



What proportion of cancer is due to obesity?

Tony Blakely, Diana Sarfati, Caroline Shaw

The evidence linking overweight and obesity to the incidence of a range of cancers has strengthened over the last couple of years. A recent review of 221 datasets published in *The Lancet* found that there were associations between increasing body mass index (BMI) and cancers of oesophagus, kidney, thyroid, and colon, as well as leukaemia, multiple myeloma, and non-Hodgkin's lymphoma.¹ In addition, increasing BMI was associated with rectal cancer and malignant melanoma among men, and cancer of the endometrium, postmenopausal breast, and gall bladder among women. These findings are consistent with, and extend, another recent major review.²

Rates of obesity and overweight increased in New Zealand during the 1980s and 1990s,³ but may have now reached a plateau.⁴ The distribution and pattern of BMI varies between population groups (Table 1).

Table 1. Proportionate BMI category distribution, within sex by ethnic groups

| BMI (kg÷m ²). | Males | | | Females | | |
|---------------------------|-------|-------|---------|---------|-------|---------|
| | Total | Māori | Pacific | Total | Māori | Pacific |
| Non-overweight* | 38.0% | 35.8% | 18.1% | 50.2% | 42.2% | 17.3% |
| Overweight † | 42.1% | 37.2% | 43.9% | 27.7% | 31.3% | 34.8% |
| Obese ‡ | 19.9% | 27.0% | 38.0% | 22.1% | 26.5% | 47.8% |
| | 100% | 100% | 100% | 100% | 100% | 100% |

Source: 2002/03 Health survey estimates from Tracking the Obesity Epidemic Report.³

* BMI <25.0 for European/Other, <26.0 for Māori and Pacific.

† BMI 25.0 to 29.9 for European/Other, 26.0 to 31.9 for Māori and Pacific.

‡ BMI ≥30.0 for European/Other, ≥32.0 for Māori and Pacific.

Men are more likely to be overweight than women, but women are more likely to be obese. Māori and Pacific are more likely to be obese than European/Other, and there is an evolving socioeconomic gradient in BMI distribution (those in lower socioeconomic groups more likely to be overweight or obese) which is stronger for women, but increasingly evident for men.

Similarly, the burden of cancer is not evenly distributed in New Zealand. Compared with non-Māori, Māori are both more likely to be diagnosed with cancer and to die from it.⁵⁻⁷ This is true both for all cancers combined, and for many specific cancer sites.

Inequalities in cancer mortality rates between Māori and non-Māori have increased throughout the 1980s and 90s. Pacific people also have higher incidence and mortality, and there is a gradient of increasing incidence of cancer with increasing deprivation. Those in the most deprived quintile have at least a 25% higher rate of cancer incidence than those in the least deprived group.⁸

These three pieces of evidence combine to suggest that increasing BMI currently has an impact on cancer incidence in New Zealand, that this impact will increase over time, and (in addition) it will have an increasing role in driving disparities in cancer incidence between ethnic and socioeconomic groups in the next generations.

In this editorial we synthesise the New Zealand data on obesity and cancer rates with international estimates of the relative risk of cancer by BMI or overweight/obesity category. Our objective is to estimate the contribution of obesity to cancer for the total population, and provide indicative estimates by ethnic group. It must be emphasised that our estimates are limited by the quality of data we have available and the assumptions we are required to make. For example, the relative risk estimates are from overseas studies conducted in the past— it is not guaranteed that they will apply to ethnic groups in New Zealand into the future.

The source reference for these relative risks¹ attempted to rule out confounding by important factors such as smoking, but there may be residual confounding by other lifestyle risk factors that were variously modelled (e.g. physical activity, hormone replacement therapy).

We use the population attributable risk percent (PAR%, also known as population attributable fraction) as an indicative estimate. Our calculations assume complete reversibility of risk, and that everyone moves to a BMI of less than 25 (or less than 26 for Māori or Pacific)—obviously not a realistic policy outcome in the foreseeable future. The PAR% is, therefore, the percentage reduction in the cancer incidence under such a scenario.

Finally, we have used overall population statistic inputs and rates—had we been able to use all of relative risks, percentage obesity, and cancer rates by narrow age groups, and a linear modelling of BMI rather than categorical, the results might differ modestly. These caveats issued, we still believe it is useful to gain an *indicative* understanding of the possible impact of obesity on cancer to inform policy decisions.

The first column of Table 2 shows the relative risk estimates by cancer site for a 5-unit increase in BMI from Renehan et al.¹ The PAR% are shown by sex, for the total population, and for Māori and Pacific people.

Obesity appears to have a substantial contribution to the incidence of oesophageal, colon (male), gallbladder (female), endometrial, renal, and thyroid cancer with PAR% of 15% or more. This means that if the total population moved to a BMI of less than 25 (or less than 26 for Māori or Pacific), we might expect the incidence of these cancers to reduce by at least 15%.

For the total population we estimated the PAR% for all cancers combined as 5% for males and 4% for females. For Māori, the PAR% was a little less at 4% for Māori males, due to Māori male cancers being more likely to be cancers that are not weight related, and a little more for Māori females (5%). Any contribution of overweight and obesity to total cancer inequalities between Māori and non-Māori in the future will therefore depend on both overweight and obesity distributions by ethnic group, and the changing 'background' incidence and relative shares of cancers that are less related to weight—especially lung cancer.

