



‘Diabesity’ down under: overweight and obesity as cultural signifiers for type 2 diabetes mellitus

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Although overweight and obesity are increasingly seen as the key ‘risk factors’ for Type 2 diabetes mellitus (T2DM), the relationship between them is complex and not well understood. There are many ‘risk factors’ for T2DM, including ageing, genetics, previous gestational diabetes, a family history of the disease, etc. the interplay of which is not entirely clear. While weight gain is a common symptom of T2DM and the disease appears to be more prevalent among ‘obese’ people, individuals from a broad range of weights (including those considered ‘healthy’) can develop the disease. However, in recent years, the idea that fatness is *the* risk factor and/or central *cause* of T2DM has become increasingly prevalent and naturalized in popular, academic, and public health discourses in Australia. In these convergences, the complex etiology of the disease and limitations in current knowledge are blurred or reconstituted. To date, the potency of overweight and obesity as cultural signifiers for T2DM and its consequences has received little attention. Drawing on an analysis of government reports, journal articles, and media coverage published since 1998, this article sets out to trace and unpack some of the contours of these convergences, while recognizing their entanglement in earlier moralizing discourses, which continue to have considerable salience.

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Introduction

A friend aged in her early 40’s recently recounted her experience in hospital after having her first child. Like most women, she had gained weight during her pregnancy, placing her pre-delivery weight at around 95 kg. One of the nurses working on the maternity ward came into the room my friend shared with several other mothers and their newborns. The nurse was looking for a woman who had gestational diabetes. My friend explained that the nurse looked around the room and then came straight over to her bed and asked, ‘Are you the woman with gestational diabetes?’ She was not. It was the younger (mid 20s) and physically much smaller woman in the bed across the room.

At a recent lunch time gathering, another friend, also in her 40s, revealed that she had been diagnosed with type 2 diabetes mellitus (T2DM) – something she was not expecting and was clearly surprised by. ‘How can I have bloody diabetes’, she exclaimed. ‘I’m not overweight; I’ve never been overweight. In fact I’ve always been small, like all my life. I eat well and I exercise, I mean, I drink a bit, sure, hell, we all

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have our vices, but I'm not fat, never have been. So how is it that I have bloody diabetes?' These incidents reveal a growing conflation between overweight, obesity, and diabetes in the public imagination – and it is this union and its appearance across a range of fields and discourses and media that is the focus of this paper.

T2DM is a condition marked by high levels of glucose in the blood, caused by a reduced capacity or inability to produce insulin – a hormone generated in the pancreas that controls blood glucose levels (WHO 1999). The disease often goes undetected for years and early symptoms include increased tiredness, thirst, urination, hunger, and weight gain.

It is the most common form of diabetes and occurs mostly in people aged 50 and over (AIHW 2012, 298). It is strongly associated with ageing, genetics, a family history of the disease, previous gestational diabetes, ethnicity, nutrition, and poverty – the interplay of which is not well understood (Gard and Wright 2005; Ridderstråle and Groop 2009, 111; Vassy and Meigs 2012, 191; Wilson et al. 2007). Having a parent with diabetes 'increases one's risk for the disease by 40%' (Köbberling and Tillil 1982, 2003). Having two parents with the condition increases this to 70% (Kaprio et al. 1992; Meigs et al. 2000; Poulsen et al. 1999; Ridderstråle and Groop 2009, 111; Vassy et al. 2011, e77; Wilson et al. 2007).

It is thought that T2DM is both polygenetic and heterogeneous: that multiple genes are involved and that different combinations are at play in different groups or individuals (Poulsen et al. 1999; Doria, Patti, and Khan 2008, 186; Rich 1990; Kahn, Vicent, and Doria 1996). Vassy et al. (2011, e77) suggest that while parental history (which includes familial and ethnic clustering) and specific gene variants play a significant role, there are likely to be 'modest associations' with the environment and unknown genetic factors. Evidence is also emerging that second-generation anti-psychotics – increasingly used to treat anxiety and depression – create metabolic changes that bring on diabetes and lead to significant weight gain to a even greater degree than first generation drugs (Allison et al. 1999; Leucht et al. 2009; Zimmermann et al. 2003, 193–194).

