



## Why so Fat ?

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### Abstract

The epidemic of severe obesity is a worldwide social burden, involving all ages and all ethnic communities. Among the possible reasons for the severe obesity epidemic, it might be considered a biological “adaptation” of the human phenotype to its genotype programmed in the Palaeolithic age for a daily energy balance usually above 3000 kcal/day. To test the hypothesis we compared 90 control subjects (60 F, 30 M, 23.9 ± 2.5 yrs, BMI 22.2 ± 1.7 Kg/m<sup>2</sup>, TEE 1913 ± 370 kcal/die) and 90 young severely obese patients (60 F, 30 M, 23.9 ± 2.4 yrs, BMI 47.5 ± 5.9 kg/m<sup>2</sup>, TEE 3049 ± 579 kcal/die), all recruited from a Southern Italy population.

Data show a mean TEE in severely obese patients slightly above 3000 Kcal/day, close to the one expected to correspond to our Palaeolithic genotype.

In the present human environment, severe obesity, at least in some cases, might be interpreted, in absence of other bio-behavioural compensation, as the natural phenotypic response to our ancestral genotype.

### Keywords

Severe obesity, Palaeolithic diet, Obesogenic environment, Adaptation

### Introduction

The epidemic of severe obesity (i.e., body mass index > 40 kg/m<sup>2</sup>), now involving > 5 percent of the adult US population [1], has almost reached, at least in this country, the prevalence of diabetes, thus becoming a real social burden [2].

When focusing on severe obesity an intriguing question is why so many people, and not only in the US, reach such a massive increase in body fat, apparently in the absence of any biological and/or cultural-behavioural control/inhibition?

The simplest and most frequently advocated explanation is that body fat, unlike protein and carbohydrate, stores are virtually unlimited and poorly regulated [3]. We would like to discuss a possible speculative explanation for the diffusion of severe obesity and the uncontrolled increase in body fat depots [4-7].

Our hypothesis goes back to the interaction between the human genotype, definitely determined in the Palaeolithic age, until about

150.000 years ago [8], and the dramatic changes observed mostly in the last 30 - 40 years in terms of demography, environment, life style etc, particularly in Westernized human populations [9].

The selection of the human genotype has been determined for a hunter - gatherer lifestyle typical of Homo Sapiens, until 10.000 years ago i.e., at the end of the Palaeolithic age, when the human population all over the planet counted – at its demographic peak, – about ten million individuals [10].

If we accept as reasonable the assumptions proposed for instance by Hambrecht [11] nowadays, with a human population of seven billion people mostly living in metropolitan areas [9], individual average energy expenditure per kg body weight should be about 40 percent that of our Palaeolithic ancestors.

As to food (i.e., energy) intake, Bellisari, Eaton and others [8,12-13], have reasonably reconstructed the average diet of Palaeolithic foragers. The most reliable Palaeolithic diet – again, the one shaped for our genotype – compared with current recommendations appears to have a high calorie and protein content about 3000 Kcal per day of which 30 - 50% represented by proteins, the main protein food source being wild game. “This energy intake (i.e., about 3000 Kcal/day) would approximately correspond to the expected energy requirements/expenditure of our aborigine/Paleolithic ancestors”. In terms of nutrient composition, this aborigine diet is low in saturated fats, simple sugars and sodium, vice versa rich in animal proteins, polyunsaturated omega 3 fatty acids, vitamins and minerals, in particular potassium and calcium. If compared to current western diets dietary thermogenesis by the Palaeolithic diet is certainly higher than contemporary diets. Furthermore in the wildness, the availability of fat (from animal source) and simple sugars (from wild honey for example) was rare and rather periodic or seasonal [14]. However, as a matter of fact, nowadays a high protein diet for the whole human population does not appear ecologically sustainable nor has indication [15-16]. In conclusion expected energy expenditure according to our ancestral genotype should be of at least 3000 Kcal/day, mostly due to the continuous and heavy daily physical exercise and, to a lesser degree, to the composition of this “hyperproteic” diet.

