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Obesity – a natural consequence of human evolution

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Abstract: Obesity is considered a major epidemic of the 21st century. In developed countries, about 1/3 of adults are obese and another 1/3 overweight according to the oversimplified measure – the Body Mass Index. More precise indicators of adiposity: waist circumference, skinfolds, underwater weighing and absorptiometry indicate similar levels of fatness. Obesity per se does not necessarily lead to pathological states, nor to premature mortality. Recent results of large sample studies indicate that more than 1/3 of people classified as obese by fatness indices are physiologically normal. Others, however, suffer from a number of pathological conditions, common among them being the metabolic syndrome and cardiovascular disease. The classical explanation for increasing obesity is the positive energy balance – too much food intake and too little exercise. It seems, however, that this explanation is too simplistic. In societies, and in families, exposed to overeating and lazy lifestyles, about 1/3 of individuals have normal body mass and low levels of fatness, while others become obese. There is, therefore, individual variation in propensity for obesity. We have identified two specific variables differentiating fatness. People who have large lean trunk frames – large volumes of abdominal cavities and thus large gastrointestinal tracts – put on more subcutaneous fat than those with smaller trunk frames (Henneberg and Ulijaszek 2010). This may be a result of larger volumes of food required for antral extension to release ghrelin, or larger surface area of small intestines for food absorption. The second variable is concentrations of Alanine Transaminase, an enzyme responsible for conversion of an amino acid to a carbon skeleton that can be used in fat synthesis. Our study of 46000 young Swiss males (Henneberg, Rühli, Gruber and Woitek 2011) found consistent correlation between levels of Alanine Transaminase and body weight in groups of normal body mass individuals, overweight individuals and moderately obese individuals. Coupling this finding with the fact that among vegetarians, even those living in North America with overabundance of food and low levels of exercise, obesity and overweight are much less common than among non-vegetarians, we have now hypothesized that the increased obesity of modern affluent societies is a result of consumption of animal protein when energy needs are already covered by carbohydrates and fat consumed concurrently. Until the advent of agriculture, humans relied on consumption of a variety of terrestrial and aquatic animals supplemented by relatively small amounts of plant foods. In this situation our bodies became adapted to use proteins as a source of energy, and became efficient at storing occasional surpluses of amino acids by their deamination and conversion to fats. In the modern diets carbohydrates are abundant and provide, together with fats, energy required by human bodies, proteins after deamination are efficiently converted to fats. When new types of crops are introduced to mass production of cheap foods our bodies may not be able to react correctly to all their contents and some of the ingredients may cause additional fatness. An example of widespread recent introduction of industrially processed soybean products that correlates with prevalence of obesity across countries of the world is discussed.

Key words: adiposity, gut size, alanine transaminase, protein, vegetarian

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Introduction

Human obesity is now a problem worldwide; in 32 countries more than 25% of adults have Body Mass Index values in excess of 30 kg/m^2 (World Health Organisation 2013). Among those are countries of different levels of affluence, located on different continents and having different cultural backgrounds. For over 50 years dietary abundance and lazy lifestyle requiring less physical activity have been considered the main causes of this phenomenon (Armstrong et al. 1951) though now it appears also in environments that do not seem conducive to the accumulation of body fat (Sawaya 1995, Cabarello 2005). Although nearly a half of individuals classified clinically as obese because their BMI equals or exceeds 30 kg/m^2 are metabolically healthy (Ortega et al. 2013), the remainder develop serious challenges to their health: hypertension, cardiovascular disease, hyperglycaemia and dyslipidaemia as well as tendency towards cancer (Terry 1923, Gofman 1952, Chiang and Pearlman 1969, Kannel et al. 1979, Brown et al. 2000, Stein and Colditz 2004, Hsieh 2011).

It can be hypothesized that in terms of human biology causes of obesity are multitudinous and human propensity to accumulate excessive amounts of fat in their bodies has its roots in our evolution. Evolution proceeds by altering individual variation of organisms both in terms of their central tendencies and its

