THE HUMAN BIOLOGY OF OBESITY, AND ITS RELEVANCE TO INSURERS

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No human community functions without its insurance industry. People everywhere “insure their lives” by seeking marriage partners, or surround themselves with family members, with the longest possible life expectancy. Everybody concludes social and economic deals that take the mortality risks of the contractors into account. Age, sex, and body build have probably always constituted the corner stones of such risk assessments. The present review deals with one of these attributes — body build, especially obesity — and examines its value as a predictor of mortality to modern underwriters, in the light of what is presently known about its biology.

Causes of obesity

That Obesity is the result of food intake having exceeded the metabolic energy expenditure of an individual is a truism. It is also a platitude of singular uninformativeness. A person’s every physical feature is a result of positive energy balance: one’s muscle mass, bones and skin, the existence of limbs, the displacement of one’s brain and the volume of the packed blood cells. These self-evident truths contribute as much to the understanding of obesity, as they do about the prevention of phocomelia, the treatment of polio deformities, or the management of prostatic hypertrophy.

The human neonate converts about 10% of its solid food intake into tissue. This value decreases very rapidly, however, to less than 1% during the first 2 years of life. It remains at approximately 0.8% during most of the rest of childhood. To conclude from this that the energy budget will balance, and growth will stop, when the food intake of a child is reduced by 0.8% is clearly erroneous. Much greater reductions than 0.8% can be brought about merely by substituting brown for white bread in a child’s diet, or by withdrawing a favorite cool drink or condiment from the menu. In fact, most children in the world probably eat about 80 - 90% of what their counterparts in the U.S.A. eat, yet they grow. Nor are they obviously less active than American children. Often the contrary. Children in many Third World environments have to walk large distances to school, frequently after having first attended to a variety of arduous domestic chores, such as carrying water and firewood, or herding the cattle to distant pastures. But they remain in positive energy balance. Food-energy intake probably needs to be reduced to less than 20% or 30% of that eaten by Western middle class children before growth will stop in childhood.

The apportionment of the energy budget under various types of nutritional stress is, therefore, clearly a great deal more subtle and versatile than is generally appreciated.

Humans are unusual among land vertebrates in that they have considerable fat deposits under the skin. The human baby is born with it, and at 9-12 months it constitutes, on average, about 25% of the infant’s mass. Relative fat mass decreases during the second year of life, to average about 10% of total body mass through most of childhood. The fat organ increases in size during puberty in both sexes, but, more importantly, it changes shape. In boys it tends to thin out on the limbs and the limb girdles, while thickening in the loins to produce the torso of a typical young man. In girls there is an overall increase in the thickness of the subcutaneous fat layer during puberty, with particular emphasis on the hips, thighs, and, of course, the breasts. Interestingly, the fat layer in the loins tends to be thinner than it is in boys. When growth in height ceases at the age of 18 years adipose tissue constitutes, on average, 12% of the mass of boys (the normal, 95% probability limit, range is from 3% to 28%), and 24% of the mass of girls (normal range: 17% to 34%).

The remodelling of the body does not cease at 18 years. In both men and women fat and muscle continue to accumulate well into middle age. Connective tissue and skin probably never stops growing, except terminally. Indeed, the growth of the nose and ears, and skin of the face with age is well recognized by cartoonists, who invariably depict older people (especially women) with larger noses and ears than they depict younger people. But the hands and feet also grow, as does the skin all over the body, causing the characteristic creases and wrinkles of old age. In men the fat organ hypertrophies on the trunk, especially in the abdomen. In women the remodelling of this organ occurs particularly on the proximal portions of the limbs, the breasts, and, to a lesser extent, in the abdomen. All of these changes are associated with a positive energy balance of, on average, 0.5%, which tails off, in men, to zero at about the age of 50 years (though isoenergetic remodelling of the body continues unabated into old age). Most women remain in positive energy balance for an additional 20 years.

The average 25 year-old therefore weight 4 - 5% more than the average 20 year-old. At the age of 45 years the average weight of both men and women, on a wide variety of nutritional regimens, worldwide, is 20% higher than it is at 20 years.
Few other land animals carry as much fat on their bodies as people do, especially not under the skin. This type of body architecture is more characteristic of animals that spend much of their time in water, such as seals, penguins, ducks, dolphins, whales and hippos. A fat organ which envelops almost the entire body, just below the skin, is encountered in land mammals only among the pigs, bears, the musk ox, reindeer, and people. The evolutionary significance of this distribution is not known.

