimorphism	Canine dimorphism	0.47	0.52	6	1.00		
NWM	2D:4D dimorphism	Neonatal dimorphism	0.13	0.72	7	1.00		
NWM	2D:4D dimorphism	Group size	0.11	0.73	13	0.40		
NWM	2D:4D dimorphism	ECV dimorphism	1.45	0.27	7	0.32		

Appendix 4.4: Moran's I values for Chapter 4.

Species Variable	Moran's I			
	Expected	Observed	p	sd
Log Male 2D:4D	-0.024	0.234	<0.001	0.036
Log Female 2D:4D	-0.024	0.256	<0.001	0.035
Log Species 2D:4D	-0.024	0.251	<0.001	0.036
Log 2D:4D dimorphism	-0.023	0.040	0.057	0.033
Log Male body weight	-0.024	0.549	0.001	0.035
Log Female body weight	-0.024	0.523	0.001	0.035
Log Species body weight	-0.024	0.541	0.001	0.035
Log Body dimorphism	-0.024	0.286	0.001	0.036
Log Male canine size	-0.041	0.485	0.001	0.056
Log Female canine size	-0.041	0.581	0.001	0.059
Log Species canine size	-0.041	0.554	0.001	0.058
Log Canine dimorphism	-0.041	0.341	<0.001	0.501
Log Male neonatal weight	-0.033	0.487	0.01	0.049
Log Female neonatal weight	-0.033	0.496	0.01	0.049
Log Species neonatal weight	-0.033	0.497	0.01	0.048
Log Neonatal weight dimorphism	-0.033	0.937	0.01	0.049
Log Male ECV	-0.033	0.507	0.001	0.049
Log Female ECV	-0.033	0.477	0.001	0.049
Log Species ECV	-0.033	0.498	0.001	0.049
Log ECV dimorphism	-0.032	0.048	<0.001	0.048
Log Total group size	-0.041	0.798	<0.0001	0.014

Appendix 6

Appendix 6.1: Source references for testosterone values. F = females; M = Males.

Species	T	Sex	Reference	Species	T	Sex	Reference
<i>Homo sapiens</i>	Serum	M	McCamant <i>et al.</i> 1987.	<i>Pan troglodytes</i>	Faecal	M	Kutsukake <i>et al.</i> 2009.
<i>Homo sapiens</i>	Serum	M	Weiss <i>et al.</i> 1983.	<i>Pan troglodytes</i>	Salivary	M	Kutsukake <i>et al.</i> 2009.
<i>Homo sapiens</i>	Serum	M	Trojesen & Sandnes 2004.	<i>Gorilla gorilla</i>	Serum	M	Coe <i>et al.</i> 1992.
<i>Homo sapiens</i>	Serum	M	Trojesen & Sandnes 2004.	<i>Pongo pygmaeus</i>	Serum	M	Coe <i>et al.</i> 1992.
<i>Homo sapiens</i>	Serum	M	von Eckardstein <i>et al.</i> 2001.	<i>Hylobates lar</i>	Serum	M	Coe <i>et al.</i> 1992.
<i>Homo sapiens</i>	Serum	M	Krithivas <i>et al.</i> 1999.	<i>Hylobates lar</i>	Faecal	M	Rafacz <i>et al.</i> 2009.
<i>Homo sapiens</i>	Serum	M	Coe <i>at al.</i> 1992.	<i>Symphalagus syndactylus</i>	Serum	M	Coe <i>et al.</i> 1992.
<i>Homo sapiens</i>	Serum	F	Weiss <i>et al.</i> 1983.	<i>Symphalagus syndactylus</i>	Faecal	M	Rafacz <i>et al.</i> 2009.
<i>Homo sapiens</i>	Serum	F	Trojesen & Sandnes 2004.	<i>Cercopithecus aethiops</i>	Serum	M	Steklis <i>et al.</i> 1985.
<i>Homo sapiens</i>	Serum	F	Trojesen & Sandnes 2004.	<i>Cercopithecus aethiops</i>	Serum	M	Whitten & Turner 2004.
<i>Homo sapiens</i>	Salivary	M	McIntyre <i>et al.</i> 2003.	<i>Cercopithecus aethiops</i>	Serum	M	Coe <i>et al.</i> 1992.
<i>Homo sapiens</i>	Salivary	M	Roney <i>et al.</i> 2003.	<i>Macaca mulatta</i>	Serum	F	Turner <i>et al.</i> 1989.
<i>Homo sapiens</i>	Salivary	M	Kempel <i>et al.</i> 2005.	<i>Macaca mulatta</i>	Serum	F	Perachio <i>et al.</i> 1977.
<i>Homo sapiens</i>	Salivary	F	Roney <i>et al.</i> 2003.	<i>Macaca mulatta</i>	Serum	F	Mello <i>et al.</i> 2004.
<i>Homo sapiens</i>	Salivary	F	Kempel <i>et al.</i> 2005.	<i>Macaca mulatta</i>	Serum	F	Wilson <i>et al.</i> 1982.
<i>Pan paniscus</i>	Serum	M	Coe <i>et al.</i> 1994.	<i>Macaca mulatta</i>	Serum	M	Herndon <i>et al.</i> 1981.
<i>Pan troglodytes</i>	Serum	F	Machataschke <i>et al.</i> 2006.	<i>Macaca mulatta</i>	Serum	M	Phoenix <i>et al.</i> 1977.
<i>Pan troglodytes</i>	Serum	M	Kutsukake <i>et al.</i> 2009.	<i>Macaca mulatta</i>	Serum	M	Goodman <i>et al.</i> 1974.
<i>Pan troglodytes</i>	Serum	M	Martin <i>et al.</i> 1977.	<i>Macaca mulatta</i>	Serum	M	Gordon <i>et al.</i> 1976.
<i>Pan troglodytes</i>	Serum	M	Coe <i>et al.</i> 1992.	<i>Macaca mulatta</i>	Serum	M	Perachio <i>et al.</i> 1977.
<i>Pan troglodytes</i>	Faecal	M	Muehlenbein <i>et al.</i> 2004.	<i>Macaca mulatta</i>	Serum	M	Robinson <i>et al.</i> 1975.
<i>Pan troglodytes</i>	Faecal	M	Seraphin <i>et al.</i> 2008.	<i>Macaca mulatta</i>	Serum	M	Rose & Bernstein 1972.

