THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

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This Issue in the Journal

Addictive overeating: lessons learned from medical students' perceptions of Overeaters Anonymous
Ria Schroder, Doug Sellman, Jane Elmslie

Obesity is a serious and debilitating medical condition which can impair both physical and psychological health. Addictive behaviours, especially tobacco smoking and compulsive alcohol use are accepted as important causes of ill health in modern society. To conceive that something as fundamental to human survival as food has the potential to trigger compulsive behaviour that may seriously compromise health and wellbeing is perhaps counterintuitive. This study viewed the concept of 'addictive overeating' through the eyes of a group of fifth-year medical students who attended Overeaters Anonymous meetings as part of their course requirements. The study findings highlight the value of developing empathy in trainee doctors towards patients with obesity and suggest that exposure to groups such as Overeaters Anonymous during training may better equip doctors and other health professionals to become more therapeutically effective with this major health problem.

Ethnic-specific body mass index cut-off points for overweight and obesity in girls
J Scott Duncan, Elizabeth K Duncan, Grant Schofield

This study follows on from a previous publication that demonstrated the considerable differences in body fat at a given height and weight in girls from NZ's five major ethnic groups. As our current classification system depends on a weight-based index (BMI), this means that our estimates of overweight and obesity in girls could be inaccurate. In this paper, we provide the first ethnic-adjusted BMI thresholds for detecting overweight and obesity in Maori, Pacific Island, East Asian, and South Asian girls. We conclude that current BMI thresholds should be lowered for South and East Asian girls and raised for Maori and Pacific Island girls to represent the same level of body fatness as European girls. Such changes could result in dramatic changes to our understanding of obesity in these population groups.

Overweight and obesity prevalence among adult Pacific peoples and Europeans in the Diabetes Heart and Health Study (DHAHS) 2002–2003, Auckland New Zealand
Gerhard Sundborn, Patricia A Metcalf, Dudley Gentles, Robert Scragg, Lorna Dyall, Peter Black, Rod Jackson

This paper describes and compares proportions of overweight and obese Pacific (Samoan, Tongan, Niue, Cook Islands) and European New Zealanders who participated in the DHAHS 2002–2003 survey. 1011 Pacific people were surveyed of whom almost all are now overweight or obese.

Healthier vending machines in workplaces: both possible and effective
Delvina Gorton, Julie Carter, Branko Cvjetan, Cliona Ni Mhurchu

Vending machines typically supply snack foods with poor nutritional quality, with few if any healthier choices. *Better Vending for Health Guidelines* were developed by a collaboration of health organisations in Auckland to guide provision of healthier options in vending machines. The guidelines were trialled in a hospital workplace to determine their impact on nutrition content and sales of vending products. The amount of total energy, saturated fat, and sugar sold decreased substantially while total sales volume was not affected. The guidelines appear to be an effective way of encouraging provision of healthier options in vending machines.

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The right diet—'sweet as'?

Anne-Thea McGill

When it comes to medical treatment of any condition we are well used to the adage 'above all do no harm' but perhaps we should make sure that we 'really do good'. The complicated, expanding fields of obesity prevention and management are doing less well than those of tobacco management.[1]

Giving an (artificial) agent that does no overt or immediate harm, but no good either, is not adequate. On further examination, there appear to be a range of concerns with agents generally assumed to help weight loss and obesity related disease. These include food additives designed to enhance qualities such as sweetness.[2,3] These additives are variably tested for toxicity,[4] efficacy[2] yet extensively for industrial application.[5] Also problematic are modified food extracts (nutraceuticals),[6] alternative therapies,[7] supplements[8] (vitamin, mineral and other) and drugs.[9–12] Some of these agents may not even be harmless or stop weight gain, and some have noticeable adverse effects.

The problem may be the widely held assumption that obesity is the result of a simple energy equation; 'if energy in is greater than energy out then fat gain results'. The presumption is that energy in food, as conventionally measured by calimetry, is 'burned' uniformly and predictably by complex organisms such as humans, irrespective of overall nutrition.

It is also presumed that body fat *per se* is one of the major risks for Type 2 diabetes mellitus [TIIDM] cardiovascular disease [CVD] and cancers.[13] But are we sure about the 'energy in/energy out' equation? Can Western food-type low-energy 'diets' or weight management programmes be confidently associated with sustained weight loss, reduced weight gain, and/or health improvement?

In the carbohydrate domain alone, low energy diets, especially those advised for TIIDM patients in the community, usually include sugar replacements (artificial sweeteners or non-nutritive sweeteners [NNS]). Arguably, they contribute to generally lower energy intakes results.[14–16] These novel chemicals (and there are more in the production pipeline) are much cheaper to insert into food and beverages, and worth $US 147bn to the global NNS market.[17]

There is pressure (and controversy) involved in formulation, extraction and marketing of ever greater volumes, and types of NNS.[5] Aspartame (a methyl ester of a phenylalanine/aspartic acid dipeptide)—discussed in this issue of the *Journal* by Dr Magnuson (http://www.nzma.org.nz/journal/123-1311/4024)—is one of 17 sweeteners permitted in New Zealand, and is widely reported as present in 6000 American food/beverage items.

Aspartame is often added in combination with other NNS, although the amounts in each item are not required to be revealed on packet labels or to the Federal Drug Agency (FDA).[2] Along with many 'convenience' food additives this agent has a long

history of controversial studies, (of which some concerned with cancer are covered in this issue) and registration by the FDA was delayed. Some of its toxicity profile is reviewed, although the article does not mention aspartame's Maillard reactions under certain conditions.[18,19] (Maillard reactions are non-enzymatic 'reducing sugar' and amino acid reactions occurring in biologic systems e.g. in food; on standing, but especially on heating, lending the browning colour and strong flavours).

Aspartame should not be heated as it decomposes and loses its sweetness. Maillard reactions with asparagine (derived from aspartic acid) have been shown to produce carcinogens such as acrylamide.[20] Even in stored powder/fluid beverage mixes, aspartame can degrade over time, form Maillard products and requires chemical and pH buffering. Although a few studies show ingestion of Maillard reaction products in food increased serum antioxidant activity,[21] many are associated with oxidative stress and related degenerative disease including TIIDM, CVD and Alzheimer's disease.[22–25] In TIIDM patients, where *in vivo* high levels of advanced glycation (Maillard) end-products [AGEs] form due to hypergylcaemia, foods low in these products are advised.

Weight loss has been shown with aspartame use in controlled energy decreased diet studies, as reviewed in one paper,[26] and critiqued as not including certain studies.[15] Concerns about whether NNS are associated with weight gain date back at least to 1986, when an epidemiological study showed weight gain in users, and recently another prospective study showed similar results.[14,15] Furthermore, diet sodas have been associated with incident metabolic syndrome and TIIDM.[16]

In reviewing the mechanisms of action of NNS, research on artificial sweetening of foods in a series of studies in rats indicates that sensory experiences of NNS do not predict energy intake consequences and can alter the ability to control food intake.[27] Unfortunately, body weight in various studies not only does not decrease or remain the same but the interference with energy intake prediction appears to result in body weight gain.[3] This has been shown with a number of sweeteners (sucralose and AME-k), and can blunt or underestimate future energy prediction when sweetness is associated with energy take.[27,28]

Furthermore, adverse gut flora changes are recorded.[29] Well-known clinical nutrition researchers on taste and sugar effects in nutrition discuss the many studies of efficacy and possible mechanisms of NNS in a wide-ranging overview.[2] They and others query whether the increase in sugar and sweet drinks consumption may be enhanced since the widespread use of NNS, especially when NNS are consumed without an energy controlled diet.[2,3,14-16,30]

Thus much dietary advice for both fat loss and reducing CVD is about just reducing energy, although which is the most efficacious macronutrient (carbohydrate or fat) for weight loss remains unresolved.[31–34] We do know that people who eat diets high in fruit, vegetable and fibre as advised by various CVD (NCEP Therapeutic Lifestyle Changes,[35,36] Dietary Approaches to Stop Hypertension (DASH)[37,38]) and cancer[39,40] societies, and low in man-made chemicals[41]do better at decreasing degenerative disease and risk factors, and improving their health overall.

The fact that they also lose weight (or maintain lower weights) is put down to energy dilution, as fruit and vegetables have higher percentages of fibre and water. However,

the most studied, healthy dietary pattern, the traditional Mediterranean diet, is proportionately high in energy from fat (approximately 38%, equivalent to a typical Western diet[42]) but this is almost all oil from cold pressed olives. In addition, there are large quantities of vegetables, fruit , variable levels of dairy and fish and, of course, nutrient-containing fibre.[42–49]

The 'energy in: energy out' equation itself may have to be re-examined. We now have better scientific evidence of the longest running experiment at our door step - that of human diets through evolution and history.[50] Since the advent of controlled clinical trials none, to my knowledge, has tested the effect on weight and health of people who are allowed to eat as much whole food as they want, although some are getting closer to this type of eating pattern.[51] (Whole food is minimally-processed animal protein, nuts, fruits, vegetables, and a little raw honey—and they can be preserved: cooked, frozen, tinned, dried, fermented and pickled.)

The catch is that highly bred and /or processed food stuffs that are intrinsically highly energy dense, palatable (and often made chemically more so with NNS and other additives), would need to be nearly totally excluded. Starch or sugar products, refined and processed from grain, tuber and similar plant sources, often combined with industrialised fats/oils, would be returned to 'two or three festive occasions a year-type' foods.

Confectionary, biscuits and refined breads are not staple energy foods. A modest intake of whole grain or thick skinned, old fashioned, potato starches may be required for the young, slim and active. In simple terms, the re-introduction of *any* type of micronutrient-dense whole food/traditional diets with greatly increased proportions of many cofactors, vitamins and minerals would allow food energy, and body fat, to be mobilised.

Such high volume diets are filling (satiating), generally self-limiting and difficult to over-consume, and interestingly, not incompatible with the major parts of dietary recommendations.[52] Any 'excess' energy combined with plenty of plant (with fibre) and free-range animal derived nutrients, would contribute to tissue repair, reducing degenerative disease.[53,54] To re-normalise this micronutrient dense pattern of eating, with its complex tastes, flies in the face of processed food industries that market over-palatable, damaged food.

Our first sweetener producers (bees) also pollinate approximately one-third of our food plants, and are seriously threatened by industrial farming practices.[54] However, demand is already spurring on the budding, environmentally and technologically smart, rather than factory, farming industry.[54,56,57]

Importantly, even now (anecdotally) very overweight people and TIIDM patients whose glycaemia is out of control, with some help (counselling, medication) in controlling urges to eat processed energy-dense food, are making this realistic, although difficult, choice. They know, once told about evolution, that they are really following common prudent dietary advice and that they: lose weight, are more healthy, are more inclined to be physically active, are not hungry and they can have plenty of sweet (and dried) fruit.

There is now little need to consume artificial sweeteners, to 'portion control' or to 'count calories'—in or out.

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Bariatric surgery: a dilemma for the health system?

Jonathan Foo, Robyn Toomath, S Kusal Wickremesekera, Simon Bann, Richard Stubbs

It has been 5 years since Professor Iain Martin commented in a *NZMJ* editorial that bariatric surgery should be accessible for patients through the public system.[1] An important caveat mentioned in that editorial was his concern regarding the mortality and morbidity associated with bariatric surgery.

Since that time, surgical techniques and perioperative management have been refined to reduce complications to very low levels. Yet, over the same time period, only minor progress has been made with the provision of public funding of bariatric surgery. The lack of publicly funded bariatric surgery is not due to a falling rate in obesity; in fact, the World Health Organization has recently recognised obesity as being one of the five greatest risk factors for global mortality.

New Zealand is part of this obesity pandemic, with the prevalence of obesity having risen from 11% in 1977 to 26.5% in 2007, and with 2% of males and 2.8% of females currently being classified as morbidly obese (BMI ≥40).[2] This increase in the prevalence of obesity has occurred with significant disparity between ethnic groups, with Māori and Pacific Island groups having a three to six fold increase in rates of morbid obesity.[3]

The increase in rates of obesity is associated with increasing rates of serious comorbidities, including Type 2 diabetes, hypertension, dyslipidaemia, polycystic ovary disease, musculoskeletal problems and obstructive sleep apnoea. All of these conditions continue to affect quality and length of life despite current efforts at treatment. Furthermore, it has been estimated that for every 5-point increase in BMI over 25 there is a 30% increase in risk of mortality.[4]

Obesity also carries significant socioeconomic implications with those affected often being poorer, less well educated and less likely to marry.[5] Indeed, the economic burden of obesity for society is large and will undoubtedly escalate. This relates not only to the direct cost of healthcare for such individuals, but also the loss of valuable human resource and diminished consumer spending within society.

Although it is laudable that we have been developing national healthy eating and lifestyle strategies, treatment strategies for severe obesity must also be explored. While conventional approaches including dietary and lifestyle modifications, pharmacotherapy and behavioural therapies can achieve short term weight loss of between 1.1 and 6.5kg, attrition rates of 20-45% are observed.[6]

While such levels of weight loss may be of value for the overweight, they are of limited value to those with morbid obesity. For too long society has attributed obesity to sloth and gluttony, while ignoring what are probably far more important, if ill understood factors. The increasing prevalence of obesity is probably due to an "obseogenic" environment with behavioural and important biological influences.

The morbidly obese, at the extreme end of the scale, are thought to have a significant genetic component to their disorder. Weight regulation is controlled centrally by the hypothalamus and brain stem and both the brain and gut work in concert to counter any attempts to lose weight. Thus, weight loss provokes a compensatory response from the gut neuroendocrine axis to increase the orexigenic hormone, ghrelin and decrease satiety hormones such as peptide YY, cholecystokinin and leptin. Internal autoregulation therefore causes the body to resist weight loss and if large amounts of weight are lost, post-starvation hyperphagia occurs with disproportionate fat regain.

What then can be done? Surgery can provide an answer. Large volumes of bariatric surgery are being performed worldwide with the American Society for Metabolic and Bariatric Surgery reporting that 220,000 bariatric procedures are now being performed annually in the United States. This represents a paradigm shift over the last few decades, and one which New Zealand needs to join. A growing number of New Zealand surgeons are offering a variety of procedures, but with few exceptions, these are being performed in the private sector. Surgical mortality can be expected to be less than 1% with major morbidity of less than 5%.[7]

Numerous international studies have now established improved life expectancy following bariatric surgery[8,9] and the effect on comorbidities following surgery is nothing short of extraordinary. A recent meta-analysis shows Type 2 diabetes completely resolving in 77%, hyperlipidaemia improving or resolving in 70%, hypertension resolving in 62% and obstructive sleep apnoea resolving in 86%.[10] These potential benefits are reflected in the Ministry of Health's projections that bariatric surgery would be cost neutral after 5 years and potentially cost saving by 8 years.[11]

A Canadian experience which followed 1000 patients who had undergone bariatric surgery for 5 years, and 6000 who had not, revealed a cost neutral position after only 3.5 years.[12] Given the accuracy with which Canadian individual healthcare costs can be tracked, this particular cost analysis is of real value.

The New Zealand National Service and Technology Review Advisory Committee from the Ministry of Health recently recommended that a minimum of 915 bariatric procedures (0.5% of the severely obese population) be performed annually. Despite this recommendation and its potential benefits, bariatric surgery remains largely confined to the private sector, for much of the country.

The almost non-availability of bariatric surgery in the public sector is of serious concern, when 1 in 4 of New Zealand adults are classified as obese and nearly 40% of adults in our most disadvantaged regions (NZDep2006 quintile 5) are obese.[2] Such individuals not only have a high rate of obesity but also high rates of dyslipidaemia, diabetes and an elevated risk of cardiovascular disease.

This cohort of patients will experience debilitating chronic disease over the course of their lives. Bariatric surgery provides a means to alleviate that debility, and can be expected to be cost neutral within 5 years.

Prejudice aside, it is a moral, practical and fiscal duty of the New Zealand health system to make bariatric surgery accessible for the morbidly obese.

Competing interests: None known.

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Addictive overeating: lessons learned from medical students' perceptions of Overeaters Anonymous

Ria Schroder, Doug Sellman, Jane Elmslie

Abstract

Aim To explore medical students' perceptions of Overeaters Anonymous (OA).

Method 72 fifth-year medical students' reports of their experiences of attending OA meetings were analysed using thematic analysis. Students were required to submit these reports in order to complete a compulsory component of the Addiction Medicine run in their medical training at the University of Otago, Christchurch during 2002–2007.

Results Analysis of the students' reports highlights an emerging understanding of the concepts of addiction in general and addictive overeating in particular. This understanding is reflected in the students' acceptance of addictive overeating as a potential disorder and their increased empathy and confidence in working with patients with this disorder.

Conclusions Improving treatment for people with obesity is a major contemporary health challenge. Addictive overeating could be a critical element in understanding the nature of obesity but has not been the subject of extensive research to date. Medical students in this study discovered that addiction to food is not just a theoretical construct but fits with the actual experience of people. The poignancy of these narratives illustrates how such information can promote greater understanding of medical and other life issues which may benefit their identification and treatment.

Addictive overeating can be conceived as an ego-dystonic compulsion to overeat, despite the person recognising the harm it is causing, similar to the compulsion to use drugs in drug addiction.[1] An increasing body of evidence now exists to suggest that highly pleasurable foods (particularly those high in sugars, salt and fats) have the potential to initiate compulsive behaviour which can result in a clinical state of addictive overeating.[1–7]

While the availability of energy dense fast foods and the cost differential between 'healthy foods' and 'junk foods' are clearly established risk factors for the development of obesity, there remains considerable scepticism about the possibility that some people might be "addicted to food". The terms "food addiction" and "addictive overeating" are viewed by many as convenient excuses for excessive food intake and clinicians rarely consider the role that addictive overeating may have in the development of overweight and obesity.

In contrast, drug addiction and other associated compulsive behavioural disorders such as pathological gambling are recognised as mental disorders that can severely impact the lives of the addicted person, their family and friends. Consequently, advances have been made in research and clinical intervention to assist people to

recover from these addictions. To date, however, this same perspective has not been applied to addictive overeating. In a climate where disbelief and negative stereotypes have dominated, little information exists that portrays the lived experiences of people who have problems with controlling their food intake.

One forum, however, that has been committed to hearing about the lived experiences of overeaters worldwide is Overeaters Anonymous (OA). OA is a self-help group for compulsive overeaters and follows the same 12 Step principles as Alcoholics Anonymous. Members of OA consider compulsive eating to be a threefold disease comprising physical, emotional and spiritual components that can be arrested through abstinence from compulsive eating, but cannot be cured.[8,9]

Regardless of whether or not one subscribes to the particular theoretical model that underlies OA, it is clear that OA has provided a forum for overeaters to meet for support and to feel safe to share their stories. OA has also provided a forum in which clinicians and researchers are able to learn more about overeating by hearing the experiences of people who identify as overeaters. To date however, the lessons that have been learned about overeating from OA have been mostly restricted to those people who have had direct contact with OA and its members.

The aim of this paper is to examine this collective experience through the lens of a group of fifth-year medical students who had attended an OA meeting as part of their course requirements. A systematic analysis involving a qualitative research method was used to analyse the students' observations about people's experiences of food addiction and the impact this addiction can have on individuals' daily lives.

Method

Background—As part of the course requirements for the Addiction Medicine component of their training, all fifth-year medical students at the University of Otago Christchurch are required to attend one self-help group meeting and to write a report about this experience. The data presented in this paper are extracts from reports written by the fifth-year medical students in 2002–2007 who attended an OA meeting. Over this period 72 (19.5%) out of a total of 370 students chose to attend an OA meeting. Of the remaining 298 students, 214 (57.8%) attended Alcoholics Anonymous (AA), 44 (11.9%) Narcotics Anonymous (NA), 27 (7.3%) Gamblers Anonymous (GA), 10 (2.7%) AL-ANON and three (0.8%) Rational Recovery.

In 2007 permission was given by the University of Otago Student Body to use the submitted reports to conduct a thematic analysis. The anonymity of all students was maintained by removing all student names and identifying details from the reports prior to analysis.

Representatives from OA who had been involved in teaching the students and facilitating their access to the OA groups also gave permission for the student reports to be analysed and for this paper to be written.

Procedures—All reports were read and initially coded by RS, who is not involved in the teaching or examination of the Addiction Medicine course. Thematic analysis is a qualitative method of analysis which requires in-depth examination of the data to allow common themes to emerge.[10] Initial coding revealed a number of minor categories that were summarised into preliminary themes. After further analysis the preliminary themes were condensed into the three major themes discussed in this paper. Independent rating was achieved by the second author comprehensively reading all reports and providing feedback on initial and final themes.

Participants—Of the 72 students, 53 were female (73.6%). A stable interest in attending OA remained across the years with nine students attending in 2002, 14 in 2003, 12 in 2004, 13 in 2005, 12 in 2006 and 11 in 2007.

Results

Three main themes emerged from the analysis: 'Conceptualising Food as Addictive', 'The Experience of Being Addicted to Food' and 'Impact on Education and Practice.' Each theme is discussed in turn below.

Conceptualising food as addictive—For the most part, the concept of food as a substance to which people can become addicted, is only beginning to be more widely considered and researched. Given this context it was not surprising that most of the medical students who attended OA initially did so because of their uncertainty about the concept of food as an addictive substance.

For many this was revealed as their failure to have ever contemplated food as a potentially addictive substance.

> I had no idea that people could be addicted to food, and not only to nice food, but just any food (frozen, out of the rubbish bins). It also seemed to me that all of the other groups work towards abstinence, but food is something that is essential to our lives, so I was curious as to how people could recover from the addiction, and eat 'normal' again. *Female, 2007*

Lack of familiarity with the concept of addictive overeating was also discussed by OA members as they shared their experiences. Many commented on the lack of understanding they had of the disease they now term addictive overeating.

> …she thought she couldn't possibly be as "weak-willed as an alcoholic," having thought that someone with alcohol dependence should obviously just stop and it would solve all their problems. That was before she realized that she had the same problem with food. *Female, 2007*

Others indicated how they perceived that other people such as health professionals also lacked this knowledge.

> Most of the participants had seen multiple professionals and attempted many therapies (GP's, psychiatrists, religions, group therapy, alternative therapies and endless diets) to try to work out what was wrong with them. *Female, 2003*

Lack of knowledge of addictive overeating was also reflected in the students' surprise about the types of people who attended OA. Over 60% of students reported that the people who attended OA meetings were not who they had expected. Most had expected to see overweight, middle aged females and were greatly surprised when they attended groups of mixed gender, mixed weight ranges (mainly comprising normal weight rather than overweight people) and mixed age ranges. Students were also surprised at the large numbers and the length of time many members had been attending groups. Some members reported OA attendance in excess of 20 years.

The experience of being addicted to food—The stories that OA members told about their lives and their relationships with food prior to OA depicted the features of salience, dyscontrol, compulsive use and tolerance associated with addiction.

In contrast to most other addictions, OA members described their addiction to food as something that began at a very young age with many recalling problems with overeating in childhood. For some, their eating behaviour became a major focus of others' attention and intervention was sought very early on in life.

> …She had always had a problem with food and her mother had told her as a little girl that she wished she would eat "normally". …Originally her mother tried to prevent her. However eventually she decided that if she got sick enough it might cure her. *Female, 2004*

For others, it was something they had simply grown up with and it was not until later in life that they realised their thoughts about food and their eating behaviour were different.

> …several people shared that their problem started at a very young age and that for a long time they did not realise that other people "weren't obsessed with food 24/7" and weren't overeating like they were. *Female, 2007*

The power of food in OA members' lives became apparent as they described their overwhelming desire to eat food.

> "…I could not stop. I would eat everything, Tim Tams, ice cream, frozen food, food covered in ants, food from the rubbish bin. At least I didn't eat the food from the street, I wasn't that bad." *OA participant as quoted by female student, 2005*

The salience of these desires was seen by OA members as extremely oppressive.

> They talked about how their obsessions were very disturbing for them, often present from the moment they woke up until they went to bed. The only way to relieve the thoughts would be to eat and continue eating at every possible moment. In order to satisfy their obsession some of the women stole food or money or had no concern for what they ate, admitting to eating cat food or eating out of rubbish bins. *Female, 2002*

Attempts to control this obsession were common and it was not unusual for extreme measures to be taken in an attempt to prevent overeating.

> People described in detail how their lives were totally consumed by eating: "every hour of every day" said one lady. Another talked of how she had to spray her garbage bag with Spray n' Wipe so that she would not sneak out and eat secretly. *Male, 2004*

Alongside attempts to control eating, came stories of the lengths that people would go to obtain food.

> At boarding school she told how she would steal food from fellow students and risk punishment for sneaking food into her dormitory. …The stealing and obsessive behaviour continued into her adult life… … the breaking point came when she found herself trapped in her room lying in a food-smeared bed wanting to die. *Female, 2004*

The consuming nature of addiction was also evident from the extreme distress OA members experienced.

