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Obesity and diabetes: questions remain but action should not be delayed

Jim Mann, Kirsten McAuley, Rachael Taylor

That obesity and its comorbidities, especially type 2 diabetes (T2DM), have reached epidemic proportions in New Zealand and globally, has been established beyond doubt. This issue of the *Journal* includes papers that highlight some of the many outstanding questions that must be answered if there is to be any serious hope of stemming the tide of these epidemic diseases of the 21st Century.

Rush and colleagues[1] have carried out studies of body composition in young New Zealand European, Pacific Island, and Asian Indian men and have confirmed both that the relationship between percentage body fat and body mass index (BMI) is different among the three groups (possibly due to differences in muscularity), and that Asian Indians have more abdominal fat that the other ethnic groups. The latter observation might explain the high rates of cardiovascular disease amongst people of Indian descent.

The Rush study confirms earlier observations that people of Pacific Island descent have a lower percentage body fat than Europeans for any given BMI and that the reverse applies for Indians. Hence, the suggestion that different BMI cut-offs for the definitions of overweight and obesity from those applied to European populations, should apply to Pacific Island and Indian populations.

While this may be appropriate if one wishes to compare body composition in different population groups, it is important to remember the original, and still clinically the most important, reason for the introduction of BMI cut-offs to define the categories of overweight and obesity—the identification of individuals who are at increased risk of the comorbidities associated with excess adiposity.

The cut-offs were principally determined from prospective epidemiological studies in predominantly European populations.[2] Given that for the relatively common comorbidities, there is a gradient of risk with increasing adiposity, the levels used to define overweight and obesity are inevitably somewhat arbitrary. Were it true that a given level of adiposity was associated with a comparable level of risk in all populations, it would be appropriate to adjust the categories according to degree of adiposity. However as there may well be inherent differences in risk in different racial groups it is potentially misleading, at least from a clinical point of view, to suggest the redistribution of BMI cut-offs on the basis of percentage or amount of body fat as measured by dual-energy X-ray absorptiometry.

Indeed, some evidence suggests that susceptibility amongst different racial groups to adverse health effects varies—even if the degree of adiposity is similar. A pilot study from Dunedin has suggested that for comparable levels of adiposity Maori have a greater degree of insulin resistance than New Zealanders of European descent.[3] If confirmed (a large study is currently underway), this suggests that it would be inappropriate to recommend higher BMI cut-offs for the definitions of overweight and

obesity in Maori, despite the fact that for any given BMI Maori may have greater lean body mass and less fat mass than Europeans.

While it might be inappropriate to use higher BMI cut-offs for Maori, it may well be appropriate to suggest lower cut-offs for Asian Indians given their well recognised high cardiovascular risk at lower levels of BMI. In general, Asian Indians have greater levels of total and central fat than Europeans for a similar BMI. [2] Ideally, this issue should be resolved by prospective observations on non-European populations for whom body composition data are available. However as such data are unlikely to become available in the near future, further body composition studies of Asian and Pacific Island populations should be undertaken in conjunction with measurements of associated comorbidities, especially those related to abnormal carbohydrate metabolism, which are particularly relevant in New Zealand.

Thus, it seems appropriate to retain current cut-offs at present, while acknowledging that Asian Indians may be at increased risk of cardiovascular disease even if their BMIs are within the currently defined normal range.

Another issue relating to measurement is highlighted in the paper by Hohepa and colleagues.[4] There is no doubt that physical inactivity is a major contributing factor to the obesity epidemic, and that increasing physical activity facilitates weight loss and weight maintenance and increases insulin sensitivity.[5] Indeed, promotion of physical activity is a pivotal component of public health messages as well as management of overweight and obese individuals.

The New Zealand Government intends to expend substantial sums of money on a national programme aimed at increasing physical activity in schools. However, without appropriate instruments for measuring activity, it will not be possible to accurately relate trends in obesity with trends in physical activity, and evaluation of such interventions will be impossible. Thus, the call made by Hohepa and colleagues for accurate measurement tools is strongly endorsed. There is also a need to establish with greater certainty the level and type of physical activity, which is most likely to facilitate reduction of overweight and obesity and their comorbidities.

Current advice in New Zealand centres around the recommendation to have at least 30 minutes of physical activity most days of the week without any clear indication of the level of activity. There has also been the suggestion that this amount of activity can be achieved by having several shorter periods of activity (‘snacktivity’) which may be added together to achieve the required amount.

While this may be a useful approach to initiate physical activity in previously inactive individuals, and while any level of activity is undoubtedly better than none, there is evidence that longer and more intensive levels of activity than currently recommended are required to improve insulin sensitivity in insulin-resistant individuals.[6] Furthermore, a combination of endurance and resistance training may be preferable to larger amounts of only one type of activity.[7]

Until further evidence is available regarding optimal type and amount of exercise, it may be appropriate to indicate (in both public health messages as well as in advice to individual patients) that current recommendations represent minimal requirements and that whenever possible the level of activity should be sufficient to raise the pulse rate.

Ni Mhurchu and colleagues draw attention to a somewhat neglected set of comorbidities associated with obesity.[8] They report on the impaired health-related quality of life (HRQOL) experienced by overweight and obese individuals. Of particular importance is the fact that HRQOL measures did not improve appreciably with weight loss. However only 23 of the 250 participants lost more than 5% of their baseline weight. Thus the overall finding is perhaps not surprising. It seems likely, therefore, that a greater than 5% weight loss is necessary both to reduce the risk of progression from impaired glucose tolerance (IGT) to T2DM, and probably also other comorbidities of obesity, as well as improve quality of life.

Much attention has been focussed on the need to prevent and treat obesity in childhood, and indeed this must be one of the greatest health priorities in New Zealand. However the paper by Rose and colleagues reminds us that adults too require attention.[9] They found that more than half of over 3,000 women screened are at 'high risk' of developing T2DM. Such statistics along with observations such as those by Tipene-Leach and colleagues[10] showing that a comparable proportion of adult Maori living in a rural environment have T2DM, impaired fasting glucose, IGT, or insulin resistance endorse the New Zealand guidelines for the detection and management of cardiovascular risk published in 2003.[11] All adult New Zealanders should be screened for T2DM and pre-diabetic states by age 45 (male) and 55 (female), with high-risk individuals screened earlier.

With current health care costs relating to T2DM approaching NZ$400 million and expected to rise to more than NZ$1,000 million by 2021, and evidence showing that appreciable risk reduction by lifestyle modification is achievable, urgent implementation of lifestyle modification programmes is essential.

The questions posed by the papers presented here need not delay the implementation of existing knowledge. Perhaps the greatest issue remaining to be solved is how to persuade at-risk individuals and populations to make the necessary changes.

Author information: Jim I Mann, Professor in Human Nutrition and Medicine; Kirsten A McAuley, Senior Research Fellow; Rachael Taylor, Lecturer in Human Nutrition; Departments of Human Nutrition and Medicine and Edgar National Centre for Diabetes Research, University of Otago, Dunedin

Correspondence: Professor Jim Mann, Department of Human Nutrition, University of Otago, PO Box 56, Dunedin. Fax: (03) 479 7958; email: jim.mann@stonebow.otago.ac.nz

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