Appendix 6.1: Source references for testosterone values continued. F = females; M = Males.

Species	T	Sex	Reference	Species	T	Sex	Reference
<i>Macaca mulatta</i>	Serum	M	Rose <i>et al.</i> 1972.	<i>Macaca fuscata</i>	Serum	M	Eaton & Resko 1974.
<i>Macaca mulatta</i>	Serum	M	Lacreuse <i>et al.</i> 2009.	<i>Macaca fuscata</i>	Serum	M	Matsubayashi & Enomoto 1983.
<i>Macaca mulatta</i>	Serum	M	Mello <i>et al.</i> 2004.	<i>Macaca fuscata</i>	Serum	M	Barrett <i>et al.</i> 2002.
<i>Macaca mulatta</i>	Serum	M	Poblano <i>et al.</i> 2004.	<i>Macaca fuscata</i>	Serum	M	Coe <i>et al.</i> 1993.
<i>Macaca mulatta</i>	Serum	M	Muehlenbein <i>et al.</i> 2002.	<i>Macaca fuscata</i>	Faecal	M	Barrett <i>et al.</i> 2002.
<i>Macaca mulatta</i>	Serum	M	Phoenix 1980.	<i>Macaca fuscata</i>	Faecal	M	Muroyama <i>et al.</i> 2007.
<i>Macaca mulatta</i>	Serum	M	Arslan <i>et al.</i> 1984.	<i>Macaca fuscata</i>	Faecal	M	Barrett <i>et al.</i> 2002.
<i>Macaca mulatta</i>	Serum	M	Coe <i>et al.</i> 1992	<i>Papio hamadryas</i>	Serum	F	Longcope <i>et al.</i> 1988.
<i>Macaca mulatta</i>	Salivary	M	Arslan <i>et al.</i> 1984.	<i>Papio hamadryas</i>	Serum	M	Taranov & Goncharov 1985.
<i>Macaca fascicularis</i>	Serum	M	Coe <i>et al.</i> 1993	<i>Papio hamadryas</i>	Serum	M	Coe <i>et al.</i> 1992.
<i>Macaca fascicularis</i>	Serum	M	Czoty <i>et al.</i> 2008.	<i>Papio hamadryas</i>	Faecal	M	Bergman <i>et al.</i> 2006.
<i>Macaca fascicularis</i>	Serum	M	Dang & Meusy-Dessolle 1981.	<i>Papio hamadryas</i>	Faecal	F	Beehner <i>et al.</i> 2005.
<i>Macaca fascicularis</i>	Serum	M	Tan & Kwan 1987.	<i>Mandrillus sphinx</i>	Serum	M	Wickings & Dixon 1992.
<i>Macaca fascicularis</i>	Serum	M	Fouquet <i>et al.</i> 1984.	<i>Mandrillus sphinx</i>	Serum	M	Dixson & Anderson, 2004
<i>Macaca fascicularis</i>	Serum	M	Zumpe & Michael 1987.	<i>Ateles geoffroyi</i>	Serum	M	Cerda-Molina <i>et al.</i> 2009.
<i>Macaca fascicularis</i>	Serum	M	Michael <i>et al.</i> 1987.	<i>Ateles geoffroyi</i>	Serum	M	Coe <i>et al.</i> 1992.
<i>Macaca fascicularis</i>	Serum	M	Arslan <i>et al.</i> 1984.	<i>Alouatta caraya</i>	Faecal	M	Moreland <i>et al.</i> 2001.
<i>Macaca fascicularis</i>	Salivary	M	Arslan <i>et al.</i> 1984.	<i>Alouatta caraya</i>	Faecal	F	Moreland <i>et al.</i> 2001.
<i>Macaca arcoides</i>	Serum	M	Koos-Slob <i>et al.</i> 1979.	<i>Callithrix jacchus</i>	Serum	M	Baker <i>et al.</i> 1999.
<i>Macaca arcoides</i>	Serum	M	Goldfoot <i>et al.</i> 1975.	<i>Callithrix jacchus</i>	Serum	M	Coe <i>et al.</i> 1992.
<i>Macaca arcoides</i>	Serum	M	Nieuwenhuijen <i>et al.</i> 1987.				
<i>Macaca arcoides</i>	Serum	M	Coe <i>et al.</i> 1993				

Appendix 6.1: Source references for testosterone values continued. F = females; M = Males.