> One particular quote struck me, "Overeating is a career...in that you career all over the place". …Several people mentioned the desperation and anguish to the level where they even considered suicide. *Male, 2005*

Negative emotional reactions sparked yet more problems as many described how they were both an antecedent to and a consequence of overeating.

> Many spoke of the shame and embarrassment associated with overeating. A common theme was related to the cyclical nature of the disease, after a binge people always felt as if that would be the last time, that they would not let this happen again, and then when it did happen again, they would feel even worse and the cycle would continue. *Female, 2006*

Many OA members also indicated that the area of their lives that was most commonly affected was their relationships with significant others.

> …for many years he concealed his problem with food from his friends and family and refused to admit it to himself. This pattern of behaviour caused him to isolate himself more and more from his friends and family. *Male, 2006*
>
> … there were many, many ruined relationships represented at overeaters anonymous (OA). *Male, 2003*

> One lady talked about strained relationships with her children, and how she 'neglected' her daughter because all she wanted to do at night was to climb into bed early and just eat for the rest of the evening. *Female, 2004*

Similar to other addictions, OA members also described comorbid addictions to other substances either as a co-existing addiction or as a replacement addiction. In addition members described the many attempts they had made to stop their overeating.

> Listening to the individual stories I found that this addiction was astoundingly similar to alcohol and other drug addictions. These people were completely powerless over food. …But like in other addictions people commonly had to hit rock bottom before turning around or seeing the light. *Female, 2007*

Impact on education and practice—Many of the students commented on the value of attending OA and expressed how it had been enlightening and had served as a valuable learning experience.

> …it would be one of the most powerful experiences I have had within the med school. As a result I believe that now, through having had this experience, I would be able to advise patients who have an addiction about the appropriate paths they should take. *Female, 2003*
>
> To be honest, it was something that showed me more of the human side of addictions and gave a glimpse of broken lives that no amount of theory-based learning can adequately convey. *Female, 2004*

Furthermore, the potential long term benefits were highlighted in the students' reflections about what this experience would mean for their future practice as medical professionals.

> I think attending this self help group was not only beneficial for me understanding addiction, but also providing me with information for overeaters and people who have problems with food to not be too scared in opening a subject I was not comfortable with before, to ask them about it and inquire into it…. *Female, 2003*

Discussion

To conceive that something as fundamental to human survival as food has the potential to trigger compulsive behaviour that impacts severely on an individual's health and well being is somewhat counterintuitive. "Addictive overeaters" themselves appear to struggle to understand what is happening for them and this appears to be mirrored in the slow evolution of research on overeating from an addiction perspective.

The purpose of this study was to view OA through the eyes of a group of 72 fifth-year medical students. We found that a general lack of awareness existed among medical students and consumers alike about the concept of addictive overeating or addiction to food prior to attending OA. These findings also suggest that ignorance about addictive overeating extends far beyond the participants in the present study as OA members reported that they experienced a lack of understanding from other health professionals, their own friends and families and society in general. A failure to entertain the possibility that such a condition exists is further reiterated in the research literature where the concepts of food addiction and addictive overeating have only recently begun to be more widely discussed and researched.[1,4]

Hearing the narratives of the OA members' experiences with food, weight gain and compulsive eating appears to have enabled the medical students to move beyond their own experiences and begin to appreciate the serious implications of addictive

overeating for sufferers. Similarly OA members recalled their own growing acceptance and understanding of their eating behaviours through their participation at OA.

The destructive and all-consuming nature of compulsive eating is evident from these discussions and from a qualitative perspective at least, appears to strongly resemble other substance and behavioural addictions. Our findings are in agreement with early findings in the quantitative literature that suggest a strong similarity between food and other substance addictions, including the compulsive nature of consumption and associated negative life consequences.[1-7]

The students' accounts of the real life experiences of people living with a compulsion to overeat highlight how a willingness to listen can benefit both clinicians and patients and may help to bridge the gap between the theoretical construct of addictive overeating and effective treatment. Their resounding positive feedback about attending an OA meeting makes clear both their increased understanding of addictive overeating and their confidence in assisting patients presenting with such a disorder.

These results suggest that exposure to the lived experiences of people with addictive overeating may help doctors to be more compassionate and confident in their dealings with patients who overeat, to the benefit of both parties. In fact medical students, and students training in other health disciplines, may benefit greatly from the opportunity to interact with potential client groups in a non clinical setting.

Removing the onus on students to provide a solution for patients' problems and allowing them to just listen and learn about the reality of living with a particular disorder is likely to provide them with a different viewpoint on which to base their practice. This method of teaching is not only appropriate for students in training but may also be useful for ongoing professional development with qualified practitioners.

Finally, the results suggest that at least for some people, abstinence from problem food(s) may be a more effective therapeutic strategy than the more widely promoted principle of moderation encouraged by both mainstream health providers and commercial weight loss companies. Controlled studies comparing these different approaches are clearly required.

Competing interests: None known.

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Ethnic-specific body mass index cut-off points for overweight and obesity in girls

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Abstract

Aim To develop ethnic-specific body mass index (BMI) cut-off points for overweight and obesity in girls from New Zealand's five major ethnic groups.

Methods A total of 1676 girls (41% European, 21% Pacific Island, 15% East Asian, 13% Māori, and 11% South Asian) aged 5–16 years participated in this study. BMI was determined from height and weight, and body fat percentage (%BF) was obtained from hand-to-foot bioelectrical impedance measurements. Using stepwise multiple regression, a series of ethnic-specific BMI cut-off points were developed that corresponded to the equivalent %BF of European girls at the BMI reference values provided by the International Obesity TaskForce (IOTF).

Results The adjusted cut-off points for overweight and obesity ranged from an average of 3.3 and 3.8 $kg.m^{-2}$ (respectively) lower than the IOTF standards in South Asian girls to 1.5 and 1.9 $kg.m^{-2}$ higher in Pacific Island girls.

Conclusion We conclude that the ethnic-specific BMI cut-off points developed in this study are more appropriate than universal definitions of overweight and obesity for predicting excess adiposity in New Zealand girls.

Rapid and widespread increases in childhood overweight and obesity are a serious public health concern for many countries. To facilitate the comparison of prevalence estimates between populations, the International Obesity TaskForce released a series of body mass index (BMI) cut-off points defining overweight and obesity in young people that adjust for natural differences between sexes and across age groups.[1] However, there is a growing body of evidence indicating that universal BMI thresholds often correspond to different body fat levels in children from diverse ethnic backgrounds.[2–8] Indeed, several studies have concluded that ethnic-specific BMI cut-off points would provide a more appropriate representation of overweight and obesity in multiethnic populations.[2-4,9,10]

Previously, we have shown that adjusting universal BMI standards for ethnicity improves diagnostic accuracy when screening for overweight in Māori, Pacific Island, East Asian, and South Asian girls.[4] A logical progression is the formulation of ethnic-specific cut-off points that correspond to the equivalent level of body fat in all ethnicities, thereby standardising BMI-based estimates of overweight and obesity. Rush et al[8] used regression analysis to demonstrate the increase in BMI required for Māori and Pacific Island girls to match the same body fat percentage (%BF) as European girls at a range of BMI values. To our knowledge, no studies have derived ethnic-specific BMI cut-off points from the existing IOTF standards. The purpose of the present study was to propose adjusted BMI-for-age curves in Māori, Pacific

Island, East Asian, and South Asian girls that correspond to the observed %BF of European girls at the IOTF cut-off points for overweight and obesity.

Methods

The participant recruitment and body composition methodology has been described in detail elsewhere,[4] and consequently only a brief description is given here. A total of 1676 participants aged 5-16 y (school years 1-10) were randomly selected from 39 primary, intermediate, and secondary schools in Auckland, New Zealand. The ethnic composition of this sample was 680 European (40.6%), 355 Pacific Island (21.2%), 216 Māori (12.9%), 243 East Asian (14.5%), and 182 South Asian (10.9%). BMI was calculated as weight (kg) divided by squared height (m^2). Fat-free mass measurements were obtained using a hand-to-foot bioelectrical impedance analyzer (Model BIM4, Impedimed, Capalaba, Australia) and prediction equations previously validated in all ethnic groups within the present study.[8,11] Fat mass was derived as the difference between fat-free mass and body weight, and %BF was calculated as 100 × fat mass/weight. Ethical approval for this study was obtained from the Auckland University of Technology Ethics Committee. Written informed consent was provided by each participant and her legal guardian.

Data were analysed using SPSS for Windows v12.0.1 software, (SPSS Inc., Chicago, IL). The relationship between BMI and %BF was assessed using stepwise multiple regression, with age (rounded to nearest half-year) and ethnicity as independent variables ($P_{in} > 0.05$, $P_{out} > 0.10$). BMI was log transformed due to the curvilinear relationship between BMI and %BF. To minimise colinearity complications, the $\log_{10}$BMI variable was centered about the mean by subtracting 1.31 (mean $\log_{10}$BMI) from each value. The variables for ethnicity were dummy coded E_1-E_4. For South Asian $E_1 = 1$, $E_2 = 0$ $E_3 = 0$ $E_4 = 0$; for Pacific Island $E_1 = 0$, $E_2 = 1$ $E_3 = 0$ $E_4 = 0$; for East Asian $E_1 = 0$, $E_2 = 0$ $E_3 = 1$ $E_4 = 0$; and for Māori $E_1 = 0$, $E_2 = 0$ $E_3 = 0$ $E_4 = 1$.

Results

Table 1 presents the coefficients of the stepwise multiple regression in the order the independent variables were entered into the equation. The final regression equation was as follows: $\%BF = [59.5 \times (\log_{10}BMI - 1.31)] + [4.50 \times E_1] - [1.84 \times E_2] + [1.39 \times E_3] - [0.164 \times age] - [0.731 \times E_4] + 28.9$; with an R^2 of 0.669 and a SEE of 3.73%. Hence, at a fixed BMI and age, South Asian ($E_1 = 1$) and East Asian ($E_3 = 1$) girls averaged 4.50% (95% CI: 3.89-5.11%) and 1.39% (0.841-1.94%) more body fat (respectively) than European girls, while Pacific Island ($E_2 = 1$) and Māori ($E_4 = 1$) girls averaged 1.84% (1.32-2.35%) and 0.731% (-0.014-1.31%) less body fat.

The variance in %BF explained by each term in the equation was as follows: $\log_{10}$BMI – 1.31, 58.3%; E_1, 11.1%; E_2, 2.8 %; E_3, 1.5 %; age, 1.0%; and E_4, 0.4%. Significant ($P < 0.05$) interactions between age and $\log_{10}$BMI, and between age and E_3, were excluded as they resulted in only minor improvements to the regression equation. There were no significant interactions between $\log_{10}$BMI and any of the dummy variables, indicating that there were no differences in the regression slopes between the ethnic groups. In other words, ethnic differences in the association between BMI and %BF were similar across the entire BMI distribution.

Table 1. Stepwise multiple regression with %BF as the dependent variable

Log_{10}BMI		E_1		E_2		E_3		Age		E_4		Intercept			
β	*SE*	*β*	*SE*	*β*	*SE*	*β*	*SE*	*β*	*SE*	*β*	*SE*	*β*	*SE*	r^2	*SEE (%)*
51.5	1.02	–	–	–	–	–	–	–	–	–	–	27.2	0.100	0.602	4.09
53.5	0.967	4.60	0.304	–	–	–	–	–	–	–	–	26.7	0.099	0.650	3.83
55.9	1.01	4.29	0.302	-1.76	0.243	–	–	–	–	–	–	27.1	0.113	0.660	3.78
56.2	1.00	4.60	0.306	-1.49	0.247	1.40	0.272	–	–	–	–	26.8	0.126	0.666	3.75
58.9	1.21	4.66	0.305	-1.62	0.248	1.55	0.273	-0.153	0.0394	–	–	28.6	0.476	0.668	3.73
59.5	1.23	4.50	0.311	-1.84	0.263	1.39	0.280	-0.164	0.0395	-0.731	0.297	28.9	0.490	0.669	3.73

E_1= 1 for South Asian, E_2 = 1 for Pacific, E_3 = 1 for East Asian, E_4 = 1 for Māori.

SE standard error, SEE standard error of estimate, r^2 = explained variance.

β = unstandardised coefficients.

To demonstrate the ethnic variation in BMI thresholds for overweight and obesity, the expected %BF at each IOTF age and sex specific cut-off point was first calculated using the regression equation in European participants. Body fat levels increased steadily with age before reaching a plateau in older girls (range: overweight, 25.2% [5 years] to 36.1% [16 years]; obese, 28.1% to 41.0%). These %BF data were entered into the reversed equation to determine the BMI levels for Pacific Island, Māori, East Asian, and South Asian participants that correspond to the IOTF cut-off points for overweight and obesity in European children (Table 2).

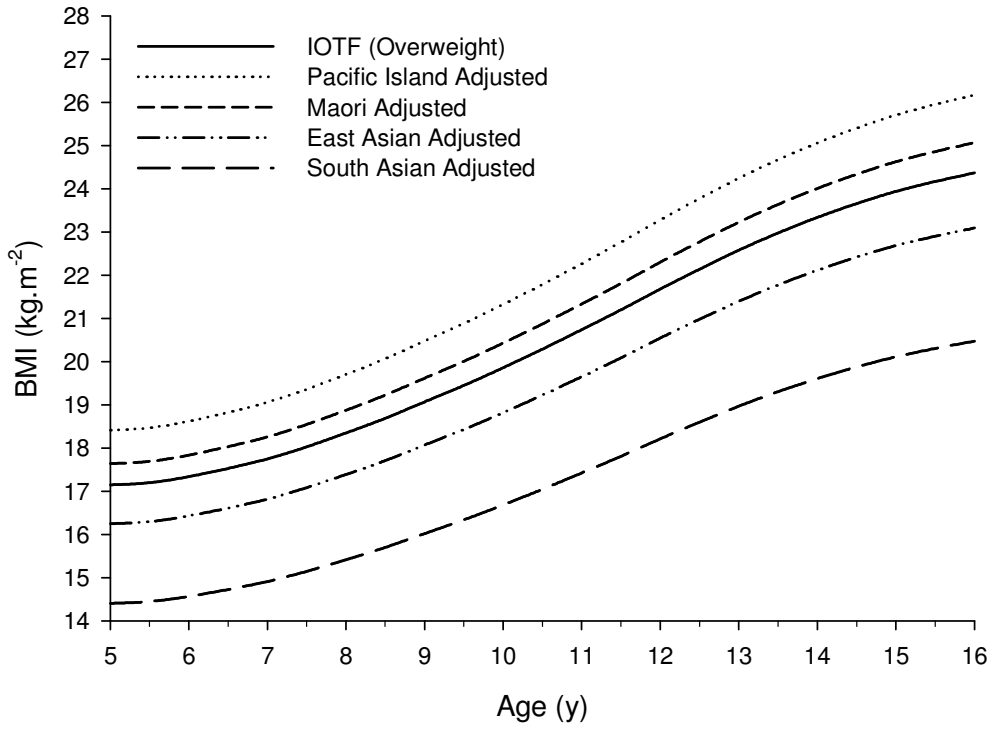
Table 2. Ethnic-specific BMI cut-off points equivalent to the predicted %BF of European children at the IOTF BMI cut-off points for overweight and obesity

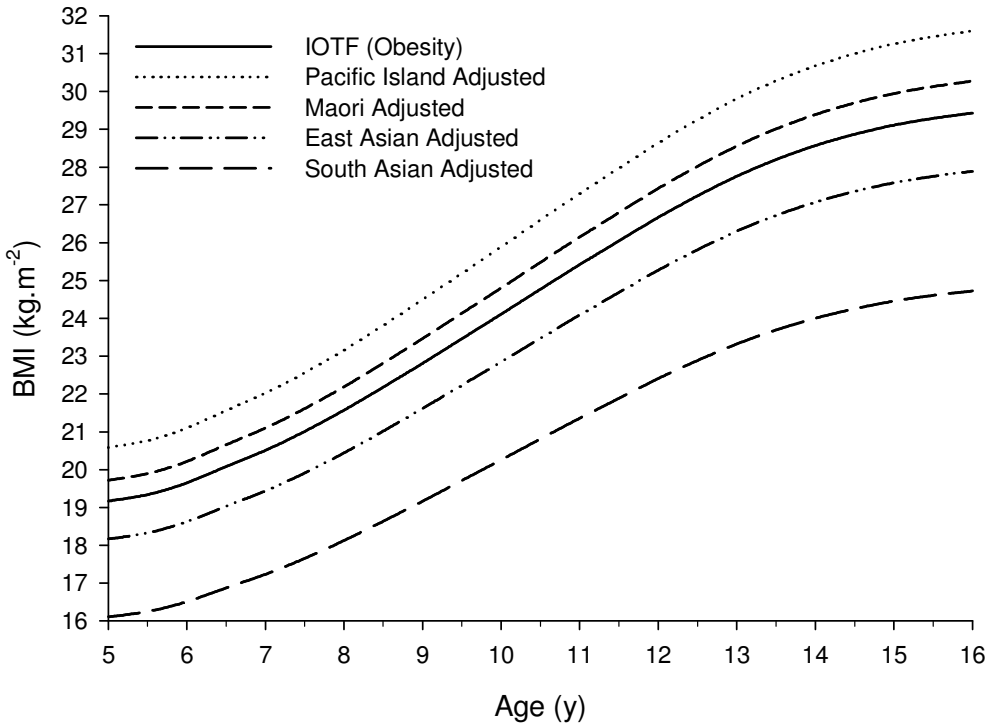
Age (y)	IOTF		Pacific Island		South Asian		East Asian		Māori	
	Overweight	Obesity	Overweight	Obesity	Overweight	Obesity	Overweight	Obesity	Overweight	Obesity
5	17.15	19.17	18.42	20.58	14.41	16.11	16.25	18.17	17.64	19.72
5.5	17.20	19.34	18.47	20.77	14.45	16.25	16.30	18.33	17.69	19.89
6	17.34	19.65	18.62	21.10	14.57	16.51	16.43	18.62	17.84	20.21
6.5	17.53	20.08	18.82	21.56	14.73	16.87	16.61	19.03	18.03	20.66
7	17.75	20.51	19.06	22.02	14.91	17.23	16.82	19.44	18.26	21.10
7.5	18.03	21.01	19.36	22.56	15.15	17.65	17.09	19.91	18.55	21.61
8	18.35	21.57	19.70	23.16	15.42	18.12	17.39	20.44	18.88	22.19
8.5	18.69	22.18	20.07	23.82	15.70	18.64	17.71	21.02	19.23	22.82
9	19.07	22.81	20.48	24.49	16.02	19.16	18.07	21.62	19.62	23.46
9.5	19.45	23.46	20.89	25.19	16.34	19.71	18.43	22.23	20.01	24.13
10	19.86	24.11	21.33	25.89	16.69	20.26	18.82	22.85	20.43	24.80
10.5	20.29	24.77	21.79	26.60	17.05	20.81	19.23	23.47	20.87	25.48

11	20.74	25.42	22.27	27.30	17.43	21.36	19.65	24.09	21.34	26.15
11.5	21.20	26.05	22.76	27.97	17.81	21.89	20.09	24.69	21.81	26.80
12.0	21.68	26.67	23.28	28.64	18.21	22.41	20.54	25.27	22.30	27.44
12.5	22.14	27.24	23.77	29.25	18.60	22.89	20.98	25.81	22.78	28.02
13.0	22.58	27.76	24.25	29.81	18.97	23.32	21.40	26.31	23.23	28.56
13.5	22.98	28.20	24.68	30.28	19.31	23.69	21.78	26.72	23.64	29.01
14.0	23.34	28.57	25.06	30.68	19.61	24.00	22.12	27.07	24.01	29.39
14.5	23.66	28.87	25.41	31.00	19.88	24.26	22.42	27.36	24.34	29.70
15.0	23.94	29.11	25.71	31.26	20.11	24.46	22.69	27.59	24.63	29.95
15.5	24.17	29.29	25.95	31.45	20.31	24.61	22.90	27.76	24.86	30.13
16.0	24.37	29.43	26.17	31.60	20.48	24.73	23.09	27.89	25.07	30.27

Pacific Island girls averaged the highest BMI cut-off points for overweight and obesity, equivalent to 1.5 and 1.8 $kg.m^{-2}$ greater than the IOTF recommendations, respectively. In contrast, the South Asian cut-off points were lowest at an average of 3.3 and 3.9 $kg.m^{-2}$ below the IOTF criteria. Figure 1 shows the ethnic-specific BMI cut-off points for overweight and obesity in each group alongside the established IOTF standards.

Figure 1. IOTF and ethnic-specific BMI curves for overweight and obesity in girls aged 5–16 years





Discussion

This study provides the first ethnic-specific BMI criteria for standardising youth overweight and obesity in a multiethnic population. The diverse ethnic composition of the sample enabled a wide range of body types to be compared: Pacific Island and Māori phenotypes are defined by a relatively low fat-to-fat-free mass ratio,[4,8] whereas Asian individuals tend to display the opposite characteristics.[3,4,7] In agreement with these observations, our results indicated that Māori and Pacific Island BMI thresholds need to be raised to match the equivalent body fatness of European girls at the IOTF BMI cut-off points, while East and South Asian thresholds need to be lowered. The adjusted BMI-for-age curves for South Asian girls, in particular, were over 3 $kg.m^{-2}$ lower than the existing IOTF definitions of overweight and obesity. Thus, it is likely that a significant proportion of overweight or obese South Asian girls are currently classified as normal weight by the IOTF criteria. This underestimation may prohibit health services from identifying those most at need, and may reduce the probability that 'at risk' individuals will initiate the lifestyle changes necessary to prevent fat accretion.

Although Pacific Island girls are also liable to be misclassified, it could be argued that overestimation of overweight/obesity status is preferable to overlooking those with excess adiposity. Nevertheless, labelling a young person as overweight raises the possibility of adverse psychosocial effects,[12] and consequently every effort should be made to ensure that only those with excess body fat are classified as overweight or obese. The cut-off points for Māori and East Asian girls were closer to the IOTF criteria than the Pacific Island and South Asian curves; however we suggest that each

non-European curve showed sufficient divergence to warrant inclusion in an ethnic-specific BMI classification system for overweight and obesity in New Zealand girls.

To our knowledge, only one other study has modified the IOTF classification system to suit a specific population. Kim et al[9] proposed a series of BMI percentiles for Korean girls aged 8 to 18 years that corresponded to the IOTF thresholds for overweight and obesity in Asian adults at age 18 years (23 and 25 $kg.m^{-2}$, respectively). Similar to our observations in East and South Asian girls, the adjusted BMI-for-age curves for Korean girls were substantially lower than the IOTF cut-off points. However, it remains uncertain if these ethnic-specific BMI values correspond to excess adiposity in Korean individuals.

An advantage of the present study was the use of %BF to standardise the classification of weight status across diverse ethnicities. The %BF of European girls at the IOTF BMI cut-off points was selected as the appropriate criteria given that the IOTF standards are applied most frequently to children and adolescents of European descent. Nevertheless, these criteria are not necessarily the most suitable markers of increased health risk. While several studies have posited single %BF thresholds ranging from 20 to 30% that are associated with an elevated risk of negative health outcomes,[13-16] the age-related increase in %BF observed in female children and adolescents raises concerns about the appropriateness of a single %BF point.

In the present study, %BF values corresponding to the IOTF criteria in European girls were age-dependent, ranging from 25% to 36% for overweight and from 28% to 41% for obesity. The %BF-for-age curves reported in another investigation of the IOTF BMI cut-off points in New Zealand (predominantly European) children aged 3-18 years showed a similar overall pattern in female subjects: 20-34% for overweight and 26-46% for obesity.[17] The findings from both studies suggest that a single %BF threshold may underestimate health risk in younger girls and overestimate health risk in older girls.

Clearly, there is a need to elucidate the associations between adiposity, age, and negative health outcomes in young people. While the delayed onset of many obesity-related complications makes this a challenging task, an understanding of the unhealthy body fat levels in a variety of ethnic groups would facilitate the development of ethnic-specific BMI thresholds for overweight and obesity that are closely linked with disease risk. Indeed, data that enable the comparison of established risk factors in youth, such as blood pressure, blood lipids, or other blood markers, with total and regional body fatness in different age and ethnic groups is urgently required. More information about the role of fat distribution in the risk of disease would also be valuable given the evidence that peripheral and central adiposity have significantly different effects on health outcomes.[18]

A limitation of this study is that only female children and adolescents were assessed. It is possible that ethnic differences in the association between %BF and BMI follow different patterns in boys. However, Rush et al[19] showed that the significant differences in body composition among European, Māori, and Pacific Island girls are not evident among boys from the same ethnic groups. This suggests that ethnic adjustments to the IOTF BMI cut-off points may not be necessary for Māori and Pacific Island boys. In contrast, there is evidence that differences in the %BF-BMI ratio exist between European and both East and South Asian boys.[3,7] Further research

is required to establish the appropriate BMI thresholds for overweight and obesity in boys from these ethnicities.

