PSYCHOLOGY MISCELLANY

No.35 - May 2012

Kevin Brewer

ISSN: 1754-2200

Orsett Psychological Services
PO Box 179
Grays
Essex
RM16 3EW
UK

orsettpsychologicalservices@phonecoop.coop

This document is produced under two principles:

1. All work is sourced to the original authors. The images are all available in the public domain (most from http://commons.wikimedia.org/wiki/Main_Page). You are free to use this document, but, please, quote the source (Kevin Brewer 2012) and do not claim it as you own work.

2. Details of the author are included so that the level of expertise of the writer can be assessed. This compares to documents which are not named and it is not possible to tell if the writer has any knowledge about their subject.

Kevin Brewer BSocSc, MSc

An independent academic psychologist, based in England, who has written extensively on different areas of psychology with an emphasis on the critical stance towards traditional ideas.

A complete listing of his writings at http://kmbpsychology.jottit.com.

CONTENTS

1. UNDERSTANDING THE ISSUES AND CHANGING KNOWLEDGE ABOUT THE EVOLUTION OF HUMANS

1.1. INTRODUCTION

The traditional view has been of the evolution of apes to humans as a relay race of species of hominids where each one developed something new. It is now accepted that many different species existed at the same time.

Furthermore, for many years the view was held that Homo neanderthalensis (Neanderthals) evolved into Homo sapiens, whereas now it is accepted that they were a separate species that existed at the same time as humans (at least for a while). There was an overlap of about 15 000 years between humans and Neanderthals existing in Europe, for example (Wong 2009) (figure 1.1).

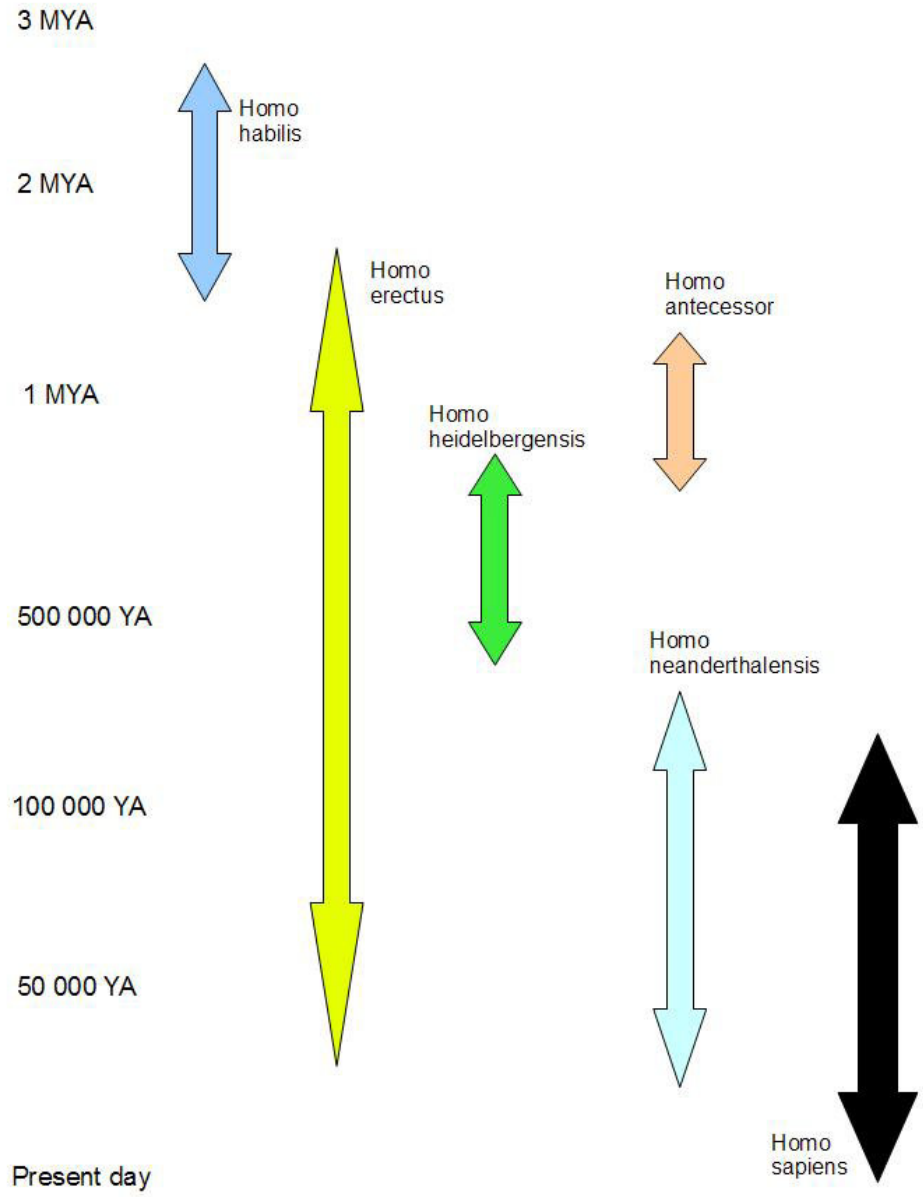
"The classic view of human evolution doesn't emphasis adaptability. It focuses more on the idea that we were inevitable: that famous march from ape to human. It's a ladder of progress with simple organisms at the bottom and humans at the top. This idea of inevitability runs deep in our societal assumptions, probably because it's comforting - a picture of a single, forward trajectory, ending in modern humans as the crown of creation" (Potts quoted in Neimark 2011 p57).

In fact, 90 000 - 70 000 years ago (YA) there were few Homo sapiens and the species almost died out [1] [2]. But why did we survive? One recent answer is the ability to adapt to change ("variability selection"; Potts 1998).

Marean (2010) sees the ability to adapt as humans moving from the grasslands of east Africa to places like the coast of southern Africa (Cape Floral Region), where

[1] This accounts for the low genetic diversity of humans today.

[2] Marean (2010) dated the "bottleneck" as between 195 000 and 123 000 YA in the last Ice Age ("Marine Isotope Stage 6"; MIS6).



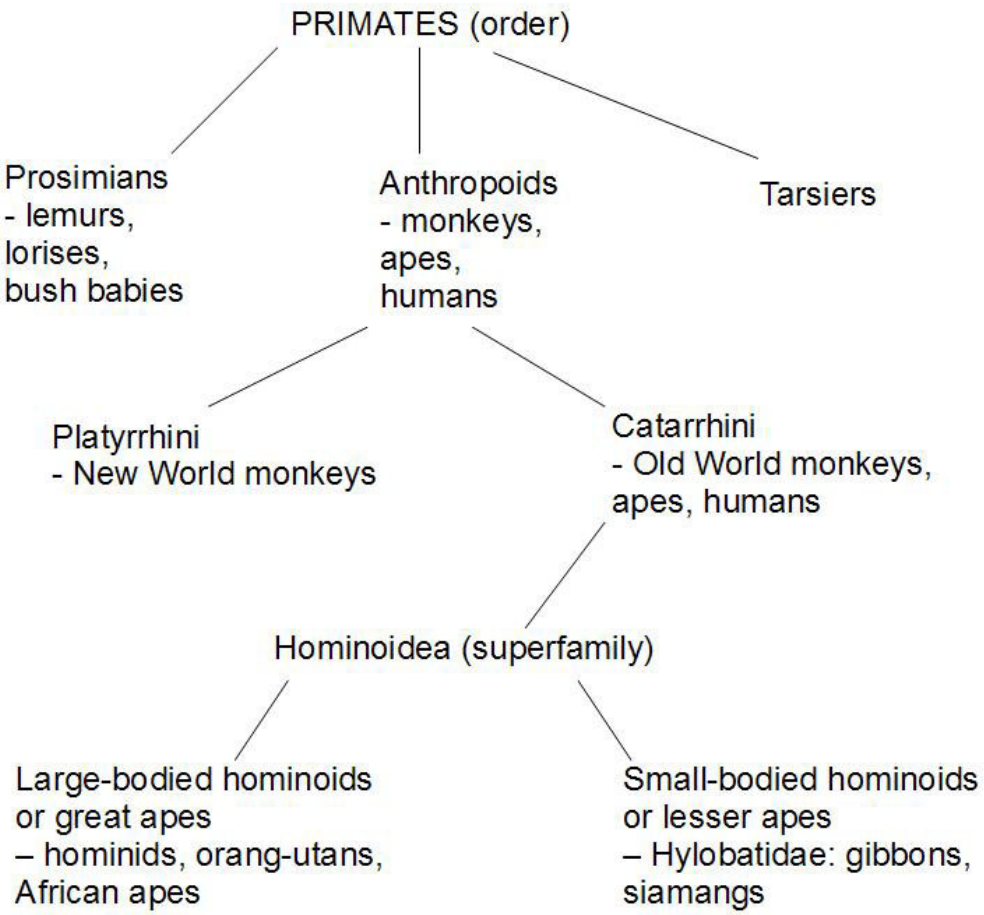
(Based on Wong and Deak 2009)

Figure 1.1 - Approximate dates of selected hominids.

caloric-dense, nutrient-rich protein was available from shellfish and carbohydrates from geophytes (eg: tubers).

1.2. FROM APES TO HUMANS

Human beings are members of the order Primate along with monkeys and apes (figure 1.2).



(Source: Grehan and Schwartz 2009)

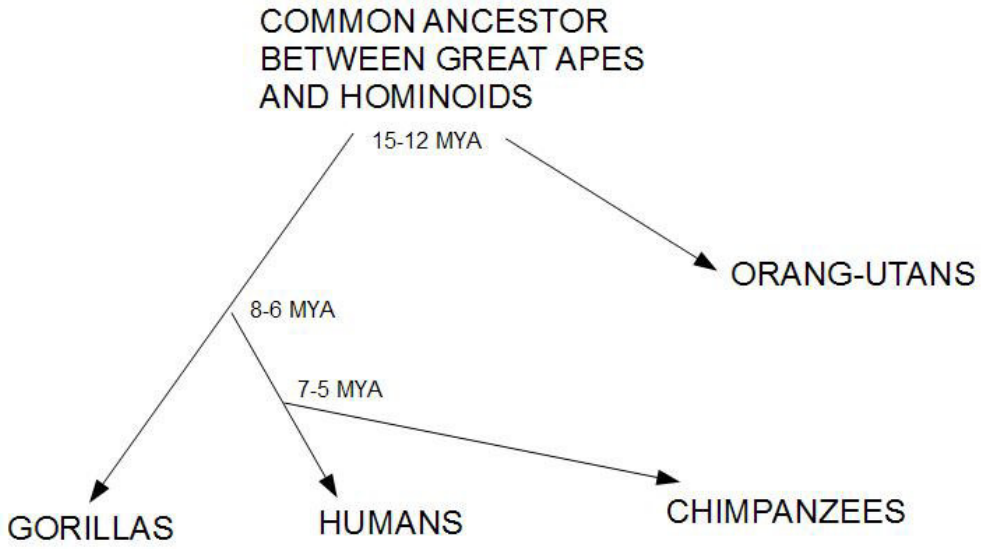
Figure 1.2 - Humans place in order Primate.

The traditional view is that the human's nearest relative among the apes is the chimpanzee (figure 1.3).

However, Grehan and Schwartz (2009) argued that it is the orang-utan. They present a different evolutionary path (figure 1.4) based upon two challenges to traditional evidence.

i) DNA - Humans share 98.4% of their DNA with chimpanzees and 96.5% with orang-utans. Grehan and Schwartz (2009) argued similarity in DNA is less important because orang-utans evolved rapidly after splitting from the common ancestor of humans and orang-utans.

ii) Anatomy - Grehan and Schwartz (2009) placed Homo sapiens, orang-utans and australopithecines as a "clade" (and chimpanzees and gorillas as another - "African apes" clade) because of the anatomical similarities like thick



(Based on Figure 2.1 p19 Robson et al 2006)

(MYA = million years ago)

Figure 1.3 - General pattern of evolution of humans from great apes [3].

tooth enamel, long hair, male facial hair, and concealed ovulation.

Schwartz has been arguing that orang-utans are the nearest relative to humans for many years (eg: Schwartz 1984). Most of the scientific community has rejected the idea because the evidence is overwhelming in favour of chimpanzees. When is a different idea to the accepted one a challenge that spurs science to learn more or is complete nonsense? There have been many ideas held as accepted by science that were shown later to be wrong.

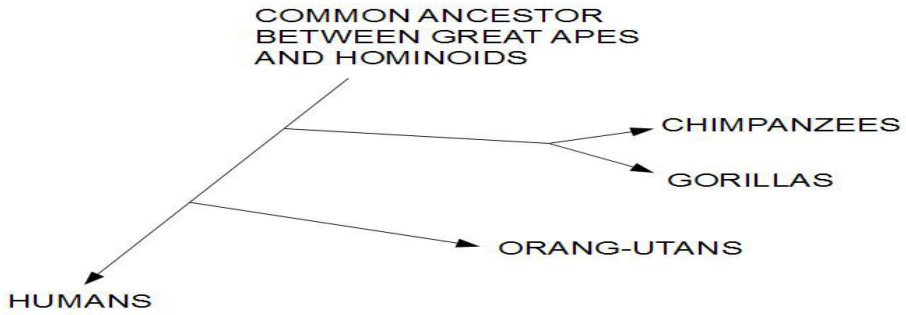
Humans differ from the great apes in other key ways:

a) Lifespan - Human foragers without modern medical support have a maximum lifespan closer to 80 years compared to 50 for chimpanzees and gorillas (Robson et al 2006) [4].

b) Age at first birth - Among human foragers it is

[3] Hominoids tends to be used to cover apes and hominids to mean great apes, though the latter term is often used to cover and humans and relative species like Homo habilus.

[4] Estimates for hominids include Australopithecus afarensis 45 years and Homo erectus 60 years (Gibbons 2008). This information comes from counting the microscopic lines on the surface of teeth that are laid down weekly.