Although diabetes appears to be prevalent among those classified as 'obese' (BMI \geq 30), people from a broad range of body weights and BMI's (including those considered 'healthy') can and do develop the disease (McCarthy et al. (1996, 5)). The largest national study into diabetes prevalence in Australia estimated that 44% of participants with impaired glucose were obese – suggesting that 56% were not – and that 15.9% of those who were obese did not have impaired glucose (Dunstan et al. 2001, 1).

The Australian Government's bi-annual health reports are based on self-reported data, and they too have shown a rise in the incidence of T2DM from 193,000 (1.3%) in 1989–1990 to 818,000 (3.9%) in 2007–2008 (AIHW 2012, 299, 2010). However, while an increase in disease incidence has played a role here, it is widely acknowledged that a significant aging population, increased life expectancy, rising community awareness, improved detection, survival, and improved access to health care services are important contributors (AIHW 2010, 154, 2012; Vassy and Meigs 2012, 191; Wilson et al. 2007; Zimmet, Alberti, and Shaw et al. 2001:78). Indeed, McCarthy et al. (1996, 1) have argued that diabetes 'has evolved as a major health problem because of increasing life expectancies' in many parts of the world, including Australia, which has a large aging population.

Despite the complex causal pathways that lead to diabetes, the disease is increasingly represented in deceptively simple terms. Since 2000 researchers, commentators and policy-makers in Australia have linked the rise in diabetes directly to increases in obesity in the same populations (Barr et al. 2006; Colagiuri et al. 2006; Duke,

Colagiuri, and Colagiuri 2009; Dunstan et al. 2001; Zimmet 2001). Over the last decade, government health reports, strategies and plans focused on diabetes, consistently identify obesity/diabetes as a major and escalating health problem that is now commonly referred to as an ‘epidemic’.

As Gard and Wright (2005, 101–102) have pointed out, ‘much of the rhetoric around the so-called ‘obesity epidemic’ is based on the spurious claim that obesity causes or is one of the important multiple causes of non-insulin dependent diabetes mellitus or ‘ischemic heart disease’. And yet, the potency of overweight and obesity as a cultural signifier for diabetes in Australia, and its iatrogenic consequences for those diagnosed with or deemed ‘at risk’ for the disease, are topics that have received little critical examination to date.

This paper sets out to map the perceived linkages between T2DM and body weight in Australia. An extensive literature search was undertaken in September 2012 for sources published in, or about, Australia since 1988 using the databases LexisNexus, ProQuest, and Factiva. Searches for ‘diabesity’, ‘obesity risk diabetes’, ‘obesity causes diabetes’ and ‘weight or fat causes diabetes’ were used and a total of 81 items were located: 42 media stories, 22 peer-reviewed articles, and 17 government and non-government reports. This includes national health reports and policy briefs from health agencies, diabetes research centers and associations (NGO’s), journal articles, and media reports and commentaries. This analysis is based on repeated critical readings of these texts to examine the ways in which the relationship between diabetes and overweight/obesity are discussed while tracking this chronologically, through time. I am particularly interested in how – and when – discourses on overweight, obesity, and diabetes began to converge in Australia. Of course, it is acknowledged that diabetes, weight, and diet have a long and complex history – the nature and archeology of which is well beyond the scope of this paper. However, this analysis suggests that certain convergences and framings that have become naturalized in recent years began to appear from at least the late 1990s. The paper also highlights some of the potential implications of this convergence in light of recent research examining the relationship between weight stigma, discrimination, and diabetes management.

Configuring the relationship between diabetes and obesity: slippages and framings

Diabetes was not part of the Australian national health agenda until 1998, when it was named as the fifth Nation Health Priority Area (NHPA), alongside cardiovascular health, cancer control, injury prevention and control, and mental health (CDHC and AIHW 1999). Over the last 15 years and despite the ongoing uncertainty about the etiology of diabetes, overweight and obesity are increasingly presented not only as risk factors, but also as a contributors to and generators of the disease, across a spectrum of publications and mediums. While weight and diabetes have had a long association, the confluences exposed here began to appear in Australia in the late 1990s.