Nevertheless, the life of our Palaeolithic ancestors, besides the regular and intense physical activity, was also characterized, in at least some regions of the world and in the dry seasons of the year, by alternating periods of feast and famine [17-18] which may have

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**Table 1:** Individual characteristics, body composition, REE and TEE in 90 (60 females and 30 males) obese patients and in 90 (60 females and 30 males) normal weight subjects.

		Study population (n.180)			Males (n. 60)			Females (n.120)		
		Obese (n.90)	CTR (n.90)		Obese (n.30)	CTR (n. 30)		Obese (n. 60)	CTR (n. 60)	
		Mean (SD)	Mean (SD)	p	Mean (SD)	Mean (SD)	p	Mean (SD)	Mean (SD)	p
Age	years	23.9 (2.4)	23.9 (3.5)	0.882	23.9 (2.4)	23.9 (5.4)	0.975	23.9 (2.4)	23.9 (2.1)	0.839
Weight	kg	130 (21)	61.2 (76)	0.000	145 (22)	68.2 (7)	0.000	123 (17)	58 (5)	0.000
Height	cm	168 (8)	167 (7)	0.800	173 (7)	173 (6)	0.825	162 (6)	162 (5)	0.547
BMI	kg/m <sup>2</sup>	47.5 (5.9)	22.2 (1.7)	0.000	48.2 (6.4)	22.9 (2.2)	0.000	47.1 (5.7)	21.9 (1.3)	0.000
FFM	kg	65.3 (11.7)	45.0 (8.7)	0.000	76.6 (11.2)	54.7 (7.4)	0.000	59.7 (6.9)	40.1 (3.9)	0.000
FM	kg	65.1 (13.1)	16.3 (4.6)	0.000	68.0 (14.7)	13.5 (4.8)	0.000	63.7 (12.1)	17.7 (3.9)	0.000
FM	%	49.8 (4.9)	26.9 (7.7)	0.000	46.8 (5.1)	19.9 (6.8)	0.000	51.4 (4.1)	30.5 (5.4)	0.000
REE	kcal/die	2345 (446)	1472 (285)	0.000	2785 (413)	1792 (237)	0.000	2125 (261)	1311 (128)	0.000
REE/ FFM	kcal/kg	36.1 (4.2)	32.8 (3.1)	0.000	36.7 (5.3)	33.0 (3.8)	0.002	35.8 (3.6)	32.8 (2.8)	0.000
RQ		0.857 (0.033)	0.829 (0.139)	0.058	0.861 (0.027)	0.827 (0.167)	0.269	0.856 (0.036)	0.830 (0.125)	0.125
TEE	kcal/die	3049 (579)	1913 (370)	0.000	3621 (537)	2330 (308)	0.000	2762 (339)	1705 (167)	0.000

FM: Fat Mass, FFM: Fat Free Mass, REE: Resting Energy Expenditure, RQ: Respiratory Quotient, TEE: Total Energy Expenditure (REE × 1.3)

also conditioned our genotype versus an efficient nutrient/substrate utilization.

Nowadays our genotype “survives” in a totally different environment “i.e., the so called obesogenic environment”, which has heavily affected and modified our energy expenditure. Under such external pressures, besides the already referred dramatic reduction in physical exercise, the composition of our diet has also changed: more saturated fats and simple sugars, less proteins and omega 3 fatty acids, more “artificial foods [19] etc”. In other words, an increase in the absolute intake of fat, a poorly thermogenic nutrient, and a shift from proteins to simple sugars, for the more thermogenic ones. These changes have facilitated a number of individual adaptive (potentially pathological) responses, such as hyperinsulinemia, insulin resistance etc. in other words the metabolic syndrome. Severe obesity, we speculate, represents, in absence of an efficient cultural control, the natural, immediate phenotypic response to the conflict between human genotype and present life style in an overcrowded urbanized world [5-7,20].

“To test the hypothesis we have measured resting energy expenditure by indirect calorimetry in a group of young adult severely obese patients to verify if their daily energy expenditure corresponds, although in a totally different environment, to the one expected for our aborigine/Paleolithic ancestors”.