(Source: Lawton 2009)

Figure 1.4 - Evolution of great apes and humans according to Grehan and Schwartz (2009).

about 20 years old which is later than the 10-15 years old in the other great apes (Robson et al 2006) [5].

Delayed reproduction (and long childhood or slow growth) allows for larger and stronger mothers who can have many children, but it is a risky strategy in evolutionary terms as the women has longer to survive before reproduction. This strategy occurred somewhere between Homo antecessor (800 000 YA) and the first Homo sapiens (200 000 YA) (Gibbons 2008).

c) Gestation period - This is longer in humans (average 270 days compared to less than 260 days in the great apes) (Robson et al 2006). This longer gestation means that human babies are heavier at birth.

d) Length of life after last birth - Humans live longer after the potential last birth (ie: menopause).

e) "Stacking" - This refers to the unique characteristic of human mothers who bear another baby before the previous one can feed themselves (ie: "nutritional independence" not until 6-7 years old; Gibbons 2008 [6]). This is only possible if mothers are given help by fathers, and post-menopausal and adolescent females. The presence of the grandmother being key to the welfare of the grandchildren (Robson et al 2006) [7]. Though great ape mothers with dependent infants may be accompanied by older offspring (weaned sub-adults or juveniles), the latter feed themselves.

---

[5] Estimated at 11.5 years for Australopithecus afarensis and 14.5 years for Homo erectus (Gibbons 2008).

[6] Borrell (2010) reported that anthropologists feel that indigenous individuals living in the Amazon region cannot survive independently until 18 years old.

[7] Hawkes et al (1998) proposed this idea as part of the explanation for the evolution of grandmothering.

1.3. TO MODERN HUMANS

The idea that there was a simple linear progression from the common ancestor of chimpanzees and modern humans has been discredited over time with the increasing number and variety of fossil finds [8] [9]. When Charles Darwin was writing in the mid-nineteenth century, "fossil support for human evolution was almost absent" (Klein 2009). Two key historical findings came after Darwin's death - Eugene Dubois (1892) first specimen of Homo erectus, and Raymond Dart (1925) first specimen of an australopithecine (Klein 2009) [10].

The date since the last common ancestor between apes (gorillas and chimpanzees) and humans has increased with newer finds. For most of the twentieth century it was assumed to about one million years ago, then 2 MYA in the 1960s, 4.4 MYA, and currently 7-6 MYA (Klein 2009).

Australopithecines are distinguished from chimpanzees by their bipedalism, among other differences, 4.5 - 2 MYA (Klein 2009). There are debates about the different species of this time period (appendix 1A).

There are many species of australopithecines [11], and the genus, Homo, evolved from one of them 2.5 MYA (table 1.1). Stone tool "technology" is often used to aid this distinction (though there are disagreements among scholars) (Klein 2009) (figure 1.5) [12].

Around 1.7 MYA Homo habilis (earliest species of Homo) evolved into Homo ergaster or Homo erectus (figure 1.6)(with the former leaving Africa 2 - 1.6 MYA). This movement "Out-of-Africa" led to three evolving human lineages by 500 000 - 400 000 YA: Homo sapiens in Africa [13], Homo neanderthalensis (Neanderthals) in Europe, and

---

[8] It must be remembered that there are a limited number of fossils from which conclusions are being drawn. For example, knowledge of Homo erectus mostly comes from a single skeleton of a boy who died near Lake Turkana, Kenya approximately 1.6 MYA ("Turkana boy") (Gibbons 2008).

[9] However, increasing knowledge has not answered all the questions and ended all the debates, and in fact, it has opened new controversies. Some of the main issues include (Wong and Deak 2009):

- Sahelanthropus tchandensis (7 MYA) - bipedal or not.
- Australopithecus (ancestor to Paranthropus and Homo genuses) - social structure similar to humans, chimpanzees, or gorillas?
- Homo habilis - include in Homo genus or Australopithecus?
- Homo ergaster - why did they leave Africa in the first out-of-Africa?

[10] The first Neanderthal fossil was found in 1856 (Wong 2009).

[11] "The relationships of australopiths to one another are very uncertain..., and some australopiths appear to be more closely related to Homo than others..." (Grehan and Schwartz 2009 p1827).

[12] Stone tools have been found with australopithecines which challenges the idea that tools were unique to the Homo genus (Barras 2012).

[13] "Becoming human" involved two separate stages - the distinctive morphology (ie: physical body) around 200 000 YA (anatomical human), and the appearance of symbolic thought around 100 000 YA (cognitive human). Symbolic thought was present when humans appeared, but took time to be exploited in the same way that birds had feathers for millions of years before using them for flight (Tattersall

Homo erectus in eastern Asia (Klein 2009) [14] [15] [16].

Recently, a fourth possible lineage of Homo floresiensis (nicknamed the "hobbit") was discovered on the island of Flores, Indonesia (Morwood et al 2004), but this is disputed (Culotta 2006).

Modern research techniques have put forward other species as well including the Denisovans in Siberia (Robson 2012d). Genetic analysis of bones found in 2008 in Denisova Cave in the Altai Mountains in southern Siberia (which had been occupied as early as 280 000 YA) shares little with Neaderthals and humans suggesting a separate species (though a common ancestor with Neanderthals) (Reich et al 2010).

After recent analysis of fossils from Longlin Cave (Guangxi Province), China, Curnoe et al (2012) reported "an unusual mixture of modern human traits" (both common to later Homo but also some unusual features). The researchers hesitated to call it a separate species at this time.

About 50 000 YA Homo sapiens moved "Out-of-Africa" (or "Out-of-Africa 2"; Klein 2009). This stage of evolution is documented by molecular genetics (Cann et al 1987) as well as by fossils [17]. DNA family trees have been produced using the genes of living humans (eg: Li et al 2008).

It is now accepted that different species existed at the same time, like Homo sapiens and Neanderthals, rather than one after another, but the amount of contact and/or interbreeding is contested (Klein 2009) [18] [19].

2009).

[14] Some researchers have suggested that Homo erectus was "neither chimp-like nor human-like but perhaps somewhere in between"; it fits the Homo genus and australopithecines in different ways (Gibbons 2008).

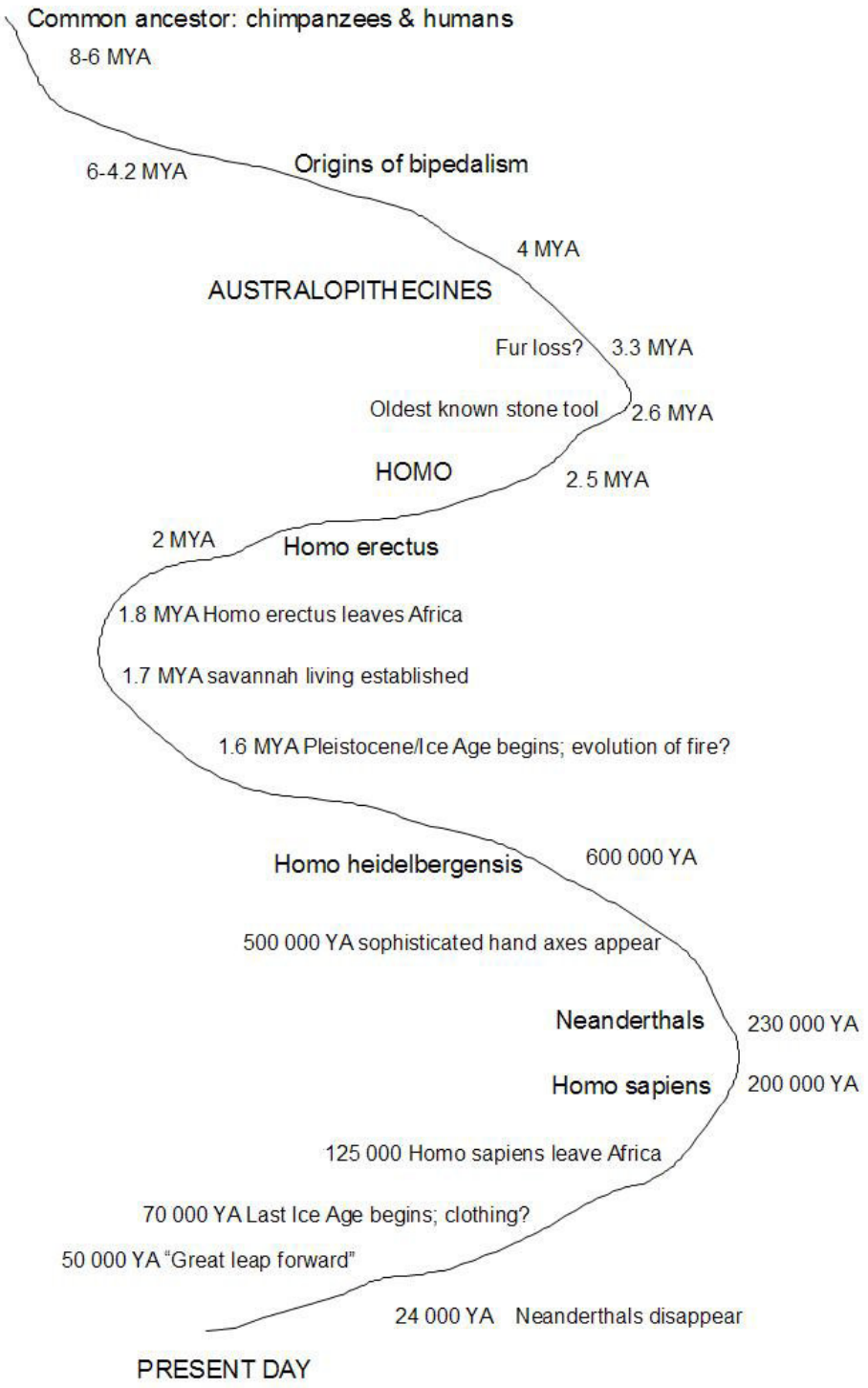
[15] General DNA analysis has revealed common ancestors for Neanderthals and humans at 800 000 YA, and a split at between 440 000 and 270 000 YA. While mitochondrial DNA show a common ancestor at 500 000 YA (Reich et al 2010). There are debates over the fossil evidence as to when and how a split occurred (appendix 1B).

[16] Differences between Neanderthals and humans include brain shape, developmental aspects, and obstetric features (Hublin 2009).

[17] Mitochondrial DNA (mtDNA) is used to show inheritance lines because it is inherited maternally and does not recombine (as with other DNA). mtDNA is 16 500 bases long of which 500 bases long ("control region") does not code for anything. Mutations of the latter occur at regular and predictable times. For example, an average of 10 000 years per mutation; thus three mutations shared by individuals means a common ancestor 30 000 YA (Cocker 2005).

[18] The Rift Valley in East Africa is traditionally viewed as the "birthplace" of humans as most fossils have been found there. But recent finds have been made in South Africa suggesting that "parallel" evolution of humans occurred around the continent (Brahic 2011).

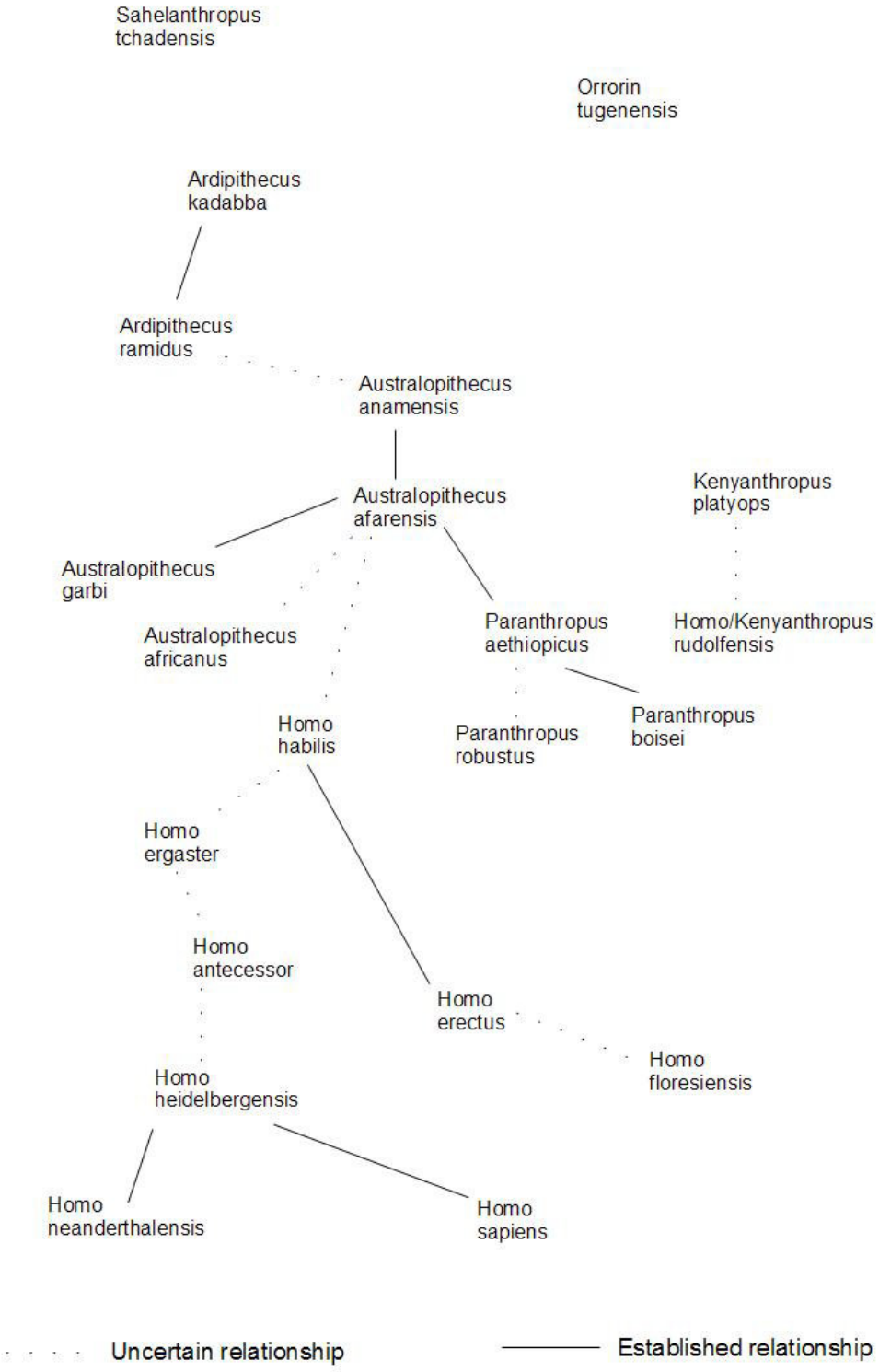
[19] This is called the "multi-regional evolution theory of modern human origins"; eg: humans mated with Neanderthals in Eurasia and Homo erectus in eastern Asia (Wong 2011).



