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Review article

Obesity, waist–hip ratio and hunter–gatherers

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Obesity is a rapidly growing global problem. It is not simply the result of eating too much, and not all types of obesity have the same significance. Obesity is in part genetic, and one particularly important genetic type of obesity is the tendency to 'truncal obesity',—that is, a raised waist-to-hip ratio. Such obesity is powerfully associated not only with a tendency to diabetes, but also to cardiovascular disease, ('Syndrome X'). Interestingly, this is the type of obesity seen in every hunter–gatherer (HG) population around the globe. Such people are intolerant of carbohydrate, especially refined carbohydrate, especially in the excessive amounts typically consumed in affluent societies. In such pure HG communities, rates of diabetes can be as high as 50%, when the 'Western' lifestyle is adopted. Many of us, however, share some of their genes and their carbohydrate intolerance—perhaps as many as 20 or 30% of the world's population. Pregnancy can uncover this characteristic, and obesity and glucose intolerance in pregnancy are rapidly burgeoning problems. Quite contrary to the common nutritional dogma of encouraging regular carbohydrates, it is suggested that pregnant women with a high waist-to-hip ratio should be strongly advised to adhere to a low-glycaemic-index diet. Additionally, many dietary interventions, some of them derived from observation of HG populations, are of proven benefit in reducing the expression of glucose intolerance and may well help in tackling the obesity epidemic.

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Obesity is a growing problem

Obesity is a serious global epidemic. To quote the European Taskforce:[1] 'Estimates suggest that 1.1 billion adults are overweight, 312 million of whom are obese. The prevalence of obesity has doubled or tripled in less than two decades, while in children this is rising at an even faster rate'. The same problem is appearing in all developed countries.[2,3] Amidst a host of complications that result, of particular concern to this article is the effect of obesity and glucose intolerance in pregnancy.

Obesity is not just eating too much

Some people are fat because they eat too much and exercise too little. But is all obesity of one type, related to this one combination? And is there just one solution? Why are some people like stick insects, whatever they eat? Why does weight not vary for some people, despite dietary fluctuation? In famine, why do some children develop marasmus more quickly than others? Why are women with polycystic ovary syndrome (PCOS) often plump? Most interestingly, why is truncal obesity of such distinct prognostic significance, compared to more generalised fatness?[4,5]

Clearly, the calories-to-exercise ratio is only part of the story, and later in the article the idea is explored that the ability to burn off excess calories as heat is an important safeguard against obesity. In part, this is genetically determined, and in part it can be influenced by what we eat. Thus certain types of calories in certain people may be doubly damaging—the burden of the calories, plus the metabolic effect of that food in slowing heat production.

Obesity is in part genetic

Much is known in relation to the causes of obesity, but many pieces of the jigsaw are still missing. It is clear that some of the factors have a genetic origin, which might find expression in a number of ways: some people may for instance have genetically determined modification of the obesity-related structures—for instance, the hypothalamic hunger centre or satiety centre.[6] Production of enzymes is in part genetically determined, and some enteric hormones influence obesity e.g. cholecystokinin produces satiety,[7] and ghrelin stimulates appetite.[8] There is a complex interaction of glucagon, insulin and blood sugar level that has a profound effect not only on satiety, but also on metabolism.[9,10]

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Other more subtle genetic factors influencing glucose tolerance include control of the 'carboxypeptidase E' system. This enzyme is necessary for the breakdown of neuropeptide Y,[11] and genetic mutations are associated with middle-age, truncal obesity and glucose intolerance. Chromosome 19 is increasingly quoted as being the locus of some of the hereditary tendency to glucose intolerance.

Leptin production is also in part genetically determined. Leptin is made by adipocytes—the more fat you have, the more leptin you produce.[12] Leptin stimulates 'nucleopeptide Y', which suppresses appetite.[23] Importantly, it also stimulates an increase in basal metabolic rate (BMR). A relative paucity therefore leads to the genetic tendency to obesity. Production of the leptin receptor is also genetically mediated.

