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Scientific article for use with Question 7

A source of 'thinspiration' – *media, body image and dieting* – but can your own body fat make you thin?

1. Hoping to shift a few pounds? You could subject yourself to a gruelling exercise regime, try the latest fad diet (anyone for cabbage soup?), take the newest blockbuster weight-loss pill or . . . simply chill out in the fridge for a couple of hours a day. Recent findings have suggested that specialised fat stores known as brown adipose tissue (BAT), which are activated by the cold, can help control body weight and may be a target for new anti-obesity therapies.
2. Obesity really is a big problem – currently in the UK almost two-thirds of adults and one-third of children are overweight or obese. The treatment of obesity is estimated to cost the NHS £4.2 billion per year.
3. Obesity is defined as a surplus of body fat which is detrimental to health. This fat, or white adipose tissue (WAT), is located underneath the skin and around the internal organs and stores excess energy in the form of triglycerides. WAT located around the abdomen (giving rise to the 'apple' body shape) is considered more dangerous than fat stored around the hips and thighs as it is strongly correlated with type 2 diabetes, heart disease and certain types of cancer.

The media and body image

4. Over the past four decades women in industrialised countries have become wider, yet the ideal female physique, as depicted by the media, has become thinner. Twenty-five years ago, the average fashion model was only 8% thinner than the average woman. Today that figure has risen to 23%.
5. More worryingly, body dissatisfaction is now appearing at increasingly younger ages. In a study by University of Central Florida, nearly half of the 3 to 6 year-old girls said they worried about being fat. Around one in three would change a physical attribute, such as their weight. The use of diet pills among 15-17 year olds has doubled in a five-year period. Female preoccupation with physique and body dissatisfaction does not appear to diminish across the age spans and is now even exhibited by pensioners.
6. Yet body dissatisfaction is more than a psychological and cultural issue. The methods used by an increasing proportion of girls and women to alter body shape are more than mere stylistic exercises in aesthetics. Body dissatisfaction has serious implications for female health and is a major risk factor for a variety of disorders. As body dissatisfaction rises in increasingly younger children, so too has the prevalence of eating disorders and dieting. In the past decade, there has been an 80% rise in the number of young girls admitted to hospital with anorexia in England with a mortality rate of between 10–20 per cent.
7. A review of 25 studies entitled 'The effect of experimental presentation of thin media images on body satisfaction' concluded, as have others, that 'body image was significantly more negative after viewing thin media images. This negative effect is frequently reported to be both strong and immediate.
8. Analysing the brain activation patterns of females being exposed to media images is now illuminating the biological landscape of body dissatisfaction. Neuroscientists at Brigham Young University in Utah examined subconscious feelings about body image through fMRI analysis of the brains of healthy men and women who were assessed psychometrically as being confident with their bodies.