(MYA = million years ago; YA = years ago)

(Based on information in Editor 2012)

Figure 1.5 - Approximate time line of human evolution.



(Not to scale in relation to time)

(Based on Wong and Deak 2009)

Figure 1.6 - Key species in the evolution of humans.

The traditional view (though not clearly established in the fossils) is that Australopithecus afarensis gave rise to Homo habilis (ie: the move from short legs and "tree-climbing hands" to longer legs and "toolmaking hands") (Wong 2012). But a large fossil find in modern day northern Republic of South Africa (Malapa area) has led to a revision. This new species, Australopithecus sediba [20], is proposed as the direct ancestor of Homo, but of Homo erectus (not Homo habilis) (Berger et al 2010) [21].

These finds suggest that the move from Australopithecine to Homo was not an all in one, but a mixture of differences. For example, Australopithecus sediba had "tree-climbing hands" with long thumbs (a characteristic of "tool-making hands") (Wong 2012).

Table 1.1 - From Australopithecus to Homo.

The belief that considerable biological changes occurred to humans as they populated the world after leaving Africa has been challenged by recent genome studies. The speed of beneficial genetic mutations through a population appears to have been relatively rare in the past 60 000 years (Pritchard 2010) [22]. Saying that, it is estimated that 7% of human genes have evolved in the last 5000 years (Ward 2009).

One example of evolution established from genome studies relates to the ability to digest carbohydrate lactose (sugar in milk) [23]. In the past humans were not able as adults to digest it (and thus not drink fresh milk [24]), but with the development of diary farming this changed (Pritchard 2010).

1.4. ISSUES AND DEBATES

1. Bipedalism/walking upright on two legs.

One of the key differences between humans and the great apes is locomotion. They are "terrestrial knuckle walkers" while we are bipedal with the physiological adaptations (eg: tibia in lower leg held upright to foot versus angled in apes; shoulders pulled back; legs lengthened; pelvis adapted; Douglas 2012a) [25].

There is fossil evidence that Australopithecines (4.4 MYA) walked habitually (Bramble and Lieberman 2004).

---

[20] From the ancestor Australopithecus africanus (Wong 2012).

[21] As always not all scholars agree (Wong 2012).

[22] In theory, a beneficial mutation could take only a few hundred years to become the norm in a population, but more likely thousand of years. For example, if a mutation allows the carrier to have 10% more children than non-carriers, this mutation will increase in frequency in the population from 1% to 99% in two hundred generations (approximately 5000 years) (Pritchard 2010).

[23] LCT (gene for lactose) (Pollard 2009).

[24] Babies are able to tolerate lactose, but this is turned off by the genes after weaning (Pritchard 2010).

[25] Bramble and Lieberman (2004) argued that the human body evolved for endurance running 2 MYA.

Charles Darwin believed that hominoids became bipedal to free their hands for tool-making and use. But bipedalism came before tools (1-2 million years before) (Douglas 2012a).

However, it does leave the hands free to carry things, and aid in the access to more food as well as making the spotting of predators on the savannah easier (Douglas 2012a).

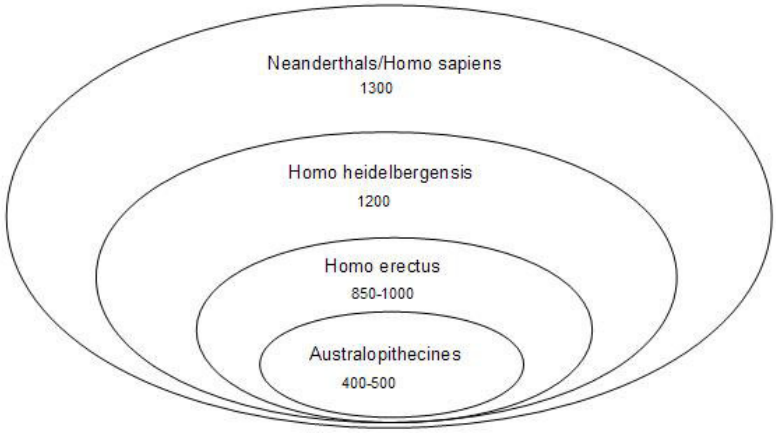
Schultz (2007) reported the idea that it is better support for carrying a child, particularly heavier children. Primate infants tend to cling to the mother's fur which is limited by the weight of the baby and the strength of the fur.

Wheeler (1984) argued that walking upright allowed air to circulate more effectively around the body and helped its cooling in hot climates (along with the fur loss and evolution of sweating).

2. Growth of brain size.

The different hominids varied in brain volume size with it increasing to Homo sapiens (figure 1.7). There was a three-fold increase in absolute brain size and in encephalisation quotient (EQ) (ratio of brain volume to body mass) between Homo habilis and Homo sapiens (Bailey and Geary 2009).

The australopithecine's brain was larger than the chimpanzee's (as they are today) on both measures. There is a further expansion overall to Homo habilis, but also increases in areas like the frontal and parietal lobes (Bailey and Geary 2009).



(Source of information: Editor 2012)

Figure 1.7 - Representation of brain volume size (cm^2).

In terms of physiology, a single gene mutation of a

jaw muscle that constrains the skull's growth occurred around 2.4 MYA (Stedman et al 2004). Meat and fish eating appeared 2 MYA also, and these would sustain brain expansion (Braun et al 2010), as well as the evolution of cooking later (Robson 2012b) [26].

Three main explanations are put forward for the increase in brain size (Bailey and Geary 2009):

a) Climate variation - Brain volume is larger the further away from the equator (where there is greater seasonal variation in temperature).

b) Ecological models - The demands of complex foraging and hunting including tool use drove brain evolution.
The environment was also important in that it was tectonically active (Tectonic Landscape Model; eg: Reynolds et al 2011). Such complex landscapes are to the benefit of intelligent and adaptable species over fast or strong ones (Marshall 2010).

c) Social competition - Living in social groups and the need to manage information about other individuals (eg: who can be trusted) was the motivation for brain evolution.

Bailey and Geary (2009) argued that multiple pressures drove the evolution of brain size with social competition being the most important. The researchers collated data on latitude/climate, and population density (number of fossils found in an area) for 175 hominid skulls. Large population density predicted large skull finds generally, but at a high population density the skull was smaller.

This general trend of increasing brain size has been reversed in the last 20 000 years with a small decline in average volume (1500 cm^3 to 1350 cm^3) (McAuliffe 2011). The reasons for this decline might include warmer climate, advent of agriculture, large communities, food shortages, domestication, or co-operation instead of aggression (McAuliffe 2011). This is not necessarily a sign of declining intelligence because the wiring of the brain has become more efficient. On the other hand, there is evidence that brain volume has started to increase in the last two or three centuries (McAuliffe 2011).

[26] Cooking may have been present as early as Homo erectus (Wrangham 2009).

3. Technological development.

Much of the understanding about the different species of hominids comes from the increasing sophistication of stone tools found.

Though the first stone tools found date back to 2.5 MYA, there is a gap of one million years before the tool is adapted (Douglas 2012b). However, a lot of cognitive evolution was occurring during that period (eg: cognitive complexity that underpins later language development) as well as new perceptual-motor abilities (in manipulating the tools) (Faisal et al 2010).

During this one million years other tools may have been made from materials that have perished (as only stone tools are left for fossil hunters). On the other hand, life may have been so difficult that experimentation with new ideas/things was just too risky (Douglas 2012b).

4. Evolution of language.

Dunbar (2004) placed the evolution of language between 1.6 million and 600 000 YA.

Evidence for the evolution of language is concluded from anatomical evidence. For example, Homo heidelbergensis do not have the balloon-like organ connected to the voice box that was used to produce booming noises, but stopped language development (Robson 2012a). Other changes include neural connections to the tongue, diaphragm and chest muscles, and the presence of the FOXP2 gene (Dominguez and Rakic 2009).

But the appearance of language did not automatically mean that complex ideas accompanied it. Language could have evolved to sing around the camp fire at night and encourage group bonding (Dunbar 2004). Furthermore, hand gestures make have evolved before speech (Robson 2012a).

5. Loss of fur.

Hair covering the body is unique to mammals (Jablonski 2010), and it serves a number of functions from insulation to camouflage to use in visual communication (eg: hair raised as sign of aggression).

The loss of fur has been possibly dated to 3.3 MYA (ie: before the Homo genus) (based on studies of the co-evolution of lice) (Douglas 2012c).

Can we learn about fur loss from mammals that do not have fur?

a) Living underground as with the hairless naked mole rat - not relevant to humans.

b) Living in water like marine mammals - the "aquatic ape theory" (appendix 1C) argues that human evolution included a semi-aquatic phase (of 1-2 million years) living in marshes and on the coast which led to fur loss.

c) Very large body size and risk of overheating - though humans do not have less surface area relative to overall body mass which makes it difficult to lose heat as with elephants, for example, overheating is important in the evolution of fur loss, particularly to cope with the heat on the savannah (Ruxton and Wilkinson 2011) [27].

Key for humans is sweating to reduce heat, which is more effective without a fur covering. Furthermore, humans have more eccrine glands in the skin that produce sweat, whereas other mammals that sweat have more sebaceous and apocrine glands. These glands located near the base of hair follicles produce an oily sweat which is less effective at heat dissipation, whereas eccrine glands produce watery sweat directly through the skin (Jablonski 2010).

As human ancestors moved to the savannah from wooded environments, due to climate change beginning 3 MYA, getting food (which was now probably meat) required more activity in a hot environment. In other words, running after moving targets in the sun compared to foraging for fruits in the shady woodlands. This change has led to the suggestion that Homo ergaster (1.6 MYA) was the first to be furless (Jablonski 2010). This information has been ascertained indirectly from fossils through changes in the body that appear to show greater movement (eg: ankle, knee and hip joints).

In relation to sexual selection, fur harbours parasites [28] whereas unblemished skin advertises good health to potential mates.

From gene studies, Rogers et al (2004) calculated that a variant of the MC1R gene (responsible for skin pigmentation) appeared 1.2 MYA. This variant produced dark pigmentation (ie: the basis of black skin seen today). Human ancestors were assumed to have pink skin covered by black fur (Jablonski 2010).

After fur loss, the next question relates to the adoption of clothing by hominids. Based on analysis of the genes of body lice (which live in clothing [29]),

---

[27] But heat loss at night or in cold temperatures would be greater (Pagel and Bodmer 2003).

[28] Pagel and Bodmer (2003) suggested that fur loss occurred to remove lice and fur-dwelling ectoparasites that carry disease.

[29] Body lice evolved from head lice which were present earlier (Kittler et al 2003).

Kittler et al (2003) calculated that they evolved around 70 000 YA (110 000 - 30 000 YA) suggesting that this was when clothing became common. This would go with the movement of humans to colder climates out of Africa.

6. Out-of-Africa.

Homo sapiens leaving Africa 125 000 YA is a major event in terms of the population of the world leading to today [30]. A number of reasons have been proposed for the migration.

One is overcrowding in the Horn of Africa (Atkinson et al 2009) based on analysis on modern day mitochondrial lineages [31]. Humans currently alive originate from 4 lineages called L0, L1, L2 and L3 (corresponding to "ancestral mothers"; Douglas 2012d). Only the L3 group was outside Africa suggesting that they experienced the population growth that prompted migration.

This group are more likely to have the DRD4-7R gene which is associated with novelty seeking (Matthews et al 2011).

Mellars (2006) saw the trigger for population growth and migration as increases in the complexity of technological (eg: tools), economic (exchange of goods), social (size of communities), and cognitive behaviours between 80 000 and 60 000 YA. This has been called the "great leap forward".

7. Homo sapiens interbreeding with others.

If there were different species [32] of Homo around at the same time, did they interaction and/of interbreed?

Molecular genetic analysis shows that 1-4% of the genome of current humans of non-African descent (L3 mitochondrial lineage) is from Neanderthals (Green et al 2010), and current Melanesians have 7% from Denisovans

---

[30] "Our understanding of the origins of modern human populations (ie: Homo sapiens) has made massive strides in the past two decades. We now know from studies of both the DNA patterning of present-day world populations and surviving skeletal remains that populations that were essentially 'modern' in both a genetic and an anatomical sense had emerged in Africa by at least 150,000 years ago..." (Mellars 2006 p9381).

[31] Mellars (2006) gives a word of caution: "Demographic reconstructions based on DNA studies of present-day human populations are notoriously problematic and controversial, with the data from African populations being no exception. Debates over the rates of mutation of different genetic loci, the effects of adaptive selection on DNA patterns, and the potential complications of demographic dispersals and back migrations between different regions, all serve to complicate the surviving fingerprints of demographic history in ways that have still to be fully resolved..." (p9381).

[32] A species can be defined as a "group that cannot mate and produce viable offspring with other species" (Jones 2012b). This definition means that Neanderthals could be viewed as a sub-species of Homo sapiens if mating occurred (Jones 2012b).

(Reich et al 2010).

8. Homo sapiens and the fate of Neanderthals.

Neanderthals became extinct 24 000 YA, but did Homo sapiens contribute to that process [33]?

