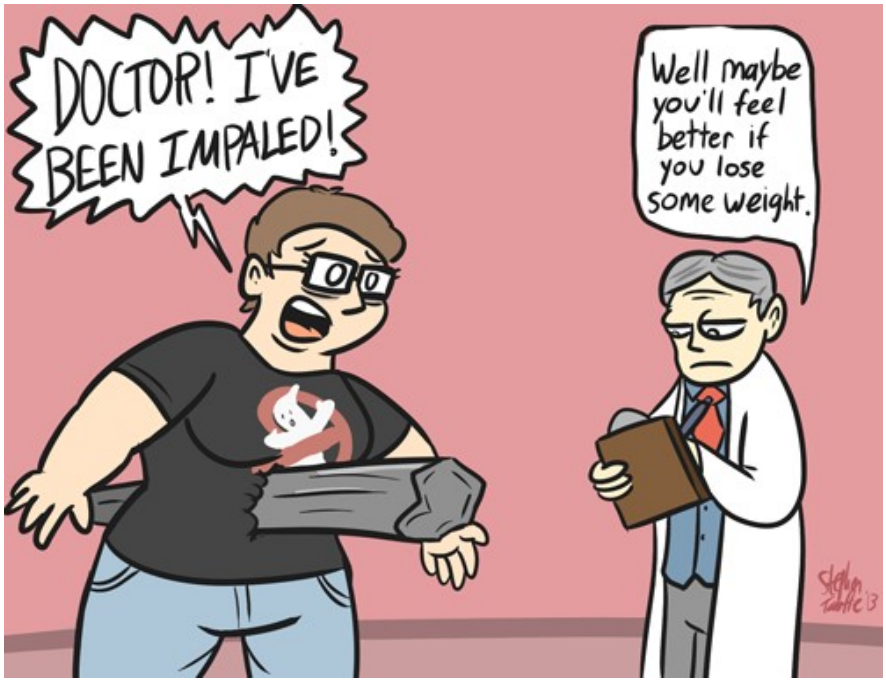

Morbidly Obsessed

The Junk Science of 'Obesity'

Kim Bryan

In western cultures fatness has long been regarded with distaste. Associated with deviance in morals or personal conduct, for those of larger size, ostracism, ridicule or exclusion are common experiences. Mostly, these ideas derive from a primitive but persistent cultural aversion to images of human disability, ugliness or imperfection. Today, owing, I suggest, to its appropriation by the medical establishment, the 'obesity' brand is fuelling discriminatory practices which impact directly on the life chances and wellbeing of the target group.

When scientific claims appear to reinforce longstanding cultural phobias, it is reasonable, given the damaging experiences historically of some marginal people, to expect alarms to sound and critical faculties to become engaged. In the case of obesogenics, we could not be more wrong. The study of fatness as a disease has become established across social and political media firstly here and then across the planet without any serious debate in the public forum over the substance of this claim. Scientists clinging to orthodoxies of the past, who believe for instance that women or non Europeans are less intelligent, that mental illness can be cured by surgery or that homosexuality can be 'reoriented', would, in modern academia, be likely to find themselves 'no platformed'. Enjoying the advantage however of being 'current', and not associated therefore with a 'backward' past, obesogenics is sweeping aside all before it.

What, exactly, is inhibiting our willingness or ability to scrutinise its claims? Have we accepted the mantra that today's medicine is benign, operating independently of society? If

this is the case then first and foremost it is the medical basis of the current war on fat that must be examined and tested against the standards that would normally be applied to any area of scientific study. While there will be multiple factors that will explain public belief in the 'deviance' of any group, this essay will focus only on the doctrines of 'obesity' science, looking specifically at how it assesses and presents 'evidence'.

Defining the Disease

In the diagnostics of the fat war the currency of the 'problem' is BMI, the weight-height chart constructed to quantify the extent of an individual's 'fatness'. Any theory of human biology which is authentically scientific begins with natural laws, building from what we know about ourselves and our world. It should be noted from the outset therefore that in its adoption of body typology, obesogenics does the reverse, openly and shamelessly overriding biology in favour of socially constructed norms.

The obesogenic doctrine that diversity of body size is rooted in unnatural processes not only overrides living reality but is at odds spectacularly with core scientific knowledge. Over many decades studies conducted on identical twins have produced a consistent body of data showing strong predisposition in body size and shape quantifiable statistically to that which determines height (Stunkard et al, 1986, Wardle, 2008). Furthermore biologists have explained long ago the natural processes that regulate appetite as well as individual body size and shape, and why attempts to modify or alter either by 'lifestyle' changes have little chance of long term success.