In 1998, *The National T2DM Strategy and Implementation Plan for Australia* was released by Diabetes Australia (Colagiuri, Colagiuri, and Ward 1998). The focus was on people, providers, policy, and programs and called for a National Diabetes Task Force to oversee the execution of the plan (Colagiuri, Colagiuri, and Ward 1998, xv). In this Strategy, T2DM is clearly framed as ‘a major public health concern’ and as ‘reaching epidemic proportions’ (Colagiuri, Colagiuri, and Ward 1998, x). Although there is a call for increasing biomedical research into the disease, and un-modifiable and modifiable risk factors are described (including obesity and overweight), the

greatest emphasis is on ‘lifestyle’ interventions’ (Colagiuri, Colagiuri, and Ward 1998, 18). Indeed, the authors assert that ‘[L]ifestyle factors such as overweight, inactivity and diet predispose to its [diabetes] development’ (Colagiuri, Colagiuri, and Ward 1998). The Plan’s release followed that of the much publicized WHO report that announced the emergence of a global obesity epidemic (WHO 1998).

The National Strategy was endorsed in 1999 with the Federal health minister calling for the country to ‘Defuse the diabetes time bomb!’ and stating that ‘Australians are facing a diabetes epidemic within 10 years if immediate changes are not made to our lifestyle and diet’ (Australian Government 1999). In that same year, a new study was commissioned to determine diabetes and obesity prevalence in a randomly selected population that would provide baseline data for future interventions and the beginning of a longitudinal study.

Published in 2001, *The Australia Diabetes, Obesity and Lifestyle Study 2000 (AusDiab – 2000)*¹ saw the emphases on weight given significantly more prominence (Dunstan et al. 2001). The title of the study, ‘Diabetes and Associated Disorders in Australia – 2000: The Accelerating Epidemic,’ evidences this shift in emphasis towards obesity/diabetes. It is also earliest use of the ‘diabetesity’ found in an Australian government document².

Throughout the document, diabetes is referred to as an ‘epidemic’ of some ‘magnitude’ that is being driven by increasing rates of overweight and obesity (Dunstan et al. 2001, x). The WHO (1998) report proclaiming the existence of a ‘global obesity epidemic’ is given considerable prominence in the study and is the primary source of evidence for an obesity epidemic in Australia. Following an introduction and background chapter detailing what diabetes mellitus is, the next and third chapter in the AusDiab Study is called ‘Overweight and Obesity’, suggesting to the reader the import of these ‘factors’. It begins with the following opening statement: ‘Since obesity is strongly linked to Type 2 diabetes, the term ‘diabetesity’ has been recently used to embrace the two conditions’ (Dunstan et al. 2001, 13). So connected are the two conditions in this framing that a new term is now to be used to encompass both: diabetesity. The authors then detail rates of overweight and obesity in Australia and conclude that:

The principal finding [of the study] was the high prevalence of being overweight, both in the pre-obese and obese ranges amongst the AusDiab participants. ... The likelihood is that the increased levels of obesity have been a significant contributing factor in the escalating prevalence of diabetes. The epidemic of obesity must be curtailed in order to reduce the impact on diabetes, as well as other obesity-related conditions. (Dunstan et al. 2001, 13)

In this framing, overweight and obesity are not symptoms of diabetes but have become a ‘significant contributing factor’; diabetes has emerged as an ‘obesity-related condition’ of epidemic proportions and the two ‘conditions’ have been merged into diabetesity. As noted above, however, the AusDiab Study (2001) also reported that only 44% of participants with impaired glucose were obese – suggesting that 56% were not (Dunstan et al. 2001, 1).

Furthermore, the later chapters of the report are entitled: dyslipidemia, hypertension, smoking, physical activity, risk factors for cardiovascular disease, and indicators of renal disease. None are dedicated to non-modifiable ‘risk factors’, or to the role of an aging population or improvements in diagnosis as factors contributing to diabetes prevalence in the population. Notably, while hypertension is reported as present in 63% of participants with glucose impairment, it is covered *after* the chapter on obesity and overweight.