This could include actual killing them at one extreme, to passive extermination from diseases carried by Homo sapiens, or simply outsmarting them and taking the available food. In the latter case, the Neanderthals had more brain capacity for vision (to see in the dark), and less for complex thought which put them at a disadvantage compared to Homo sapiens (Robson 2012 c).

Finlayson (2009) preferred to blame climate change for the extinction of the Neanderthals. Between 65 000 and 25 000 YA the climate varied greatly between ice and mild, and though Neanderthal populations bounced back after each ice period, eventually they were not able to do so (Wong 2009).

It was not just the cold that defeated Neanderthals. In fact, their short and stocky build conserved body heat, and worked well when ambush hunting solitary large animals in the cold forests. In warmer times with open grasslands, they were disadvantaged (particularly compared to humans).

Humans also did better in the competition for food because they ate a variety of animals and plants, while analysis of bone chemistry of Neanderthals suggests that the diet was limited to large animals. Neanderthals are viewed as "one-trick ponies" (Wong 2009) - they had one way of doing things which was great unless circumstances changed.

But some researchers have questioned this view. For example, Stringer et al (2008) found evidence of Neanderthals at Gorham's Cave and Vanguard Cave in Gibraltar eating marine mammals like seals, and shellfish.

In fact, there is a blurring of the line between Neanderthals and humans in a number of ways. For example, archaeological finds in recent times suggest that Neanderthals made jewellery, which could hint at symbolic thought (assumed to be distinct in Homo sapiens). In caves in south-east Spain painted scallops and cockleshells from 50 000 YA have been found (Zilhao et al 2010). These finds were 10 000 years before Homo sapiens are dated in Europe (Choi 2010).

---

[33] An assumption has tended to be that Neanderthals were not very intelligent compared to humans, though this is being challenged.

Differences that advantaged humans to survive and Neanderthals to go extinct are relatively small, including (Wong 2009):

a) Use of needles to sew clothes (and make them better and stronger).

b) Division of labour that meant human males hunted and females remained safe with the children.

c) Longer living humans allow the passing down of knowledge.

d) Less calories need - it has been estimated that Neanderthals needed 100-350 calories more per day (Froehle and Churchill 2009).

9. Evolution of humans today.

In the last few hundred years there have been so many changes for humans that were culture-based (ie: not evolution) including medical advances, which has led to the popular lay belief that humans are no longer affected by natural selection. But "That's just plain false" (Stephen Stearns quoted in Holmes 2009).

Evolutionary changes are long-term and hard to spot compared to the swift cultural and social changes.

Because few individuals die young today (ie: predator risk low), the selection pressures will not be related to survival as in the past (eg: running faster to escape predators), but work upon genes that affect the number of children (ie: fertility or reproductive behaviour) (eg: later age of menopause) (Pritchard 2010).

For example, data from the Framingham Heart Study suggests that shorter, heavier women have more children, and if this trend is extrapolated into the future, by 2409 women will be 2 cm shorter and 1 kg heavier than today (Holmes 2009). The Framingham Heart Study has collected data on the residents of Framingham, Massachusetts, USA, since 1948, and in particular 2238 women who had passed menopause (Byars et al 2010).

To suggest that evolution has stopped for humans assumes that the body is perfect for its environment, but there are many aspects that are far from that including DNA replication making mistakes, vulnerable brain cells (eg: epaulette sharks can survive for one hour without oxygen), or the fact that the vagina and urethra are near the anus increasing the risk of infection (Ainsworth and LePage 2007).

Ward (2009) outlined possible future evolutionary

scenarios:

- Stasis - "minor tweaks".
- Speciation - new human species.
- Symbiosis with machines.
- Extinction.

1.5. WHAT MAKES HUMANS UNIQUE

Homo sapiens have evolved in the recent past of the earth's history. If hominids have existed for seven million years, then Homo sapiens for only 1% of that time, and the great human achievements for 5-10% of that period (ie: up to 7000 years) (Cacioppo et al 2007).

What accounts for the achievements of humans in terms of our evolved bodies?

a) Genes - No: few differences in the number of genes to other primates/great apes (Cacioppo et al 2007).

But the 1% difference in DNA masks 30 million point mutation differences with 80% of 30 000 human genes (proteins) being affected (Glazko et al 2005) [34]. For example, the FOXP2 gene seen as crucial in the development of language (allows for the subtle facial movements needed) only differs by two amino acids in humans and chimpanzees (Jones 2012a) [35].

Other key genetic differences found recently include the 118 bases known as human accelerated region 1 (HAR1) involved in development of the cerebral cortex, ASPM (which controls brain size), HAR2 (or HACNS1) (controlling wrist and thumb development in the womb), and AMY1 (allowing the digestion of starch) (Powell 2009).

Molecular genetic analysis in recent years has shown uniquely human gene families, probably originating from "mobile genes" (insertion and deletion variations; INDELs) that randomly move around the genome during duplication (Polavarapu et al 2011).

b) Frontal cortex of brain - No: similar ratio of frontal cortex to total brain matter as other primates (Cacioppo et al 2007).

c) Number of cortical neurons in brain - Not really: more than most mammals, but less than whales and

---

[34] "The way to evolve a human from a chimp-human ancestor is not to speed the ticking of the molecular clock as a whole. Rather the secret is to have rapid change occur in sites where those changes make an important difference in an organism's functioning" (Pollard 2009 p47).

[35] It is believed to have appeared 500 000 YA (Pollard 2009).

elephants (Cacioppo et al 2007).

d) Small specialist differences in response to living in social groups - Yes: greater information-processing capacity of the brain due to, for example, greater number of synapses (Cacioppo et al 2007).

1.6. CONCLUSIONS

This is a time of change in the understanding of the evolution of humans. There are a number of issues and debates that will continue to be important:

- The use of molecular genetic studies compared to fossil finds.
- The divergence from a common ancestor of apes.
- Small evolutionary changes make big differences.
- The link between Australopithecus and Homo.
- The different Homo species (many alive at the same time).
- Fossils finds outside East Africa (eg: South Africa, and East Asia).

1.7. APPENDIX 1A - DIFFERENT SPECIES BETWEEN APE AND HUMAN

The complexity of the situation in terms of the evolution from ape to human is shown by the varied species named from fossil finds over the years (figure 1.8), and the debates over their acceptance as hominids (Grehan and Schwartz 2009).

MILLION YEARS AGO

Khoratpithecus (two species: K. chiangmuanensis and pirivai; 13 - 10 MYA; Thailand)

12

Dryopithecus (3 species from Europe 12-9 MYA: D. fontani, brancoi, and crusafonti)
Sivapithecus 3 species: S. indicus, sivalensis, and parvada; 12.5 - 8 MYA; Asia)
Ankarapithecus (formerly Sivapithecus meteai) (10.7 - 10.6 MYA; central Turkey)

10

Hispanopithecus laietanus (10 -9.5 MYA; Spain)
Ouranopithecus macedoniensis (9 MYA; Greece)
Lufenpithecus (L. lufengensis and keiyuanensis; 9 - 7 MYA; China)

8

Gigantopithecus (3 species: G. blacki, bilaspurensis, and giganteus; 7.8 - 7.5 MYA in Asia)
Sahelanthropus tchadensis (7-6 MYA; Chad)

MILLION YEARS AGO

6

Orrorin tugenensis (6 MYA; Kenya)
Ardipithecus (two species from Ethiopia: A. ramidus (4.4 MYA) and A. kadabba (5.8 - 5.2 MYA))

4

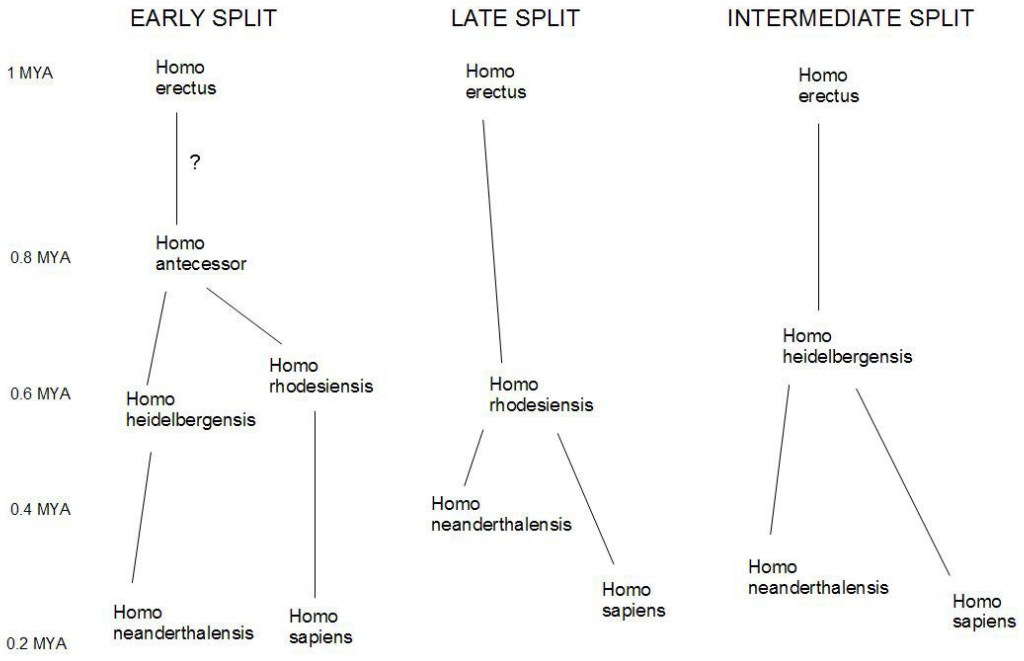
Australopithecus-Paranthropus (at least 10 species in east and south Africa 4.5 - 2 MYA)
Kenyanthropus platyops (3.5 MYA; Kenya)

(Information from Grehan and Schwartz 2009)

Figure 1.8 - Approximate time periods of different species of early hominids and where fossils found.

1.8. APPENDIX 1B - SPLIT BETWEEN NEANDERTHALS AND HUMANS

Hublin (2009) outlined three possible models for the split between Neanderthals and humans based on the fossil evidence of the time (figure 1.9).



(Homo rhodesiensis = large-brained ancestor of Homo sapiens in Africa and root of Neanderthals in Europe; Hublin 2009)

(Based on Hublin 2009 figure 1 p16024)

Figure 1.9 - Three possible models of the Neanderthal-human split.

1.9. APPENDIX 1C - AQUATIC APE THEORY

The aquatic ape theory (AAT) was first proposed by Alister Hardy in 1960, and championed by Elaine Morgan (eg: Morgan 1997). The main evidence being common anatomical features between humans and aquatic/semi-aquatic mammals like no fur, and fat deposits directly under the skin.

Jablonski (2010) pointed out three reasons why the AAT is generally seen as wrong:

i) There is no simple connection between fur and the environment in which the animal lives.

ii) The semi-aquatic environments were full of predators (eg: crocodiles) that would have severely threatened humans living in them.

iii) It is an overly complex explanation to suggest that humans evolved from land-based to water-based, and then back to land-based. The common view is that humans were always land-based. Furthermore, why are so-called aquatic adaptations still maintained by land-dwelling humans (Pagel and Bodmer 2003)?

Pagel and Bodmer (2003) pointed out that though fossils have been found near water, no "aquatic ape" fossils have been unearthed to date.

1.10. REFERENCES

Ainsworth, C & LePage, M (2007) Evolution's greatest mistakes New Scientist 11/8, 36-39

Atkinson, Q.D et al (2009) Bayesian coalescent inference of major human mitochondrial DNA haplogroup expansions in Africa Proceedings of Royal Society B 276, 367-373

Bailey, D.H & Geary, D.C (2009) Hominid brain evolution Human Nature 20, 67-79

Barras, C (2012) Humanity's roots New Scientist 24/3, p54

Berger, L et al (2010) Australopithecus sediba: A new species of Homo-like Australopith from South Africa Science 328, 195-204

Borrell, B (2010) All in the family Scientific American August, p33

Brahic, C (2011) Humans - who do you think you are? New Scientist 24-31/12, p27

Bramble, D.M & Lieberman, D.E (2004) Endurance running and the evolution of Homo Nature 432, 345-352

Braun, D.R et al (2010) Early hominin diet included diverse terrestrial and aquatic animals 1.95 MYA in East Turkana, Kenya Proceedings of National Academy of Sciences, USA 107, 22, 10002-10007

Byars, S.G et al (2010) Natural selection in a contemporary human

population Proceedings of National Academy of Sciences, USA 107, sup 1, 1787-1792

Cacioppo, J.T et al (2007) Social neuroscience: Progress and implications for mental health Perspectives on Psychological Science 2, 2, 99-123

Cann, R.L et al (1987) Mitochondrial DNA and human evolution Nature 329, 31-36

Choi, C.Q (2010) Heavy brows, high art Scientific American March, 10-11

Cocker, J (2005) Who was (and who wasn't) Mitochondrial Eve? Association for the Teaching of Psychology (ATP) Newsletter June, 13-16

Culotta, E (2006) Sceptics seek to slay the "hobbit", calling Flores skeleton a modern human Science 313, 1028-1029

Curnoe, D et al (2012) Human remains from the Pleistocene-Holocene transition of southwest China suggest a complex evolutionary history for East Asians PLoS ONE 7, 3, e31918

Dominguez, M.H & Rakic, P (2009) The importance of being human Nature 462, 169-170

Douglas, K (2012a) Why did we become bipedal? New Scientist 24/3, 36-37

Douglas, K (2012b) Why was technological development so slow? New Scientist 24/3, 36-37

Douglas, K (2012c) Why did we lose our fur? New Scientist 24/3, p39

Douglas, K (2012d) Why did we go global? New Scientist 24/3, 40-41

Dunbar, R (2004) The Human Story London: Faber & Faber

Editor (2012) Riddles of the past New Scientist 24/3, 35-42

Faisal, A et al (2010) The manipulative complexity of Lower Paleolithic stone toolmaking PLoS ONE 5, 11, e13718