If, in our determination to fight fat, we are guided by science not society, it is difficult to explain how a substantial body of established knowledge pointing to the normality of size development and diversity would give way to a powerful orthodoxy predicated upon its precise opposite.

The Character of the Disease

To the extent that it regards size 'modification' as medically 'reasonable', it follows that obesogenics assumes that body size is a core indicator of important traits, in this case the health or well being of an individual. Once again the belief defies decades of scientific data suggesting body size is a largely passive ingredient in the health prognosis of individuals.

In relation to fitness, the usual standard in western societies of 'health', laboratory based studies suggest the benefits of exercise do not depend on weight or weight loss (Blair, 2007) while elite sport (not incidentally associated with longer life) is performed by athletes of all sizes. Until the eighties few practitioners would have questioned the idea that our better health, physical performance and longer life - we run faster, jump higher, last longer - is attributable at least in part to overcoming the diseases associated with malnourishment. Yet today's ideal of 'healthy living' appears to be based on the entirely

contrary idea that the life patterns of well nourished people are problematic.

In arguing for a wholesale change in western lifestyles ‘obesity’ scientists claim that since the 1970s overall growth in body size has accelerated proving it is no longer normal or healthy and has an origin in some unnatural cause. For the vast majority of us, the ‘growth’ to which they refer is exaggerated by failure to control for age and changes to the BMI measures which 'expand' the diagnosis. Use the original measures and our body growth levels over the last few decades are similar to those of height. Growth is also found disproportionately at the atypical margins among those who will carry the strongest predispositions to look different from the ‘norm’ (Campos et al, 2006, p55). It is at the margins that we find an exaggerated tendency to repetitious dieting or weight cycling, the element that multiple studies show is the ‘single biggest predictor of weight gain’ (cited *Scientific American*, 2007). Far from being welcomed, attempts to integrate this body of established knowledge into medical practice, along with recent studies suggesting our body growth is slowing, are overwhelmed by the power of the moralising 'panic' narrative with 'good' news generally followed by mounting levels of alarm (Rokholm 2010).

By conferring on the socially constructed idea of ‘overweight’ the status of medical pathology, obesogenics is able to simply portray the presence of ‘fat’ as the ‘proof’ that something is ‘wrong’. Without this presupposition, rooted in belief systems linking large to ‘excess’, the broader changes claimed by ‘obesity’ scientists simply do not exist. It was in the 1970s that calorie (and fat) intake first began to dip not increase as is often supposed. If we're looking to 'explain' our unwanted growth, an ‘explosion’ in dieting rather than 'overeating' is a better fit (cited Gard and Wright, 2005, p116). Given that the body changes we’ve seen cannot be shown to be unnatural and the trend of the past forty years is calorie reduction not increase, it is hard to see how the claim that western society is threatened by an epidemic of ‘obesity’ (meaning gluttony) is based on science.

Targeting the Diseased

In light of the comparative health of well nourished societies, it is difficult to understand why the diversity of body size which accompanies improved nutrition should be regarded by anyone as a problem let alone a crisis.

Given their belief in the ‘pathology’ of the fat condition, it is no surprise that the obesogenic search for ‘answers’ is focusing on the thinnest section of the population: children. The *Thousand Families Study* is one of many generational studies showing the strong link between increasing body size and ageing with eighty percent of fat people acquiring their fatness not in childhood but in adult life. In finding ‘little tracking from childhood overweight to adulthood obesity’ TTFS also found ‘no excess adult health risk from childhood or teenage overweight’. According to Gard (2007) ‘no study in the history of medical science has ever established a causal link between childhood fatness and adult ill health or premature death’ (p8). In contrast the impact on children of calorie restriction even at lower levels is not disputed, resulting in adverse health outcomes which can be long

term or irreversible. In light of this, it is difficult to see how the current 'health' programmes aimed at making fat children thinner are medically based.

Given their belief in the magnitude of the 'disease' it is common for anti-fat campaigners to 'explain' it in terms of broader social problems. It may be reasonable, in light of mounting levels of prejudice, to expect more fat people on welfare and in lower paid jobs and, in due course, thinness to become correlated with success. In spite of major inequalities in health, however, recent UK based studies into 'obesity' and social class, conducted by the *Food Standards Agency* (2007) and by Norfolk PCT (2007) suggest that the 'relationship' between poverty and BMI is marginal at most, amounting to a couple of pounds. Both studies also failed to find the 'link' between food choices and body sizes upon which the 'abnormality' narrative is predicated. If practitioners are linking poverty to BMI through what they see as common behaviours or characteristics, then, in the fullness of time, 'evidence', may well materialise 'linking' 'obesity' to criminality, antisocial behaviour and low intelligence.