## Description

We selected, from our database of obese outpatients, 90 young severely obese subjects (60 females, 30 males) in a stable body weight since at least 6 months, age range 20 - 30 years, who underwent measurements of resting energy expenditure (REE) by indirect calorimetry (VMax Sensor Medics) and body composition was evaluated by bioelectrical impedance (BIA). Bio impedance analysis (BIA) was performed at 50 kHz (Human Im plus II, DS Medica) in standard conditions. Fat Free Mass (FFM) and fat mass (FM) were

estimated using the prediction equations developed by Kushner [21] this sample represents a quite young adult population but may well correspond to the middle age group of active hunter-foragers of the Palaeolithic age, as well as to comparable surviving aborigines. This sample of obese subjects has been compared with an age- and sex-matched group of 90 (60 F, 30 M) normal weight individuals, living in the same metropolitan area. All patients and controls are Caucasians living in Southern Italy.

Results are expressed as mean and standard deviation. The statistical analysis was performed using one - way ANOVA test (SPSS-PC vers.15) for differences in mean values between genders.

When compared with normal weight individuals, the obese group had markedly increased BMI (+115%), REE (+65%) and calculated TEE (+48%) values (Table 1).

When REE was corrected for fat free mass (REE/FFM) the increase observed, compared to normal weight individuals was only 11%. This last figure represents the adaptive increase in energy demands for unit of FFM due to the overall increase in body mass in severely obese patients.

If we multiply REE of severely obese patients by 1.3, a coefficient currently used for sedentary people, the expected total daily energy expenditure (calculated TEE) in obese individuals would be 3621 ± 537 kcal/day in men and 2762 ± 339 kcal/day in women, a value actually not too far from the one calculated for normal weight physically active Palaeolithic foragers as well as for living aborigines.

Obesity, in particular severe obesity, has reached an uncontrolled epidemic diffusion all over the world [22], despite intensive studies it appears still unsuccessful any intervention to reverse this trend in obesity prevalence. Failure to conservative treatments has been also interpreted as considering obesity a biological adaptation (with pathological consequences) to our obesogenic environment rather than a true disease [20,23]. On the other hand in potentially unhealthy

adaptations like this, intervention strategies should be aimed more at prevention than strictly to treatment, by reducing exposure to obesogenic environmental factors at least since childhood [24].

In this study we have explored the hypothesis that severe obesity represents a phenotypic adaptation of our genotype, selected in a highly demanding environment as was the one during the Palaeolithic pre-agriculture period, when exposed – in particular during the last decades – to an “obesogenic environment”. This environment is characterized by a dramatic reduction in physical activity [25], continuous exposure to highly caloric meals, an huge consumption of “artificial foods” like caloric sweeteners and sugar alcohols [19], exposure to chemical pollutants with endocrine disrupting capacities [26] etc.

The calorie expenditure of severely obese individuals, evaluated in this study and in agreement with literature data [27], appears to correspond to the one expected on the basis of our ancestral genotype but the calorie balance is obtained in a totally different way represented by a huge amount of fat mass, a consequent increase in fat free mass, metabolic syndrome onset with its complications, and a final increase in REE.

Severe obesity in this environment might well be interpreted, at least in some cases, as the consequence of the phenotypic (pathological) adaptation to our ancestral genotype in the absence of any bio-behavioural compensation. This could be one reason why, with an apparently unjustifiable mechanism, many individuals become so fat and are resistant to conservative treatments.

Effective educational preventive campaigns against obesity, starting since early developmental ages, associated with a continuous struggle against urban sprawl appear in our opinion an urgent policy to counteract chronic not communicable diseases, including severe obesity, epidemic.

## Conflict of Interest

All authors disclose any financial and personal relationships with other people or organisations that could inappropriately influence their work.