The AusDiab Study – 2000 exhibits a conflicted and at times contradictory position on the relationship between weight and diabetes, describing the disease as follows:

Type 2 diabetes is characterised by resistance to insulin's action and impaired insulin production by the pancreas, either of which may predominate. It is the most common form of diabetes accounting for more than 85% of persons with diabetes in Australia. It has a strong genetic (familial) propensity, which is unmasked by lifestyle factors such as obesity (hence the term "diabesity") and lack of exercise. In most instances, the molecular or metabolic causation is not yet known. (Dunstan et al. 2001, 2)

This description exemplifies the kinds of slippage that occur in the framing of diabetes and its convergence with obesity and overweight. While the authors highlight the significance of genetics, they also argue that 'lifestyle' or environmental factors can 'unmask' or activate diabetes. In doing so, they make it clear that overweight or obesity alone would *not* cause diabetes, unless the person already has a genetic predisposition. However, this also undermines the use of the term 'diabesity' and the direct connection it implies between diabetes and obesity. The authors then go on to conclude that the actual causes of diabetes are 'not yet known' and, in doing so, effectively undermine everything they have said before.

The flourishing of diabesity and the wilting of other 'risk factors' or 'causes'

The AusDiab Study – 2000 signaled the emergence of a rising 'enemy' in the fight against diabetes: overweight and obesity. It gives considerable prominence to the view that overweight and obesity (and the obesity 'epidemic') are behind increases in the incidence of diabetes, masking the complex etiology of the disease. And this is exactly how it was taken up in the media. The study received strong coverage at the time, alongside other reports about Australia's alleged obesity epidemic. Headlines included 'Weighty problems set to spark 'diabesity' epidemic' (O'Dwyer 2001). Interviews with the authors stated the AusDiab Study – 2000 had shown that a 'diabesity epidemic would hit Australia within 10 years' (O'Dwyer 2001). By early 2002, some commentators were linking Australia's emerging 'diabesity epidemic' to a global epidemic of diabesity that was 'expected' to be upon us by the end of the century and would 'rival the HIV-AIDS epidemic' (Advertiser 2002).

The influential and heavily cited AusDiab Study – 2000 also produced a five-year follow up study of the same cohort, called *The Australian Diabetes, Obesity and Lifestyle Study (AusDiab 2005)*: Tracking the accelerating epidemic, its causes and outcomes (Barr et al. 2006). Again, overweight and obesity are the focus of a dedicated chapter appearing immediately after the definition of diabetes. In this report, the authors describe the disease as follows, with *no* explicit mention of non-modifiable factors:

Diabetes is a disease with mixed aetiology. There are many risk factors for the development of the disease including obesity, hypertension, sedentary lifestyle, dyslipidaemia and the metabolic syndrome ... (Barr et al. 2006, 6)

The AusDiab Study – 2005 (2006) reports that in the five years since the first study, the incidence of new diabetes in the cohort increased by a modest 0.8%. It also reported that those aged less than 65 years gained 1.8 kg, and those aged over 65 years lost 0.8 kg over the period – again a modest change (Barr et al. 2006, xi). Nevertheless, the authors continue to use the language of 'epidemic' and to link not only diabetes, but now also pre-diabetes, to the 'obesity epidemic' in Australia (Barr et al. 2006, 7).

Every two years, the Australian Government, via the Australian Institute of Health and Welfare (AIHW) produces a report on the health of the nation, based on self-reported data. A critical review of these reports from their inception in 1988 reveals an important shift. In the earlier reports ‘diet, relative weight and physical inactivity’ along with under nutrition are identified as ‘factors influencing risk’ for T2DM (AIHW 1988, 90, 116–119, 1990, 1992). However, from 2000 onwards, and following the WHO report (1998), overweight and – in particular – obesity emerge as *the* most significant risk factors, with purported increases in diabetes rates increasingly linked to increases in the girth of the population.

In the 1988–2006 reports, explanations of the onset of diabetes include a range of ‘risk’ factors both modifiable and non-modifiable. However, from 2002 the AusDiab – 2000 Study is cited extensively, and it is here that obesity begins to emerge as an important driver of diabetes in Australia. For example, in the AIWA Report of 2002 we find the following:

Dunstan et al. (2001) suggest that the increased prevalence of obesity in Australia has been a significant contributing factor in the increasing prevalence of diabetes, in particular Type 2 diabetes (p. 68).

