Commentary: Body weight and mortality in the late 19th century

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On 18 June 1908, Brandreth Symonds presented the results of an extraordinarily large prospective study of weight and mortality to the annual meeting of the Medical Society of New Jersey. The analysis related the overall and cause-specific mortality among thousands of men and women accepted as life-insurance policyholders to weight and height as measured when they had applied for the policy, and was the product of a pioneering collaboration between insurance companies across the USA and Canada. Although the analysis is now over a century old, there is much in it that seems presciently relevant to the current time.

Early evidence from life-insurance mortality studies

During the first half of the 20th century, life-insurance mortality studies provided much better evidence about some causes of premature death than mainstream epidemiological studies did. Large actuarial studies investigated the effects on mortality not only of weight, but also of alcohol, smoking, glycosuria and blood pressure. The main motivating force behind these studies was not the improvement of public health, but insurance company profits: by more accurately predicting who would die early and thus be expensive, a company could appropriately calibrate premiums and contain costs. But the studies depended crucially on the work not only of actuaries but also of medical doctors—including Brandreth Symonds, who since 1903 had been Medical Director of the Mutual Life Insurance Company of New York—and, thankfully, some of the results were published in medical journals, to the potential boon of patients and the public. The financial incentive to collect detailed information on applicants, together with the newly introduced technique of storing information on punch cards, soon produced rafts of analysable prospective data on a scale that would not be seen in mainstream medical research for another half century.

Signal features of the 1908 analysis

By modern standards, the transcript of Symonds’ 1908 presentation lacks the detail necessary to make confident aetiological inferences, but it does describe many features that today would be considered serious strengths for assessing cause and effect. The first was genuinely large scale: there were 1499 male deaths in the most obese group alone, which is a high number even by the standards of recent epidemiological studies. Second was a careful dissection of results for weight not only by height and by sex but also by age. This dealt with potential confounding by these variables, while also informatively displaying modification of associations by them. Third, the results may have been unusually free of contamination by the effects of pre-existing disease on weight at the time of measurement (i.e. reverse causality), because
of the powerful financial incentive for the insurance company to reject at the outset anyone with even a hint of disease. (Although people with established disease were sometimes offered policies at higher premiums, such people were routinely excluded from mortality analyses.) A detailed personal and family medical history was taken, and a wide-ranging physical examination was done; any family history, symptom or sign suggestive of underlying disease—including very low weight for height, recent weight loss, or chronic cough—could be enough for the person to be refused an ordinary policy. Not many modern prospective studies are so rigorous in excluding people who appear to be in less than ideal health at the start. Fourth, the results may also have been less contaminated by the effects of cigarette smoking, since that habit was not yet widespread, and few cigarette smokers had been smoking for long. Mass production of cigarettes became possible only in the 1880s and, by 1910, per capita consumption in the USA was, though rapidly increasing, still less than a 20th of the level that would be reached in the 1970s. Other forms of tobacco were consumed, but not as heavily or as frequently as cigarettes later were, nor, probably, with the depth of inhalation into the lungs. Reverse causality and confounding by smoking are two problems that plague modern epidemiological studies of weight and mortality, and both result in overestimation of death rates among lean people.

**Half-way between Quetelet and Keys**

Modern epidemiological studies of weight more often use the ratio of weight to height squared than, as Symonds did, weight for a given height, but the two approaches are approximately equivalent. Symonds’ 1908 presentation was, as it happens, delivered almost at the half-way mark between Adolph Quetelet’s 1842 description in English of the empirical principle that underpins this equivalency, and Ancel Keys’ influential 1972 paper that dubbed the ratio ‘body mass index’ (BMI). Quetelet’s 1842 publication was the translation of an influential work he had first published 7 years earlier in Paris. In the translation, he observed that ‘the weight of developed persons, of different heights, is nearly as the square of the stature’; a consequence, he surmized, of men and women not increasing equally in all dimensions (noting that if they did, weight would be as the cube of height). Quetelet did not, though, much use the ratio weight to height squared, if at all, whereas he did use liberally the simpler ratio of weight to height; nonetheless, the ratio of weight to height squared has sometimes been called the Quetelet index. However, that ratio is now more commonly known by the name coined in Keys’ 1972 paper.

This article also demonstrated that BMI is generally a better measure of adiposity than other simple weight-height indices are, and although it was not the first paper to do so, it was probably the one that had the most decisive influence.