It should be remembered that the ethnic-specific BMI cut-off points proposed in this study were generated from girls living in New Zealand. The existence of population-specific differences in physical characteristics within girls from the same ethnic background would reduce the international applicability of our results. Thus, cross-validation of the regression equation in other samples of Māori, Pacific Island, East Asian, and South Asian girls is recommended, especially in those living in countries other than New Zealand. It would also be worthwhile to establish the ethnic adjustments necessary to standardize overweight and obesity in girls aged 2-4 and 17-18 years, thereby covering the entire age range of the IOTF BMI standards. Nevertheless, we suggest that the ethnic-specific BMI cut-off points provided in this study represent the most appropriate definition of overweight and obesity for New Zealand girls aged 5-16 years.

In summary, this study provides the first ethnic-specific BMI cut-off points for defining overweight and obesity in a multiethnic population of female children and adolescents. Our results showed that the current IOTF standards for overweight should be raised by an average of 1.5 and 0.6 $kg.m^{-2}$ for Pacific Island and Māori girls (respectively), and lowered by an average of 3.3 and 1.1 $kg.m^{-2}$ for South and East Asian girls. Similarly, the ethnic-specific BMI cut-off points for obesity were (on average) 1.8 and 0.7 $kg.m^{-2}$ greater than the IOTF thresholds for Pacific Island and Māori girls, and 3.9 and 1.3 $kg.m^{-2}$ lower for South and East Asian girls. Application of the proposed adjustments will reduce the misclassification of excess adiposity common in ethnic groups that differ from the typical European phenotype.

Competing interests: None known.

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Overweight and obesity prevalence among adult Pacific peoples and Europeans in the Diabetes Heart and Health Study (DHAHS) 2002–2003, Auckland New Zealand

Gerhard Sundborn, Patricia A Metcalf, Dudley Gentles, Robert Scragg, Lorna Dyall, Peter Black, Rod Jackson

Abstract

Aim This paper describes and compares proportions of overweight, obese, and average BMI and their relationship with physical activity for Pacific ethnic groups (Samoan, Tongan, Niue, Cook Islands) and European New Zealanders by gender who participated in the 2002–03 Diabetes Heart and Health Study (DHAHS).

Methods The DHAHS was a cross-sectional population based study of people age 35-74 years carried out in Auckland between 2002-03. A total of 1011 Pacific people comprising of 484 Samoan, 252 Tongan, 109 Niuean, 116 Cook Islanders and 47 'Other Pacific' (mainly Fijian) and 1745 European participants took part in the survey. Participants answered a self-administered questionnaire to assess their participation in physical activity, perceived weight, and their perception of their current weight. Following this participant's height and weight was measured for calculation of BMI. Ethnic-specific cut offs were used for classification of overweight (Pacific ≥26.0–<32.0, European ≥25.0–<30.0) and obesity (Pacific ≥26.0, European ≥32.0).

Results Approximately 95% of Pacific men and 100% Pacific women were *'overweight or obese'*. Proportions of obesity were for men: all Pacific 53%, Samoan 58%, Cook Island 23%, Tongan 60%, and Niuean 49%; and for women: all Pacific 74%, Samoan 75%, Cook Island 69%, Tongan 78%, and Niuean 76%. Pacific people were as accurate at estimating their body weight as Europeans, and included similar proportions who under-estimated their weight. The Cook Islands group were most likely to accurately report their weight and were significantly less likely to underestimate their weight. A significantly higher proportion of Pacific people reported that they were heavier than a year ago (22.7%) compared to Europeans (17.2%), but significantly fewer Pacific people (55.6%) reported thinking that they were overweight compared to Europeans (64.9%). After adjustment for possible confounding variables, older Pacific adults were over 11 times more likely to be obese than their Europeans counterparts.

Conclusion The continued rise in overweight and obesity in older Pacific adults means that almost all are now overweight or obese. This raises concerns about interventions focussed on overweight and obesity, and will require the adoption of a total Pacific population 'environmental change' approach rather than dietary or physical activity interventions targeted to overweight individuals.

Overweight and obesity is a global concern with approximately 1.6 billion adults overweight and 400 million adults obese in 2005.[1] Physiologically, the main drivers

of this epidemic are a combination of an unhealthy diet, and inadequate physical activity resulting in an energy imbalance and the storing of energy as adipose tissue (body fat). An intermediate affect can be observed as raised blood pressure, raised blood glucose levels, abnormal blood lipids and eventual overweight and obesity. Annually, 2.7 million deaths have been attributed to low or inadequate consumption of fruit and vegetables and a further 1.9 million attributed to inadequate physical activity.[1]

In New Zealand reducing obesity has been listed as the third highest *'Priority population health objective'* by the New Zealand Ministry of Health, following objectives to reduce smoking and improve nutrition.[2] Pacific children and adults in New Zealand are 2.5 times more likely to be obese,[3] and Pacific children are eleven times more likely to be extremely obese than their European counterparts.[4] In light of these figures, public health interventions that not only aim to prevent but also curb current trends of overweight and obesity are a priority area for New Zealand and especially Pacific communities in New Zealand.

Many locally and nationally funded health strategies have been developed to encourage a healthier diet and to increase physical activity as a means of addressing the obesity epidemic.[5–7] Collaborative interventional research designed to impact on overweight and obesity in the community and school settings in New Zealand and in the Pacific is also being undertaken[8] together with an evaluation into its effectiveness.

There has been debate on the accuracy of using a single measure of Body Mass Index (BMI) as a measure of obesity for people of different ethnic groups. This has led to the use of different levels of BMI for ethnic groups that categorize 'overweight' and 'obesity'. Ethnic-specific measures should ensure greater accuracy as they account for differences observed in body type (composition). Swinburn et al found "that at any given body size Polynesians are significantly leaner than Caucasians and that specific Polynesian standards for defining obesity needed to be developed".[9] In response to these findings Swinburn's recommendations were adopted for the 1997 National Nutrition Survey. For Pacific people, BMI categories were higher than for New Zealand European and Others. These were 'overweight': Pacific people $26 \geq$ BMI $<$ 32 kg/m^2 and for New Zealand European other $25 \geq$ BMI < 30 kg/m^2, and obesity for Pacific people BMI ≥ 32 kg/m^2 compared to BMI ≥ 30 kg/m^2 for New Zealand European and others.

There is little information on perceptions and attitudes of body weight in New Zealand. Furthermore, information specific to Pacific ethnic groups on obesity and overweight is scarce. This study aims to address these information gaps by reporting ethnic-specific proportions of overweight, obese and average BMI by gender. The influence that physical activity has on overweight, obesity and average BMI was investigated.

Methods

This paper reports on analyses from the Diabetes Heart and Health Study (DHAHS). The aim of the DHAHS was to investigate the prevalence of cardiovascular and diabetes risk factors in a representative sample of Aucklanders aged 35-74 years, between January 2002 and December 2003. Participants were recruited using two sampling frames: one was a cluster sample where random starting point Auckland area addresses were obtained from Statistics New Zealand and the probability of selection was proportional to the number of people living in that mesh block (response rate 61.3%); and

the other was a random sample taken from the November 2000 Auckland electoral rolls stratified into 5 year age bands and included all people living in the Auckland area, with the exception of the Franklin and Rodney electorates (response rate 65%). Participants were interviewed in places close to where they lived. All completed a self-administered questionnaire and a series of health measurements were made.

All participants received information in the mail with instructions of where and when to attend the survey centres. Participants completed questionnaires covering socioeconomic status (SES), and demographic information. In addition several body measurements were taken (height, weight, waist and hip circumference).

Initially participants were asked if they knew what their body weight was, following this height and weight measurements were taken. Further questions asked whether they thought they were lighter, heavier or the same as one year ago, and if they thought they were underweight, overweight or just right.

Overweight and obesity were classified using standard BMI cut-offs as well as ethnic-specific BMI cut-offs. The ethnic-specific BMI cut-offs used to classify overweight were: European 25.0—<30.0 kg/m², Pacific 26.0– <32.0 kg/m²; and obesity: European ≥ 30.0 kg/m², Pacific ≥ 32.0 kg/m². Justification for the use of higher BMI cut-offs for Pacific is due to a greater lean body mass found in Pacific people at any given BMI compared to European.[9]

Leisure-time exercise was assessed using a three-month physical activity recall questionnaire that has previously been validated.[10] One question asked if participants had engaged in any vigorous activity at least once a week, in the past three months, long enough to cause them to breathe hard or sweat. The other question asked if they had engaged in any regular moderate activity (that did not cause them to breathe hard or sweat). Those who answered no to both were categorised as inactive. Further analyses measured and quantified exercise time in minutes per week.

NZDep2001 score was determined for each participant based on their area of domicile and was classified in quintiles. Quintile 1 represents areas with the least deprivation and quintile 5 areas with the most deprivation.[3] The New Zealand Index of social deprivation (NZDep20001) was created from Census 2001 data; it describes the deprivation by small geographic areas and is used as a proxy for individual deprivation.

Classification of ethnicity gave priority to Pacific over European ethnicity. This is similar to the method used by Statistics New Zealand.[11] Participants who indicated belonging to more than one Pacific ethnic group were assigned to one ethnic group only. Those who were of Pacific and non-Pacific or non-Maori were assigned into their respective Pacific ethnic group. Those who belonged to more than one Pacific ethnic group were assigned to the smaller Pacific group as done by census 2001. [11] This gave priority firstly to Niuean, followed by Cook Island, Tongan, and lastly Samoan ethnicity. Small numbers of Fijian (n=27) and 'Other Pacific' (n=27) participants meant that analysis of their results could not generate reliable findings. Analyses were performed for the entire Pacific cohort (n=1011) which included 'Fijian' and 'Other Pacific' participants, as well as ethnic-specific analyses for the main Pacific ethnic groups (Samoan, Cook Island, Tongan and Niuean). Ethical approval was obtained from the Auckland Ethics Committees.

Statistical analysis was undertaken using SAS version 9.1. Participant data were weighted according to the sampling frame that they were obtained from and means, standard errors and prevalence's calculated using dual frame sampling methodology. [12-14] SAS survey procedures (SURVEYMEANS, SURVEYREG, SURVEYFREQ AND SURVEYLOGISTIC) were used to calculate weighted means, adjusted means, percentages and odds ratios, respectively.[15] The Rao-Scott modified Pearson Chi squared test was used where appropriate. Analyses have compared all Pacific ethnic groups to their European counterparts.

Results

Mean BMI, waist-hip and waist measurements

Mean BMI, waist-to hip ratios and waist measurements are presented in Table 1 for Europeans and the Pacific ethnic groups by sex. Pacific men and women had significantly greater mean BMI and waist measurements than their European

counterparts. Tongan men and all Pacific women had significantly greater waist-to hip ratios than Europeans.

Tables 2 presents mean BMI and waist measurements for Europeans and the combined Pacific ethnic group by age group and sex. The older age groups in Pacific men had progressively lower BMI, whereas for European men, there was no change. For Pacific women a similar observation was made, that as age increased BMI tended to decrease. However, for European women the opposite was observed. With the exception of Pacific men, all groups had larger waist measurements in the older age categories.

The prevalence of overweight / obesity

Figures 1 & 2 present the proportions of overweight and obese by ethnic group for men and women using ethnic-specific body mass index classification. For Europeans, men were more likely to be *'overweight or obese'* than women, however, in contrast, Pacific women were more likely to *'overweight or obese'* than Pacific men.

Cook Island men had a similar level of obesity to European men and were the only Pacific group that were more likely to be overweight than obese. For women, almost all Pacific ethnic groups were either overweight or obese compared to approximately 60% of European women.

The combined Pacific ethnic group had significantly different prevalence patterns to Europeans in all weight categories. Approximately 96% of all Pacific people were either *'overweight or obese'* compared to 67.4% of Europeans ($p < 0.0001$). This relationship remained the same when assessing *'overweight or obesity'* by age group.

Prevalence (%) of overweight and obesity using ethnic-specific BMI thresholds for men (Figure 1) and women (Figure 2)

Fig. 1

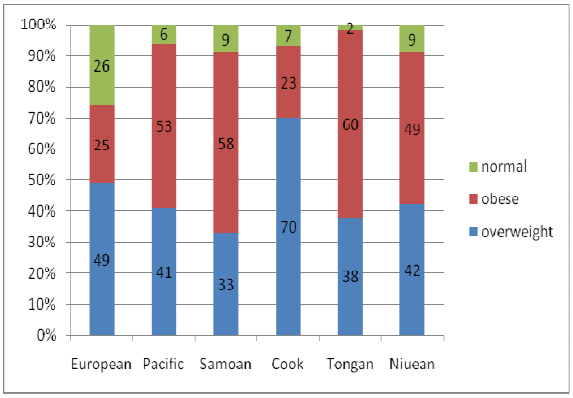


Fig. 2

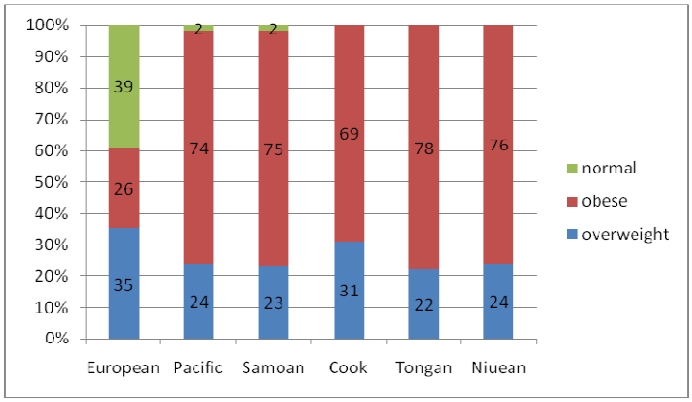


Table 1. Comparison of BMI and waist/hip measurements among *men & women* aged 35–74 years (age adjusted). Values are mean (SE) or percent

Variables Men	European (n=863)	Samoan (n=246)	Tongan (n=123)	Niuean (n=49)	Cook Islands (n=46)
BMI (kg/m²)	27.6 (0.16)	33.0 (0.47)***	34.4 (0.88)***	32.0 (0.75)***	30.8 (0.81)*** †
Waist-to hip ratio	0.93 (0.002)	0.94 (0.01)	0.97 (0.01)*** †	0.93 (0.01)	0.93 (1.01)
Waist (cm)	97.8 (0.39)	105.9 (1.11)***	110.1 (2.05)*** †	102.1 (1.57)** †	100.2 (2.93)†
Women	**(n=882)**	**(n=238)**	**(n=132)**	**(n=60)**	**(n=70)**
BMI (kg/m²)	27.3 (0.19)	36.3 (0.57)***	36.3 (1.05)***	35.3 (1.27)***	35.4 (0.78)***
Waist-to hip ratio	0.81 (0.002)	0.87 (0.01)***	0.87 (0.01)***	0.85 (0.01)***††	0.87 (0.01)***
Waist (cm)	87.1 (0.45)	105.1 (1.06)***	106.4 (1.73)***	101.0 (1.80)***	102.7 (2.00)***

*p<0.05, **p<0.01, ***p<0.0001 compared to European; †p<0.05, ††p<0.01, ††† p<0.0001 compared to Samoan.

Table 2. Anthropometry by ethnicity, sex and age

Variables	Age range	Males		Females	
		European	Pacific†	European	Pacific†
BMI (kg/m²)	<45	27.4 (26.8-27.9)	33.7 (32.5-34.9)	26.8 (26.0-27.6)	35.6 (34.5-36.6)
	45-54	27.9 (27.2-28.6)	33.2 (31.7-34.7)	27.1 (26.4-27.8)	36.3 (34.5-38.0)
	55-64	27.5 (27.0-28.0)	32.8 (31.6-34.1)	27.8 (27.1-28.5)	35.9 (33.5-38.3)
	65+	27.5 (26.9-28.1)	31.0 (29.3-32.7)	27.8 (27.0-28.6)	33.7 (32.6-34.9)
Waist (cm)	<45	96.2 (94.7-97.6)	105.4 (102.8-108.8)	85.0 (83.2-86.8)	101.7 (99.3-104.1)
	45-54	98.4 (96.8-99.9)	106.4 (103.0-109.8)	86.2 (84.5-87.9)	105.2 (102.4-107.9)
	55-64	98.0 (96.7-99.3)	106.9 (103.7-110.1)	89.1 (87.5-90.8)	105.1 (103.0-107.2)
	65+	99.5 (98.0-100.9)	105.8 (99.9-111.7)	90.7 (88.8-92.6)	108.2 (101.8-114.5)
Total number		**863**	**487**	**882**	**524**

BMI: Body mass index = weight (kg) ÷ height (m²); †Mostly of Samoan, Tongan, Niuean or Cook Island ethnicity.

BMI levels

Figure 3. European men and women's average BMI by age group

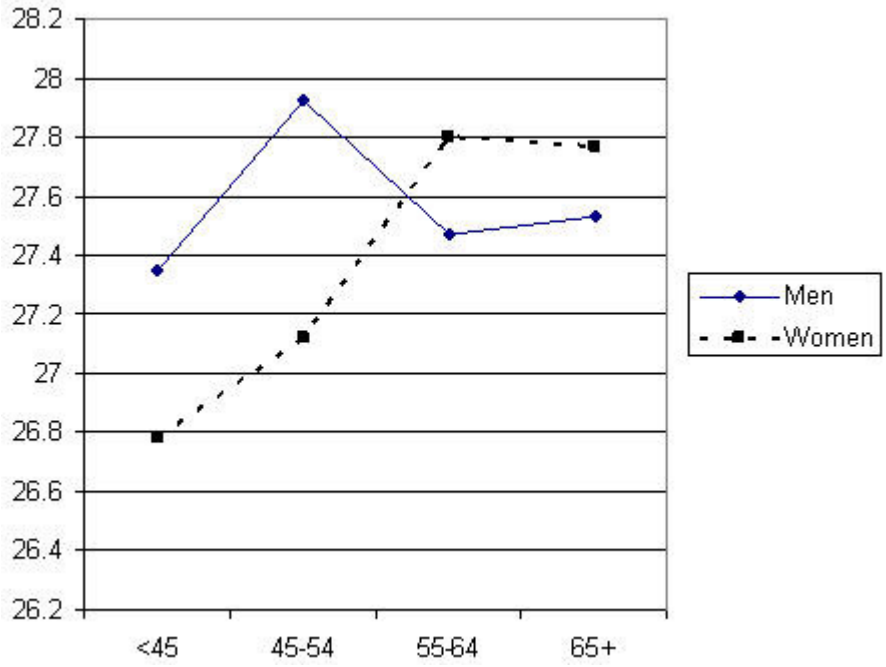
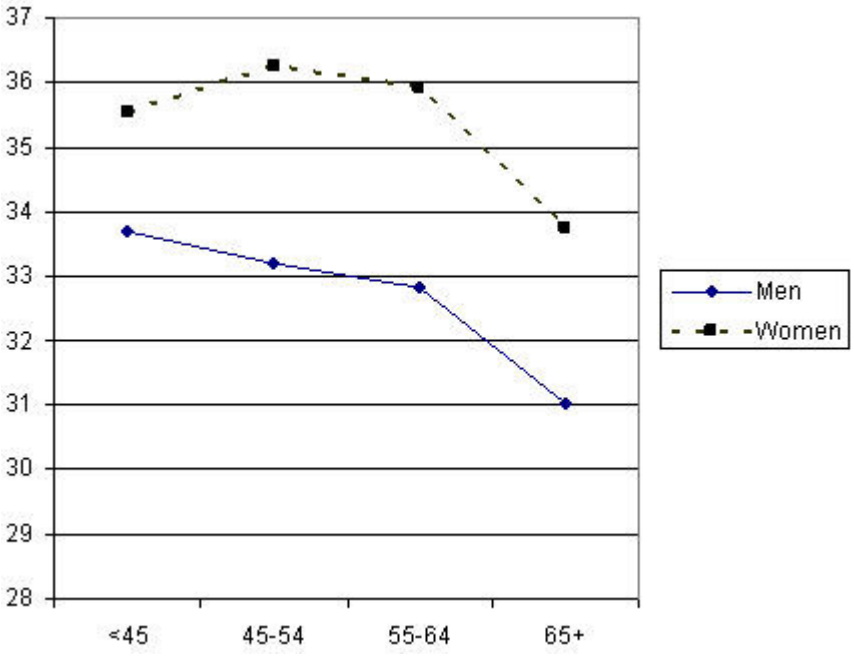


Figure 4. Pacific men and women's average BMI by age group



Average BMI levels of European and Pacific people are plotted in Figures 3 and 4 by age and gender. For Europeans (Figure 3), males in the two younger age groups (<45 and 45-54 years) had higher BMI levels than their female counterparts with the opposite experienced in the older age groups (55-64 and 65+ years). In contrast, Pacific women had higher BMI levels at every age group than their male counterparts (Figure 4). Mean BMI increased up to age 55-64 years in European women and remained relatively static with age in European men. In contrast, BMI decreased with age in Pacific men, whereas it was relatively stable in Pacific women until age 55-64 years and then decreased.

When age and sex adjusted BMI levels were compared between Pacific participants who were sedentary (did not indicate having participated at least once a week any activity—moderate or vigorous that caused them to breath hard or sweat) to those who were highly active (indicated both moderate and vigorous activity), BMI was higher but not significantly. (Moderate & vigorous activity BMI = 33.3kg/m², neither 34.83kg/m², p 0.7).

Perceptions of body weight

Table 3 compares self-reported estimates of body weight to measured body weights. Results show that women were generally more likely to know their actual weight than men. For women, there was very little ethnic difference in this knowledge (range 12.2%), however, for men the ethnic difference was more pronounced. Niuean men were least aware of their current weight (41.3%), compared to Cook Islands men (72.3%) who were most aware, encompassing a 31.1% range which was approximately three times greater than the range for women. With the exception of the Cook Island group under-estimation of body weight between all other ethnic groups were quite similar. Men were more likely to under-estimate their body weight than women. Cook Islands men and women were significantly less likely to under-estimate their body weight than their European counterparts.

Table 3. Correct estimation and underestimation of self-reported body weight

Group	Correct estimation of body weight (±2 kg)			Under-estimation of body weight (>2 kg)		
	Total (%)	Men (%)	Women (%)	Total (%)	Men (%)	Women (%)
European	63.1	59.7	66.4	28.3	28.9	27.8
All Pacific	57.9	50.6*	65.2	28.8	33.7	23.7
Samoan	55.3	49.9	60.8	29.3	30.4	28.2
Cook Island	70.9	72.3	71.0	12.4†	7.1†	15.4*
Tongan	64.7	57.4	73.0	28.8	35.2	20.2
Niuean	57.3	41.3*	69.3	26.7	38.0	18.4

*p<0.05; †p<0.0001, compared to Europeans of same sex.

Perceptions of weight by ethnic group are presented in Table 4. Significantly more Europeans reported maintaining a stable weight, whilst significantly more Pacific people reported being heavier compared to one year ago. Significantly fewer Pacific people reported being overweight than Europeans.

Table 4. Weight now versus 1 year ago and perceptions of weight

Weight now versus 1 year ago	European (%)	Pacific (%)
Lighter	13.5	17.3
Same	69.2	60.2†
Heavier	17.2	22.7*
Perception of your weight		
Under-weight	5.2	7.6
Just right	30.1	36.2*
Overweight	64.9	55.6†

*p<0.05; †p<0.01, compared to Europeans.

Table 5. Percent measured normal, overweight, and obese in each 'self-perception of body weight' category by ethnic group (age and sex adjusted)

Objective measure	Self-reported assessment of weight					
	European (N=1745)			Pacific (N=1011)		
	Under-weight (2.8%)	Right-weight (23.6%)	Over-weight (73.6%)	Under-weight (5.3%)	Right-weight (29.6%)	Over-weight (65.1%)
Obese	4.7%	2.5%	33.7%	42.4%	40.6%	75.0%
Overweight	2.0%	24.1%	49.0%	21.1%	49.8%	25.0%
Normal	93.3%	73.4%	17.3%	36.5%	9.6%	0.0%

Note: Ethnic-specific cut-offs for overweight and obese were: European—overweight (25<30 kg/m²), obese (≥30 kg/m²), and Pacific—overweight (26<32 kg/m²), obese (≥32 kg/m²).

Of those who self-reported being *'Under-weight'*, 6.7% and 63.5% of European and Pacific people were *'overweight or obese'*, respectively. Of those who self-reported being the *'Right-weight'*, 26.6% of Europeans and 90.4% of Pacific were *'overweight or obese'*. Of those who self-reported being *'Overweight'*, 82.7% of Europeans and 100% of the Pacific group, were *'overweight or obese'*.