The AusDiab Studies (2001, 2005) have been cited extensively in all government health reports up to the present day. Although the term ‘diabesity’ was not generally used, these studies helped establish a position in which obesity is deemed to contribute to increasing rates of diabetes. In 2008, obesity was added to the list of Australian Nation Health Priority Areas (NHPA). In that same year, obesity was clearly identified as a central or primary cause of rises in the incidence of diabetes, in the AIHW government health report, for example:

there is likely to be strong growth in the level of diabetes over the next 20 years, mostly as a direct result of increasing levels of obesity. (AIHW 2008, 158)

By 2010, however, the AIHW Report explained the increase in diabetes as follows: ‘Over the longer term and accounting for population changes, Type 2 diabetes is projected to become the leading cause of disease burden by 2023, partly attributable to the expanding problem of overweight and obesity’ (AIHW 2010, 58). By 2012, we find this: ‘The proportion of Australian adults who do not already have Type 2 diabetes but are overweight or obese is an indicator of Australian adults who are at risk for Type 2 diabetes’ (AIHW 2012, 266). Once again, the 56% of people who (according to the 2001 AusDiab Study – 2000) are not obese but have diabetes are ‘displaced’ from the discourse above in lieu of an emphasis on fat and risk.

Obesity = diabetes = diabesity = epidemic(s)

It is also noteworthy that over time, when listing the risk factors for diabetes in Government reports and websites (as well as NGOs like Diabetes Australia), obesity and/or overweight became increasingly prominent: listed first in most instances or second after aging. For example, diabetes and its risk factors are discussed at several points throughout the 2010 AIHW report, but only *once* are genetics, age, family history, and ethnic background mentioned in association with diabetes (152). The authors then immediately conclude:

[W]hile these risk factors cannot be changed, there are also a number of modifiable risk factors for Type 2 diabetes – notably obesity, physical inactivity and an unhealthy diet (Shaw and Chisholm 2003). Therefore, Type 2 diabetes is highly preventable. (AIHW 2010, 152)

For those with a family history of the disease or genetic susceptibility to it, asserting that diabetes is preventable for those willing to modify their lifestyle overstates the situation and the challenges faced in ‘managing’ the disease, while overlooking factors beyond individual control.

Repositioning overweight and obesity as the generator of diabetes and diabetes has also been replicated in the media. For example, ‘Diabetes on rise, experts say’ is the headline from an interview with Paul Zimmet, one of the authors of the AusDiab studies. In the interview, Zimmet stated, ‘JUNK food and soft drinks should be banned from hospitals, schools and other public institutions to help stem growing ‘diabetes’ (Sydney Morning Herald 2006). Later in the story and directly below Zimmet’s credentials as the director of the International Diabetes Institute, we find this statement, ‘Obesity is strongly linked with type-2 diabetes’; no other factors are listed (Sydney Morning Herald 2006).

Today, this re-positioning and re-signification is ubiquitous on commercial medical websites and those of national sports celebrities where health is the central theme. This, for example, is from well-known health and fitness celebrity Guy Leech’s web site:

Type 2 diabetes is the most common form of diabetes. It was once called, “Mature onset diabetes” but inline with increasing obesity trends and the lowering of the age when sufferers become afflicted with this illness this name has been dropped. In fact, so close is the relationship between obesity and Type 2 diabetes that the term “diabetes” has been coined (www.guyleechfitness.com).

Alongside the framing of weight as a, or *the* central ‘risk’ factor for diabetes, there are also examples of obesity being named as a cause or *the* cause of diabetes. For example, Cornes et al. (2009:75) introduce their article on genetic variants in diabetes and BMI by stating that: ‘obesity causes or exacerbates many health problems including type 2 diabetes mellitus, cardiovascular disease, certain forms of cancer, respiratory complications and osteoarthritis’. Again, this is also evident in media coverage on the disease. For example, Associate Professor Maarten Kamp, a diabetes expert from the state of Queensland, has commented that ‘Obesity is a very important factor in contributing to diabetes, it’s certainly the main factor that’s contributing to the epidemic of diabetes that we have’ (NineMSN 2006).