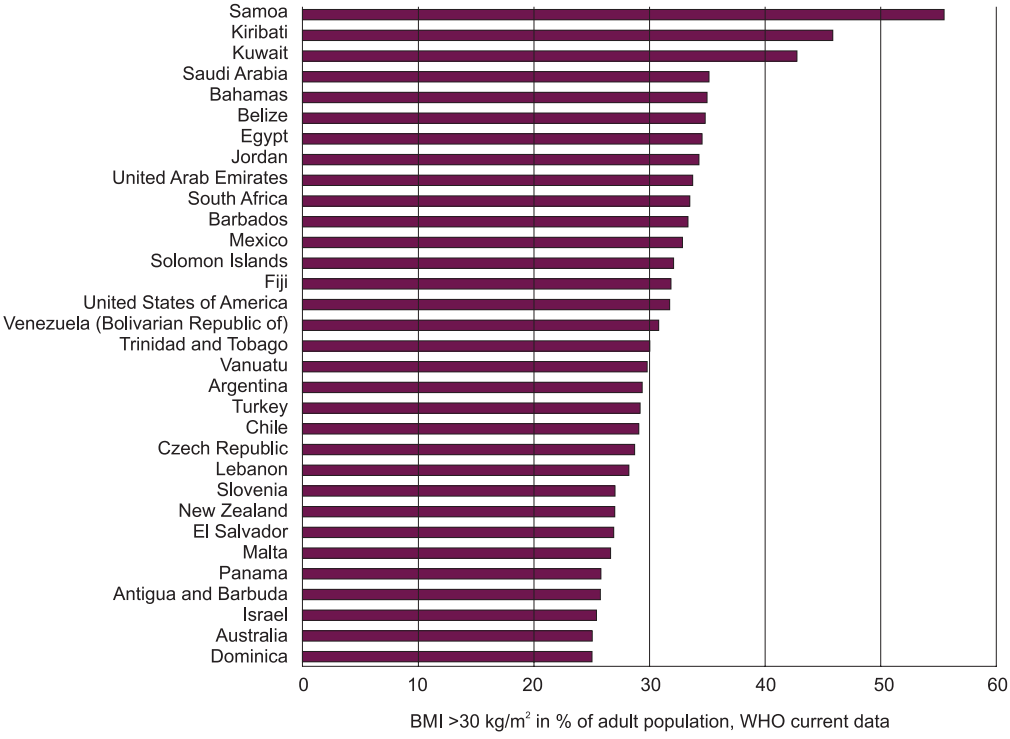

Fig. 1. The prevalence of obesity in those countries that have at least 25% of adults with Body Mass Index values exceeding 30 kg/m^2

ranges. Interindividual variation is the necessary condition for the operation of natural selection and a universal property of all living organisms. Thus variation in anatomical and metabolic characteristics that predispose people to excessive fatness needs to be considered. In the present day obesogenic environments some individuals develop excessive adiposity while others do not. In the last century the opportunity for natural selection decreased dramatically (Stephan and Henneberg 2001, Saniotis and Henneberg 2011) and thus many alleles on the over 17000 loci related to obesity (Kunej et al. 2012) may have accumulated in the situation of relaxed selection.

Human diet in the past

For the vast majority of the approximately 5 million years of hominin evolution our diet comprised what could be extracted from natural environments by gathering, scavenging and hunting. This is often labeled as "palaeolithic diet" and was significantly influenced by hunting (Thieme 2005). The main characteristics of this diet were: scarcity of carbohydrates, especially simple sugars, and reliance on protein from various sources – gathering of terrestrial invertebrates, fishing, scavenging and hunting of a variety of vertebrates. Fats do not occur in large quantities in plants, nor in wild animals. Thus our metabolic system adapted to processing animal protein as a source of energy. This requires a complex system of enzymes that break protein into peptides and then into separate amino acids that are deaminated to obtain their carbon skeletons that can be eventually broken down to pyruvate. Pyruvate can be converted into Acetyl CoA to obtain energy in the citric acid cycle. Any surplus of pyruvates may be stored as fats (*de novo lipogenesis*) while Acetyl CoA may also be metabolized into cholesterol.

Table 1. Comparison of human gut length and energetic value of food ingested with some other mammals. Data from various sources described in Henneberg et al. 1998

Animals	Gut length to body length	Energy intake (kJ/day/kg)
Ungulates	20:1	320–560
Baboons	8:1	230–300
Humans	5:1	120–180
Felids	4:1	50–90

In terms of the total energy of food consumed and the size of the gastrointestinal system relative to body size humans are closer to carnivores than to other primates (Henneberg et al. 1998, Table 1).