It is also not known when hominids first developed the subcutaneous fat layer. It does not occur in any of the other primates. The earliest surviving human art (e.g. the statuettes of Venus of Willendorf, and Venus of Lespugne) clearly indicates that a substantial layer of subcutaneous fat already characterized the mature women 30,000 years ago, when people subsisted predominantly by foraging and hunting. Today, though people have spread to every continent, and therefore eat diets that vary greatly, both in quantity and quality, the human fat organ develops remarkably uniformly. The same features distinguish the silhouettes of the maidens from the matrons in Zululand as they do in Sicily, and the youths from the elders among the Polynesians as they do among the Eskimos (Inuit).

The accumulation of fat (and muscle) during adulthood cannot be prevented by a reduction in food intake of only 0.5%. Far greater differences in food consumption occur cross-culturally, yet everywhere people utilize approximately 0.5% of their daily energy intake for the remodelling of the body contours. It requires reductions to well below 50% of the Recommended Daily Allowance (RDA) to remain in strict energy balance during adulthood, and thus prevent the “middle-ages spread” which is dreaded so much in Western societies.

Attempts at experimentally hastening the accumulation of fat in adults, have been uniformly unsuccessful. Ethan Sims and his coworkers in Burlington, Vermont, studied the effects of deliberate overeating on 4 student volunteers. The initial motivation for the experiment was to determine which of the blood biochemical deviations from normal seen in “spontaneous obesity” could be reproduced in “experimental obesity”; and hence to determine whether these deviations were the cause or the result of the obesity. Sims argued that since these students were in apparent energy balance (a 0.5% positive energy balance is undetectable, even over the course of a year), any increase in food intake would put them into substantial positive energy balance. Sims argued, furthermore, that all of the excess food intake would be deposited as fat.

Though each of the 4 men claimed to have eaten to tolerance (25 MJ/day as opposed to their normal intake of 12 MJ/day) for 3 - 5 months, none gained more than 12% on his starting weight. Sims had expected close to a 50% increase in mass.

As the researchers could not be certain that the students had not, unwittingly, increased their physical activity while overeating, permission was obtained to recruit volunteers for these experiments at the Vermont State Prison. Here it was not only possible to measure exactly how much was eaten, but also to monitor daily physical activity.

During the next 10 years Sims and his coworkers studied 19 men in the prison. Each subject was studied for an initial period of 6 weeks while he at his normal 8 MJ/day prison rations. After it was established that weight remained constant on this diet, the subjects were encouraged to eat as much as they could for 30 weeks. The average food intake per person during this period was 14 MJ/day, with some eating as much as 40 MJ/day for the entire period. (The normal RDA for young men engaged in light physical activity is 11 MJ/day.) However, the average increase in body mass was limited to 21%, all of which was gained during the first 2 months. There was no further increase in weight thereafter, despite continued overeating (even in those consuming 40 MJ/day!). The stout and lean men had equal difficulty in gaining weight (though the experimenters did not recruit grossly obese volunteers). The composition of the diet did not noticeably influence the total weight gained. The tissue gained in these, and similar experiments, consisted of 3 parts of fat to 2 parts of muscle. On cessation of the experiments all the subjects, students and prisoners alike, automatically returned to their original weights within a few weeks. They did not need to “diet” to lose the weight they had gained during the experiment.

Considering that the prisoners were eating nearly 35% less food than they would have eaten at home, (8 MJ/day in prison versus 10-12 MJ/day at home) it is likely that their 21% weight gain was the sum of the recovery from relative undereating in prison and the gain from overeating. If this interpretation is correct then it means that normal, middle class, young men (the experiments have not been carried out on women) can increase their body mass by not much more than 10% through deliberate overeating. The increase lasts only for as long as the person overeats, after it is rapidly lost.