9. It is known that when humans engage in serious self-reflection, activity increases in the medial prefrontal cortex (mPFC). It is suspected that this increased activation can betray subconscious thoughts.
10. In this study the healthy women looked at images of models in skimpy bikinis. Some images were overweight, some very thin. On viewing each image the women were told to imagine that it was them. When presented with overweight images, the mPFC showed in *all* of the women. Merely imagining that they might be overweight seemed to lead women to question their sense of self, even though they claimed afterward that the test was boring or meaningless.
11. However, men showed no significant mPFC activation while processing either type of equivalent male image. The researchers concluded that there are 'sub-clinical' issues with body image among healthy women and a much finer line between women with and without eating disorders than previously thought.
12. Related findings are also reported from Hiroshima University where healthy women and those with eating disorders were presented with morphed images of themselves and that of another woman. The pre-frontal cortex and the amygdala (implicated in processing emotional reactions such as fear, threat, anxiety and emotional responses to pain) were 'significantly activated' in healthy women in response to their own fat-image'.
13. Even the printed word elicits similar neurological reactions. The study 'Gender differences in brain activity generated by unpleasant word stimuli concerning body image' found that in women, words such as 'obesity', 'corpulence' or 'heavy' were accompanied by increased activation in the amygdala, while the left side of the mPFC (associated with decision making and rational thought) became inactive. In men the response was the reverse.
14. The authors believe that the mPFC is responsible for the gender differences in the processing of words concerning body image, and may also be responsible for gender differences in susceptibility to eating disorders.
15. As visual media of thin female physiques reaches further across the globe the neurological alterations cited above may be increasing among large sections of a population and at younger ages. In a landmark study, a multidisciplinary team from Harvard Medical School travelled to Fiji to evaluate the impact of the introduction of television on body satisfaction and disordered eating in adolescent girls. In Fiji, the ideal body weight for females has always been very full, while going thin – as Fijians refer to weight loss – is a cause for concern, not admiration. Dieting has been rare.
16. In 1995 television arrived and within three years everything changed. The percentage of subjects with pathologically high scores on a test for disordered eating more than doubled from 12.7% to 29.2% and three-quarters of the study population reported that they felt 'too big or fat'. Dieting among teenagers who started to watch television increased dramatically to include two in every three girls and the rate of self-induced vomiting to control their weight, which had been rated as non-existent before television arrived, leaped to 11.3 per cent of that population. The girls openly cited thin female characters in American programmes as inspirations for changing their bodies. Comments included 'I feel fat . . . I just admire them. I want their body, I want their size'. The researchers describe the 'dramatic increase' in disordered eating. 'The impact of television appears especially profound. Western media imagery may have a negative impact upon body image and disordered eating attitudes and behaviours, even in traditional societies in which eating disorders have been thought to be rare'.
17. A British study has found that more than one in four adults in the UK is trying to lose weight 'most of the time' either due to body dissatisfaction or genuine obesity. Yet dieting is increasingly being found to be a major biological event, causing significant metabolic alterations, in some cases paradoxically leading to disease.

18. It has been recently reported that restricting calories to 1200 kcal per day increases the total output of cortisol in females. Cortisol is important for regulating changes in behaviour and functions such as glucose metabolism and the inflammatory process. Prolonged exposure to high levels, however, can lead to higher blood pressure, suppressed thyroid function, impaired immunity, and increased intra abdominal fat – all of which contribute to chronic disease states such as heart disease, diabetes and cancer.
19. The study also found that monitoring calories increased perceived stress and concluded: 'Dieting may be deleterious to psychological well-being and biological functioning'. Stress has been linked to over-consumption of calorie-rich foods and concomitant weight gain in rodents, primates and humans.
20. Intermittent 'yo-yo' dieting is increasingly practised in response to body dissatisfaction. It is generally ineffective in achieving weight loss and the reasons for failure are becoming clearer. The brains of rats alternating between healthy and sweet (unhealthy) food, in the way many dieters do, show highly significant recruitment of neural circuits (CRF system) involved in stress reactions and promote the 'compulsive selection' of unhealthy food and the under eating of healthy foods. This change in dietary preferences is accompanied by a 'withdrawal-like state seen in drug dependence.'
21. There are possible implications for humans who diet in this way. Adaptively, this behaviour may shift food-seeking toward energy-dense, high-reward foods, while devaluing efforts to obtain less energy-rich, low-reward foods'.
22. 'This eating pattern leads to a vicious circle. The more you cycle this way, the more likely it is you cycle again.' Weight cycling averaging only 2.5kg over two-year periods among normal weight individuals is strongly linked to 'a higher risk of cardiovascular disease and death'.

Media and evolutionary adaptation

23. Heterosexual human females maintain/adapt physical appearance in accordance with sexual dimorphism – the systematic difference in form between individuals of different sex. In females, more subcutaneous fat and fat deposits mainly around the buttocks, thighs and hips are *central* to sexual selection.
24. The waist-hip ratio of any physique is very strongly correlated to male perception of female attractiveness across all cultures and throughout history. This is a key health and fertility indicator and core feature of feminine beauty. Exposure to visual images depicting attractive females is found to alter women's perception of their own sexual attractiveness and mating viability through a cognitive comparison process referred to as the 'contrast effect'.
25. The contrast effect is the enhancement or diminishment of perception, cognition and related performance as a result of previous exposure to a stimulus of lesser or greater value in the same dimension (e.g. weight, height, luminescence). Contrast effects are ubiquitous throughout human and non-human animal perception and cognition. In terms of evaluating one's own attractiveness, one appears more attractive when contrasted with a person less attractive and less attractive when contrasted with one of greater attractiveness.
26. Indeed, women are most highly satisfied with their own body image when exposed to images of females wider than themselves. Until recently these self-evaluations of body attractiveness involved comparisons with a relatively small number of other women.