Table 6. Multivariate odds ratios (95% CI) for factors that may contribute to obesity

Variables	Model 1	Model 2	Model 3
Age	1.01 (1.00-1.03)*	1.01 (1.00-1.03)*	1.01 (1.00-1.02)
Male	1.33 (1.03-1.73)*	1.34 (1.03-1.75)*	1.36 (1.04-1.76)*
Pacific	16.78 (10.57-26.57)‡	12.92 (7.82-21.35)‡	17.73 (11.27-27.91)‡
NZDep2001	–	1.07 (1.02-1.13)†	–
Non-smokers	–	–	1.63 (1.09-2.43)*
Variables	**Model 4**	**Model 5**	
Age	1.01 (1.00-1.02)* 1.37 (1.05-1.78)*	1.01 (1.00-1.02) 1.47 (1.12-1.93)†	
Male			
Pacific	15.66 (9.77-25.11)‡	11.72 (6.91-19.86)‡	
NZDep2001	–	1.07 (1.02-1.13)* 1.96 (1.28-2.99)†	
Non-smokers	–		
Exercise > 30mins a day	0.59 (0.44-0.78)†	0.59 (0.44-0.78)†	

*p<0.05, †p<0.01, ‡p<0.0001.

Five multivariate models for obesity are presented in Table 6 for the total sample. The comparison group are those participants with normal weight. In Model 1, Pacific ethnicity is associated with an increased odds of being obese of over sixteen fold compared with Europeans. Adjusting for deprivation (NZdep2001) impacts the greatest on the Pacific odds ratio lowering it to 12.92 (model 2).

The following models show that being male, having increased deprivation, and non-smoking is associated with an increased the odds of being obese and that participating in exercise is associated with a decreased odds of being obese. In the combined regression (model 5) all factors maintain their significance and Pacific people are over eleven times as likely to be obese than Europeans.

When separate models were run for Pacific people or Europeans alone, all odds ratios were in the same direction except for gender. Pacific females were more overweight than males but the reverse was true for Europeans. Participation in exercise was protective for both Pacific and European, however, the relationship was not as pronounced for Pacific compared to Europeans nor was it statistically significant. Odds ratios and 95% confidence intervals for Pacific and European were 0.74 (0.48-1.14) and 0.56 (0.42-0.76) respectively**.**

Discussion

Prevalence of overweight & obesity

A previous study by our group, the 1988-90 Workforce Diabetes Survey (WDS) found that 94% of Pacific men and 93% of Pacific women were *'overweight or obese'*. If the same BMI cut offs (overweight ≥ 25, obese ≥ 30) were used to analyse the current DHAH data, then 95% of men, and 100% of women would be *'overweight or obese'*.

Within the Pacific population there has been a large shift into the obese category, the WDS reported 55% of men and 72% of women were obese, whereas our study found that 73% of men and 83% of women were obese.

Although these studies are not directly comparable due to different study design and age categories, the results probably reflect a shift to higher levels of overweight and obesity in Pacific communities, similar to those reported for the national New Zealand population.[16] Other support for this is found when comparing the 2002/03[17] and 2006/07[3] New Zealand Health Surveys (NZHS) where the proportion of Pacific participants who were obese increased from 38% to 64% for men, and from 47% to 66% for women.

Comparing mean BMI levels between the two studies (WDS and DHAH) also shows an increase over time. On average BMI levels of Pacific men increased by 1.8 kg/m² between these studies and for Pacific women an increase of 2.9 kg/m² was observed**.** This increasing trend of *'overweight and obesity'* is in line with the total New Zealand population's trend of increasing BMI over time.[16]

Furthermore, more detailed ethnic comparisons show obesity is increasing in both Samoan and Tongan men and women. Comparing findings of the South Auckland Diabetes Project /SODP[18] study conducted during 1991-96 to the DHAH, the

proportion of obese Samoan men and women increased from 46% to 58%, and 60% to 75% respectively; and for Tongan men and women obesity increased from 49% to 60%, and 59% to 78%, respectively.

These findings clearly illustrate how almost an entire community has been adversely affected by an obesogenic environment and the findings equally clearly demonstrate the need for public health interventions to be designed for the whole Pacific community. More effective strategies may require a structural shift in resources that foster more supportive environments, rather than supporting individual-focussed nutritional and activity initiatives that struggle to operate with observable success in an inhibitory / obesogenic environment.

Perceptions of body weight

It is apparent that Pacific people have similar awareness of their weight (in kg) as European New Zealanders. Despite a larger proportion of Pacific people reporting being heavier than 1 year ago and despite their much higher prevalence of overweight and obesity compared to Europeans, significantly fewer Pacific people regarded themselves as being overweight compared to Europeans.

Over 95% of the Pacific sample (men and women combined) were classified (using BMI) as *'overweight or obese'* however only 61% reported the perception that they were overweight (Table 5)—leaving a 34% shortfall. Similar findings have been reported elsewhere, which found that Samoans residing in New Zealand and Samoa who were above normal weight did not perceive themselves as being overweight and were happy with their body size, weight and health.[19]

In contrast, 67% of Europeans surveyed in the DHAHS were categorised as being *'overweight or obese'* which was accurately reflected in their perceptions as 73% reported the perception that they were overweight.

These findings are similar to those reported from the WDS[20] and indicate that Pacific people have significantly different views on body image than Europeans with a preference for larger body size.[21,22]

Reasons identified have been that cultural preferences and established norms for a larger body size are partly responsible for these differences. The cultural preference for larger body sizes are reported to be symbolic of higher status, hierarchy and beauty. [23-25]

While this study comprised older adults most of whom were born in the Pacific (95%), research on the perceptions of young New Zealand born Pacific children have found a similar preference for a larger body size in some Pacific youth as well.[26]

Overweight & obesity and health

Previous research assessing socioeconomic, diabetes and CVD risk factors in this study found that Niueans and Cook Islands communities had a more favourable socioeconomic profile and that the Niuean community had the healthiest CVD risk profile, reporting the lowest inactivity levels, highest average exercise times, smallest waist to hip ratios, and the healthiest lipid profiles resulting in the lowest CVD risk scores of the Pacific groups.[27,28]

With regard to diabetes, Niuean men and women also had the lowest HbA1c, and prevalence of total and previously diagnosed diabetes.[27]

However, Niueans had similar levels of *'overweight or obesity'* as other Pacific groups, which questions the type of, and characteristics in which overweight and obesity has developed in this community that has resulted in a significantly healthier CVD and diabetes profile. Niueans had the lowest waist circumferences of the Pacific ethnic groups which may mean that this measure of abdominal obesity is a better indicator for overall health than weight or BMI.

Risk factors for increased BMI and obesity

Tables 7 presents multivariate regression equations that models 'obesity', using 'normal weight' individuals as the reference group. In this model it is shown that adjusting for deprivation lowers the likelihood of Pacific individuals being obese from 16.78 to 12.92 suggesting that cultural factors are more important in the development of overweight and obesity. In the combined multivariate model (Table 6, Model 5) and after adjusting for possible confounding factors, older Pacific adults were over 11 times as likely to be obese than Europeans. This finding is similar to those found in a study that compared levels of extreme obesity in children aged 5-14 years, where Pacific children were 11 times more likely to be extremely obese than their European counterparts.[4]

Conclusion

The continued increase in the prevalence of overweight and obesity in Pacific adults has resulted in almost 100% of men and women being categorised as overweight or obese. It is also apparent that perceptions of an ideal body size for Pacific adults may contribute to the higher levels of overweight and obesity in this population. Observing BMI patterns by age and gender show that the Pacific population are different to the European population and these findings warrant further investigation. As almost the entire adult Pacific population over 35 years of age in New Zealand is overweight or obese, any programme must address the whole community and focus on the environment in which they live.[29,30] Programmes targeted at health education for overweight and obese individuals would be somewhat meaningless.

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Healthier vending machines in workplaces: both possible and effective

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Abstract

Aim To develop healthier vending guidelines and assess their effect on the nutrient content and sales of snack products sold through hospital vending machines, and on staff satisfaction.

Methods Nutrition guidelines for healthier vending machine products were developed and implemented in 14 snack vending machines at two hospital sites in Auckland, New Zealand. The guidelines comprised threshold criteria for energy, saturated fat, sugar, and sodium content of vended foods. Sales data were collected prior to introduction of the guidelines (March–May 2007), and again post-introduction (March–May 2008). A food composition database was used to assess impact of the intervention on nutrient content of purchases. A staff survey was also conducted pre- and post-intervention to assess acceptability.

Results Pre-intervention, 16% of staff used vending machines once a week or more, with little change post-intervention (15%). The guidelines resulted in a substantial reduction in the amount of energy (-24%), total fat (-32%), saturated fat (-41%), and total sugars (-30%) per 100g product sold. Sales volumes were not affected, and the proportion of staff satisfied with vending machine products increased.

Conclusions Implementation of nutrition guidelines in hospital vending machines led to substantial improvements in nutrient content of vending products sold. Wider implementation of these guidelines is recommended.

Snack vending machines are part of an obesogenic environment that promotes easy access to energy-dense, nutrient-poor foods. Typically, vending machines offer few healthy options.[1] Thus, they make the *un*healthy choice the easy choice, which is contrary to the goals of public health nutrition.[2] They are therefore an appropriate target for interventions to improve the nutrition environment.

Previous research has examined the effect of reduced price and/or promotion of healthier choices in vending machines.[3-7] However, no studies to date have assessed the impact of vending nutrition guidelines on the nutrient profile of products sold from vending machines.

Nutrition guidelines have been used in a variety of interventions and situations. Positive effects have been seen in schools in the United States. Implementation of nutrition guidelines has lead to increased proportion of snacks meeting guidelines stocked in vending machines,[8] decreased consumption of snacks with low nutritional value,[9] and decreased purchasing of less healthy meals.[10]

Recent research on the feasibility of point-of-purchase interventions strongly recommended pilot studies which examine sales figures, consumer satisfaction, and

practical feasibility prior to implementation.[11] This pragmatic community intervention study aimed to assess the effect of implementation of healthier vending guidelines on the nutrient content of products sold, sales, and customer satisfaction.

Specific objectives were to develop nutrition criteria for vending machines (Better Vending for Health [BVFH] guidelines)[12] and assess their effect on the amount of energy, total fat, saturated fat, sugar and sodium sold from snack vending machines; measure total product sales; and assess staff satisfaction with vending machine product choices. Vending machines selling beverages were excluded.

Methods

Study setting—The study was carried out at two hospital sites in Auckland, New Zealand (North Shore Hospital and Waitakere Hospital) between March 2007 and May 2008. Hospital vending machines were accessible by staff and some visitors. Staff working at the hospitals included administrators, managers, health professionals, cleaning and catering staff, security, and clerical staff. At baseline 4700 staff (4000 Full Time Equivalent positions [FTE]) worked at the two hospitals.

Study phases—There were three phases to the study: development of nutrition criteria; staff surveys pre-intervention and midway through the intervention; and collection and analysis of 3 months of vending machine sales data pre-intervention and post-intervention. Sales data were linked to a specially compiled nutrient database of vending snack products. The Northern X Regional Ethics Committee stated that ethical approval was not required for the study.

Nutrition criteria development (BVFH guidelines)—Nutrition criteria for the BVFH guidelines were developed by eight nutrition professionals representing local District Health Boards, the Auckland Regional Public Health Service, the National Heart Foundation of New Zealand, and the University of Auckland. The nutrition criteria (Figure 1) focused on energy, saturated fat, sodium, and portion sizes of confectionery. They were based on existing New Zealand food and nutrition guidelines and classification systems, including the Food and Beverage Classification System for schools, and modelling of the discretionary energy allowance for snacks.

Two levels of classification were developed: 'better' and 'other' choices. Products had to meet one of these classifications to be stocked in vending machines, and the ratio of 'better' to 'other' choices could be adjusted to suit individual workplaces. To be a 'better choice' item, foods were required to contain ≤800kJ per packet, ≤1.5g saturated fat per 100g, ≤450mg sodium per 100g, and not be confectionery. 'Other choice' items were only required to meet the energy criteria (≤800kJ/packet).

Preliminary modelling work was conducted to assess how many current vending snack foods met the criteria, to ensure foods were classified in a way that was consistent with foods generally considered as healthier options, and to ensure the guidelines were appropriate and feasible. The guidelines were intended to be simple and straightforward to facilitate use by vending contractors, and able to be written into the vending contract. The vending contractor was trained how to use the guidelines, and was responsible for ensuring machines were stocked correctly.

In this study, vending machines were stocked with 50% better choices and 50% other choices. Examples of 'better choice' vending items included small packets of rice crackers, dried fruit, tuna and crackers, dried fruit bars, and soup mix sachets. 'Other choice' items included finger-sized chocolate bars, small cookies (30g) with reduced saturated fat content (1.5g), lower fat potato chips, some puffed snack products, some muesli bars, and small packets of confectionery.

'Other choices' were included in the guidelines because of limited availability of products for vending machines that met 'better choice' guidelines. Furthermore, it was recognised that allowing inclusion of some 'treat' type foods would provide more flexibility for individual worksites adopting the guidelines. The 800kJ cap on 'other choices' ensured that these treat foods were in appropriate serving sizes, and eliminated the traditional vending range of high-energy foods.

Figure 1. Criteria for snacks sold through vending machines (Better Vending for Health guidelines) at two hospital sites in Auckland, New Zealand in 2007-08

BETTER CHOICES	**ENERGY**	**SATURATED FAT**	**SODIUM**
	≤ 800Kj per packet*	≤ 1.5g/100g	≤ 450mg/100g
	Excludes confectionary items: i.e. soft / hard lollies (candy), marshmallows, licorice, chocolate, carob, or chewing gum. Sugar-free varieties are also excluded.		
OTHER CHOICES	≤ 800kJ per packet	Not restricted	Not restricted

* For packets containing more than one serve of an item, it is the packet size (not the serving size) that must meet these guidelines.

Implementation of guidelines—BVFH guidelines were gradually phased into all vending machines at the two main hospital sites over a 2-month period. Products meeting the criteria were identified from the existing vending contractor's product range, and additional suitable products were also sourced. An implementation manual was developed for vending contractors to provide guidance on how to identify suitable products.

The guidelines were introduced as part of an overall District Health Board (DHB) workplace food and nutrition policy that was in the process of being developed and implemented. Under the same policy, sugary beverages had previously been removed from DHB beverage vending machines. Staff were informed of planned changes to vending machines in a monthly staff newsletter, following a baseline staff survey. Vending products were not signposted in the machine as 'better' or 'other' choices.

Staff surveys—Two staff surveys were conducted: one before and the other midway through the intervention phase. Surveys were web-based, and staff were informed of the survey by email. The surveys assessed where staff usually obtained the food they ate at work, frequency of snack vending machine use, reasons for non-use of vending machines, food usually purchased from vending machines, number of items purchased, whether staff tried to choose healthier items, satisfaction with vending machines, suggestions for additional foods they would like in vending machines, and socio-demographic data.

The second survey, during the intervention period, also assessed self-reported change to vending machine purchases following introduction of the BVFH guidelines.

Sales data—Sales data (the number of items re-stocked) were collected from all snack vending machines across two hospital sites (n=14 machines). In addition, a stocktake of each vending machine was completed at the beginning and end of the data collection period. During the stocktake the quality of data collection was monitored and stock was checked for compliance with the guidelines.

Baseline (pre-intervention) sales data were collected for 3 complete months from March to May 2007 and intervention sales data were collected 1 year later from March to May 2008. This time period was chosen to minimise seasonal variation in vending machine purchases. The changeover to healthier vending options was carried out gradually over a 2-month period (October to November 2007).

Sales data were linked to a nutrient database, which contained the energy, total fat, saturated fat, total sugars, and sodium contents of vending products based on the mandatory nutrition information panel information on their packaging.

Study outcomes and analyses—The primary outcome was the effect of the intervention on energy density (kJ/100g) and energy per packet (kJ). Secondary outcomes were sales of total fat, saturated fat, sugars and sodium per 100g of food sold from vending machines; change in machine sales (absolute amount and dollar value); and staff satisfaction.

Product nutrient and sales data were entered into an Excel spreadsheet and extracted into SAS Version 9.1 for analysis. Descriptive analyses were undertaken for both sales data and staff surveys. Measures of variability (e.g. confidence intervals) were not calculated for sales data due to the clear difference in outcomes following intervention.

Total sales value ($) over the 3-month baseline and intervention period were estimated by multiplying product price by number of items sold per product. The price of products at the start of intervention was used for both baseline and intervention price calculations to avoid confounding due to price increases between assessment periods.

Data from the first stocktake at the beginning of the baseline period could not be used due to missing data. However, monthly stocktakes were conducted throughout, and the stocktake from the end of the first month was used as a surrogate starting point for the baseline stocktake. In order to determine whether this made a difference to sales results, a sensitivity analysis was conducted. Sales volumes were also estimated based on number of products restocked only (starting and end stock were not included) as a surrogate for sales.

Results

Staff surveys—The baseline web survey was completed by 18% of all DHB staff (n=835); 84% were female, 57% were health professionals, and 82% were NZ European or Other ethnicity (non Māori, Pacific or Asian), with a mean age of 43 years. The follow-up survey was completed by 13% of all DHB staff (n=611), with similar demographics to the baseline survey, which are generally representative of the staff population (80% female, average age 44 years[13]).

Most respondents either *never* or *infrequently* used vending machines (84% at baseline, 85% at intervention). Vending products preferred by machine users were chocolate and potato chips/crisps, and most users only bought one item at a time (84% and 85%). At least half (51% and 53%) claimed to try to choose healthier items. At baseline, 27% of staff who used vending machines were *somewhat* or *very satisfied* with the vending range, and this increased to 46% post-intervention.

After introduction of the BVFH guidelines, 87% of staff who used vending machines had noticed healthier snacks were available. Forty-seven percent thought the range had improved, 27% thought it was about the same, and 26% thought the range was worse. One-tenth (11%) reported they now used vending machines at work more frequently. Over half (54%) of the staff who used vending machines had changed their choices, with one-third (31%) reporting this change was in order to make healthier choices.

Sales data and effect on nutrient content of products sold—During the 3-month baseline period, 13,749 individual items of food were sold (611 kg total weight) through snack vending machines. In the post-intervention period, 17,425 items (611 kg) were sold, an increase of 3676 items. Total weight (kg product sold) did not change, as some products were sold in smaller-sized packets. Staff numbers also increased over the same time period, by around 400 FTE. When taking staff numbers into account, 3.4 items were purchased from vending machines per FTE over the 3-month baseline period. During the intervention period, 4.0 items were purchased per FTE, giving an average increase in sales of half a packet (0.5 items).

Implementation of the BVFH guidelines decreased average energy content per product sold from 939kJ per packet to 563kJ per packet—a 40% reduction (Figure 2). Per 100g, average energy content reduced by 24% to 1606kJ/100g. The average total fat content per 100g of products sold reduced by 32% (from 28g/100g to 19g/100g) (Figure 3).

Saturated fat also reduced, by 41% to an average of 7g/100g, primarily due to removal of most cookies and some potato chip/crisp varieties. This level is higher than the

'better choice' guideline threshold of ≤1.5g/100g because only 50% of products stocked were 'better choices', and 'other choices' did not have a specified saturated fat threshold. The average percentage of total sugars in products sold decreased by 30%.

Figure 2. Change in the amount of energy per 100g and per packet sold through vending machines at baseline (2007) and post-intervention (2008)

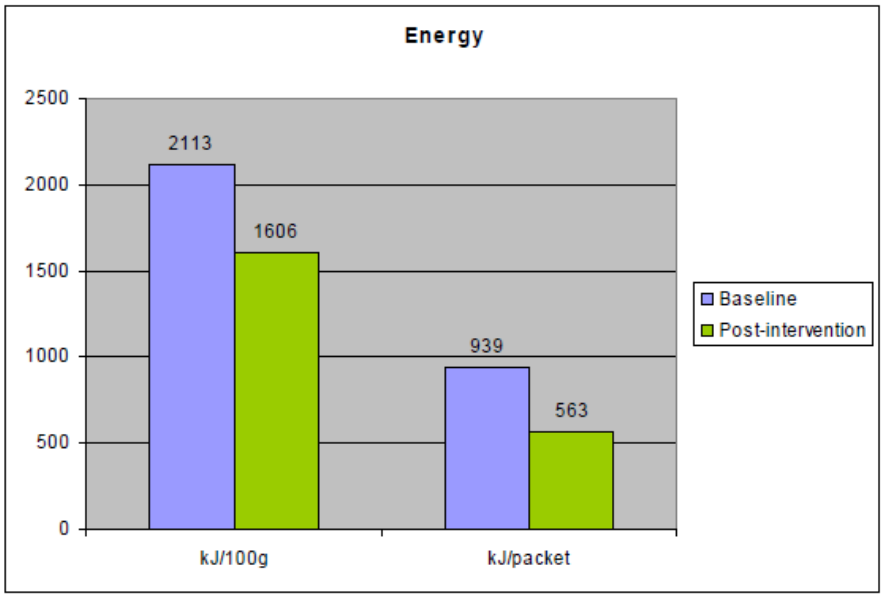
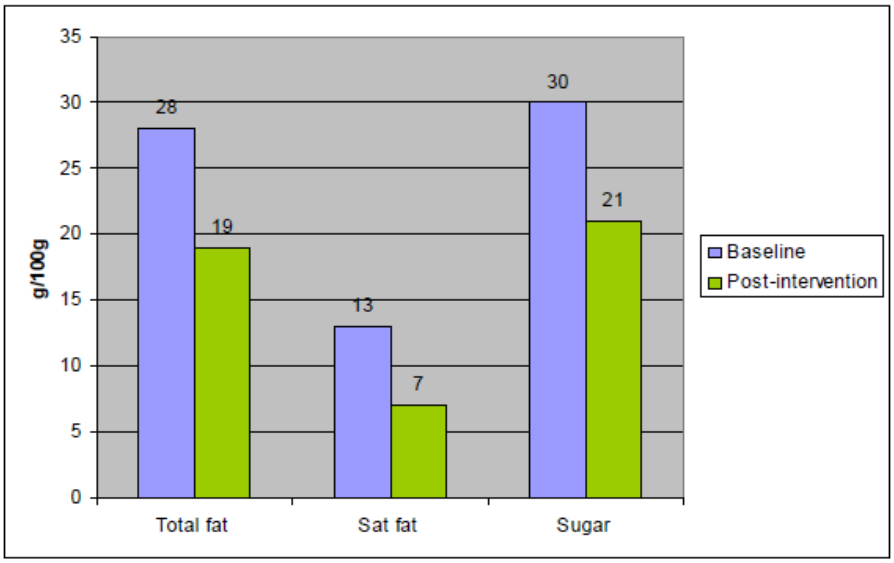
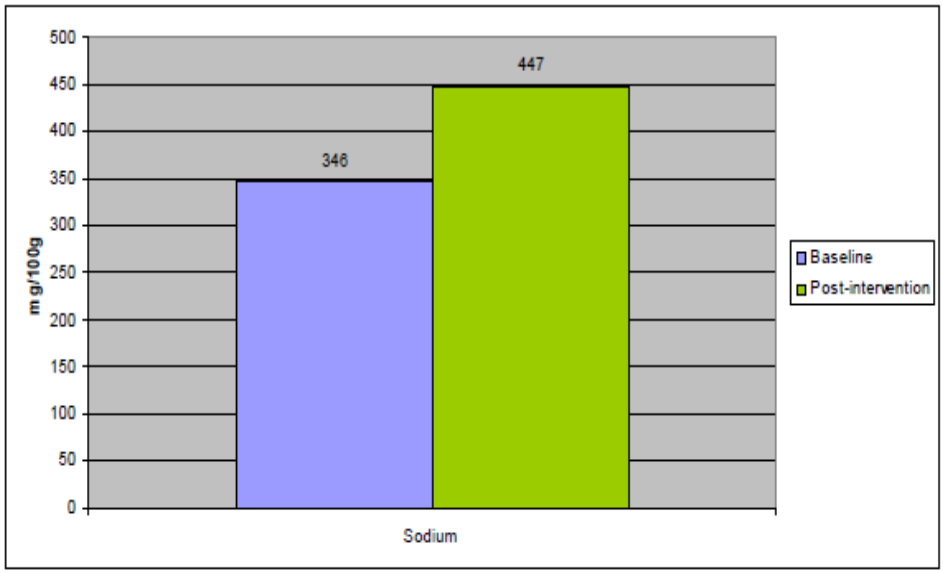


Figure 3. Change in fat and total sugars per 100g product sold through vending machines at baseline (2007) and post intervention (2008)



Sodium was the only nutrient that did not show changes in the direction expected (Figure 4). Overall, levels stayed within the guidelines for 'better choices' (<450mg/100g). However, there was an overall increase of 101mg/100g (29%) sodium in products sold. This increase was largely due to increased sales of puffed snack and potato chip/crisp products.

Figure 4. Change in sodium per 100g product sold through vending machines at baseline (2007) and post-intervention (2008)



The top-selling items (over 500 items sold) during the intervention were reduced-fat chips and puffed savoury snacks, small chocolate bars, small reduced-fat cookies, small packets of lollies, and rice crackers. Of the 'better choice' options, as well as rice crackers, apple crisps (n=328 packets sold), tuna and crackers (n=316), dried fruit (n=232), and soup mixes (n=200) were the most popular items.