Human gastrointestinal tract is only about 60% of the size of that of a chimpanzee of the same total body weight (Aiello and Wheeler 1995). This testifies to human reliance on higher quality foods than those consumed by most mammals, including primates. Food quality of humans seems to be similar to that of carnivores. Cooking can be considered an extraoral digestion that increased food quality and has diminished the requirements of food processing by the human gastrointestinal tract (Wrangham 2009). Extraoral food processing also had obvious consequences for human dentition – its size decreased since Pliocene very considerably: from some 1550 mm^2 of the total surface of the occlusal area of all teeth in *Homo habilis* to about 850 mm^2 in modern agriculturists (Henneberg 2006).

However, the hunter-gatherer diets did not produce high levels of adiposity even when they contained large quantities of animal fat. A good example of this is provided by the comparison of

Table 2. Comparison of skinfolds of Canadian Inuits (Eskimo) and citizens of Toronto

Sex	Age Group	Canadian Inuits (Eskimo) Sum of skinfolds	Toronto Citizens Sum of skonfolds
Male	18–39	20.5	40.7
	40–54	21.5	43.3
	55+	22.2	47.5
Female	18–39	31.6	42.3
	40–54	39.1	49.5
	55+	45.9	59.2

skinfolds of Canadian Inuits (Eskimo) and citizens of Toronto shown in Table 2 (Shepard et al. 1969, Schafer 1977).

This table shows that the Toronto citizens are much fatter than their Eskimo counterparts, particularly amongst the men. These native Eskimo people had a diet much higher in protein and fat but much lower in carbohydrates. This shows that people without the "Deadly combination of carbohydrates and proteins" do not gain body weight so readily, despite consuming more protein and fat.

In a recent study, we found a significant relationship between the body mass and levels of Alanine Transaminase (ALT) in young, healthy Swiss males (some 46 thousand conscripts, Henneberg et al. 2011). This shows the link between the efficiency of aminoacid processing into carbon skeletons and accumulation of body mass. The Spearman "rho" for correlation of BMI and ALT in the whole sample was 0.395 (p=0.000) while it remained similar – 0.237 – and highly significant (p=0.000) when only "metabolically healthy" individuals (N=1341) were selected on the following clinical criteria: Systolic Blood Pressure<120mmHg, Glucose<7.8 mmol/L, Cholesterol<5.0 mmol/L, and Leukocytes<7100/μL.

Table 3. Association between the type of diet and body mass index. Data from Tonsad et al. (2009)

Type of diet	Mean BMI
Vegan	23.6
Lacto-Ovo Vegetarian	25.7
Pesco Vegetarian	26.3
Semi-Vegetarian	27.3
Non-Vegetarian	28.8

When human diets include both carbohydrates and proteins, as is the case in most cultures of the agricultural background, and especially in the so-called Western culture, carbohydrates consumed are quickly digested and used to provide energy in citric acid cycles, while slower digested proteins end up as surplus pyruvates that are converted to triglycerides and stored in body fat. This hypothesis finds strong support in a large study of diets and BMIs of North American Adventists (Tonsad et al. 2009) as well as in the EPIC-Oxford study (Spencer et al. 2003). In those studies among people of relatively similar affluence and education, vegetarians had the lowest average BMI values, while people eating mixed diets had the highest BMI averages (Table 3). This table shows that the more protein one eats, in conjunction with a high carbohydrate diet, the more body fat one gathers. This shows that people without the "Deadly combination of carbohydrate and protein" do not gain body weight so readily. Data from Tonsad et al. (2009).

Variation in gut size

The variation in the size of the gastrointestinal tracts, seen in the course of overall homin evolution, is still present among modern humans. Some of us have large gastrointestinal tracts, while tracts

of others are small. Stomach volume may vary from about 1 litre to 3 litres, whereas variation in the length of human intestines extends from 3.8 m to 13.2 m. The intestinal length is correlated with body weight, but not with height (Hounnou et al. 2002) indicating that body proportions, especially the width of the trunk in relation to height are indicators of gut size.