**All-cause mortality in the late 19th and late 20th centuries**

The greater part of the mortality results in Symonds’ presentation concern overall mortality (i.e. all-cause mortality). However, overall mortality is not an entity that can in itself be directly affected by weight, as it is merely the sum of the specific diseases and injuries that kill people (which may be directly affected by weight). In places, and times, with different mortality patterns, the associations of overall mortality with weight for a given height (or with BMI) could thus be different. The association in Symonds’ 1908 report might well approximate the real association among North American men in the late 19th century (there was little quantitative information for women), so some relevant mortality rate ratios have been abstracted from the report and listed in Table 1. At age 65 years, men who weighed 20–30% more than the ‘standard’ for age and height had an overall mortality rate that was 20–30% higher than the rate for men whose weight was near the standard (mortality rate ratio 1.26). The standard weight was defined as the average weight at each height and age as reported in an 1897 analysis of a similar population; and after applying the corrections for clothing and shoes that Symonds implies would be appropriate (see Table 1 footnote), it can be calculated that the standard weight at age 65 years would have corresponded to a BMI of ~25 kg/m². The mean BMI in those whose weight was 20–30% greater than the standard could thus have been ~30 kg/m² (Table 1). Therefore, at age 65 years, a BMI of 30 compared with 25 kg/m² might have been associated with 20–30% higher overall mortality. How does this compare with more recent estimates for North America? A recent analysis from the Prospective Studies Collaboration (PSC)—a collaboration of 57 cohort studies with data on BMI, including 15 North American studies—found that 5 kg/m² higher BMI (25 kg/m² compared with 20 kg/m²) was associated with 32% (95% confidence interval 29–36%) higher overall mortality among men (mean age at death across both sexes was 67 years). In the overweight and obese range, the relative increase in mortality for 5 kg/m² higher BMI during the late 20th century may thus have been similar to, or slightly greater than, that in the late 19th century.

However, even if this was so, the absolute increase in mortality could—assuming that the relative risks in the actuarial and PSC analyses were causal—have been greater in the late 19th century, because the overall mortality rates were much greater then.
Although mortality rates are not available for the whole of the USA before the 1930s, US age-specific mortality rates in 1900 might not have been greatly different from those in England and Wales, where 54 out of each 1000 men aged 60–69 years (and 44 out of each 1000 women) died in that year, compared with 18 (and 11) in 2000 (Figure 1). If US men aged 60–69 years also had an overall mortality rate of 54 per 1000 in 1900, then men with a BMI 5 kg/m\(^2\) higher than the average would, assuming causality, have had a mortality rate that was higher than the average by \(20\%\)–\(30\%\). That implies an excess of 11–16 deaths per year for each 1000 men who had a BMI 5 kg/m\(^2\) above the average (by 2000, the average BMI at this age\(^{1}\) had reached 28–29 kg/m\(^2\)).

Thus, 100 years earlier the absolute risks associated with a given absolute higher level of BMI (in the overweight or obese range) could have been much greater, even if the relative risks were similar or a bit less.

### Cause-specific mortality in the late 19th and early 21st centuries

Only a lesser part of the mortality results were for specific diseases and injuries, and though the analysis has some frustrating defects, these results are especially interesting because they show glimmers of relationships that future studies would indicate are likely to be causal. Among the analytical shortcomings, Symonds merely gives the percentage of total deaths

### Table 1 Mortality rate ratios at different weights and ages among North American male life-insurance policyholders in the late 19th century

<table>
<thead>
<tr>
<th>Age</th>
<th>Weight, as percentage of standard weight for height(^{a})</th>
<th>Approximate mean BMI(^b) (kg/m(^2))</th>
<th>Mortality rate ratio(^c)</th>
</tr>
</thead>
<tbody>
<tr>
<td>40 years</td>
<td>100%</td>
<td>24</td>
<td>1.00</td>
</tr>
<tr>
<td></td>
<td>120–130%</td>
<td>(30)</td>
<td>1.12</td>
</tr>
<tr>
<td></td>
<td>&gt;130%</td>
<td>(33)</td>
<td>1.30</td>
</tr>
<tr>
<td>65 years</td>
<td>100%</td>
<td>25</td>
<td>1.00</td>
</tr>
<tr>
<td></td>
<td>120–130%</td>
<td>(31)</td>
<td>1.26</td>
</tr>
<tr>
<td></td>
<td>&gt;130%</td>
<td>(34)</td>
<td>1.72</td>
</tr>
</tbody>
</table>

\(^{a}\)The standard weight for a given height and age was defined as the average weight of policyholding men of that height and age, as reported in table II of the Symonds paper\(^1\) (which, in turn, was based on an earlier, 1897, analysis). The weights with the lowest subsequent mortality were said to be generally 5% below the standard.

\(^{b}\)The mean BMI of 40-year olds at the standard weight (100%) was calculated as the average of the mean BMI for 35–39- and 40–44-year olds, using the height and weight values in table II of the Symonds paper.\(^1\) The mean BMI for each of these quinquennia was calculated as the unweighted mean BMI across 16 reported heights (5 feet 0 inches to 6 feet 3 inches), after first subtracting 6.3% of weight and 1.125 inches from height to take account of clothing and shoes (see page 161 of that article). Analogous procedures were used to calculate the mean BMI of 65-year olds at the standard weight. For 120–130% and >130% of standard weight, the mean BMI was estimated by assuming a normal distribution with mean BMI equal to the BMI calculated for the standard weight, and a standard deviation of 4 kg/m\(^2\). Parentheses indicate added levels of uncertainty.