27. Females today are exposed to evolutionarily novel stimuli that deceive cognitive and neurological processes whose function developed to evaluate other females. As some females use a typical media physique to establish norms as points of comparison it has for many become a case of 'keeping up with the Boneses'.

But, if fat and 'yo-yo' diets are so bad, is it really possible for your existing stores to help you burn calories and become a potential anti-obesity drug target?

Brown adipose tissue

28. In mammals the other, less well known type of fat is BAT. Brown adipocytes (fat cells) are structurally very different from white fat cells. Although they still contain lipid, it is stored in many small droplets rather than in one large mass. Brown fat cells contain large numbers of mitochondria (the energy-producing organelles within cells) which are packed with a specialised protein: uncoupling protein 1 (UCP-1). Usually, adenosine triphosphate (ATP), the main energy substrate in living organisms, is produced by the chemical process of respiration which takes place in mitochondria. UCP-1 disrupts respiration at the electron transport chain level and prevents the production of some ATP. Hence, energy acquired from the uptake of free fatty acids and glucose from the circulation is lost as heat, rather than being stored. UCP-1 is mainly produced in BAT.

29. BAT is activated by the sympathetic nervous system (SNS) and thyroid hormones. The release of noradrenaline by the SNS stimulates brown adipocyte proliferation and local production of tri-iodothyronine (T₃, the active form of thyroid hormone) within BAT which stimulates the production of UCP-1. The SNS is activated by exposure to cold temperatures and the ingestion of high-calorie foods; hence, BAT is able to regulate both core body temperature and body weight by increasing energy expenditure.

30. BAT is commonly found between the shoulder blades and around the internal organs and blood vessels. It is present in most small mammals and the newborns of larger animals, including humans. It is particularly important for babies to be able to produce heat via BAT as they have a large body surface area and therefore lose heat more easily. They are also unable to shiver, which is the normal mechanism of generating body heat.

31. It was previously thought that in humans BAT regresses by approximately one year of age and loses its heat-generating properties, except in very rare circumstances. For example in the 1980s it was demonstrated that people who work outdoors in extremely cold conditions (in this case lumberjacks in Norway) have deposits of BAT around their neck arteries which were thought to warm blood flowing to the head. The size of these BAT 'nests' correlated with the length of time the participants worked in the cold. Recent advances in imaging have challenged the view that BAT is neither present nor functional in most adult humans. In a specialised type of positron emission tomography (PET) scanning, patients are injected with 18F-fluorodeoxyglucose (18F-FDG), a radioactive form of glucose which is taken up by metabolically active tissues.

32. Unlike glucose, once inside cells 18F-FDG only undergoes the first step of metabolism and becomes 'trapped'; its emissions can then be detected. This type of scan is usually used to detect tumours as cancer cells take up large quantities of glucose to fuel their growth. From an oncologist's perspective this technique is hampered by the fact that other metabolically active tissues such as the brain and heart, which also absorb large amounts of glucose, are labelled in addition to the tumour.

33. However, this led to an unexpected discovery, a symmetrical area of glucose uptake commonly seen on scans around the neck and shoulders, originally thought to be muscle, turned out to be BAT.

34. Three studies published recently used the technique of PET with 18F-FDG to determine the physiological relevance of BAT in adult humans. All demonstrated that BAT is present in adults (shown by 18F-FDG uptake), predominantly above the collar bones and around the neck. This distribution is different to that seen in rodents, where BAT stores are mainly located between the shoulder blades. Interestingly, on average, lean participants had more active BAT than overweight and obese participants, suggesting BAT may help protect against obesity. As expected, exposure to cold temperatures (in this case dipping your foot in icy water) increased BAT activity.
35. The probability of detecting BAT depended on the outdoor temperature at the time of scanning; detection rates were higher in the colder winter months than in the summer. Additionally, BAT was identified more readily in young women than older men, suggesting there may be age and sex differences.

So if BAT is present and active in adult humans, can it be targeted to help people lose weight?