Species	T	Sex	Reference
<i>Saimiri sceurius</i>	Serum	M	Winslow & Insel 1991.
<i>Saimiri sceurius</i>	Serum	M	Schiml <i>et al.</i> 1996.
<i>Saimiri sceurius</i>	Serum	M	Winslow <i>et al.</i> 1988.
<i>Saimiri sceurius</i>	Serum	M	McCamant <i>et al.</i> 1987.
<i>Siamiri scureus</i>	Serum	M	Pasqualini <i>et al.</i> 1986.
<i>Siamiri scureus</i>	Serum	M	Coe <i>et al.</i> 1992.
<i>Saguinus oedipus</i>	Faecal	M	Konecki <i>et al.</i> 2007.
<i>Saguinus oedipus</i>	Faecal	F	Konecki <i>et al.</i> 2007.
<i>Cebus apella</i>	Serum	M	Coe <i>et al.</i> 1994.

Appendix 6.2: Source references for androgen receptor CAGn values. F = females; M = Males.

Species	N	Sex	CAG	References
<i>Homo sapiens</i>	175	M	20	von Eckardstein <i>et al.</i> 2001.
<i>Homo sapiens</i>	205	M	23	Campbell <i>et al.</i> 2007.
<i>Homo sapiens</i>	638	M & F	21	Medland <i>et al.</i> 2005.
<i>Homo sapiens</i>	133	M	18	Rajender <i>et al.</i> 2007.
<i>Homo sapiens</i>	241	M	18	Rajender <i>et al.</i> 2007.
<i>Homo sapiens</i>	271	M	21	Rajender <i>et al.</i> 2007.
<i>Homo sapiens</i>	882	M	22	Krithivas <i>et al.</i> 1999.
<i>Homo sapiens</i>	85	M & F	23	Hong <i>et al.</i> 2006.
<i>Pan troglodytes</i>	89	M & F	20	Hong <i>et al.</i> 2006.
<i>Pan troglodytes</i>	3	F	21	Choong <i>et al.</i> 1998.
<i>Gorilla gorilla</i>	28	M & F	8	Hong <i>et al.</i> 2006.
<i>Pongo pygmaeus</i>	30	M & F	12	Hong <i>et al.</i> 2006.
<i>Hylobates agilis</i>	26	M & F	4	Hong <i>et al.</i> 2006.
<i>Symphalangus syndactylus</i>	25	M & F	4	Hong <i>et al.</i> 2006.
<i>Papio hamadryas</i>	6	M	9	Choong <i>et al.</i> 1998.
<i>Macaca fascicularis</i>	6	M & F	8	Choong <i>et al.</i> 1998.
<i>Lemur sp.</i>	5	M	4	Choong <i>et al.</i> 1998.

Appendix 6.3: Correlations between testosterone levels, 2D:4D, body weight and substrate.
 *= with body weight controlled for $F_{1,4} = 2.42$, $p = 0.19$, $\lambda = 1$.

		Non-Phylogenetic (GLM)				Phylogenetic (PGLS)			
		F	p	R ²	sd	F	p	sd	λ
Male serum T	2D:4D	0.01	0.93	0.01	10	0.02	0.88	10	0
	Weight	2.02	0.19	0.17	10	1.68	0.22	10	0
	Substrate	2.29	0.16	0.19	10	1.09	0.38	9	0
Female serum T	2D:4D	1.40	0.36	0.41	2	0.66	0.5	2	0
	Weight	0.99	0.43	0.33	2	0.49	0.55	2	0
	Substrate	1.96	0.30	0.50	2	1.28	0.38	2	1
Mean serum T	2D:4D	0.01	0.98	0.00	10	0.01	0.97	10	0
	Weight	2.21	0.17	0.18	10	1.85	0.2	10	0
	Substrate	3.04	0.11	0.23	10	1.26	0.33	9	0
Male faecal T	2D:4D	8.88	0.03	0.64	5	9.69	0.02*	5	0.63
	Weight	1.46	0.28	0.23	5	9.49	0.03	5	1
	Substrate	2.37	0.19	0.32	5	1.55	0.32	4	0.87
Mean faecal T	2D:4D	12.95	0.02	0.72	5	14.92	0.01	5	0.8
	Weight	1.25	0.32	0.20	5	6.04	0.06	5	1
	Substrate	2.48	0.18	0.33	5	1.27	0.37	4	0.67