Finlayson, C (2009) The Humans Who Went Extinct Oxford: Oxford University Press

Froehle, A.W & Churchill, S.E (2009) Energetic competition between Neanderthals and anatomically modern humans PaleoAnthropology 96-116

Gibbons, A (2008) The birth of childhood Science 322, 1040-1043

Glazko, G et al (2005) Eighty percent of proteins are different between humans and chimpanzees Gene 346, 215-219

Green, R.E et al (2010) A draft sequence of the Neanderthal genome Science 328, 710-722

Grehan, J.R & Schwartz, J.H (2009) Evolution of the second orang-utan: Phylogeny and biogeography of hominid origins Journal of Biogeography 36, 1823-1844

Hardy, A (1960) Was man more aquatic in the past New Scientist 7, 642-645

Hawkes, K et al (1998) Grandmothering, menopause, and the evolution of human life histories Proceedings of National Academy of Sciences, USA 95, 1336-1339

Holmes, B (2009) Evolution is alive and well in modern women New Scientist 24/10, p14

Hublin, J.J (2009) The origin of Neanderthals Proceedings of National

Academy of Sciences, USA 106, 38, 16022-16027

Jablonski, N.G (2010) The naked truth Scientific American February, 28-35

Jones, D (2012a) Why are we so different from chimpanzees? New Scientist 24/3, p35

Jones, D (2012b) Are some of us hybrids? New Scientist 24/3, 40-41

Kittles, R et al (2003) Molecular evolution of Pediculus humanus and the origin of clothing Current Biology 13, 1414-1417

Klein, R.G (2009) Darwin and the recent African origin of modern humans Proceedings of National Academy of Sciences, USA 106, 38, 16007-16009

Lawton, G (2009) Are orangs our nearest relative? New Scientist 20/6, 6-7

Li, J.Z et al (2008) Worldwide human relationships inferred from genome-wide patterns of variation Science 319, 1100-1104

Marean, C.W (2010) When the sea saved humanity Scientific American August, 40-47

Marshall, M (2010) Evolution by shake, rattle and roll New Scientist 13/11, 8-9

Matthews, L.J et al (2011) Novelty-seeking DRD4 polymorphisms are associated with human migration distance out-of-Africa after controlling for neutral population gene structure American Journal of Physical Anthropology 145, 382-389

McAuliffe, K (2011) The shrinking brain Discover Magazine: The Brain 23-29

Mellars, P (2006) Why did modern human populations disperse from Africa ca. 60 000 years ago? Proceedings of National Academy of Sciences, USA 103, 25, 9381-9386

Morgan, E (1997) The Aquatic Ape Hypothesis Harmondsworth: Penguin

Morwood, M.J et al (2004) Archaeology and age of a new hominin from Flores in eastern Indonesia Nature 431, 1082-1091

Neimark, J (2011) How we won the hominid wars Discover: Evolution Summer, 56-58

Pagel, M & Bodmer, W (2003) A naked ape would have fewer parasites Proceedings of Royal Society B 270, S117-S119

Polavarapu, N et al (2011) Characterisation and potential functional significance of human-chimpanzee large INDEL variation Mobile DNA 2, 13

Pollard, K.S (2009) What makes us human? Scientific American May, 44-49

Potts, R (1998) Variability selection in hominid evolution Evolutionary Anthropology: Issues, News and Reviews 7, 3, 81-96

Pritchard, J.K (2010) How are we evolving Scientific American October, 23-29

Reich, D et al (2010) Genetic history of an archaic hominin group from Denisova Cave in Siberia Nature 468, 1053-1060

Reynolds, S.C et al (2011) Landscapes and their relation to hominin habitats: Case studies from Australopithecus sites in eastern and southern Africa Journal of Human Evolution 60, 281-298

Robson, D (2012a) When did language evolve? New Scientist 24/3, p38

Robson, D (2012b) Why are our brains so big? New Scientist 24/3, p39

Robson, D (2012c) Did we exterminate the Neanderthals? New Scientist 24/3, p42

Robson, D (2012d) Are there other hominins left? New Scientist 24/3, p42

Robson, S.L et al (2006) The derived features of human life history. in Hawkes, K & Paine, R.R (eds) The Evolution of Human Life History Sante Fe: School of American Research Press/Oxford: James Currey

Rogers, A.R et al (2004) Genetic variation at the MC1R locus and the time since loss of human body hair Current Anthropology 45, 1, 105-108

Ruxton, G.D & Wilkinson, D.M (2011) Thermoregulation and endurance running in extinct hominins: Wheeler's model revisited Journal of Human Evolution 61, 169-175

Schultz, N (2007) Walking tall to carry a baby New Scientist 15/12, p9

Schwartz, J.H (1984) The evolutionary relationship of man and orang-utans Nature 308, 501-505

Stedman, H.H et al (2004) Myosin gene mutation correlates with anatomical changes in the human lineage Nature 428, 415-418

Stringer, C.B et al (2008) Neanderthal exploitation of marine mammals in Gibraltar Proceedings of National Academy of Sciences, USA 105, 38, 14319-14324

Tattersall, I (2009) Human origins: out of Africa Proceedings of National Academy of Sciences, USA 106, 38, 16018-16021

Ward, P (2009) What will become of Homo sapiens Scientific American January, 68-73

Wheeler, P.E (1984) The evolution of bipedality and loss of functional body hair in hominids Journal of Human Evolution 13, 91-98

Wong, K (2009) Twilight of the Neanderthals Scientific American August, 34-39

Wong, K (2010) Our inner Neanderthal Scientific American July, 9-10

Wong, K (2012) First of our kind Scientific American April, 20-29

Wong, K & Deak, V (2009) The human pedigree Scientific American January, 60-63

Wrangham, R (2009) Catching Fire: How Cooking Made Us New York: Basic Books

Zilhao, J et al (2010) Symbolic use of marine shells and mineral pigments by Iberian Neanderthals Proceedings of National Academy of Sciences, USA 107, 3, 1023-1028

2. SOME ORTHODOX AND UNORTHODOX IDEAS ABOUT WEIGHT GAIN AND LOSS

2.1. INCREASING OBESITY TODAY

Approximately one-third of adults in the USA are obese, and between 4-37% in different parts of Europe (Hatch et al 2010).

Body mass index (BMI) is commonly used to assess weight, and the World Health Organisation (WHO) defines ranges of BMI based on risk of health problems and/or premature death - underweight (<18.5 kg/m²), overweight (≥25 kg/m²), and obese (≥30 kg/m²). The risk of premature death among obese individuals has been "clearly identified" (Orpana et al 2010).

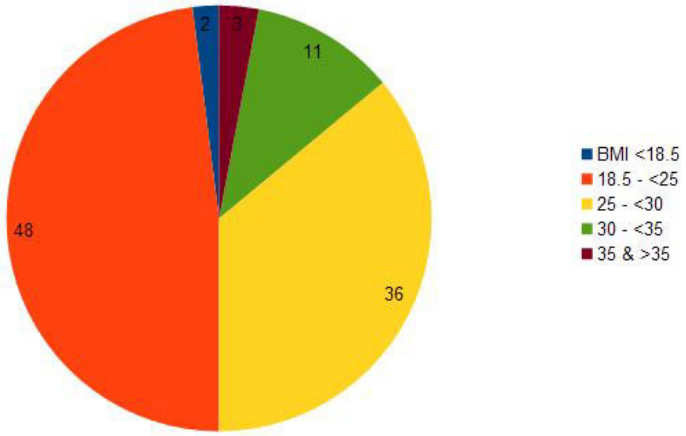
However, it is contested as to the relationship between overweight and premature death. For example, Adams et al (2006) reported that being overweight at age fifty was a risk for earlier death. But this was a retrospective study of members of the American Association of Retired Persons who were asked to recall their weight at 50. This study could be prone to recall bias and/or the "health survivor effect" (Orpana et al 2010).

On the other hand, data from the National Health and Nutrition Examination Survey in the USA (Flegal et al 2005) found no association between being overweight and the increased risk of death.

Orpana et al (2010) analysed data from a longitudinal national representative population survey of Canadian adults (National Population Health Survey) to establish the relationship between weight and risk of death. The twelve-year study began in 1994-5 and followed up the 11 326 over-25 year-old participants every two years. In total, 1 929 individual had died during the study period.

The majority of participants (48%) had a BMI in the normal range (18.5 - <25 kg/m²) (figure 2.1), and they were classed as the reference group (risk = 1.00). A relative risk higher than one equals a greater chance of early death, and less chance with a relative risk of below one. Though small in number (2% of the sample), underweight participants had the highest risk (1.73), followed by very obese (or class-II) (≥35 kg/m²) with a relative risk of 1.36. But overweight and obese (class-I)

(30 - <35 kg/m²) had lower risks of early death at 0.83 and 0.95 respectively. All the four relative risks were significantly different to the reference group.



(Data from Orpana et al 2010 table 1 p215)

Figure 2.1 - Distribution of sample (%) in terms of BMI.

It is accepted wisdom that obesity increasing in the West is due to eating more calories than are expended in exercise, and the answer to weight lose is to turn the equation around (make energy expenditure greater than calorie intake). But recent research has suggested that it may not be as simple as this. For example, Klimentidis et al (2011) found signs of obesity in non-human species living around industrialised humans.

They collected data on eight different species of animals living with or around humans in industrialised societies (in 24 different populations). These included primates (macaques, chimpanzees, vervets, and marmosets), and rodents (mice and rats) in research colonies, domestic dogs and cats, and feral rats living in cities and towns. Average mid-life body weight had risen in all groups (table 2.1), and 11 of the 24 populations were significant.

There may be a different explanation for each population (eg: over-feeding of pets; feral rats and increased refuse), but Klimentidis et al (2011) speculated that there may be an overall explanation that also applies to humans (eg: endocrine disrupters or a virus).

SPECIES	STUDY POPULATION	PERCENTAGE INCREASE IN WEIGHT PER DECADE
Macaques	Wisconsin National Primate Research Center, USA; 1971-2006 (n = 65)	5.33
Chimpanzees	Yerkes National Primate Research Center, USA; 1985-2005 (n = 46)	33.6
Mice	National Toxicology Programme data in USA; 1982-2005	12.46
Cats	1989-2001	9.72
Feral rats	1948-2006 captured in urban USA	6.88

(Source: Klimentidis et al 2011 table 1 p1629)

Table 2.1 - Examples of species and body weight increase.

2.2. IDEAS ABOUT WEIGHT GAIN AND LOSS

1. Virus.

A common cold virus, adenovirus-36 (Ad-36) has been found to increase fat inside cells, and the number of fat cells in the body. Obese individuals were nearly three times more likely to have Ad-36 antibodies (a sign of current or past infection) than non-obese individuals (30% vs 11% respectively)(Atkinson et al 2005).

Over 30 weeks, 25 young male rats injected with Ad-36 gained significantly greater body weight than 25 controls despite the same food intake (figure 2.2) (Pasarica et al 2006). Similar results have been found with chickens, mice, and marmosets (Pasarica et al 2006).

Another ten microbes have also been reported to increase fat (Dhurandhar et al 2004).

	CONTROLS	EXPERIMENTAL RATS
Pre-test	161	162
	↓	↓
12 weeks after infection	515	518
	↓	↓
30 weeks	588	628 *
Food intake at week 30	26.36 grams per day	26.05

(* $p<0.008$)
(Source Pasarica et al 2006 table 2 p1909)

Figure 2.2 - Mean weights (grams)

2. Stress.

Stress and the stress hormone corticotropin-releasing factor (CRF) lead to the consumption of calorically dense foods (Pankevich et al 2010). Dieting is a stressor, and so post-dieting individuals will rebound with weight gain.

Pankevich et al (2010) found that mice restricted to 75% of average calorie intake for three weeks showed high-fat food binge eating behaviour when subsequently given access to unlimited food. Ultimately they gained weight compared to their starting body weight before the calorie restriction. The researchers reported that the stress (calorie restriction) had altered the brain chemistry (eg: orexin) which motivated the subsequent binge-eating.

3. Warm houses.

Bo et al (2011) found that middle-aged adults living in houses with the highest thermostat temperatures were twice as likely to become obese over the next six years. Put simply, shivering burns energy.

Bo et al (2011) reported that a number of modern lifestyle factors were associated with obesity in an Italian study. Just over 1500 45-64 year-olds from six family physicians in the province of Asti [36] were questioned about their lifestyle including average sleep duration, house temperature during autumn/winter, number of hours watching television per day, use of air conditioning in the summer, and number of weekly visits to restaurants. Obesity was measured by BMI.

After adjusting for socio-demographic variables, obesity was significantly associated with less exercise, greater number of weekly visits to a restaurant (4 or more times) [37], less sleep (less than 6.5 hours per day) [38], and having the highest average house temperature (>20°C compared to <18°C) [39].

[36] North west Italy, between Turin and Genoa.

[37] Restaurant food tends to have higher energy density and more fat with larger portion sizes than food prepared at home (Bo et al 2011).

[38] Less sleep or "sleep debt" produces physiological changes, like reduced leptin and increased ghrelin, cortisol and orexin secretion, which stimulate appetite and food intake (especially for energy-dense, high-carbohydrate foods) (Bo et al 2011).

[39] Cold temperature stimulates the burning of brown adipose tissue and use of calories, which does not happen with warm house temperatures, and a warm house discourages exercise and going outside in cold weather (Bo et al 2011).

4. High-protein diet.

There are many claims about such diets marketed as the "Atkins diet" or the "Dukan diet" (Young 2011), but research does support such protein diets in reducing overall calorie consumption.

The "protein leverage hypothesis" (Simpson and Raubenheimer 2005) proposes that a reduction in the amount of protein in relation to fat and carbohydrate in the Western diet in recent years (1960 onwards) is associated with an increase in total energy intake [40]. Not only in humans, but in other species studied (eg: rodents, pigs, non-human primates), when protein is decreased there is an overall increase in amount eaten to maintain the protein intake (Gosby et al 2011).