36. Body weight is determined by the fine balance between calories consumed and energy expended – if you burn more calories than you eat, you lose weight. Numerous attempts have been made to find a wonder drug which increases energy expenditure and burns fat. In the past, very high levels of thyroid hormones or drugs which stimulate the SNS were administered in an attempt to stimulate BAT but both had unpleasant side effects.
37. Dinitrophenol (DNP), a highly toxic industrial chemical, became popular in the 1930s when it was reported to cause dramatic weight loss by increasing metabolism by up to 50%. By affecting respiration in a similar way to UCP-1 (para 28), DNP caused energy generated in mitochondria to be lost as heat rather than being stored as fat. DNP was banned in 1938 when thousands of people reported side effects such as blindness, blood disorders and death due to uncontrollable heat production.
38. A less dangerous potential alternative was highlighted in 2005 when researchers found that fucoxanthin, the compound which gives seaweed its brown colour, increased the production of UCP-1 in WAT from a previously low level. This reduced the amount of abdominal fat in rodents. Although fucoxanthin is available to buy online as a slimming aid, no studies in humans have yet been carried out.
39. The recent identification of BAT in humans suggests that it is a potential target tissue for anti-obesity therapies. However, it might be of limited use in obese people who have smaller BAT stores to begin with. It would be useful if these stores could be increased and new BAT tissue formed, to boost calorie-burning capacity.
40. PRDM16 is a protein which is thought to control the development of brown adipose tissue. It is expressed at much higher levels in BAT compared to WAT and 'knocking out' PRDM16 in BAT causes abnormal tissue development and a loss of heat-producing capacity. When PRDM16 is artificially over-expressed in the precursors of white fat cells it changes their fate and induces them to become brown fat cells instead. This causes the cells to express characteristics of BAT such as an increase in UCP-1. This phenomenon was demonstrated in mice engineered to produce high levels of PRDM16 protein in their white fat stores, which resulted in BAT formation.

41. Although brown and white fat cells are very different, it was commonly assumed that they originate from the same precursor. However, it was recently discovered that brown fat cells arise from the same progenitor as muscle cells, whereas white fat cells emerge from an independent source. As in white fat cell precursors, increasing the expression of PRDM16 in muscle cells causes them to differentiate into brown fat cells. Therefore a drug which increases PRDM16 in either white fat cell precursors or muscle cells could be a potential future anti-obesity therapy – it may increase BAT stores, leading to increased energy expenditure and weight loss. Alternatively, PRDM16 could be used to transform stem cells into brown fat cells in a test tube, which could then be transplanted into humans. However, it must be noted that the effectiveness of any weight loss therapy is limited as the body has many compensatory mechanisms in place to ensure your weight stays constant.
42. Recent findings have shown that BAT is active and present in adult humans and demonstrated that it may be a target for future anti-obesity treatments. So, this winter try turning down the central heating and embracing the cold weather and maybe the pounds will fall off!

Gene expression

43. Many genes can be regulated by changes in behaviour and environment. Nutrition and diet-induced changes in gene expression are now increasingly reported. When overweight women were randomly assigned to a moderate-fat, moderate-carbohydrate diet or a low-fat, high-carbohydrate hypoenergetic diet for 10 weeks a total of 52 genes were expressed differently in adipose tissue as a result of the intervention. This was irrespective of the type of diet.
44. Tenomodulin (TNMD) is a transmembrane glycoprotein. TNMD gene expression in human fat tissue was down-regulated during diet-induced weight loss, with a 65% decrease after 18 weeks of dieting. In both breast and abdominal tissue, significantly reduced expression of genes is observed in the lipid metabolism and glycolytic pathways following dietary energy restriction.
45. The chronic dieting of anorexia nervosa is also accompanied by genetic changes in adipose tissue. Decreased body fat content of patients with anorexia nervosa was accompanied by a reduction in mRNA coding for fat synthesis, the proteins adiponectin, leptin and interleukin-6 together with CD68 expression. In addition, resistin mRNA expression was increased.
46. But in understanding the link between exposure to visual media of thin female physiques and the onset of eating disorders, to what extent can body dissatisfaction induce epigenetic changes which activate psychiatric disorders such as anorexia nervosa? Harvard Medical School molecular biologists examining epigenetic alterations of the dopaminergic system in major psychiatric disorders, believe that environmental factors can influence DNA methylation patterns and hence alter gene expression. Such changes can be especially problematic in individuals with genetic susceptibilities to specific diseases.
47. Recent reports provide compelling evidence that both hyper- and hypo-DNA methylation changes of the regulatory regions play critical roles in defining the altered functionality of genes in major psychiatric disorders. Some psychological distress is now found to leave changes in DNA structure for genes that control our stress response.
48. A disturbed expression of dopaminergic genes has now been identified in eating disorders. Can early or prolonged body dissatisfaction also leave epigenetic marks on DNA?