The obesity/diabetes or diabetes epidemic ‘down under’

Reference to the emergence of a ‘twin’ or ‘diabetes epidemic’ fuelled by obesity has become commonplace in Australia, driven in no small part by the AusDiab Studies (2001, 2005), media commentary by its authors and government health priorities, reports, and education campaigns. Indeed, there appears to be broad social and political acceptance of the view. For example, since the late 1990s, Australian Federal Health Ministers have asserted that ‘Australians are facing a diabetes epidemic within 10 years if immediate changes are not made to our lifestyle and diet’ or that ‘Australia’s ageing, increasingly overweight and inactive population means the nation is well on the way to a diabetes epidemic’ (Australian Government 1999). More recently, headlines stating,

‘The Federal Government has reaffirmed its commitment to tackling the diabetes epidemic’ (Brimbank Weekly 2010) and reports of new ‘frontiers’ under threat, ‘Diabetes epidemic looms in regional Queensland’ frequently appear (ABC 2011a)

The idea that a diabetes epidemic is being driven by the earlier and ongoing ‘obesity epidemic’ has gained considerable ground in the media and in public health. By 2011, a senior commentator for the Dietitians Association of Australia speaking at their National Conference expressed her concern about the ‘twin epidemics’ of obesity and diabetes as follows:

We are looking at an avalanche really, of people who are, who have become obese and are going to develop diabetes. It has very serious consequences for our health budget and for our community and for those individuals. (ABC 2011b)

The causative undertone of the ‘twin’ or ‘diabetes epidemic’, i.e the ‘obesity-leads-to-diabetes’ frame, is again highly visible, as the quote suggests.

In the ways the term diabetes is used in Australia, the independent identities of obesity and diabetes are merged even further and become the looming and threatening ‘diabetes epidemic’. For example, Prof. Rob Moodie, head of Australia’s National Preventative Health Taskforce, which is assigned with ‘turning around’ the obesity epidemic in Australia, described the situation as follows:

Its going to require a massive effort to stop the increases in overweight and obesity ... also that obviously has a major impact on levels of diabetes so there is a notion of a sort of ‘diabetes’ epidemic – the double epidemic of obesity and diabetes – [as] something that we have to take very seriously. (ABC 2008)

Paul Zimmet has suggested that the UN declare diabetes ‘an international health disaster’ (The Australian 2006) and that rising international rates ‘threaten to consume world economies and bankrupt health systems’ including Australia’s (Zimmet 2000; 2001). In an editorial in the *Medical Journal of Australia*, Zimmet and James (2006, 187–88) argue that our consumption of unhealthy foods and ‘ever more sedentary leisure’ sees Australia ‘facing a seemingly unstoppable juggernaut of obesity and diabetes’. Zimmet has also stated that Indigenous Australians and other Aboriginal Metis, or First Nation peoples are at great risk of partial ‘if not total extinction within this century’ from diabetes (The Australian 2006; Zimmet 2001). As Fee (2006) has demonstrated, heavily racialized statements like these, with their imagery of death and extinction, are part of larger, post-colonial interpretations of obesity and diabetes in which notions of ‘race’ are used uncritically, as a proxy for presumed genetic differences.

At another level, linking diabetes and overweight/obesity together in such politically charged ways, serves to elevate the role of overweight and obesity as risk factors or, more disturbingly, as causes of diabetes. Conflating the two in the term ‘diabetes’ or ‘diabetes epidemic’ implies a single, all pervasive cause for diabetes nationally and globally, namely obesity (overweight/ obesity). According to Haslam (2012), this usage masks the fact that:

Obese individuals have a host of other comorbidities to contend with in addition to type 2 diabetes, [that] not everyone who is obese will develop type 2 diabetes, [and] not everyone with a high risk of type 2 diabetes is necessarily obese. (334)

Yet, despite the problems with this conflation, it has become thoroughly naturalized amongst Australian researchers, policy-makers, media, and the public. Today, there is an annual ‘Diabetes and Diabetes Day’ in Australia – an education event where Allied Health Professionals provide information to the public – and leading Australian academics in the field of diabetes are on the editorial board of a new academic journal called *Diabetes in Practice* (<http://www.diabetesinpractice.co.uk/>).

Consequences of the conflation

The growing emphasis on diabetes and/or the obesity/diabetes epidemic must be located within a broader cultural context in which overweight and obesity have come to act as powerful cultural signifiers for poor health and a range of diseases. Increasingly, the larger-than-average body has come to signify a person who, by virtue of their girth, engages in risky behavior and an unhealthy irresponsible lifestyle that places them at risk for a range of health issues and diseases (Austin 1999; Bell and McNaughton 2007; Bell, McNaughton, and Salmon 2009, 2011; Campos 2004; Campos et al. 2006; Gard and Wright 2005; Jutel 2001, 2008; Le Besco 2011; McNaughton 2011; Monaghan 2008; Murray 2008; Petersen and Lupton 1997).