Between baseline and post-intervention, the total sales value increased by a total of NZ$1538. Per staff FTE, sales over the 3-months were NZ$5.70 for baseline sales and NZ$5.53 post-intervention. The average price per item sold at baseline was NZ$1.66 and the average price per item sold under the BVFH guidelines was NZ$1.40. The reduced price was due to the lower cost of smaller packet sizes, especially the chocolate bars. Some 'better choice' items in the intervention were more expensive than average, such as cooked rice meals ($3), and tuna and crackers ($2.50). Potato chip/crisps and puffed savoury snacks remained the same price.

Discussion

This study examined a pragmatic intervention aimed at improving the food environment by introducing healthier snack choices into workplace vending machines. Introduction of the BVFH guidelines led to a substantial decrease in energy, total fat, saturated fat, and sugars sold through vending machines. Over a 1-year period, this was equivalent to the removal of approximately 12,400MJ, 210kg of total fat, 130kg of saturated fat, and 220kg of sugars from vending machines at these sites.

Furthermore, machine sales did not decrease (which is an important consideration for vending contractors), and staff reported improved satisfaction with the vending range. Thus, implementing the BVFH guidelines more widely could make a small but important difference to the diet of people who frequently buy snacks from vending machines.

This research adds to the existing body of evidence on vending machines by showing the effectiveness of nutrition guidelines. To date, much vending research has focused on pricing or promotion interventions.[5,6,14-16] Lowering the price of healthier options, however, appears to lead to increased sales of both healthier and less healthy options.[5]

Likewise, promoting healthier choices may increase sales of both healthy and less healthy choices.[15] The current study did not use promotions or price discounts to encourage 'better choices', and instead focused on assessing the effect of increased availability of healthier options, and elimination of products that did not meet the BVFH guidelines.

Whilst introduction of the BVFH guidelines resulted in beneficial changes in most nutrients, the sodium content of products sold post-intervention increased, mainly due to potato chip/crisp or puffed snack sales. As these were a top-selling item, vending machines were stocked with a wide selection. There can be substantial variation in sodium content between flavours in a product range and between crisp or puffed snack products. Including flavours or products with lower sodium contents could therefore potentially reduce the amount of sodium sold. Consideration could also be given to introducing sodium criteria for 'other choices'. Nevertheless, average sodium remained below the guideline threshold and the overall impact of the BVFH guidelines on nutrition was still largely positive.

Introduction of the BVFH guidelines did not result in any substantial changes to usage of vending machines or amount of product purchased. Whilst one-tenth of vending machine users reported that they were using vending machines more frequently, one-third used them less often, and the overall usage remained very low (85% of staff infrequently or never used vending machines).

Average sales per staff member only increased by half a packet over the 3 months, with no change in total weight of product sold. Whilst there was an increase in total sales value, this may have been largely driven by an increased staff FTE. If the FTE had not increased, total sales value may have decreased due to the lower average price per item, due to smaller packet sizes. There was demand from around half of the staff who replied to the survey for healthy options to be supplied in vending machines, although a minority opposed changes.

The BVFH guidelines served to introduce a range of healthier options for those who previously did not have that choice, whilst still providing some 'treat' type options in appropriate serving sizes. It has been said that to succeed in changing diets, healthier foods must first be available,[17] and the BVFH guidelines assisted in increasing availability in vending machines.

Of the nine products that sold over 500 items in the 3-month intervention period, only one was a 'better choice' item (rice crackers). The remaining items were 'other choice' products. Thus, there is potential to improve the range of 'better choice' items to increase demand, although there are currently limited options from which to select, mainly because products are often made in specific packet sizes for vending machines.

Should the BVFH guidelines be implemented widely across worksites, schools and other locations in New Zealand, they should provide an incentive for food manufacturers to reformulate products to meet 'better choice' criteria, thus improving the range of options available. Some manufacturers have demonstrated willingness to reformulate to meet nutrition guidelines such as these, as was the case following introduction of national nutrition guidelines for food and drinks sold in schools in New Zealand.[18] As a wider range of 'better choice' products become available, the ratio of 'better' to 'other' choices stocked in vending machines can be increased to provide more 'better choice' options.

Successful implementation of the BVFH guidelines depends on active participation of both worksites and vending contractors. For this study, initial support for the vending contractor in identifying suitable products was provided by a public health dietitian at the DHB. Similar support could be provided by public health organisations to assist worksites and vending contractors in successfully implementing the BVFH guidelines elsewhere.

Vending machines are likely to remain a part of the nutrition environment. This study therefore provides some reassurance to vending contractors and host institutions regarding the feasibility and acceptability of introducing healthier products into vending machines, in the context of the limitations discussed below.

These results should be used to support the implementation of the BVFH guidelines on a wider scale. Examples of suitable venues would be schools that host vending machines (in combination with the Food and Beverage Classification system), leisure facilities such as gyms, and other worksites. Further research into the additional effects of price and/or promotion interventions on sales in vending machines with the BVFH Guidelines would be useful.

Study limitations and strengths—The strengths of the study include its assessment of the implementation of healthier vending criteria in a real-world setting; its multi method design; the use of sales data as a robust, objective measure of effect; and the extended length of time over which sales data was collected, which minimised any effect of seasonal variability.

The staff survey achieved very low response rates and cannot be considered representative; however, demographics of respondents did not appear to differ substantially from the general hospital staff population (in terms of sex, age, profession, and ethnicity). The study was also conducted in a hospital/health provider

setting, and thus results may not be generalisable to all workplaces. Nevertheless, staff at the hospitals comprised a diverse range of health professionals, administrative, clerical and manual workers.

Price changes could potentially have influenced sales, but this likely worked in both directions. Prices for some of the products reduced due to smaller packet sizes, some remained the same, and others increased.

Finally, we did not assess dietary intakes so cannot estimate the impact of the intervention on overall dietary intake of individuals. It is possible that some compensation may occur at other times of the day that could minimise the overall impact of the vending intervention.

Conclusion

Introduction of healthier vending guidelines led to improved nutrient profile of products sold through worksite vending machines, increased staff satisfaction with the product range, and had no adverse impact on total sales. The results show such guidelines are feasible and acceptable for both consumers and vending contractors. Similar interventions with a wider reach are indicated.

Competing interests: None known.

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*A copy of the guidelines are available at:
http://www.arphs.govt.nz/promoting_health/downloads/BVFH%20A4%20Booklet%20-%20FINAL%20-%20110108.pdf

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Aspartame—facts and fiction

Bernadene Magnuson

In September of 2008, I was invited to New Zealand by the New Zealand Nutrition Foundation to discuss the results of a recent review on the safety of aspartame published in the scientific journal *Critical Reviews in Toxicology*.[1] Unfortunately urban myths surrounding this sweetener are causing undue concern and many New Zealanders are choosing to switch from diet versions of products to high calorie sugar-sweetened versions. This is of concern in a nation where obesity and diabetes, which have well-document health risks, are on the rise. Therefore, I welcomed the opportunity to present the scientific facts about aspartame, in order to help New Zealanders make informed decisions.

As lead author for a team of nine independent internationally esteemed toxicologists, I reviewed hundreds of studies on aspartame safety, providing me with a clear understanding of the science. This review was powerful because as a team, we arrived at our conclusions based on the totality of all safety studies available—published and unpublished (pre-market safety evaluation studies).

The key findings of the review[1] with respect to aspartame safety were:

- Aspartame is completely broken down in the intestine to components found in other foods.
- Aspartame consumption (even at levels much higher than consumed by the highest users) has virtually no impact on blood levels of amino acids, methanol or glucose.
- Aspartame safety is clearly documented and well established through extensive laboratory testing, animal experiments, human clinical trials and epidemiological (population) studies.
- There is no evidence from numerous well conducted studies that consumption of aspartame at levels found in the human diet are associated with conditions of nervous system, behaviour, or other illness.
- Aspartame does not cause mutations, and there is no credible evidence that it causes cancer.

Therefore, the overall conclusion of the study is that, based on the current information available, aspartame is safe to consume even at levels much higher than the highest users are currently consuming.

The problem with many of the "aspartame toxin" stories is that they contain just enough science to make them sound plausible, so I wasn't surprised to hear that even some medical professionals have been questioning the safety of aspartame.

To counter some of the misinformation, I make five key points:

- Firstly, when aspartame undergoes digestion in the gut lumen and epithelial cells lining the gut, the breakdown products are phenylalanine and aspartic acid (two amino acids present in many protein-containing foods) and a small amount of methanol (also present in most fruits and vegetables).[2,3] The amounts of these are much less than found in other foods. For example, aspartame from a can of diet soft drink provides less methanol than a banana, and far less (only 20%) than from the same amount of tomato juice.[2,4] Because aspartame *never* enters the bloodstream as a whole,[3,5] studies where aspartame is directly injected into the body, or added to cells grown in a dish, cannot be used to assess safety for humans. This also explains why aspartame cannot possibly cross into the fetus during pregnancy or into breast milk. In fact studies show that amounts normally consumed in the diet are safe during pregnancy and lactation.[6-12]

- Secondly, it is necessary to explain how our body deals with the methanol produced when the body digests aspartame. The human body is well-equipped to use small amounts of methanol routinely produced from foods and drugs. First, alcohol dehydrogenase in the liver converts it into formaldehyde, which is used within seconds or converted to formic acid, which in turn is used by the body or converted into water and carbon dioxide for excretion.[13] The fact that formaldehyde and formic acid are breakdown products of aspartame sounds scary. But the body is very efficient at using up formaldehdye (it actually needs it for some reactions and therefore produces it endogenously in much greater amounts than we could ever produce by ingesting aspartame),[14] and so formaldehdye never builds up in the body. If the body doesn't need it, it converts formaldehye to formic acid within minutes. In most cases the formic acid will be either excreted in the urine, or broken down to carbon dioxide and water. However, this takes more time and if there is a lot of methanol (or formaldehye) coming into the body, formic acid can build up and that causes the adverse effects seen in methanol poisoning.[13]

  So when the safety of aspartame was being assessed, many studies were conducted to examine whether the consumption of aspartame would affect blood methanol, formaldehyde or formic acid levels in humans. People consuming up to 200 mg aspartame/kg body weight (the normal daily consumption is 5 mg/kg) had a small increase in blood methanol, but this was 100X lower than the amount needed to cause methanol poisoning), no change in formic acid levels (there is always a small amount in blood) and formaldehyde was not detected. Studies in infants and children showed the same thing. People given 10 mg aspartame /kg body weight (about double a normal daily amount), every hour for 6 hours were monitored and there was no change in blood methanol, or any other metabolites.[3,5] So, the amount of aspartame in diet drinks or foods produces so little methanol that there is no chance it could cause a build up of formic acid and cause adverse effects.

- Thirdly, another urban myth is that the methanol in aspartame is handled differently from the methanol in foods, because it is not consumed with ethanol, as it often is in other foods. As I've already explained, there isn't

enough methanol produced from consuming aspartame to cause methanol toxicity. However the statement is worthy of discussion because it is based on science, but is incorrectly extrapolated. Ethanol is also metabolised in the liver by alcohol dehydrogenase. This is why methanol poisoning (high blood methanol, high blood formic acid), is treated clinically by administering ethanol. This stops further production of formic acid as alcohol dehydrogenase will preferentially metabolise ethanol, and slow the methanol metabolism. This gives the body time to breakdown the excess levels of formic acid before more is produced.

So the argument that the ethanol protects against methanol poisoning is correct, but this only is relevant when there is sufficient levels of methanol to cause a build-up of formic acid, and when there is sufficient ethanol to offset the metabolism of methanol. When people consume foods and drinks containing aspartame, such a small amount of methanol is released and metabolized, that there is no change in blood methanol or blood formic acid levels, so it makes absolutely no difference if you concomitantly consume ethanol or not.

- Fourth, allegations have been made that industry-funded studies always find no adverse effects while "independent" studies find adverse effects. This argument is both misleading and false. For example, three studies in mice conducted by the US National Toxicology Program (an independent group) concluded that aspartame is not a carcinogen.[15] And a recent large scale US National Cancer Institute epidemiological study (also independent) came to the same conclusion.[16] In addition, industry-initiated research most often examines the effects of ingesting aspartame—so as to test what happens when aspartame is consumed in foods and drinks. Many "independent" researchers study unrealistic situations such as injecting aspartame directly into the bloodstream, brain or other organs, *and/or* use doses far beyond what anybody could conceivably consume. At doses thousand of times what human consume, as with any compound, adverse effects will be seen. This is often a result of creating an unnatural and imbalanced intake of amino acids.
- Lastly, two studies in rats conducted by the Ramazzini Foundation[17–19] are often upheld as scientific proof of adverse effects due to aspartame. The first study reported an association between aspartame and leukaemia and lymphomas, and the second reported increased cancers in rats fed aspartame for their lifetime, and whose mother was fed aspartame during pregnancy. This research, which is in contrast to all previous studies finding no effect of aspartame on cancer, has been carefully reviewed by numerous international food safety authorities and other experts. All found serious flaws in the research methodology and interpretation of results, which are discussed at length in our most recent review[1] as well as a subsequent letter to the editor.[20]

  Some of the flaws included:

  - The experimental animals were housed unconventionally, without the treatment groups being in the same environment. This resulted in some

groups contracting high rates of respiratory infection—a known risk factor for lymphoma and leukaemia.

- When the pathology slides used to draw conclusions about the rates of leukaemia and lymphoma by the researchers were examined by independent reviewers, they did not draw the same conclusions.
- The researchers did not provide information about the baseline rat diet used in their studies. It is known that it is not the conventional "rat chow" diet, and that nutrient levels were not re-adjusted depending on dose of aspartame used, as they are in conventional studies. Therefore there is a possibility that some findings could have been due to nutritional deficiencies in some groups.
- The researchers most recently reported a cancer risk from prenatal exposure to aspartame, without providing any data on aspartame intake (or indeed any other parameter) during pregnancy in rats.

In addition, 14 previous studies in various animal models found no evidence of aspartame causing or promoting cancer development.[1] Thus the independent reviews all agree that there is no credible evidence that aspartame is carcinogenic.

I hope that this article helps clarify the scientific facts about aspartame so that New Zealanders can make fully informed choices about their consumption of aspartame sweetened foods and beverages in future.

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The New Zealand sugar (fructose) fountain: time to turn the tide?

Simon Thornley, Hayden McRobbie, Gary Jackson

In 2005 New Zealanders drank and ate, on average, over half a cup (158g) of sucrose (sugar) per day. In contrast, less than 40g a day (about one and a half tablespoons) are recommended by the World Health Organization to prevent dental caries, obesity and chronic disease.[1] The growth of global sugar demand has been well documented,[2,3] but is it a major concern? Here we review the composition of sugar, its physiological properties, consumption trends, risks it poses to New Zealanders' health, and, finally, should we attempt to turn the tide?

By *sugar* we mean white table sugar, or crystalline sucrose, derived from sugar cane or beet. Sucrose is also found in less concentrated form in fruits and vegetables, and consists of a single molecule of naturally occurring glucose and fructose, chemically linked.

Trends in sugar intake

A national increase in demand for sugar has occurred in the last 40 years. In 1961 daily per capita consumption of sugar was 126g, which increased to 158g in 2005; a 25% increase occurring mostly between 1980 and 2005.[4] In New Zealand adults, between 8–10% of daily energy consumed came from sucrose consumption, whereas children (aged 5 to 14) consumed between 10–16% of daily energy from this source.[5] In children, 25% came from beverages, with powdered drinks the main source (45%), followed by soft drinks (33%) and cordials and fruit drinks (15%).[6] The other 75% was attributed to a wide variety of foods. Evidence suggests that soft drink consumption is a growing source of sugar in the New Zealand diet with sales increasing 4% per annum in the early 2000s.[7]

What is wrong with sugar?

Traditional arguments mounted against sugar focus on it as a source of "empty calories".[8] This statement is true, refined sugar lacks any micronutrients, present in less refined sugar cane derivatives (molasses). However, having "empty calories" does not distinguish sugar from other *refined* carbohydrates, such as starch. Sugar, like any other purified carbohydrate, has an energy density of 17kJ/g (equivalent to protein), compared to fat, which has over twice the energy per unit mass (34kJ/g). Therefore its caloric content may not be the main contribution of sugar to adverse health outcomes.

In exploring such adverse effects on health, we consider sugar's chemical constituents. One half of the sucrose disaccharide consists of glucose, commonly found in starch as long chains (present in flour and potatoes) and other disaccharides such as lactose (found in dairy products). Some diets are based on the physiological properties of glucose. The 'glycaemic index' (GI), for example, measures the

physiological response of serum glucose to a carbohydrate load.[9] Low glycaemic index diets have been shown, in meta-analyses, to both reduce risk of chronic disease risk and weight in obese people.[10,11] Serum glucose is commonly measured in plasma to detect diabetes, and average serum glucose level over three months, measured by HbA_{1c} correlates with increased risk of mortality.[12] Sugar, however, has a moderate GI (68), mainly due to its high glucose component, so advocates of GI based diets downplay the role of sucrose in weight gain and chronic disease.[9]

The other half of the sucrose molecule, fructose, has little effect on GI, but is it benign? Fructose receives scant attention in nutritional science or medical practice. While free fructose naturally occurs in honey and fruit the most common source is as a disaccharide, in sugar. What is unusual about sugar is its concentration of fructose. For example, banana typically contains 6% fructose by weight (from both free fructose and sucrose). Sugar, in contrast, has an equal ratio of fructose to glucose (50% by weight). In the United States, high fructose corn syrup often replaces sucrose in food manufacturing. It is simply fructose and sucrose in their elemental form, rather than as a disaccharide. In this article we draw a distinction between *concentrated fructose* present in refined sugar, and the lower concentrations in naturally occurring sources.

The sugar-refining process not only concentrates sucrose but removes substances which slow its digestion and absorption. Amongst these elements are polyphenols which inhibit digestive enzymes in the human gut. Cross over studies indicate that polyphenol rich meals reduce the glycaemic index of matched carbohydrate loads.[13] Polyphenols therefore favourably slow the absorption of glucose, with likely similar effects on fructose.

After ingestion, fructose is absorbed from the mid to distal small bowel and almost completely metabolised by the liver, independent of insulin. Unlike glucose, fructose does not stimulate insulin release. Metabolism of fructose depletes intracellular energy stores (ATP), and induces uric acid production.[14] The principal products of hepatic fructose metabolism are triglycerides, which are then released into the circulation.

While fructose is processed, conversion of glucose to glycogen (glycogenesis) in the liver is blocked. The reduction in glucose metabolism, in turn, causes insulin levels to rise so that glucose is taken up in alternative sites, such as muscle tissue. Such high insulin levels leads to compensatory insulin resistance in muscle tissue.[14] This mechanism may explain how fructose has little acute effect on serum glucose levels, but importantly, impairs glycaemic control after long-term exposure to high doses. Further details of fructose physiology are presented elsewhere.[14]

Animal studies have documented adverse metabolic effects of refined fructose consumption. Rodents fed on high fructose and sucrose diets, but not high glucose diets develop features of the metabolic syndrome, such as hyperinsulinaemia, hyperuricaemia and hypertriglyceridaemia.[14]

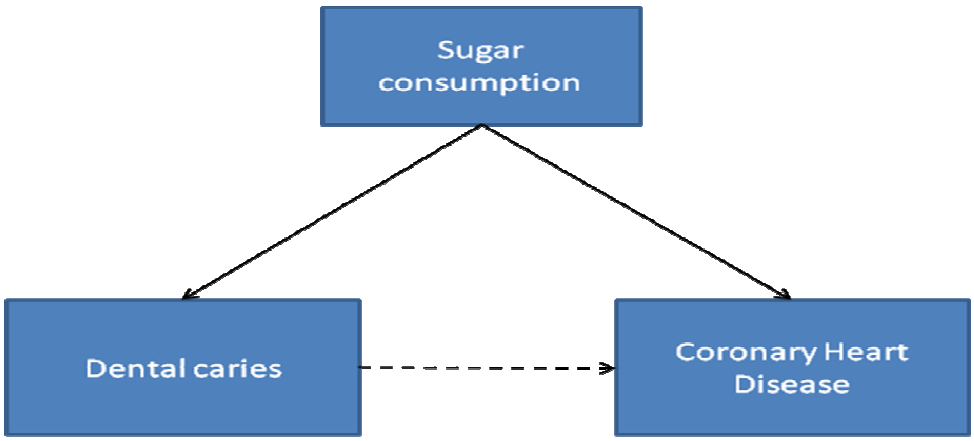
In contrast to animal data, links between fructose and adverse health outcomes have not been so convincingly demonstrated in humans, although longer exposure studies and higher doses of fructose (>200g/day or the equivalent of two cups of sugar) tend to produce clearer adverse health outcomes. For example, small intervention studies

have shown that high doses of fructose provoke insulin resistance within one week,[15] whereas smaller doses (<100g/day) may conversely improve glycaemic control.[14] Whilst 200g is greater than the average New Zealand daily intake (80g/day), variation in consumption means that a substantial proportion of New Zealanders are likely to ingest more than 100g/day. A rise in systolic blood pressure of 7mmHg was observed after intake of 200g of fructose per day for 14 days in a randomised trial (n=74).[16] This study also found adverse effects on triglycerides, fasting insulin and metabolic syndrome outcomes. Other effects include modest weight gain in some short term studies.[17] The health effects of long term, high dose exposure of fructose, which occurs in some subsets of the population have not been studied in experimental trials.

Perhaps the best described effect of fructose consumption is deterioration in lipid profiles. A meta-analysis indicated that fructose worsens serum triglycerides in experimental studies of patients with diabetes, compared with control diets.[18] Increasing evidence supports the association between triglycerides and coronary heart disease, although abnormal triglyceride rich lipoproteins are commonly associated with other adverse lipid abnormalities. Uncertainty still exists over which of these fractions is causally associated with coronary disease.[19]

The best known ill-effect of sugar is dental caries. The British Nutrition Foundation stated that *"the evidence establishing sugars as an aetiological factor in dental caries is overwhelming. The foundation of this lies in the multiplicity of studies rather than the power of any one"*.[20] Starch, and other nutrients, in contrast, show little effect. If sugar causes dental decay, and is linked to coronary risk factors, we expect and have indeed found published associations with disease outcomes (after controlling for established risk factors).[21] Although explanations for this relationship have focused on the putative pro-inflammatory role of oral bacteria, the relationship may be explained by sugar intake (Figure 1).

Figure 1. A plausible causal diagram explaining nature of the association between dental caries and coronary artery disease. Solid arrows indicate proposed direction of causation while dashed arrow shows apparent association



Given that sugar-sweetened soft drinks make up a large proportion of added sugar in modern diets, intake of such beverages may be a proxy for sugar exposure. Systematic reviews of the effect of these drinks consistently show associations with adverse outcomes. For example, a meta-analysis of longitudinal studies investigating the correlation between such drinks and increased body weight, showed an *r*-value of 0.09 (*P*=0.001).[22] One randomised study (considered a more compelling design for assessing causation than observational studies) in which obese adolescents were given either supplemental diet soft drinks or no intervention found a beneficial effect, reducing body mass index.[23]

Sucrose and addiction

Why is sugar consumption rising? Evidence points to sugar possessing rewarding qualities similar to drugs of abuse. Addiction is defined as a *loss of control*, usually associated with the intake of specific drugs that induce consumption of increasing amounts of the substance after initial exposure.[24] Alcohol and opiate dependence, or addiction, are often perceived by society as 'serious addictions' usually because impaired social relationships and work performance coexist.

Conversely, dependence on nicotine and caffeine, for example, are considered 'lesser addictions' as they do not necessarily dominate the addict's life. These 'lesser' addictions do, nevertheless, share many of the other clinical features of more severe syndromes. For example, the repeated quenching of unpleasant withdrawal symptoms from substance use leads to strong negative re-inforcement of such behaviour, shifting drug ingestion from consciously initiated to automated actions. Some readers may be familiar with the relief of mild caffeine withdrawal symptoms; such as irritability and reduced concentration; which often follow the drinking of a cup of coffee.

The biological basis of addiction offers clues to why some substances are rewarding. Symptoms of addiction are linked to part of the brain responsible for subconscious control of behaviour and motivation.[27] The dopaminergic mesocorticolimbic projection, present in the midbrain, is most often implicated. Human and animal studies show changes in this region after exposure to addictive substances. For example, an intravenous bolus of cocaine results in a spike in extracellular dopamine by blocking re-uptake by nerve terminals in the nucleus accumbens. Also, drug induced dopamine release in this projection is associated with "feeling high".[28]

Is sugar consumption similar to other addictive behaviours? Although by no means widely accepted in nutritional circles, evidence supports such a link. Other articles more fully evaluate the evidence for obesity sharing features of addiction,[25 26] so we only briefly discuss the salient evidence. Of all the food groups, carbohydrate is commonly ascribed addictive properties,[29] and within this food group, sugar. In humans, carbohydrate craving has often been reported in obese people,[29] although a full withdrawal syndrome has not been described. We portrayed one case of an obese woman who recounted a likely food withdrawal syndrome after abstinence from sugar and white flour[30] whose symptoms resolved after about one month. This pattern is similar to the temporality of symptoms observed in other addiction syndromes after abstinence. Obese people also show anatomical changes similar to people who suffer from drug addiction, with increased density of dopamine receptors in reward centres compared to controls.[26]

Is action justified?