Variation in the size of the lean trunk, measured as bi-iliocristal hip width, biacromial shoulder width, chest depth from xiphoidale to iliospinale and chest width from thoracolaterale to thoracolaterale, correlates with variation in skinfold thickness on abdomen, in subscapular position and on triceps (Table 4) even when the possible influence of skin overlying these bony points is corrected for (Henneberg and Ulijaszek 2010). We have hypothesised that this correlation is a result of the larger trunk dimensions, especially those of the hips and the lower chest, that indicate larger size of the gastrointestinal tract. Larger gastrointestinal tract may facilitate better absorption of nutrients or require greater volume of food to cause antral extension of the stomach that will release amounts of ghrelin sufficient to produce sensation of satiety. Our initial observations on adults

Table 4. C*oefficients* of correlation between lean body frame dimensions and skinfold thickness when age is kept constant[1]

Variable	Females N=1250				Males N=61			
	Triceps skinfold	Subscapular skinfold	Abdominal skinfold	Sum of skinfolds	Triceps skinfold	Subscapular skinfold	Abdominal skinfold	Sum of skinfolds
Stature	0.04	–0.01	–0.03	0.00	0.08	0.00	0.15	0.09
	0.00[2]	*–0.06**	*–0.08**	*–0.05*	*0.12*	*–0.11*	*0.17*	*0.06*
Ankle circumference	0.40[3]*	0.34*	0.34*	0.45*	0.39*	0.38*	0.41*	0.41*
	*0.49**	*0.42**	*0.36**	*0.47**	*0.31**	*0.26**	*0.37**	*0.35**
Wrist Circumference	0.38*	0.38*	0.40*	0.49*	0.17	0.39*	0.49*	0.43*
	*0.45**	*0.47**	*0.44**	*0.51**	*0.30**	*0.42**	*0.53**	*0.48**
Biacromial diameter	0.21*	0.18*	0.23*	0.27*	0.36*	0.33*	0.47*	0.44*
	*0.25**	*0.22**	*0.22**	*0.26**	*0.43**	*0.35**	*0.52**	*0.50**
Biiliocristal diameter	0.37*	0.35*	0.42*	0.47*	0.36*	0.43*	0.43*	0.42*
	*0.45**	*0.45**	*0.47**	*0.51**	*0.26**	*0.47**	*0.44**	*0.44**
Chest width	0.49*	0.50*	0.48*	0.55*	0.46*	0.57*	0.58*	0.60*
	*0.51**	*0.54**	*0.53**	*0.59**	*0.32**	*0.51**	*0.55**	*0.55**
Chest depth	0.55*	0.60*	0.55*	0.64*	0.36*	0.56*	0.51*	0.54*
	*0.55**	*0.63**	*0.60**	*0.66**	*0.28**	*0.51**	*0.50**	*0.49**

[1] Australian Adults Study. Data collected in 2002 (Henneberg and Veitch 2003, 2005), analysed by Henneberg and Ulijaszek and published for females only (Henneberg and Ulijaszek 2010)
[2] Spearman rho in italics
[3] Underlined are coefficients in the cell where the rho indicates >10% of variance explained
* Statistical significance at $p<0.05$;
** statistical significance at $p<0.001$

Table 5. Coefficients of correlation between lean body dimensions and skinfold thickness for sexes combined[1]

Variable	Triceps skinfold	Subscapular skinfold	Abdominal skinfold	Suprailiac skinfold	Average skinfold	Average trunk skinfold
Body height	0.03	0.05	0.15*	0.15*	0.12	0.14*
Trunk length	0.04	0.08	0.10	0.10	0.07	0.09
Wrist circumference	0.33**	0.36**	0.39**	0.38**	0.39**	0.40**
Biepicondylar width	0.23**	0.32**	0.38**	0.37**	0.38**	0.37**
Biacromial width	0.22**	0.26**	0.26**	0.28**	0.30**	0.27**
Chest width	0.32**	0.32**	0.31**	0.27**	0.32**	0.33**
Biiliocristal width	0.42**	0.45**	0.45**	0.47**	0.48**	0.47**
Chest depth	0.40**	0.47**	0.46**	0.46**	0.48**	0.48**
Frame size	0.30**	0.33**	0.35**	0.36**	0.35**	0.36**
Trunk size	0.39**	0.42**	0.43**	0.43**	0.45**	0.44**

[1] Australian schoolchildren and youths aged 6-18 years (N=229). Data from an unpublished Honours thesis by E. Durdin 2007.
* statistical significance at $p<0.05$;
** statistical significance at $p<0.01$

were confirmed by studies of Australian (Durdin 2007) and South African schoolchildren (Lucas and Henneberg 2013).