Conclusions

49. There has been a decided shift in position by scientists and prominent medical bodies in considering media images as being causative. For example, some think that: 'The media is a causal risk factor for body dissatisfaction, negative effect and eating pathology'.
50. The Royal College of Psychiatrists has issued a 'Statement on the influence of the media on eating disorders' (2010). 'The media has a role in both providing a social context for the development and maintenance of eating disorders . . . achieved by propagating unobtainable body ideals and the acceptability of dieting leading to lowered mood, body dissatisfaction and eating disorder symptoms. There is a lack of reality-based imagery'.
51. In their 'Summary and Call for Action', they demand the 'use of role models throughout the mass media that cover a diversity of weight, shape, age . . . cessation of the use of underweight models . . . raising awareness of use and extent of digital manipulation of images through use of a kite mark'.
52. There is good reason for these strong reactions as few realise the sheer prevalence and mortality rate of anorexia nervosa. In the UK, 1.4m females currently have an eating disorder. Of these, 140,000 have anorexia and with a 10% mortality rate, approximately 14,000 will die. If only 5% of these deaths could hypothetically be attributed mainly to the effects of exposure to thin media physiques, this equates to 700 deaths. This is far greater than the number of women in the UK killed each year through domestic violence (approximately 100) or from heterosexually contracted HIV/AIDS (38) per annum.
53. A decade ago, the British Medical Association's Board of Science and Education demanded 'a more responsible editorial attitude towards the depiction of extremely thin women as role models'. Yet matters have become much worse. Traditionally, suggestions that media images are causative are deflected by the rationalisation that 'the media merely reflects society and are being used as a scapegoat for body dissatisfaction and eating disorders'.
54. However, the biological sciences have now provided a deeper understanding of the precise role of media physiques in these pathologies. In other areas of child and public health, exposure to causative risk factors for disease is ultimately controlled through legislation. As thin media physiques are now a biologically based medical issue, it raises the question of whether assertive guidance should in future emanate from the Department of Health as opposed to the Department for Culture, Media and Sport.
55. There is already a precedent for media policy and legislation based upon the biological characteristics of people represented on television screens, for example, there is racial diversity and gender legislation. The BBC, for instance, 'is committed to reflecting the diversity of the UK audience . . . in its output on TV, on radio and online.' At the same time, an ongoing issue is the de-selection of females whose biological characteristics include grey hair and/or wrinkles on all electronic media networks in most Western industrialised countries.
56. Following this logic, female physique is yet another biological parameter which could be considered an aspect of diversity. Both the BMA and the RCP have called for media physiques 'that cover a diversity of weight, shape, age . . .' Implementing this policy will require a new-found enlightenment towards images deemed harmful, ie. the incorporation and active exclusion of media physiques according to their degree of risk to young female viewers. While most concern has surrounded thin fashion models, greater risk may lie in the more everyday ambient images of 'permarexic' – visibly unhealthily thin – children's television presenters, actresses and innocuous newsreaders who form the backdrop to the visual lives of girls and women.

57. At a practical level, a minimum standard of risk acceptability would have to be established based upon visual physique parameters such as waist-hip ratio and dress size. Alternatively, a roughly accurate proportion of female media images of an average UK dress size 16 could be ensured.
58. This may seem an extraordinary form of social medicine but the evidence suggests that 'a lack of reality based imagery' in media is causing health problems in a very large number of women and young girls. And so it appears that while men eat food, women have a relationship with food. This relationship has grown increasingly dysfunctional. Forty years after the debut of body politics, biology is explaining more precisely why fat is a feminine issue.

Acknowledgments

'A source of thinspiration – the biological landscape of media, body image and dieting',
(Aric Sigman), Vol 57, No 3 October 2010 p117–121 (ISSN 0006–3347)

'Can your fat make you thin?' (Kylie Beale), Vol 57, No 2 June 2010 p77–79 (ISSN 0006–3347)

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