Gosby et al (2011) experimentally manipulated protein content in a study of 22 lean volunteers in Sydney, Australia [41]. The 28 food items (table 2.2, figure 2.3) offered were adjusted to contain 10%, 15% or 25% energy as protein in three four-day study periods (ie: repeated measures design) [42]. Participants were allowed to eat as much as they wanted with free access to a full refrigerator (while outside food sources were forbidden). The protein content of the food was disguised from the participants.

	Study day 1	Study day 2	Study day 3	Study day 4
Breakfast		**Savoury breakfast muffin**	**Savoury breakfast muffin**	**Savoury breakfast muffin**
8.30–10.00am				
		Apricot yoghurt muesli	Raspberry yoghurt muesli	Apricot yoghurt muesli
		Pear, raspberry & coconut bread	**Banana bread**	**Pear, raspberry & coconut bread**
Lunch	Tuna bake	Mexican wrap	Tandoori wrap	Sweet potato wrap
1pm				
	Beef and vegetable pastry	Teriyaki sushi roll	Beef and vegetable pastry	Pasta salad
	Salad & dressing	Salad & dressing	Salad & dressing	
	Fruit salad yoghurt	**Apple crumble muffins**	**Fruit salad yoghurt**	**Apple crumble muffins**
Dinner	Goulash	Mushroom Pasta	Pasta Bolognaise	Hokkien noodles
6.30pm				
	Cheese Scones	Chow mein mince	**Cheese Scones**	Massaman curry
	Salad & dressing		Salad & dressing	
	Orange & poppyseed cake	**Chocolate, apple & ricotta cake**	**Orange & poppyseed cake**	**Chocolate, apple & ricotta cake**
	Custard	**Custard**	**Custard**	**Custard**
Snacks	**Savoury scones**	**Cheese scones**	**Savoury scones**	**Cheese scones**
all day				
	Carrot cake	**Raspberry yoghurt**	**Apricot muffins**	**Raspberry yoghurt**

Foods offered during the 10%, 15% and 25% protein 4-day *ad libitum* study periods. The methodology used to design each of these foods and the final nutritional information can be found elsewhere [22]. Some foods were only available in one meal sitting ('meal time foods': not bold), whereas others were available to participants anytime once served ('anytime foods': bold). Anytime foods were 'snack' foods and foods that were first served at a meal and could be kept if not eaten or finished at that meal; these foods were labelled for identification and kept in a refrigerator to which participants had free access.

doi:10.1371/journal.pone.0025929.t006

Table 2.2 - Example of menu of food items.

[40] In the USA, between 1961 and 2000, dietary protein declined from 14% to 12.5% while non-protein energy intake increased 14% (Simpson and Raubenheimer 2005).

[41] Sixteen women and six men recruited at the universities or via the local newspaper with a BMI between 18-25 kg/m².

[42] Participants were resident at the Woolcock Institute of Medical Research during each four-day period (with approximately one week between each study period).

The 3 photos on the left column are the 10%, 15% and 25% versions (top to bottom) of each food given to participants at breakfast on study day 2. In the right hand column the three photos are the 10%, 15% and 25% versions (top to bottom) of each food given to participants at dinner on study day 2. Participants were offered a set amount of each food that was the same on each study period. The plates were the same for a particular food on each study period.

(Source: doi:info:doi/10.1371/journal.pone.0025929.g004)

Figure 2.3 - Photographs of some food items.

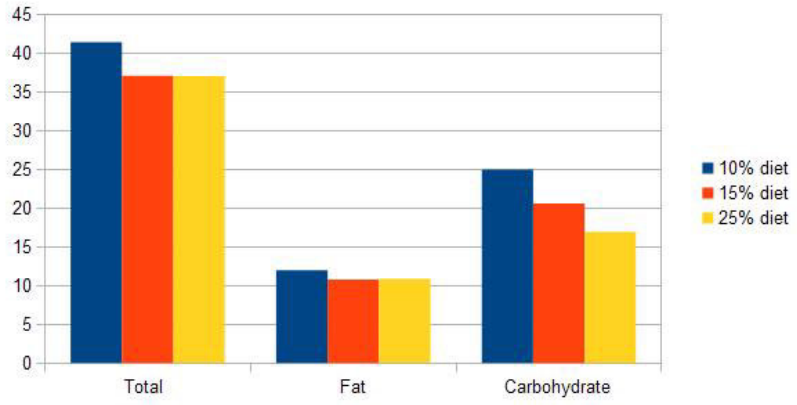
Food intake was measured by subtracting the weight of food after eating from the weight before, and this was converted into energy intake. Self-reports of hunger and fullness were taken regularly.

The participants ate significantly more food (approximately 12% more energy) when the energy as protein was less, and specifically more carbohydrate and

fats (figure 2.4). The difference in energy intake between the 10% and 25% diets would produce an equivalent of 1 kg weight increase per month.

A significantly greater hunger score (out of 100) was reported on the fourth day of the trial after the 10% protein breakfast than the 255 breakfast.

Gosby et al (2011) concluded: "It follows from our results that any change in the nutritional environment that encourages dilution of dietary protein with fat and/or carbohydrate will promote increased total energy intake and thus increase the risk that obesity might develop. Many sources of such encouragement exist in the modern westernised environment. Some are economic - fat and carbohydrate are cheaper than protein...; others reflect an increasing reliance on processed foods which are often higher in fat and refined carbohydrate than unprocessed foods, and yet other influences include our evolutionary heritage, which has left us with a predilection for foods with a high fat and sugar content... To make matters worse, it appears that the beneficial side of protein leverage – reduced intake on high percent protein diets – may be diminished in westernised countries in which the variety and availability of foods, especially snack foods, is greater than it has ever been in our evolutionary history" (p7).



(Source: Gosby et al 2011 table 1 p2)

Figure 2.4 - Energy intake (MJ) based on protein diet.

5. Endocrine disrupters.

Complex endocrine and other chemical signalling mechanisms are involved in resting and non-resting energy expenditure, and these are altered by chemicals in the environment. Endocrine disrupting chemicals (EDCs) or "obesogens" are "molecules that inappropriately regulate

lipid metabolism and adipogenesis [43] to promote obesity" (Grun and Blumberg 2006), and include diethylstilbestrol (DES), bisphenol A (BPA), and phthalates (found in plastic packaging).

EDCs can alter weight homeostasis in a number of ways including regulation of fat cell development, thyroid function, sex hormones, and in the womb (in utero) (Hatch et al 2010).

Hatch et al (2010) found a positive correlation between body weight and the amount of six phthalate metabolites in urine in the USA. Data on over 4000 adults were collected in 1999-2002. Confounding variables like physical activity and diet were controlled for in the statistical analysis. The association was stronger for men than women.

However, this association or correlation is not causation. It is possible that the variables could work either way - EDCs cause obesity or being obese causes the body to produce more EDC metabolites, for example.

6. Light at night.

Artificial light at night interferes with the circadian rhythms, which disrupts "natural" sleep and eating patterns. Furthermore, this disruption of the circadian rhythms is linked to obesity in mice, for example.

Fonken et al (2010) kept mice in one of three different conditions for eight weeks - 24 hours of continuous light (LL), 16 hours of light and 8 hours of dim light (DM), or 16 hours of light and 8 hours of darkness (LD). There was a significant increase in body mass of the mice in the LL and DM conditions relative to the LD condition despite equivalent calorie intake and daily activity. The light at night seemed to produce a desynchrony between metabolic activity and food intake, and this accounted for the increase in body mass.

7. Polluted air.

Exposure to air pollution in the form of fine particulate matter is a risk factor for health (eg: cardiovascular problems), and for fat storage.

Xu et al (2010) compared three-week old mice reared in controlled laboratory conditions receiving polluted or filtered air for six hours per day, five days a week, for ten weeks. The mice in the polluted condition showed a greater gain in body weight compared to the filtered condition (controlling for normal or high fat diet).

[43] Storage of fat.

8. Sleep deprivation.

In a four-year longitudinal study in New Zealand, Carter et al (2011) found that young children who had less sleep were more likely to be overweight. The Family Lifestyle, Activity, Movement and Eating (FLAME) study followed children born between 19 July 2001 and 19 January 2002 at the Queen Mary Maternity Unit, Dunedin. Of them, 244 were seen every six months between the ages of 3 and 7 years old for this study.

Sleep deprivation and physical activity were measured with activity monitors attached by belts to the waist of the children for sample periods, and the data divided into fifteen-second segments. Parents also kept activity logs including bedtimes and wakings.

Form the data, statistical analysis showed that each additional hour of sleep between 3-5 years old was associated with a reduction of 0.49 in BMI at age 7 (or 0.39 controlling for diet and physical activity) (ie: 0.7 kg in weight) and a 61% reduction in risk of being overweight or obesity.

Sleep deprivation reduces the secretion of leptin, which suppresses appetite, and increases ghrelin (that stimulates it) (Young 2011).

9. Chewing gum.

There are claims that chewing gum can suppress appetite [44] and increase energy expended [45]. Shikary et al (2012) investigated these claims in an eight-week randomised clinical trial [46]. Two hundred and one overweight and obese volunteers in the Birmingham area of Alabama, USA, were randomised to the experimental condition (chewing sugar-free gum for 90 minutes/day in six sessions [47], and printed material on good nutrition [48]) or the control condition (printed material only). Regular gum chewers, smokers, individuals already on weight loss programmes, and those with health problems were excluded from the study.

Adherence in the experimental condition was assessed from participants' gum-chewing diaries of minutes per day, and by counting the empty gum wrappers [49]. BMI and waist circumference were taken at baseline and at week 8.

---

[44] Eg: 8% less eaten after 15 minutes per hour chewing gum (Hetherington and Boyland 2007).

[45] Eg: 11 kcal/hour (which equals a loss of 5 kg/year) (calculations based on data from seven non-obese individuals) (Levine et al 1999).

[46] Details at http://www.clinicaltrials.gov/ct2/show/NCT00971347?term=NCT00971347&rank=1.

[47] Twenty minutes each after breakfast, lunch, and dinner, and 10 minutes each mid-morning, mid-afternoon, and mid-evening.

[48] "Finding Your Way to a Healthier You" (Dietary Guidelines for Americans 2005).

[49] Adherence was over 90%.

Waist circumference decreased significantly between baseline and week 8 in the experimental group (mean reduction of 1.4 cm), but there were no significant differences between the conditions. However, Shikany et al (2012) did not measure any changes in diet or physical activity during the study period.

The findings of this study are different to other studies (as detailed in table 2.3), and this may be due to differences between the studies like:

- Laboratory study versus free-living/natural environment.
- Normal weight or overweight/obese participants.
- Timing (when in day), frequency, and duration of chewing gum.

Hetherington & Boyland (2007)	Julis & Mattes (2007)
• 60 normal weight participants. • Laboratory experiment. • Lunch, then snack 3 hours later. • Gum chewed for 15 minutes per hour. • Gum chewers ate significantly less of snack.	• 47 overweight and obese participants. • Free-living study. • No chewing vs chewing sugar-sweetened gum for 20 minutes two hours after lunch vs chew gum for 20 minutes when hungry. • No difference between groups in food eaten.

Table 2.3 - Differences in methodology between two other studies.

Jabr (2012) reported work by Robert Doyle to develop a chewing gum that releases human peptide YY (hPYY). This hormone seems to signal satiety to the hypothalamus and it is released from intestinal cells into the bloodstream. Individuals given a dose of hPYY two hours before lunch ate 30% less than controls at a free buffet.

10. Laughter.

Genuine voiced laughter (ie: "laughing out loud") produces a 10-20% increase in energy expended compared to the resting state [50]. This converts to 10-15 minutes of laughter expends 10-40 kcal depending on body weight and laughter intensity (Buchowski et al 2007).

[50] This is the equivalent to writing or playing cards, whereas jogging increases energy expended by 100% (Buchowski et al 2007).

Buchowski et al (2007) recruited 31 men and 63 women (aged 18-34 years) in Nashville, USA, to watch humorous or non-humorous [51] film clips in friendship pairs for ninety minutes. This was done in an air-tight room that measured energy expenditure (whole-room indirect calorimeter) through oxygen consumption (VO_2) and carbon-dioxide production (VCO_2). More oxygen is consumed and carbon-dioxide produced as energy is expended.

Resting energy expenditure (kcal/min) was based on sitting in a reclining position for thirty minutes.

The average energy expended during a laughter episode was 1.32 kcal/min (or 5.53 kJ/min) which was significantly higher than during the resting state (and non-humorous film clips).

Table 2.4 summarises the main strengths and weaknesses of this study.

Strengths

1. Individuals watched the films with a friend, which was more realistic, because laughter is greater in social situations than in isolation and with friends than with strangers (Buchowski et al 2007).

2. Controlled measurement of energy expended.

3. Participants were told that the study was about their emotional reactions to film clips, and this reduced the risk of "demand characteristics" (ie: laughing to please the experimenters).

4. Able to gain measurements of energy expended in a way not possible in a real-life laughter situation.

5. Participants were asked to give written consent before the study, and again after the debriefing. This second occasion allowed the right of withdrawal of data by participants after they discovered the study's purpose.

6. A variety of individuals were involved in the study in terms of body weight (41 - 139 kg) and BMI (17.0 - 41.1 kg/m^2).

Weaknesses

1. Sample - young adults (probably mainly students) which limits the generalisability of the findings to other groups.

2. The calorimeter measured VO_2 and VCO_2 for two people rather than for an individual. The energy expended in the room was divided by two, but each individual may have expended different amounts of energy. Buchowski et al (2007) admitted: "Small EE [energy expenditure] measurement errors are inherent but mostly systematic and should not affect the difference between EE at rest and laughter EE measured in a strictly controlled environment" (p136).

3. The degree of friendship between the pairs was not controlled as the volunteers were simply asked to bring a friend to the study. This

[51] Documentary about England's landscapes.

could lead to variations in social comfort, and consequently in laughter duration and rate.