Observational studies document a range of adverse associations with sugar or soft drink consumption, and limited numbers of experimental studies indicate that such associations are likely to be causal. Unlike other addictions with adverse health effects (such as tobacco), no regulation discourages consumption, and in medical circles, little appreciation of such adverse health effects have surfaced. Other authorities noted similar evidence of adverse outcomes from sugar consumption, yet conclude with no restrictions.[31] Our opinion is that sugar is contributing to obesity, diabetes and associated cardiovascular disease, and by its addictive nature will resist restraint.

Other counter arguments may suggest that sugar is ubiquitous and unlikely to pose a significant health threat, because it only consists of naturally occurring sugars, albeit in a more concentrated form. Sir Richard Doll reflected on a similar point, when in 1947, a cause for an epidemic of lung cancer was sought. Several exposures were mooted - from pollution to arsenic - however smoking was discounted because it

"*...was such a normal thing and had been for such a long time it was difficult to think that it could be associated with any disease*."[32]

From a public health view point, we must consider possible negative consequences of taking action. Restricting sugar intake is unlikely to cause unintended adverse nutritional effects because sugar is devoid of trace micronutrients. At worst, reducing sugar consumption is likely to improve oral health; at best, it will lower rates of obesity, diabetes and cardiovascular disease.

For the clinician, patients with risk factors for, or established, coronary artery disease are likely to benefit from advice to severely limit sugar intake, noting common sources to avoid. Doctors may warn patients that symptoms such as craving, irritability, and limited concentration may peak in the first days after abstinence, but wane after about one month. Careful monitoring for improvement and change in drug requirement (for oral hypoglycaemics and antihypertensives particularly) is prudent in the early stages. Simple quit techniques may also help such as removing the substance from easy access to reduce the likelihood of relapse.

Experience from public health initiatives to reduce smoking prevalence indicates that individual treatment has only a weak effect compared with a more comprehensive population approach such as food reformulation and economic incentives to change behaviour. We agree with arguments to consider incentives to reduce sugar in manufactured foods, such as taxation or legislation, or directly taxing high-sugar beverages themselves.[33] Revenues from such a strategy can be directed to promote healthier food and drinks.

Other population level measures, drawn from tobacco control, include restricting sales and marketing of sugar sweetened products, particularly to younger consumers. Whilst we acknowledge that such a move may be unpopular, the negative externalities that accrue from escalating health care costs of obesity and diabetes, require bold and assertive action if we are to reverse this tide.

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THE NEW ZEALAND MEDICAL JOURNAL
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Bariatric surgery: a cutting-edge cure for Type 2 diabetes?

James Coleman, Simon Phillips

Abstract

Type 2 diabetes is rapidly becoming a global health crisis. It is associated with multiple comorbidities and is placing an ever increasing financial burden on society. There is now a need to explore new methods of tackling this problem. A growing pool of evidence suggests that bariatric surgery has the potential to cure Type 2 diabetes in a select population and provide additional benefits for many of its associated comorbidities. Whilst there are various proposals that explain these phenomena, current research suggests the cause is mainly due to increased post surgical release of insulin promoting gut hormones. The aim of this paper is to introduce some of the complex issues surrounding the use of bariatric surgery in Type 2 diabetes and highlight the controversial aspects encompassing this topic.

Type 2 diabetes is reaching epidemic proportions. As the next generation of frontline doctors, it will be our responsibility to manage this problem using the most effective methods available.

The prevalence of Type 2 diabetes in the UK and globally is approximately 3.5% and this is expected to reach 4.4% by 2030.[1,2] Of these patients, 80% are clinically obese [body mass index (BMI) >30 (kg/m^2)] and 20% of all patients with diabetes will develop microvascular and/or macrovascular complications.[2] This problem currently places a huge financial burden on the UK's National Health Service (NHS), with provision of healthcare for Type 2 diabetics costing £1.3 billion per year. The cost to society in benefit payments and revenue is much greater at an estimated £6.5 billion per year.[2]

Bariatric surgery is a rapidly developing field that has the potential to cure Type 2 diabetes in the majority of individuals with a BMI >35 and reduce the burden of diabetic complications, comorbidities[3] and cost to society.[2]

What is bariatric surgery?

Bariatric surgery is used in the management of the morbidly obese (BMI >40 or >35 with comorbidities such as Type 2 diabetes or hypertension)[4] but it is not widely available in the UK. It involves surgically restricting food intake and/or inducing a malabsorptive state. Whilst there are many different types of surgery, the majority performed are either the laparoscopic Roux-En-Y gastric bypass or laparoscopic gastric band surgery.

Roux-En-Y gastric bypass is a mixed restrictive and malabsorptive procedure that bypasses the stomach, duodenum and proximal jejunum. This surgery is associated with an immediate resolution of Type 2 diabetes.

Laparoscopic gastric banding is a restrictive procedure where an adjustable band is placed around the upper region of the stomach. Here, the beneficial effects occur over a longer period and are associated with gradual weight loss.

What is the proposed mechanism of action?

Type 2 diabetes is associated with insulin resistance and insulin insufficiency. The exact mechanisms are unclear, but obesity and lack of physical activity contribute to insulin resistance. Recent studies have focused on the insulin-mediating action of hormones released from the gastrointestinal tract. Broadly there are two proposals; the distal hypothesis and the proximal hypothesis.

The distal hypothesis proposes that incretin hormones, for example glucagon-like peptide (GLP-1), are secreted by cells within the distal jejunum and ileum in response to intestinal nutrients, improving the efficacy of insulin. This is due to a combination of increasing the secretion of insulin by enhanced proliferation of β cells in the pancreas and decreasing insulin resistance.[5,6]

In the proximal hypothesis it is thought that a counter-regulatory signal, an anti-incretin hormone, is triggered by the passage of nutrients through the foregut and released from the duodenum and proximal jejunum. Its action is to increase insulin resistance by interfering with the incretin mechanism.[5,6] However, recent evidence suggests that this mechanism plays a lesser role relative to the distal hypothesis.[6]

In health there appears to be a correct balance between incretin and anti-incretin hormone secretion. In Type 2 diabetes, the incretin and anti-incretin hormone ratio is skewed, resulting in an imbalance favouring increased insulin resistance.

With gastric bypass surgery the delivery of nutrients is diverted from the foregut to the distal intestine. This increases the secretion of incretin hormones, decreases the release of anti-incretins, and shifts the ratio back to normal glucose homeostasis.

Research outcomes

There is growing evidence that bariatric surgery may effectively cure Type 2 diabetes. In a 2009 meta-analysis of 621 studies, there was an immediate, complete resolution of Type 2 diabetes in 78.1% of patients[7] and improvement or resolution of Type 2 diabetes in 86.6% of patients following the surgery.[7]

For all patients undergoing bariatric surgery there are additional benefits that include resolution of hypertension in 61.7%, resolution of sleep apnoea in 85.7% and an improvement of hypercholesterolaemia in 70% of patients.[3] In the severely obese, bariatric surgery has also been shown to have better outcomes for weight loss over 15 years compared to non-surgical controls (average loss of weight; gastric bypass 27%, controls <2%).[8] It has also been shown that there is a decreased overall mortality in these surgical patients compared to controls over a 16-year period.[8]

Complications

A major issue related to bariatric surgery is whether it is safe. As with any operation there are associated risks, but these should be assessed in light of the perceived benefits.

There are both general and specific complications associated with the surgery.

The general complications include: risk of infection, venous thromboembolism and anaesthetic problems. In 4.3% of patients there will be at least one major adverse outcome within 30 days of the procedure.[9] Nevertheless, the current overall mortality from bariatric surgery is <1% and the laparoscopic approach has the potential to reduce the perioperative risk even further.[10]

The specific complications of bariatric surgery include leaks of gastrointestinal contents, nutritional deficiencies such as iron, folate and calcium deficiency associated with the Roux-en-Y bypass procedure and dumping syndrome associated with gastric banding surgery.

Conclusions

We acknowledge that this is a controversial topic and lifestyle modifications should be the cornerstone of management in all patients with Type 2 diabetes.

Whilst bariatric surgery is recognised as an invasive, expensive procedure requiring rigorous assessment, it has the potential to cure the majority of patients with Type 2 diabetes. It is cost-effective and significantly reduces the risk of diabetic complications and comorbidities. Whilst surgical treatment for Type 2 diabetes is still in its infancy, there are huge implications for future research with the potential of solving a growing epidemic.

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Preoperative diagnosis of cholecystocolonic fistula on ERCP

John P Rice, Bret J Spier, Anurag Soni

Cholecystocolonic fistula (CCF) is usually an incidental finding found during surgical cholecystectomy. While diagnostic testing for CCF is often insensitive, its preoperative diagnosis would alter surgical management (partial colectomy and cholecystectomy versus cholecystecomy alone).

We describe a patient with cholecystitis, cholangitis and pneumobilia who had a CCF diagnosed by endoscopic retrograde cholangiopancreatography (ERCP).

Case report

A 70-year-old immune suppressed man, having recently completed treatment for recurrent large cell lymphoma, developed right-sided abdominal pain, profound diarrhoea and temperature to 103°F (39.5°C), but was otherwise stable haemodynamically. Total bilirubin was 1.6 mg/dL (reference range, 0.0–1.4 mg/dL) and alkaline phosphatase was 514 U/L (35–130 U/L). He was pancytopaenic with a platelet count of 23 K/uL (160–370 K/uL) and was receiving anticoagulation (INR 3.1) for atrial fibrillation and a deep vein thrombosis.

On admission, CT of the abdomen revealed air (white arrow) and stones (black arrowhead) within the gallbladder along with marked gallbladder wall thickening and possible common bile duct stone (black arrow) (Figure 1A). Pneumobilia was also identified (white arrow) (Figure 1B).

Figure 1A. Oral contrast abdominal CT revealing air (white arrow) and stones (black arrowhead) within the gallbladder along with marked gallbladder wall thickening and possible common bile duct stone (black arrow)

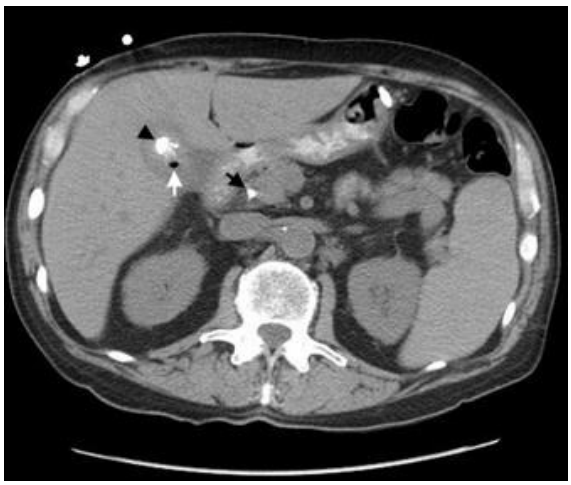
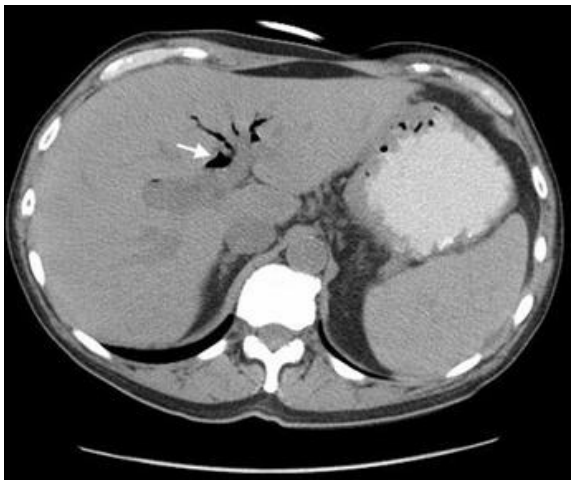


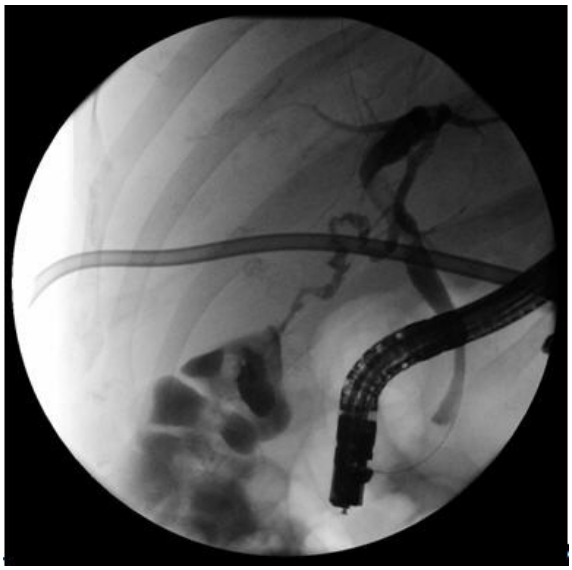
Figure 1B. Abdominal CT demonstrating pneumobilia (white arrow)



Given concern for biliary obstruction with associated cholangitis, an ERCP was performed on hospital day #1, revealing extravasation of contrast from the gallbladder into the colon, thus indicating the presence of a cholecystocolonic fistula (Figure 2). A biliary stent was placed.

By hospital day #3 his liver tests had normalised and fevers subsided. The patient subsequently underwent cholecystectomy and partial colectomy.

Figure 2. ERCP revealing extravasation of contrast from the gallbladder into the colon, a cholecystocolonic fistula



Discussion

Cholecystocolonic fistula (CCF) is the second most common cholecystoenteric fistula.[1–3] In several large series of patients undergoing elective cholecystectomy, CCF is discovered in 0.06 to 0.14% of cases.[1–3] The most common aetiology of CCF is acute cholecystitis, however it has also been described after gastric surgery, cholecystostomy tube placement, post-traumatic, gallbladder carcinoma and iatrogenic causes.[4]

Many patients with CCF are asymptomatic and are only diagnosed during cholecystectomy. The classic symptomatic triad of diarrhoea, right upper quadrant abdominal pain, and cholangitis is uncommon. Diarrhoea is felt to be the most common complaint, occurring in 71 percent of cases.[5] Pain and cholangitis occur less frequently. Other presentations include gallstone ileus/obstruction, liver abscess, and bleeding.[4]

Preoperative diagnosis of CCF is difficult and is achieved in only about 7.9% of patients.[2] A number of different imaging and endoscopic modalities have been described anecdotally including plain X-ray, CT scan, ultrasound, barium enema, colonoscopy, and ERCP.[4] Currently, there is no gold standard, nor reliable method for the nonoperative diagnosis of CCF.

Treatment of CCF is almost always surgical. In uncomplicated cases of CCF, a laparoscopic approach may be feasible, but conversion to an open procedure is not uncommon.[6] In addition, colostomy can generally be avoided. Patients with CCF complicated by bleeding or gallstone ileus often require a more “customised” and extensive surgery.[4] Endoscopic therapy occasionally may be beneficial.[7,8] In some cases of gallstone ileus, colonoscopy has been used to manually extract an impacted gallstone in the sigmoid colon.[7]

In summary, cholecystocolonic fistula is a relatively uncommon complication of gallbladder disease and is most often found incidentally during cholecystectomy. CCF cannot be reliably diagnosed symptomatically, however most patients will complain of diarrhoea. Similarly, imaging and endoscopic procedures have low diagnostic sensitivity. The treatment for most cases of CCF is surgical and its presence may alter management.

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An unusual presentation of staphylococcal tricuspid valve infective endocarditis

Arindam Pande, Biswadip Ghosh, Shantasil Pain, Rathindra Nath Karmakar, Anirban Ghosh, Sandip Saha

Infective endocarditis can present with highly variable and often non-specific clinical features.[1] Here we are reporting an interesting case with uncommon characteristics of infective endocarditis.

Case report

A 28-year-old married lady presented with low-grade intermittent fever, exertional dyspnoea and gradual swelling of whole body starting from lower limbs of 2 weeks duration. It was associated with generalised weakness, cough with whitish expectoration and orthopnoea. She had a history of medical termination of pregnancy, 4 weeks back. It was followed by per vaginal bleeding for 5–6 days. Five days prior to admission, she was transfused with 4 units of whole blood.

Physical examination revealed mild pallor, temperature of 102^{o}F (38.8°C) and features of CHF with bilateral pleural effusion and ascites. She was found to have normocytic normochromic anaemia with an ESR of 120 mm in 1st hour and transudative pleural effusion. Routine urine analysis showed albumin 3+, RBC 15 per HPF, pus cells 30 per HPF, a few granular and hyaline casts. Spot urine albumin to creatinine ratio was 3055.8mcgm per mg. ANA and ELISA for HIV were non reactive.

Echocardiography showed a pedunculated mass of about 10mm length and 6mm globular head, attached to anterior tricuspid leaflet with a severe normotensive tricuspid regurgitation. Blood culture revealed profuse growth of *Staphylococcus aureus* in all the 3 samples. Kidney biopsy showed features of diffuse proliferative glomerulonephritis.

Antibiotic therapy with ceftriaxone and gentamicin was initiated. Follow up investigations showed a rising creatinine level (up to 4mg per dl) though the patient became afebrile. Gentamicin was withdrawn and intravenous linezolid was started following sensitivity report. Oral prednisolone in the dose of 1mg per kg body weight was started as urine examinations showed persistent proteinuria and creatinine level kept uprising.

Following continuation of this therapy the albuminuria subsided, serum creatinine values gradually returned to normal. Repeat blood culture was found to be sterile. Steroid was gradually tapered off. After about 3 weeks of therapy, repeat echocardiography showed a hyper mobile vegetation of the same dimensions as the previous one at the same site. The patient was then transferred to the cardiothoracic surgery department, where she was transplanted with a bioprosthetic valve.

She was stable in the postoperative period and is now doing well.

Figure 1. Vegetation at tricuspid valve (encroaching ventricle)

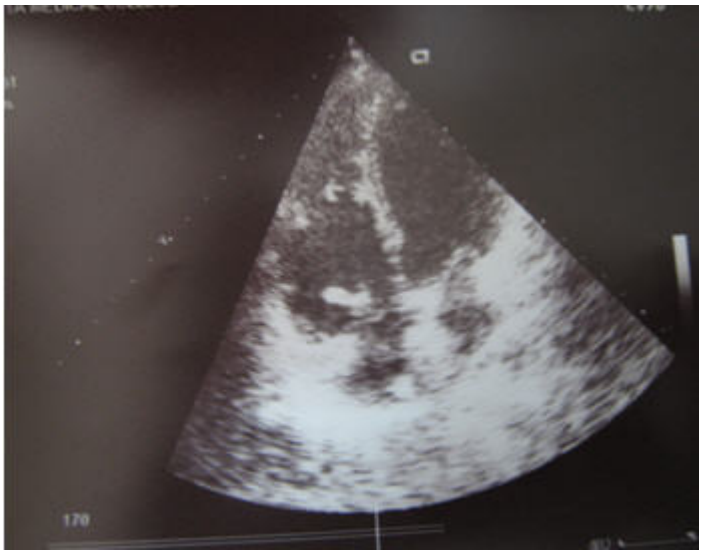


Figure 2. Vegetation at tricuspid valve (in atrium)

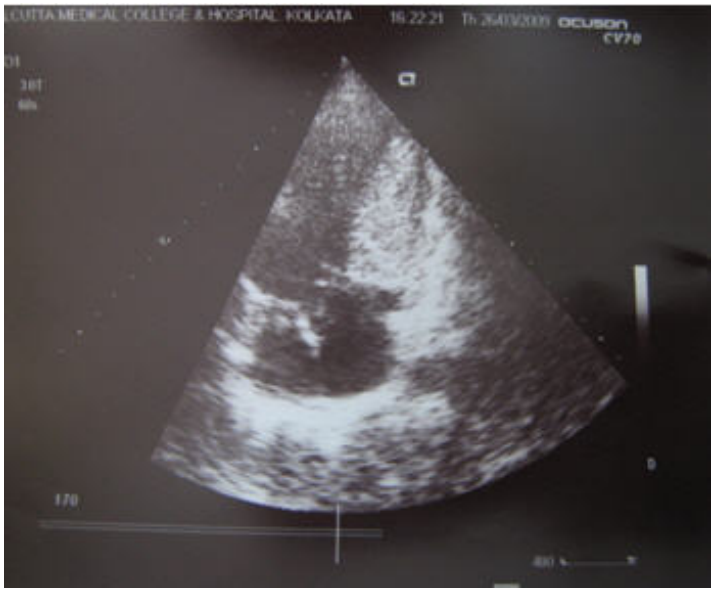
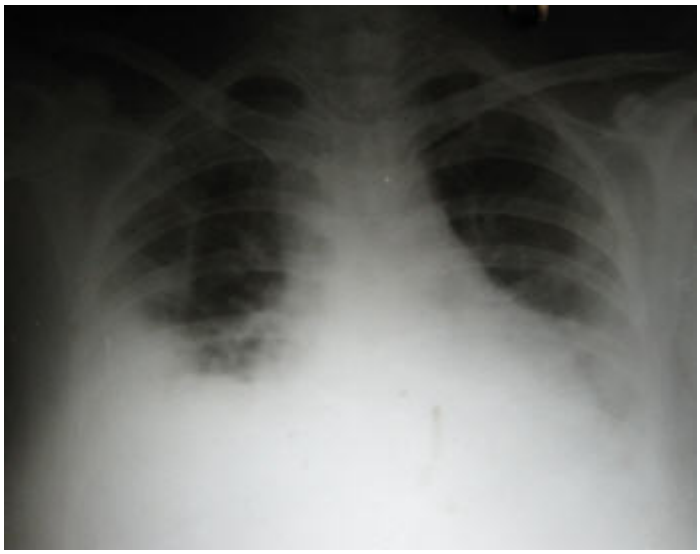


Figure 3. Bilateral pleural effusion



Discussion

In infective endocarditis, glomerulonephritis has been seen to be associated in up to 22% cases in United States.[2,3] It was focal in about 8% and diffuse in about 14% of the cases.[2,3] It has also been seen that *S. aureus* is the most common etiologic agent in those cases.[2,4] With *S. aureus* IE limited to tricuspid valve systemic complications are rare[5] though our patient had glomerulonephritis. Again *S. aureus* IE is characterised by a highly toxic febrile illness,[6] contrary to our case.

The case was also unique in multiple other ways. This non-HIV patient had an unusual combination of tricuspid valve endocarditis in the setting of an intrinsically normal tricuspid valve in a non-drug user. Our patient had received multiple parenteral injections and the longstanding indwelling intravenous cannula may have led to seeding of the tricuspid valve, a setting somewhat akin to that of a drug abuser.

According to a study, it was found that the frequency of isolated right sided endocarditis in patients who are non iv drug users and who don't have a pacemaker is about 2.9%.[7] In a publication in 1989, it was reported that in a series of 80 autopsied iv drug abusers having infective endocarditis, the tricuspid valve was involved in half of the victims compared with 15% of victims dying of acute endocarditis not using iv drugs.[8] In our case, the renal impairment did not improve with antibiotics alone, a short course steroid had to be instituted. Ideal treatment strategy in this regard is not clearly defined. In addition to antibiotic treatment, plasmapheresis and steroids have been used with variable results.[9,10]

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THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

A case of rhinodynia and asthma

Tarek Darwish

Clinical

A 42-year-old African American female with a history of asthma presented to the Emergency Department with a 2-week history of cough and worsening shortness of breath and wheezing. She denied any fever or chills. The patient was admitted for asthma exacerbation management. A presenting chest radiograph (Figure 1) and subsequent chest and abdominal radiographs (Figure 2 and 3) were obtained.

Figures 1 and 2. Chest radiographs

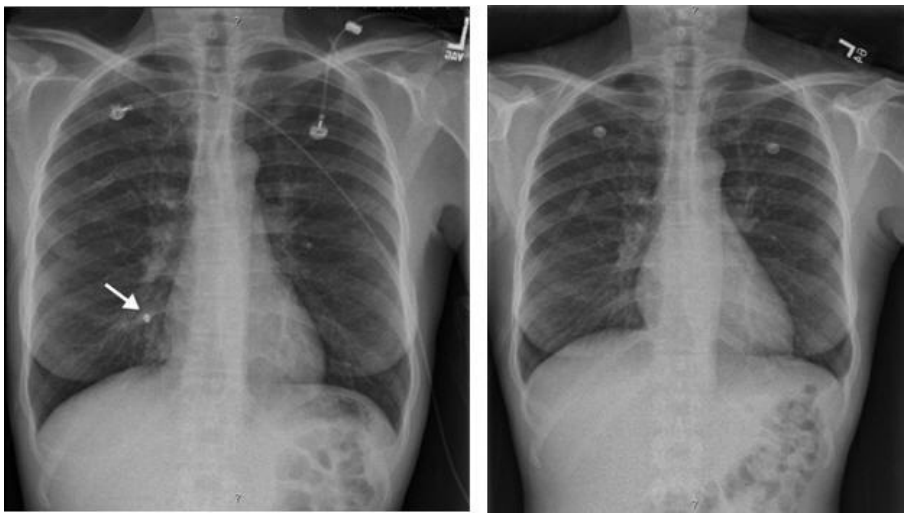
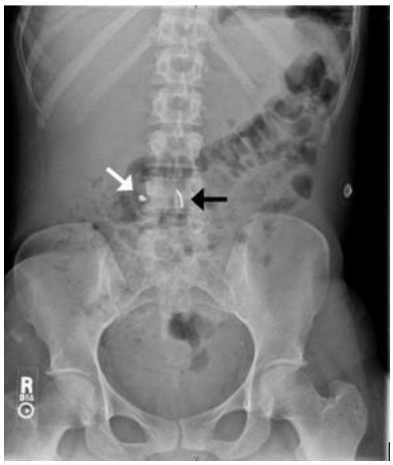


Figure 3. Abdominal radiograph



What is the abnormality?