This indicates that the total daily metabolic energy expenditure can be facultatively increased depending on the dietary intake. The adaptive advantages are obvious. Food quality and quantity vary, for all animals, in time and space. In some locations (or during certain seasons) food quality may be very high, and a relatively small intake will provide all the protein, minerals and vitamins needed for health. In other locations (or during certain seasons) food quality may be very low, and a relatively small intake will provide all the protein, minerals and vitamins needed for health. In other locations (or during certain seasons) food quality may be very low, and a relatively small intake will provide all the protein, minerals and vitamins needed for health. This would be disastrous if the excess food-energy could not be burnt away under these circumstances, but accumulated inexorably as fat. Conversely, it would be equally deleterious if the metabolic rate could not be reduced during periods of famine. Indeed, if the metabolic rate was unadjustable then three quarters of the world’s population (including the inmates of Vermont State Prison), would be in perpetual negative energy balance. This is clearly impossible. Professional ballerinas, in fact, regularly consume diets which supply less energy (less than 6 MJ/day) than the textbooks day is required for basal metabolism, yet they live in continuous dread of gaining weight, despite being engaged in one of today’s physically most strenuous professions.
Definition of obesity

Seen from a broad biological perspective, being human means being obese. Our subcutaneous fatty layer is not only biologically unusual, it also seems to be superfluous. There appears to be no good biological reason why we could not have been created with the body composition of a zebra, or a vervet monkey, both of whom have shared the African plains with people since the common ancestor of all humankind lived there over 200,000 years ago. There is certainly no reason to suspect that a breed of humans totally lacking in subcutaneous fat would be at a selective disadvantage to the present 100% “obese” variety.

More commonly, however, it is not the fat-free zebra or vervet monkey that is presented as our biological ideal, but the 12% and 24% body fat contents of postpubertal boys and girls respectively. (Indeed, “Desirable Weight Tables”, in use today by the medical profession and beauty specialists, are based on this ideal.) These amounts of fat are more than adequate for accurately distinguishing the two sexes, and they are associated in many people’s minds with the exuberance, and apparent indestructibility, of youth. By these criteria, obesity changes from a general human characteristic to a disease primarily of middle age, which affects just over half population between the ages of 40 and 60 years.

The incidence of obesity is, however, arbitrarily halved if the ideal is not the postpubertal boy and girl, but the fully mature 30 year-old man and woman. The “disease” vanishes altogether in societies that venerate the experience and discernment of middle age.

The dispassionate view, on the other hand, does not accept arbitrary, ethnocentric ideals as bases for biological definitions. If humans walk upright, then it is “normal” for humans to have a bipedal gait. If the hair on people’s heads turns grey with age, then it is “normal” for human hair to become grey with age! If babies are born enveloped in an almost uniform layer of subcutaneous fat, then that must be normal for human babies. A baby born without subcutaneous fat is abnormal. A baby born with more fat than is usual for humans is also abnormal. It is similarly normal for people to gain weight during adulthood. By these criteria, it is only the 2.5% of persons at the upper extreme of their age-, and gender-specific weight-for-height distribution who may legitimately be termed “obese” or “over-weight”.

A very small proportion of this obesity (usually referred to as “morbid obesity”) is caused by neurological lesions in and around the hypothalamus, or by unusual endocrine abnormalities. These causes are, however, exceedingly rare, and can safely be ignored. The remaining cases of obesity are predominantly of constitutional origin. The unfortunate genetic lucky-dip draw which endows some people with more (and other people with less) than their fair share of fat-genes. Indeed there are few risk factors (e.g. occupation, habits, body build, blood biochemistry, blood pressure and many illnesses) which rival age as predictor of impending death. Thus, shuffle-board players have a higher mortality rate than American football players, and university professors are less likely to survive their next birthday than are their undergraduate students.

It is not only the risk of mortality that changes with age, but also the spectrum of diseases which can affect the individual. Hence parents of teenage children have a higher incidence of hypertension and heart attacks than the parents of toddlers. The incidence of cancer, non-insulin dependent diabetes mellitus, gall-bladder disease, degenerative lung conditions, gout, diverticulitis, and cerebrovascular accidents is considerably higher among Caucasian men who comb their hair with a parting than among those who do not.

The often quoted observation that obesity is “clearly associated with increased mortality, hypertension, hypercholesterolemia, non-insulin dependent diabetes mellitus, various cancers and other medical problems,” may, therefore, be nothing more than a re-affirmation of obesity’s occurrence predominantly in older people. If this is true, then, surprisingly, the above observation (that obesity is associated with hypertension, diabetes etc.) implies that people who die from obesity and its “complications”, have lived longer (i.e. are older) than those who die of other causes. (This conundrum loses its mystery if the words “grey hair” are substituted for “obesity” in this paragraph.)