When we examine its relationship with food, obesogenics appears intrinsically disordered. Mixed with our longstanding body dysmorphia modern anxiety about food is medically toxic. In contrast to what is popularly believed, decades of studies have failed to link any food or food group causally to disease except in highly abnormal quantities and fruit is as theoretically capable of 'causing' cancer as meat. Like 'overweight', our 'gluttony' is a fabrication, the product of an artificial standard which, in playing to our subconscious fears, encourages an aversion to intuitive and therefore nutritious eating.

It is true we consume more fat and calories than our doctors say we should. Yet the diet we actually eat is also matched rather well to the balance of nutrients consumed by preceding generations, or at least the rich among them. While 'diet' is often cited as a major cause of premature death, comparable to smoking, almost all diet related disease is in fact malnutrition in the elderly (BAPEN, 2005). Whatever the risks of 'over consumption' they do not come near those associated with smoking or the morbidity intrinsic to low social status. The question once again is why, when our bodies are so good at regulating our consumption to our needs, our doctors appear to be operating by a very different standard.

According to Gard and Wright (2005) among its many roots in irrational fears, obesogenics draws on a longstanding inclination to view 'modern' society as 'sick' or 'soft', a theme of popular culture for so long it is oblivious to reason. However, the belief that fatness is 'proof' of 'increasing' idleness in western society is similarly not supported by the balance of evidence which suggests that fatter people are more active than the thin and that the overall level of activity of the population generally has not declined. While they may be fond of telling us how much time we spend looking at our computers, the context provided by the balance of data is always avoided: activity levels in general are unrelated to how much TV we watch or other aspects of 'sedentary' modern living (pp22-32).

'Scientists' (nearly all funded by the diet/lifestyle industry and drugs companies) who treat the 'idleness' of larger people as worthy of testing, are failing to interrogate the

assumptions driving their work, a baseline test of scientific integrity. Since, under the most basic laws of nutrition, energy levels drop when calorific intake is reduced, idleness, like malnutrition, is unlikely to be overcome by dieting. Where we are guided by the artificial doctrine of 'weight control', the healthy imperative to eat well and keep active becomes, for most of us, unhealthy.

The Case for Malnutrition

Given the personal experiences of so many dieters, it's no surprise that the high 'failure' rate of intentional malnutrition (dieting) is the one truth obesogenics has not managed entirely to bury. Given the association of ageing, specifically middle age, with body size increase, we should not be surprised to discover another truth, less well known, that eighty percent of studies into dieting and morbidity have suggested that regardless of BMI deliberate weight loss increases mortality especially in the over fifties (Gaesser, 2002, p136). Aside from its association with totalitarian states - Nazi Germany patented the lifestyle based model of 'responsible' citizenship - using medicine to eliminate or amend naturally occurring human differences contains extraordinary potential for multiple levels of well documented physical and mental harm (Heuer and Puhl 2010). Next time you are told to lose weight, you might ask yourself (or your doctor) why he or she is telling you to do something that science has shown conclusively is bad for you.

It is reasonable to assume there is an association between increasing affluence and increasing body size. In the West we have fewer malnourished people and more older ones than we did fifty years ago. What is absolutely not reasonable by any scientific standard is the dogma that this is an aberration and that deprived or under nourished societies, individuals or communities are in some respect 'healthier'. Health practitioners or campaigners who compare our 'modern' lifestyle unfavourably to conditions of war, poverty or famine (see Rohrer, 2010) have submitted, I suggest, to phobias about ageing and control which view fasting or denial as an intrinsically 'good' or morally superior condition. In its relationship with food, Paul Campos suggests obesogenics is mirroring the belief systems of anorexia, attitudes more common among high status individuals (2004). If he is right, the progress of the war on 'obesity' will likely mirror the recidivist nature of the disorder. An increasingly unrealisable ideal will lead to an increasingly restrictive model of nutrition, forced, without informed consent, onto otherwise healthy citizens.

The Misappliance of Science

In promoting the concept of larger body size as disease, it has been necessary for obesogenicists to encourage the belief that when fat people die they have at some level died of 'obesity'. To this end extraordinary liberties have been taken with scientific data.