Answer

The chest radiograph shows an aspirated rounded metallic body in the right lower bronchus (Figure 1).

After questioning the patient, she admitted that her *nose piercing* had loosened 2 days prior to admission and that she had felt a piece migrate posteriorly into her nose. A bronchoscopy was planned on the next hospital stay. However, the ring was not visualised despite repeated chest radiograph (Figure 3). Thus, the bronchoscopy was cancelled.

An abdominal radiograph was performed. It noted a rounded metallic density in addition to a linear one (represents a *belly button ring*); both were located in the patient's mid abdomen (Figure 3). These findings supported our belief that the patient had swallowed the ring backing while she was coughing heavily.

Serial abdominal radiographs ultimately confirmed the successful elimination of the ring backing with the patient's bowel movement.

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THE NEW ZEALAND MEDICAL JOURNAL
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Painful foot swellings

Nadir Goksugur, Betul Sereflican, Berna Kılıç, Kutay Ozturan

Clinical

A 70-year-old man was admitted to the hospital with painful erythematous swelling of left fifth digit and multiple nodules on both feet (Figure A). The metatarsophalangeal joint of the left first toe was severely affected (Figure B) and was firm on palpation. On the left foot, there were three ulcerated areas (Figure C).

A white, pasty material was exuded from the small orifice of the ulceration (Figure D). This “toothpaste”-like material was also seen in the base of the big ulceration. An AP left foot radiograph was performed (Figure E).

Figure A

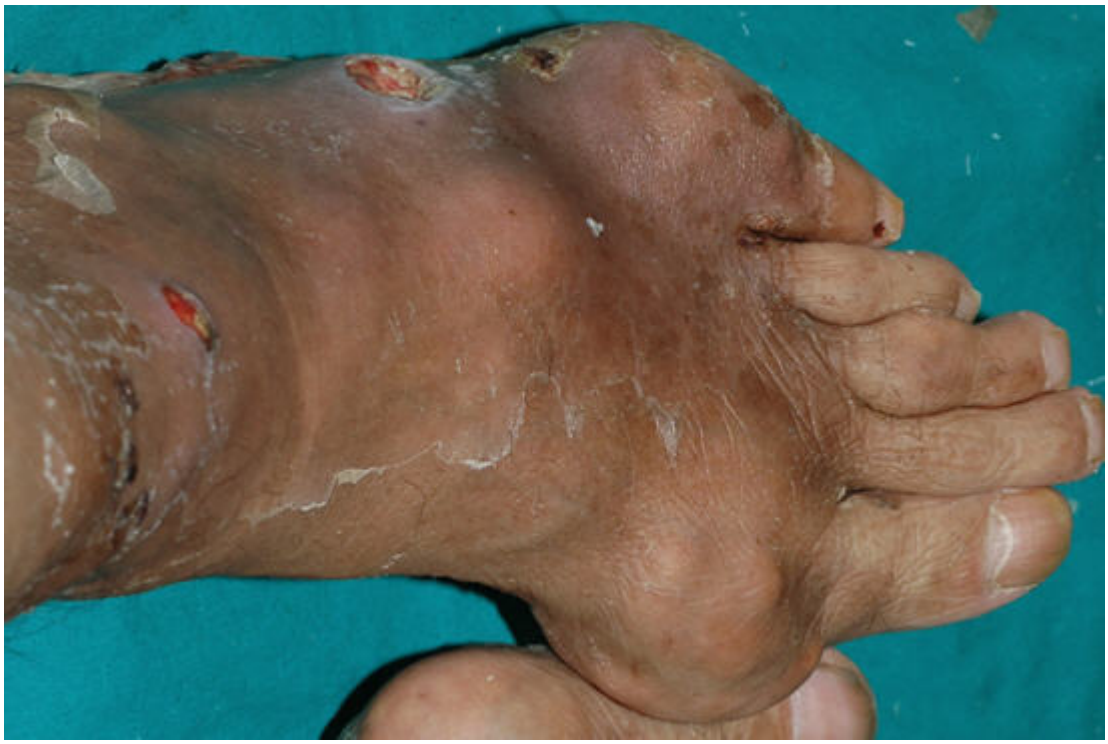


Figure B

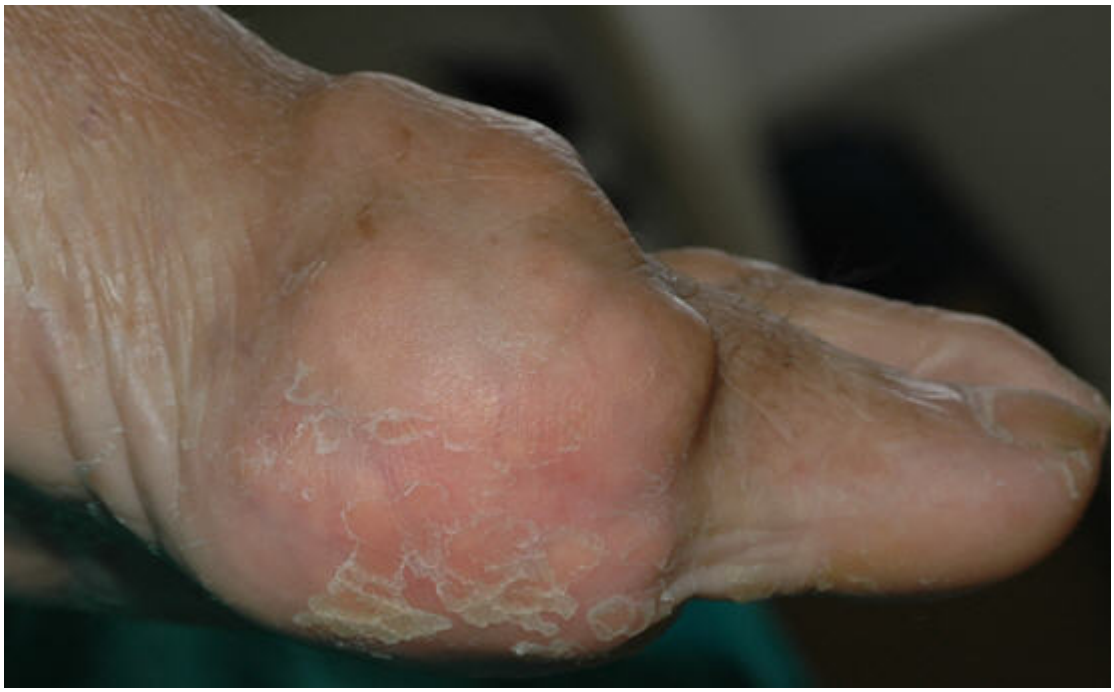


Figure C

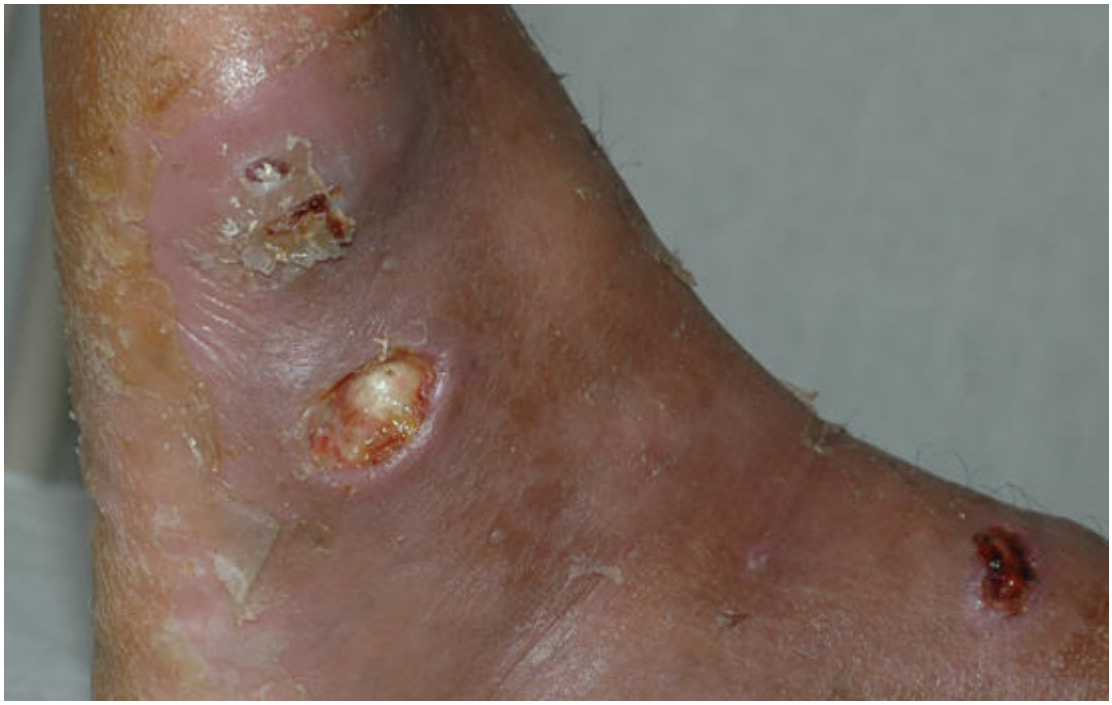


Figure D

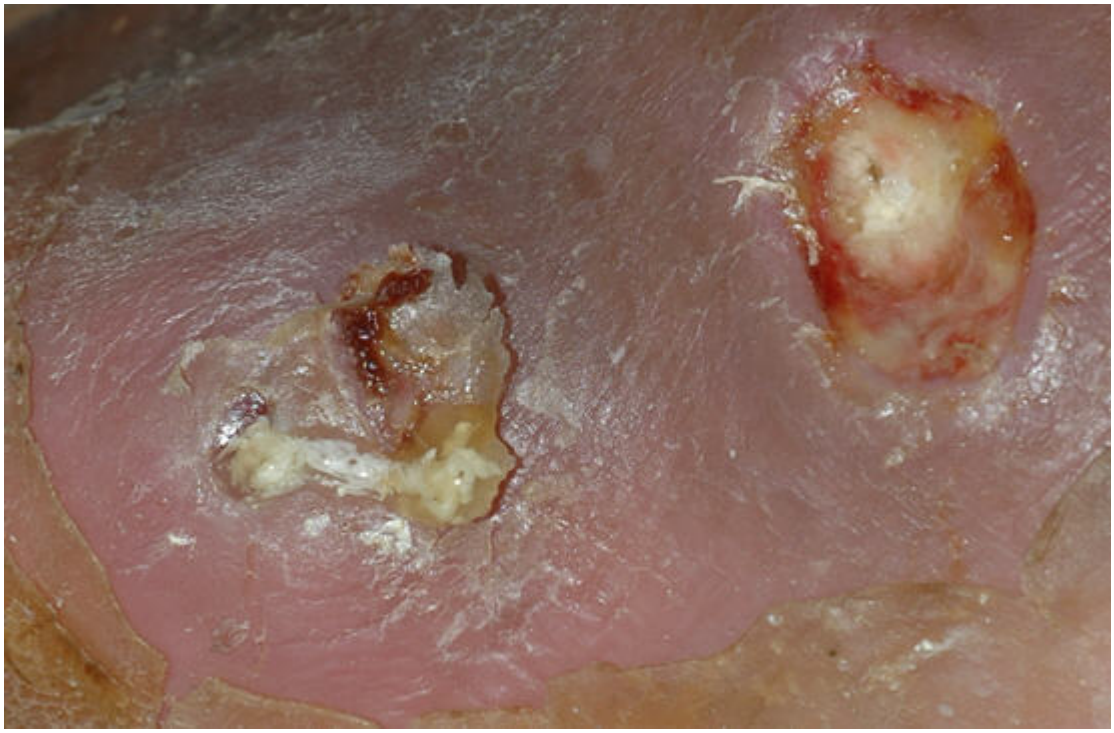


Figure E

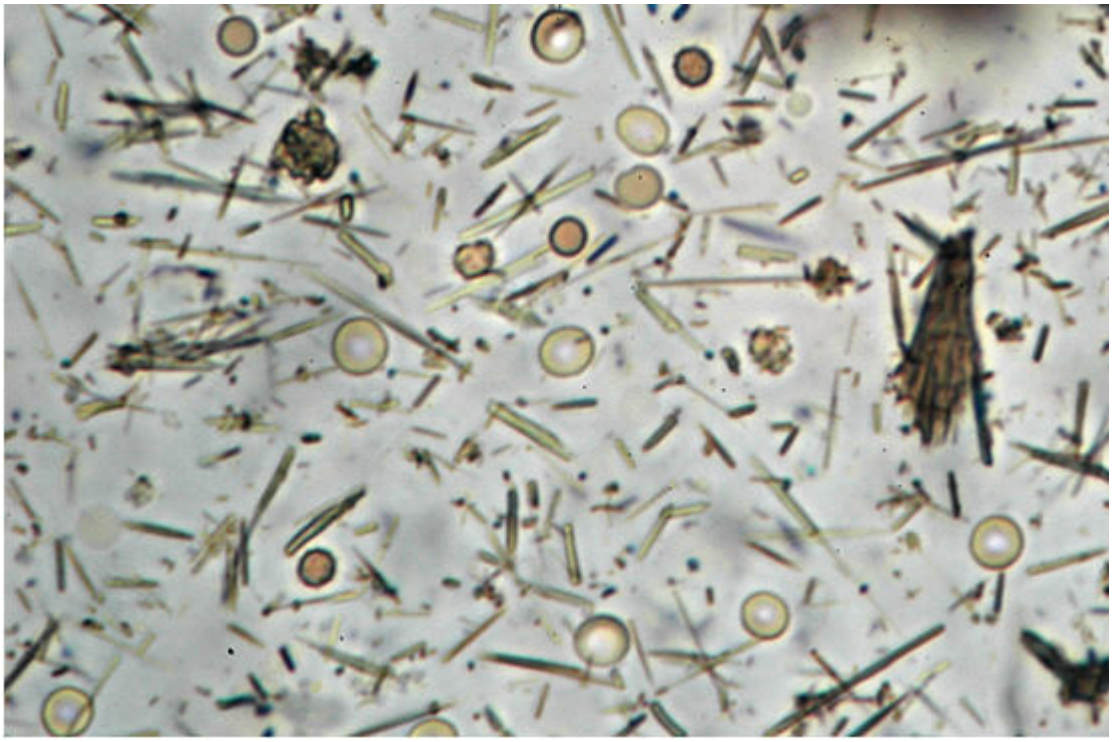
What is the diagnosis and what would microscopy show?

Answer

These findings are consistent with *tophaceous gout*. Light microscopic examination of the exuded material showed abundant needle-shaped *monosodium urate crystals* (Figure F).

A radiographic evaluation (Figure E) demonstrated soft-tissue swellings (arrowhead), bony erosions and cystic changes (arrows) involving metatarsophalangeal joints.

Figure F



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THE NEW ZEALAND MEDICAL JOURNAL
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Deaths of King Edward VII and Thomas Hocken

Published in NZMJ 1910 May;8(34):42–43.

EDITORIAL.

THE news of the death of King Edward VII. came as a stunning shock to the mass of the public; but doctors who knew better the dangers which hang over a man whose constitution has been shattered by serious illnesses; who thought how from time to time the hearts of his subjects had been filled with the keenest anxiety as the hourly bulletins were issued, telling of the long struggle while he lay between life and death; of how the exigencies of his busy and arduous life necessitated a heavy drain on the toughest constitution, and of how much the recent conflict between the Lords and Commons must have helped to age a man to whom England and her future was ever the leading thought—we who knew and realised what all this meant were not so completely taken by surprise when the fatal news came. The sorrow we feel, however, is none the less real because we were better prepared for the end. We mourn for the dead King, and at the same time we can with thankful hearts, knowing the true worth of his son, say" God Save the King."

OBITUARY.

With great regret we have to record the death of Dr. Hocken, one of the oldest, best known and must respected doctors in Dunedin. In a future issue we hope to give a detailed account of the great research work to which he has devoted so many years. At a time of life when must men would seek, ease and leisure, he threw himself with the utmost ardour into the task of collating early records of New Zealand history, and of him it may be truly said he died in harness.

NZMJ Note: According the the Alexander Turnbull Library website (http://www.nzhistory.net.nz/media/photo/alexander-turnbull-library-circa-1930s) Thomas Hocken, in 1908, ‘rescued’ the Treaty of Waitangi from the basement of the Government Buildings. He discovered the ‘damaged’ and ‘presumably rat eaten’ documents amid a heap of old papers and rubbish.

Proceedings of 'Tinnitus Discovery': Asia-Pacific Tinnitus Symposium, 11–12 Sept 2009, Auckland, New Zealand

Download the full 167-page document separately from here:
http://www.nzma.org.nz/journal/123-1311/4040/content.pdf

THE NEW ZEALAND MEDICAL JOURNAL
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The US Food and Drug Administration (FDA) and concerns about direct-to-consumer drug or device advertising (DTCA)

The FDA concerns about DTCA have been a worry but has been exacerbated by the internet and development of networking sites and blogs. The advertisements typically contain just the brand name of the product, a reference to the disease or condition it treats, potential benefits, and a link to a product web site. Such advertisements can also boost inappropriate prescribing, with physicians reporting that half of the requests they receive for advertised drugs are inappropriate choices for the patients. These points have come to the attention of the FDA following a review of the subject. Obviously a vey difficult problem. One contributor to the review (a physician) points out that buying the wrong bar of soap may not matter but inappropriate drugs may matter very much. Good luck to the FDA reviewers. As New Zealand is the only other country with DTCA the matter is of importance to us as well.

JAMA 2010;303:311-13.

Chronic obstructive pulmonary disease (COPD)—are there childhood causes?

This international study group note that early life environment is most important for the development of asthma and atopy and they speculate that it may well be true for COPD as well. Over 13,000 subjects in 29 centres were randomly selected and underwent spirometry in 1991-3 and nine years later. They correlated the spirometry results with childhood disadvantage factors—maternal, paternal or childhood asthma, maternal smoking and childhood respiratory infections. They report that those with early life disadvantage have permanently lower lung function, no catch-up with age but a slightly larger decline in lung function and a substantially increased COPD risk. I note that the results were the same if subjects with asthma were excluded. I was surprised that they report that the effect was as large as heavy smoking.

Thorax;2010;65:14-20.

Peptic ulcer bleeding and low-dose aspirin therapy

Low dose aspirin is widely used for its antiplatelet activity in those with cardiovascular risk factors. A dilemma arises when such a patient has bleeding from the upper gastrointestinal tract. After the bleeding has been contained and proton pump inhibition is established the question arises—should aspirin be withheld or restarted? This prospective study addresses this problem. Seventy-eight patients received aspirin, 80mg/d and 78 received placebo for 8 weeks immediately after endoscopic therapy. All patients received a 72-hour infusion of pantoprazole followed by oral pantoprazole.

The outcome—recurrent ulcer bleeding within 30 days was 10.3% in the aspirin group and 5.4% in the placebo group. But patients who received aspirin had lower all-cause mortality rates than patients who received placebo (1.3% vs 12.9%). Of the 10 patients in the placebo group who died, 3 died of gastrointestinal complications and 2 died of pneumonia. The remaining 5 died of vascular complications. The researchers recommend that those with the highest risk (i.e. secondary cardiovascular risk patients) should restart aspirin as soon as possible—maybe within 7 days. An editorial reviewer endorses their conclusions but points out that clinicians will still have to make very difficult decisions and common sense must prevail.

Ann Intern Med 2010;152:1-9 & 52-3.

Can angiotensin receptor blockers (ARB) protect against Alzheimer's disease or dementia?

Recent abstracts have reported on the ineffectiveness of tarenflurbil (19/2/10) in gingko (5/3/10) in the prevention of dementia. What about benefits of antihypertensives, in particular ARB in this respect. Intuitively effective management of hypertension should help preserve brain function and theoretically ARB should be the best in preserving the brain because of their alleged neuroprotective effects. This study involves 819491 men aged 65years or more who were treated with ARB or ACE inhibitors or other cardiovascular drugs. The researchers report a hazard rate of 0.76 for the incidence of dementia for the ARB group compared with other cardiovascular drugs and 0.81 compared with lisinopril. They also note a significantly lower admission rate to nursing home care and death in the ARB treatment cohort. An editorial writer is not convinced pointing out that "the absence of changes in blood pressure during follow-up further clouds interpretation. The non-random allocation of treatment is also a serious problem." The researchers also note that there are several confounding factors including misclassification and lack of precision in the reasons for nursing home admissions.

BMJ 2010;340b:5465 & BMJ 2009;339b:5235.

Magnetic resonance imaging in fetal medicine

Ultrasound has been and remains the mainstay of imaging of the fetus. Ultrasound is used for routine dating, screening, and evaluation of growth. In this review article the place of magnetic resonance imaging (MRI) is discussed. The problem with standard MRI has been the long picture acquisition times which had made it unsuitable for imaging a mobile fetus. However, modern MRI scanners have improved scan times and signal to noise ratios such that a single slice image can take less than a second to acquire. Obviously this makes fetal MRI feasible. The reviewer still regards ultrasound as the first-up test and emphasises that ultrasound and MRI are complementary. Apparently the MRI images are exquisitely anatomically detailed and parents find them easier to understand. Excellent points, however you have to have one of these modern scanners available and have trained staff to interpret the images, which may not be so simple.

Postgrad Med J 2010;86:42-51.

THE NEW ZEALAND MEDICAL JOURNAL
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Australian dust causing respiratory disease admissions in some North Island, New Zealand Hospitals

On 24 and 25 September 2009 the dust which had so dramatically affected Sydney and the South East Coast of Australia crossed the northern half of the North Island of New Zealand. Unusually, because of the particular weather patterns at the time, the dust arrived from the north east. As a result of this the national threshold concentration for PM_{10}, of 50µgm^{-3} (24 hours average) was exceeded. PM_{10} is a term used to describe dust particles 10µm in size or less. They are small enough to enter the bronchi of the lungs and have been known to cause respiratory problems.

Peaks in concentration at Whangarei were greater than 250µgm^{-3} and were at 350µgm^{-3} at the Khyber Pass dust monitoring station in Auckland. Within the Waikato region peak dust concentrations were variable but did generally decline with latitude going south. At Ngaruawahia they were approximately 300µgm^{-3} and 250µgm^{-3} at Hamilton. A clearer trend could be seen with 24 hour averages which dropped from 113µgm^{-3} at Ngaruawahia to only 16µgm^{-3} at Turangi.

Within Northland PM_{10} concentrations started to increase at around 6pm at Kaitaia and 9pm in Whangarei on the evening of 24 September. They peaked for about 3 hours and had dropped to normal background concentrations by 8am and 11am the following morning respectively. In Auckland concentrations started to increase at around 9pm on the 24th, peaked for about 4 hours and returned to background levels by noon on the 25th. In the Waikato the concentrations of dust started to increase around midnight, on the 24th, in Hamilton but not until 8am on the 25th in Putaruru and Tokoroa. They peaked for approximately 5 hours around Hamilton and had returned to normal by 3pm on the 25th. Further south, in Tokoroa, the dust concentrations peaked for 2 hours and were at background levels by 5pm.

To determine any health effects, from the dust, respiratory admissions to individual hospitals across Northland, Auckland and Waikato for September 2009 were examined. Although PM_{10} has also been associated with cardiovascular disease this relationship was not examined in this instance. Only at the Waikato Hospital in Hamilton was any effect evident. In all other areas no increase in respiratory disease admissions were apparent, either as individual diseases or when grouped to those that may be aggravated by dust inhalation. However on 27 September 2009 (2 days after the dust had passed) there were 10 asthma admissions (diagnostic code J459, asthma unspecified) at The Waikato Hospital. The mean number of admissions for the month was 2.23 per day with a 95% confidence interval of 1.56 to 2.9.

With higher PM_{10} concentrations and a higher population it would be expected that Auckland would be the most likely area to have demonstrated any effect on health of the dust. It is interesting that the only area which seems to have witnessed any effect is the Waikato.