\(^{c}\)As reported for 5 feet 6 inch-tall 40-year olds and for 5 feet 11 inch-tall 65-year olds. The mortality rate ratios are, however, likely to have been generalizable to other heights. [The mortality rate ratio for the heaviest 65-year olds (1.72) may have been based on particularly small numbers of men, and therefore might be inexact.]
attributable to each specific disease or injury rather than (as he did for overall mortality) the ratio of observed to expected deaths in each weight category; and the central comparison group was the entire male mortality experience of (just) one insurance company, and so would have included the mortality experience both of those defined as overweight (>120% of standard weight) and of those defined as underweight (<80% of the standard). But despite these handicaps, some illuminating comparisons are possible. The proportion of deaths in overweights relative to that in underweights was 5.3 for cirrhosis (i.e. cirrhosis formed a 5.3 times higher proportion of total deaths for overweights than it did for underweights), 5.2 for diabetes, 2.6 for ‘diseases of the skin and cellular tissue’, 2.1 both for ‘general paralysis and other forms of mental alienation’ (much, no doubt, due to syphilis) and for ‘Bright’s disease and nephritis’, 1.7 for ‘cerebral congestion and hemorrhage – cerebral softening’, 1.5 for ‘organic diseases of the heart’, and 1.3 for suicide. A century later, a wealth of epidemiological, clinical and laboratory evidence shows that obesity can be considered a cause of heart disease,12,16,22 stroke,12,23 diabetes22 and kidney disease.12,24 Cirrhosis ought now to be added to that list:12 independently of alcohol consumption, obesity can cause persistently heavy fluxes of free fatty acids to the liver, causing triglyceride deposition and, eventually, fibrotic scarring and dysfunction.25

There were a few causes of death in the 1908 analysis that formed a smaller proportion of deaths among overweights than underweights, most notably tuberculosis, respiratory diseases and cancer. Symonds suggested that overweight gives some protection against death by tuberculosis, but appreciated that the evidence is heavily muddied by the weight loss caused by tuberculosis itself. The inverse association for total cancer mortality differed qualitatively from a (weak) positive association in the PSC12 a century later, but the types of cancer common in each of those two eras would have been substantially different.

The causal links between obesity and ischaemic heart disease are now especially clear:22,26 obesity raises blood pressure, disturbs blood cholesterol and causes diabetes, and each of these can cause atherosclerosis. Apart from noting (as he did) the higher diabetes mortality among overweight men, Symonds could not have really understood these causal mechanisms in 1908. The routine bedside measurement of blood pressure was only then beginning to become widespread following Korotkov’s description 3 years previously of the auscultatory method of measuring blood pressure.27 Symonds himself helped to establish the link between weight and blood pressure in a 1923 report that analysed cross-sectional actuarial data on about 150 000 men.7 In it, higher weight was associated with higher systolic blood pressure (SBP) throughout the entire weight range, and 10 kg/m² higher BMI (estimated from average weights and heights in that report) seems to have been associated with ~6–7 mmHg higher SBP, which is only slightly weaker than in the PSC12 a century later. The central role of blood cholesterol in atherosclerosis was not widely accepted until the second half of the 20th century.28

One of the inescapable limitations of Symonds’ analysis of specific diseases was his reliance on disease rubrics that are now—for good reason—obsolete. The second most common cause of death, nearly as frequent as tuberculosis, was ‘cerebral congestion and hemorrhage – cerebral softening’, which we can take to mean stroke. Between the 1760s and early 1900s, stroke was considered by many to be a consequence of congested brain vessels29 (i.e. distended cerebral vessels, excessive cerebral vascularity)—a theory that could sometimes have had tragic consequences, as it suggested such risky treatments for stroke as venesection and carotid ligation.29,30 ‘Cerebral congestion and hemorrhage – cerebral softening’ may seem a historical oddity to us; however, in a small but telling twist, Symonds felt precisely the same about diagnoses in John Graunt’s ‘Bills of Mortality’ (1662), some two-and-a-half centuries before his own time, remarking in a manual for trainee medical assessors10 that, ‘The causes of death stated in these bills of mortality are very quaint and indicative of the state of medicine at the time. Who would recognise croup as “the rising of the lights”?’. In a similar vein, how many of today’s conventional diagnoses will seem quaint—or dangerous—by, for example, the second centenary of Symonds’ presentation, in 2108?

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References
Commentary: The quest for weight standards

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Symonds’ article1 presents a table of standard weights for height for men that were derived from 1897 life insurance data from the USA and Canada. As has been the case with other life insurance height–weight tables,2–8 it is not completely clear how Symonds’ table was derived and exactly what his standards represent. As noted by Rothstein,9 the goals of life insurance companies tend to be somewhat different to those of health researchers and include finding accurately and inexpensively measured factors with good predictive values rather than looking for causal factors. Characteristics such as sex, age, occupation and possibly even body weight, may be good predictors without being in themselves causes of mortality. For purposes of assessing causality in health research, life insurance

11 Human Mortality Database. University of California, Berkeley (USA), and Max Planck Institute for Demographic Research (Germany). http://www.mortality.org or http://www.humanmortality.de (12 December 2009, date last accessed).