4. Some pairs were same-sex and some were mixed-sex. Average energy expended did vary between the type of pair (eg: 10% increase with laughter in male-male pairs compared to an 8% increase in female-female and mixed-sex pairs) suggesting that the laughter varied.

5. An artificial situation unlike a comedy show, for example, where laughter naturally takes occurs.

6. No details given of the humorous film clips used as what is perceived as funny varies between individuals.

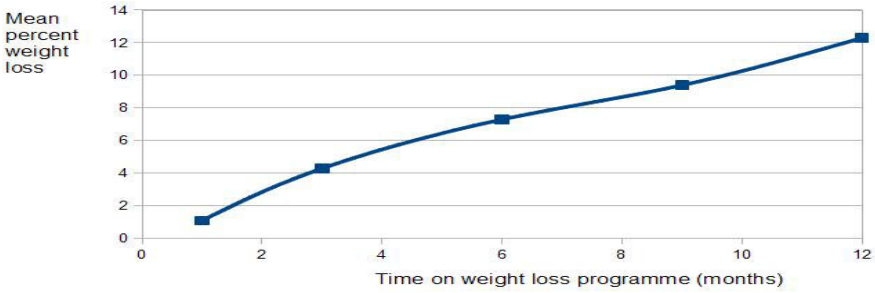
Table 2.4 - Main strengths and weaknesses of Buchowski et al (2007) study.

2.3. MORE ORTHODOX IDEAS

These recent ideas are interesting, and time will tell about them, but, in the main, losing weight still depends on energy expenditure being greater than calorie intake.

There are many commercial weight loss programmes, and few have been assessed in large-scale studies. One exception is the "Jenny Craig Platinum Programme", which tailors the food (1200-2000 kcal per day) and physical activity (30 minutes on five or more days a week) to the client's needs.

Finley et al (2007) analysed the data of 60 164 adults (18-79 years old) enrolled on the programme in the USA between May 2001 and May 2002. Overall, those who remained on the programme for 52 weeks (6.6% of starters [52]) [53] lost most weight (mean of 12% of baseline body weight; 12.6 kg [54]) (figure 2.5).



(Data from Finley et al 2007 table 1 p295)

Figure 2.5 - Time on weight loss programme and mean weight loss.

[52] Drop-out was defined as six or more consecutive weeks missing weight data.

[53] Finley et al (2007) calculated that one-year membership of the programme cost $1480 including enrolment fee and specialist food (pre-packaged Jenny Craig branded foods).

[54] This compares at one year to 5 kg lost in a study of the Weight Watchers programme (Heshka et al 2003), and 6.5 kg with the Take Off Pounds Sensibly (TOPS) programme (Garb and Stunkard 1974).

Researchers are interested in the developmental origins of obesity - namely, what happens in the womb and in the early years of life. Casazza (2011) described the importance of four critical periods in childhood for adult obesity - intra-uterine (in the womb), early post-natal, pre-puberty, and during adolescence (figure 2.6).

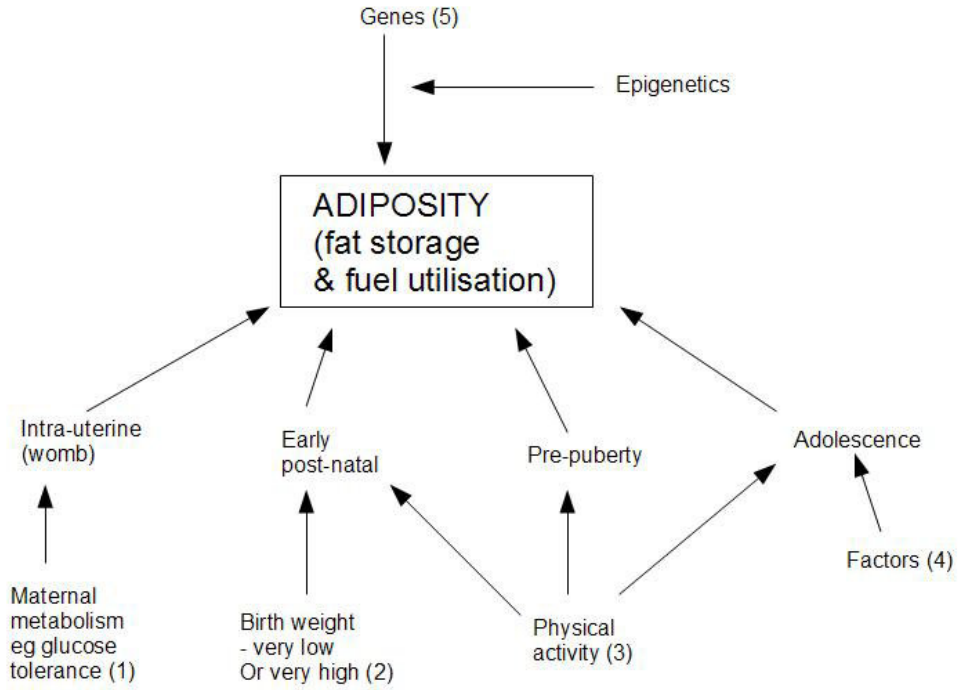


Figure 2.6 - Model of the developmental origins of obesity with four critical periods.

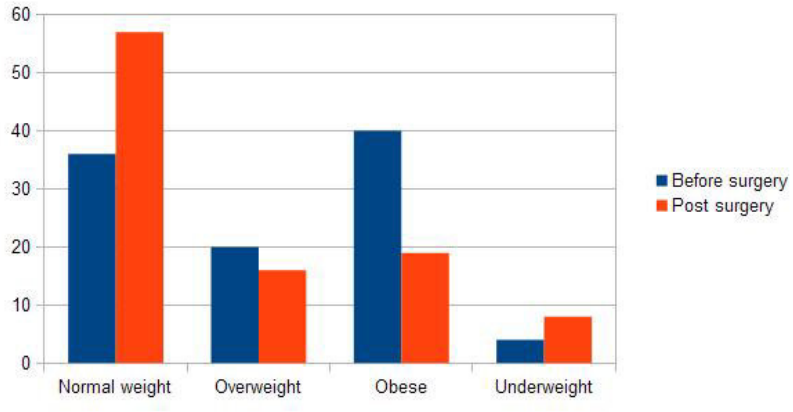
Key to figure:

(1) Children born to overweight and obese women have a greater risk of becoming obese themselves as compared to children born to normal weight mothers. It appears that the weight of the mother during pregnancy "programmes" the embryo for obesity, not through maternal adipose tissue but via other metabolic health abnormalities (eg: impaired glucose tolerance) from being overweight or obese (Chandler-Laney and Bush 2011). Overweight and obese women are more likely to have reduced insulin sensitivity [55], and so maternal glucose concentrations will be high and foetal overgrowth could occur through altered programming of energy balance regulation leading to the accumulation of fat mass (Chandler-Laney and Bush 2011).

Changes in the mother's weight between pregnancies gives a "natural laboratory" to study this issue. For example, Getahun et al (2007) compared consecutive pregnancies by women who had normal weight for the first pregnancy, but were obese by the second one (appendix 2A), while Kral et al (2006) [56] studied pregnant women

[55] Maternal insulin production usually counters the increase delivery of glucose to the foetus.
[56] This was a follow up of women who underwent surgery between 1982 and 2001 (eg: Marceau et al 2004).

before and after weight loss surgery. In the latter study, overweight and obesity among 172 children [57] born to 113 obese mothers post surgery [58] was no more than the average (approximately one-third), whereas 45 children born to 34 obese mothers prior to surgery were significantly more likely to become obese (approximately 60%) (figure 2.7). The BMI of the mothers were matched at conception. But the study was based on telephone interviews and mothers' self-reports of their children's heights and weights (to calculate BMI).



(Data from Kral et al 2004 table 1 p e1646)

Figure 2.7 - Weight categories of children (%) based on mothers' surgery status.

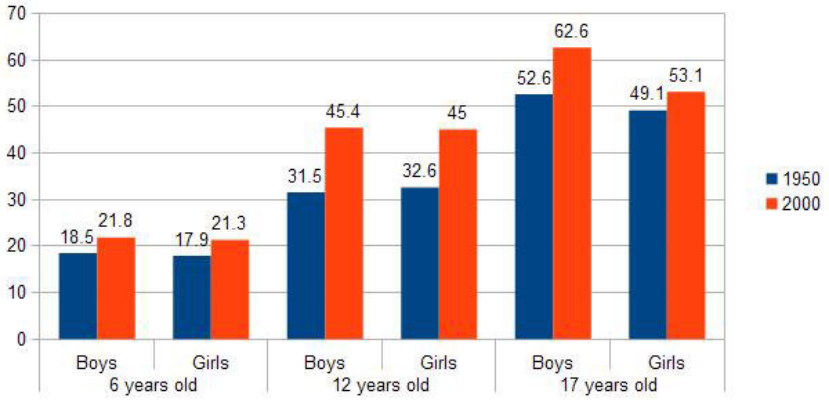
(2) Low birth weight (<2500 g) and high birth weight (>4000 g) are associated with later health problems and increased body weight and BMI (in the latter case in particular) (Willig et al 2011). A U-shaped curve between birth weight and obesity risk (Cardel et al 2011). Individuals in nutrition-restricted womb environments had more fat stored centrally on the body, which is a risk factor for cardiovascular disease (Kensara et al 2005).

However, Willig et al (2011) have challenged BMI as an appropriate measure of children's body size. BMI can overestimate body fat in taller children and underestimate it in shorter ones.

Willig et al measured fat mass, fat-free mass, and central adiposity in 256 7-12 year-olds in Birmingham, Alabama, USA, using dual-energy x-ray absorptiometry (DXA). Higher birth weight was significantly associated with increases in these three measures (but fee-fat mass was not significant after adjusting for physical activity).

(3) Using annually collected data on Japanese children from 1950 to 2000, Kagawa and Hills (2011) reported small increases in average body weight in each decade for 6-17 year-olds. For boys, average height increased by 5.6% to 13.3% over the period, and weight increased by 17.8% to 44.1%, while for girls the increases were 3.5% to 11.7% and 8.1% to 39.2% respectively (figure 2.8).The increases were seen as due to improved diet and increased energy intake, and less physical activity.

[57] All children were older than two years in age.
[58] Billopancreatic diversion to reduce the size of the stomach.



(Source: Kagawa and Hills 2011 table 1 p10 and table 2 p11)

Figure 2.8 - Average weight (kg) in 1950 and 2000 for selected ages.

(4) Cardel et al (2011) explained fat accumulation in adolescence as a combination of the "Big Two" (excess energy consumption and reduced physical activity), and five other factors:

- Metabolic programming in the womb and early childhood.
- Hormonal environment - the hormonal changes at puberty can alter the energy balance, particularly if the adolescent is stressed at the same time.
- Endocrine disruptors.
- Parental feeding practices - eg: restricting "unhealthy" or snack foods in childhood can be counterproductive leading to excess consumption when these foods more freely available in adolescence or adulthood, while "pressure to eat" nutrient-dense foods can lead to later lower BMI scores (Fisher and Birch 2002).
- Built environment - less "health-promoting resources" (eg: recreational facilities) and fear of crime discourage walking around cities. Cities also have more fast food restaurants and convenience stores (eg: each additional store per 10 000 capita associated with 0.15% increase in overweight; Powell et al 2007; appendix 2B).

(5) Twin, adoption, and family studies show the heritability of BMI varying from 40-90% with the importance of genes increasing with age (Rokholm et al 2011). However, there is room for the influence of the environment in the form of epigenetics (ie: environmental influences on the genetic development of the foetus). For example, sheep given increased nutrition in the womb had more fat 30 days after birth and a larger appetite than controls (Muhlhausler et al 2006).

Godfrey et al (2011) measured the DNA methylation (evidence of epigenetic processes) in the umbilical cord tissue at birth, and compared it with fat tissue of the children at nine years old in the Princess Anne Hospital study in Southampton, England. Methylation of specific genes was associated with fat mass.

Epigenetics also has transgenerational effects. Stein and Lumey (2000) reported that mothers who experienced the Dutch Famine during

World War II in the third trimester of their pregnancy has children with lower birth weight and increased risk of insulin resistance, and also their grandchildren had lower birth weight (irrelevant of the food available during their mother's pregnancy). The experience in the womb of famine altered the behaviour of the genes in the future generation "through interacting and complicated pathways, and there are still a lot of details about possible mechanisms that need to be unravelled" (Rokholm et al 2011 p31).

It has been found that genetically identical mice reared in the same environment show differences in behaviour. This goes against the accepted wisdom that behaviour is due to genes or environment, or more appropriately, genes and environment. It seems that the same genes can affect the embryo and/or later development through processes like "alternative splicing" (where a single gene can produce different proteins), epigenetics (changes in protein synthesis without changing the information in the genes), or "jumping genes" (mobile elements) (genes moving around within the genome) (Gage and Muotri 2012).

Mobile elements were first discovered in corn plants. Under stress certain regions of the genome can move or copy themselves elsewhere. So identical twins developing from the same egg could have different gene activation patterns because of "jumping genes". The brain is the organ of the body where this process appears to happen most often. An average of 80 "jumping" events could occur during development (Gage and Muotri 2012).

"Jumping genes" increase the risk of introducing potentially fatal genetic flaws, so what are the evolutionary advantages of this process? One suggestion is as a way to combat DNA invaders (eg: viral parasites) by changing the genome and disabling such invaders (Gage and Muotri 2012).

If weight gain can be reduced by increasing physical activity, it is necessary to measure such activity. But this is not straightforward. Physical activity involves elements like duration, intensity, frequency, and type. It can be measured by subjective methods like diaries/activity logs or by recall, or by objective methods based on physiology like heart rate or body temperature (Bonomi and Westerterp 2012).

Bonomi and Westerterp (2012) outlined common objective measures of physical activity:

i) Pedometers - counting the number of steps taken during walking or running.

Advantages:

- Unobtrusive.
- Can use for long periods without inconvenience.

- Better than recall.