Types of obesity, waist–hip ratio, and hunter–gatherers

Furthermore, not only is it the case that women may be genetically programmed to be obese, but also that the programming might be for one of two main types of obesity: general fatness of female distribution (breasts, bottom, thighs, upper arms) or alternatively 'truncal obesity'. Truncal (also called 'central') obesity is where the fat deposition is mainly around the abdomen and the viscera. This produces a large waist in relation to hip size—i.e. a large waist-hip ratio (WHR).[13,14]

It is now abundantly clear that such a distribution of fat is associated with both diabetic tendency[14,15] and with increased cardiovascular mortality,[16–20] ('Syndrome X'[21]). The evidence is also powerful that such a tendency is in part genetic[22,23] and that even *in utero* influences begin to exert an effect on expression of the tendency.[24,25] Indeed, whole populations can share this tendency. HGs, for instance, are mainly not obese when they live their traditional lifestyle. When such people are exposed to high, refined carbohydrate intake; however, they develop truncal obesity and a vastly increased risk of diabetes—up to 50% in some populations. This tendency to a high WHR and to carbohydrate intolerance is shared by all hunter–gatherer (HG) populations throughout the world: Canadian Inuit, Native Americans, Mexican Indians, Pima Indians, South American Indians, Middle-Eastern Nomads, African Pygmies, Australian Aborigines, Maoris, South Sea Islanders, etc..[26–43]

Evolutionary and carbohydrates

Homo sapiens evolved into its modern form 100 000 to 200 000 years ago. At that time, all of the species were HGs. About 20 000 years ago, agriculture was begun, and more stable populations evolved, with (presumably) different genetic tendencies favoured. Some populations stayed as HGs (and have continued as such to this day), but the 'agriculturalists' began to grow cereals as well as herd livestock. For these people, the amount of carbohydrate in an average diet increased from very little, to about 30% of the dietary energy intake.[44–51] Over the centuries, as populations grew, this proportion gradually increased for the agriculturalists because cereal staples could be grown cheaply, and stored, and so would help stave off starvation for the poorest people in these cultures.

However, for the well fed, it is only in the last half century that there has been a sudden explosion in eating carbohydrates as the major component of the diet. Not only that, but the 'carbs' are now being eaten in a much more refined form and in excess of need.[52] Even vegetables such as pea and carrot have been bred to be sweeter.[15] In evolutionary terms, the pace of change is staggering. For instance, between 1990 and 2000, the amount of potato in the UK, which was processed to produce starch (to make crisps, etc.) increased from 50 to 90%.

The result has been a major challenge to the human genotype—and much more of a challenge to those who are genetically HGs. Few of us can even trace our origins three hundred or four hundred years, so it would be folly to assume that because we do not hunt and gather now, that we are not of that genotype. The evidence is in the waistline: the average waist size of a female in the reproductive years went up by 8 inches between 1950 and 2000.

HG genotype, PCOS, and evolutionary advantage

What could be the evolutionary value in glucose intolerance and truncal obesity? It is important to remember that HGs naturally eat very little carbohydrate. If, eating carbohydrate were to 'turn down the thermostat' of their metabolism, they would become energy efficient—the 'thrifty genotype'.[53,54] Deposition of truncal and visceral fat would then lower the energy needs. This might be useful if the next meal were 20 miles away. If this meant that they did not produce as much heat, this may well not matter in many climates. Presumably there might also be related advantages to turning up the metabolic burner after a fatty meal.

It is interesting to speculate whether women with polycystic ovaries—who form 20% of the population—are a subgroup of HGs! Certainly, they share the tendency to truncal obesity, glucose intolerance, and increased cardiovascular risk. If, indeed, PCOS is part of the same genotype, then it can be imagined that the tendency not to menstruate or be fertile in times of plenty would be an advantage, if such women became fertile in times of meagre food supplies, when the less energy-efficient women were beginning to become amenorrhoeic.

The inescapable fact, however, is that in the 21st century, at least one-fifth of women (and quite possibly many more)

have a genetic tendency to glucose intolerance and yet live in a culture of refined carbohydrate bombardment.