Although scientists have known for a long time that arterial fat is completely unrelated to body fat, in larger people 'obesity stakeholders' (see Cooper) continue routinely to link the 'fatty deposits' found in heart attacks to 'overweight'. While, in general, studies suggest fat people are at the same or slightly lower risk of cancer, stakeholders have been broadly

successful too in getting ‘overweight’ established as a risk factor for the disease. In the case of breast cancer for instance only three of the sixteen studies cited by the *World Cancer Research Fund* in its 2009 report actually suggested any statistically significant link to large body size (compared to eight saying the reverse) and overall the ‘links’ to low body weight were stronger (cited Luik and Basham, 2009). It is hard to explain why the WCRF and breast cancer advice continues to list ‘obesity’ as a ‘factor’. Unless of course we are dealing with practitioners who are less interested in treating the cancer than ‘curing’ the ‘obesity’.

In spite of the frequency of the claim, no study has shown a ‘surge’ in cases of type two diabetes in children aside from the discredited 2005 report that found the condition almost entirely in high risk children from non white ethnic groups. Widespread screening and changes to the diagnostic measure of type two diabetes (the basis incidentally of false claims of soaring rates) have increased greatly the probability of diagnosis compared to the past. Nonetheless, the disease, which is substantially inherited, has not actually been ‘linked’ to weight except at high end BMI and rarely is account taken of the weight gain often indicated with the condition. Diabetes is one of twenty one diseases commonly ‘linked’ to ‘obesity’ (including CVD, colorectal and breast cancers) that Graubard and Flegal (2009) found had no actual statistically significant mortality link to BMI. Other common measures of ‘fatness’, in particular a high waist measurement and high body fat, were found to be correlated with lower overall mortality.

One reason for the paucity of references to the role of dieting and of poverty in exacerbating type two diabetes, as well as the unnecessary deaths among the poor and elderly attributable to systemic failings in medical care, may be that like cancer and CHD, weight focused practitioners are less interested in treating the disease than 'correcting' the deviant 'behaviour’ (Aphramor, 2015). Osteoporosis is a disease that kills more women every year through directly related fractures than colorectal and breast cancer combined. In spite of its high mortality, it appears not to be subject to much ‘prevention’ at all, being virtually absent from the ‘healthy lifestyle’ advice we receive from our practitioners except where they are trying to link it to ‘obesity’.

In the case of thinner people or those following weight loss regimes, disease correlations are never treated as causal, discounted on the perfectly reasonable grounds that ‘the thin’ includes people with chronic morbidities and eating disorders who are not representative. People whose weight is inflated by disease or disability are regarded differently however: they remain ‘fat’ rather than ‘ill’, their ‘unrepresentative’ diseases ‘translated’ into ‘obesity’ (see Shriver, 2009).

The fat may well be the only group subject to social discrimination who do not in fact die younger than everyone else. Contrary to media reports at the time the large 2009 Canadian study did not show ‘benefits’ to ‘overweight’ only; many of the ‘obese’ scored as well as or better than the ‘normal’. Its findings are consistent with the great majority of studies conducted since the 1960s showing little variation in mortality across most of the BMI range with the ‘better’ outcomes falling in the middle of the population among the

‘overweight’ and mildly ‘obese’. The BMI with the ‘best’ score in terms of life expectancy, 27, is also the average of Britain’s population. The picture of BMI as non pathological except at the margins was found in the 2013 JAMA ‘study of studies’ to be near unequivocal (Flegal et al).

The obesogenic doctrine that fat people ‘die younger’, and the wild claims built around it, is achieved by the simple expedient of ‘spreading’ the prognosis of people at the margins where more people with illness or disability will be found, across millions who are well; a ‘reading’ of the figures that would produce the same outcome for those of ‘normal’ weight. While multiple individual variables affect our health and mortality, epidemiological studies suggest that for all but a tiny number of people, body size does not figure among them. Invariably this core data is marginalised in favour of short term studies showing benefits which over the longer term disappear or reverse, 'extrapolations' which ignore the negative effects of the measures indicated and 'correlations' which fail to control for significant morbidity indicators such as class, race, disability, life experiences, as well as dieting history (Kendrick, 2014).

In spite of the far greater impact of malnutrition, frequently, the war on ‘obesity’ targets the ‘cost’ of fat (ie ‘over’ nutrition) as a burden on society. While the fat are expected to subsidise the treatment of the non fat it is regarded as unreasonable if not outrageous for the care of people who happen to be large to be the responsibility of all of us. In cases where dysfunction in diet is causal, which are rare, the same double standard is usually applied. Patients whose health is compromised by eating disorders are viewed as deserving of understanding and support while ‘supersized’ individuals whose weight is inflated by genetic, metabolic or other disorders (and this is nearly all of them) are ridiculed in media as failed people lacking willpower.