New components of diets

Palaeolithic diets did not contain dairy products, nor considerable amounts of starches and refined sugars. Fresh milk contains, besides fat and proteins, significant quantities of sugar in the form of lactose. Most humans lose the ability to digest lactose after weaning and as adults do not tolerate lactose. Some individuals, however, have a gene that makes them lactose tolerant throughout their entire lives (Bersaglieri et al. 2004). This increase in genetic polymorphism occurred as a result of fast acting selection where fresh milk became a significant source of food. Similarly, the modern human genome contains more copies of the gene coding for amylase – an enzyme important for carbohydrate digestion – as a result of starches becoming staple food. Higher number of copies of the AMY1 gene is found in populations long exposed to starchy diets (Santos et al. 2012). These genetic adaptations to new components of diets, although produced by relatively fast selection, took a number of generations to be established. Steady supply of fresh milk and large quantities of starches became available as a result of the development of new food production technology in the Neolithic – animal husbandry and agriculture. Thus modern humans became fairly quickly genetically adapted to new technologies of food production. The effect of those adaptations is modern human ability to absorb kinds of nutrients that were not digestible, or poorly digestible, by earlier populations of *Homo sapiens*. Although this ability

could be beneficial in situations where there is a shortage of food, under conditions of overabundance of large scale food production they lead to overeating and its consequence – adiposity.

Although microevolutionary processes can adapt people to the use of new agriculturally produced foods, such adaptations take at least several generations, and as evidenced by lactase and amylase gene polymorphisms, do not adapt all individuals to the same degree, so that individuals vary in their responses to digestion of same quantities of same foods.

Soy is among crops whose production dramatically increased in the last decades. Traditionally, soybeans were used in Asian cusines where through fermentation they yielded such products as soy sauces and miso soups. Soy protein was converted into tofu. During the second part of the 20th century, soy became cultivated in large quantities by mainstream Western agricultural enterprises while its beans became industrially processed to obtain large quantities of soybean oil, soy flour, soy milk, soy protein and lecithin. Soy products permeated the industrially produced human food chain to the extent that they are even found in the finest Swiss chocolates and in pharmaceuticals. Since soy is produced and processed cheaply it became appreciated as a source of inexpensive fats – oil and margarines – and proteins, and as such it competes on the free market with animal proteins and fats. Like many other plant parts, soybean contains, in addition to potentially useful molecules, a quantity of potentially harmful substances such as phytoestrogens genistein and daidzein, and topoisomerase type II poisons (Yamashita et al. 1990, Hengstrel 2002). Soy also contains a number of phytates that may act as antinutrients and prevent absorption of zinc, iodine and other elements as well as the soy oil may be converted during heating to potentially detrimental unsaturated hydroxyaldehydes (Seppanen and Csallany 2006).

Widespread consumption of industrially processed soy products is statistically correlated with levels of obesity as reported by WHO using the BMI>30 kg/m^2 criterion (Roccisano and Henneberg 2012). Suffices it to quote from that work's Table 1 that the average prevalence of obesity in English-speaking "Westernised" countries (N=9) is 30.4% while in the European Union (including EEA and Switzerland, N=29) it is 14.1%. Gross Domestic Product (GDP) per capita and caloric consumption per capita in those two groups of countries are comparable (US$22,725 and US$31,120, 3127 and 3347 kcal per day per person). The only difference is that the "Westernised" English-speaking countries have soy consumption of 32 kg per capita per year, exactly double that of EU countries (16 kg per capita per year). Prevalence of obesity – 18.4% in the 20 poor countries of Latin America (GDP per capita US$ 8,135, caloric consumption per person per day 2640 kcal) exceeds that in EU countries, but the soy consumption per capita per year in those countries is nearly 30 kg. The exact mechanism through which soy consumption contributes to the increase in obesity is not known yet. It may be suggested that phytoestrogens of soy are the active factor as oestrogen is known to interact with fatness in female reproductive cycles.

Summary

Our metabolic adaptations to millions of years long interactions with the environment, even though modified by rapid mi-

croevolutionary changes adjusting us to the agricultural and pastoralist technologies of food production, did not obliterate variability in human responses to availability of various nutrients and did not adjust our organisms to vagaries of industrial mass production of new food items that may contain poorly known deleterious substances. Thus a substantial part of human populations reacts to the new nutritional situation in inappropriate ways developing excessive adiposity with its detrimental health consequences. While studying responses of our bodies to the new situation we should focus our attention on qualities of those individuals who, despite being exposed to obesogenic environments, do not become obese. These qualities should be considered desirable in all members of modern populations.

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Author contribution

MH drafted the text, JG read and altered it. Both authors contributed to the conceptual content of this paper and both provid.

Conflict of interest

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