Diet, refined carbohydrates, and truncal obesity

Therefore, it is possible to be genetically programmed to have truncal obesity—given the right environmental circumstances—and this tendency can be counter-balanced by the systems in the body which, if activated, can suppress appetite, increase BMR, and favour wasteful heat production.

It may be that many environmental and lifestyle factors could influence these systems—e.g. smoking, stress, exercise, and alcohol consumption. Of vital interest, however, is the effect of diet on heat production and thus on the expression of truncal obesity.

The Atkins diet[55] famously postulated the severe view that complete exclusion of carbohydrates (inasmuch as that is possible) has a profound effect on metabolic 'wasting' of excess energy—so profound in fact, that dietary fat can be eaten with alacrity. Too much carbohydrate in the diet is bad for some people, especially those with a tendency to glucose intolerance.[56] Furthermore, reduced carbohydrate in the diet has been shown to have beneficial effects in certain circumstances.[57–64]

But carbohydrate has less than half the calories of fat, gram for gram, so how can it be more fattening?

Carbohydrates, exercise, and heat production

The answer lies in the effect of carbohydrates on heat production. It is incontrovertible that exercise is a vital and effective part of any weight-loss programme.[65] Take in 3000 calories and burn off only 2000, and the rest will be stored as fat. Exercise is an important stimulant to this burning. However, regular gym visitors find it dispiriting to realise that the 20 minutes just spent on the rowing machine barely burns enough calories to cover the last packet of crisps.

However, exercise has an added effect on calorie consumption, over and above the calories used in the activity. Aerobic exercise promotes an upregulation of metabolism. The work itself produces heat as a by-product, which the body dissipates by peripheral vasodilation. However, the vasodilation does not necessarily close down immediately. As heat continues to be lost, the body is capable of short-circuiting the need for energetic exercise by allowing fat to be burnt directly to heat production, rather than to production of high-energy phosphates.[66,67] This occurs in infants, for instance, by activation of 'mitochondrial uncoupling protein' in brown adipocytes (under adrenergic stimulation).[68] This is a considerably more wasteful use of the energy in fat than providing energy for work. By analogy, a litre of petrol will take half a ton of car 5 or 6 miles. Thrown on a fire, however, the same litre will produce a very temporary burst of heat.

Thus, exercise is an important trigger to the key element of weight control—heat production. Anything, which influences this metabolic process will also affect weight. Thus, dietary carbohydrate in HGs might not only cause the tendency to obesity (and glucose intolerance) by excess calories, but also by a downregulation of metabolism.

Carbohydrates and glycaemic index

It seems unlikely, however, that all carbohydrate is always bad, for all people. Certainly, it has long been held that a healthy diet should include five portions of fruit and vegetable,[69,70] and many fruit and some vegetables are rich in carbohydrate. However, the effect of the carbohydrate in its complex, natural form on insulin metabolism is dependent on the type of carbohydrate. Oats and pearl barley, for instance, have less readily available carbohydrate, and so are said to have a lower 'glycaemic index' (GI).[71] Even the species of plant can make a difference—the (durum) wheat used for pasta has a lower GI than that used for bread, for instance. The type of food processing also influences the GI—wholemeal bread, for example, has almost the same GI as white bread, because the preparation of the flour makes the carbohydrate more readily available. Whole-grain bread, however, has a significantly lower GI, and also contains natural vitamins, of which grains are an important source.[72]

A study of the GI of foods can be surprising and enlightening. The ubiquitous potato, for instance, comes off badly. When peeled, it adds very little to the diet except energy and stomach filling[73,74]—and carbohydrates filling the stomach do not produce the same degree of satiety as fats.[75] When baked in its jacket, the situation is no better. True, it is low in fat, but the vitamins are destroyed, and the GI significantly elevated by the baking, such that the result is almost pure starch.

It seems very sensible to suggest, therefore, that women with a tendency to glucose intolerance should limit the total amount of carbohydrate they eat, and what they do take should be of a complex, whole-grain,[76] low-GI form.

Dietary manipulation and glucose intolerance

Interestingly, it is not only the GI of food, which can affect glucose tolerance. Numerous foods and supplements have been shown to have significant benefit in reducing insulin resistance in susceptible individuals. Much information has come from studying the diet of HGs, which is naturally anti-diabetic.