Disadvantages:

- Step count accuracy reduced by slow walking and/or greater body weight.
- Not able to show non-walking/running energy expenditure.

ii) Accelerometers - measuring the acceleration of the human body during movement.

Advantages:

- Sensitive to any movement.
- Small and lightweight to use.
- Better than recall.

Disadvantages:

- Sophisticated technology that captures the intensity, duration and type of movement is demanding on battery life or else the equipment becomes obtrusive if larger.
- Can confuse sitting and standing.

iii) Activity monitors - more sensitive accelerometers that are able to distinguish movement and sedentary activities using the detection of gravity acceleration.

Issue - number of sensors and their placement on the body can alter accuracy (eg: waist, arm, ear).

iv) Activity monitors and physiological measures - combined methods give a more accurate measure of energy expended.

2.4. APPENDIX 2A - GETAHUN ET AL (2007)

Getahun et al (2007) used self-reported maternal pre-pregnancy BMI from 146 227 women in the state of Missouri, USA, between 1989 and 1997, at the time of their first two consecutive singleton births.

The women were divided into groups for the purposes of analysis:

1. Same pre-pregnancy weight category for 1st and 2nd pregnancies:

a) Underweight (BMI <18.5 kg/m²) (n = 6827 births).
b) Normal weight (BMI 18.5 - 24.9 kg/m²) (n = 71 867).
c) Overweight (BMI 25 - 29.9 kg/m²) (n = 12 535).
d) Obese (BMI >30 kg/m²) (n = 12 219).

2. Women who gained weight between 1st and 2nd pregnancies:

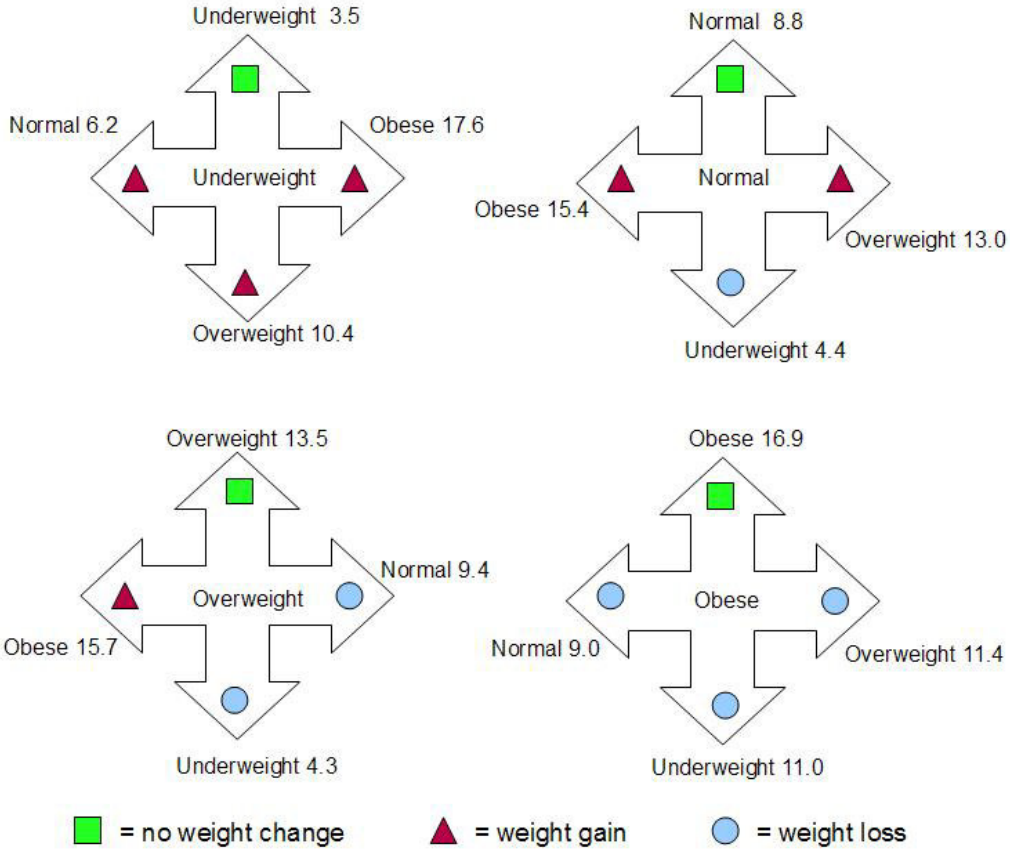
a) Underweight to normal weight (n = 5786).
b) Underweight to overweight (n = 211).
c) Underweight to obese (n = 91).
d) Normal weight to overweight (n = 13 492).
e) Normal weight to obese (n = 2376).
f) Overweight to obese (n = 6827).

3. Women who lost weight between 1st and 2nd pregnancies:

a) Obese to overweight (n = 2797).
b) Obese to normal weight (n = 1199).
c) Obese to underweight (n = 82).
d) Overweight to normal weight (n = 5971).
e) Overweight to underweight (n = 117).
f) Normal weight to underweight (n = 3830).

The main outcome measure was the birth weight of 2nd pregnancy, and particularly if it was large-for-gestational age (LGA) (defined as above 90th centile for sex).

Compared to group 1b (normal weight both), there was a greater risk of a LGA baby especially among groups 2c, 2f, 2d, and 2b (ie: weight gained), and less of a risk among groups 3e and 3f (ie: lost weight), for example (figure 2.9).



(Data from Getahun et al 2007 table 1)

Figure 2.9 - Percentage of second births LGA based on weight category at first and second pregnancies.

2.5. APPENDIX 2B - POWELL ET AL (2007)

Unhealthy food consumption (ie: high intake of fat, sugar, snacks, and fast food, and low intake of fruit and vegetables) are a key risk for overweight and obesity. Environmental factors in the modern Western world influence such eating patterns, most notably access to the particular foods.

The type of store in the local area is important as:

- larger food stores and chain supermarkets are more likely to stock healthy foods than smaller and independent food stores/supermarkets.
- larger stores and supermarkets offer food at cheaper prices.

Powell et al (2007) found statistically significant associations between chain supermarkets in the local area

and lower BMI among adolescents, and between convenience stores and higher BMI. The researchers used data from the Monitoring the Future (MTF) surveys in the USA, which are performed annually since 1991 with 30 000 adolescents. This gave the data on BMI. ZIP codes areas of the participants' schools were scored for number of chain supermarkets (mean 0.3 per 10 000 population), independent supermarkets (mean 0.26), convenience stores (mean 2.2), grocery stores (mean 3.3), and fast-food restaurants (2.6) using business lists.

Controlling for individual variables, an additional chain supermarket per 10 000 capita was associated with 0.6% less overweight, and an extra convenience store with 0.15% increase in overweight, according to Powell et al's calculations.

In terms of group differences, the association between chain supermarkets in the local area and lower BMI was three times higher for African-American adolescents than for White and Hispanic ones. It was also higher for all adolescents in families where the mother worked full-time as compared to part-time or not working. Combined the association was powerful (ie: African-American adolescents whose mothers worked full-time).

2.6. REFERENCES

Adams, K.F et al (2006) Overweight, obesity, and mortality in a large prospective cohort of persons 50 to 71 years old New England Journal of Medicine 355, 763-768

Atkinson, R.L et al (2005) Human adenovirus-36 is associated with increased body weight and paradoxical reduction of serum lipids International Journal of Obesity and Related Metabolic Disorders 29, 281-286

Bo, S et al (2011) Contributors to the obesity and hyperglycaemia epidemics. A prospective study in a population-based cohort International Journal of Obesity 35, 1442-1449

Bonomi, A.G & Westerterp, K.R (2012) Advances in physical activity monitoring and lifestyle interventions in obesity: A review International Journal of Obesity 36, 167-177

Buchowski, M.S et al (2007) Energy expenditure of genuine laughter International Journal of Obesity 31, 131-137

Cardel, M et al (2011) Contributors to paediatric obesity in adolescence: More than just energy imbalance 3, 17-26 (Download at http://www.benthamscience.com/open/toobesj/V003-SI0001TOOBESJ.htm)

Carter, P.J et al (2011) Longitudinal analysis of sleep in relation to BMI and body fat in children: The FLAME study British Medical Journal 342, d2712

Casazza, K.R (2011) Editorial: Developmental origins of obesity: A critical periods perspective The Open Obesity Journal 3, 1-3 (Download at http://www.benthamscience.com/open/toobesj/V003-SI0001TOOBESJ.htm)

Chandler-Laney, P.C & Bush, N.C (2011) Maternal obesity, metabolic health, and prenatal programming of offspring obesity The Open Obesity Journal 3, 42-50 (Download at http://www.benthamscience.com/open/toobesj/V003-SI0001TOOBESJ.htm)

Dhurandhar, N et al (2004) Obesity of infectious origin: A review Growth, Genetics and Hormones 20, 33-39

Finley, C.E et al (2007) Retention rates and weight loss in a commercial weight loss program International Journal of Obesity 31, 292-298

Fisher, J.O & Birch, L.L (2002) Eating in the absence of hunger and overweight in girls from 5 to 7 years of age American Journal of Clinical Nutrition 76, 226-231

Flegal, K.M et al (2006) Excess deaths associated with underweight, overweight and obesity Journal of the American Medical Association 293, 1861-1867

Fonken, L.K et al (2010) Light at night increases body mass by shifting the time of food intake Proceedings of the National Academy of Sciences, USA 107, 43, 18664-18669

Gage, F.H & Muotri, A.R (2012) What makes each brain unique Scientific American March, 20-25

Garb, J.R & Stunkard, A.J (1974) Effectiveness of a self-help group in obesity control. A further assessment Archives of Internal Medicine 134, 716-720

Getahun, D et al (2007) Changes in pre-pregnancy body mass index between the first and second pregnancies and risk for large-for-gestational-age birth American Journal of Obstetrics and Gynecology 196, 6, 530, e1-8

Godfrey, K.M et al (2011) Epigenetic gene promoter methylation at birth is associated with child's later adiposity Diabetes 60, 5, 1528-1534

Gosby, A.K et al (2011) Testing protein leverage in lean humans: A randomised controlled experimental study PLoS ONE 6, 10, e25929 (Freely available at http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0025929)

Grun, F & Blumberg, B (2006) Environmental obesogens: Organotins and endocrine disruption via nuclear receptor signalling Endocrinology 147, supplement 6, S50-S55

Hatch, E.E et al (2010) Association of endocrine disruptors and obesity: Perspectives from epidemiologic studies International Journal of Andrology 33, 2, 324-332

Heshka, S et al (2003) Weight loss with self-help compared with a structured commercial program: A randomised trial Journal of the American Medical Association 289, 1792-1798

Hetherington, M.M & Boyland, E (2007) Short-term effects of chewing gum on snack intake and appetite Appetite 48, 397-401

Jabr, R (2012) Gumming up appetite Scientific American February, p12

Julis, R.A & Mattes, R.D (2007) Influence of sweetened chewing gum on appetite, meal patterning adn energy intake Appetite 48, 167-175

Kagawa, M & Hills, A.P (2011) Secular changes in BMI and obesity risk in Japanese children: Considerations from a morphologic perspective The Open Obesity Journal 3, 9-16 (Download at http://www.benthamscience.com/open/toobesj/V003-SI0001TOOBESJ.htm)

Kensara, O.A et al (2005) Foetal programming of body composition: Relationship between birth weight and body composition measured with dual-energy X-ray absorptiometry and anthropometric methods in older Englishmen American Journal of Clinical Nutrition 82, 980-987

Klimentidis, Y.C et al (2011) Canaries in the coal mine: A cross-species analysis of the plurality of obesity epidemics Proceedings of the Royal Society B 278, 1626-1632

Kral, J.G et al (2006) Large nutritional weight loss from obesity

surgery prevents transmission of obesity to children who were followed for 2 to 18 years Pediatrics 118, 6, e1644-1649

Levine, J et al (1999) The energy expended in chewing gum New England Journal of Medicine 341, p2100

Marceau, P et al (2004) Outcome of pregnancies after bilopancreatic diversion Obesity Surgery 14, 318-324

Muhlhausler, B.S et al (2006) Increased maternal nutrition alters development of the appetite-regulating network in the brain FASEB Journal 20, 8, 1257-1259

Orpana, H.M et al (2010) BMI and mortality: Results from a national longitudinal study of Canadian adults Obesity 18, 214-218

Pankevich, D.E et al (2010) Caloric restriction experience reprograms stress and orexigenic pathways and promotes binge eating The Journal of Neuroscience 30, 48, 16399-16407

Pasarica, M et al (2006) Human adenovirus 36 induces adiposity, increases insulin sensitivity, and alters hypothalamic monoamines in rats Obesity 14, 11, 190501913

Powell, L.M et al (2007) Associations between access to food stores and adolescent body mass index American Journal of Preventive Medicine 33, S301-307

Rokholm, B et al (2011) Developmental origins of obesity - genetic and epigenetic determinants 3, 27-33 (Download at http://www.benthamscience.com/open/toobesj/V003-SI0001TOOBESJ.htm)

Shikany, J.M et al (2012) Randomised controlled trial of chewing gum for weight loss Obesity 20, 547-552

Simpson, S.J & Paubenheimer, D (2005) Obesity: The protein leverage hypothesis Obesity Review 6, 2, 133-142

Stein, A.D & Lumey, L.H (2000) The relationship between maternal and offspring birth weights after maternal pre-natal famine exposure: The Dutch Famine Birth Cohort Study Human Biology 72, 4, 641-654

Willig, A.L et al (2011) Birth weight is associated with body composition in a multi-ethnic paediatric cohort The Open Obesity Journal 3, 4-8 (Download at http://www.benthamscience.com/open/toobesj/V003-SI0001TOOBESJ.htm)

Xu, X et al (2010) Effect of early particulate air pollution exposure on obesity in mice: Role of $p47^{phox}$ Arteriosclerosis, Thrombosis, and Vascular Biology 30, 2518-2527

Young, E (2011) You may scoff New Scientist 24/31 December, 62-64