## References

1. Ogden CI, Carroll MD, Curtin IR, McDowell MA, Tabak CJ, et al. (2006) Prevalence of overweight and obesity in the United States, 1999-2004. *JAMA* 295: 1549-55.
2. [www.who.int/hpr/NPH/docs/gs\\_obesity.pdf](http://www.who.int/hpr/NPH/docs/gs_obesity.pdf)
3. Flatt JP, Tremblay A (1997) Energy expenditure and substrate oxidation. In: Bray GS, Bouchard C, James WPT (eds), *Handbook of Obesity*, New York, Marcel Dekker, 513-522.
4. Colicchio P, Tarantino G, Del Genio F, Sorrentino P, Saldalamacchia G, et al. (2005) Non-alcoholic fatty liver disease in young adult severely obese non diabetic patients in South Italy. *Ann Nutr Met* 49: 289-295.
5. Berghofer A, Pischon T, Reinhold T, Apovian CM, Sharma AM, et al. (2008) Obesity prevalence from a European perspective: a systematic review. *BMC Public Health* 8: 200.
6. Rabin BA, Boehmer TK, Brownson RC (2007) Cross-national comparison of environmental and policy correlates of obesity in Europe. *Eur J Public Health* 17: 53-61.
7. Lobstein T, Frelut ML (2003) Prevalence of overweight among children in Europe. *Obes Rev* 4:195-200.
8. Bellisari A (2008) Evolutionary origins of obesity. *Obesity reviews* 9: 165-80.
9. Drewnoski A, Popkin BM (1997) The nutrition transition: new trends in the global diet. *Nutr Rev* 55: 31-47.
10. Leakey RE, Lewin R (1977) *Origins*. London: Macdonald and Jane's, 1- 264.
11. Hambrecht R, Stephan Gielen (2005) Essay hunter-gatherer to sedentary lifestyle. *The Lancet* 366: 60-61.
12. Eaton SB, Konner MJ (1985) Paleolithic Nutrition: A Consideration of its Natural and Current Implications. *N Engl J Med* 312: 283-89.
13. Cordain L, Eaton SB, Miller JB, Mann N and Hill K (2002) The paradoxical nature of hunter-gatherer diets: meat-based, yet non-atherogenic. *Eur J Clin Nutr* 56: 542-56.
14. Cordain L, Brand Miller J, Eaton SB and Mann N (2000) Macronutrient estimation in world-wide hunter-gatherer diets. *Am J Clin Nutr* 72: 1589-1590.
15. Pimentel D, Pimentel M (2003) Sustainability of meat-based and plant-based diets and the environment. *Am J Clin Nutr* 78: 660-663.
16. Contaldo F, Pasanisi F (2006) High-protein diet, obesity, and the environment. *Am J Clin Nutr* 83: 387.
17. Contaldo F, Pasanisi F (2004) Xhooxham, the Young Lady of a Bushman Tribe, and “Globesity”. *Nutrition* 20: 949-950.
18. Diamond J (2003) The double puzzle of diabetes. *Nature* 423: 599-602.
19. Payne AN, Chassard C, Lacroix C (2012) Gut microbial adaptation to dietary consumption of fructose, artificial sweeteners and sugar alcohols: implications for host-microbe interactions contributing to obesity. *Obes Rev* 13: 799-809.
20. Chaput J P, Doucet E, Tremblay A (2012) Obesity: a disease or a biological adaptation? An update. *Obes Rev* 13: 681-691.
21. Kushner RF (1992) Bioelectrical impedance analysis: a review of principles and applications. *J Am Col Nutr* 11: 199-209.
22. Flegal KM, Carroll MD, Ogden CL, Curtin LR (2010) Prevalence and trends in obesity among US adults. *JAMA* 303: 235-241.
23. Tremblay A, Doucet E (2000) Obesity: a disease or a biological adaptation? *Obes Rev* 1: 27-35.
24. Egger g (2011) Obesity, chronic disease and economic growth: a case for 2 big picture “prevention. *Adv Prev Med* 149-158.
25. Ng SW, Popkin BM (2012) Time use and physical activity: a shift away from movement across the globe, *Obes Rev* 13: 659-680.
26. Tang-Peronard JL, Anderson HR, Jensen TK, Heitmann BL (2011) Endocrine-disrupting chemicals and obesity development in humans: a review, *Obes Rev* 12: 622-636.
27. Kee AL, Isenring E, Hickman I, Vivanti A (2012) Resting energy expenditure of morbidly obese patients using indirect calorimetry: a systematic review. *Obesity reviews* 13: 753-765.