Table 2. Relative risk of developing cancer for each 5 unit increase in BMI, and PAR% of each cancer due to having a BMI \geq 25 for European/other or \geq 26 for Māori and Pacific

| Cancer type | Male | PAR% [†] | | | Females | PAR% [†] | | |
|---|------|-------------------|-------|---------|---------|-------------------|-------|---------|
| | RR * | Total | Māori | Pacific | RR * | Total | Māori | Pacific |
| Oesophageal adenocarcinoma | 1.52 | 32% | 35% | 42% | 1.51 | 30% | 33% | 44% |
| Colon | 1.24 | 17% | 19% | 24% | 1.09 | 6% | 7% | 11% |
| Rectal | 1.09 | 7% | 8% | 10% | 1.02 | 1% | 2% | 3% |
| Gall bladder | 1.09 | 7% | 8% | 10% | 1.59 | 33% | 37% | 48% |
| Pancreas | 1.07 | 6% | 6% | 8% | 1.12 | 8% | 10% | 9% |
| Melanoma | 1.17 | 13% | 14% | 18% | 0.96 | -3% | -3% | -6% |
| Postmenopausal breast | | | | | 1.12 | 8% | 10% | 14% |
| Endometrial | | | | | 1.59 | 33% | 37% | 48% |
| Renal | 1.24 | 17% | 19% | 24% | 1.34 | 21% | 24% | 33% |
| Thyroid | 1.33 | 23% | 25% | 30% | 1.14 | 10% | 11% | 16% |
| Mulltiple myeloma | 1.11 | 8% | 9% | 12% | 1.11 | 8% | 9% | 13% |
| Non-Hodgkin's lymphoma | 1.06 | 5% | 5% | 7% | 1.07 | 5% | 6% | 9% |
| Leukaemia | 1.08 | 6% | 7% | 9% | 1.17 | 11% | 13% | 19% |
| Weighted [‡] PAR% across cancers | | 5% | 4% | - | | 4% | 5% | - |
| Ischaemic heart disease and stroke [#] | 1.29 | 20% | 22% | 28% | 1.29 | 19% | 21% | 30% |

* Relative risks, sourced from Renehan et al (2008).

[†] Population attributable risk percents, assuming: full risk reversibility as given by the RRs; a counterfactual scenario where everyone had a BMI less than 25 for European/Other or less than 26.0 for Māori and Pacific; that the difference in average BMI between each of the three categories is equal to 5.0, allowing a simple PAR% calculation with the given RRs. This latter assumption is relatively robust, based on distributional data shown in the Tracking the Obesity Epidemic Report³ (workings available from authors on request).

[‡] We used the incidence rates as given in the Unequal Impact Report⁶, Tables 6.2 and 6.3, for each cancer site divided by the total cancer incidence.

[#] Using a common relative risk estimate of 0.95 for each unit *decrease* in BMI from the GBD Comparative Risk Assessment project⁹, which translates into a 1.29 relative risk for a five-unit increase in BMI.

We might anticipate that if the distribution of cancers affecting Māori and Pacific proportionately shift to more obesity-related cancers (due to reductions in lung, cervical, stomach, hepatocellular, and other cancers) then the PAR% will increase.

By way of comparison, we also estimated the PAR% of being overweight or obese (compared to a non-overweight BMI) for ischaemic heart disease and ischaemic stroke, using relative risk estimates from the WHO comparative risk assessment project.⁹

The PAR% were 20% and 19% for total males and females, and higher for Māori and Pacific. It must be noted, however, that it is difficult to disentangle BMI from physical activity and dietary factors such as saturated fat, or fruit and vegetables, in the diet as a causal and unconfounded risk factor for cardiovascular disease (CVD). Moreover any progress to the counterfactual (total population with BMI less than 25

or 26) would be likely to involve simultaneous changes in these other associated risk factors.

Such arguments would also apply to cancers, but not to the same extent as for CVD. So while this PAR% for CVD should also be treated as indicative, it does suggest that the impact of overweight or obesity is considerably larger on the incidence of cardiovascular disease than on cancer incidence.

It is biologically plausible that being overweight or obese is a causal factor for some cancers, independent of other risk factors. There are a number of possible mechanisms.¹ First, insulin-like growth factors increase the risk of some cancers. Second, levels of sex steroids that increase cancer risk (e.g. oestradiol) vary with weight. This is likely to be at least part of the explanation for increased risk of breast cancer among obese (postmenopausal) women. Third, adiponectin secreted from visceral fat adipocytes, with blood levels inversely proportional to BMI, are probably antiangiogenic and anti-inflammatory. Fourth, there may be specific mechanical risks associated with weight, for example reflux is a risk factor for oesophageal adenocarcinoma.

It is important not to feed a climate of fear and hysteria about obesity, but equally we need to consider its likely impact on health and disease. It is also imperative to consider the obesogenicity of our environments and culture, not just victim blame.

It seems likely that overweight and obesity will be increasingly responsible in the future for both disease burden in New Zealand, and also for an increasing contribution to ethnic and socioeconomic inequalities in health. Cancer is involved in that mix, along with CVD and diabetes.

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