Comparisons of health hazards across age groups are therefore calculated to mislead. Misrepresentations are sure to arise if epidemiological data are not meticulously categorized by age, or if comparisons are not obsessively confined within age classes.

Since the mortality rate of people under the age of 30 years is exceedingly low (less than 1 per thousand per year in modern industrialized countries) the differences between the mortality risks of lean and chubby young people can only be evaluated in extremely large populations. Surveys of the required magnitude are therefore generally beyond the scope of academicians, and reliance has to be placed on the pooled statistics of the American Life Insurance companies. Though far from ideal, these statistics are our only source of information about the effects of body build on mortality in young adults. They show that the risk of death is lowest in persons with the average build for their age and sex. Mortality is approximately 20% higher at both extremes the normal range (i.e. the 95% probability limits).

Studies of the health and mortality risks of obesity in middle age abound, because deaths occur at least five times more frequently than in 20 year-olds. Two types of result have been reported:

1. The risk of death is lowest in persons with the average build for their age and sex.
2. The heaviest (most “overweight”) risk dying.

None of the studies support the claim that corpulent, middle
aged people have higher 10 and 20 year mortality rates than lean persons of the same age, and sex. Indeed the opposite is true. 4, 19-20, 23 This remains true even after the data are corrected for the slimming and mortality effects of cigarette smoking. 20

The investigations which show that mortality is negatively correlated with weight (or, with fat mass) in middle age (i.e. that "overweight" middle aged people live longer than similarly aged average or lean people do) have generally involved 5,000 or fewer subjects, who were thoroughly screened at the beginning of the study to exclude pre-existing disease. The studies involving close to a million or more subjects have generally been more representative of the ambulatory population at large, in that there was little or no exclusion on the grounds of pre-existing disease. In these surveys the risk of death was lowest in persons with the average mass for their age and sex. Together, these investigations show that it is not only normal to gain weight during adulthood, but that it is also healthy! The ideal body mass for a 20 year-old is, therefore, not the ideal body mass for a 45 year-old. The latter is ideally 20% heavier than the average 20 year-old. Indeed, if there are no clinical signs of disease, then it would seem that it is healthiest for middle aged persons to be as much as 50% heavier than the average 20 year-old. 4, 19-20, 23

Statistics on the effects of body build on mortality in the over 65 year-old age category are scant. The available evidence indicates that body build has no influence on mortality in this age group. 2

Morbidity Risks

Interest here clearly focuses on coronary heart disease. Insurance company statistics have generally indicated that it is more common in stout than in lean persons. However, it is well-known that such prophesies tend to be self-fulfilling in the insurance industry. Thus, if a category of persons has to pay extra premiums because these individuals are considered to be poor insurance risks, then such people generally do not take out insurance. Those who do apply for insurance, under these circumstances, usually have special reasons (which make the extra premiums seem worthwhile) for doing so.17 Thus, earlier this century, insurance companies charged women extra premiums for life insurance. This was because their data showed that women were poorer insurance risks than men. Yet, it is now well known that women have always had longer life expectancies than men. Clearly the insurers were attracting only the poor risk cases when they charged women extra for life cover, thus confirming their preconceptions.17

Non-insurance data shows that the body mass indexes (BMI = body mass/height2) of US soldiers under the age of 40 years who died of myocardial infarctions were no different from those of the men killed in action, or through accidents.17 In the New England town of Framingham the incidence of episodic pain in the chest (presumed to be of coronary origin) was more common in stout than in lean persons of the same age.120 But neither myocardial infarction nor death from that disease was significantly correlated with body mass within age-categories. Similar results characterize the majority of population studies carried out elsewhere in the USA, and in Europe.17 The common view that coronary heart disease is an affliction of stout people must therefore be another example of comparisons across age-categories.

The conclusion is obvious. To avoid death or myocardial infarction in middle age, it is best to be at least 20% heavier than the average 20 year-old. In terms of the ubiquitous, so-called "Desirable Weight Tables", this means that to enjoy optimal physical health, the middle aged man or woman should be comfortably "overweight" (BMI equal to, or greater than, 26 kg/m2).

Acknowledgments

This review was adapted from 2 articles written on this topic for the South African Journal of Continuing Medical Education, with permission. The research was supported by the Staff Research Fund of the University of Cape Town.

References