HGs often eat fish as a major nutrient, and fish oil contains varying amounts of omega-3 fatty acids,[77–79] (n-3FA), which

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seem to decrease insulin resistance and increase BMR. N-3 FA are types of poly-unsaturated fatty acids, but other fats can also affect glucose tolerance—e.g. substituting mono-unsaturated fatty acids for carbohydrates in the diet reduces insulin resistance.[80–82] Given the multifaceted nature of insulin resistance, if any single intervention can be shown to make a difference by itself, then that intervention should be worthy of close scrutiny, even if it is a nutrient.

Chromium (Cr) seems to be a particularly important supplement for those with insulin resistance. Dietary Cr intake is poor in the UK (few people achieve the recommended allowance of 50 mg per day) and a diet high in refined carbo hydrate (CHO) causes a large increase in Cr excretion. Supplementing Cr in such people significantly improves insulin resistance.[83–90]

Soluble fibre in foods binds CHO in the gut and thus improves glucose metabolism.[15] This may be the mechanism whereby certain foodstuffs have proven to have anti-diabetic qualities, despite the CHO content. Examples include Korean ginseng,[91] flax seed,[92] fenugreek,[93] and many other plant products.[94–100] The addition of folic acid and anti-oxidants to the diet have benefits not only for CHO metabolism, but also for the raised CV risk of glucose intolerant people (e.g. by reduction of homocysteine levels).

Some natural diets put many of these elements together—for instance the 'Mediterranean diet'[101]—and this significantly benefits cardiovascular mortality.[102] Fish seems to be a particularly important component of such diets,[103] and meat fat seems to be bad.[14]

Does it matter?

If some parts of the population have a genetic tendency to truncal obesity and glucose intolerance, which finds expression when they exercise little and eat the wrong foods, does this matter?

Emphatically, yes! It matters firstly to the woman's long-term health. Her life expectancy is considerably truncated in proportion to the degree of truncal obesity.[104,105] The extra weight carried causes additional health problems such as degeneration of joints and complications of surgery.

It matters further because glucose intolerance is related to significantly higher levels of infertility and miscarriage, both of which improve with weight loss.[106–112]

And it matters because perinatal mortality is considerably increased in the presence of glucose intolerance.[113] Fetal anomaly[114] is only one aspect of this—in the West Midlands in 2004, one-third of perinatal deaths occurred in women with a body mass index over 30,[115] (22% of the population—and rising). We simply do not fully understand the effects of borderline,and often undetected, glucose intolerance on the metabolism and development of the fetus.

Summary

So the message is clear: a high WHR is common, is in part genetically determined, is associated with glucose intolerance, which is exacerbated by pregnancy,[116] and is modifiable by lifestyle interventions. It is made worse by high, refined carbohydrate in the diet and by lack of exercise, both of which are endemic in the UK.

Allowed to go its own way, it is bad for reproductive health, for any children a woman may bear, for her life expectancy, and for her general health.

What can be done?

I make a simple suggestion. Adult women should measure their WHR, and should have it measured for them at booking antenatal visits and other appropriate opportunities. The higher it is above the accepted upper limit (0.85), the more they should be advised to be on a low-GI diet (which has no known downsides, except in famine). They should restrict the total proportion of carbohydrate in the diet, and what carbohydrates they do eat should be in the complex, whole grain, low-GI form.

Glucose intolerance should be screened for in pregnancy,[117] and if found, it is worth considering supplementation with omega-3 fatty acid, chromium, and folic acid. Other supplements are also quite possibly of benefit, including garlic, Korean ginseng, and flax seed oil. (Some proprietary preparations, e.g. Vitabiotic's 'CardioACE'—contain many of these ingredients—n-3FA, folic acid, garlic, chromium, anti-oxidants, etc.). It would also help women to know which fruit and vegetables were relatively 'anti-diabetic' (e.g. parsnip and turnip), and which were pro-diabetic, e.g. potato.

However, the message can be put more simply: if you have a fat waist, then cut the carbs! ■

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