For example, in the 1980s and 1990s, Watson (1993, 248) and Crawford’s (1984, 70–71) US-based studies, found that those whose body weights were in the highest ranges were consistently assumed to be the persons most likely to be ‘unhealthy’. In an Australian study on cholesterol, participants commonly assumed a direct link between body shape and cholesterol: fatness, inactivity, and over-nutrition equaled high cholesterol for many (Lupton and Chapman 1995, 488). Davison, Smith, and Frankel’s (1991) research into heart disease demonstrated that ‘obese’ men and women were deemed the most likely ‘contenders’ for heart disease (see also Backett, Davison, and Mullen 1994, Davison, Frankel, and Smith 1992). Such prejudices are not limited to the lay public, as there is considerable evidence to suggest that health care providers hold negative and prejudicial attitudes towards overweight patients, assuming that weight is the source of their ills (Anderson and Wadden 2004; Ferraro and Holland 2002; Oberrieder et al. 1995; Teachman and Brownwell 2001).

The idea of an obesity/diabetes epidemic therefore crystallizes broader assumptions about the dangerousness of fat and the idea that poor health is simply the result of changeable, risky behaviors which in turn, have created another potentially more deadly scourge in the form of a ‘diabetes’ epidemic. Overweight and obese people are imagined either as diabetic or as inevitably becoming diabetic. In this framing, the overweight or obese body/person signifies a diseased body/person, or a body/person with high disease potential – diseases that are self-inflicted through risky behavior. There is little room here for the idea of weight as a symptom of diabetes, for the notion that some overweight or obese people never develop the disease, or that some people whose body weight is considered ‘normal’ will be diagnosed with T2DM.

Moralising obesity/diabetes: blame, shame and individual responsibility

This convergence of weight with diabetes opens the way for what Finerman and Bennett (1995) called ‘a blame-oriented model’, wherein responsibility and blame for ‘disease, its onset and outcomes are ... ascribed to the afflicted’. Diagnostically, the response to the onset of T2DM or indications that it may be developing is glycemic control, via diet, weight loss, exercise, medications or insulin, often in combination.

However, as the authors of AIHW report (2010) note, ‘Type 2 diabetes *may* be managed with *changes* to diet and exercise, oral glucose lowering drugs, insulin injections or a combination of these’ (AIHW 2008, 3, my emphasis). As this statement suggests, for some, diabetes is not easily managed by diet, exercise and/or medications, although this is rarely stated explicitly in the literature examined here. Furthermore, disease progression can occur even with optimal management.

As van der Does (1997, 34) notes, ‘in Type 2 diabetes, definite conclusions about the role of long-term glycaemic control in preventing complications cannot be drawn yet’. Nevertheless, weight reduction or management is often a central aim of glucose control. However, if patients are not losing weight or struggling to do so, health staff may lose patience or express a lack of empathy, shaming or further stigmatizing them (Cossrow, Jeffery, and Mc Quire 2001; Teixeira and Budd 2010). Patients may also blame themselves for their ‘failure’ or be made to feel responsible for their diabetes in a way that is shaming or stigmatizing. Conversely, a lack of emphasis on the limitations of current management strategies leads some patients to develop unrealistic expectations that cannot be met or to believe they have been cured as Parry et al. (2006, 100) have shown.

Conversations about weight, weight loss, diet, and exercise are commonplace in health care encounters around T2DM and several studies suggest that experiencing or expecting weight discrimination may be causing people to evade or delay health care or treatment. For example, Teixeira and Budd (2010) suggest that the shame of not having lost weight and the fear of being reprimanded are a key factor in T2DM patients postponing or avoiding follow-up visits. Peyrot, McMurry, and Kruger (1999) study into possible psychological barriers to diabetes care, also found that many participants with T2DM felt very anxious and ashamed about their weight.