Although dust concentrations were still high in the morning they had peaked in Northland and Auckland before most people were out and about. In the Waikato,

however, particularly south Waikato, the dust concentrations were still at or close to their peak when people started their day on the morning of 25th September. The dust was not particularly apparent, at least in Hamilton, (although some people did witness dust settling on their cars during the day) and the authors do not believe the increased admissions resulted from a psychosomatic effect from seeing the dust.

This small observation supports advice that people, especially those with asthma or other respiratory diseases, should stay in doors when PM_{10} concentrations are high.

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THE NEW ZEALAND MEDICAL JOURNAL
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A new opportunity to eliminate policy incoherence in tobacco control in New Zealand

The Māori Affairs Select Committee is undertaking an inquiry into "the tobacco industry in Aotearoa (New Zealand) and the consequences of tobacco use for Maori".[1] Our submission to this Committee emphasised the primacy of tobacco endgame policies—e.g. to have a sinking lid on tobacco imports so as to achieve a negligible level of smoking (<1% prevalence) by 2020. Nevertheless, we also argued that supplementary incremental steps might be needed if political leaders do not adopt endgame strategies. So with this in mind we briefly reviewed current key tobacco control policy interventions supported by central government.

The interventions we considered are those largely described on the Ministry of Health website and in other documents.[2] We particularly aimed to identify those central government interventions which could be strengthened by reducing the extent to which they were being constrained or countered by other government policies. That is, we classified these interventions as "coherent" where there was no such constraint or conflict, and "incoherent" where a policy was subject to such constraints and conflicts.

From the generated list of 12 intervention areas, we identified at least 4 where some level of policy incoherence appeared to exist (Table 1). Three of these interventions involving incoherence were within the most important four areas of tobacco control (as per current incremental approaches). Besides the specific intervention areas, there is the strategic contradiction of government encouraging and requiring tobacco companies in New Zealand to profit from selling an addictive and highly hazardous product (through the provisions of the Companies Act), while also having the reduction of tobacco use as a government health aim.

Making these current policies more coherent would support tobacco endgame policies. Such endgame policies could include a sinking lid of quotas on tobacco imports, and/or large regular (6-monthly) tax hikes, with both approaches aiming to achieve negligible levels of smoking prevalence within a decade (albeit with home-grown tobacco for personal use still being permitted).

Failing such endgame approaches being supported by the Select Committee, the Committee should at least recommend rapid resolution of these areas of policy incoherence and increase the intensity of all effective tobacco control interventions. This would mean that at least the incremental approaches for ending the tobacco epidemic could proceed more effectively.

Table 1. Tobacco control interventions supported by central government and classification in terms of policy incoherence

Tobacco control intervention	Evidence of policy incoherence	Description of the coherence / incoherence
Top 4 interventions[3]		
Tobacco taxation to raise tobacco prices (to reduce youth uptake and promote quitting)	Yes	Tax/price policy has been inadequately implemented with no real increase in levels of tax since 2001.[4 5] Tax/price policy is also partly undermined by government policy to permit duty free sales of tobacco and to allow for personal supplies of tobacco to be carried into NZ from overseas. This also results in substantial loss of government revenue that could be used for tobacco control.[6] Allowing roll-your-own tobacco to be sold at essentially cheaper prices also undermines the price policy.[7] Using all tobacco tax revenue for general purposes with no dedicated fraction for tobacco control may also partly undermine government arguments that the tax is a health protecting measure. Furthermore, the lack of any dedicated component of tobacco tax used to help smokers quit can be considered ethically problematic.[8]
Complete restrictions on tobacco sponsorship and nearly complete restrictions on tobacco marketing	Yes	The important marketing measures of point-of-sale displays, branding and use of positive imagery and wording on the tobacco packaging itself, continues to be permitted.
Smokefree environments (especially indoor public settings and school premises)	Yes	Allowing smoking in cars with young children present – despite this setting potentially having extremely high levels of second-hand smoke.[9 10] There is also a stark contrast with other in-vehicle laws designed for public safety purposes: seat-belts, child safety restraints and a ban on the use of cell phones when driving. New Zealand is becoming out-of-step with other jurisdictions in this area.[11]
Mass media campaigns	No	There is a coherent policy that links cessation promotion campaigns well with the Quitline service. Nevertheless, these mass media campaigns are still under-funded and do not adequately exploit the synergies of co-interventions (e.g., smokefree law changes[3]), nor use more innovative approaches such as targeting the tobacco industry itself).
Other interventions		
Commerce Commission warning in 2008 on the misleading nature of "light and mild" descriptors	Yes	The government allows the tobacco companies to use other misleading descriptor words (e.g., "smooth") and allows the colour-coding of packs.[12] There is good evidence that many NZ smokers are being mislead by these messages on packaging, and misperceptions that these tobacco products are less harmful to health are common.[13]
Age restrictions on tobacco sales	Possibly	This policy is possibly being undermined by not having a more controlled system around tobacco retailing. For example, if retailer licenses were required to sell tobacco, then these could be withdrawn when there was evidence of illegal sales to youth. Similarly, there could be minimum ages for shop attendants and no sales could be permitted by retailers in close proximity to schools. Permitting point-of-sale tobacco displays and permitting additives to tobacco (eg, sweeteners and flavours) also partly undermines this policy.
Widely available and heavily subsidised pharmacotherapies (eg, NRT)	Possibly	This policy appears fairly coherent and the link with the Quitline provides a distribution system for nicotine replacement therapy (NRT). Nevertheless, the uneven retail availability (compared to tobacco) is possibly problematic (ie, there is no requirement for retailers to sell NRT products if they sell tobacco). Also, a possible form of policy incoherence is the lack of public education to reduce substantial confusion among smokers about nicotine being the major cause of cancer from cigarettes.[14]

Tobacco control intervention	Evidence of policy incoherence	Description of the coherence / incoherence
Requirements for graphic health warnings on tobacco packaging	No	This appears to be a coherent policy but the failure to fully utilise this intervention (which costs taxpayers nothing once the policy is developed) might mean that more tax payer funds need to be spent on mass media campaigns to compensate. For example various design problems with the current NZ warnings exist,[15 16] and in particular they are small compared to those used in some other countries (up to 80% of the front of the packet in Uruguay[17]). Some other countries also use stronger "fear arousal" and "loss-framed" themes in their graphic warnings (eg, Brazil[17]) which are probably more effective.
Funding the Quitline service	No	Policy appears coherent and there is relatively high use of the Quitline by international standards and linkage with information on cigarette packets.[16]
Vending machine controls	No	Policy appears coherent.
Developing "New Zealand Smoking Cessation Guidelines"	No	Policy appears coherent.
ABC approach for smoking cessation (framework and work programme)	No	This Ministry of Health (MoH) policy appears coherent.

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Competing interests: Although we do not consider it a competing interest, for the sake of full transparency we note that some of the authors have undertaken work for health sector agencies working in tobacco control.

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The extent of YouTube videos with smoking and smokefree words

Background—As restrictions on tobacco advertising increase, tobacco companies look for new ways to advertise their products. The internet remains largely unregulated, and one potential internet advertising forum is YouTube. YouTube is a site for the sharing of videos, and has become an extremely popular entertainment destination.[1] YouTube is the biggest player in the online video market, holding almost 43% of the market share.[2] YouTube claim that 52% of 18 to 34 year olds share videos often with friends and colleagues [YouTube data apparently for the USA].[3] A previous study used YouTube to search for the term *smoking*, and found 29325 videos.[1]

Aim—To determine the extent of videos available on YouTube that include smoking, quitting or smokefree terms.

Method—We entered terms related to smoking into the YouTube search engine during late January 2010. The terms used were a sample chosen from an earlier study which looked at adolescents' exposure to tobacco on the internet,[4] and were selected in order to provide a balance between pro-smoking, smokefree and commercial terms. The terms from the earlier study were; "*cigarette*", "*smoker", "tobacco", "cigar", "smoking", "quitting smoking", "cessation", "nicotine", "menthol", "quitline" "British American Tobacco" and "Philip Morris*. In addition, in we searched for the term "*smokefree*".

The number of videos found for each search term was recorded.

Results—For results, see Table 1.

Table 1. Search terms and number of videos found

Smoking terms	Number of videos
"cigarette"	73,400
"smoker"	43,400
"tobacco "	30,300
"cigar"	34,600
"smoking"	188,000
Quitting/smokefree terms	
"quitting smoking"	24,100
"cessation"	3880
"nicotine"	6630
"smokefree"	2640
"quitline"	206
Commercial terms	
"menthol"	1540
"British American Tobacco"	135
"Philip Morris"	1430

Discussion—The amount (if not the proportion) of tobacco content on YouTube appears to be increasing. In contrast to a 2007 search of YouTube using the term *smoking*, which returned 29325 videos,[1] our search returned 188,000. However, the total number of videos on YouTube has also increased, and so the proportion of videos with tobacco content may not have changed.

Caution is needed in interpreting our data, as for instance "smoking" is used for other substances besides tobacco. Many of the same videos would come up when using different search terms, so totals cannot be added.

Policy implications—Website operators can be encouraged to add pro-tobacco imagery or brand content to the material they will remove. YouTube will remove a page if it violates their guidelines.[5] YouTube relies on users to notify them about content which is in breach of its policies. There is currently no tobacco relevant policy.

National law in most countries currently do not require pro-tobacco messages to be removed from the internet. However, the Framework Convention on Tobacco Control (FCTC) requires

an effective ban on tobacco advertising and promotion, including sponsorship (Articles 13.1 and 13.2) unless countries are obstructed by their constitution. This includes internet promotion (Article 13.58). Nearly all countries (168) except the USA have ratified the FCTC.[6]

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In defence of Linda Bryder's book *A History of the 'Unfortunate Experiment' at National Women's Hospital*

Debate over this book has unfortunately centred on criticism of Professor Bryder, but has uniformly failed to address the issues raised by her, that "there never was an 'unfortunate experiment' ". Professor Skegg, Charlotte Paul, Barbara Brookes and now Barbara Heslop....all from Otago University...and R Jones, all avoid comment on the following points.....most of which are taken as direct quotes from the McIndoe Paper, Metro Article or the Cartwright Inquiry Report.

Fact 1; The 1984 McIndoe Paper was a retrospective audit of patients presenting to NWH with abnormal smears during 1955-76. (McIndoe Paper p 452 para 1). It was never a prospective study. Yet this 'audit' was the genesis of the Metro Article with accusations of an 'unfortunate experiment at NWH'.

Fact 2; Groups 1 and 2 were never two separate groups of women 'treated differently'.

To quote the McIndoe paper 1984, page 452 para 7, *"Follow up cytology was used as the basis for division of the patients into 2 groups. Group 1 consisted of patients with normal cytology at follow up at 2 years, whereas group 2 patients had persistent equivocal or abnormal cytology follow up at that time"*.

The two groups were created in 1984, on the basis of outcome of treatment, not on treatment received.

Fact 3; The Metro article states that (p 60, para 5); *"12 of the total number of women had died of invasive cancer, 4 or 0.5% of group 1 women and 8 or 6% of group 2 women who had little or no treatment"*.

Yet McIndoe paper p 454 para 4 states that the 131 women they designated *as group 2, had received principal management by cone excisions in 88, and total hysterectomy in 33.* Because of continuing abnormal smears they had a further 78 cone biopsies and 29 hysterectomies...ie a total of 228 major treatments in 131 women....whom the Metro article claims had 'little or no treatment', as the basis for their claim of an 'Unfortunate Experiment'.

Fact 4; Sandra Coney even owns up to this fundamental mistake in her own words, in her book (The Unfortunate Experiment Penguin 1988), p 17; …. *"..we had made mistakes in the reading of their report..." ..."The first mistake concerned how the authors had divided the women into two groups"....." the key factor in establishing the two groups had actually been whether the women had positive or negative cytology, that is, whether there had been a cure or signs of continuing disease..."*

Yet they continue to claim that 'little or no treatment' was the basis of higher cancer rates.....Dr Jones most recently in NZ Doctor 2009.

This inaccuracy was accepted by the Cartwright Inquiry (p 95 & 150);

"*The McIndoe paper distinguishes between two groups, and 22% of those whose abnormalities were 'untreated' developed invasive cancers*".

This is wrong!

Were they treated adequately?

McIndoe Paper p 453, para 6; *"The 817 patients in group 1 remained clinically and cytologically normal for the four years after the initial biopsy, irrespective of whether or not there was evidence of complete excision of CIS."*

And

p 454 para 4; *"The 131 patients in group 2 continued to produce abnormal cytology consistent with cervical neoplasia irrespective of initial management or the histologic completeness of excision.*

Yet to Quote Dr Jones (NZDoctor 2009); *"The detai*l (of McIndoe paper) shows *women who continued to have abnormal smears, a result of inadequate treatment, had a 25-fold increased risk of cancer."*

This is simply not what his own (McIndoe) paper presents.

Professor G Seber (Univ AK Mathematics Dept) did an independent analysis of the figures from the McIndoe paper, (1990 together with P Mullins) which showed that the division into group 1 or 2 had no relationship to the initial management.

That is, the percentage of hysterectomies is similar for women who ended up in group 1 or group 2. The differences in outcome are related to ongoing disease not their management...the 25-fold increase Jones 'claims' above arises from ongoing disease...not treatment.

The irony of all this, is that the McIndoe Paper actually reinforces Professor Green's clinical perception....that the initial management had no bearing on subsequent disease.

Linda Bryder is Professor of History at Auckland University, with a special interest in medical history. While preparing to write a history of NWH, she had to deal with this 'unfortunate experiment'. She became interested, then intrigued and then finally flabbergasted by where that research led her. Despite claims by her detractors -who almost all seem to be based at Otago University- of superficial, non- professional work, she has done her homework....they just don't like her conclusions.

There never was an unfortunate experiment, 'lambs to the slaughter' and Frankenstein experiments....just a spectacular misrepresentation of the facts.

Helen Overton
GP, New Zealand & Senior Medical House Officer, Denmark

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The legacy of Sir Charles Hercus

Your excerpt from Dr Colquhoun's paper[1] of 1910—100 years ago—showing the complete mystery of the cause of Graves' disease, is a timely reminder of the monumental contribution made by Sir Charles Hercus, Dean of the Otago Medical School. He fostered research by setting up disease-removing full-time research units, manned by hand-picked people with especial research talent.

In this way, Hercus got rid of:

- Hydatids,
- Iodine-deficiency goitre and its associated cretinism,
- Ricketts, by milk in schools,
- Bovine tuberculosis, by Pasteurization of milk

…And discovered the cause of Graves' disease. It is an autoimmune disease and has pioneered discovery of this great category of diseases, which are now ripe for prevention or cure by negating the microbial triggers or selective destruction of the pathogenic forbidden clones.[2]

This is a wonderful legacy from a very great man, whose methods should be copied by today's leaders.

Hercus was descended from Shetland Island Vikings. The Vikings invading Normandy, found the French language better than their own, so they adopted it! They also copied the French in riding horses. This virtue of preserving and adopting anything good that was better than what they had themselves, contrasts strikingly with the Mongols. As well as plaguing the Chinese, the Mongols ruthlessly destroyed anything different from their own, including the great, civilised Muslim Arab Empire set up by Mohammed.[3]

Endowed with Norman virtues, Hercus was fearless in seeking to use all available talented people for getting rid of disease. His greatest asset was Dick Purves, with Honours degrees in Physics and Chemistry, who could take medical problems back to their basis in mathematics, physics or chemistry.

Hercus and Purves abolished New Zealand's goitre endemic, saving countless thyroid operations and the birth of cretinous children. Then with Walter Griesbach and Tom Kennedy, Purves solved pituitary cytology, developed Endocrinology, discovered antithyroid drugs for medical treatment of thyrotoxicosis and set up the research with radioactive iodine that led to discovery that Graves' disease of the thyroid is an autoimmune disease. These were monumental research achievements.

For seeking talent Hercus applied to the medical course the formal university tradition of a Bachelor's degree, based on attendance at lectures followed by passing an examination, then a Master's degree, based on writing a thesis which contributed to knowledge. Accordingly, Hercus had his medical students write a thesis in the 5th year of the course, its great virtue being that the student chose the topic, rather than

having it suggested by a supervisor. This enabled originality to be shown. I wrote a thesis on asthma that showed fledgling research ability and caused Hercus to recruit me as an apprentice to Purves 3 years later.

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Health Practitioners Disciplinary Tribunal – Professional Misconduct (Med08/102P)

Charge

Dr Heather Margaret MacDonald, registered medical practitioner of Auckland (the Doctor) was charged with professional misconduct by a Professional Conduct Committee (PCC).

The charge raised quality of care issues, particularly with regard to the prescribing of morphine, record keeping issues, an allegation of insufficient handover of care to another practitioner, and an allegation of an inappropriate intimate/sexual relationship.

Background

The Doctor and the patient were at all material times employees at NS Ltd. The Doctor was required to provide medical services to fellow employees as well as being the Medical Director at NS Ltd.

During 2005, the Doctor saw the patient on a professional basis. In August, the patient presented with back pain, complicated subsequently by a suspected decompression injury. From 4 January 2006, the Doctor provided the patient with oral and parenteral morphine for the pain.

On 16 February 2006, the patient had an accident on his motorbike, injuring his right foot and exacerbating the back pain. Later investigations showed annular tears to discs in the back.

Over time, the administration of morphine increased. Both controlled drug prescriptions and Practitioners Supply Orders were used to obtain the morphine, in oral and injectable form. The patient was able to self administer. The Doctor used an exercise book to record the patient's dosage, because the Doctor understood there were confidentiality issues when using the MedTech notes at NS Ltd. The patient took significant time off work and this led to employment issues.

The Doctor referred the patient to a muscoskeletal physician for the assessment of back pain, but there was delay in this occurring. The patient was also referred to other specialists – a neurologist, an endocrinologist, a psychiatrist, and a Hospital. On 21 May 2006, the Doctor and the patient attended another Doctor Dr H, with the intention that Dr H would assume the patient's care. However, the Doctor continued to provide medical services.

Some weeks later, the Doctor and the patient entered into an intimate relationship and from early August 2006 were living together. The Doctor continued to prescribe and administer morphine. The Doctor said she was using a morphine reduction template, with the intention of reducing the patient's dosage. The patient was allowed to self administer injectable morphine and to personally sign the record as to administration

The patient became ill in September 2006, and was admitted on two occasions to Hospital. On 16 September 2006 the patient was discharged but was to be re-admitted two days later. The patient died on 17 September 2006. A pathologist found a combined toxicology of morphine, methadone, tramadol and diazepam was the cause of death.

Finding

The Tribunal found the Doctor guilty of professional misconduct.

Reasons for Finding

The Tribunal found the Doctor made multiple and very significant errors of judgement:

- Unacceptable and inappropriate prescribing of morphine in large doses, with a poor attempt at reduction, via an inadequate morphine reduction template.
- Inadequate referrals to clinical specialists, and to staff at a hospital, again over a period of months, which resulted in colleagues being misled.
- The failure to maintain, accurate, complete and/or adequate clinical notes, and records of prescriptions.
- Failure to handover care of the patient adequately.
- Allowed a patient to self administer injectable morphine, and to personally sign a record as to administration.

The Tribunal found the Doctor had an inappropriate sexual relationship with the patient which made a bad situation worse. It resulted in the Doctor being unable to apply her professional judgment objectively and in the interests of a vulnerable patient.

Penalty

The Tribunal ordered that the Doctor be suspended from practice for a period of nine months. The following conditions were imposed on her practice:

- During the period of suspension, the Doctor undertake a Medical Council sexual misconduct assessment (SMAT) and undertake such courses and treatments as the Medical Council may direct; she was further to comply with such conditions as the Medical Council may impose as a result of that programme.
- Following the period of suspension, the Doctor comply with such conditions as the Medical Council may impose upon her, relating to the SMAT assessment.
- The Doctor is to meet the costs of the above assessment and conditions.

Following the period of suspension the Doctor continue to actively participate in the Maintenance of Professional Standards Programme for continuing vocational registration with RNZCGP, involving active regular participation in a peer group.

An order of censure was imposed. The Tribunal expressed its strong disapproval for the conduct which occurred. The Tribunal ordered the Doctor to pay $120,000 in respect of the costs of the hearing.

Appeal

The Doctor appealed all the penalty orders except the order that she be censured. She also contended that the Tribunal ought to have suppressed her name and identifying particulars.

The High Court made the following orders:

- The order for suspension was confirmed but was varied so that it was reduced from nine months to five months.
- The order censuring the Doctor was confirmed.
- The order of costs was reduced from $120,000 to $100,000.
- The condition requiring the Doctor to undertake a SMAT assessment was reversed.
- The condition requiring the Doctor to participate in the Maintenance of Professional Standards Programme was confirmed.

The Doctor was not granted name suppression.

The full decisions relating to the case can be found on the Tribunal web site at www.hpdt.org.nz
Reference No: Med08/102P.

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James Fraser Warwick Macky

FRCS, FRACS, OBE—8 December 1920–9 February 2010—Urologist

Warwick passed away on 9 February in the company of his wife Elizabeth; children Peter, Rebecca, Sarah, Josephine; and his grandchildren.

Warwick was educated at Wanganui Collegiate and gained his medical degree in Melbourne, receiving the Ryan Prize in Surgery in 1943. He was a Captain in the New Zealand Medical Corps 1945–46 and Surgeon Lieutenant in the RNZNVR from 1950–55.

Postgraduate study was undertaken in Melbourne in 1947, gaining his Master of Surgery that year and also being awarded the Gordon Craig Scholarship. Further postgraduate study followed in England where he passed the FRCS in 1947. He specialised in Urology working at the Westminster Hospital.

He was awarded the FRACS in 1950 and throughout his life was dedicated to the advancement of what he referred to as the craft. Warwick returned to New Zealand and was Tutor Specialist in Surgery at Greenlane Hospital 1950–51. He was then appointed Visiting Urologist in Auckland Hospital, becoming senior Urologist and Head of Department until he retired in 1985. He set up the Ormond Clinic with consulting rooms and day stay capability for private Urology at a time when the term day stay had not been coined. The Ormond clinic was a nurturing ground for younger consultants for some 30 years.

Warwick was very proactive in both College and Urological Society affairs. He served on the New Zealand Committee of the College from 1955–1963, College Council 1965–1977 and was Vice President 1975–1977. He was an examiner in Urology 1966–75, New Zealand Censor 1975–77, and a member of the Court of Honour 1981–2010.

Warwick was on the Executive of the Urological Society and President in 1965–1966. He hosted the Society's Annual Conference in Auckland in 1966 and invited David Innes-Williams, a Paediatric Urologist, to not only be guest speaker at the meeting but also to work in the Auckland Unit for 4 weeks.

The international contacts that Warwick developed were legendary and resulted in a steady stream of Urologists coming to Auckland including Willard Goodwin, Frank Hinman, Victor Politano, James Glenn from the USA, Dicky Reese-Mogg, JP Williams, John Blandy, Richard Turner-Warwick, John Wickham and Bill Hendry from the United Kingdom.

He was also a member of the British Association of Urological Surgeons and a member of the Academy of Medical Services of Argentina. Warwick was invited to join G.U Surgeons, an accolade granted to a very select few. He was awarded the OBE for services to Surgery and the community.

Warwick was instrumental in establishing the Marion Davis now the Ernest and Marion Davis Memorial Library which was the original postgraduate medical library in Auckland, and became the home of the College and sister Colleges in Auckland. This was not only a library but also a conference centre.

He was Visiting Medical Officer to the Crippled Children's Society (Dadley Foundation) from 1966–72 and was also co-founder and Director of the Southern Cross Medical Society, serving as Chairman. He was a director of Brightside and Huia hospitals. Outside medicine Warwick was instrumental in the founding of St. Kentigern School and was its Chairman for 37 years.

Warwick had a lifelong love of sailing and his yacht "Ilex" was the place where his children and grandchildren learned to sail, three going on to become world champions. In this as in all his life he was ably supported by his wife Elizabeth.

His was a life of immense service, he was always the gentleman but did have a somewhat impish sense of humour. His wisdom and mentorship has been personally valued over the years and will be sorely missed.

Vale Warwick Macky.

Russell McIlroy FRACS wrote this obituary.

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Graham Aitken Nuffield Trust: Graham Aitken Nuffield Medical Postgraduate Travelling Scholarship

Applications are invited from well qualified New Zealand medical graduates in the 25-35 age group for the above Scholarship.

The purpose of the Scholarship is to provide travel funds to enable New Zealand graduates to further their clinical medical training and research interests in the United Kingdom.

The Scholarship will provide a travel contribution of up to the equivalent of three return air fares to the U.K., together with allowances amounting to not less than $5,000, based on the applicant's circumstances. The Scholarship has a current maximum of $20,000

Candidates for the Scholarship must submit a training or research programme for approval together with the name of a person in the U.K. who will provide salary and facilities.

For further information please consult the Deans of the Schools of Medicine or write to

Professor A.D.Campbell, Honorary Secretary, Managing Trustees, Graham Aitken Nuffield Trust, C/- Department of Chemistry, University of Otago, P.O. Box 56, Dunedin. adcamp@otago.net.nz

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