It is difficult to see the reasons why health practitioners and academics would adopt or accept ‘standards’ like these unless of course they are proceeding from the belief that fat death is different from thin or deserves to be treated differently. The belief that morbidity in the fat is a pathological condition of the larger body is a result I suggest not of evidence but of ‘response’; it is the attitude of practitioners to the fat body that differs from the ‘norm’ applied to everyone else.

Conclusion

The ‘obesity’ story – fat is the ‘proof’ we’re unhealthy and it’s ‘caused’ by ‘bad’ lifestyle choices – is contradicted by decades of independent scientific study suggesting size diversity is benign, naturally occurring and resistant to manipulation by dietary ‘choices’. By extension the ‘cure’ - the ‘balance’ of nutrition and energy the theory says will produce the ‘healthy’ body size - will be ‘found’ in most of us only in a life governed by hunger.

Where health policy is disrupted by socially constructed ‘ideals’ of what is ‘desirable’ at the expense of what is real, the result is not simply failure but long term harm. As we have seen historically in medical mistreatment of the poor, women, non white minorities, the

disabled, the mentally ill and the sexually 'deviant', the impact on health policy of ideology, for I believe that is what we are seeing, creates failed or abusive practice because it shifts the imperative to 'treat' beyond those who are actually sick.

Stretching the 'responsibility' to 'change' so far beyond the small minority whose lives may be improved by managed weight loss opens markets for 'providers' but across the community any benefit will be outweighed significantly by the potential for harm. Based on idealized 'models', millions of people with a normal life expectancy face the risks of 'treatment' with drugs, malnutrition or surgery based on nothing more than the imperative to change their BMI or other arbitrary 'norm'.

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Appended:

'How We Know It's Not Science': science v superstition model
'You know How to Whistle, Don't You?: medical modelling of lesbianism

SCIENCE	SUPERSTITION
What We Are	What We Want to Be
What We Know	What We Feel
Lead by Evidence	Lead by 'Opinion'
Disinterested	Self Interested
Questioning	Dogmatic
Flexible	Inflexible
Moderate	Extreme
Considered	Emotional
Invites Scrutiny	Fears Scrutiny

Science v Superstition Model
Kim Bryan, January 2016

You Know How to Whistle, Don't You?

Medical 'explanations' for lesbianism

1857	*Tardieu*	homosexuals as criminally deviant
1860	*Ulrichs*	homosexuals a 'third sex'
1893	*Moll*	'unrequited' homosexual women suffer disturbance of the nervous system and outbursts of fury
1894	*KrafftEbing*	lesbianism a cerebral anomaly indicating an inherited diseased condition of the central nervous system
1896	*Tarnovsky*	homosexuality a result of nervously disturbed, hysterical, insane or diseased parents
1897	*Ellis*	female 'inversion' the more common; lesbians boyish, nervy and having deep voices and the ability to whistle
1908	*Carpenter*	female homosexuals 'fiery, active, bold and truthful, with defects running to brusqueness and coarseness'
1917	*Adler*	lesbianism a 'protest' against male objectification of women
1917	*Wolfe*	lesbians 'avoiding' the responsibilities of marriage; 'predatory' (unmarried) lesbians, a threat to family life
1920s	*Freud*	lesbianism an 'abnormality'/ underdevelopment caused by unresolution of Oedipal feelings; characterised by penis envy, mother fixation
1936	*Terman, Miles and Kelly*	two lesbian types, perverted and inverted, the first outside natural gender alignment, the second emotionally repressed in gender identity

1936	*Ellis*	sex segregation, disappointment or failure in heterosexual love or excessive masturbation
1936	*Hirschfield*	an incurable genetic disease
1941	*Henry*	failure of parents to enforce gendered roles creating a man in a woman's body
1947	*Thompson*	an underlying disorder 'triggered' when there are no available men
1954	*Caprio*	a narcissistic extension of auto eroticism triggered by a childhood or adolescent trauma
1963 1965 1976	*Socerides,* *Romm and* *Wilbur*	'contributory' factors- sexual abuse, ambivalence towards parents, fear of men, 'clitoral fixation'
1970	*MacDougall*	rebellion against mother role resulting in a psychotic identification with the father
1978	*Socerides*	a masochism leading to a failure to recognise or focus upon the 'object'
1988	*Siegal*	narcissism again, caused by the mother 'interfering' in her daughter's identification
1989	*Quinodoz*	a defence against psychotic and Oedipal anxieties

http://www.lesbianinformationservice.org/medrole.htm
January 2004

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Edited version of essays written in 2017

Published by 'National Bolshevism UK'
2nd March 2021