In a similar vein, a survey undertaken in Aotearoa–New Zealand by Simmons et al. (2007) indicated that obesity was an obstacle to patients seeking diabetes support, along with other health conditions and economic barriers. Another study by Drury and Louis (2002) indicates a direct correlation between avoidance or delay of health care with women’s perceptions of weight and stigma. Although this study did not consider men’s experiences, broader arguments about men, service use, and weight stigma would suggest that they too, may be avoiding health care (Bell and McNaughton 2007, Monaghan 2008).

Teixeira and Budd (2010) argue that obesity stigma is a likely barrier to ongoing diabetes management and that health providers need to improve their sensitivity, devote more time to interactions with overweight patients, and reflect more self critically on their own assumptions about weight and how these are being communicated to clients. They also call for health staff to employ particular counseling strategies with their ‘obese’ T2DM patients that will improve disease management and reduce patients’ experience of weight stigma.

Although Teixeira and Budd (2010) assume that most diabetics are overweight, overlooking those who may not be overweight but are still at risk for the disease, their points regarding the potentially negative impacts of weight stigma on T2DM patients echo a larger body of literature suggesting that the stigma associated with particular conditions and practices (e.g. HIV/AIDS, illicit drug use, tobacco use, etc.) often creates substantial barriers to health care. Studies into the stigmatizing of drug users, smokers, consumers of alcohol, and those with HIV–AIDS strongly indicates that stigmatizing and marginalizing people in these ways is likely to leave them alienated, anxious, and removed from the kinds of services that they might need and potentially with poor mental and physical health as a result (Krieger 2000; Kvernmo and Heyerdahl 2003; Link and Phelan 2001; Mason 2001; Puhl and Heuer 2009, 2010; Todd and Fisher 1993).

There are a number of studies indicating that the more insidious, subtle daily expressions of discrimination provide a powerful baseline from which patients evaluate their health care experiences (Browne 2003; Johnson et al. 2004) and that this may discourage people from seeking support or attending follow-up visits.

While there have been calls internationally and in Australia to de-normalize fatness through the use of shaming and stigma, the taxing of ‘junk’ food, removal of overweight and/or diabetic children from their parents and so on, the ethical and iatrogenic consequences of this are likely to be considerable as I and others have shown (Bell, McNaughton, and Salmon 2009, 2011; McNaughton 2011). Like Burris (2008, 475), I would argue that stigma cannot be used in an ethical way, because it is, by definition, an ‘arbitrary and cruel form of social control’ and a form of violence. Following Burris’ (2008, 475) I would also ask: ‘where is there good evidence that inculcating a sense of a spoiled identity is a good way to get people to adopt healthier behaviours?’

Conclusion

Clearly, the idea that fatness is the most significant ‘risk factor’ or *central* cause of Type 2 diabetes mellitus has become pervasive and thoroughly naturalized in Australian media representations, public health campaigns, popular discourse, and scholarly research. In this framing, the gaps in current knowledge around the causes of diabetes or the role of weight in insulin resistance, or as a symptom of T2DM are elided. Factors that are not linked to ‘lifestyle’ are less open to change (and intervention), or beyond individual control – such as genetics, poverty, family history etc. – are given less emphasis or omitted entirely. In the weight-causes-diabetes framing and in the creation of a new condition ‘diabesity’ in which the two fully merge, the complexity and multiplicity of causes associated with the disease are lost or rendered invisible. All that can be seen are obesity, diabetes, and the unhealthy lifestyle that allegedly created them. Here, weight, and to a lesser degree diabetes, are framed as primarily self-inflicted: the result of wholly changeable and highly risky lifestyle factors – notably over-nutrition and physical inactivity.

This ‘fatness causes diabetes’ linkage overstates the evidence, turns symptoms into causes, and understates the complexity of the issue (also see Burris 2008). Given that T2DM can emerge in people from a range of weight ranges – including those considered ‘healthy’ – such a correlation may have ethical implications for their experience and interpretation of the symptoms of the disease, its prevention, and diagnosis.

Notes

1. Data was collected from a stratified sample of blood tests, taken from 11,247 Australians aged 25 years or over, residing in 42 randomly selected urban and non-urban areas (Census Collector Districts) of the six states of Australia and the Northern Territory (Dunstan et al. 2001:6).
2. Haslam (2012, 331) has demonstrated that the term ‘diabesity’ was invented and first used by Sims (1973), in reference to the ‘mutual causes of impaired glycaemic control and weight gain’ and their amelioration.

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